The Prevalence of Disc Displacement in Symptomatic and Asymptomatic Volunteers Aged 6 to 25 Years

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The purpose of this investigation was to determine a possible association between disc displacement and temporomandibular disorders (TMD). Fifty-six Brazilian asymptomatic volunteers (25 males and 31 females) and 181 symptomatic TMD patients (112 females and 69 males) participated. Volunteers did not have temporomandibular joint pain, limited jaw opening, joint sounds, or previous TMD treatment. Bilateral temporomandibular joint magnetic resonance imaging scans were obtained from all subjects. Joints were classified as normal or having disc displacement. Asymptomatic volunteers had 28 (25%) joints with disc displacement; 10 (18%) had unilateral and 9 (16%) had bilateral disc displacement. Of the TMD patients, 25 (13.8%) had bilateral symptomatic but normal joints. Fifty-one (28.2%) had unilateral and 105 (58%) had bilateral disc displacement. Odds ratios (12.2 [95% confidence interval = 6.1 to 24.4, P = .001]) suggest a strong association between disc displacement and TMD. This study suggests that disc displacement is relatively common (34%) in asymptomatic volunteers and is highly associated with patients (86%) with TMD.

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Temporomandibular disorders (TMD) is common in adults and has been reported to occur in children. A few studies[1,2] have suggested that disc displacement (DD) may not be uncommon in symptomatic children. The prevalence of DD in young adult autopsy specimens was 12%.[3] Epidemiologic studies[4–11] have suggested that signs and symptoms are present in children.

Dibbets and van der Weele[12] have documented the presence of osseous abnormalities in 11% and 35% of subjects equal to or less than 22 years of age and 23 to 24 years of age, respectively. Stewart and Standish[13] reviewed 346 patients with jaw-related disorders. Twenty-three patients were 19 years old or younger. Six (26%) were diagnosed as having degenerative joint disease (DJD) when tomograms were evaluated. These studies suggest DJD to be common in children.

There are opposing points of view as to the contribution of internal derangements to TMD. The prevalence of occult derangements ranges from 0% to 34% in asymptomatic volunteers.[14–18] The most recent investigation[16] suggests a prevalence of internal derangement to be 33% in asymptomatic subjects. Hellsing and Holmlund[19] suggest that internal derangement is an anatomic variant. Mills et al[20] have studied partial displacements in rabbits. Morphologic changes in the disc and posterior attachment are similar to findings in humans.[20] A recent study[21] suggests that patient complaints are not correlated with the degree of pathology. Disease is defined as an "objective biologic event" that involves disruption of specific body structures or organ systems caused by pathologic, anatomic, or physiologic changes. Internal derangement is often associated with anatomic changes in the articular disc, the articular eminence, and the mandibular condyle. There are frequently changes in mandibular movement and function; when this represents a pathologic event or becomes painful is uncertain. Schiffman et al[22] have suggested that there is no correlation between the severity of derangement and pain in a sample of TMD patients. This would suggest that a joint with derangement does not have to be chronic (ie, DJD) to be painful and that the degree of pathology may not be as important as the presence of the derangement. The prevalence of internal derangement is greater in symptomatic patients than in asymptomatic volunteers.[23–26] This would suggest that internal derangement makes some contribution to mandibular dysfunction, the severity of which does not correlate with pain.

The recognition of derangement or the potential of derangement may be important when performing dental treatments. Derangement may represent a risk factor that may compromise treatment outcomes. Patients may have pain during orthodontic treatment, restorative care, or oral surgery. Several studies cite the prevalence of temporomandibular joint pain after orthognathic surgery. Kahnberg[27] reported 60% of patients, symptom-free prior to surgery, had symptoms after treatment. Kerstens et al[28] reported that 11.5% of patients developed symptoms after surgery. Link and Nickerson[29] reported that 38 of 39 patients who had orthognathic surgery had internal derangements. Eriksson et al[30] reported that 11 of 21 consecutive orthognathic surgical patients had disc displacement. It is not completely understood why symptoms may develop during or after orthodontic treatment, restorative treatment, or oral surgery procedures. The prevalence of occult derangements may represent a risk factor and may be one explanation why symptoms develop in a patient previously asymptomatic. If derangements are very common in symptomatic patients, it should be no surprise that occult derangements are present prior to dental treatment or trauma.

