Indian J Physiol Pharmacol 2007; 51 (4) : 333–344

ROLE OF TEMPORALIS MUSCLE OVER ACTIVITY IN CHRONIC TENSION TYPE HEADACHE : EFFECT OF YOGA BASED MANAGEMENT

R. BHATIA, G. P. DUREJA*, M. TRIPATHI**, M. BHATTACHARJEE***, R. L. BIJLANI AND R. MATHUR¤

*Departments of ¤Physiology, *Anaesthesia and **Neurology, All India Institute of Medical Sciences, Ansari Nagar, New Delhi – 110 029*

(Received on May 10, 2007)

Abstract : The role of central versus peripheral mechanisms has always been questioned while explaining the etiopathogenesis of chronic tension type headache (CTTH). The following study was done to study the role of muscle spasm in CTTH. 15 patients of CTTH and 7 age matched controls were included in the study and their m. temporalis EMG was recorded for one minute each during rest, mental activity and maximal voluntary contraction and subjective pain scoring was done by visual analogue scale. The results revealed significant overactivity of m.temporalis in CTTH patients at rest when compared with control subjects (P=0.01 and 0.03 left and right side respectively). After respective interventions namely non steroidal anti inflammatory drugs, botulinum toxin injections and yogic life style course, the EMG records revealed decrease in the mean EMG amplitude of m.temporalis during rest and mental activity more significantly after yoga based interventions (P= 0.03) and subjective pain scores decreased from 7.00 ± 2.10 to 2.00 ± 1.26 (P=0.02) supporting the beneficial effect of such non invasive techniques.

Key words : botulinum toxin yoga subjective pain scores

INTRODUCTION

The origin of pain in chronic tension type headache (CTTH) still remains controversial. Both, central (psychogenic) and peripheral (myofascial) dysfunctions have been implicated in its genesis (1–3). The former contention gains support from a few negative reports about any difference in EMG of pericranial muscles at either rest or during voluntary activity (4, 5), and an attenuation of the multi synaptic reflex namely the second exteroceptive suppression period (ES2) of temporalis muscle (that is the inhibition of voluntary EMG activity of the temporalis muscle induced by trigeminal nerve stimulation) (6–8). The latter observation is indicative of a disturbance in limbic control of the brainstem centres which, controls the excitability of medullary ES2 inhibitory inteneurons via serotonergic and presumably opioidergic mechanisms (9).

¤Corresponding Author
***Present address : Department of Physiology, JIPMER, Puducherry – 605 006

Moreover, nociceptive flexion reflex, which is influenced by endogenous opioid and serotonergic systems, is decreased in CTTH patients thereby, supporting an abnormality in these systems (10). The involvement of former system is further strengthened by the reports of an increase in metenkephalin levels in cerebrospinal fluid of these patients (11). Furthermore, a central dysfunction is favoured by the precipitating factors for headache in these patients include stress, mental tension and tiredness (12).

In the absence of a clear understanding about the CTTH pathophysiology the treatment is often incomplete. The current management strategies either target the peripheral mechanism by advocating muscle relaxants or central mechanism by advocating mental relaxation techniques, placebo or antidepressant therapy. The latter psychotherapeutic measures indicate the putative role of limbic system in CTTH.

We have studied the role of oral non-steroidal anti-inflammatory drugs, muscle relaxant (botulinum toxin) and yogic lifestyle management of chronic tension type headache to address the issue of central versus peripheral genesis of CTTH. Non steroidal anti-inflammatory drugs (NSAID) and botulinum toxin act peripherally by decreasing pain, muscle tenderness and spasm whereas, the yogic lifestyle exerts its influence centrally via probably limbic area and by alleviating the stress/mental tension related component of chronic pain. The degree of muscle spasm was assessed by recording EMG of pericranial muscle temporalis, during rest, mental activity and maximum voluntary contraction.

MATERIALS AND METHODS

The study proposal was approved by the Ethics committee for clinical research of All India Institute of Medical Sciences, New Delhi and in accordance with the declaration of Helsinki (13). The informed written consent of the patients was obtained.

