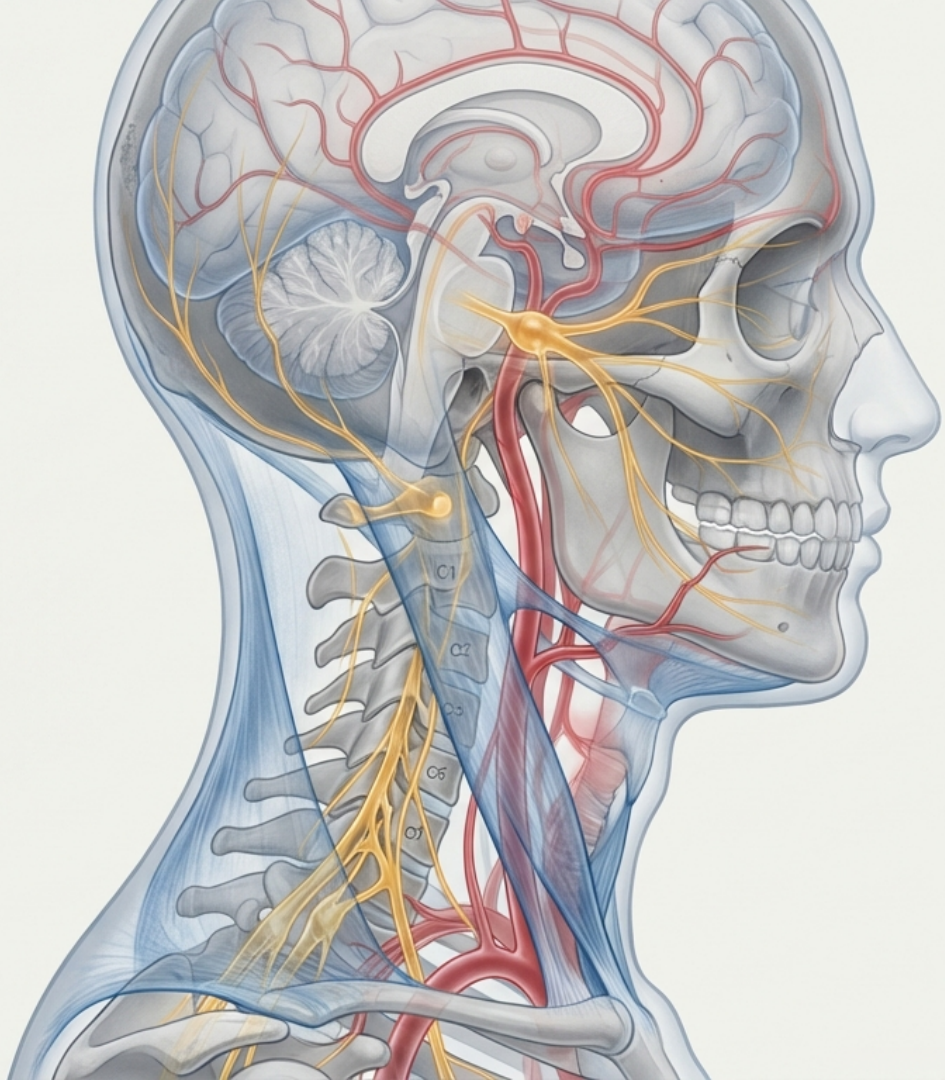
The Origin of Migraines: An Evolutionary Investigation

From Vascular Theory to the Neuromyofascial Dynamics of Chronic Pain

While the symptoms of migraine have remained constant throughout human history, the scientific understanding of their mechanism has undergone three distinct paradigm shifts: Vascular, Chemical, and Structural.

Dr. G. Blair Lamb

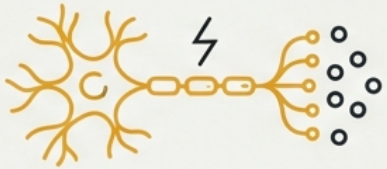


Three Eras of Scientific Understanding

1950s - 1980s:
The Vascular Era

Focus on blood vessel dilation and vasoconstriction. Pain viewed as a vascular event.

