Triazolam Improves Sleep but Fails to Alter Pain in TMD Patients

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Patients with chronic orofacial pain often report disturbances in sleep, leading to the hypothesis that nocturnal motor hyperactivity of the muscles of mastication may contribute to the nociceptive process. This hypothesis was tested in a controlled study to evaluate the relationship between sleep stages, patient self-report of pain in the orofacial region, and nocturnal masticatory muscle activity. Twenty subjects participating in a two-period, within-subject, crossover study received triazolam or placebo for 4 nights. Sleep, pain, and mandibular range of motion were assessed at baseline, following the first period, and again following the second period; a 3-day washout period separated the two treatments. Subjective report of sleep quality was significantly improved following triazolam in comparison to placebo as measured by category scales for sleep quality, restfulness, and sleep compared to usual. The amount of time spent in stage-2 sleep was also significantly increased by triazolam. No improvement was seen in pain as measured by palpation with an algometer, in scales for sensory intensity and the affective component of pain, or in daily pain diaries. Mean facial muscle electromyographic activity for 30-second epochs averaged over the entire period of sleep did not reveal any differences in muscle activity across the three conditions. These data indicate that improvements in sleep quality and alterations in sleep architecture do not affect nocturnal facial muscle activity or subsequent pain report in temporomandibular patients, thereby failing to support the hypothesized relationship between sleep disturbances and chronic orofacial pain.

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Chronic orofacial pain occurs commonly and yet remains a therapeutic enigma that has been subjected to a wide variety of treatment modalities based on a diverse spectrum of etiologic hypotheses. It is frequently observed that patients complaining of temporomandibular joint (TMJ) and/or masticatory muscle pain also report disturbances in the quality and quantity of their sleep. Patients may complain of difficulty in falling asleep, frequent awakening, or nonrestorative sleep.[1] A variety of clinical conditions related to chronic pain have also been linked to sleep disturbances, including nocturnal bruxism, fibromyalgia, and nocturnal myoclonus, as well as depression, headache, chronic fatigue syndrome, and posttraumatic conditions involving the central nervous system.[2–4] Occlusal appliances are frequently prescribed to protect the dentition from the impact of nocturnal muscle hyperactivity based on the apparent association between sleep disturbances, nocturnal bruxism, and chronic orofacial pain.[5,6]

Studies suggesting that drugs that improve sleep quality also reduce pain symptoms in chronic pain patients apparently confirm the relationship between sleep and chronic pain.[7,8] Hypnotics, including benzodiazepines, have significant actions on sleep stages[9–15] to counteract sleep-disrupting influences. Typical changes induced by benzodiazepines include reduced rapid eye movement (REM) and nonrapid eye movement (non-REM) sleep, improved sleep efficiency, increased REM latency, and decreased frequency of awakenings. Benzodiazepines may also enhance motorneuron inhibition mediated by the gamma-aminobutyric acid (GABA)–benzodiazepine receptor complex, thereby reducing the expression of abnormal motor events (parasomnias) during sleep.[1,3,9–12]

In the present study, the relationship between sleep quality and chronic pain in patients with temporomandibular disorders (TMD) was evaluated by quantifying the effect of a short-acting benzodiazepine on sleep and on patient report of pain. Triazolam, a short acting triazolobenzodiazepine, was chosen as the experimental drug based on its efficacy as a hypnotic agent with a short duration of action.[16] The half-life of triazolam is approximately 3 hours, with peak therapeutic effects observed at 2 to 4 hours.[16,17] Triazolam improves sleep time, decreases daytime sleepiness, and improves morning stiffness and pain in patients with rheumatoid arthritis.[18] It was hypothesized that triazolam would improve sleep patterns in patients with TMD, leading to decreased nocturnal muscle hyperactivity and less pain upon awakening or during the day.