CTTH patients were selected from the Pain and Neurology clinics of All India Institute of Medical Sciences (AIIMS). Their EMG was recorded before and after one month of intervention namely, a non-invasive yogic life style, oral non steroidal anti-inflammatory drugs (NSAID) or intramuscular injections of botulinum toxin.

Groups

The patients (n=21) were selected on the basis of the criterion laid for CTTH by International Headache Society (14), while the controls (n=7) comprised of healthy age matched volunteers. The patients of CTTH, were divided into three groups namely NSAID, Botulinum toxin (Botox) and Yoga groups based on the option of the patients (incidentally the patients with a prolonged history of severe headache had opted for yoga).The patients (aged 18–50 years) of CTTH, having episodes of headache for more than 15 days a month to 180 days a year for at least 2 years were included in the study (Tables I and II). Both male and female patients were included in the study (Table III). The patients requiring analgesic medication for more than three times a month, muscle relaxants, addiction to morphinomimetic drugs, or practicing yoga were excluded from the study.

TABLE I: Age profile of headache patients.

Age range (yrs)	*15-20*	*20-25*	*25-30*	*30-35*	*35-40*	*40-45*
n	2	2	5	4	1	1

The table depicts age profile of headache patients. Most patients belonged to young age group ranging from 15-35 years.

TABLE II: Duration of pain in headache patients.

Duration of pain (yrs)	<1	1	2	3	4	5	6	7	8	9	10
n	0	0	2	1	1	4	2	2	2	0	1

The table depicts duration of pain in chronic tension type headache patients. Note that most of the patients had a history of headache for more than 5 years.

TABLE III: Sex wise distribution of controls and headache patients.

Groups	*Male*	*Female*	*Total*
Control	5	2	7
NSAID	3	3	6
Botox	1	2	3
Yoga	4	2	6
Total	13	9	22

The table depicts sex wise distribution of control and headache patients.

Yogic life style management training

Each session extended from 0830 h to 1130 h. Every session consisted of lectures on yoga, meditation, fundamentals of nutrition along with individualised advice on diet and physical activity (Tables IVa and IVb). While, a practical session included training on various yogic postures (*asanas*), meditation and other relaxation techniques by the experts. At the end of 2 weeks of training, they were advised to continue the same regime and to report 2 weeks later (Visit II).

TABLE IVa: Details of recommended physical postures (YOGASANAS) (Duration: 1 hour approx).

I. Breathing techniques	
Dog breathing: breathing through mouth	1 min (3 times)
Tiger breathing: breathing through mouth, back concave breathing through nose back convex	6 times
Hands in and out breathing	6 times
Hands interlocked, kept on chest, stretching 3 position breathing	4 times in each position
Ankle stretch breathing	5 times
II. Loosening exercises	
Neck rolls	6 times
Shoulder rotation clockwise and anti-clockwise	6 times each
Elbows fold in and out	6 times
Wrist movements: up, down; left, right; clockwise, anti-clockwise	6 times each
Close and open fist, stretch fingers	6 times
Rotate arms alternatively	6 times
Twist body at waist	6 times
Sideways bend-alternate sides	6 times
Rotate knees clockwise and then anti- clockwise	6 times
Toes pointing outwards-up and down, sideways, clockwise and anti-clockwise ankle rotation	6 times each
III. Quick relaxation in Shavasana	
Focus on stomach	
Focus on breathing	
Synchronise movements of stomach and breathing	
Stomach out for breathing in Stomach in for breathing out	

Contd.....

IV. Asanas
(a) Standing Ardhachakrasana Padahastasana Ardhkatichakrasana Vrikshasana
(b) Sitting Ardhamatsyendrasana Paschimaanasana Konasana
(c) Lying on stomach Makarasana Bujangasana Dhanurasana
(d) Lying on back Uttitapadasana Sarvangasana Matsyasana Setubandhasana Pavanmuktasana
V. Deep relaxation in Shavasana
Voluntaraly relaxing each part of body by focussing attention on it, proceeding from the toes to the head.
VI. Pranayam
Bastrika Nadi shuddhi Bhramari
VII. Quick relaxation in Shavasana
Turn to side and sit up End with chanting in a mediative posture

TABLE IVb: Life style management course schedule (Integral Health Clinic, AIIMS).