1990s - Present:
The Neural Era



Focus on Neuropeptides (CGRP) and the Trigeminal Ganglion. Pain viewed as a chemical cascade.

The Missing Link:
The Neuromyofascial Era

Focus on the structural "driver"—post-traumatic fibrosis—that initiates the chemical cascade.

Previous eras were not wrong, but incomplete. NMF Science identifies the physical origin of the established chemical mechanisms.

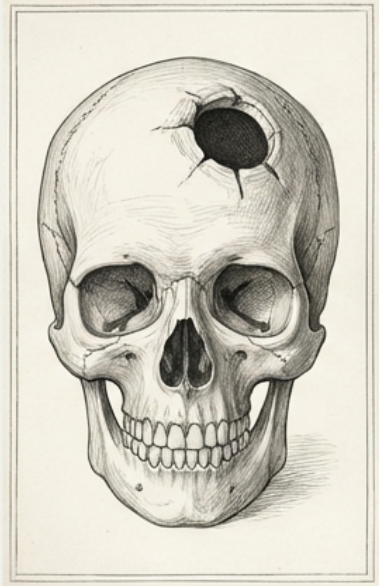
A 7,000-Year Search for Relief

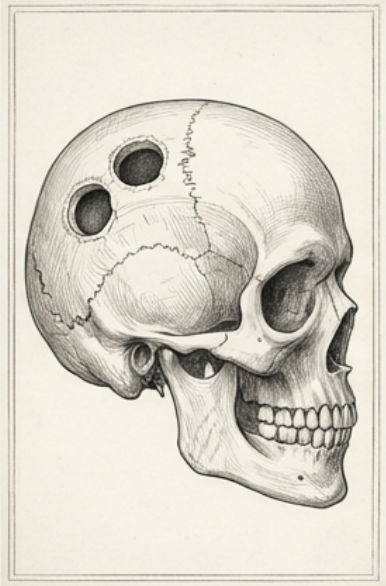
Archaeological evidence dates back 7,000 years across Greek, Roman, Peruvian, and Chinese history.

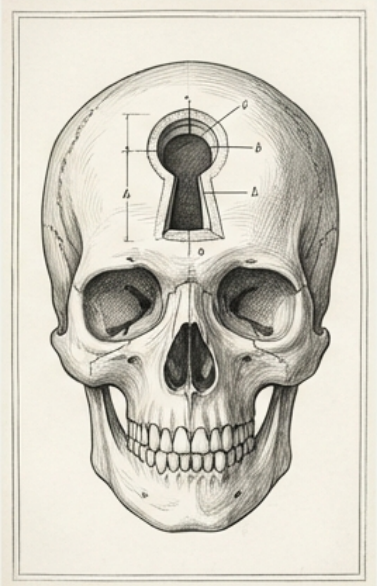
The Procedure: **Trepanation** (early craniotomy).

The Indication: Head trauma, infection, and chronic headaches.

The Insight: Since the dawn of medicine, **physical intervention on the skull structure was linked to headache relief**.







Clinical Editorial

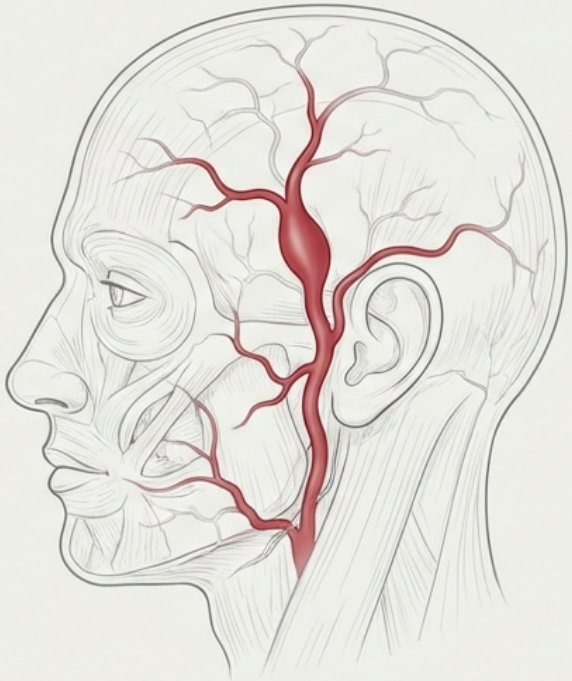
The Vascular Theory (1950s – 1980s)

Core Theory: Migraines were believed to be caused by intracranial and extracranial blood vessel dilation.