Materials and Methods

Patient Population

Twenty patients, aged 24 to 55 years, each of whom had been diagnosed with a painful TMD involving the muscles of mastication and/or the TMJ, were included in the study. Each patient was evaluated by the principal investigator (DD) and assigned a primary diagnosis of TMD in accordance with the Research Diagnostic Criteria for Temporomandibular Disorders.[19] Inclusion criteria included unilateral or bilateral pain associated with the muscles of mastication and/or the TMJ with or without an accompanying decrease in mandibular range of motion. In cases where joint pathosis was suspected, confirmation was accomplished by magnetic resonance imaging.

Patients excluded from the study were those with clinical depression requiring immediate psychiatric care, those suspected of having a trigeminal or glossopharyngeal neuralgia or a neurologic deficit, patients with significant systemic disease, patients allergic to the study drug, and those involved in litigation related to their TMD. Patients were screened for depression by means of the Beck Depression Inventory. Patients were advised to refrain from the use of nonsteroidal anti-inflammatory agents, antidepressants, hypnotics, and any other analgesics for a period of 10 days prior to the commencement of the study and throughout the course of the study.

Treatments

The study was conducted over a 2-week period and used a randomized double-blind, two-period, crossover design allowing for a within-subjects analysis of data. A baseline sleep observation was conducted during the week prior to the study on the same night of the week as data collection during the study. Subjects were randomized to active drug or placebo for the 4 nights of the study, which concluded with sleep monitoring during the fourth night and data collection in the clinic on the morning of the fifth day. Three days over the weekend were allowed for drug washout, followed by the alternative treatment during the second week of the study. Subjects were given a supply of 0.125 mg triazolam tablets or matching placebo and instructed to take the drug 1 hour before bedtime. The dose was titrated based on subjects' daily report of drowsiness, with two tablets (0.25 mg triazolam or placebo) taken on the first night and escalation in one-tablet (0.125 or placebo) increments each subsequent night to a maximum of .50 mg on the third and fourth nights. Compliance with the drug regimen was confirmed by verbal report during daily conversations with the subjects to adjust their dose.

Dependent Measures

On the first day of each of the two 5-day treatment periods, subjects completed graphic pain-rating scales for measurement of pain intensity and discomfort.[20, 21] Pain pressure thresholds were measured by means of a pressure algometer (Somedic 900831, Farsta, Sweden) applied to two painful areas over the masseter or temporalis muscles on one side of the face.[22,23] Their locations were marked on the skin with delible ink, and a template of clear acetate was made of the location of these painful areas relative to facial landmarks so that the same locations could be approximated

on subsequent visits. The algometer tip was placed on the painful areas and pressure was applied gradually. The subject was advised to press a button when the "pressure turned to pain." The pressure was then recorded (in kilopascals) for each of the two painful points. Mandibular range of motion measurements (in millimeters), consisting of maximum interincisal opening with and without pain, maximum lateral movements with and without pain, and maximum protrusive movement with and without pain, were also recorded.

Three nights of sleep monitoring were conducted per patient by means of an ambulatory polysomnographic recording at home: a baseline recording was conducted during the week prior to drug administration, a second observation was conducted following 4 nights of drug or placebo treatment, and a final recording was conducted at the end of the second week following 4 nights of the alternative treatment. Electroencephalographic (EEG) and electromyographic (EMG) data were collected by means of a Medilog 9000-II recorder with eight data-collection channels (Oxford Instruments, Clearwater, FL). In addition, electrodes were attached bilaterally to the masseter muscles to measure electrical activity during the monitoring period. The EEG data were subjected to automated sleep analysis using an Oxford 9200 8/16-channel advanced EEG monitoring replay system (Oxford Instruments, Clearwater, FL).

Each subject was also given sleep and pain diaries.[24,25] Subjects were instructed to complete the sleep diary each morning upon arising. Sleep diary data included subjective measures of sleep quality, sleep duration, and periods of awakening. Subjective estimates were made by selecting a number (0 to 5) arranged horizontally and anchored by descriptors at each end of the scale. Overall sleep quality was categorized from extremely poor to extremely good. Subjects estimated how rested they were upon awakening, ranging from "not rested at all" to "well rested." They also rated the sleep compared to normal, which could range from "much worse than normal" to "much better than normal."