Week 1		
Mon/Tue	*History Taking*	
Wed	9:00 AM	Fasting blood samples for FPG & Cholesterol (Pre)
	9:30 AM	Lecture: Introduction to yoga
	11:00 AM	Practice: Shavasana / yoga nidra
Thu	8:30 AM	Practice: Asanas
	9:30 AM	Break (Breakfast, music)
	10:00 AM	Lecture: meditation
	11:00 AM	LSGA (loosely structured group activity)
	11:15 AM	Practice: Meditation
	11:30 AM	Individualized advice: 2 patients
Fri	8:30 AM	Practice: Asanas
	9:30 AM	Break (Breakfast, music)
	10:00 AM	Lecture: Fundamentals of nutrition
	11:00 AM	LSGA
	11:15 AM	Practice: Meditation
	11:30 AM	Individualized advice: 2 patients
Sat	8:30 AM	Practice: Asanas
	9:30 AM	Break (Breakfast, music)
	10:00 AM	LSGA
	10:15 AM	Practice: Meditation
Week 2		
Mon	8:30 AM	Practice: Asanas
	9:30 AM	Break (Breakfast, music)
	10:00 AM	Film: Samatvam
	11:00 AM	LSGA
Tue	8:30 AM	Practice: Asanas
	9:30 AM	Break (Breakfast, music)
	10:00 AM	Film: stress management
	11:00 AM	LSGA
Wed	8:30 AM	Practice: Asanas
	9:30 AM	Break (Breakfast, music)
	10:00 AM	Lecture: About your illness
	11:00 AM	LSGA
	11:15 AM	Practice: Meditation/shavasana
	11:30 AM	Individualized advice: 2 patients
Thu	8:30 AM	Practice: Asanas
	9:30 AM	Break (Breakfast, music)
	10:00 AM	Lecture: vogic attitude in daily life
	11:00 AM	LSGA
	11:15 AM	Practice: Meditation/shavasana
	11:30 AM	Individualized advice: 2 patients
Fri	8:00 AM	Fasting blood samoles for FPG & Cholesterol (Post)
	8:30 AM	Practice: Asanas
	9:30 AM	Break (Breakfast, music)
	10:00 AM	Lecture: Stress management
	11:00 AM	LSGA
	11:15 AM	Practice: Meditation/shavasana
	11:30 AM	Individualized advice: 2 patients

Botulinum toxin injections

Botulinum toxin (BOTOX; Allergen, Inc, USA) was injected intratramuscularly after selecting appropriate tender points on the muscle. Botulinum toxin (50–60 U) was divided into 10–12 equal doses. Normal saline was used as a vehicle for injecting botulinum toxin.

EMG recordings: EMG of m. temporalis was recorded on Biopac Student Lab V3.6.2 (BSL) system (Biopac System Inc., CA, USA) through surface electrodes (Silver-Silver chloride disc electrodes, Nessler Medizintechnik, Germany). After cleaning the skin overlying the muscle one of the electrodes was placed 10mm lateral to the external angle of orbit and the other directly above the first while, the reference electrode was placed on the frontal bone (centre of the forehead).

EMG was recorded in sitting posture, during rest, mental activity and maximal voluntary contraction of the muscle till the onset of fatigue. The subjects were instructed to remain re1axed or serially subtract 7 from 100 or maximally clench their jaws till they felt, tired, respectively .The EMG records were obtained and the data was saved for off-line analyses.

Study plan

A written consent was obtained from the patient for their participation in the study. A detailed history regarding their headache was recorded. On each visit the blood pressure was recorded, the subjective assessment of headache was done by visual analogue scale (VAS) and the EMG of m. temporalis was recorded for 1 min each in various above mentioned states. EMG of the age matched volunteers (control group) of either sex (both male and female) was recorded only on the first visit.