The purpose of the present study was to evaluate the prevalence of disc displacement in asymptomatic subjects aged 6 to 25 years and to compare these findings to symptomatic TMD subjects to determine whether an association between disc displacement and TMD exists.

Materials and Methods

Fifty-six asymptomatic Brazilian volunteers (112 joints, 25 males and 31 females) and 181 symptomatic TMD patients (362 joints, 112 females and 69 males) from Eastman Dental Center Department of Temporomandibular Joint Disorders were included in this study. All volunteers were selected on the basis of not having temporomandibular joint (TMJ) pain, limited jaw opening, joint sounds, or previous TMD treatment. The joints were palpated during open and close maneuvers for the presence of vibrations that might be suggestive of joint noise. A stethoscope was also used to evaluate for the presence of sound. A vibration transducer was not used because this would have introduced false positive and negative findings.[14] The symptomatic patients were selected from 181 consecutive patients aged 6 to 25 years and were evaluated at the Eastman Dental Center Department of Temporomandibular Joint Disorders. The symptomatic TMD patients all had localized TMJ pain. They also may have had one or more of the following complaints: joint sounds (clicking/crepitation); limitation of jaw opening; jaw locking; or headache. To be included in this study, patients had to have pain greater than 4 on a visual analog scale, where 0 represented no pain and 10 represented the most intense pain they could imagine.

There were no asymptomatic or symptomatic subjects who had rheumatoid arthritis or generalized joint disorders. All asymptomatic and symptomatic subjects in this study had bilateral TMJ magnetic resonance imaging (MRI) scans acquired in paired oblique sagittal (closed and open) and oblique coronal (closed) planes along the long axis of the condyle, as described by Musgrave et al.[31] Magnetic resonance imaging assessment of each subject was performed with a 1.5-tesla MR system (Signa scanner, General Electric Medical Systems, Milwaukee, WI) utilizing high resolution 6 × 8-cm surface coils with the jaw in the closed position and at maximal mouth opening. An axial localizer, with a 52-second scanning time, was performed with a TR of 400 milliseconds, a TE of 16 milliseconds, a field of view (FOV) of 18 cm, a slice thickness of 3 mm, and a 256 × 128 scanning matrix. These images were acquired in the sagittal and coronal planes of both TM joints in the closed jaw position utilizing a TR of 2,000 milliseconds, TEs of 19 and 80 milliseconds, 3-mm image slice thickness, FOV of 10 cm, and a scanning matrix of 256 × 192. This was followed by paired oblique open jaw images with a TR of 1500 milliseconds, and TEs of 19 and 80 milliseconds. The MR studies were independently assessed by two readers (RFR, RWK) using established criteria for disc displacement and blinding as to clinical information.

The joints were classified as having *(1)* normal disc position (Figs 1a to 1c) or *(2)* DD (lateral, medial, anterior, anterolateral, or anteromedial). These included joints with DD with reduction (Figs 2a to 2c), DD without reduction, and DD without reduction associated with DJD (Figs 3 to 5).[23–26]

Statistical Analysis

Joints were classified as normal or having DD. Odds ratios (OR) and corresponding 95% confidence intervals (CI) were calculated to evaluate the strength of the association between DD and TMD (Proc FREQ, SAS Institute, Cary, NC). Statistical significance was accepted when $P < .05$.

Fig 1a Sagittal scan of 16-year-old asymptomatic male volunteer with normal disc position. In the closed-mouth position, the posterior band *(large white arrow)* is located superior to the condyle head, the thin zone *(small white arrow)* is located between the condyle and the posterior wall of the articular eminence (e), and the anterior band *(open arrow)* is located anterior to the condyle (c).

