Many Faces of Migraine

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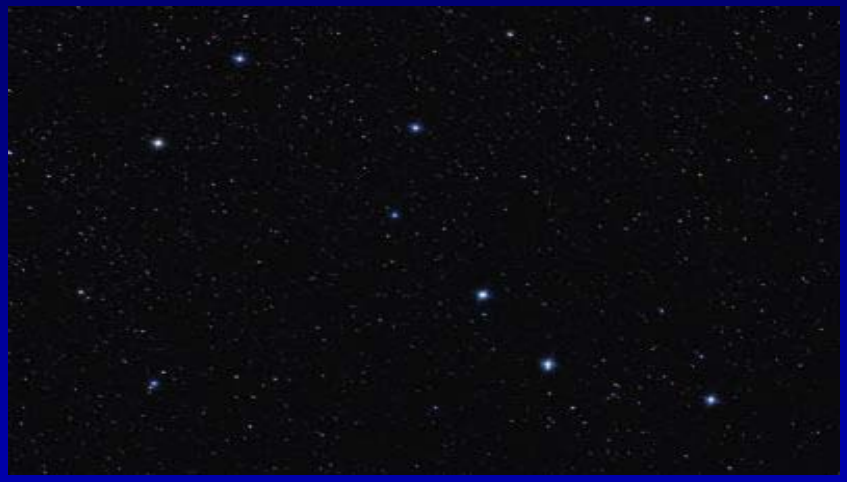
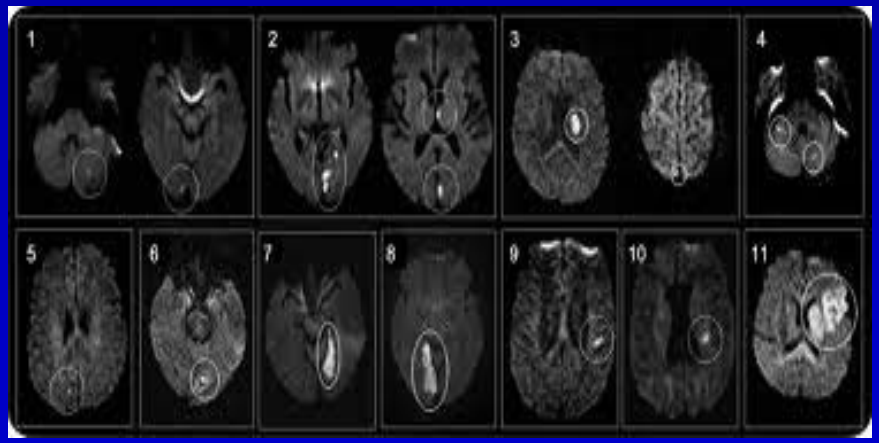
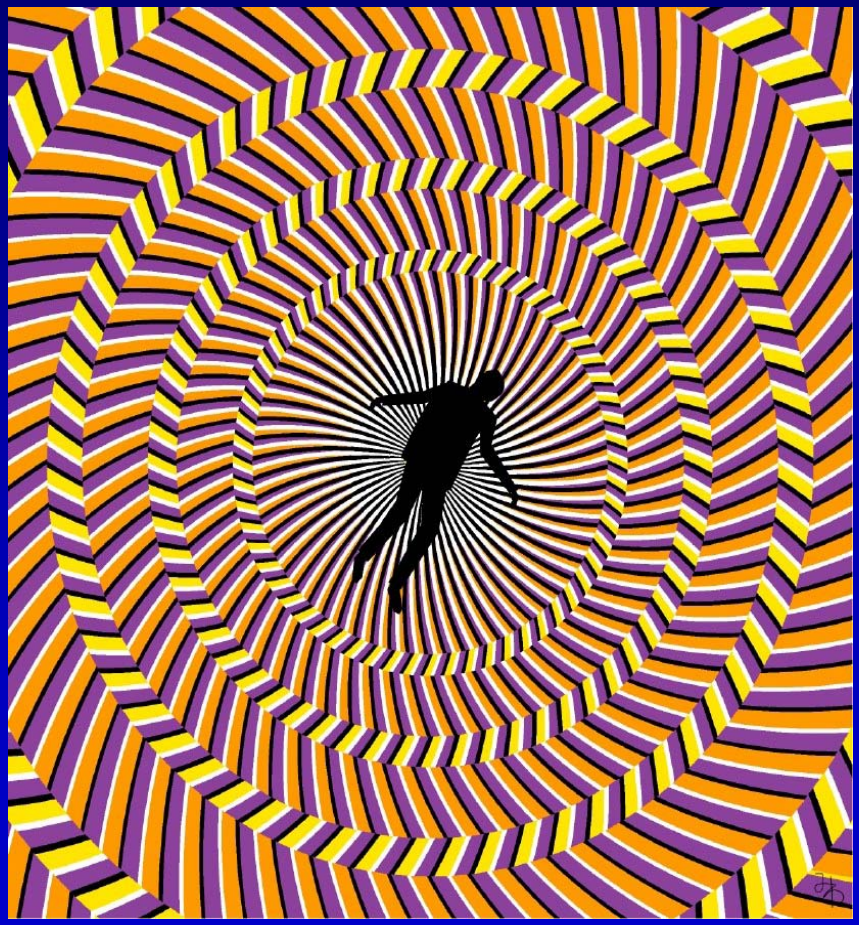
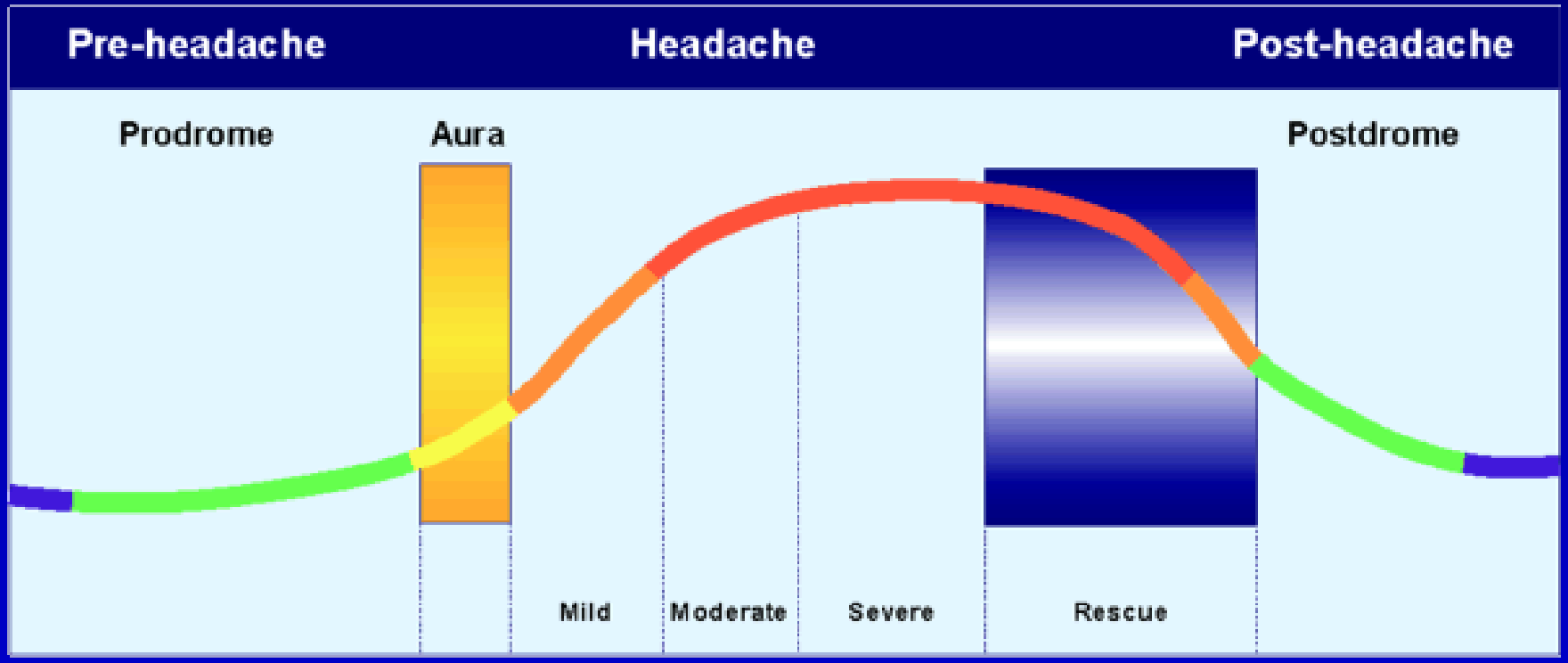






Classical migraine

Migraine phenomena



Migraine Syndrome

- Premonitory phase
  - Mental-Hypothalamic-Autonomic syndrome
- Aura phase
  - Neurological syndrome
- Headache phase
  - Migration and increase in severity of headache
  - Associated symptoms: nausea, vomiting, photophobia phonophobia
- Resolution phase
  - Subsided of headache
  - Reversed of promonitory phase

Premonitory symptoms (prodrome)

