In a 1987 article from Neurology (Schoenen J, Jamart B, Gerard P, Lenarduzzi P, Delwaide PJ. Exteroceptive suppression of temporalis muscle activity in chronic headache. Neurology. 1987 Dec;37(12):1834-6), it was shown that:

1. Clenching the teeth fires the temporalis muscle, and its tone can be measured with surface EMG. [SURFACE]

2. Electrical stimulation of the sensory fields of the Trigeminal nerve (face) sends sensory information into the trigeminal cervical nucleus.

3. This sensory information to the trigeminal cervical nucleus is then sent to the pontobulbar reticular formation (lateral and/or medial medullary tegmentum).

4. The pontobulbar reticular formation (lateral and/or medial medullary tegmentum) is inhibitory to the motor nucleus of the Trigeminal nerve, which causes a significant reduction of the temporalis EMG activity.

5. The efficiency of this inhibitory reflex is dependent upon summation from the Hypothalamus-Periaqueductal-Raphe Magnus Nucleus.

6. Since the Hypothalamus-Periaqueductal-Raphe Magnus Nucleus loop also is the primary nociceptive, measuring the effectiveness of the sensory field stimulation driven inhibition of temporalis tone as measured with a surface EMG may represent an objective measurement of the efficiency of the Hypothalamus-Periaqueductal-Raphe Magnus Nucleus descending nociceptive inhibitory pathway.

7. An abnormality is technically termed “shortened duration of the ES2”
Brainstem-mediated antinociceptive inhibitory reflexes of the temporalis muscle were investigated in 82 patients (47 F, 35 M, mean age 28.3 years) with acute posttraumatic headache (PH) following whiplash injury but without neurological deficits, bone injury of the cervical spine or a combined direct head trauma on average 5 days after the acceleration trauma.

Latencies and durations of the early and late exteroceptive suppression (ES1 and ES2) and the interposed EMG burst (IE) of the EMG of the voluntarily contracted right temporalis muscle evoked by ipsilateral stimulation of the second and third branches of the trigeminal nerve were analyzed and compared to a cohort of 82 normal subjects (43 F, 39 M, mean age 27.7 years).

Highly significant reflex alterations were found in patients with PH with a shortening of ES2 duration.

These findings indicate that acute PH in whiplash injury is accompanied by abnormal antinociceptive brainstem reflexes. We conclude that the abnormality of the trigeminal inhibitory temporalis reflex is based on a transient dysfunction of the brainstem-mediated reflex circuit mainly of the late polysynaptic pathways.

The reflex abnormalities are considered as a neurophysiological correlate of the posttraumatic (cervico)-cephalic pain syndrome.

They point to an altered central pain control in acute PH due to whiplash injury.

THESE AUTHORS ALSO NOTE:

INTRODUCTION

“Eighty percent of patients with whiplash injury suffer from acute posttraumatic headache (PH)”
An adequate objective biological marker for PH is the inhibitory temporalis antinociceptive reflex (ITR), which is involved in central pain processing.

Also, the inhibitory jaw-opening reflex is used as an electrophysiological marker of the descending pain control system.

The basics of the descending pain control system consists of various brain stem structures including the periaqueductal grey (PAG) matter and the nucleus raphe magnus (RMN), which are responsible for endogenous pain control on the trigeminal and spinal level.

The basic concept is that an impairment of the PAG-RMN descending inhibitory pain control system results in more pain and a proportionate shortening of inhibitory temporalis antinociceptive reflex (ITR), an inhibitory trigemino-trigeminal reflex.

“It was the purpose of this study to investigate the ITR in patients with acute PH following whiplash injury compared to a control group of healthy human volunteers.”

SUBJECTS

Surface EMG recordings of the ITR were performed on 164 individuals, 82 with acute whiplash injury (<14 days). [SURFACE]

Exclusion criteria for this study included direct head trauma, direct neck trauma, cervical spine fracture, neurologic deficits, previous brain disorders history of headache (e.g. migraine, chronic tension-type headache) or psychiatric disorders (e.g. depression).