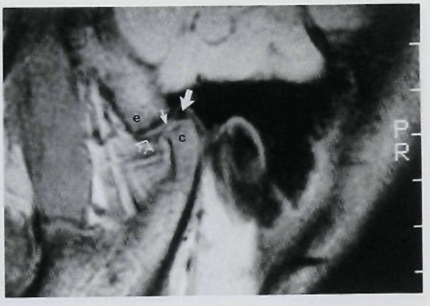


Fig 1b Sagittal scan (same volunteer as in Fig 1a) in the open-mouth position. The posterior band *(large white arrow)* is located posterior to the condyle (c). The thin zone *(small white arrow)* is located between the condyle and the apex of the articular eminence (e). The temporal and condylar parts of the posterior attachment can be seen clearly *(arrowheads)*.

Fig 1c Coronal scan (same volunteer as in Fig 1a) in the closed-mouth position. The disc is not visualized on the medial or lateral poles *(arrows)* of the condyle (c).

Fig 2a Sagittal scan of 12-year-old asymptomatic female volunteer with anterolateral disc displacement with reduction. In the closed-mouth position, the deformed posterior band *(arrow)* is located anterior to the condyle (c). The thin zone and anterior band are not well visualized (e = articular eminence).

Fig 2b Sagittal scan (same volunteer as in Fig 2a) in the open-mouth position. The three parts of the reduced disc are well visualized: the posterior band *(large white arrow)*; the thin zone *(small white arrow)*; and the anterior band *(open arrow)* (c = condyle, e = articular eminence).

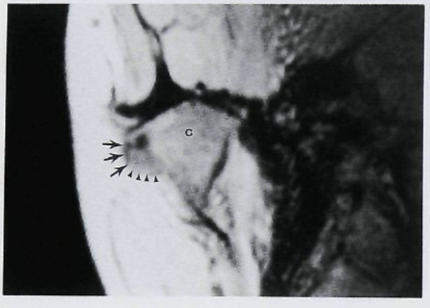
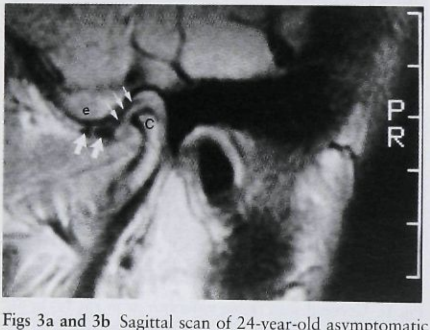
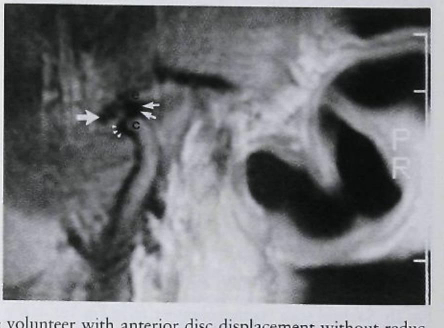


Fig 2c Coronal scan (same volunteer as in Fig 2a) in the closed-mouth position. The partially laterally displaced disc *(arrows)* can be seen lateral to the condyle (c). This may represent capsular hypertrophy *(arrowheads)*.





Figs 3a and 3b Sagittal scan of 24-year-old asymptomatic male volunteer with anterior disc displacement without reduction. *(Left)* Parasagittal scan in the closed-mouth position. The decreased signal from the posterior attachment *(small arrows)* suggests fibrotic changes. The deformed disc *(large arrows)* is located anterior to the condyle head (c). *(Right)* Parasagittal scan in the open-mouth position. The fibrotic posterior attachment is located between the condyle and apex of the articular eminence (e) *(small arrows)*. The deformed disc *(large arrow)* can be seen anterior to the condyle (c). There is a decreased signal on the anterior aspect of the condyle *(arrowheads)*, which suggests early osteophyte formation.

