Tension-type headache: current research and clinical management

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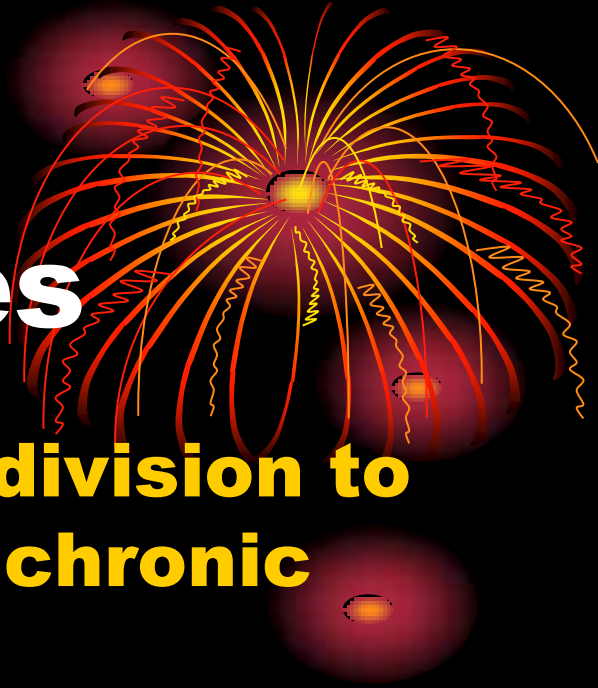
Introduction

- Most common form headache
- Indicate some kind mental or muscular tension
- Exact pathogenesis unknown
  - Peripheral myofascial mechanism
  - Central dysregulation pain
- Several secondary headache as TTH

Classification and diagnostic features



- Dx change at ICHD-II : subdivision to three group ; episodic and chronic
- Episodic:
  - Infrequent - less then one/month
  - Frequent -
  - Difficult distinguish Migraine without aura
- Chronic <-> Medication- overuse headache

Panel 1: Tension-type headache (episodic form)—general diagnostic criteria (B–E)

B **Headache lasting from 30 min to 7 days**

C **At least two of the following pain characteristics:**

1 Bilateral location

2 Pressing or tightening (non-pulsating) quality

3 Mild or moderate intensity

4 Not aggravated by routine physical activity, such as walking or climbing stairs

D **Both of the following:**

1 No nausea, vomiting (anorexia can occur)

2 No more than one of photophobia or phonophobia

E **Not attributed to another disorder**

From the 2nd edition of the International Classification of Headache Disorders (ICHD-II).[2]

Panel 2: Tension-type headache—specific diagnostic criteria

2·1 Infrequent episodic tension-type headache

A At least 10 episodes that occur on less than 1 day per month (less than 12 days per year) that fulfil criteria B–D

2·2 Frequent episodic tension-type headache

A At least 10 episodes that occur on one or more days per month but less than 15 days per month for at least 3 months (12 or more days and less than 180 days per year) that fulfil criteria B–D

2·3 Chronic tension-type headache

A Headache that occurs on 15 or more days per month, on average for more than 3 months (180 or more days per year) that fulfils criteria B–D

B Headache that lasts hours or may be continuous

C Both of the following:

1 No more than one of photophobia, phonophobia, or mild nausea

2 Neither moderate or severe nausea nor vomiting

2·4 Probable tension-type headache

A Episodes that fulfil all but one of criteria A–D for 2·1, 2·2, or 2·3

B Episodes that do not fulfil criteria for 1·1 (migraine without aura)

C Not attributed to another disorder

From the 2nd edition of the International Classification of Headache Disorders (ICHD-II)[3]

Panel 3: Proposal for stricter diagnostic criteria for TTH

B Headache lasting from 30 min to 7 days

C At least three of the following pain characteristics:

