Generalized Joint Laxity and Temporomandibular Disorders

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Temporomandibular disorders (TMD) has been suggested to be of multifactorial etiology. One factor that has been suggested is laxity of joint ligaments. The purpose of this study was to evaluate the relationship between generalized joint hypermobility and TMD. Thirty-eight asymptomatic volunteers and 62 symptomatic patients were included in this study. All asymptomatic volunteers did not have temporomandibular joint pain, limited jaw movement, joint sounds, or previous TMD treatment. All subjects had bilateral magnetic resonance imaging scans in the sagittal closed and opened and coronal closed positions. The Beighton test was used to score joint laxity with a laxity score of ≥ 4 to define generalized joint laxity. The symptomatic group had an increase in joint laxity as compared to asymptomatic control subjects (odds ratio 4.0 [95% confidence interval = 1.38 to 10.95, P = .01]). There were no differences in laxity between male and female symptomatic subjects (P > .05). This study suggests a positive correlation between generalized joint laxity and TMD.
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key words: hypermobility, joint instability, joints, temporomandibular joint

Hypermobility of the joints is frequently encountered in patients with inherited connective tissue disease. To study the effects of genetic mutations, a model of a disease that has maximum expression should be evaluated. This is true in Ehlers-Danlos syndrome (EDS), but it also occurs in osteogenesis imperfecta (OI), Marfan syndrome, and in some chondrodysplasias, especially pseudoachondroplasia.[1–4] All of these disorders are usually considered to be a result of laxity of supporting ligaments because of the failure of their structural components, particularly collagen, which is responsible for the obvious features of the disease. Moderate joint laxity also occurs without involvement of other tissues and has been suggested to predispose to the development of osteoarthritis.[5] Most studies demonstrate that women are more lax than men.[6] Differences in mean joint mobility scores have also been reported in specific ethnic groups.[6–9] Higher joint mobility has been reported to occur in Asian populations as compared to whites and certain variants of major collagen gene disorders.

Temporomandibular disorders (TMD) are generally believed to be of a multifactorial origin.[10] Frequently mentioned etiologic factors include occlusal disharmonies, psychologic profile, muscu-

loskeletal injuries, parafunctional habits, distress, and connective tissue laxity.[10–13] Several studies have reported an association between generalized joint laxity and signs and symptoms of TMD,[14,15] and they have been suggested to be significantly more prevalent in individuals with TMD as compared to asymptomatic control subjects.[15] Consistent with this observation, it has been suggested that patients with TMD and generalized joint laxity report significantly more general muscular skeletal complaints than do asymptomatic control subjects.[16] This suggests that generalized joint laxity may represent a risk factor in the development of TMD. A possible manifestation of joint laxity of the temporomandibular joint (TMJ) may be altered disc position in symptomatic patients.[17] Disc abnormalities in the absence of pain have been reported in other joints including the knee, cervical spine, and lumbar spine.[18–21] It may be possible that disc abnormalities may represent a joint phenotype that predisposes to joint dysfunction including TMD. Altered collagen metabolism may be important in joint laxity. Higher ratios of collagen type III to type III + I have been reported in patients with TMJ derangement and generalized joint laxity than in control subjects.[14,22] A strong genetic link suggesting altered collagen metabolism (skipping of exon 6) has been reported in EDS and other collagen disorders.[23–25]

The purpose of the present investigation was to compare the prevalence of joint laxity of symptomatic TMD patients to that of asymptomatic volunteers.

Materials and Methods

Sixty-two adult symptomatic TMD patients and 38 asymptomatic volunteers were included in this study. The symptomatic patients were selected from patients seeking treatment for TMD. Asymptomatic volunteers were accepted into the study following completion of the following: *(1)* a TMJ subjective questionnaire documenting the absence of jaw pain, TMJ noise, disc displacement without reduction, and a positive history for TMD; and *(2)* a clinical TMJ and dental examination as described by Roberts et al[26] for signs and symptoms, associated with TMD or internal derangement. All asymptomatic volunteers demonstrated maximal mouth opening of at least 40 mm. Of the 38 volunteers, 13 had disc displacement (6 bilateral and 7 unilateral). Two had laxity scores equal to or greater than 4.

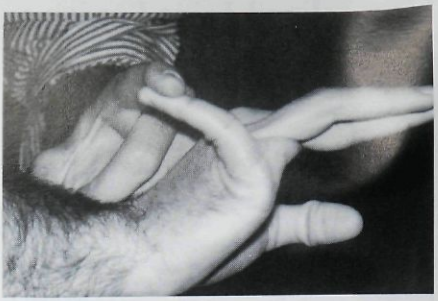

Fig 1a Extension of the wrist and metacarpal phalanges (all fingers or the little finger) so the fingers are parallel to the dorsum of the forearm. Note that the finger does not reach parallel; this would be a negative finding.