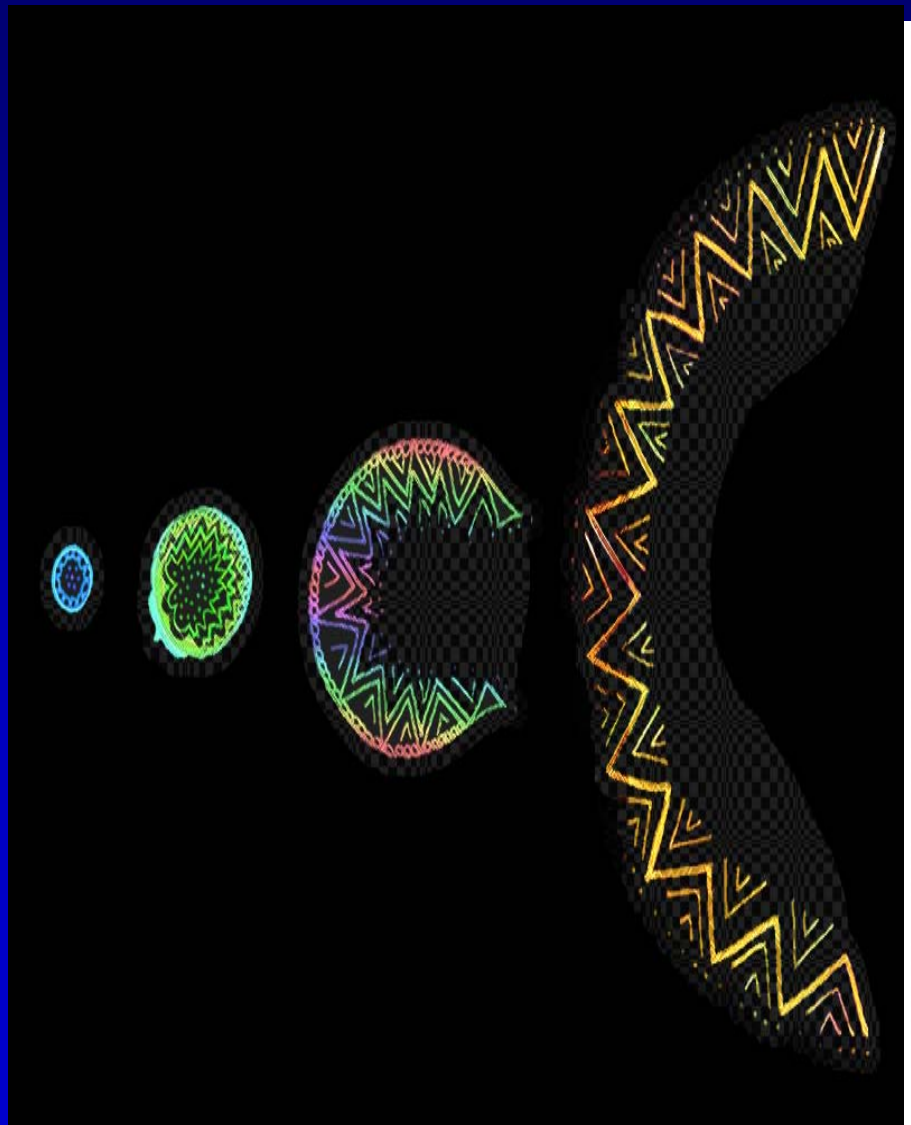
Mental manifestations	Hypothalamic autonomic manifestations
Depressed	Yawning
Hyperactive	Cold feeling
Euphoric	Food cravings
Talkative	Anorexia
Irritable	Diarrhea
Restless	Constipation
Difficulty concentrating	Thirst
Sluggishness	Urination
Drowsy	Retention
Hyperosmia	

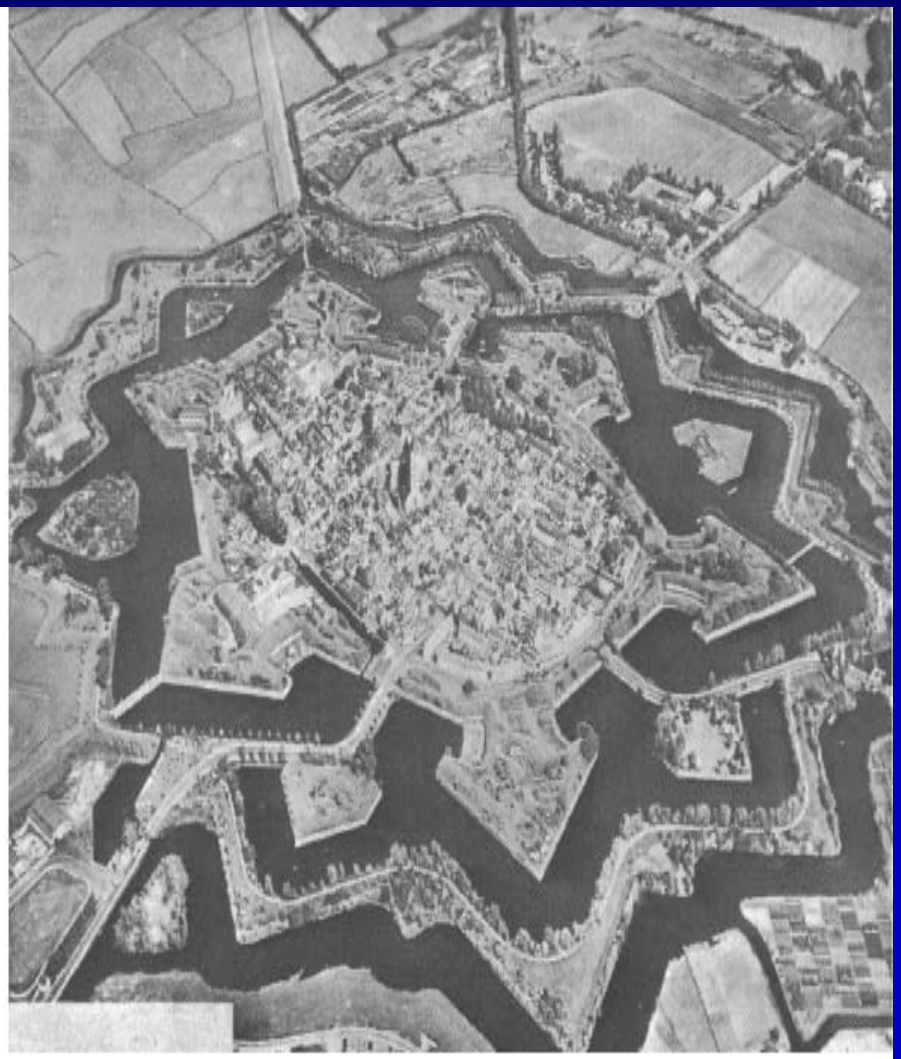
Aura – Neurological syndrome

- Typical aura
  - Visual
    - Scintillating scotoma
    - Hemianopia
    - Blindness
    - Blurring of vision
  - Paraesthesia
  - Aphasia
  - Combined
- Other aura
  - Hemiplegia
  - Brainstem symptom
    - Dysarthria
    - Ophthalmoplegia
    - Nystagmus

- Other aura
  - Oculosympathetic palsy
  - Mydriasis
  - Confusion-stupor
  - Dizziness
  - Cyclical vomiting
  - Seizure
  - Deafness
  - Recurrent of old stroke deficit

Fortification spectra



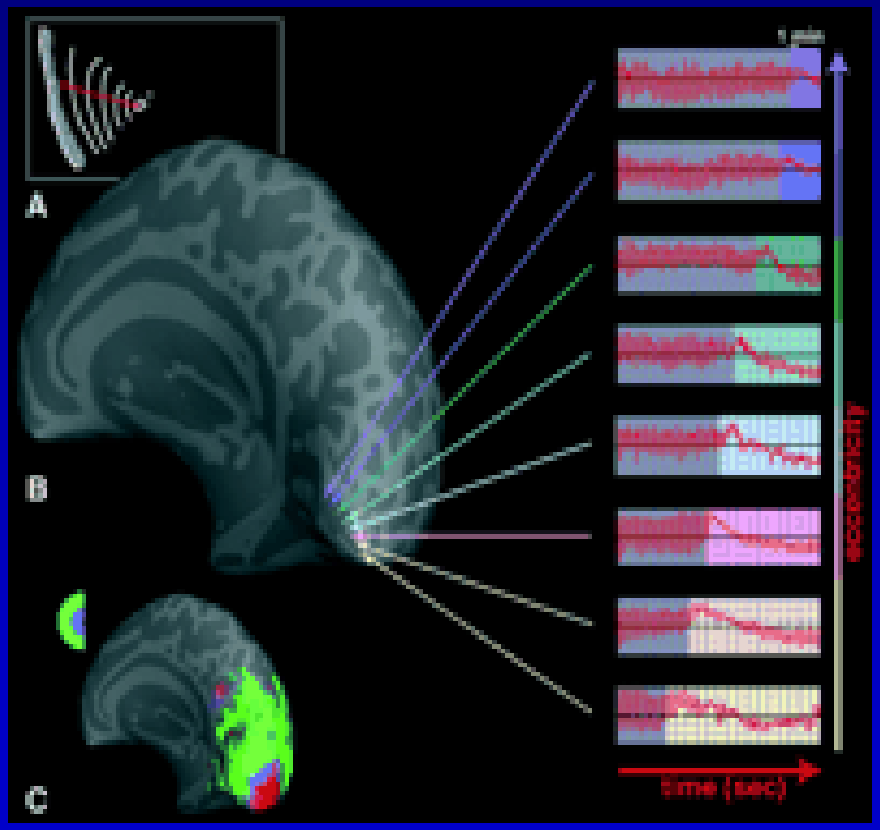


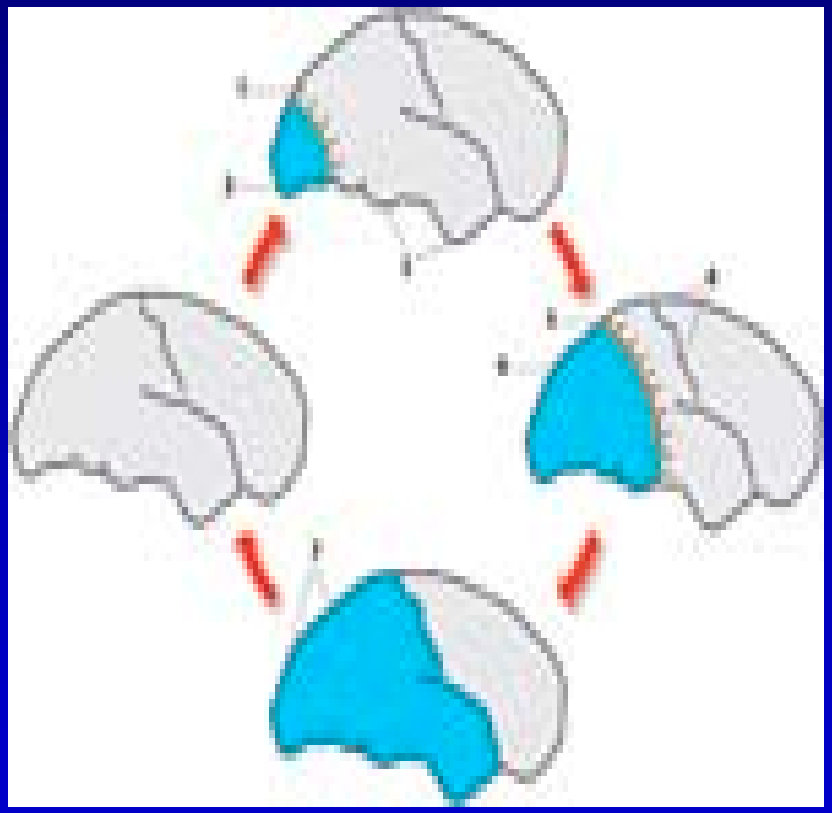
Visual distorsion

Spreading Depression

- **Spreading depression** (SD) consists of a spreading wave of depolarization associated with a reduction of the cortical activity that lasts for minutes with a propagation speed of around 3 mm/min.
- Expression "cortical spreading depression" (CSD) is widespread
- Phenomenon is not exclusively cortical—it has been recorded in various tissues including
  - Basal ganglia
  - Thalamus
  - Cerebellum
  - Tectum
  - Olfactory bulb
  - Retina
  - Spinal cord
- "Spreading depression" (SD) is a better denomination

