Editor:
LARRY J. PETERSON, DDS
Professor and Chairman
Department of Oral and Maxillofacial Surgery
The Ohio State University College of Dentistry
2131 Postle Hall
305 West 12th Avenue
Columbus, Ohio 43210-1241

Temporomandibular joint dysfunction

Connective tissue variations in skin biopsy and mitral valve function

Lilian Westling, DDS,[a] Sten Holm, PhD,[b] and Ingemar Wallentin, MD, PhD,[c]
Göteborg, Sweden

FACULTY OF ODONTOLOGY AND SAHLGREN HOSPITAL, UNIVERSITY OF GÖTEBORG

Ten women with temporomandibular joint dysfunction and general joint hypermobility (score, 4 to 8) and 10 symptom-free female volunteers without systemic laxity (score, 0 to 2) were selected for the study. A biopsy of connective tissue from arm skin found that the total collagen concentrations were lower and the proteoglycan values were higher in the hypermobile TMJ patients than in the control subjects. The mitral region of the heart was inspected by echocardiography. Eight patients and four controls had slightly abnormal echocardiographic findings. Two patients fulfilled the criteria for mitral valve prolapse. The patients had significantly more musculoskeletal complaints than did the controls. The study suggests an association between joint hypermobility, abnormal skin connective tissue composition, mitral valve malfunction, and musculoskeletal disorders in young women with TMJ dysfunction, especially internal derangement.
(ORAL SURG ORAL MED ORAL PATHOL 1992;74:709-18)

Significant correlations between systemic laxity of joints and disk displacement of the temporomandibular joint (TMJ) have been reported in several articles during the last decade.[1-7] It has also been noticed that generalized joint laxity may worsen the prognosis for the TMJ treatment.[8, 9]

The range of joint movement is determined by a variety of factors. Biochemical diversities in collagen and elastin structure may account for the variation in laxity. Collagens are widespread proteins in the body and the major constituent of skin, tendons, ligaments, joint capsules, and blood vessels. In inherited disorders of connective tissue, such as osteogenesis imperfecta, Marfan syndrome, and Ehlers-Danlos syndrome, there is collagen involvement.[10] Generalized joint hypermobility is a feature of each of these diseases. TMJ dysfunction has been reported in patients with Ehlers-Danlos syndrome.[11, 12]

The term *hypermobility syndrome* was coined by Kirk et al.[13] to denote the presence of rheumatic symptoms in otherwise healthy subjects in whom generalized joint laxity is the only observed abnormality. Subsequent reports have confirmed the associations between hypermobility and certain articular complications.[14-19] Similar observations have also been reported from pediatric clinics.[20-25]

Supported by grants from the Dental Practitioners Inc. (Praktikertjänst AB), Stiftelsen Wilhelm och Martina Lundgrens Vetenskapsfond and the Faculty of Odontology, University of Göteborg.
[a]Department of Stomatognathic Physiology, Faculty of Odontology, University of Göteborg.
[b]Associate Professor in Orthopedics, Sahlgren Hospital, Göteborg.
[c]Associate Professor in Clinical Physiology, Sahlgren Hospital, Göteborg.
7/12/37067

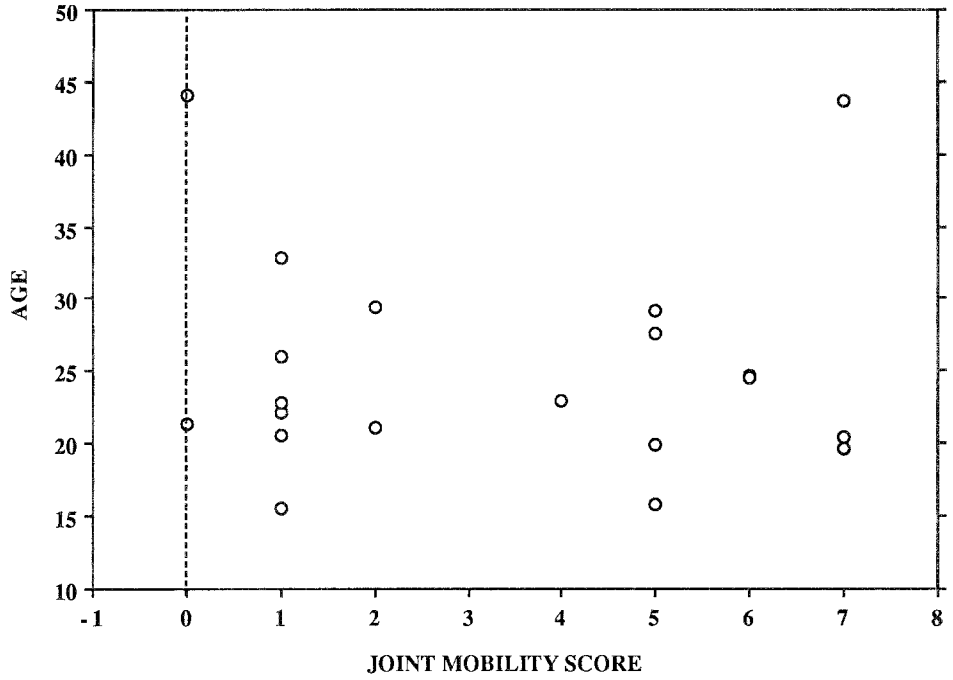

Fig. 1. Distribution of age and joint mobility scores in 10 female TMJ patients (score, 4 to 7) and 10 female controls (score, 0 to 2).