Data analysis

The EMG record of rest, mental activity and maximum voluntary contraction was analysed bin wise (10 s each) to calculate the mean EMG amplitude. The integrated EMG record of maximal voluntary contraction was further analysed for the area under the curve and time for onset of fatigue. Three alternate bins of 10 s were considered. Friedman test did not reveal significant difference amongst the three bins and therefore the mean of these bins was used for statistical analysis. Kruskal-Wallis test was utilized for comparison between the groups. The comparison between first and second visit of the same group of patients was obtained by Wilcox on Signed Rank test in all the groups. The result was considered significant at 5% level of significance i.e., $P<0.05$. Out of 21 patients, 6 could not complete the study. Therefore the results of only 15 patients are presented.

RESULTS

EMG before intervention (Visit I)

The mean EMG amplitude of left and right m. temporalis in CTTH patients was significantly higher than controls (Table V). Amongst the experimental groups, Yoga and Botox groups of CTTH patients had notably higher mean EMG amplitude (Table V).

EMG after intervention (Visit II)

During visit II the NSAID group of patients showed a significant decrease in mean EMG amplitude of m. temporalis at rest ($P=0.04$, left side; $P=0.04$, right side) (Fig. 1) as compared to visit I. During the mental activity also there was a significant decrease in the mean EMG amplitude (Table VI). While, during maximal voluntary contraction of m. temporalis the increase in

TABLE V: EMG amplitude (μV) of temporalis muscle before any intervention.

Rest	*Control*	*NSAID*	*BOTOX*	*YOGA*	*P value*
Left	12.9±5.5	26.0±21.1	48.4±42.5	54.5±36.9	0.01
Right	13.5±8.7	28.8±14.7	49.2±46.7	49.9±25.7	0.03
Mental activity					
Left	26.4±27.8	25.3±25.4	63.5±18.7	37.5±21.3	0.07
Right	19.7±13.2	30.7±27.1	49.4±32.5	76.0±87.8	0.26
Max. contraction					
Left	680.4±282.2	422.9±278.1	453.6±176.8	319.8±103.4	0.06
Right	577.2±145.2	341.2±151.6	453.2±166.8	317.8±215.1	0.14

The table depicts the mean EMG amplitude of temporalis muscle in controls and chronic tension type headache patients during rest and activity before undertaking different interventions. (NSAID, non-steroidal anti-inflammatory drugs; BOTOX, botulinum toxin and yogic lifestyle).

TABLE VI: EMG activity (μV) during mental activity before and after different interventions.

	Left temporalis			*Right temporalis*		
	Before	*After*	*P Value*	*Before*	*After*	*P Value*
NSAID	25.30±25.40	15.94±6.35	0.06	30.76±27.07	12.96±9.12	0.03
BOTOX	63.58±18.78	20.87±l1.43	0.10	49.25±46.71	28.06±2.34	0.10
YOGA	37.54±21.33	16.44±4.17	0.03	76.02±87.74	30.99±26.80	0.03

The table depicts the effect of NSAID, BOTOX and YOGA on EMG activity during mental activity.

TABLE VII: EMG activity (μV) during maximal voluntary contraction before and after different interventions.

	Left temporalis			*Right temporalis*		
	Before	*After*	*P Value*	*Before*	*After*	*P Value*
NSAID	422.26±235.67	457.08±179.55	0.7	541.20±151.66	562.46±451.93	0.3
BOTOX	453.60±176.87	359.35±211.63	0.1	453.27±166.81	242.44±l05.89	0.1
YOGA	3l9.85±103.44	408.27±180.l	0.08	3l7.89±165.35	492.59±259.0	0.06

The table depicts the effect of NSAID, BOTOX and YOGA on EMG activity during maximal voluntary contraction.

the EMG amplitude, area under the curve of the integrated EMG and the time for the onset of fatigue were not statistically significant (Tables VII and VIII). The VAS pain ratings were significantly lower during visit II. (Tables IX).

The Botox group of patients showed a statistically significant (P=0.02) decrease in the EMG activity post intervention during

TABLE VIII: Area under the curve of integrated EMG of and time for the onset of fatigue m.temporalis during maximum voluntary contraction in the various study groups.