Key Evidence (Mid-20th Century):

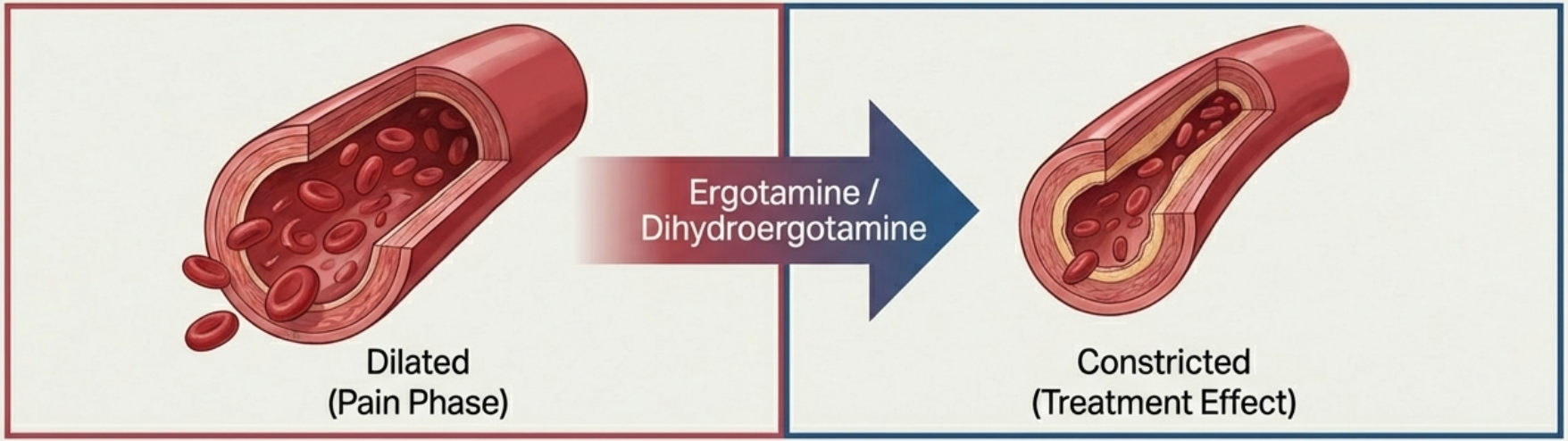
- Cerebral angiography consistently demonstrated dilation of large extracranial temporal arteries during attacks.
- Conversely, constriction was observed during the aura phase.

Key Figure: Harold G. Wolf, whose research defined the consensus of this era.



Clinical Editorial

The Era of Vasoconstriction



Pharmaceutical Context (1970s - mid-1980s): Primary treatments worked via vasoconstriction.

The Clinical Reality: These drugs offered genuine relief, and this relief coincided with a reduction in arterial pulse amplitude.

The Conclusion: Success reinforced the Vascular Theory, even though it was treating a downstream symptom rather than the origin.