It has been suggested that the hypermobility syndrome is not only a benign locomotor disorder but may be an atypical form of a hereditary disorder of connective tissue.[26, 27] Easy bruising and varicose veins have been seen more frequently in hypermobile persons.[15, 19] Echocardiographic evidence of mitral valve prolapse (MVP), a reduced upper segment/lower segment (US/LS) ratio, and reduced skin thickness were found to be significantly more common among the hypermobile patients who attended a rheumatologic clinic than among the controls.[26] Hypermobile patients with joint pain have been found to have a greater incidence of MVP and a higher type III to III + I collagen ratio than those in the general population.[28] In patients with MVP, joint laxity was significantly more common than in control groups without MVP.[29, 30] In another study,[31] hypermobility was found in more than half of the MVP patients and the hypermobile patients had an increased function of antigen B 35.

In a study of MVP patients, Kim et al.[32] found a significant correlation between internal derangement of the TMJ and MVP in women. Waite[33] reported that 67% of the subjects with MVP had some degree of TMJ dysfunction. Half of the MVP patients with TMJ dysfunction and 10% of the MVP patients without TMJ dysfunction had hyperlax joints.[33]

The aim of this work was to compare hypermobile women with TMJ dysfunction and healthy nonlax women with respect to type III to type III + I collagen ratio and proteoglycans in skin biopsy specimens, mitral valve function, and signs of inherited connective tissue disorders.

MATERIAL AND METHODS

Study population

Ten patients treated for TMJ dysfunction at the Department of Stomatognathic Physiology, Göteborg, were selected for the study. The following criteria were applied: (1) female sex, (2) age <45 years, (3) TMJ instability (disk displacement or dislocation), (4) widespread general joint hypermobility (hypermobility score ≥ 4),[14] (5) agreement to undergo echocardiography, blood tests, and biopsy examinations, (6) no verified rheumatoid arthritis or other systemic inflammatory disease that involves joints or muscles. Ten female volunteers with no symptoms or signs of craniomandibular disorder, matched for age with the patients and with no demonstrable systemic laxity (hypermobility score = 0-2), took part in the investigation. Pregnant women were excluded from the study. Distributions of age and hypermobility scores of the participants are shown in Fig. 1.

All participants were randomly numbered from 1 to 20, and analyses were carried out blindly.

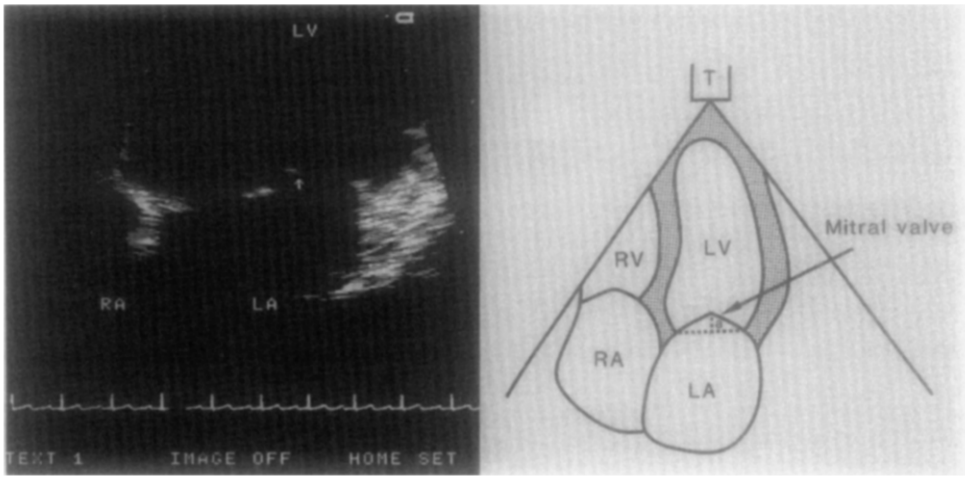

Fig. 2. **A,** Echocardiographic end-systolic still-frame of apical four-chamber view from video recording of mitral valve in closed position (case 11). *Arrow* shows coaptation point. **B.** Schematic drawing of apical four-chamber view in **A;** *a* = perpendicular distance between coaptation point and level of valve ring. Distance a = −5 mm (case 11). *RA* = right atrium, *LA* = left atrium, *RV* = right ventricle, *LR* = left ventricle.

Questionnaire

A self-administered questionnaire was completed before the clinical examinations. The questions were about general health, musculoskeletal symptoms and injuries, varicose veins, easy bruisability, orthodontic treatment, childbirth, tobacco habits, and physical exercise. No general physical examination was performed. Copies of records from rheumatologic and orthopedic clinics were requested when relevant.