Figs 4a and 4b Sagittal scan of 21-year-old asymptomatic female volunteer with partial anterior disc displacement in the lateral third of the joint. *(Left)* In the most lateral parasagittal closed-mouth section, the thin, folded, anteriorly displaced disc can be seen *(arrows)*. *(Right)* In the medial section (oblique sagittal), the posterior band *(arrow)* can be seen in a more posterior superior position (e = articular eminence, c = condyle).

Results

Tables 1 to 3 show results of the study. Of the 112 joints of the asymptomatic volunteers, 28 joints (25.0%) displayed DD. Nineteen of the 56 volunteers (33.9%) had DD: 10 (17.9%) had unilateral DD; and 9 (16.1%) had bilateral DD. Three volunteers (5.4%) had unilateral DJD: 2 had normal disc position; and 1 had disc displacement (Fig 3b). The bony changes with the 2 normal discs were similar to the slight osteophyte formation seen in Fig 3b. In the symptomatic TMD group, 25 of 181 subjects (13.8%) had bilateral symptomatic but normal joints. Fifty-one of 181 subjects (28.2%) had unilateral DD, and 105 (58%) had bilateral DD. Ten patients (5.5%) had unilateral DJD, and 26 patients (14.4%) had bilateral DJD. The odds ratio (OR = 12.2 [95% CI = 6.1 to 24.4, $P = .001$]) suggests a high prevalence of DD in patients presenting with TMD.

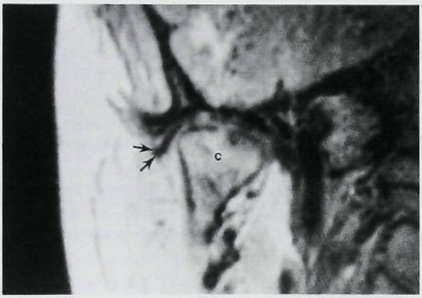


Fig 5 Oblique coronal scan of 24-year-old asymptomatic female volunteer with lateral disc displacement. The disc *(arrows)* can be seen directly lateral to the condyle (c).

Discussion

The prevalence of joint abnormalities in the absence of symptoms has been shown to occur in other joints. Disc abnormalities have been demonstrated in the knee, the cervical spine, and the lumbar spine.[32–36] It has also been suggested that subjects with TMD have a higher prevalence of other joint problems[37] and joint laxity.[38] It is possible that disc abnormalities may represent a joint phenotype that predisposes to joint abnormalities, including temporomandibular joint disc displacement. There are several studies that suggest a relationship of altered collagen metabolism and joint abnormalities. Temporomandibular disorders has been noted with increased frequency in individuals with mitral valve prolapse, indicating a possible etiologic association with altered collagen metabolism.[38,39] Aortic aneurism has been suggested to be related to collagen defects, although this is less clear.[40] Altered collagen metabolism may be important in joint laxity. Higher ratios of type III to type III + I collagen have been reported in patients with TMJ derangement and systemic joint laxity when compared to control subjects.[41,42]

Table 1 Age Range of Volunteers and Patients

Age	Asymptomatic volunteers with normal joint		Asymptomatic volunteers with DD		Symptomatic patients with TMD	
	n	%	n	%	n	%
6–7	4	10.8	2	10.5	3	1.7
8–11	9	24.3	2	10.5	12	6.6
12–15	4	10.8	5	26.3	63	34.8
16–19	11	29.7	3	15.8	98	54.1
20–23	8	21.6	5	26.3	4	2.2
24+	1	2.7	2	10.5	1	0.6
Total	37	100	19	100	181	100

Table 2 Sex and Distribution of Asymptomatic Volunteers Who Have Normal Joints or DD

Group*	Females		Males		Total	
	n	%	n	%	n	%
1/1	20†	35.7	17†	30.3	37	66.0
2/1	6	10.7	3	5.4	9	16.1
2/2	5	8.9	3	5.4	8	14.3
3/3	0	0.0	1	1.8	1	1.8
4/1	0	0.0	1	1.8	1	1.8
Total	31	55.3	25	44.7	56	100

*Groups: 1/1 = both joints are normal; 2/1 = one joint has DD with reduction and other joint is normal; 2/2 = both joints have DD with reduction; 3/3 = both joints have DD without reduction; 4/1 = one joint has DD without reduction and other joint has DJD.