Cortical spread depression

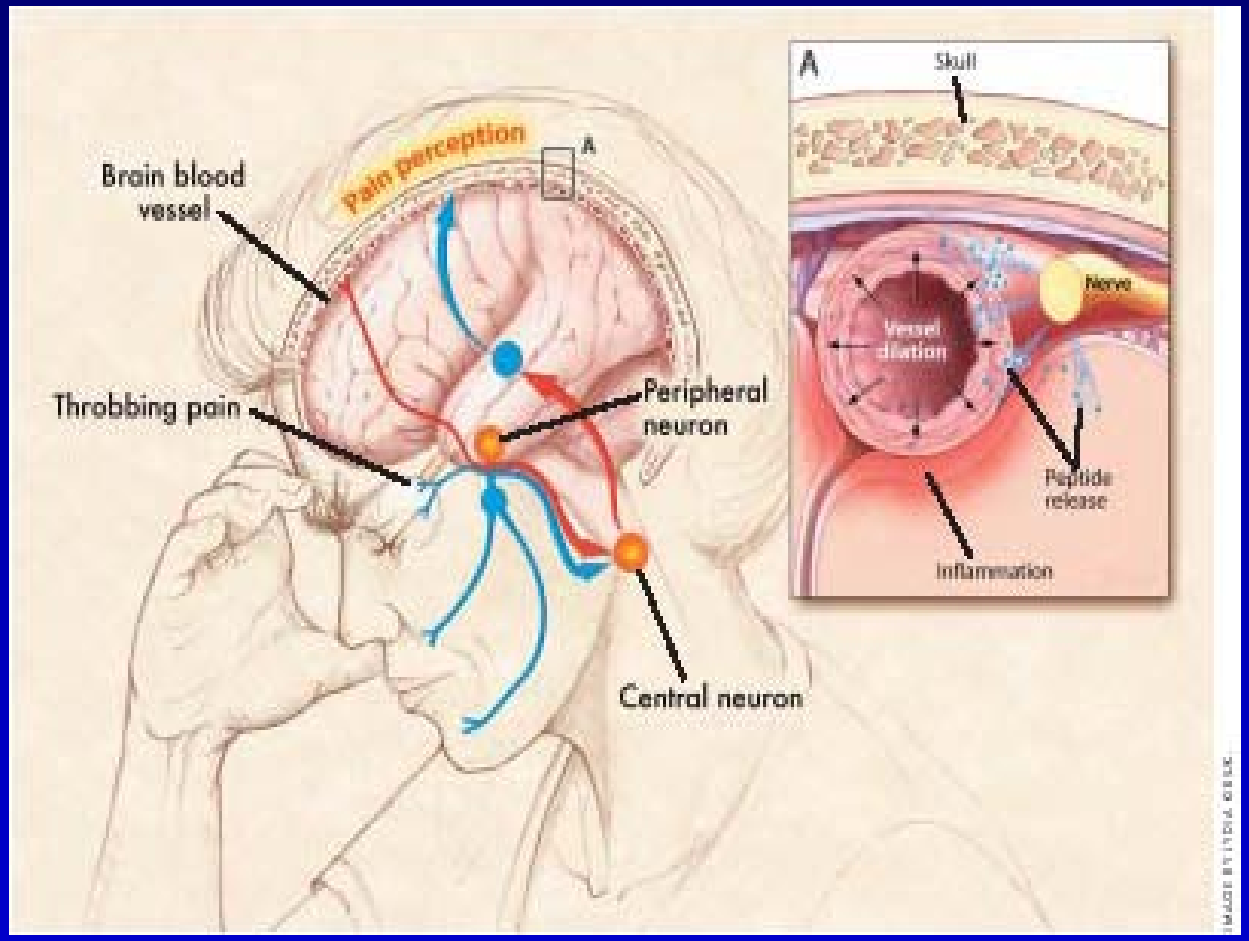




Trigeminovascular system

- Trigeminovascular system
  - Comprised of the trigeminal fibers innervating meningeal and brain vessels
- Trigeminovascular system is activated by SD, leading to
  - Plasma extravasation
  - Vasodilatation (neurogenic inflammation) in dura mater
  - Meningeal pain

Migraine headache



Migraine headache

- Unilateral or bilateral location
- Migratory of pain
- Shifting of side in various attacks
- Meningeal pain syndrome
- Pulsating quality
- Moderate or severe intensity (inhibit or prohibit daily activities)
- Aggravated by walking stairs or similar routine physical activities
- Headache lasts 4-72hr (untreated or unsuccessfully treated)

Other faces of migraine

Persistent aura without infarction (PAWOI)

Introduction

- Migraine auras are reversible visual, sensory, motor or language deficits that typically last <1 h preceding migraine attacks
- Rare condition in which the aura symptoms persist for >1 week without concomitant radiological evidence of cerebral infarction is referred to as ‘persistent aura without infarction
- Most of these patients had very frequent headache attacks, similar to chronic migraine.

Spectrum of persistent aura without infarcts

- Patients affected by genetic forms of migraine (more common)
  - Familiar hemiplegic migraine type 1 or 2 (FHM type 1 or 2)
  - Cerebral autosomic dominant angiopathy with subcortical infarcts and lacunar state (CADASIL)
  - Mitochondrial Encephalomyopathy, Lactic Acidosis, Strokelike Episodes (MELAS)
  - Aura symptoms may involve: motor, sensory or visual systems, either positive or negative.
- Patients without genetic syndromes, (rare)
  - Long-lasting visual auras plus sensory aura

Persistent aura without infarction (PAWOI)

- Persistent aura without infarction (PAWOI) first described under the designation prolonged migraine aura status
- PAWOI is said to be a possible cause of a variety of neurological symptoms, including visual snow, loss of vision, increased afterimages, and others
- Pathogenesis of PAWOI is unknown
- PAWOI is usually diagnosed solely based on the patient's present and past symptoms.

IHS criteria

- Description:
  - Aura symptoms persisting for more than 1 week without radiographic evidence of infarction.
- Comments:
  - Persisting aura symptoms are rare
  - Often bilateral and may last for months or years.
  - Reliably effective treatment is not known
  - Exclude by MRI
    - Posterior leukoencephalopathy
    - Migrainous infarction

Diagnostic criteria

- Diagnostic criteria:
  - A. The present attack in a patient with 1.2 Migraine with aura is typical of previous attacks except that one or more aura symptoms persists for > 1 week
  - B. Not attributed to another disorder

Symptoms

- Symptoms are those of a migrainous visual aura that either recurs repetitively hour-after-hour, day-after-day for weeks, months, or years, or does not abate for weeks, months, or years.
- Aura symptoms other than visual may be a sensory , but so far no one has reported a migraine aura status composed soley of non-visual aura symptoms

Variety of PAWOI

- Repetitive variety
  - Stereotyped repetitive attacks several times per hour for weeks.
- Continuous variety
  - persisting unremitting disturbances
- Continuous, strongly fluctuating variety
  - persistent but fluctuating in size or intensity, without disappearing entirely

Visual aura

After image

Visual snow

Pathophysilogy

- Persistent visual aura described as positive aura symptoms : ‘moving stars’ or ‘rippling waves’ supports central excitation
- Cortical spreading depression is a neuronal depolarization wave originating from the occipital cortex with subsequent suppression of electrical activity
- Visual cortex in persistent visual aura may be dominated by the sustained, recurrent excitatory activations piloting cortical spreading depression waves
- Persistent aura can be relieved clinically with the anti-convulsants suggesting a pathophysiological link to abnormal cerebral excitability,

Investigations

- PET showed a sustained metabolic activation in medial occipital cortex
- Magnetoencephalographic showed visual cortex maintains a steady-state hyperexcitability without significant dynamic modulation.
  - Excitability characteristic supports pathophysiological link to sustained excitatory effects possibly related to reverberating cortical spreading depression.
- Magnetic resonance spectroscopy (MRS) provided evidence for difference in metabolic ratios between proposed affected cortical region and contralateralcortical region

Investigations

- SPECT scans have shown decreased cerebral perfusion in most of the patients scanned,
  - One hemifield the decreases have been on the side opposite the perceptions
  - In some scans the perfusion decreases have been limited to the occipital lobe
  - Findings suggest that the affected cerebral regions are metabolically depressed, and are compatible with the data from more quantitative tests in patients with typical migrainous auras.
  - SPECT and perfusion MRI, documenting decreased left fronto-parieto-occipital and right occipital blood perfusion at the onset of symptom and almost complete extinction of symptoms on followup
- Metabolic implications of this are, as yet, unclear but may involve altered energy metabolism

Treatment

- Repetitive form
  - Acetazolamide may be the premier drug
- Continuous form.
  - Divalproex (valproate), lamotrigine , or topiramate
- When these oral drugs are ineffective, an intravenous injection or injections of furosemide should be tried.

Retinal Migraine

History

- Galezowski : attacks of monocular visual impairment associated with migraine headache. as “ophthalmic megrim”
- Carroll introduced the term "retinal migraine" : episodes of transient and permanent monocular visual loss, specifically in absence of migraine headache
- Most subsequent observers have used term “retinal migraine” for those cases of monocular visual impairment temporally associated with attacks of migraine.
- Some have noted that unilateral visual loss was not restricted exclusively to retina and advocated the term “anterior visual pathway migraine” or “ocular migraine”
- Many authors prefer the term “migraine associated with monocular visual symptoms” because it distinguishes between the loss of vision in 1 homonymous hemifield and that of 1 eye and includes sites other than the retina, such as the choroid or the optic nerve

IHS criteria

- Description:
  - Repeated attacks of stereotypical monocular visual disturbance, including scintillations, scotomata or blindness, associated with migraine headache
- Comment:
  - Some patients who complain of monocular visual disturbance in fact have hemianopia.
  - Some cases without headache have been reported, but their migrainous nature cannot be ascertained. Other causes of transient monocular blindness (amaurosis fugax), such as optic neuropathy or carotid dissection, must be excluded.