1 Bilateral location

2 Pressing or tightening (non-pulsating) quality

3 Mild or moderate intensity

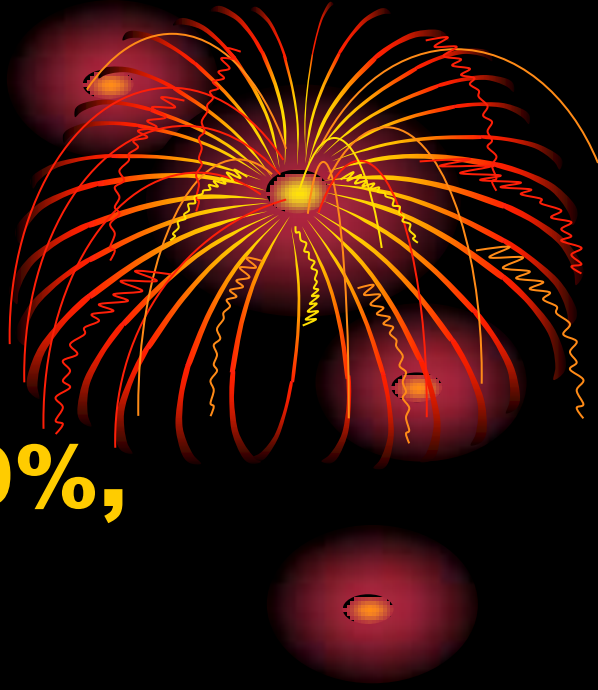
4 Not aggravated by routine physical activity such as walking or climbing stairs

D No nausea, vomiting (anorexia can occur), photopobia, or phonophobia

E Not attributed to another disorder

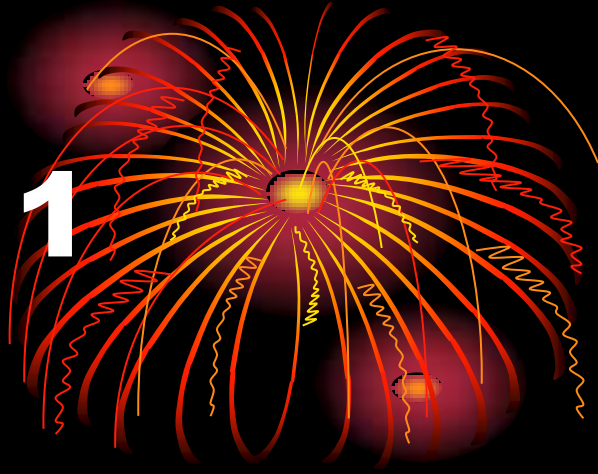
From the 2nd edition of the International Classification of Headache Disorders (ICHD-II) appendix A2.[3]

Epidemiology



- **Lifetime prevalence :79%, 3%CTTH**
- **Women higher**
- **Decline with age**
- **11.1%TTH, 3.2%migraine, 4.3%combine (from birth to age 26)**

Pathophysiology - 1



- **Myofascial factors**
  - ***Muscle activity and metabolism***
  - ***Tenderness and pain thresholds***
- **Nociceptive reflexes and pathways**
  - ***Exteroceptive silent periods in the temporalis muscle***
  - ***Blink reflex***
  - ***Biceps femoris flexion reflex***
  - ***Laser-evoked nociceptive potentials***

Pathophysiology - 2

- **Structural brain changes**
- **Neurotransmitters**
  - ***Nitric oxide***
  - ***Neuropeptides***
  - ***Serotonin (5-HT)***
- **Psychological studies**
- **Genetics**
- **A model for TTH pathogenesis**

Myofascial factors

Muscle activity and metabolism

- **EMG activity higher with CTTH (no association with headache intensity)**
- **Botulinum – decrease temporalis EMG but not CTTH headache at 12 week**
- **Not favour increased activity, muscular inflammation, or disturbed metabolism of the pericranial muscles as important pathogenic factors in CTTH**

Myofascial factors

Tenderness and pain thresholds

- **TTH: More active or latent myofascial trigger point**
- **Pressure pain threshold decrease in CTTH but not ETTH**
- **Temporal or spatial summation of peripheral stimuli might have a role in predisposed to ETTH**
- **Pain sensitivity increase in CTTH**