All study participants had bilateral high-resolution magnetic resonance imaging (MRI) scans in the sagittal (closed and opened) and coronal (closed) positions to evaluate the TM joints for the presence or absence of disc displacement.[27] On the basis of these findings, each study participant was then classified as having a normal MRI scan (no disc displacement) or an abnormal MRI scan (disc displacement). The asymptomatic volunteers with normal joints and the volunteers with disc displacements (with and without reduction) were collapsed into one group. Joint laxity was measured, as described by Beighton and Horan,[1] on a scale of 0 to 9. A score greater than or equal to 4 is classified as laxity.[6] Some studies[28,29] have suggested a score equal to or greater than 3 as the cutoff point. The examination for hypermobility was performed as described by Beighton et al[6]:

1. Extension of the wrist and metacarpal phalanges so the fingers are parallel to the dorsum of the forearm (Fig 1a)
2. Passive apposition of the thumb to the flexor aspect of the forearm (Fig 1b)
3. Hyperextension of the elbows ≥ 10 degrees (Fig 1c)
4. Hyperextension of the knees ≥ 10 degrees (Fig 1d)
5. Flexion of the trunk with the knees extended so the palms rest on the floor (Fig 1e)

If any four of the maneuvers are positive, this is considered generalized joint laxity.

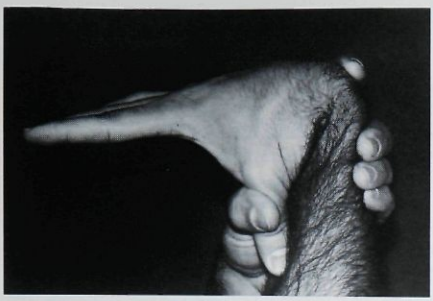

Fig 1b Passive apposition of the thumb to the flexor aspect of the forearm. Note the thumb touches the forearm; this would be a positive finding.

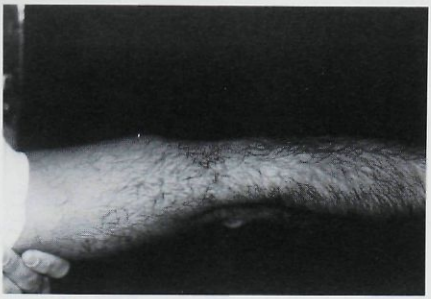

Fig 1c Hyperextension of the elbows ≥ 10 degrees. The elbow flexion does not pass 10 degrees in this subject; therefore, this is a negative finding.

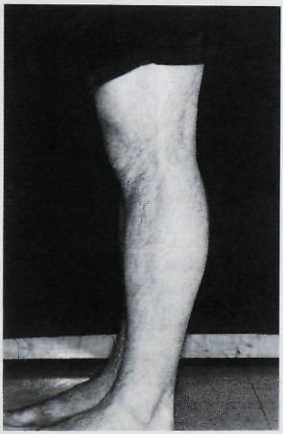

Fig 1d Hyperextension of the knees ≥ 10 degrees. Flexion does not pass 10 degrees in this subject; therefore, this is a negative finding.

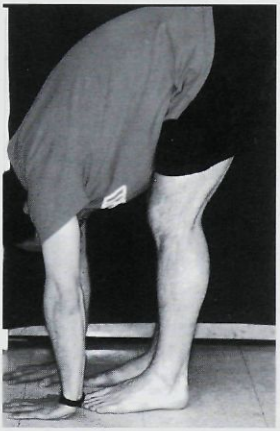

Fig 1e Flexion of the trunk with the knees extended so the palms rest on the floor. This subject is able to touch the floor; this is a positive finding.

Results

A total of 73 women and 27 men participated in this study. The mean age was 30.8 years (standard deviation [SD] = 8.2) for the symptomatic patients (n = 23) with joint laxity and 32.3 years (SD = 7.4) for those without joint laxity (n = 39). The mean age was 25.0 years (SD = 4.8) for the asymptomatic volunteers with joint laxity (n = 33) and 30.3 years (SD = 8.1) for those without joint laxity (n = 5).

Table 1 shows the distribution of the subjects based on the laxity scores. Five asymptomatic control subjects (13.2%) had a joint laxity score of equal to or greater than 4. Twenty-three symptomatic TMD patients (37.1%) had a laxity score equal to or greater than 4. Odds ratios (OR) and corresponding 95% confidence intervals (CI) suggest a higher prevalence of joint laxity in symptomatic TMD patients (OR = 3.89, 95% CI = 1.38 to 10.95, $P = .01$).