Diagnostic criteria

- Diagnostic criteria:

  A- At least 2 attacks fulfilling criteria B and C

  B-Fully reversible monocular positive and/or negative visual phenomena (eg, scintillations, scotomata or blindness) confirmed by examination during an attack or (after proper instruction) by the patient's drawing of a monocular field defect during an attack

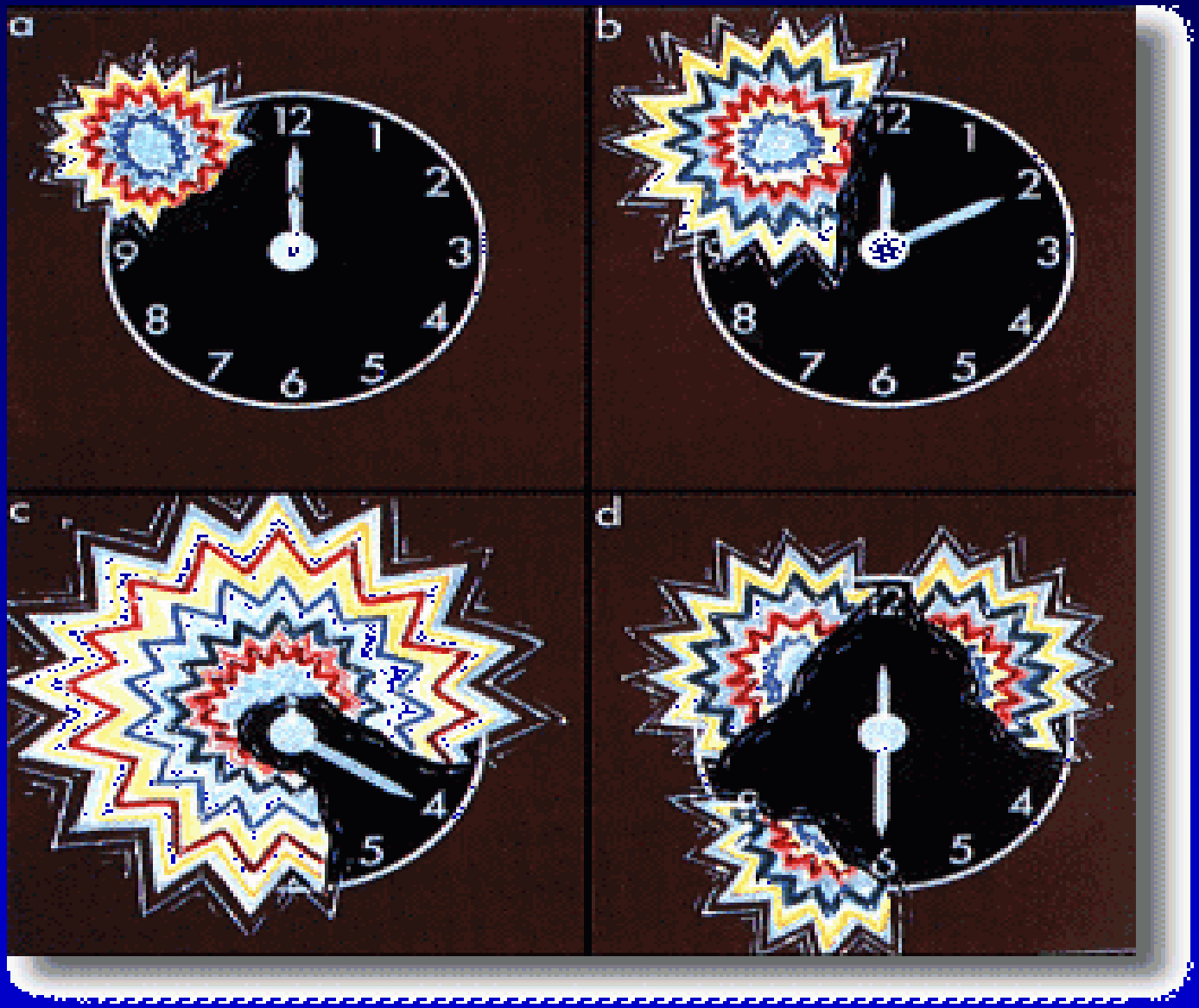
  C-Headache fulfilling criteria B-D for 1.1 Migraine without aura begins during the visual symptoms or follows them within 60 minutes

  D- Normal ophthalmological examination between attacks

  E- Not attributed to another disorder

Symptoms of retinal migraine

- Positive and negative visual phenomena
- Positive visual phenomena included
  - Flashing rays of light,
  - Zigzag lightning
  - Bright colored streaks,
  - Halos , or diagonal lines
  - Teichopsia
- Negative visual losses included
  - Blurring , “gray-outs,” and “black-outs,” causing partial or complete blindness
  - Elementary forms of scotoma were perceived as blank areas, black dots, or spots in the field of vision
  - Visual field defects can be altitudinal, quadrantic, central, or arcuate.
  - Complex patterns of monocular visual impairment, such as
    - appearance of black paint dripping down from the upper corner of the eye,
    - coalescence of peripherally located spots,
    - tunnel vision were noted rarely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Symptoms of retinal migraine

- Visual disturbance often occurs on the same side of the migraine headache and may precede, accompany, or rarely, follow it
- Duration of the visual symptoms may be as short a few seconds but usually lasts many minutes to 1 hour.
- Prolonged but fully reversible visual loss of one eye may rarely occur, sometimes lasting hours, days, or even, weeks.
- Nearly half of reported cases some series of patients with retinal migraine and recurrent transient monocular visual loss subsequently experienced permanent monocular visual loss

Symptoms of retinal migraine

- In contrast to the current ICHD-2 criteria for retinal migraine, nearly 50% of patients with monocular visual loss had a history of migraine with conventional visual aura.
- Rare cases of transient monocular visual loss have also been reported with
  - cluster headache,
  - idiopathic stabbing headache,
  - chronic daily headache,
  - cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy,
  - unspecified headache type.

Permanent monocular visual loss

- Prolonged and permanent monocular visual loss appears to occur more commonly in patients with retinal migraine than in cases of prolonged typical aura or migrainous infarction in those with conventional migraine.
- High number of patients with transient monocular visual loss, who eventually develop permanent monocular visual loss, makes retinal migraine a less benign condition than migraine with conventional visual aura.
- Therefore, although there are no data to determine the efficacy of preventative treatment for this entity, prophylactic drug therapy seems prudent, even if attacks are infrequent.

Treatment

- Insufficient data to support specific recommendations for acute and preventive medical therapy
- Therapy of the acute attack of retinal migraine should probably not include triptans or ergots because of their vasconstrictive properties.
- Prophylactic medications that have been tried with anecdotal benefit include calcium-channel blockers, tricyclic antidepressants (ie, amitriptyline or nortriptyline) and neuromodulators.(antiepileptic drugs ie, topirimate or divalproex sodium)
- Although some patients respond to beta-blockers, not usually recommend them as first-line agents because of their theoretical potential for arteriolar constriction.
- Aspirin is a logical agent because of its antiplatelet activity,

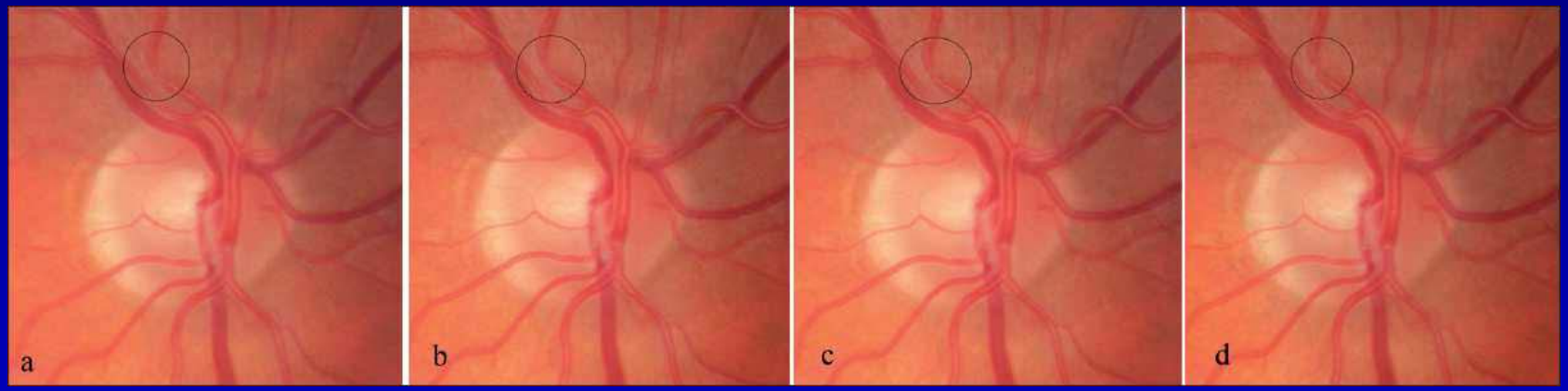
Most cases labeled as "retinal migraine" are not migraine

- Definite retinal migraine, as defined by the IHS criteria, is an exceedingly rare
- There are no convincing reports of permanent monocular visual loss associated with migraine.
- Most cases of transient monocular visual loss diagnosed as retinal migraine would more properly be diagnosed as "presumed retinal vasospasm.

Physical examinations

- Severe narrowing or occlusion of retinal arteries and veins was observed rarely diagnoses of anterior or posterior ischemic optic neuropathy
- Central serous retinopathy
- Optic disk edema
- Hemorrhages of the optic nerve, retina, or vitreous
- Optic nerve atrophy

Retinal vasospasm.