Somatometric examination

Weight, height, arm span, and lower segment (distance from top of the pubic symphysis to the floor) were measured for calculation of body proportion indices. The body mass index was calculated by the formula—weight in kilograms/height^2 in meters.[34] The proportion of height to arm span was calculated as suggested by Steele and Mattox[35]: the ratio of arm span to height is multiplied by 100. The US/LS ratio was calculated. The US is derived by subtracting LS from height.[36]

Stomatognathic examination

Besides the routine procedures to record the clinical signs, measurements were made for estimation of the maximal angle of jaw opening.[37] Radiologic examinations of the TMJs, which included submentovertical projection, posteroanterior projection, and corrected sagittal tomography, were performed on six patients. Fluoroscopy of the TMJs was performed on two patients. Diagnoses were classified in accord with the International Headache Society's Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias and Facial Pain.[38]

Blood tests and biopsy

Venous blood was taken after an overnight fast with no exposure to smoke and with minimal physical exercise. Blood status was evaluated with measures of various electrolytes, glucose, cholesterol, triglycerides, blood proteins, and enzymes. These tests were performed on an analyzer (Kodak Ektachem DT 60, Eastman Kodak Co., Rochester, N.Y.).

After the patient was given a local anesthetic, a small full-thickness scissor biopsy specimen of the upper arm skin tissue was taken and immediately frozen at −70° C. Analysis of collagen types I and III was performed as recommended by Light[39] and the cross-linking analysis as recommended by Eyre and Oguchi.[40] Proteoglycan analysis was performed as recommended by Maroudas and Thomas.[41]

One patient and two subjects in the control group refused to participate in the blood tests and biopsies.

Heart examination

An Acuson-128 computerized sonograph (Acuson, Inc., Mountain View, Calif.) was used for echocardiographic and Doppler investigation.[42] Standard views—parasternal long and short axis, apical four-chamber, two-chamber, and apical long axis—were recorded on videotape (U-Matic). The mitral region was enlarged and could be inspected in slow motion. The mitral flow and the occurrence of mitral regurgi-

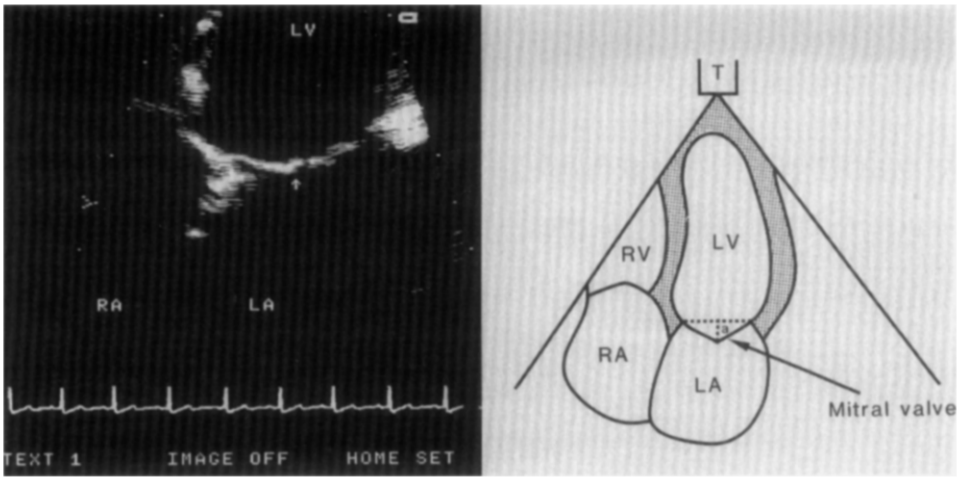

Fig. 3. **A.** Echocardiographic end-systolic still-frame of apical four-chamber view from video recording of mitral valve in closed position (case 18). Arrow shows coaptation point. **B.** Schematic drawing of apical four-chamber view in **A**; *a* = perpendicular distance between coaptation point and level of valve ring. Distance a = +2 mm (case 18). *RA* = right atrium, *LA* = left atrium, *RV* = right ventricle, *LR* = left ventricle.

Table I. Distribution of affirmative answers to the questionnaire among 10 hypermobile TMJ patients and 10 normal controls

	Patients	*Controls*
Musculoskeletal complaints	9	1*
Back pain	7	1†
Joint complaints that require medical care	5	0†
Varicose veins	1	2
Easy bruising	3	2
Raynaud's phenomenon	3	3
Orthodontics	5	2
Smoking	3	2
Regular physical training	8	7

*$p < 0.01$.
†$p < 0.05$.

tation were recorded with pulse, continuous, and colored Doppler flow.