All patients suffered from an acute posttraumatic cervico-cephalic pain syndrome with a dull pressing headache of occipital preponderance. The control group consisted of normal volunteers who were age and sex matched.

During the reflex examination the awake subjects were lying in a supine position with eyes closed. During maximal voluntary contraction the surface EMG of the right temporalis muscle was recorded [SURFACE]

RESULTS

All whiplash patients were classified as grade II according to the Quebec Task Force. 91% of these patients had a bilateral, dull pressing headache that was mainly occipital.
“The whiplash injury patients showed an impressive shortening of ES2 duration in the acute posttraumatic stage compared to the healthy, symptom-free controls.”

“The striking finding and the predominant feature of patients with PH was a significant shortening of ES2 of the ITR.”

“This ES2 shortening was independent of whether the patients took analgesics/antiphlogistics or muscle relaxants in the acute posttraumatic stage.”

“No correlations were found between ES2 duration and psychological factors such as subjective impediment by pain or depressive mood.”

DISCUSSION

The authors assessed 82 patients with acute PH due to minor whiplash injury and compared them to 82 sex- and age-matched healthy volunteers.

“We hypothesized an abnormal trigemino-trigeminal reflex behaviour supporting the idea of altered central pain processing as a mechanism in the pathogenesis of acute PH.”

The authors note that serotonin may inhibit the pain synapses in the trigeminal nucleus.

Also, serotonin may inhibit the trigeminal motor nucleus, causing the temporalis ES2 duration shortening.

This would mean that the temporalis ES2 shortening is caused by a dysfunction of the central pain control system with a disturbance of the serotonergic projections to the trigeminal motor nucleus.

Neuronal structures of the brainstem, which mediate this temporalis reflex arc, are influenced by the limbic system, the PAG, and the hypothalamus.

“Direct biomechanical lesions of those structures by an impact of the acceleration forces during the whiplash trauma are not probable in patients with minor whiplash injury.”

Therefore, the authors suggest that altered afferent input will alter the function of the PAG-RMN circuit.
Specifically, nociceptive and non-nociceptive mechanoreceptor inputs converge centrally onto wide dynamic range (WDR) neurons in the trigeminal-cervical nuclear complex, which then summate to shorten the ES2 duration.

“These WDR neurons are under direct descending inhibitory control of the PAG and RMN.”

“Bearing this in mind, it is conceivable that an augmented tonic proprioceptive and nociceptive input due to a painful sprain of the cervical muscles related to the acceleration trauma and a following activation of muscle spindles and nociceptive group III and IV afferent fibres causes a facilitation of the trigeminal motor nucleus by an activation of the (lateral) reticular system.”

“The connections of the upper cervical dorsal roots with the spinal trigeminal tract are considered as an afferent pathway of the trigemino-trigeminal circuit modulating ES2.”

The authors believe that this is a common pathomechanism responsible for the altered endogenous pain control in whiplash injury PH.

“Our studies indicate a transient traumatic alteration of the function of the nervous structures involved in the antinociceptive reflex arc in patients with whiplash injury during the early posttraumatic stage.”

“One is tempted to speculate that ES2 shortening can be considered as a biological marker of the PH.” [USING SURFACE EMG]

FROM DAN MURPHY

Importantly for many chiropractors, this study and the one in 1987 used surface EMG.

The model used is that whiplash injury alters the mechanical input into the central neural axis causing a disruption of the Hypothalamus-Periaqueductal-Raphe Magnus Nucleus descending nociceptive inhibitory pathway, resulting in both headache pain and a shortened duration of the ES2 reflex of the temporalis.

“The basic concept is that an impairment of the PAG-RMN descending inhibitory pain control system results in more pain and a proportionate shortening of inhibitory temporalis antinociceptive reflex.”