Pathophysilogy

- Vasospasm of retinal or ciliary circulation may have caused retinal or optic nerve ischemia
  - This may explain the amaurosis and rare funduscopic findings during acute attacks of retinal migraine.
- Spreading depression of retinal neurons, a phenomenon that has been demonstrated in the chick retina
  - Similarly, it is possible that those rare cases with prolonged monocular defects associated with migraine headache could have a mechanism similar to that seen in the cerebral cortex of migraineurs who have persistent aura without infarction

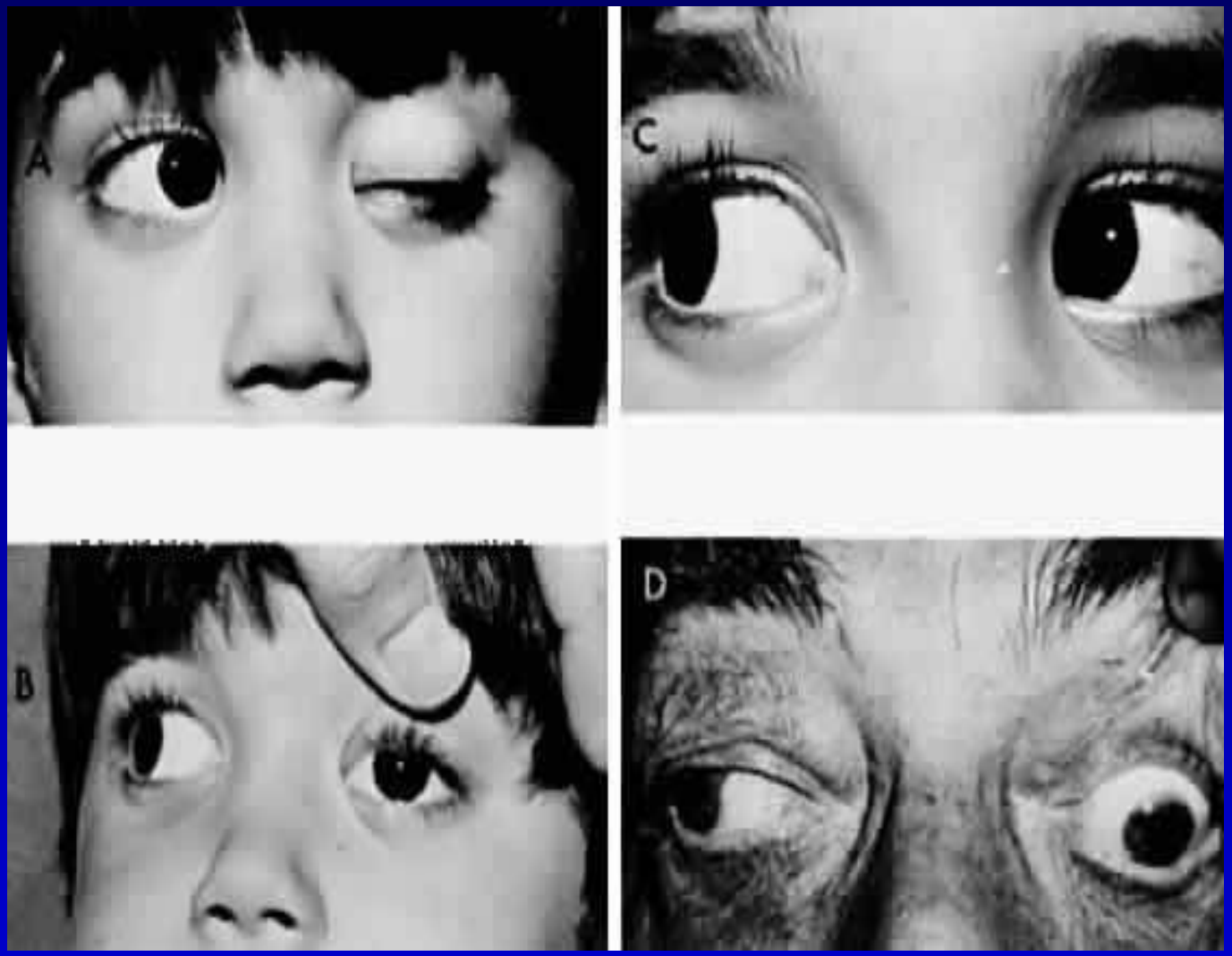
Proposed Criteria for Migraine with Monocular Visual Symptoms

- 1.4.1 Migraine with transient monocular visual symptoms
  - A. At least 2 attacks fulfilling criteria B and C
  - B. Fully reversible monocular positive and/or negative visual phenomena confirmed by at least one of the following:
    - a. Examination during the attack showing signs of ipsilateral retinal or optic nerve abnormality
    - b. The patient's drawing of a monocular field defect experienced during the attack
    - c. A description of positive or negative visual symptoms confirmed by the patient by occluding each eye individually during the attack
  - C. Headache fulfilling criteria B-D for 1.1 Migraine without aura or 1.2 Migraine with aura begins during the visual symptoms or precedes or follows them within 60 minutes
  - D. Normal ophthalmological examination between attacks
  - E. Headache and monocular visual symptoms not attributed to another disorder

Proposed Criteria for Migraine with Monocular Visual Symptoms

- 1.4.2 Migraine with permanent monocular visual loss or defect
  - A. At least one attack fulfilling criteria B and C
  - B. Irreversible monocular positive and/or negative phenomena (eg, scintillations, scotoma, or blindness) confirmed by examination during or following the attack
  - C. Headache fulfilling criteria B-D for 1.1 Migraine without aura or 1.2 Migraine with aura begins during the visual symptoms or precedes or follows them within 60 minutes
  - D. Abnormal ophthalmological examination confirming retinal vascular abnormality or optic neuropathy
  - E. Headache and monocular visual loss not attributed to another disorder
  - †Most patients experienced only one attack of permanent monocular visual loss

Ophthalmoplegic Migraine


A
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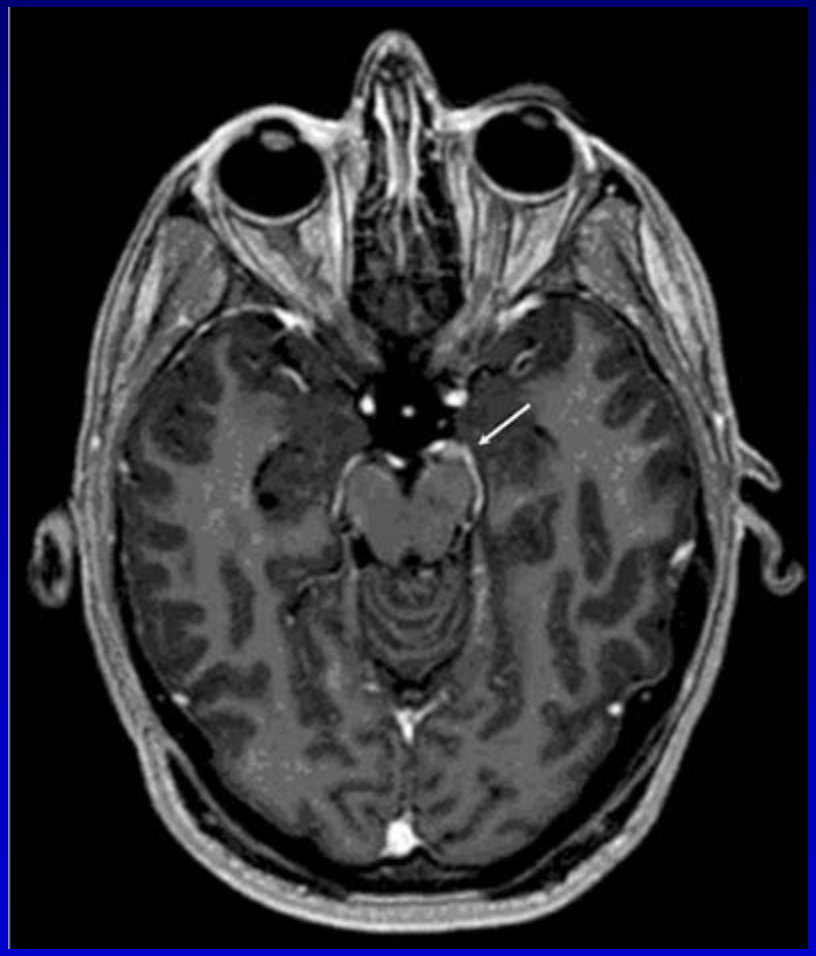
D

IHS Criteria

- Description:
  - Recurrent attacks of headache with migrainous characteristics associated with paresis of one or more ocular cranial nerves (commonly the third nerve) in the absence of any demonstrable intracranial lesion other than MRI changes within the affected nerve.
- Comment:
  - It is unlikely that Ophthalmoplegic "migraine" is a variant of migraine since the headache often lasts for a week or more and there is a latent period of up to 4 days from the onset of headache to the onset of ophthalmoplegia.
  - Furthermore, in some cases MRI shows gadolinium uptake in the cisternal part of the affected cranial nerve which suggests that the condition may be a recurrent demyelinating neuropathy

MRI shows gadolinium uptake in the cisternal part of the affected cranial nerve



MRI shows gadolinium uptake in the cisternal part of the affected cranial nerve

Diagnostic criteria

- Diagnostic criteria:

  A- At least 2 attacks fulfilling criterion B

  B- Migraine-like headache accompanied or followed within 4 days of its onset by paresis of one or more of the third, fourth and/or sixth cranial nerves

  C- Parasellar, orbital fissure and posterior fossa lesions ruled out by appropriate investigations

General aspects of OM

- Ophthalmoplegic migraine (OM), is a unique disorder characterized by recurrent attacks of ophthalmoplegia, following severe migrainous headaches.
- Condition almost always begins in childhood, and is considered to be rare in adult
- Typical clinical picture of OM includes a child with severe migraine that lasts for several days and is followed soon after, by ptosis and diplopia due to isolated oculomotor nerve palsy, as the headache remits.
- Pupillary involvement with a 'full blown' pupil is the rule in children.
- Pupillary sparing is not uncommon, especially in adults.
- Abducens nerve involvement is uncommon and trochlear nerve palsy is rare.
- Recovery is the rule.
- Rarely, deficits persist after multiple attacks

Previous concepts

- During a severe migraine attack
  - Edematous wall of the ipsilateral carotid artery compresses the oculomotor nerve inside the cavernous sinus
  - Narrowing the ostia of the vasa nervosa supplying the oculomotor nerve inside the cavernous sinus causes ischemic nerve
- Depending on the presence or absence of pupillary involvement in patients with oculomotor palsy, a compressive or ischemic etiology was suspected

Current concepts

- Gadolinium magnetic resonance imaging (GdMRI) studies provided new insights into the pathogenesis of OM
- Cisternal segment of the third nerve were most pronounced in the exit zone of the nerve from the midbrain.
- Follow-up GdMRI studies done after the resolution of ophthalmoplegia disclosed almost no enhancement.
- Benign viral infection as the possible cause of OM.
- Some patient, had recurrent attacks of OM following vaccination on three separate occasions
- Besides cases with enhancement, lack of third nerve enhancement on GdMRI is also well documented .

Current concepts

- Recurrent demyelinating neuropathy, in which an inflammatory process affecting the oculomotor nerve might have irritated trigeminal sensory fibers present in the same nerve, triggering migraine headache.
- Pronounced thickening and enhancement of the third nerve, at its origin from the midbrain was due to intraneural edema, similar to that seen in inflammatory demyelinating neuropathies.
- Severe migrainous headaches seen in almost all cases of OM were considered to be of secondary significance
- Daroff : OM from `migraine' to a `cranial neuralgia' category.
- Plethora of other case reports exhibiting similar findings on imaging, supported a post-viral demyelinating hypothesis

Theory against demyelinating disease

- All patients of OM developed against a backdrop of longstanding and uncontrolled migraine.
- History of an antecedent worsening in severity of migraine before the ophthalmoplegic attack
- Deficits develop during or within 24 h of a severe attack of migraine. Ophthalmoplegic migraine attack is severe and often lasts beyond 24 h,.
- Normal CSF examination ,even in patients with nerve enhancement.