Evaluation. The whole group was evaluated on one occasion without knowledge of whether the subjects were patients or control subjects. In the search for different signs of mitral disease the following factors were considered:

1. *Coaptation of the mitral valve.* The mitral valve was inspected frame by frame in an enlarged view. The coaptation point in relation to the mitral valve ring was measured with compasses. The level of the mitral ring was determined with the aid of a ruler, and the perpendicular distance of the coaptation point was measured in millimeters (−, 0, + where + indicated a position of the point in the left atrium). Figs. 2 and 3 illustrate normal and abnormal coaptations.
2. A. *Bulging.* The perpendicular distance of the leaflets in relation to the mitral ring was measured in late systole. Most bulging was seen in the four-chamber view compared with the two-chamber view.
2. B. *Signs of chordal stretching during systole.* If the coaptation point moved during systole toward the atrium, this was considered as an indication of stretching of the chorda (yes/no).
3. *Mitral regurgitation.* Mitral regurgitation was evaluated on a four-point scale (1 to 4). Degree <1 means very faint regurgitation during part of systole only.
4. The size of the left atrium in relation to the right one was evaluated visually, and equal size was rated 1.00.

Statistics

Differences between patients and controls in answers to the self-administered questionnaire were analyzed with the chi-squared test with continuity correction. Differences between patients and controls with respect to the parametric variables were analyzed with the unpaired *t* test. Pearson's product moment correlation coefficient was used to determine the linear correlation between the parametric variables.

RESULTS

The mean age of the patients was 24.8 years, and that of the controls was 25.6 years (Fig. 1). None of

Table II. Distribution of age, joint mobility score,[14] somatometric, and biopsy analysis values in four women TMJ patients

	Case			
	1	*8*	*17*	*18*
Age	24	27	43	29
Joint mobility score	6	5	7	5
X-ray finding	yes	—	yes	—
Somatometric variables				
US/LS	0.95	0.98	0.87	0.93
Arm-span/height × 100	104.7	99.4	101.2	98.2
Biopsy				
Total collagen*	48.1	46.8	42.6	40.7
Cross-link†	0.27	0.41	0.46	0.41
Proteoglycans‡	0.30	0.50	0.38	0.37

*In percent.
†Residues of pyridinolyne per collagen molecule.
‡Milliequivalents per gram of tissue.

Table III. Somatometric indexes: means, SD and tenth and ninetieth percentiles for 10 hypermobile TMJ patients and 10 normal controls

	All	*Patients*		*Controls*	
	Percentiles	*Mean*	*SD*	*Mean*	*SD*
Body mass index (weight/height^2)	15.9*	21.9	3.1	22.0	3.0
US/LS	0.90*	0.96	0.04	0.97	0.03
Arm span/height × 100	102.2†	100.0	2.1	100.1	1.7

*Tenth percentile.
†Ninetieth percentile.
$p > 0.05$.

the subjects had any obvious sign of connective tissue disorder.

Questionnaire

Statistically significant differences between patients and controls were found for musculoskeletal complaints (Table I). All but one of the patients had some articular complication. Five patients had complaints that required medical care: Patient 1 had been referred to and treated at a rehabilitation home for joint complaints. No diagnosis was stated. At age 15, patient 3 had a knee sprain immobilized for 20 weeks and had received physical therapy for kyphosis. Patient 7 had been treated at a rheumatologic clinic since the age of 17 because of joint pain and effusions. The laboratory data gave no evidence of any systemic inflammatory disease (antinuclear antibodies negative). However, transient rises (ESR 17) in erythrocyte sedimentation rate were noted. Because of joint instability, patient 8 had undergone surgery on the ligaments of both knees at the age of 24. Patient 17 took medicine (Voltaren) because of suspected fibromyalgia. Three of the five patients who required medical care for their musculoskeletal complaints are presented together with other findings in Table II.

Joint mobility and somatometric measurements

The distribution of the joint mobility score on the basis of age and group is presented in Fig. 1. The joint mobility score may on some occasions have been underestimated because of pain in the knees (cases 1 and 3) and because of knee surgery (case 8). Patient 7 hyperextended the right elbow to 14 degrees and the left elbow to 18 degrees.

The mean values of the somatometric measurements showed no significant differences between the patients and the controls (Table III). Two highly hypermobile patients (score = 7) (cases 6 and 17) had a US/LS ratio more than 2 SD below the mean value. One patient (case 1) had an arm span 7 cm greater than her height. This was more than 2 SD higher than the mean value of the controls (Table II).

Stomatognathic examination

Three patients had a diagnosis of articular disk displacement with reduction (and intermittent lock-

Table IV. Maximal mandibular opening angle: distribution of degrees in hypermobile TMJ patients ($n = 6$)* and normal controls ($n = 9$)†

	Mean	*Range*	*SD*
Patients	42.2 (45.3‡)	32.6 to 52.1	6.5
Controls	40.3	33.0 to 49.7	6.2

*Three missing because of pain on opening and one because of fear of dislocation.
†One missing because of orthodontic appliance.
‡Two patients with a diagnosis of chronic disk displacement without reduction excluded.
$p > 0.05$.

ing), two patients had acute disk displacement without reduction, three patients had chronic disk displacement without reduction, and two patients had TMJ hypermobility and a tendency to dislocation. Five patients evaluated their symptoms as severe or very severe.

Bruxism was reported by six patients, but it was not possible to relate that to any stomatognathic symptom or sign.