†Two volunteers with DJD had normal disc position.

Table 3 Sex and Distribution of Symptomatic TMD Patients Who Have Normal Joints or DD

Group*	Females		Males		Total	
	n	%	n	%	n	%
1/1	14	7.7	11	6.1	25	13.8
2/1	17	9.4	5	2.8	22	12.1
2/2	19	10.5	7	3.9	26	14.4
3/1	13	7.2	11	6.1	24	13.3
3/2	18	9.9	3	1.7	21	11.6
3/3	11	6.1	8	4.4	19	10.5
4/1	3	1.7	2	1.1	5	2.8
4/2	3	1.7	5	2.8	8	4.4
4/3	1	.5	4	2.2	5	2.8
4/4	13	7.2	13	7.2	26	14.4
Total	112	55.4	69	38.1	181	100

*Groups: 1 = normal joint; 2 = DD with reduction; 3 = DD without reduction; 4 = joint without reduction and with DJD.

Genetic (inherited) predisposition to altered collagen metabolism may result in a morphologic (developmental) abnormality of any joint, including the temporomandibular joint. These individuals may be at increased risk for developing TMD when also subjected to other etiologic factors. Systemic joint laxity has been reported to occur more frequently in female adolescents than in male adolescents. These findings may represent a sex-limited expression of joint laxity.[43–45] Temporomandibular disorders is much more prevalent in females. Scientific support for sex-limited joint physiology has been found in animal models. A direct link of estrogen to the development of osteoarthritis in rabbit knees has been suggested. Upregulation of estrogen receptors in cartilage might initiate the osteoarthritic changes.[43] Ben-Hur et al[46] have suggested that estrogen acts directly on chondrocytes through an estrogen receptor–mediated mechanism. In a study[47] of induced inflammatory arthritis in rodents, female mice demonstrated a greater ability to degrade cartilage. A study[48] of estrogen and progesterone receptors in the human TMJ concluded that the TMJ disc is potentially, in at least some males and females, a female sex hormone target tissue. Further investigations are necessary to elucidate the significance of estrogen receptors in the pathogenesis of joint destruction.

The TMJ may be no different than any other joint. It is susceptible to arthritic changes that may follow disc displacement. It is thought that the human intervertebral disc begins degeneration early in life.[49] The intervertebral disc in adults and adolescents does not contain vessels. In disc degeneration herniations, calcifications and posterior margin protrusions are observed on MR images. Hassler[50] suggested that the blood supply of the intervertebral disc is impaired with increasing age. He suggested that this starts early in life and may be responsible for degeneration in adolescents. Severe disc degeneration is also accompanied by secondary vascular growth. Kauppila[51] has suggested that there is an ingrowth of vessels into the area of disc herniation. It has been suggested by Boden et al[33] that there is a high prevalence of lumbar herniation in asymptomatic volunteers. Thirty-five percent of the volunteers between ages 20 and 39 years had degeneration or bulging of the disc. Two recent studies[20,52] have suggested that there is an increase in vascular elements in the completely or partially displaced rabbit TMJ disc. This may suggest that ingrowth of vessels into the intervertebral disc might be secondary to herniation. The ingrowth of vessels may represent the reparative process or reorganization of degenerated disc tissue, as described by Hassler[50] and Kauppila.[51]

Synovial and vascular tissue responses to a perforation in the disc of monkeys has been shown by Helmy et al.[53,54] Despite the body's attempts at repair through synovial migration, the products of these cells are degradative and may enhance the degenerative process. These events may partially explain the progression from asymptomatic to adaptive to dysfunctional.