Nociceptive reflexes and pathways

Exteroceptive silent periods in the temporalis muscle

- Second temporalis extero-ceptive silent period (ES2) was absent or reduced in CTTH
- ES2 are inhibited by serotoninergic pathways and activated by nicotinic cholinergic mechanisms

Nociceptive reflexes and pathways

Blink reflex

- **Positive association between R1 latency and disease duration in TTH - indicate that hypoactivity of brainstem neuron over time.**
- **Normal R2 Amp and area**
- **R2 recovery cycle was decreased (double stim) - reduced excitability of excitatory brainstem interneurons.**

Nociceptive reflexes and pathways

Biceps femoris flexion reflex

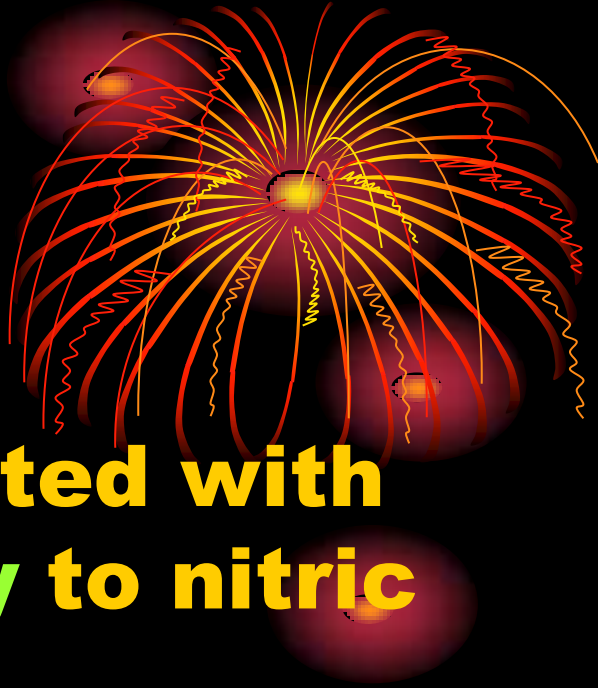
- **CTTH : Lower thresholds of the nociceptive flexion reflex and lower pressure pain thresholds**
- **Cause : Might Dysfunction of endogenous antinociceptive systems**

Structural brain changes

- Significant decreases in tissue mass (MRI)
- Reduced density of the cortical grey matter

Neurotransmitters

Nitric oxide



- **CTTH might be associated with central supersensitivity to nitric oxide**
- **L-NG-monomethyl arginine citrate (L-NMMA) – NO inhibitor , reduction of headache and muscle hardness**

Neurotransmitters

Neuropeptides

Calcitonin-gene-related peptide (CGRP)

- **A neurotransmitter that is active in the trigemino-vascular system**
- **CGRP: raised during migraine and cluster headache attacks**
- **TTH: CGRP increase while pulsating pain – suggest pathophysiology related to migraine**

Neurotransmitters

Neuropeptides

Substance P, neuropeptide Y, and vasoactive intestinal peptide

- **No differ between CTTH and health**
- **ETTH: higher Substance P and lower β-endorphin**
- **CTTH: metenkephalin in the CSF increase**

An imbalance between pronociceptive and antinociceptive mechanisms

Neurotransmitters

Serotonin (5-HT)

- **Increase 5-HT turover (opposite migraine)**
- **Plasma and platelet 5-HT concentrations are raised in patients with ETTH- but one report decrease**
- **Uptake of 5-HT in the platelets was decreased in ETTH but health in CTTH**
- **Migraine with mild headache , resemble TTH – response to sumatriptan**