Mechanism of migraine in OM general aspects

- Activation of trigeminovascular system during an attack of migraine releases neuropeptides in the vessel wall.
- This causes a sterile inflammation of the wall of vasa nervosa leading to a breach in the blood neural barrier, which is formed by endothelium of vasa nervosa.
- Leads to nerve edema and injury.
- Once the attack of OM subsides, the decrease in neurogenic inflammation of the vessel wall leads to restoration of the blood nerve barrier and decrease in nerve edema and enhancement.
- Recurrent attacks may result in nerve infarction and aberrant regeneration

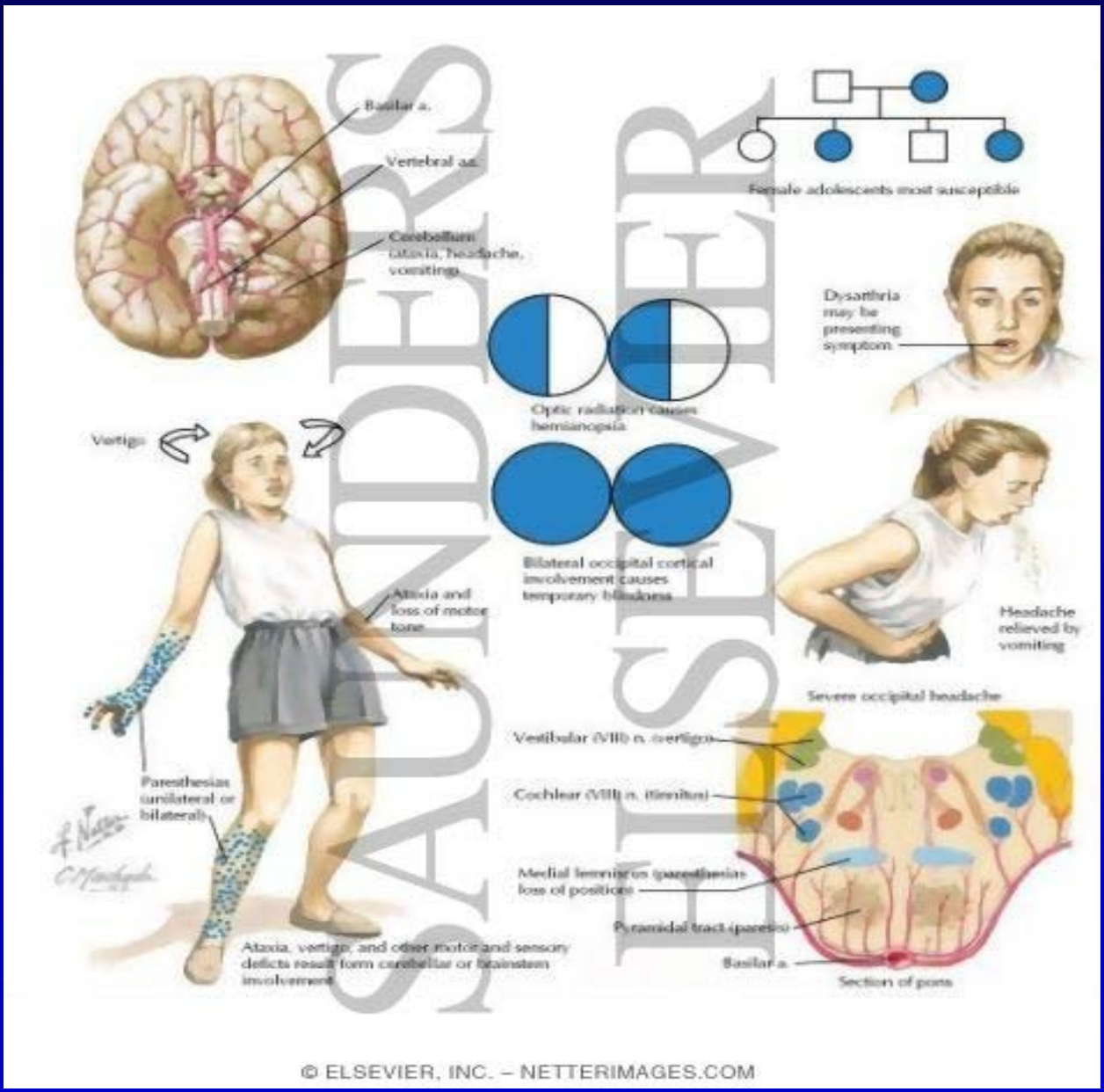
Mechanism of migraine in OM lesion at root entry zone

- Myelin of the cranial nerves at the root exit zone is of the central type, and the blood-neural barrier is deficient at this level
- During a migraine attack, the vasospasm of the vasa nervorum could lead to failure of the blood-neural barrier at the root exit zone, where it is less efficient.
- This situation may cause vasogenic edema, accounting for both the focal enlargement and the enhancement of the third cranial nerve,at the root exit zone

Ophthalmoplegic migraine

- Generally occurs in children, but adult cases have been reported
- Classification
  - Childhood variant: Characterized by severe migraine, recurrent third nerve palsy with pupillary involvement, and enhancement on GdMRI. Rarely, it is painless and lacks enhancement.
  - Adult variant: characterized by severe migraine, an antecedent worsening in severity of migraine prior to the ophthalmoplegic attack : single attacks of sixth nerve palsy. Third nerve involvement is less common and it is pupillary-sparing. Enhancement is uncommon even with third nerve involvement

Basilar - type Migraine



Basilar -type migraine (BTM).

- Basilar artery migraine (BAM).
- Bickerstaff syndrome
- Brainstem migraine
- Vertebrobasilar migraine
- Cerebellar migraine

IHS criteria

- **Description:**
  - Migraine with aura symptoms clearly originating from the brainstem and/or from both hemispheres simultaneously affected, but no motor weakness
- **Comments:**
  - Basilar-type attacks are mostly seen in young adults.
  - Almost always co-existing attacks of migraine with typical aura
  - If motor weakness is present, code as 1.2.4 **Familial hemiplegic migraine** or 1.2.5 **Sporadic hemiplegic migraine**. Patients with 1.2.4 **Familial hemiplegic migraine** have basilar-type symptoms in 60% of cases.
  - Therefore, 1.2.6 **Basilar-type migraine** should be diagnosed only when no motor weakness occurs.

Diagnostic criteria

- **Diagnostic criteria:**

A- At least 2 attacks fulfilling criteria B-D

B- Aura consisting of at least two of the following fully reversible symptoms, but no motor weakness:

- dysarthria
- vertigo
- tinnitus
- hypacusia
- diplopia
- visual symptoms simultaneously in both temporal and nasal fields of both eyes
- ataxia
- decreased level of consciousness
- simultaneously bilateral paraesthesias

C- At least one of the following:

- at least one aura symptom develops gradually over ≥5 minutes and/or different aura symptoms occur in succession over ≥5 minutes
- each aura symptom lasts ≥5 and ≤60 minutes

D-Headache fulfilling criteria B-D for 1.1 **Migraine without aura** begins during the aura or follows aura within 60 minutes

E- Not attributed to another disorder

Aura

- All patients (100%) described visual aura
- 40% with symptoms in both fields of vision and 60% with only one side of vision affected.
- Visual aura was the most common initial symptom in 2 of 3.
- Temporary blindness can be reported
- Sensory aura occur in 61%
- Aphasic aura, were present in 40%.

Aura

- Vertigo as an initial aura symptom was found in 10%
- Vertigo may manifest as illusory self, object motion, positional and head motion intolerance
- Vertigo may be associated with aural fullness and reduced balance, tinnitus
- Aura in BTM commonly becomes more typical during later mid-life

General aspects of BTM

- Basilar migraine, probably carried one of the known ion channel related mutations.
- Mutation at the FHM2 locus at theATP1A2 gene has been described in familial BTM without hemiplegia, suggesting a connection between BTM and hemiplegic migraine.
- BTM most probably represents a variation of MWA rather than another migraine subtype, as 95% of the BTM patients experience typical aura as in MWA

Treatment

- Migraine-specific medications such as the triptans and ergotamines are contraindicated for BTM
- Triptans and ergotamines are known to constrict blood vessels and were believed to likely cause safety issues if used.
- Headache specialists reported on 13 patients with basilar migraine, familial hemiplegic migraine, or migraine with prominent or prolonged aura who had received triptans. No harm was done (no adverse events) with excellent relief of headache and symptoms
- In a larger group of patients meeting criteria for BTM, no increased incidence of adverse events was reported following inadvertent or intentional triptan exposure.
- Conclusion
  - contraindication of triptans in basilar migraine should be reconsidered.
  - prominent or prolonged aura may not represent a reasonable contraindication to triptan therapy.
- .

Treatment

- Preventive medications
  - Topiramate
  - Calcium channel blocker
  - BTM is generally managed with traditional preventatives
- Although many recommend that beta blockers be avoided due to rare reports of complicating events

Disability and Prognosis

- BTM is often more debilitating than migraine with aura due to aura intensity, number of symptoms and longer length and vertiginous symptom
- More severe other migraine symptoms, such as nausea, prickly feelings on the body, sensitivity to light and/or sound, and trouble thinking clearly.
- Worse, these symptoms can come in succession - several one after the other
- Usually there's a severe throbbing headache at the back of the head on both sides
- Disabling , symptoms of BTM are usually more frightening than harmful.
- No evidence that BTM patients have any greater stroke risk than migraine with typical aura.
- Migraine with aura does have a slightly higher stroke risk than migraine without aura in those younger than 45, so optimal prevention and knowledge of stroke risk factors and their control is important.

Migraine-associated vertigo

Migraine and Vertigo (1)

- Vertigo as a major symptom in migraine
  - Basilar migraine
- Vertigo attack in migraine without typical headache
  - **Children**
    - Benign paroxysmal vertigo of childhood
    - Benign paroxysmal torticallis of infancy
  - **Adults**
    - Benign recurrent vertigo

Migraine and Vertigo (2)

Migraine-associated vertigo

- Not fulfill criteria of basilar migraine (IHS)
- Neuhauser's diagnostic criteria for migrainous vertigo

Association of migraine with other vertigo disorders?