Subjects were likewise instructed to record pain intensity and discomfort at five predesignated times during each day, using verbal descriptor scales consisting of two lists of words, one for pain intensity (eg, faint, moderate, intense) and one for the affective component of pain (eg, unpleasant, distressing, intolerable). On the fourth night, subjects underwent nocturnal polysomnographic recordings at home. The following morning, pain pressure threshold, range of motion, and pain measurements were recorded in a manner identical to the first day of the study. Pain and sleep diaries for the week were collected, and the patient was advised to return to begin week 2 of the study following a 3-day washout period. The same regimen and procedures were followed during the second week.

Statistical Analyses

Data was analyzed using the BMDP statistical software package (SPSS, Chicago, IL). Graphic pain-rating scales, range of mandibular motion, pain pressure thresholds, and EMG values were compared between drug and placebo treatments with a paired t test. Categorical data, such as the pain diaries, and subjective ratings of sleep quality were compared with the Kruskal-Wallis test. Values are reported in the text as the mean ± one standard deviation and are illustrated in the figures as the mean ± standard error of the mean. Statistical significance was accepted as $P < 0.05$.

Results

All 20 subjects enrolled completed the study. Retrospective inspection of the data revealed that one patient did not report pain at any of the study appointments, including baseline on the first day of the study, or in the pain diaries; this subject's data were omitted from the rest of the analyses. The study sample consisted of 18 women and 1 man with a mean age of 39.2 ± 9.7 years who had been experiencing TMD pain for 9.5 ± 7.3 years prior to enrollment in the study. Of these 19, 5 subjects had muscle pain without limited opening (RDC axis I, group Ia) and 14 had muscle pain with limited opening (RDC axis I, group Ib); of those with limited opening, 6 were categorized as also having an anteriorly displaced disc without reduction (RDC axis I, group IIb). Pain intensity at study entry was a mean of 5.5 (on a verbal rating scale of 0 to 10), with a range of 3 to 8.

The final mean triazolam dose achieved by the fourth night was 0.42 mg ± 0.05; an equivalent mean number of tablets (3.4) were administered during the placebo arm of the study. Triazolam resulted in a qualitative and quantitative improvement in sleep. Subjective reports of sleep quality, sleep compared to usual, and rested upon awakening were significantly improved when assessed on the morning after the final drug dose in compari-

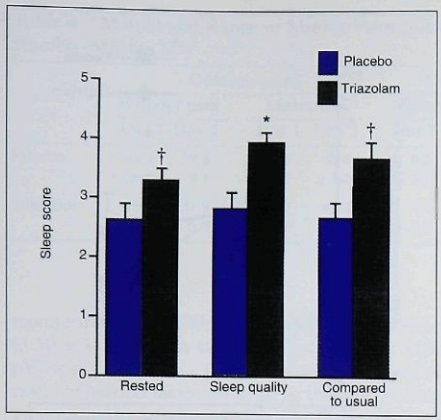

Fig 1a Subjective rating of sleep quality on separate five-point scales for the perception of rested upon awakening, quality of sleep, and sleep compared to usual (*$P < 0.05$ versus placebo; †$P < 0.01$ versus placebo).

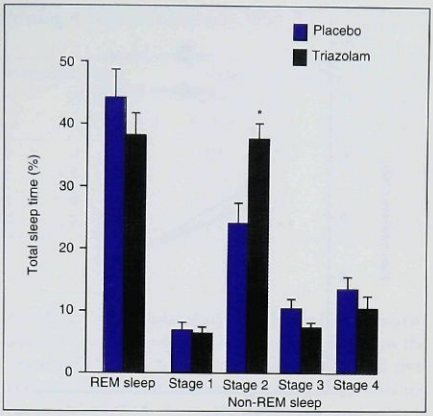

Fig 1b Percent of total sleep time in rapid eye movement (REM) sleep or the stages of non-REM sleep (*$P < 0.05$ versus placebo).