Psychological studies

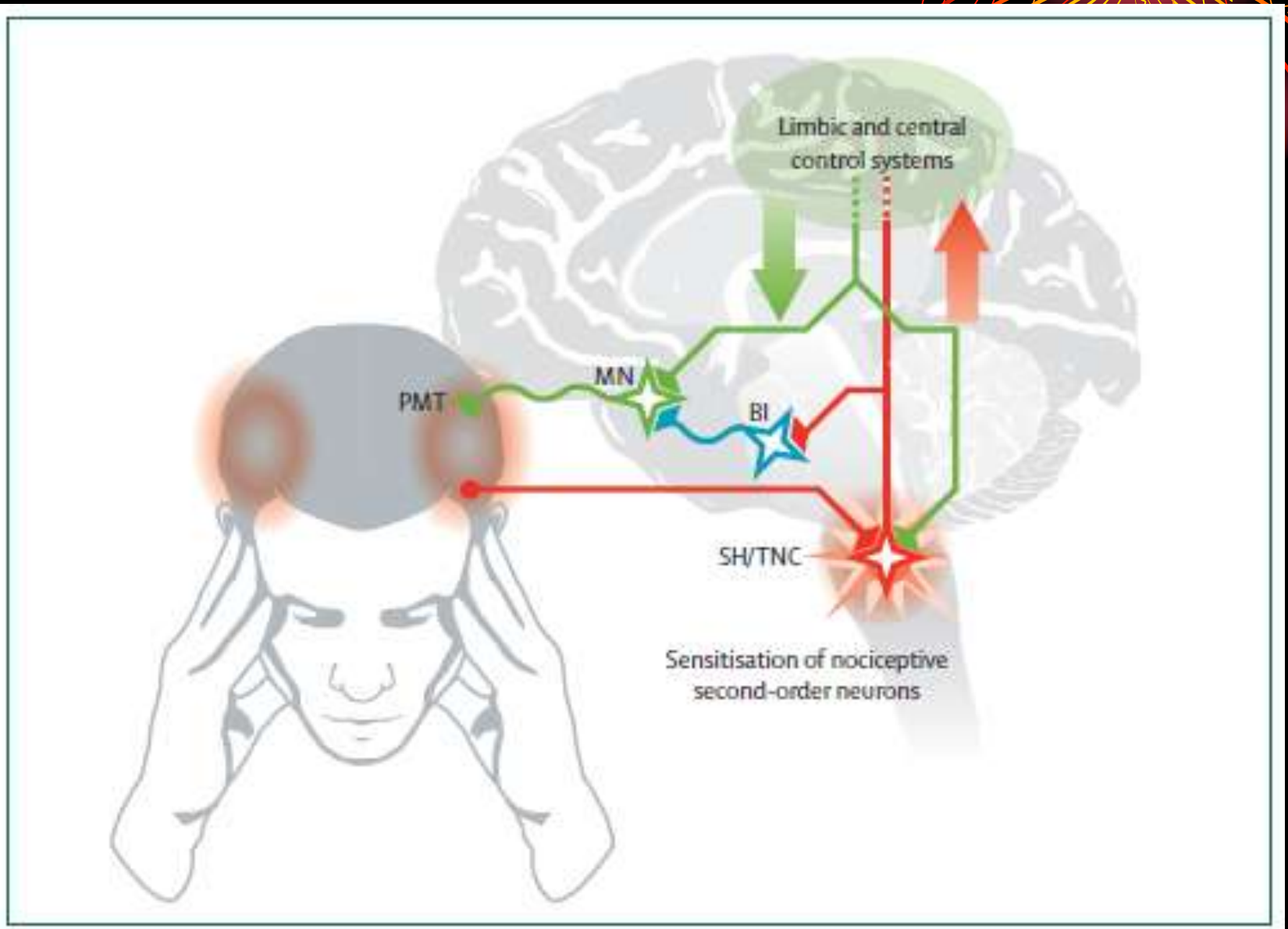
TTH Risk factor

- **Emotion disturbance, stress, mental tension**
- **CTTH: Increase frequency and severity life events – daily hassles**
- **Increase depression scale but not overt**

Genetics

- Increase genetic risk of CTTH , but not ETTH.
- One 11199 twin study. Genetic risk influence frequency ETTH, not infrequency