- Susceptibility to motion sickness
- Familial periodic ataxia
- Menière's disease
- Benign paroxysmal positioning vertigo

Terminologies
Migraine-associated vertigo

- Migraine-associated vertigo is a term used to describe episodic vertigo in patients with history of migraine or with other clinical features of migraine.
- Other terminologies include
  - Vertiginous migraine
  - Vestibular migraine
  - Migraine related vestibulopathy
  - Migraine -associated dizziness
  - Migraine -related dizziness
  - migraine-related vertigo
  - Migrainous vertigo
  - Migraine -anxiety related dizziness
  - Migraine -associated cochleovestibular dysfunction.
  - Benign recurrent vertigo
  - Benign paroxysmal vertigo of childhood

Neuhauser criteria

- Neuhauser et al elaborated the criteria for diagnosis of
- “definite” migrainous vertigo (i.e., migraine according to IHS criteria, migrainous symptoms during at least two vertiginous attacks)
- “probable” migrainous vertigo (i.e., migraine according to IHS criteria, migraine symptoms during vertigo, migraine-specific precipitants of vertigo, or response to antimigraine drugs)

Lembert and Neuhauser propose criteria for definite and probable migraine-associated vertigo

- Definite migraine-associated vertigo includes a case history of:
  - Episodic vestibular symptoms of at least moderate severity;
  - Current or previous history of migraine according to the 2004 International Classification of Headache Disorders;
  - One of the following migrainous symptoms during two or more attacks of vertigo: migrainous headache, photophobia, phonophobia, visual or other auras;
  - Other causes ruled out by appropriate investigations.

Lembert and Neuhauser propose criteria for definite and probable migraine-associated vertigo

- Probable migraine-associated vertigo includes a case history of
  - Episodic vestibular symptoms of at least moderate severity and one of the following:
  - Current or previous history of migraine according to the 2004 International Classification of Headache Disorders;
  - Migrainous symptoms during vestibular symptoms;
  - Migraine precipitants of vertigo in more than 50% of attacks, such as food triggers, sleep irregularities, or hormonal change;
  - Response to migraine medications in more than 50% of attacks; and
  - Other causes ruled out by appropriate investigations

Clinical features of definite Vestibular migraine in 33 patients

Clinical features	*%*
Vestibular symptoms	
Rotational vertigo	70
Other illusion self or object motion	18
Positional vertigo	42
Head motion intolerance	48

Clinical features of definite Vestibular migraine in 33 patients

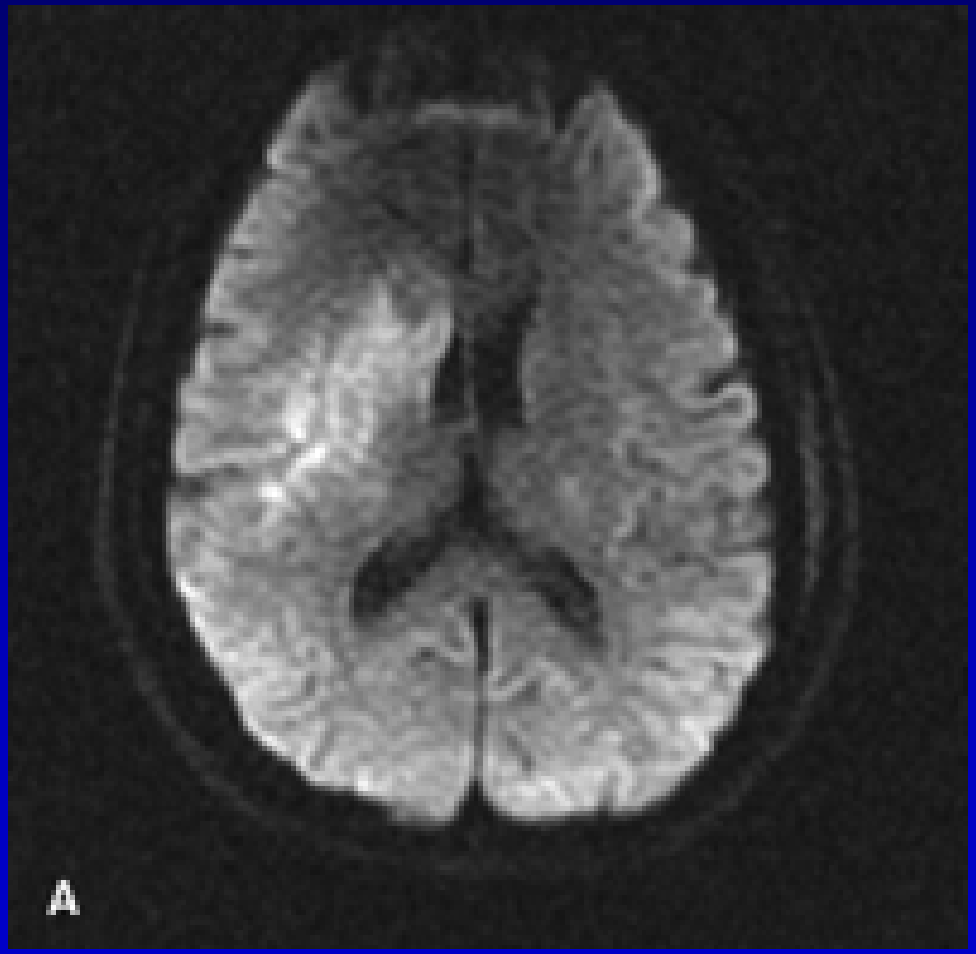
Clinical features	*%*
Duration of vestibular symptoms	
Second to 5 minutes	18
5-60 mimutes	33
1hour to 1 day	21
> 1 day	48

Vestibular migraine and abnormal brainstem signs

- Vertical nystagmus 48%
- Horizontal nystagmus 22%
- Saccadic, pursuit gaze evoke nystagmus 27%
- Moderate positional nystagmus 21%
- Spontaneous nystagmus 26%

Migrainous infarction.

Migrainous infarction



IHS criteria

- Description:
  - One or more migrainous aura symptoms associated with an ischaemic brain lesion in appropriate territory demonstrated by neuroimaging
- Comment:
  - Ischaemic stroke in a migraine sufferer may be categorised as
    - cerebral infarction of other cause coexisting with migraine,
    - cerebral infarction of other cause presenting with symptoms resembling migraine with aura,
    - cerebral infarction occurring during the course of a typical migraine with aura attack(Migrainous infarction)
  - Increased risk for stroke in migraine patients has been demonstrated in women under age 45 in several studies.
  - Evidence for an association between migraine and stroke in older women and in men is inconsistent

Diagnostic criteria

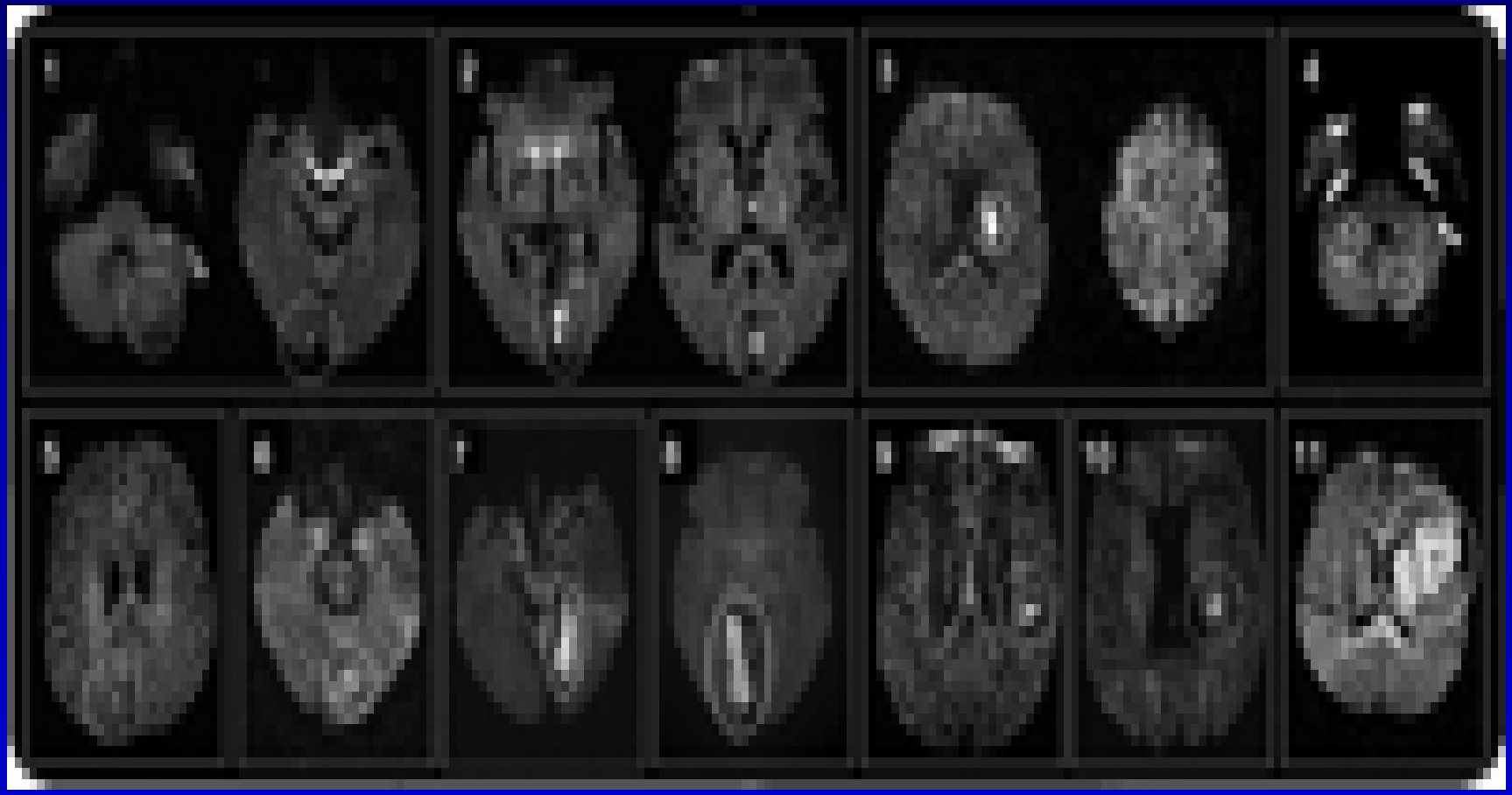
- Diagnostic criteria:

  A- The present attack in a patient with 1.2 Migraine with aura is typical of previous attacks except that one or more aura symptoms persists for >60 minutes