Table 1 Quantified Measures of Sleep Quality (Mean ± SD)

	Wake time (%)	REM latency (min)	Sleep onset (min)	Slow wave sleep (%)
Baseline	9.1 ± 6.6	39.4 ± 34.6	12.1 ± 12.7	21.5 ± 13.9
Placebo	11.1 ± 8.7	31.4 ± 27.8	12.6 ± 11.0	23.7 ± 14.5
Triazolam	5.2 ± 3.7	56.4 ± 73.4	18.6 ± 18.1	17.8 ± 11.7

son to placebo (Fig 1a). Quantal measures of sleep architecture derived from the sleep monitoring were significantly changed by the fourth night of triazolam. The amount of time in stage-2 sleep was significantly greater during the drug period compared to the amount of time in stage-2 sleep during the placebo treatment (Fig 1b). The amount of wake time, REM latency, time to sleep onset, and percent time in slow wave sleep were not significantly changed during the triazolam night of sleep monitoring (Table 1).

Pain as measured by patient self-report on the final morning of drug or placebo did not differ for estimates of sensory intensity (Fig 2a) or the affective component of pain (Fig 2b). Pain as measured by pressure algometry over the two painful sites also was similar under both drug and placebo treatment (Table 2). The mean daily pain diary scores did not differ across treatments for either pain intensity or its affective component (Table 3).

The nocturnal masseter muscle activity averaged over each 30-second epoch recorded during sleep

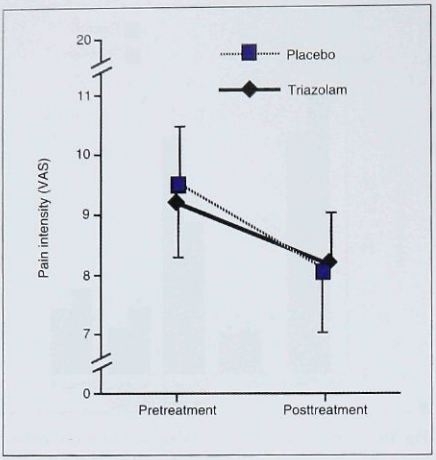

Fig 2a Subjective rating of pain intensity prior to and following each treatment (VAS = visual analogue scale).

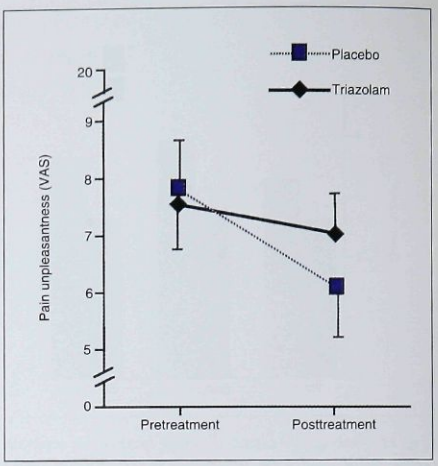

Fig 2b Subjective rating of affective component of pain prior to and following each treatment (VAS = visual analogue scale).

Table 2 Pain Pressure Thresholds (± SD) for Two Painful Sites on the Masseter Muscle

	Site 1		Site 2	
	Day 1	Day 5	Day 1	Day 5
Placebo	116.0 ± 42.8	111.0 ± 36.2	100.7 ± 38.1	107.8 ± 38.0
Triazolam	104.7 ± 39.7	119.8 ± 56.9	106.0 ± 42.3	116.5 ± 50.6

Table 3 Daily Pain Diaries for Pain Intensity and Discomfort*

	Pain intensity (scale 1–20)				Pain discomfort (scale 1–20)			
	Day 2	Day 3	Day 4	Day 5	Day 2	Day 3	Day 4	Day 5
Placebo	10.2 ± 2.4	9.2 ± 3.4	8.1 ± 2.7	8.5 ± 4.3	7.6 ± 1.7	7.2 ± 1.5	7.0 ± 2.0	7.4 ± 2.9
Triazolam	8.9 ± 3.6	9.8 ± 3.0	8.5 ± 3.4	8.5 ± 4.6	7.5 ± 2.6	7.2 ± 1.9	7.7 ± 1.4	7.4 ± 3.7

*Days 2, 3, and 4 are the mean of five daily reports; Day 5 is based on a single report in the morning (mean ± SD).