There was a prevalence of DD in 34% of our asymptomatic volunteers and 86% of our symptomatic TMD subjects. Kircos et al[15] have suggested that 32% of the joints in 26 asymptomatic adult volunteers have DD according to MRI findings. There was no reporting on a per subject basis. Tasaki and Westesson[55] examined 114 joints in 57 asymptomatic volunteers and reported a prevalence of DD in 21%. Alexander et al[56] found that 13% of symptom-free individuals had DD that was not influenced by condyle position. Hans et al[57] found that DD occurred in 11.8% of the patients that they assessed prior to orthodontic treatment (age 8 to 15 years). The results of our study are consistent with previous studies,[14–16,18] but they contradict the observations of Kaplan et al.[17] Kaplan et al[17] did not find DD in 28 joints of asymptomatic volunteers evaluated by arthrography. The probable explanation for this might be that MR imaging has a higher accuracy for the diagnosis of sideways displacements,[23–26,55] but it does not seem possible that all joints would have false negative arthrogram results. There is no clear explanation for their negative findings.

The accuracy of MRI for assessment of disc position has been reported to be 73% to 95%.[55,58–60] This findings could modify the results of the present study. If the worst-case scenario is considered (MRI has an accuracy of 73%), it can be assumed that MRI is inaccurate in 27% of the diagnoses. This may suggest that the 8 of the 28 joints with DD or the 5 of the 19 volunteers with DD may have been overestimated or underestimated (20 to 36 joints, DD; 14 to 24 volunteers, DD). This might suggest that the prevalence of DD in 6- to 25-year-olds might vary from 25% to 43%. This could change the prevalence of DD in the present study but still suggests a high prevalence of DD in asymptomatic volunteers.

Earlier studies[61,62] of symptomatic patients have suggested a high prevalence of disc displacement and TMD. Our study also demonstrates a high prevalence of DD in symptomatic subjects. There was a very strong statistical difference between asymptomatic volunteers and symptomatic subjects with regard to the prevalence of DD. Katzberg et al[1] and Sanchez-Woodworth et al[2] suggested a high prevalence of DD in symptomatic children (12% were bilateral). They also suggested that more than one third had DJD. Dibbets and van der Weele[12] have demonstrated an increase in DJD in a longitudinal investigation. These studies suggest that DD and DJD are very common in children.

Animal models for osteoarthritis have been developed to investigate its pathogenesis in other joints. Rabbits, guinea pigs, and dogs have been the animals of choice. Disease has been introduced by surgical manipulation, immobilization, or the introduction of foreign substances.[20,63–69] Tallents et al[52] have also described these findings in the rabbit TMJ. Degenerative changes in most experimental situations occur in a 1- to 12-week period. Extensive alterations in bone, collagen, and proteoglycan have been observed in all studies. The changes seen in these joints are similar to what occurs in DD and DJD in the human.

Coggeshall et al[70] produced experimental inflammation in the cat knee. The activity of types III and IV sensory afferents were recorded at rest and with passive motion. Resting discharges were higher in the inflamed group than in the control group. Passive movements activated more units in the experimental group. Guilbaud et al[71] noted in the ankles of rats with experimental arthritis that a small degree of extension or flexion produced an increased number of discharges. Schaible et al[72] demonstrated that when arthritis is present, pain frequently occurs at rest or on movement within a normal working range. These experimental events are similar to those with TMD patients who have altered jaw motion, pain on movement, and pain at rest.

The issue of importance is why do discs (lumbar, knee, cervical spine, and TMJ) have tears, herniations, or displacements. The aforementioned discussion may suggest that these are biologic events (genetic, hormonal) occurring in these tissues. Disc abnormalities seem to be involved in pain mechanisms in animals. When a patient transforms from adaptive to pain is not understood.