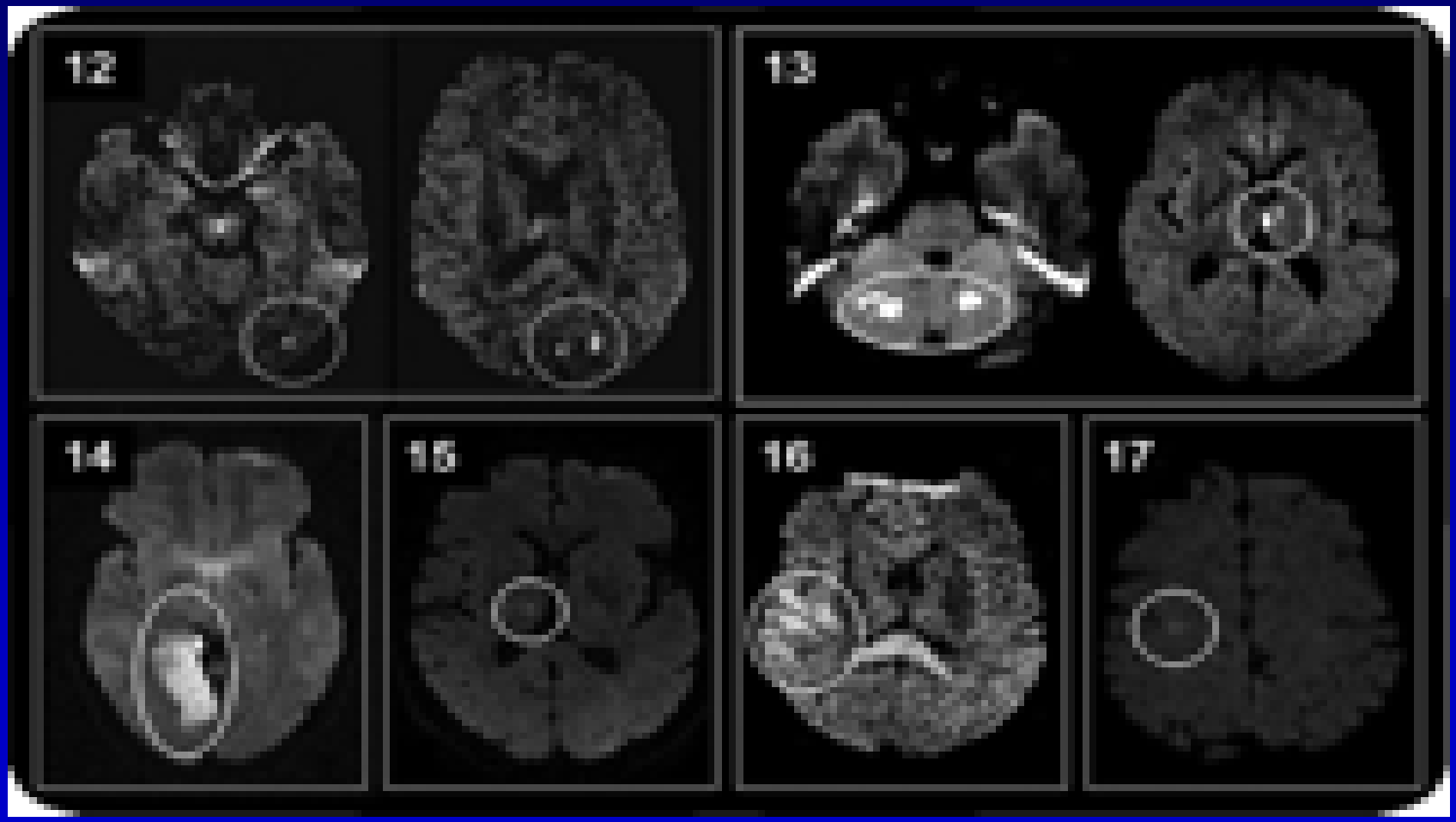
  B- Neuroimaging demonstrates ischaemic infarction in a relevant area

  C-Not attributed to another disorder

1–4 have multiple small lesions, patients 5–8 have isolated lesions in the posterior circulation territory, and patients 9–11 have isolated lesions in the middle cerebral artery territory.



12 and 13 multiple small lesions, 14 and 15 isolated lesions in posterior circulation territory, and 16 and 17 have isolated lesions in middle cerebral artery territory

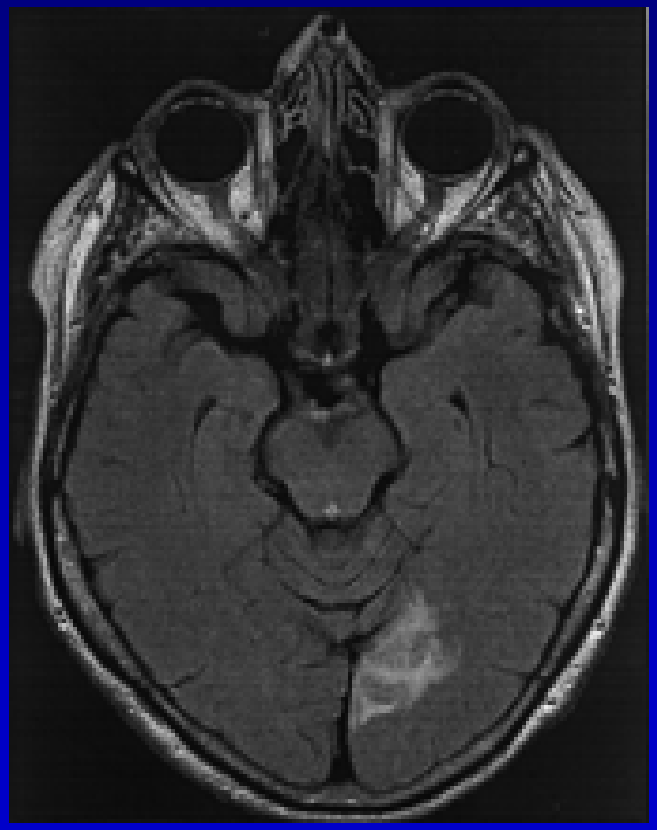


Differential diagnosis

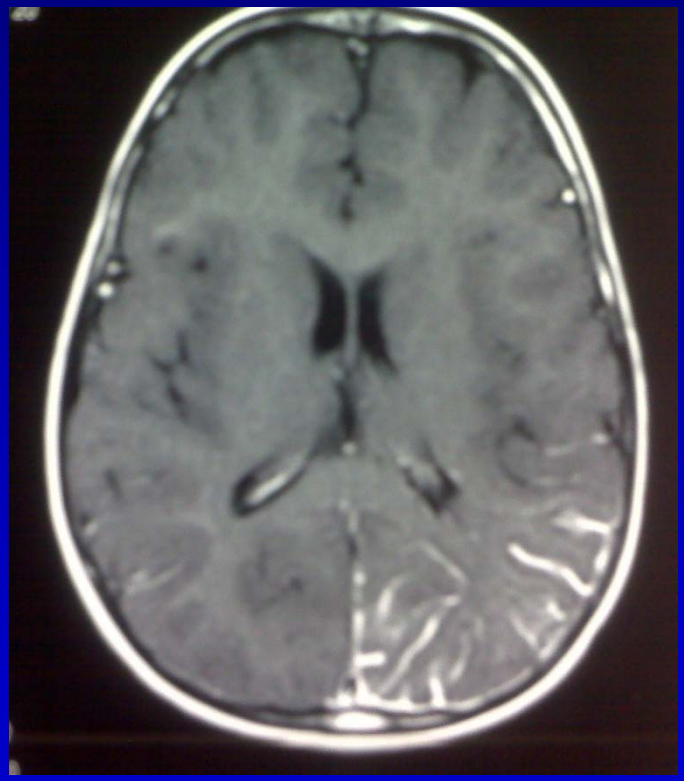
- Vascular disease common in migraine than in the general population
  - Intracranial arterial dissection
  - Patent foramen ovale (PFO).
- Secondary migraine with aura and ischaemic stroke :
  - Artero-venous malformations
  - Leptomeningeal angiomatosis, as observed in Sturge-Weber syndrome
- Aneurysm

Differential diagnosis

Migranous infarction



Sturge-Weber syndrome



Migrainous stroke.

- Best known complication of migraine
- Large hospital series, incidence of migrainous stroke may be higher than previously thought: 0.5%–1.5% of all strokes and up to 10%–14% in young patients
- Residual neurological deficit has to be similar to the migraine aura.
- Clinical signs
  - Homonymous field defect due to a posterior cerebral artery infarct, (most common)
  - Other arterial territories
  - Single or multiple lacunar infarcts
  - Retinal infarcts and ischaemic optic neuropathies have been described as complications of retinal migraine

Pathophysilogy

- Cortical spreading depression (CSD) are associated with concomitant alterations in vascular perfusion, in form of a transient hyperaemia followed by a more prolonged state of oligaemia and reduced cerebral perfusion

- During normal migraine auras, level of perfusion does not reach ischaemic threshold and is anyhow coupled with the reduced neuronal metabolism

Pathophysilogy

- In migrainous stroke this metabolic coupling is not present, so that the energetic needs of neurons exceed the vascular supply

- During migraine attacks blood platelets are overactivated and have hyperaggregability, increasing the probability of local clot formation and distal vessel occlusion.

Natural history

- Overall outcome after migrainous infarction seems favorable.
- Only a few patients reported persisting symptoms; most of them had recovered completely.
- Recurrent events were not reported .
- Frequency of migraine attacks tended to decrease after the cerebrovascular event

Migraine-triggered epilepsy

IHS criteria

- Description:
  - A seizure triggered by a migraine aura.
- Comment:
  - Migraine and epilepsy are prototypical examples of paroxysmal brain disorders.
  - While migraine-like headaches are quite frequently seen in the postictal period
  - Sometimes a seizure occurs during or following a migraine attack.
  - This phenomenon, sometimes referred to as migralepsy, has been described in patients with migraine with aura.

Diagnostic criteria

- Diagnostic criteria**:**

  A-Migraine fulfilling criteria for 1.2 **Migraine with aura**

  B- A seizure fulfilling diagnostic criteria for one type of epileptic attack occurs during or within 1 hour after a migraine aura

General aspects

- Relationship between migraine and epilepsy is complex, as the two disorders have a high comorbidity and probably share the same genetic risk factors

- Both migraine and seizures may be secondary to genetic disease or intracranial brain lesions

Pathophysilogy

- Reduced regional blood flow during CSD may also reduce the epileptic threshold.
- Repeated episodes of migraine in combination with genetic or environmental factors may lead to focal cortical injury and reorganisation, resulting in a tendency toward seizures,
- Migraine-triggered epilepsy a good response of both epileptic and migraine manifestations could often be achieved with the use of combined antiepileptic treatments eg. valproate and topiramate.

Migraine with coma

- Impaired consciousness symptoms varied from drowsiness to coma, and coma lasted from several minutes to 10 days
- Etiology of coma episodes is believed to be related to ischemic dysfunction of the rostral part of the brain stem
- Gabaergic mechanisms may be involved in dysfunction of the ascending reticular activating system, causing an alteration in consciousness
- Different types of migraine
  - Acute confusional migraine
  - Basilar artery migraine
  - Familial hemiplegic migraine;
  - Migraine with aura;

FIN