Table 4 Mandibular Range of Motion Prior to and Following 4 Nights' Treatment With Triazolam or Placebo (Mean ± SD)

	Opening (mm)				Lateral movement (mm)				Protrusive movement (mm)			
	Without pain		Maximum		Without pain		Maximum		Without pain		Maximum	
	Day 1	Day 5	Day 1	Day 5	Day 1	Day 5	Day 1	Day 5	Day 1	Day 5	Day 1	Day 5
Placebo	40.3 ± 8.0	39.5 ± 9.1	45.5 ± 6.9	45.6 ± 7.2	8.2 ± 2.8	8.4 ± 2.7	9.9 ± 2.4	10.0 ± 2.4	6.5 ± 2.6	6.9 ± 2.7	7.6 ± 2.5	8.4 ± 2.7
Triazolam	38.6 ± 10.1	37.9 ± 8.6	45.6 ± 7.2	45.3 ± 6.9	7.9 ± 2.6	7.6 ± 2.6	9.6 ± 2.1	9.7 ± 2.3	6.1 ± 2.7	6.2 ± 2.8	7.5 ± 2.6	7.7 ± 2.8

monitoring did not differ significantly from baseline (3.50 ± 1.16 μV) on either the drug (3.44 ± 1.26 μV) or placebo (3.26 ± 0.79 μV) nights. Mandibular range of motion measured prior to the start of drug administration and again on the fifth morning, including opening, protrusive, and lateral movements, did not differ across groups following the 4-day course of drug or placebo (Table 4).

Discussion

Triazolam administered over 4 nights resulted in a measureable improvement in sleep without improvement in the measured signs and symptoms of TMD. Pain pressure thresholds measured over painful muscle sites and patient self-report of both pain intensity and the affective component of pain were unchanged by administration of a near-maximum dose of triazolam. This finding is contrary to the commonly held belief that improvement in sleep quality should reduce nocturnal muscle activity contributing to muscle pain. Triazolam administration had no apparent effect on muscle activity in comparison to baseline or placebo, such that muscle activity may have continued unabated, resulting in similar levels of daytime pain if related to nocturnal muscle activity. Comparison to a nonpain population under similar experimental conditions is needed to determine if the patient group was actually experiencing muscle hyperactivity or if the baseline levels were consistent with normal nocturnal muscle activity.

Bruxism is thought to occur during REM-related transitions to lighter stages of sleep, while high-amplitude parafunctional activity occurs predominantly during REM sleep.[26] REM sleep is thought to confer a degree of protection from self-inflicted injury because of strong somatic motorneuron inhibition. In contrast, non-REM or para-REM sleep stage transitions may be associated with a barrage of uninhibited excitatory neuronal activity resulting in unimpeded parafunctional motor activity with the potential to produce injury to the organism.[27] It is hypothesized that pain and dysfunction observed in some patients with chronic orofacial pain is the result of fatigue and muscle injury associated with uninhibited mandibular parafunctional activity during the non-REM or para-REM sleep transitions. The lack of change in the amount of time spent in these stages in this study may have permitted the putative mechanisms of nocturnal muscle hyperactivity to continue. Conversely, the hypothesized relationship between sleep architecture and subsequent TMD pain may not be valid.