Conclusions

There is a high prevalence of DD in asymptomatic children and young adults (34%); there is also a clear association between disc displacement and TMD. Our findings also support the notion that DD is highly correlated with TMD pain. The progression (if any) from nonpainful to painful DD is not understood.

Acknowledgments

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Resumen

La Prevalencia del Desplazamiento del Disco Articular en Sujetos con Sintomatologia de la Articulation Temporomandibular y Voluntarios Libres de Sintomas: Comprendidos entre los 6 y 25 Anos de Edad

El propósito de la presente investigacion fue el determinar la posible asociacion entre desplazamiento del disco articular (DD) con desórdenes de la articulación temporomandibular (DTM). Cincuenta y seis voluntarios brasilenos libres de sintomas de la articulación temporomandibular (25 hombres y 31 mujeres) y 181 pacientes con síntomas (112 mujeres y 69 hombres) formaron parte del estudio. Ninguno de los voluntarios (asintomaticos) presento dolores de la articulación temporomandibular, limitaciones en la apertura, sonidos articulares o tratamiento previo de la ATM. Todos los sujetos fueron sometidos a exámenes bilaterales de la articulación mandibular por medio de resonancia magnetica (MR). Las articulaciones fueron clasificadas como normales o con desplazamiento del disco articular. Los márgenes de probabilidad (odds ratios [OR]) fueron calculados para evaluar la associacion entre DD and DTM. En el grupo de voluntarios asintomáticos 19 sujetos (34%) o 28 articulaciones (25%) presentaron desplazamiento del disco articular. Siendo más especificos, de estos 19 sujetos, 10 (18%) presentaron un desplazamiento unilateral del disco articular y 9 (16%) un desplazamiento bilateral. En el grupo de pacientes con desórdenes temporomandibulares 25 (13.8%) presentaban sintomatologia bilateral, pero articulaciónes normales; 51 (28.2%) con desplazamiento unilateral y 105 (58%) con desplazamineto bilateral del disco articular. Los margenes de probabilidad (OR = 12.2 [95% CI = 6.1 – 24.4, P = .001]) sugirieren una fuerte asociacion entre DD y DTM. Este estudio sugiere que el desplazamiento del disco articular es relativamente común (34%) en voluntarios asintomáticos y que esta intimamente relacionado con pacientes (86%) que presentan desórdenes temporomandibulares.

Zusammenfassung

Die Hänfigkeit der Diskusverschiebung im Kiefergelenk des symptomatischen und symtlosen Ingendlichen im von 6 vis 25 Jahren

Der Zweck dieser Untersuchung war einen Zusammenhang zwischen Diskusverschiebung (DD) und TMD nachzuweisen. 56 asymptomatische Freiwillige (25 Maenner und 31 Frauen) und 181 symptomatische TMD-Patienten (112 Frauen und 69 Maenner) wurden in die Studie einbezogen. Die Freiwilligengruppe hatte keinen TMJ-Schmerz, keine begrentzte Kieferoeffnungsfaehigkeit, oder vorherige TMD Behandlung. Alle Personen wurden auf TMJ MR hin untersucht. Die Gelenke wurden als normal oder zwischen DD und TMD klassifiziert. Es wurden Wahrscheinlichkeiten berrechnet um den Zusammenhang zwischen DD und TMD zu beurteilen. In der asymptomatischen Freiwilligengruppe hatten 28 (25%) Gelenke und 19 (34%) DD. Davon hatten zehn (18%) unilaterale und 9 (16%) bilaterale DD. Von den TMD Patienten hatten 25 (13.8%) bilaterale Symptome aber normale Gelenke. 51 (28.2%) hatten unilaterale und 105 (58%) hatten bilaterale DD. OR (OR = 12.2 [95% CI = 6.1 – 24.4, P = .001]) legt einen starken Zusammenhang zwischen DD und TMD nahe. Diese Studie deutet darauf hin, dass DD relativ verbreitet in asymptomatischen Personen ist und in starkem Zusammenhang mit TMD (86%) steht.