Seven patients had TMJ tenderness on palpation via the auditory meatus. Each of the five patients with severe musculoskeletal problems had posterior TMJ tenderness.

Radiographic examinations were performed on six patients, and in four (15, 19, 24, and 43 years of age) a pathologic condition—erosion, flattening, sclerosis, and/or osteophytes—could be found.

It was not possible to measure the passive mandibular opening in four patients—in three patients because of severe pain on opening and in one patient because of fear of dislocation. The other patients had, on average, a greater mandibular opening angle than the controls (Table IV). Two patients with chronic nonreducing disk displacement were sufficiently recovered to carry out the passive opening measurement. The mean value of active mandibular opening for the six measured patients was 51.2 mm (range, 41 to 60) and for the ten controls the mean was 53.3 mm (range, 44 to 64).

Blood tests and biopsy

The blood test values were all within normal ranges, and there were no significant differences between patients and controls.

Skin biopsies revealed lower values of total collagen, hydroxyproline, and hydroxypyridine in the patient group compared with the controls, whereas collagen type III to III + I ratios and proteoglycans showed higher values in the patients (Table V). The total collagen concentrations in two patients (cases 17 and 18) were lower than the mean value minus 2 SD in the control group (Table II). The proteoglycan value in case 17 was higher than the mean value plus two SD (Table II). These tendencies were mostly strengthened in patients with other musculoskeletal complaints or with severe clinical disorders of the TMJ such as posterior TMJ tenderness or pathologic x-ray findings.

Total collagen was significantly and negatively correlated to proteoglycans ($r = -0.74$; $p < 0.001$). The correlations between collagen type III to III + I ratios and values from the skin biopsies are shown in Table VI. The joint hypermobility score had a weak negative correlation with the total collagen value ($r = -0.38$, $p > 0.05$) and a weak positive correlation with the proteoglycans ($r = 0.38$, $p > 0.05$).

The values from the skin biopsies developed differently on the basis of age. In the control group, collagen parameters increased with age ($r = 0.72$, $p < 0.05$) and the concentration of proteoglycans decreased ($r = -0.44$, $p > 0.05$). The situation in the patient group was found to be the opposite, i.e., a decrease in collagen ($r = -0.60$, $p > 0.05$) and an increase in proteoglycan concentration ($r = 0.45$, $p > 0.05$) with an increase in age.

In those cases with suspected pathologic conditions that were found during the echocardiographic investigation, the mean values of the biopsy tests showed a pattern similar to that of the TMJ patient group (Table V), i.e., low collagen and high proteoglycan concentrations compared with those with no pathologic echocardiographic findings.

The upper segment/lower segment lower segment ratio was positive correlated to the collagen parameter ($r = 0.75$, $p < 0.001$).

Heart examination

Eight patients and four controls had some suspected findings in the echocardiographic and Doppler investigation.

1. In a clinical context, only three persons would have had the diagnosis of slight MVP—two patients (cases 3 and 18) and one subject from the control group (case 12) the last one because of some chordal stretching. However, when different signs of mitral function were considered, 10 persons (six patients and four controls) had a coaptation point at the ring level or toward the atrium (Table VII). At the level <-2 mm, four controls and one patient (case 17) had coaptation into the ventricle. Conversely, coaptation into the atrium $\geq +2$ mm was noted in two patients (cases 3 and 18) (Table VII).
2. A. Bulging of any of the leaflets (≥ 2 mm) was seen in five patients and two controls. Bulging of the posterior leaflet only was seen in four patients and one control, and bulging of the an-

Table V. Biopsy analysis values: means, SD, and tenth and ninetieth percentiles for nine hypermobile TMJ patients and eight normal controls

	All	*Patients*		*Controls*	
	Percentiles	*Mean*	*SD*	*Mean*	*SD*
Collagen type III: III + I	39.5§	33.2	6.5	31.7	6.9
Total collagen*	43.4‖	50.5	7.0	54.8	4.4
Cross-link†	0.20,‖ 0.55§	0.40	0.11	0.41	0.06
Proteoglycans‡	0.39§	0.33	0.08	0.27	0.07

*, † and ‡See Table II.
§Ninetieth percentile.
‖Tenth percentile.

terior leaflet was seen in two patients and two controls (Table VII). On summation of all coaptation points, the difference between the groups was obvious (−1; −28). Similarly, summation of the bulging of the leaflets amounted to 21 mm and 18 mm, respectively, in the patient group, versus 8 mm and 13 mm in the control group (Table VII).

2. B. Signs of chordal stretching during systole were seen in six persons—four patients and two controls.
3. Slight mitral regurgitation was seen in six persons—two patients and four controls. In all of these, the amount would have been considered as physiologic regurgitation in a clinical context.
4. Slight enlargement of the left atrium was seen in three patients and one control.

In the patient group, age was significantly correlated to the coaptation point of the mitral valve leaflet ($r = -0.64$, $p < 0.05$).