Stage-2 sleep was significantly increased at the expense of nonsignificant reductions in REM sleep and stage-3 and stage-4 sleep. Parasomnias are a group of unusual sleep-related syndromes that produce movements or behaviors during sleep. These include somnambulism, somniloquy, pavor nocturnus (night terrors), enuresis, nightmares, nocturnal seizures, confused partial arousals, nocturnal tics, and myoclonus.[11] Abnormal sleep behaviors or parasomnias disturb normal sleep architecture and may be a reflection of abnormal central nervous system activity that influences sleep architecture. These phenomena seem to be associated with the transition and emergence from early non-REM deep sleep (stages 3 and 4) to lighter stages of sleep (stages 1 and 2) and have been proposed to account for the observed disturbances in sleep architecture in patients afflicted with parasomnias including nocturnal bruxism.[28] By increasing the amount of time in stage-2 sleep at the expense of stage-3 and stage-4 sleep, the number of sleep disturbances associated with these transitions was probably decreased, resulting in the reported improvement in sleep.

It should be noted that several of the older patients (greater than 45 years) reported impaired memory and confusion upon arising the morning after receiving a dose of .50 mg of triazolam. This is

consistent with reports in the literature suggesting that benzodiazepines in general and triazolam in particular may produce behavioral and cognitive alterations in older patients at higher doses.[19,20] However, adverse effects, such as anterograde amnesia, may not be unique to triazolam, since hypnotically equivalent doses of triazolam have not been shown to produce these effects more frequently than other short-acting hypnotics.[29]

The results of this controlled evaluation with triazolam fail to support a relationship between improvement in sleep and a decrease in the symptoms of TMD in the patient sample studied. It is possible, however, that hypnotics have value in a sample restricted to patients satisfying EMG criteria for nocturnal bruxism[26] in which intensive, possibly within-subjects, experimental approaches are used.[30] Similarly, the relatively short duration of drug administration in the present study (4 nights) would not have detected a possible beneficial effect occurring with a longer duration trial of triazolam.

As with many therapies for TMD based on unverified hypotheses, a failure to demonstrate efficacy in a controlled trial indicates that caution is required if treatment of patients is continued with this modality unless patients are fully informed of the possible adverse effects of the treatment and agree to accept these risks in the absence of a confirmatory body of scientific evidence.