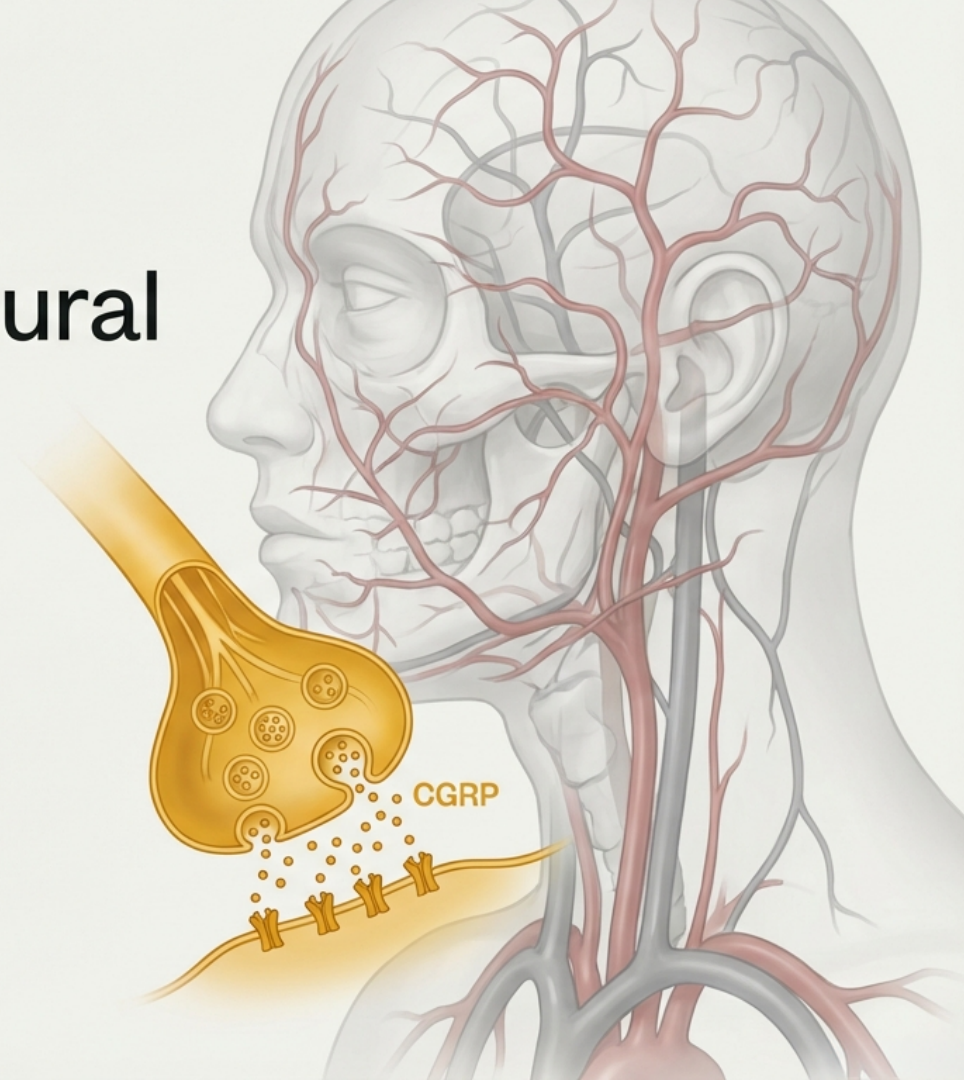
Clinical Editorial

The Paradigm Shift: From Vascular to Neural

The Shift (1990s): The scientific model moved away from simple vasodilation toward a **neuropeptide-mediated** neural mechanism.

The Culprit: Calcitonin Gene-Related Peptides (**CGRP**).

Context: Research demonstrated that vascular changes were secondary to neural activation, specifically involving the Trigeminal Nerve system.



Clinical Editorial

Evidence for the CGRP Mechanism

Goadsby et al (1990)

Found substantial elevation of CGRP in blood taken from the external jugular vein during spontaneous migraine attacks.

Goadsby & Edvinsson

Demonstrated that triptans normalized CGRP levels. Showed that experimental activation of the trigeminal ganglion directly released CGRP.

Olerson (2004)

Used CGRP receptor antagonists to effectively stop acute migraines.

Lassen (2008)

Showed that IV administration of CGRP provoked migraines in patients.

Clinical Editorial

The Unanswered Question

Current Consensus: The origin of the CGRP release is generated by the activation of the trigeminal ganglion.

The Gap: What activates the ganglion? Theories suggest “nerve root compromise” or “mechanisms yet to be determined.”

We identified the gun (Ganglion) and the bullet (CGRP), but not who pulls the trigger.

Clinical Editorial

Enter Neuromyofascial (NMF) Science

Definition: NMF Science is the study of post-traumatically induced myofascial fibrosis.