GROUPS		*Before*	*After*	*P value*
		NSAID		
Area under thecurve	Lt	2236.86±1705.16	2756.69±2103.21	0.24
	Rt	1635.00±1072.68	2013.6±1168.35	0.28
Time for onsetof fatigue	Lt	26.73±12.55	34.85±21.04	0.17
	Rt	27.43±14.60	34.69±16.9	0.12
		BOTOX		
Area under thecurve	Lt	1574.07±303.4	1304.9±1144.78	1.0
	Rt	22.55±12.66	786.86±275.12	1.0
Time for onsetof fatigue	Lt	22.55±12.66	34.00±15.83	0.1
	Rt	33.06±12.15	29.85±10.11	1.0
		YOGA		
Area under thecurve	Lt	1102.33±55.84	1304.91±1144.78	0.6
	Rt	1280.03±319.69	1533.26±963.41	0.17
Time for onsetof fatigue	Lt	20.44±4.71	27.54±7.56	0.08
	Rt	20.93±6.64	27.16±6.62	0.08

The table depicts the area under the curve (μV sec) of integrated EMG and time for onset of fatigue (sec) in NSAID, BOTOX and YOGA groups of patients.
*Lt – Left Temporalis
*Rt – Right Temporalis

rest (Fig. 1). The observed apparent decreases in the mean EMG amplitude during mental activity and maximum voluntary contraction, the area under the curve and the time taken for the onset of fatigue were not statistically significant probably because of small sample size (Tables VII and VIII). The EMG amplitude was even lesser than that in control subjects. The subjective pain ratings were higher (Table IX) and the patients complained of pain at the site where botulinum toxin was injected.

TABLE IX: Visual analogue scale rating of headache pre and post intervention.

Group	*VAS (before)*	*VAS (after)*	*P value*
NSAID	7.40±1.50	3.67±1.97	0.026*
BOTOX	8.67±l.50	5.67±2.08	0.109
YOGA	7.00±2.10	2.00±1.26	0.027*

The table depicts the comparative values of VAS before and after different interventions. The NSAID and Yoga groups reported significant improvement in their pain status following the respective interventions. (*P<0.05 is significant by Wilcoxan Signed Rank test).

During visit II in yoga group of patients EMG was significantly (P=0.03) reduced as compared to their pre-yoga (Visit I) EMG at rest (Fig. 1). During mental activity also a significant decrease in the mean EMG amplitude was observed (Table VI). While the increase in EMG amplitude during maximal contraction, area under the curve of the integrated EMG and the time for the onset of fatigue was also not statistically significant

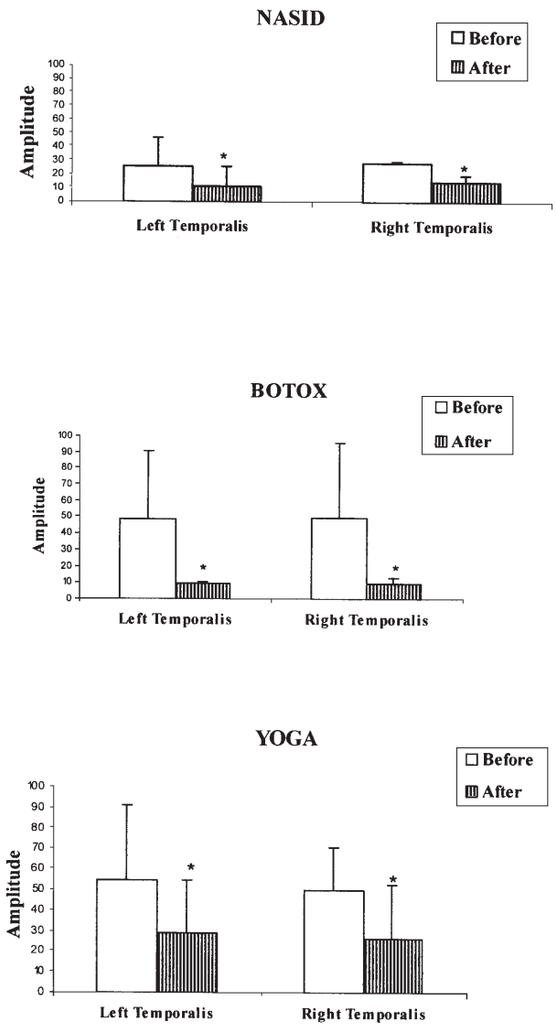

Fig. 1 : Effect of different treatment modalities namely NSAID medication, BOTOX injection and life style management course on EMG amplitude of temporalis muscle at rest.