Table 1 Laxity Score for Symptomatic and Control Subjects With a Laxity Cutoff of ≥ 4

	Laxity ≥ 4	Laxity ≤ 3	Total
No. of subjects	5 (13.2%)	33 (86.8%)	38
No. of patients	23 (37.1%)	39 (62.9%)	62

Table 2 Laxity Score for Symptomatic and Control Subjects With a Laxity Cutoff of ≥ 3

	Laxity ≥ 3	Laxity ≤ 2	Total
No. of subjects	8 (21.1%)	30 (78.9%)	38
No. of patients	30 (48.4%)	32 (51.6%)	62

Table 3 Range (Mean ± SD) of Jaw Motion of Control and Symptomatic Subjects*

	All subjects	A–N, laxity	A–N, no laxity	A–DD, laxity	A–DD, no laxity	Symptomatic patients, laxity	Symptomatic patients, no laxity
Maximum opening	43.7	46.8	47.2	43.5	44.7	42.3	42.3
	±7.6	±7.6	±6.7	±3.5	±7.8	±8.8	±7.3
Right lateral	8.9	8.6	8.4	4.5	10.2	9.2	8.9
	±3.2	±3.7	±3.3	±3.5	±3.5	±3.1	±3.0
Left lateral	9.0	7.8	9.4	7.0	10.2	8.6	9.3
	±3.3	±4.8	±3.4	±4.2	±3.5	±2.8	±3.3
Protrusive movement	7.2	8.0	7.9	7.5	8.4	7.4	6.3
	±2.5	±2.8	±2.4	±3.5	±3.1	±2.1	±2.3

*A = asymptomatic, no report of temporomandibular joint dysfunction; N = normal MRI scan, no disc displacement; DD = disc displacement (with or without reduction).

There were 28 patients with unilateral disc displacement, 33 with bilateral disc displacement, and 1 patient with bilateral symptomatic but normal joints.

As shown in Table 2 the scores were again generated, using a laxity score of equal to or greater than 3 to permit comparisons to previously published studies. Eight asymptomatic control subjects (21.1%) and 30 symptomatic TMD patients (48.4%) had laxity scores equal to or greater than 3 (OR = 3.51, 95% CI = 1.42 to 8.69, $P = .007$).

Table 3 shows the range of movement for all subjects and each study group. There were no statistically significant differences between groups except for protrusive movement in symptomatic patients, which was greater than that for the nonlax patients ($P < .05$). There were no statistically significant differences with respect to sex, age, prevalence of trauma, or unilateral versus bilateral disc displacement.

Discussion

Disc displacement with and without reduction has been suggested to be an etiology of TMJ pain and dysfunction. Presently there have been no conclusive studies that document the etiology of displacement (genetic, trauma, age). Disc displacement has been demonstrated to increase with age.[30–36] Studies on younger individuals suggest the prevalence of disc displacement to be between 7% and 12%.[30,31] Disc displacement in older populations ranges from 25% to 67%.[32–36] It has been suggested that derangement is present in asymptomatic children and young adults,[37] and in children presenting for orthodontic treatment.[38,39] These studies only describe the prevalence, not etiology. Disc displacement has been suggested to be more common in symptomatic TMD patients than in asymptomatic control subjects without disc displacement.[40] Disc displacement also occurs in symptomatic children and young adults.[37,41,42] Children are more joint lax than are adults,[6] but how this may contribute to disc displacement is unknown.

The failure of collagen fibers, the ratio of collagen type I to type III,[22] and the presence of certain risk factors (trauma, gender) might offer some explanation. If disc displacement is a result of repeated loading and repair that results in tissue becoming less capable of withstanding load, then why asymptomatic children have disc displacement may be questioned.[37–39] To date, there is no clear understanding of why discs are displaced in the TMJ or other joints[18–21] in the absence of pain.

There also is no clear understanding of why joints suddenly become painful after a traumatic event.

Mutations in procollagen genes may be expressed in a wide spectrum of rare and common human diseases. Kuivaniemi et al[23] have suggested that there are more than 70 mutations in the two structural genes for procollagen type I (COL1A1 and COL1A2) found in osteogenesis imperfecta and other tissues rich in collagen type I. Nichols et al[25] have evaluated an autosomal dominant inherited connective tissue defect that causes extreme joint hypermobility, premature osteoporosis, and late onset fractures. They found deletion of 54 bps comprising exon 9. They also evaluated a family of 13 affected and unaffected family members. All of the affected and none of the unaffected individuals were found to carry the deletion.

Temporomandibular disorders has been noted with increased frequency in individuals with mitral valve prolapse, indicating a possible etiologic association with altered collagen metabolism.[43] Aortic aneurism has been suggested to be related to collagen defects, although the relationship is less clear.[44]

No sex differences between the lax and nonlax subjects with respect to TMD status were found in the present study. The small number of men may have contributed to this result.