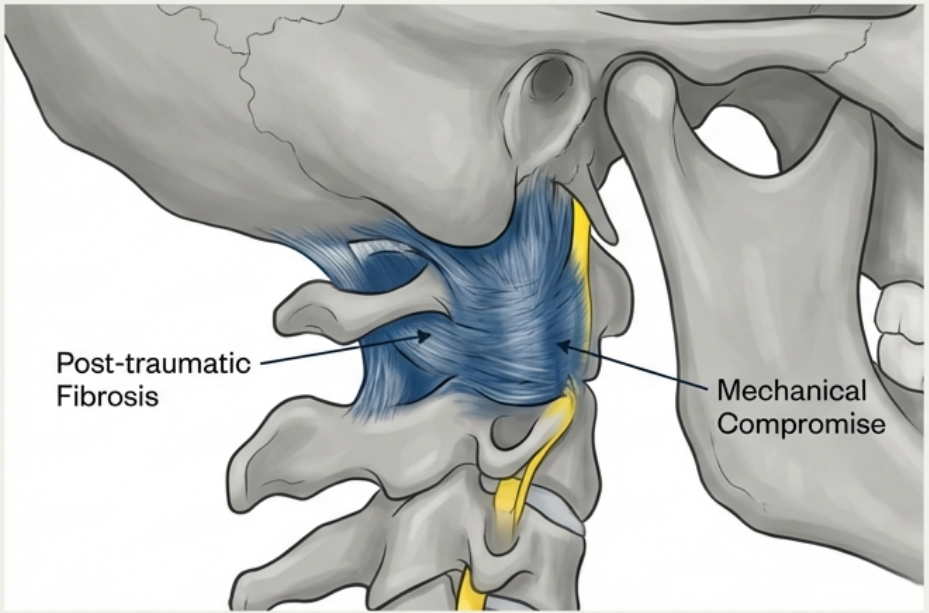
Origin: Investigated by Dr. G. B. Lamb starting in 1995.

Scope: Fibrosis surrounding the spine and limbs.

Pathology: Fibrotic tissue contributes to neurological complications including Migraine, Thoracic Outlet Syndrome, and nerve entrapment.

CLINICAL EDITORIAL

The Structural Driver of Classical Migraine

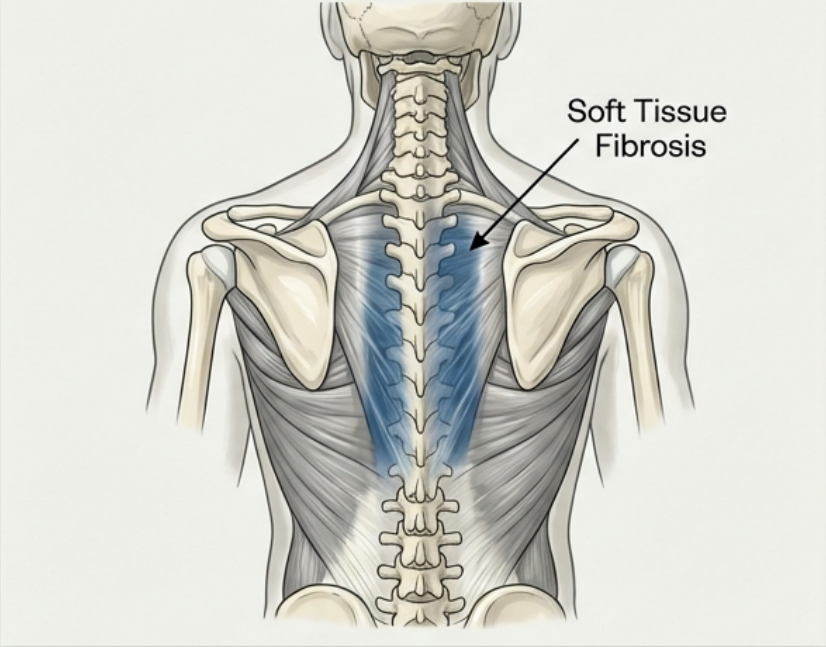


Mechanism: Post-traumatic fibrosis forms at the Craniocervical Junction.