(Table VIII). The pain ratings on VAS also improved after the intervention and patients reported a significant relief from their symptoms. (Table IX).

DISCUSSION

The results of our EMG study in CTTH patients suggest an overactivity of m.temporalis (both left and right sides) at rest which decreased significantly after undertaking any of the proposed interventions, including analgesic medication (NSAID), muscle relaxant (botulinum toxin injections) or lifestyle management course (Yoga). EMG during mental activity also decreased significantly while only post yoga EMG revealed an improvement in the ability to clench jaws during maximal voluntary contraction of m. temporalis. The area under the curve of the integrated EMG and the time for the onset of fatigue during maximum voluntary contraction were not statistically significant after any of the interventions. The results of subjective pain scores between pre and post intervention also revealed an improvement after botulinum toxin injections, NSAID administration and yoga, in this order.

We selected only CTTH amongst other headaches namely; common migraine, migraine with interparoxysmal headache and cluster headache for our study because of possible variation in their aetiology. However, the term tension type headache embraces a number of commonly used terms including tension headache, muscle contraction headache, psycho myogenic headache, stress headache, ordinary headache and psychogenic headache (14). The profile of our patients is in *pari passu* with that described for CTTH patients for example our patients belonged to either sex and of almost all age groups (15–45

years). The headache was described characteristically as a band like pain experienced in frontal, parietal and occipital regions; the duration of attacks ranging from few hours to several weeks (15 days per month for 6 months or 180 days per year) which was not associated with any underlying pathology.

We selected NSAID administration, local botulinum toxin injections and life style management course as treatment options for CTTH patients. These interventions also provided cues for the relative roles of central and peripheral components in the genesis of headache as they differed in their mechanism of action. NSAID acts at the peripheral level by inhibiting enzyme cyclooxygenase (COX-1 and COX-2) thereby blocking the prostaglandin (prostacyclin and thromboxane A2) generation (15). NSAIDs are the first choice of treatment in CTTH because of their easy availability, low cost and scope for self medication. However, their intake may cause hypersensitivity reactions in some of the patients and prolonged use may lead to serious side-effects such as damage to gastric mucosa amongst several others (16).

Recently, skeletal muscle relaxant botulinum toxin has emerged as a useful treatment modality to relieve muscle spasm of CTTH. It is believed to act selectively on peripheral cholinergic nerve endings and block the release of acetylcholine from presynaptic cholinergic nerve endings, without affecting the neuronal conduction or acetylcholine synthesis or storage (17). Once injected, the type A neurotoxin molecule selectively binds to the motor nerve terminal through high affinity receptors. After internalisation, the light chain of neurotoxin molecule is released into the cytoplasm of the nerve terminal and it then acts to block vesicle fusion in the nerve membrane, there by preventing the release of acetylcholine into the neuromuscular junction. Botulinum toxin temporarily blocks acetylcholine release by specific proteolysis of SNAP-25 (Synaptosome associated protein, 25 kD). Evidence indicates that chemical denervation of neuromuscular junction by this toxin results in expansion of end plate. region and growth stimulation of collateral axonal sprouts (l8). The invasive nature of therapy, the high cost and the need to repeat the treatment after about 6 months are the primary limitations of such an intervention.

The life style management course has been specifically designed to train the patients in yogic practices, life style, transcendental meditation and awareness about their disease. It is primarily based on the traditional Indian system of medicine and serves as a combined approach towards the mental and physical well being of the individual. The beneficial effects of these techniques have objectively been monitored by several researchers in the past five decades. A decrease in muscle activity (EMG), blood pressure, heart rate and an increase in skin resistance and skeletal muscle blood flow are some of the commonly reported effects (19). Besides, EEG changes suggestive of increased intensity of slow alpha waves and occasional theta wave activity are consistent with generalised decreased sympathetic activity which is mediated through an integrated hypothalamic and limbic response (20). Meditation has recently

been shown to increase blood flow to neural sites including limbic system like hippocampus along with sensory and higher order association regions (21).