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References

1. Boutros NN, Montgomery MT, Nishioka G, Hatch JP. The effects of severe bruxism on sleep architecture: A preliminary report. Clin Electroencephalogr 1993;24:59–62.
2. Dahl RE. The pharmacologic treatment of sleep disorders. Psychiatr Clin North Am 1992;15:161–178.
3. Chase MH, Morales FR. The atonia and myoclonia of active (REM) sleep. Annu Rev Psychol 1990;41:557–548.
4. Dao TT, Lund JP, Lavigne GJ. Comparision of pain and quality of life in bruxers and patients with myofascial pain of the masticatory muscles. J Orofacial Pain 1994;8: 350–356.
5. Solberg WK, Clark GT, Rugh JD. Nocturnal electromyographic evaluation of bruxism patients undergoing short term splint therapy. J Oral Rehabil 1975;2:215–223.
6. Clark GT, Beemsterboer PL, Solberg WK, Rugh JD. Nocturnal electromyographic evaluation of myofascial pain dysfunction in patients undergoing occlusal splint therapy. J Am Dent Assoc 1979;99:607–611.
7. Quijada J, Valenzuela A, Povedano J, Fernandez A, Hernandez W, Gutierrez A, et al. Comparison of tenoxicam and bromazepam in the treatment of fibromyalgia. Pain 1996;65:221–225.
8. Goldberg D, Mayskiy M, Mossey C, Ruthazer R, Schmid C. A randomized double-blind crossover trial of fluoxetine and amitriptyline in the treatment of fibromyalgia. Arthritis Rheum 1996;39:1852–1859.
9. Borbely AA, Achermann P. Ultradian dynamics of sleep after single dose of benzodiazepine hypnotics. Eur J Pharmacol 1991;195:11–18.
10. Borbely AA, Akerstedt T, Benoit O, Holsboer F, Oswald I. Hypnotics and sleep physiology: A consensus report. Eur Arch Psychiatry Clin Neurosci 1991;241:13–21.
11. Aldrich MS. Sleep disorders. Curr Opin Neurol Neurosurg 1992;5:240–246.
12. Ware JC, Morewitz J. Diagnosis and treatment of insomnia and depression. J Clin Psychiatry 1991;52:55–61.
13. Tani YK, Yoshino I, Kobyashi E. Electroencephalographic study of parasomnia: Sleeptalking, enuresis, and bruxism. Physiol Behav 1966;1:241–243.
14. Parrino L, Terzano MG. Polysomnographic effects of hypnotic drugs. Psychopharmacology 1996;126:1–16.
15. Mendelsen WB. Effects of flurazepam and zolpidem on the perception of sleep in insomniacs. Sleep 1995;18:92–96.
16. Hardman JG, Limbird LE, Molinoff PB, Ruddon RW, Gilman AG. Goodman and Gillman's Pharmacologic Basis of Therapeutics, ed 9. New York: McGraw-Hill, 1996: 361–370.
17. Physicians' Desk Reference, ed 49. Montvale, NJ: Medical Economics, 1995:2552–2554.
18. Walsch JF, Muehlbach MJ, Lauter SA, Hilliker NA, Schweitzer PK. Effects of triazolam on sleep, daytime sleepiness, and morning stiffness in patients with rheumatoid arthritis. J Rheumatol 1996;23:245–252.
19. Dworkin SF, LeResche L (eds). Research Diagnositic Criteria for Temporomandibular Disorders: Review, Criteria, Examinations and Specifications, Critique. J Craniomandib Disord Facial Oral Pain 1992; 6:327–334.
20. Gracely RH, Dubner R. Reliability and validity of verbal descriptor scales of painfulness. Pain 1987;29:175–185.
21. Gracely RH, Reid KI. Orofacial Pain Measurement. In: Fricton J, Dubner R (eds). Orofacial Pain and Temporomandibular Disorders. New York: Raven Press, 1995:117–143.
22. Svensson P, Arent-Nielsen L, Nielsen H, Larsen J. Effect of chronic and experimental jaw muscle pain on pain pressure thresholds and stimulus response curves. J Orofacial Pain 1995;9:347–356.
23. Reid KI, Gracely RH, Dubner R. The influence of time, facial side, and location on pain pressure thresholds in chronic myogenous temporomandibular disorders. J Orofacial Pain 1994; 8:258–265.
24. Haythornthwaite JA, Hegel MT, Kerns RD. Development of a pain diary for chronic pain patients. J Pain Symptom Manage 1991;6:65–72.
25. Rechtshaffen A, Kales A. A manual of standardized terminology, techniques, and scoring for sleep stages of human subjects. Bethesda, MD: National Institutes of Health, publication 204.
26. Ware JC, Rugh JD. Destructive bruxism: Sleep stage relationship. Sleep 1988;11:172–181.
27. Wruble MK, Lumley MA, McGlynn FD. Sleep-related bruxism and sleep variables: A critical review. J Craniomandib Disord Facial Oral Pain 1989;3:152–158.

28. Nishioka GJ, Montgomery MT. Masticatory muscle hyperactivity in temporomandibular disorders: Is it an extrapyramidally expressed disorder? J Am Dent Assoc 1988;116: 514–520.
29. Mendelson WB, Jain B. An assessment of short-acting hypnotics. Drug Saf 1995;13:257–270.
30. Lobbezoo F, Soucy JP, Hartman NG, Montplaisir JY, Lavigne GJ. Effects of the D2 receptor agonist bromocriptine on sleep bruxism: Report of two single-patient clinical trials. J Dent Res 76;1997:1610–1614.