The Trigger: This fibrosis acts as the driver site, physically compromising the nerve root.

The Cascade: This mechanical irritation triggers the Trigeminal Ganglion to release CGRP.

“Complex Patterns: Post-Concussion & Cluster Migraines” in Founders Grotesk



- **Target Profile:** Post-concussion syndrome and cluster migraines.
- **NMF Discovery:** Complex patterns are often driven by soft tissue thoracic fibrosis, lower than the classical migraine site.
- **Pathology:** Soft tissue myelopathic pathology contributes to complex craniofacial pain.

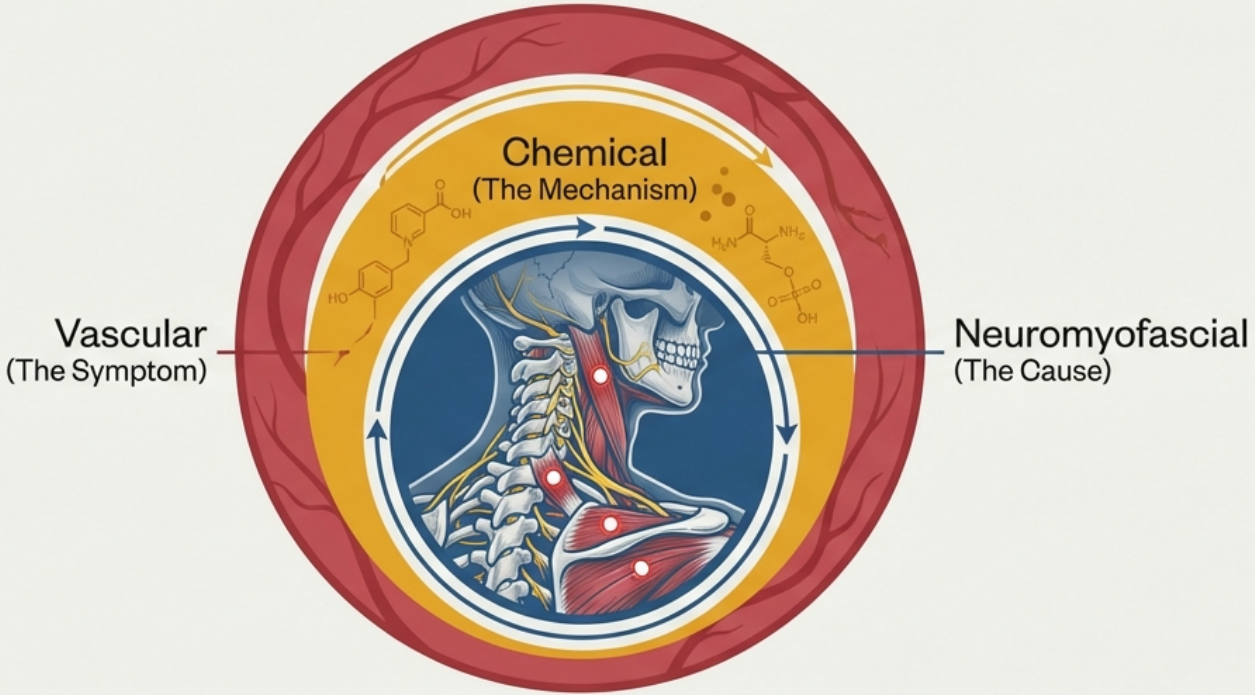
Detection and Recovery

Auditing

Release

- **Methodology:** NMF Science uses specific auditing methods (examinational and procedural) to detect fibrotic sites that standard imaging often misses.
- **Treatment Goal:** Detect the specific sites of post-traumatic fibrosis and release the injured tissue.
- **Outcome:** Recovery of the craniocervical junction removes the mechanical trigger, preventing the chemical cascade.

A Unified Theory of Migraine



Modern medicine has successfully mapped the chemical signature of migraine (CGRP).

NMF Science completes the picture by identifying and treating the structural pathology that triggers it.

 | References: Wolf, H.G. (Vascular Theory); Goadsby et al. (1990); Goadsby & Edvinsson (CGRP); Lassen (2008); Olerson (2004).