It is generally recognised that there may be myogenic and psychogenic factors of variable importance in the pathogenesis of CTTH (2, 3, 8). The question of muscle activity is to be considered as a cause, consequence or just one of the factors influencing the genesis of CTTH (1).

It appears from the results of our study that the peripheral factors are predominantly significant since both the interventions namely NSAID and botulinum toxin, acting peripherally had improved the headache status. However, yoga too is believed to influence peripheral sites including muscle relaxation which explains the improvement in the headache status of yoga group of patients. There is a robust evidence now to suggest that meditation and yogic asanas involve and influence various neural sites involved in sensory imagery (hippocampus and higher order association regions) and executive systems (dorsolateral prefrontal cortex, anterior cingulate gyrus, striatum, thalamus, pons, and cerebellum (21) respectively. Therefore, it will be inappropriate to ignore the central actions of yoga. It is obvious from yoga group of patients who reported a dramatic improvement in the general. feeling of well being which was missing in both NSAID and Botox groups. The former group complained of frequent changes in medicines and their dosages besides a gastrointestinal distress while Botox group of patients complained of pain at the injection sites and a corresponding higher VAS score. Besides, their EMG revealed a flaccid state of the jaw clenching muscles, which is disturbing to the patient. Contrary to botulinum toxin, yoga not only significantly decreased EMG amplitude but also improved the quality of life, suggesting a remarkable beneficial effect of these simple, inexpensive and non-invasive interventions in CTTH.

If the mechanism underlying CTTH were solely peripheral, there should be a reduction in the dosage, improvement in the drug efficacy and the general well being of the patient with time which unfortunately failed to happen in our patients. Therefore, it can logically be derived from the aforesaid that the central component is predominant in the genesis or rather in the progression of the disease especially at this juncture when their headache has attained a state of chronicity.

It is not easy to comment about the primary site in the genesis of CTTH of as well as to establish a cause-response relationship since CTTH patients have a prolonged history (2–10 years) and a higher frequency of headache episodes (180 times per year for 2 years at least). This is by definition included in CTTH. It is therefore true that these patients suffer from chronic pain syndrome with all its attributes of negative valence. The negative valence including anger, frustration, anxiety, fear, and depression in chronic pain are under the limbic control (22). Moreover, a suppression or absence of second exteroceptive suppression period (ES2) of the temporalis muscle in CTTH patients indicates an involvement of limbic system. It is quite possible that the relief in symptoms of yoga group of CTTH patients was due to its influence on limbic system including several

others (hippocampus along with sensory and higher order association cortices) (21).

There is a possibility that several neural and neurochemical alterations in chronic painful conditions influence the limbic and other neural systems which in turn activates peripheral mechanisms. These peripheral mechanisms may be partially blocked by medical interventions such as NSAID and botulinum toxin leading to an experience of improvement although it is only symptomatic. However, the neurochemical changes of the chronic pain continue and a vicious circle of pain leading to pain continues.

The role of limbic system is further strengthened by the recent studies utilizing PET technique to demonstrate the role of hippocampus along with sensory and higher order association cortices in yogic practices (21). This also strengthens the efficacy of psychological approaches including deep breathing exercises, proper sleep, progressive relaxation, hypnosis or deep relaxation therapy and biofeedback techniques in the management of chronic pain including chronic headaches (22). These interventions are proposed to relieve the subject of pain by the supraspinal levels mainly the limbic system and are known to modulate the endogenous pain control system.

Which out of the two mechanisms is predominant cannot be said by our study design or for that matter in CTTH patients wherein by definition the duration of pain should be more than two years. However, to resolve the issue of peripheral versus central genesis patients of shorter duration of headache but similar pattern, at the very onset of headache and before it attains a state of chronicity should be studied. Further studies regarding possibility of such headaches becoming chronic should be explored in the early cases by studying their pain modulation status *vis-a-vis* their tendency to catastrophizing pain. It may help these patients in the earlier stages. We are actively pursuing this approach.