DISCUSSION

The study has given several indications that the connective tissue assembly is different among women TMJ patients with widespread general joint hypermobility compared with normal and symptom-free women. The small number of subjects in the study showed few statistically significant correlations. The same pattern, however, was found for the differences between the biopsy test values in TMJ patients and controls, in subjects with or without general musculoskeletal complaints, and in subjects with or without suspected pathologic findings in the echo-Doppler investigation. This data is provided in Tables V and VII.

Questionnaire

The most striking difference between patients and controls was in regard to musculoskeletal complaints. Even the youngest patients had widespread discomfort. This relationship indicates that the TMJ dysfunction in a hypermobile person seems to be a manifestation of a general locomotor disorder and strengthens the concept of a connective tissue disturbance. Joint pain and effusions together with transient rises in erythrocyte sedimentation rate have previously been reported in studies of hypermobile persons with no signs of systemic disease.[13, 15] Traumatic synovitis has been associated with overuse of hypermobile joints.[13]

Table VI. Pearson's product moment correlation coefficient *(r)* between collagen type III to III + I ratio and other biopsy analysis values in 17 women (16 to 44 years)

	All n = *17*	*Patients* n = *9*	*Controls* n = *8*
Total collagen*	−0.61§	−0.56	−0.75‖
Cross-link†	−0.62§	−0.39	−0.80‖
Proteoglycans‡	0.50‖	0.52	0.46

*, †, ‡ See Table II.
§$p < 0.01$.
‖$p < 0.05$.

Similar associations between musculoskeletal complaints and TMJ dysfunction have previously been reported.[3, 5] Although signs of TMJ osteoarthrosis were noted in four of the patients, no evidence of osteoarthrosis was found in other joints. The relative risk of severe knee osteoarthrosis is, however, significantly increased in persons with a history of knee injury.[43]

Because of the small number of subjects in the study, the distribution of some self-reported symptoms, such as easy bruisability and Raynaud's phenomenon, which on the whole appear infrequently in a population, must be interpreted with caution.

Somatometric measurements

One patient (case 17) among the 20 measured subjects had a diverging US/LS ratio (Table II). The normal range values in Scandinavian populations are, however, not well verified. The extreme value of the

Table VII. Distribution of echocardiographic findings in 10 women TMJ patients and 10 controls

Case No.	*Coaptation point* (mm)*	*Bulging* (mm)*		*Chordal stretching**	*Left: right atrium size**
		Posterior	*Anterior*		
1	0	2	2	yes	1.25
3	7	0	9	no	1.00
6	−1	5	1	yes	1.00
7	0	0	0	no	1.00
8	−2	7	0	no	1.00
15	0	0	0	yes	1.00
17	−5	0	1	no	1.10
18	2	3	3	yes	1.00
19	−2	1	0	no	1.00
20	0	3	2	no	1.10
Patient totals	−1	21	18	4 yes	3 >1.00
2	0	5	5	no	1.00
4	−8	0	−1	no	1.00
5	0	2	2	no	1.25
9	−3	−1	−2	no	1.00
10	−2	0	0	yes	1.00
11	−5	−2	−2	no	1.00
12	0	2	0	yes	1.00
13	−2	0	0	no	1.00
14	0	2	3	no	1.00
16	−3	0	−2	no	1.00
Control totals	−23	8	3	2 yes	1 > 1.00

*For detailed examination see section on Material and methods, Heart examination: 1, 2. A, 2. B, and 4 respectively.

somatometric measurement and the extreme collagen test value (Table II) may, together with her high mobility score, indicate a connective tissue syndrome. The preliminary diagnosis of fibromyalgia may well agree with these findings.

It has been proposed that tall, thin persons tend to be more loose jointed than those with a short stocky physique. No significant differences in height, weight, or body mass index between lax and nonlax subjects were found. These results are in accord with those of Beighton et al.[14]

Mandibular opening capacity

A vertical opening of more than 55 mm has been proposed as a reasonable criterion to determine the existence of hypermobility of the mandible in women.[44] In this study, four controls and three of six patients had a voluntary mandibular opening that exceeded 55 mm. In the measurements of maximal mandibular opening angle, however, the hypermobile patients had a greater mean value compared with the controls (Table IV). The great opening capacity found in two patients with chronic nonreducing disk displacement agreed with findings by Johansson and Isberg[45] and suggested TMJ hypermobility. In a double-contrast arthrotomographic investigation, Johansson and Isberg classified the TMJ as hypermobile when condylar translation exceeded the anterosuperior insertion of the TMJ capsule on the temporal bone. A great mandibular opening capacity that results from a condyle that surpasses the anterosuperior capsule insertion may not exclude the presence of a nonreducing disk.

Blood tests and biopsy

The syndrome of joint hypermobility and changes in the mitral valve function and collagen distribution are known to be inherited and to mainly affect women.[16, 26] There is also evidence that the Ehlers-Danlos syndrome involves a low type III skin collagen production in patients with MVP.[46] In accordance with the study of Handler et al.,[28] we find in our study an increased ratio of collagen type III to III + I in the patient group, which may be an indication of a basic collagen abnormality. The TMJ patients in this study fulfilled the criteria of joint hypermobility. Many of them showed a streaky, slightly loose skin structure. However, the study group is too small to permit definite conclusions about the quality of the skin collagen and, despite a relatively homogeneous patient

group, the individual variations must be taken into account.