Joint laxity was not age dependent in the present study. Beighton et al[6] demonstrated that the prevalence of laxity decreases with age. Laxity was higher in children than in adults. In their study, 25% of the sample were lax between the ages of 20 and 44 years using a cutoff of ≥ 3. In the present study 21.1% of the control subjects (see Table 2) demonstrated laxity with a laxity score of ≥ 3, which suggests similarity of the samples. The mean age of the asymptomatic volunteers with laxity was 25 years and without laxity was 30 years, with a range of 21 to 33 years. The mean age of the symptomatic patients was 30 years with laxity and 32 without laxity, with a range of 22 to 53. Six symptomatic subjects older than age 40 years were equally divided between lax and nonlax classifications.

We evaluated the presence of bilateral disc displacements and the risk of having joint laxity. One could speculate that the presence of bilateral derangements might be an increased expression of the disease. Our data suggest that laxity was no more prevalent in bilateral derangement patients. A possible exception to this was found in women with unilateral derangement. They were less likely (P = .069) to have laxity. Although this value is not statistically significant, future studies will evaluate this with an increased sample size.

Macrotrauma has also been suggested as a contributing factor in the development of TMD. This may represent a risk factor, not an etiologic factor.[40] In the present study, trauma was no more prevalent in lax subjects compared to nonlax subjects.

Conclusion

Joint laxity was prevalent in the present study of TMD patients. Laxity accounted for only 37% of the symptomatic TMD patients (for score equal to or greater than 4) and 48% when the cutoff score was lowered to equal to or greater than 3. This would suggest that laxity is not the only etiologic factor in TMD and there are probably other genetic contributions to altered collagen metabolism that may represent potential genetic markers.

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Resumen

Laxedad generalizada de las articulaciones relacionada con desordenes de la articulacion temporomandibular

Se ha sugerido que los desordenes de la articulacion temporomandibular posseen un origen multifactorial. Uno de los factores sugeridos es laxedad de los ligamentos de la articulacion. El proposito de este estudio fue evaluar la relacion entre hipermobilidad generalizada de las articulaciones con Desordenes de la Articulacion Temporomandibular (DATM). Un total de treinta y ocho voluntarios asintomaticos, y sesenta y dos pacientes con sintomas de la ATM fueron incluidos en este estudio. En el grupo de voluntarios asintomaticos ninguno presento: dolor de la ATM, limitaciones en la mobilidad mandibular, sonidos articulares or tratamiento previo de la ATM. Todos los pacientes poseian resualtados de analisis bilaterles por Resonacia Magnetica en el plano sagital con la boca abierta y cerrada, y en el plano coronal con boca cerrada. El test the Beighton fue empleado para evaluar laxedad de las articulaciones (generalizada); un resultado ≥ 4 fue considerado como laxedad generalizada de las articulaciones. El grupo de pacientes sintomaticos presento un incremento en la laxedad de las articulaciones cuando fue comparado con el grupo control (sin sintomas). El Indice de Probabilidad (OR + 4.0 [95% CI = 1.38–10.95, $P = .01$]). No se encontro ninguna differencia entre hombres y mujeres en el grupo de pacientes sintomaticos ($P > .05$). El presente estudio sugiere que existe una relacion positiva entre Laxedad Generalizada de las Articulaciones con los Desordenes de la Articulacion Temporomandibular.

Zusammenfassung

Generalisierte Gelenkerschlaffung Und Kiefergelenkserkrankungen

Man nimmt an, daß kiefergelenkserkrankungen eine Vielzahl von Faktoren zugrunde liegen. Als einer dieser Faktoren wird die Erschlaffung der Gelenksligamente angenommen. Ziel der vorliegenden Studie war, die Beziehung zwischen generalisierter Gelenkshypermobilität und Kiefergelenkserkrankungen zu untersuchen. Die Studie umfaßt 38 beschwerdefreie Freiwillige und 32 Patienten mit Beschwerden. Beschwerdefrei bedeutete den Ausschluss von: Kiefergelenksschmerzen, eingeschränkte Beweglichkeit des Kiefers, Gelenkgeräusche und Behandlung dieser Symptome. An allen Probanden wurden beidseitig Kernspintomographien der Sagittalenin geöffneter und geschlossener Position durchgeführt. Der Beighton-Test wurde angewendet, um über die aus einer Punkteskala von 1–4 ermittelten Werte die generalisierte Gelenkserschlaffung zu definieren. Die Probandengruppe mit Beschwerden wies, verglichen mit der beschwerdefreien Kongrolgruppe, eine erhöhte Gelenkserschlaffung auf. Die odds ratio war (OR = 4.0 [95% CI = 1.38 + 10.95, $P = .01$]). Bei den Probanden mit Beschwerden konte in Bezug auf Schlaffheit kein Unterschied zwischen männlich und weiblich festgestellt werden ($P = .05$). Diese Studie unterstützt die These, daß zwischen genelisierter Kiefergelenkserschlaffung und Kiefergelenkserkrankungen ein Zusammenhang besteht.