A model for TTH pathogenesis

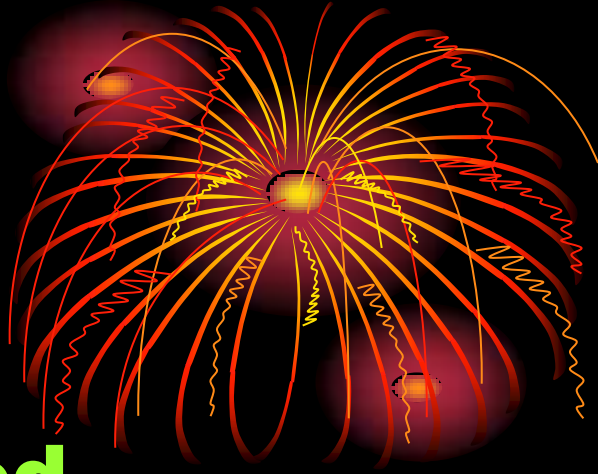


- Emotional mechanisms increase muscle tension through the limbic system, reduce tone in the endogenous antinociceptive system.
- Long-term potentiation or sensitisation of nociceptive neurons and decreased activity in the antinociceptive system gradually lead to CTTH
- Genetic components are likely to promote the psychological and central changes that lead to CTTH
- ETTH: environmental factors are the main cause

Pathogenic models:

- propose that TTH and Migraine are the opposite ends of a phenotypic spectrum of the same disorder