Of interest is the finding that the collagen concentration decreased and the proteoglycans increased in the patient group as their ages increased, which was opposite the normal pattern. These findings confirm the association between a less collagen-enforced structure and the ability to stretch and form the more proteoglycan-rich tissue, a situation enhanced by the hypermobility score, which showed a negative correlation with the collagen and a positive correlation with the proteoglycan concentration.

Heart examination

Only two patients fulfilled the accepted criteria of MVP, namely, coaptation into the left atrium. Coaptation at the ring level (0) was seen in many of the controls but is not enough for the diagnosis of MVP. Overdiagnosis of MVP is also a well-known pitfall in young persons,[47] and this study underlines the risk of such overdiagnosis. However, the aim of the present study was not to define the limits between normal and pathologic mitral function but to determine whether differences could be seen between hypermobile TMJ patients and a nonlax symptom-free control group. The great differences in the summations of coaptation points and bulging indicate that such differences exist and point to a different behavior in the mitral leaflets (Table VII). The chordae tendineae are essential to the proper function of the mitral valve. The difference in chordal stiffness between the groups may be explained by variations in the collagen structure and suggests a relationship between joint hypermobility and chordal stretching. The mechanical behavior of chordae is supposed to be consistent with the behavior of other collagenous structures.[48]

REFERENCES

1. Bates RE, Stewart CM, Atkinson WB. The relationship between internal derangements of the temporomandibular joint and systemic joint laxity. J Am Dent Assoc 1984; 109:446-7.
2. Hecht C, Adair S. Generalized joint hypermobility and TMJ signs and symptoms in children [Abstract]. J Dent Res 1985;64:232.
3. Harinstein D, Buckingham RB, Braun T, et al. Systemic joint laxity (the hypermobility joint syndrome) is associated with temporomandibular joint dysfunction. Arthritis Rheum 1988; 31:1259-64.
4. Westling L. Craniomandibular disorders and general joint mobility. Acta Odontol Scand 1989;47:293-9.
5. Westling L, Carlsson GE, Helkimo M. Background factors in craniomandibular disorders: with special reference to general joint hypermobility, parafunction and trauma. J Craniomandib Disord Facial Oral Pain 1990;4:89-98.
6. Westling L, Mattiasson A. Background factors in craniomandibular disorders: reported symptoms in adolescents with special reference to general joint hypermobility and oral parafunctions. Scand J Dent Res 1991;99:48-54.
7. Westling L, Mattiasson A. General joint hypermobility and temporomandibular joint derangement in adolescents. Ann Rheum Dis 1992;51:87-90.
8. Solberg WK. Temporomandibular disorders: clinical significance of TMJ changes. Br Dent J 1986;160:231-6.
9. Helkimo E, Westling L. History, clinical findings, and outcome of treatment of patients with anterior disk displacement. J Craniomandib Pract 1987;5:269-76.
10. Prockop DJ, Kirvikko KI. Heritable diseases of collagen. N Engl J Med 1984;311:376-85.
11. Goodman RM, Allison ML. Chronic temporomandibular joint subluxation in Ehlers-Danlos syndrome: report of case. J Oral Surg 1969;27:659-61.
12. Gosney MBE. Unusual presentation of a case of Ehlers-Danlos syndrome. Br Dent J 1987;163:54-6.
13. Kirk JA, Ansell BM, Bywaters EGL. The hypermobility syndrome: musculoskeletal complaints associated with generalized joint hypermobility. Ann Rheum Dis 1967;26:419-25.
14. Beighton P, Solomon L, Soskolne CL. Articular mobility in an African population. Ann Rheum Dis 1973;32:413-8.
15. Bird HA, Tribe CR, Bacon PA. Joint hypermobility leading to osteoarthrosis and chondrocalcinosis. Ann Rheum Dis 1978; 37:203-11.
16. Grahame R. Heritable disorders. In: Scott JT, ed. Copeman's textbook of the rheumatic diseases. New York: Churchill Livingstone, 1978:835-7.
17. Scott D, Bird H, Wright V. Joint laxity leading to osteoarthrosis. Rheum Rehabil 1979;18:167-9.
18. Finsterbush A, Pogrund H. The hypermobility syndrome: musculoskeletal complaints in 100 consecutive cases of generalized joint hypermobility. Clin Orthop 1982;168:124-7.
19. AL-Rawi ZS, AL-Aszawi AJ, AL-Chalabi T. Joint mobility among university students in Iraq. Br J Rheum 1985;24:326-31.
20. Bird HA, Wright V. Joint hypermobility mimicking pauciarticular juvenile chronic arthritis. Br Med J 1978;3:402-3.
21. Scharf Y, Nahir AM. Hypermobility syndrome mimicking juvenile chronic arthritis. Rheum Rehabil 1982;21:78-80.
22. Biro F, Gewanter HL, Baum J. The hypermobility syndrome. Pediatrics 1983;72:701-6.
23. Gedalia A, Person DA, Brewer EJ, Giannini EH. Hypermobility of the joints in juvenile episodic arthritis/arthralgia. J Pediatr 1985;107:873-6.
24. Lewkonia RM, Ansell BM. Articular hypermobility simulating chronic rheumatic disease. Arch Dis Child 1983;58:988-92.
25. Arroyo IL, Brewer EJ, Giannini EH. Arthritis/arthralgia and hypermobility of the joints in schoolchildren. J Rheumatol 1988;15:978-80.
26. Grahame R, Edwards JC, Pitcher D, Gabell A, Harvey W. A clinical and echocardiographic study of patients with the hypermobility syndrome. Ann Rheum Dis 1981;40:541-6.
27. Child A. Joint hypermobility syndrome: inherited disorder of collagen synthesis. J Rheumatol 1986;13:239-43.
28. Handler CE, Child A, Light ND, Dorrance DE. Mitral valve prolapse, aortic compliance, and skin collagen in joint hypermobility syndrome. Br Heart J 1985;54:501-8.
29. Evans P, Huges D, Smith S, Boineau J. Joint laxity associated with the mitral valve prolapse syndrome [Abstract]. Circulation 1976;54:405.
30. Pitcher D, Grahame R. Mitral valve prolapse and joint hypermobility: evidence for a systemic connective tissue abnormality? Ann Rheum Dis 1982;41:352-4.
31. Onrasik M, Rybar I, Rus V, Bosak V. Joint hypermobility in primary mitral valve prolapse patients. Clin Rheumatol 1988;7:69-73.
32. Kim SM, Cohen SG, Douglas P. Relationship between internal derangement of TMJ and mitral valve prolapse [Abstract]. J Dent Res 1989;68:416.
33. Waite PD. Evaluation of 49 mitral valve prolapse patients for maxillofacial skeletal deformities and temporomandibular joint dysfunction. ORAL SURG ORAL MED ORAL PATHOL 1986;62:496-9.