Resumen

Efectos del Triazolam: El triazolam mejora el sueño pero no altera el dolor en los pacientes con desórdenes temporomandibulares

Los pacientes con dolor crónico orofacial a menudo se quejan de alteraciones en el sueño, lo que conduce a la hipótesis de que la hiperactividad motora nocturna de los músculos de la masticación puede contribuir al proceso nociceptivo. Esta hipótesis fue probada en un estudio controlado que evaluó la relación entre los estados de sueño, el auto-reporte del paciente en relación al dolor en la región orofacial, y la actividad muscular masticatoria nocturna. Los 20 participantes de este estudio de dos períodos, de estilo cruzado y de intraindividuos; recibieron triazolam o placebo por 4 noches. Las evaluaciones iniciales, lo mismo que las efectuadas después del primer y segundo períodos incluyeron el sueño, dolor, y la escala de movimiento mandibular. Los dos tratamientos fueron separados por un período de tres días. El reporte subjetivo de la calidad del sueño mejoró significativamente luego del uso del triazolam en comparación al placebo, de acuerdo a las medidas de las escalas de categoría en relación a la calidad del sueño, tranquilidad, y sueño en comparación al acostumbrado. La cantidad de tiempo que se midió durante la segunda etapa del sueño también fue mayor, al usar el triazolam, esto fue significativo. No se detectaron mejorías en relación al dolor al hacer medidas de palpación con un algómetro, en las escalas para la intensidad sensitiva, y el componente afectivo del dolor; ni en los diarios donde se registró el dolor. La actividad electromiográfica muscular facial media registrada en períodos de 30 segundos durante todo el período de sueño no mostró ninguna diferencia en la actividad muscular al analizar las tres condiciones. Esta información indica que la mejoría en la calidad del sueño y las alteraciones en la arquitectura del sueño no afectan la actividad muscular facial nocturna o los reportes de dolor subsiguientes en los pacientes con problemas temporomandibulares, lo cual no soporta la hipótesis que relaciona los disturbios del sueño y el dolor orofacial crónico.

Zusammenfassung

Triazolam verbessert den Schlaf, kann aber den Schmerz bei TMD Patienten nicht verändern

Patienten mit chronischem orofazialem Schmerz berichten oft über Schlafstörungen, was zur Hypotheses fuhrt, dass nächtliche motorische Hyperaktivität der Kaumuskeln zum nozizeptiven Prozess beiträgt. Diese Hypothese wurde in einer kontrollierten Studie getestet, um die Beziehung zwischen Schlafstufen, Selbstreport des Patienten von Schmerzen in der orofazialen Region und nächtlicher Kaumuskelaktivität herauszufinden. Zwanzig Personen, die in einer zweizeitigen Crossover-Studie teilnahmen, erhielten Triazolam oder Placebo für 4 Nächte. Schlaf, Schmerz und das Ausmass der Unterkieferbeweglichkeit wurden zu Beginn, im Anschluss an die erste Periode und nochmals nach der zweiten Periode beurteilt; eine 3-tägige Auswaschperiode trennte die zwei Behandlungen. Der subjektive Bericht der Schlafqualität war signifikant verbessert nach Triazolam im Vergleich mit Placebo, gemessen mittels einer Kategorieskala für die Schlafqualität, Ruhe und Schlaf verglichen mit normalerweise. Die Zeit, welche im Stufe-2 Schlaf verbracht wurde, war ebenfalls erhöht beim Triazolam. Keine Verbesserung wurde für die Schmerzen beobachtet, gemessen durch Palpation mit einem Algometer, in Skalen für die sensorische Intensität und die affektive Schmerzkomponente oder in Schmerztagebüchern. Die durchschnittliche elektromyographische Gesichtsmuskelaktivität für 30-Sekundenzeiträume gemittelt über die ganze Schlafperiode enthüllte keine Unterschiede in der Muskelaktivität über die drei Zustände. Diese Daten deuten darauf hin, dass Verbesserungen der Schlafqualität und Veränderungen der Schlafarchitektur keine nächtliche Gesichtsmuskelaktivität oder späteren Schmerzreport bei temporomandibulären Patienten bewirken, damit versagt die Unterstützung der Hypothese der Beziehung zwischen Schlafstörungen und chronischen orofazialen Schmerzen.