REFERENCES

1. Jensen R. Pathophysiological mechanisms of tension type headache: a review of epidemiological and experimental studies. *Cephalalgia* 1999; 19: 602–621.
2. Hatch JP, Moore PJ, Cyr-Provost M, Boutros NN, Seleshi E, Borcherding S. The use of electromyography and muscle palpation in the diagnosis of tension type headache with and without pericranial muscle involvement. *Pain* 1992; 49: 175–178.
3. Schoenen J, Gerard P, De Pasque V and Sianard-Gainko J. EMG activity in pericranial muscles during postural variation and mental activity in healthy volunteers and patients with chronic tension type headache. *Headache* 199l; 31: 324–334.
4. Pikoff H. Is the muscular model of headache still viable? A review of conflicting data. *Headache* 1984; 24: 186–198.
5. Olesen J, Jenson R. Getting away from simple muscle contraction as a mechanism of headache. *Pain* 1991; 46: 123–124.
6. Wang W, Schoenen J. Reduction of temporalis exteroceptive suppression by peripheral electrical stimulation in migaine and tension type headaches. *Pain* 1994; 59: 327–334.
7. Schoenen J, Jamart B, Gerard P, Lenarduzzi P,

Delwaide PI. Exteroceptive suppression of temporalis muscle activity in chronic headache. *Neurology* 1987; 37: 1834–1836.

8. Schepelmann K, Dannhausen M, Kotter I, Schabet M and Dichgans J. Exteroceptive suppression of temporalis muscle activity in patients with fibromyalgia, tension type headache and normal controls. *Electroencephalogr Clin Neurophysiol* 1998; 107: 196–199.
9. Thomas Martin Wallasch, Martin Reinecke, Hans Dieter. Langohr. EMG analysis of late exteroceptive suppression period of temporalis muscle activity in episodic and chronic tension type headache. *Cephalalgia* 1991; 11: 109–112.
10. Langmark M, Bach FW, Jensen TS, Olesen J. Decreased nociceptive flexion reflex threshold in chronic tension type headache. *Arch Neurol* 1993: 50: 1061–1064.
11. Olesen J, Bach FW, Langmark M and Sechner NH. Plasma and cerebrospinal fluid beta endorphin in chronic tension type headache. *Pain* 1992; 51: 163–168.
12. Clark T,. SakaiS, Merril R, VF Flack, McCreayC. Cross correlation between stress, pain, physical activity ,and temporalis muscle EMG in tension type headache. *Cephalalgia* 1995; 15: 511–518.
13. Covino BG, Dubner R, Gybels J, Kosterlitz M. Ethical standards for investigation of experimental pain in animals. *Pain* 1980; 9: 141–143.
14. Headache Classification committee of the International Headache. Society. Classification and diagnostic criteria for headache disorders, cranial neuralgias and fascial pain. *Cephalalgia* 1998; 8(suppl.7): 19–45.
15. Bausbum AI, Julius D. Towards better pain control. *Sci Am* 2006 Jun; 294(6): 6–7.
16. Kangasniemi PJT, Nyrke AH Lang. Femoxetine-a new 5HT uptake inhibitor-and propranolol in prophylactic treatment of migraine. *Acta Neural Scand* 1983; 68: 262–267.
17. Silberstein S, Mathew N, Saper J, Jenkins S. Botulinum toxin type A as a migraine preventive treatment. *Headache* 2000; 40: 445–450.
18. Smuts JA, Baker MK, Smuts HM. Botulinum toxin type A as a prophylactic treatment in chronic tension-type headache. *Cephalalgia* 1999; 19: 454–458.
19. Proceedings of biofeedback society of America : 9th annual meeting Albuquerque New Mexico, 1978.
20. Herbert Benson, Malvea BP, Graham JR. Physiological correlates of meditation and their clinical effects on headache: an ongoing investigation. *Headache* 1973; 23–24.
21. Lou HC, Nowal M, Kjaer TW. The mental self. *Prog Brain Res* 2005; 105: 197–204.
22. Wade JB, Price DD, Hamer RM, Schwartz SM and Hart RP. An emotional component analysis of chronic pain. *Pain* 1990; 40: 303–310.