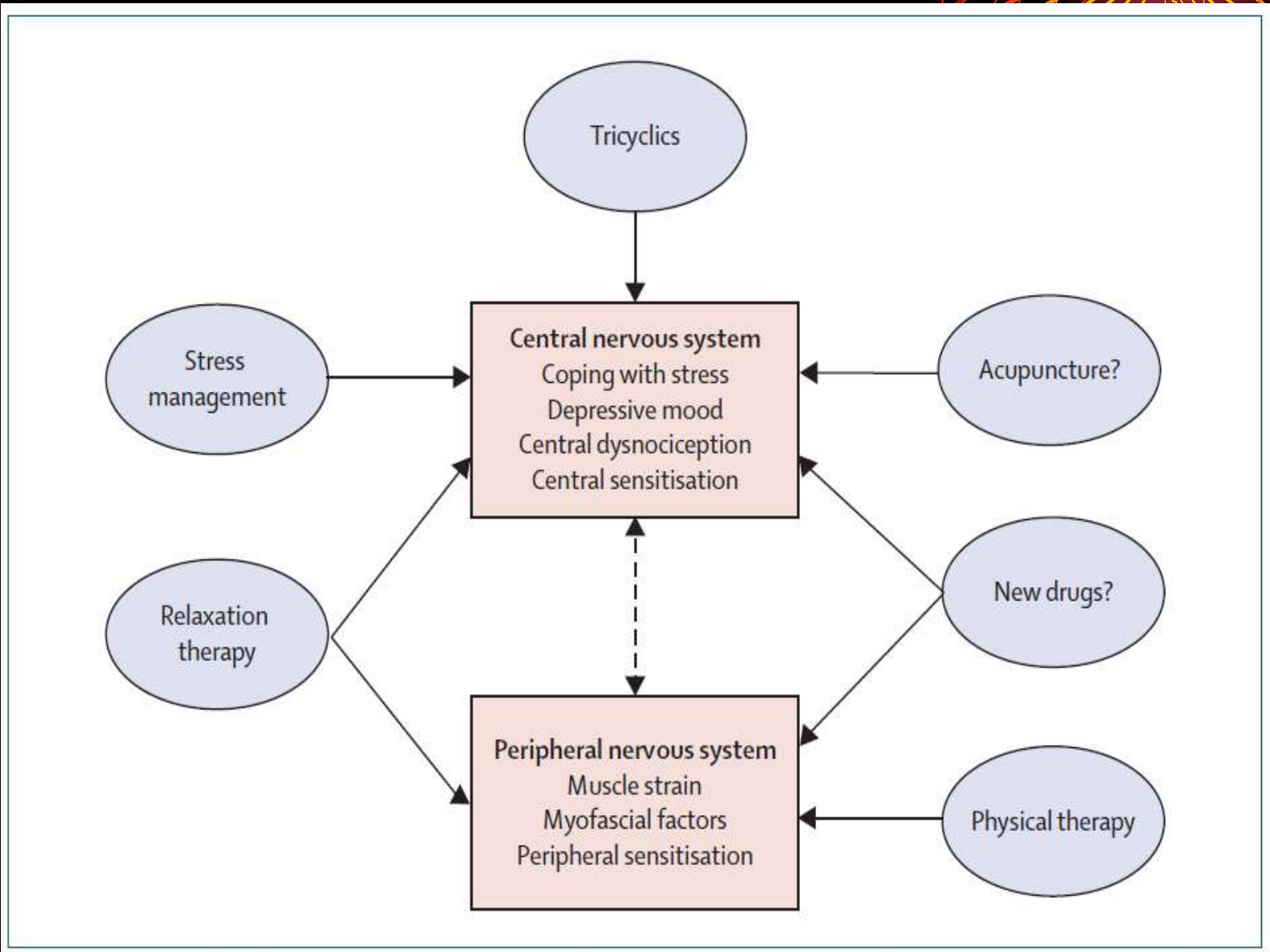
Treatment



Tx for TTH can subdivided

- **Shortterm, abortive (mainly pharmacological) treatment of each attack**
- **Long-term, prophylactic (pharmacological or non-pharmacological) treatments**

Treatment


Tricyclics
Stress management
Central nervous system
Coping with stress
Depressive mood
Central dysnociception
Central sensitisation
Acupuncture?
Relaxation therapy
New drugs?
Peripheral nervous system
Muscle strain
Myofascial factors
Peripheral sensitisation
Physical therapy

Acute pharmacotherapy

- First-line: NSAID
  - Aspirin (500 mg or 1000 mg)
  - Ibuprofen (800 mg)
  - naproxen sodium (825 mg)
  - Lumiracoxib (200mg bid)
- combination
  - Caffeine (130 mg or 200mg)
  - Sedatives, or tranquillisers

Prophylactic pharmacotherapy

- Tricyclic antidepressants-
  - most wide use- not all superior efficacy to placebo
  - Increases in serotonin by inhibition of its reuptake, endorphin release, or inhibition of NMDA receptors
  - Discontinuation of treatment after 6 months, regardless of the efficacy

Prophylactic pharmacotherapy

Selective serotonin reuptake inhibitors (SSRI)

- **Not as effective for prevention of TTH**
- **Can be tried in subgroups of patients not tolerate TCA**
- **Venlafaxine: 50% or more improvement (mild headache)**

Prophylactic pharmacotherapy

- **Tizanidine (6–18 mg per day)- antispasmodic drug ,need combine amitriptyline**
- **Topiramate: anticonvulsant – 100mg per day ,effective**
- **Botulinum toxin : no role in prophylactic ETTH**

Non-pharmacological treatments

- Relaxation and EMG biofeedback therapies
- Cognitive behavior intervention
- Stress management therapy and amitriptyline (≤100 mg day) was more effective in patients with CTTH

Non-pharmacological treatments

others

- Various physical therapy: positioning, ergonomic instruction, massage, transcutaneous electrical nerve stimulation
- Physiotherapy and spinal manipulation + nucal latex band
- Acupuncture : controversial
- Oromandibular treatment : occlusal splints, therapeutic exercises

Conclusion

- Clinically and pathophysiologically heterogeneous
- Pathogenesis:
  - Episodic TTH - pericranial myofascial mechanisms
  - CTTH: inadequate endogenous pain control
- Tx: ETTH – NSAID

  CTTH - ?!