34. Symmons DPM, Van Hemert AM, Vandenbroucke JP, Valkenburg HA. A longitudinal study of back pain and radiological changes in the lumbar spines of middle aged women. I. Clinical findings. Ann Rheum Dis 1991;50:158-61.
35. Steele MF, Mattox JW. Correlation of arm-span and height in young women of two races. Ann Human Biol 1987;14:445-7.
36. McKusick VA. The Marfan syndrome. In: McKusick VA, ed. Heritable disorders of connective tissue, 4th ed. St Louis: CV Mosby, 1972:72-3.
37. Westling L, Helkimo E. Maximum jaw opening capacity in adolescents in relation to general joint mobility. J Oral Rehabil 1992. In press.
38. McNeil C, ed. Craniomandibular disorders: guidelines for evaluation, diagnosis, and management. Chicago: Quintessence Publishing Inc, 1990:15-7.
39. Light ND. Estimation of types I and III collagens in whole tissue by quantification of CNBr peptides on SDS polyacrylamide gels. Biochim Biophys Acta 1982;702:30-6.
40. Eyre DR, Oguchi H. The hydroxypyridinium crosslinks of skeletal collagens: their measurements, properties and a proposed pathway for formation. Biochem Biophys Res Commun 1980;92:403-10.
41. Maroudas A, Thomas H. A simple physicochemical micromethod for determining fixed anionic groups in connective tissue. Biochim Biophys Acta 1970;215:214-6.
42. Feigenbaum H. Echocardiography, 4th ed. Philadelphia: Lea & Febiger, 1986.
43. Felson DT. The epidemiology of knee osteoarthritis: results from the Framingham osteoarthritis study. Semin Arthritis Rheum 1990;20(suppl 1):42-50.
44. Plunkett GAJ, West VC. Systemic joint laxity and mandibular range of movement. J Craniomandib Pract 1988;6:320-4.
45. Johansson A-S, Isberg A. The anterosuperior insertion of the TMJ capsule and condylar mobility in joints with and without internal derangements. In: Johansson-Garnier A-S. Temporomandibular joint internal derangement: tissue reactions and topographical relations with implication on pain. Stockholm: Karolinska Institutet, 1990.
46. Jaffe AS, Geltman EM, Rodey GE. Mitral valve prolapse: a consistent manifestation of type IV Ehlers-Danlos syndrome—the pathogenetic role of the abnormal production of type III collagen. Circulation 1981;64:121-5.
47. Malcolm AD. Mitral valve prolapse associated with other disorders: casual coincidence, common link, or fundamental genetic disturbance? Br Heart J 1985;53:353-62.
48. Kunzelman KS, Cochran RP. Mechanical properties of basal and marginal mitral valve chordae tendineae. Trans Am Soc Artif Intern Organs 1990;36:405-8.

Reprint requests:
Lilian Westling, DDS
Department of Stomatognathic Physiology
Faculty of Odontology
University of Göteborg
Box 33070
S-400 33
Göteborg, Sweden