Clinical Signs of TMJ Osteoarthrosis and Internal Derangement 30 Years After Nonsurgical Treatment

Reny de Leeuw, DDS
Research Fellow
TMJ Research Group

Geert Boering, DDS, PhD
Professor and Chairman

Boudewijn Stegenga, DDS, PhD
Research Associate
TMJ Research Group

Lambert G.M. de Bont, DDS, PhD
Associate Professor

Department of Oral and Maxillofacial Surgery
University Hospital
PO Box 30.001
9700 RB
Groningen, The Netherlands

Correspondence to Dr de Leeuw

To evaluate the clinical course of temporomandibular joint osteoarthrosis and internal derangement, 99 patients who had received nonsurgical treatment for temporomandibular problems were reexamined 30 years after the original treatment. Results were compared with information obtained from patients' clinical examinations before and a few years after the original treatment as well as information from the clinical examination of 35 control subjects. During the first years after treatment, the main signs of temporomandibular joint osteoarthrosis and internal derangement decreased significantly. In the next three decades, very few changes were noticed. Clicking and crepitus were the most common remaining signs. These signs, however, did not appear to be bothersome to the patients. It was concluded that the disorders in question eventually reach a state of quiescence.
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Osteoarthrosis (OA) and internal derangement (ID) are common disorders afflicting the temporomandibular joint (TMJ). The clinical course of TMJ ID is generally described in several consecutive stages.[1–5] The initial stage is characterized by reciprocal clicking, which is defined as a click that occurs on opening or protrusion followed by a click on closing.[6] This clicking is widely assumed to be related to anterior disc displacement. The disc snaps into and out of position on every open-close-clench cycle, which causes the opening and closing click, respectively. As this situation continues, the disc becomes more deformed and may not snap back into place on every opening movement. This stage of intermittent locking may progress to a permanent disc displacement. A permanently displaced disc restricts the range of condylar translation and is frequently accompanied by pain. Once the disc is permanently displaced, radiographically detectable osteoarthrotic changes usually become manifest. As this situation becomes more chronic, the range of opening will increase, because the posterior and lateral attachments of the disc are gradually stretched.[6] This stretching, along with thinning of the disc attachments, may result in perforation or rupture of the disc or its attachments. This leads to erosion of the articular cartilage of the condyle and the temporal bone and usually manifests clinically as crepitus.[7,8] Adaptive changes of the retrodiscal tissue to the joint's functional demands, resulting in pseudodisc formation, have also been described.[7,9] According to this supposed course, the signs and symptoms of the disorder tend to subside eventually, and the disorder may reach a stage of quiescence.

Since the authors' department of the university preserved the records of a considerable number of patients who suffered from

OA and ID 30 years ago, a retrospective investigation as to whether this stage of quiescence is actually reached could be performed. The purpose of this study was to evaluate whether clinical signs of TMJ OA and ID change in the long run and, if so, to what extent.

Material and Methods

Patients

For this study, 99 patients (16 men and 83 women; mean age 58.2 years) who had received nonsurgical treatment for OA and ID of the TMJ 30 years ago and 35 control subjects (8 men and 27 women; mean age 58.9 years) were examined. The selection procedures for both patients and control subjects have been described previously.[10,11] The control subjects, who matched the patients with regard to age, sex, and dental status, were free from present and previous TMJ symptoms.

Information from the 30-year-old records revealed that at that time, 15 out of the 99 patients were diagnosed with bilateral derangements. Thirty-nine patients had a reducing disc displacement (RDD), six of which were bilateral. Sixty patients had a permanent disc displacement (PDD), three of which were bilateral. Six patients with RDD in one joint and PDD in the other joint were included in the latter group. Altogether, 51 joints were diagnosed with RDD and 63 joints were diagnosed with PDD based on anamnestic, clinical, and radiographic findings.

Clinical Data

For each patient, the following clinical variables were extracted from the 30-year-old data recorded before treatment (T1) and that recorded 2 to 4 years after (T2):

1. Presence of clicking, crepitus, and pain on jaw movement; deviation from the midline on protrusion
2. Mouth opening measured in mm as maximal interincisal distance, maximal lateral excursions, and maximal protrusion

The same variables were measured at the clinical examination of the recall visit (T3). At this clinical examination the following variables were also recorded:

1. Presence of joint or muscle tenderness during palpation as indicated by the patient
2. Joint mobility measured by digital palpation of the lateral aspect of the joint during maximal protrusive movement followed by maximal opening from the maximally protruded position

At T3, the variables were recorded in conformity with the operational definitions described by Stegenga et al.[12] Mouth opening was considered restricted when the interincisal distance was less than 35 mm as suggested by the American Association of Oral and Maxillofacial Surgeons.[13] The same rule was applied to the 30-year-old records. Because mouth opening was not measured in mm in all cases at that time, this variable was also considered restricted either when it was the main complaint of the patient or when it was otherwise specifically mentioned. Additionally, at T3, digital palpation was used to assess joint mobility. According to this method, joint mobility was considered restricted when the condylar head could not be clearly palpated during maximal protrusion followed by maximal mouth opening.

Data of the clinical examination obtained at T3 were compared with data obtained from the 30-year-old records to determine whether and to what extent clinical signs of OA and ID had changed. To value the number of persisting signs in patients, the clinical data of the patients obtained at T3 were also compared with those of the control group.

Statistical Analysis

Student's *t* test was used to test differences in mandibular movement ranges (over time and between groups) and mobility scores (between groups). The other data were analyzed using nonparametric tests. The Mann-Whitney U test was used to test differences between groups, and the Wilcoxon test was used to test changes in time within a group. The level of significance was preset on $\alpha = .05$. All analyses were performed with SPSS/PC+™.

Results

Comparison Over Time

The mean time between measuring moments T1 and T2 was 3.1 ± 1.7 years and between T2 and T3 it was 28.7 ± 1.1 years. Fifteen patients, with 19 affected joints, had not been examined clinically at T2. For evaluation over time, data for 33 patients of the RDD group and 51 patients of the PDD group were available. Of these patients, 41 joints were diagnosed with RDD and 54 with PDD.

Table 1 Percentage of Joints With Signs of ID Before (T1), 2 to 4 Years After (T2), and 30 Years After Nonsurgical Treatment (T3)

	Measuring moment					
	RDD (N = 41)			PDD (N = 54)		
Signs	T1	T2	T3	T1	T2	T3
Clicking	92.7	58.5*	43.9	13.0	9.6	18.5
Pain on movement	43.9	17.1*	2.4*	87.0	13.2*	3.7
Restriction of movement	19.5**	4.9	4.9	87.0	24.5*	9.3*
Crepitus	2.4	24.4*	31.7	13.0	42.3*	46.3

*$P < .05$ T1 vs T2, T2 vs T3.
**In 7.5% most likely because of PDD in the opposite joint.

Table 2 Mean Maximal Movement Ranges and Standard Deviations (mm) Measured Before (T1), 2 to 4 Years After (T2), and 30 Years After Nonsurgical Treatment (T3)

	Measuring moment					
	RDD (N = 33)			PDD (N = 51)		
	T1	T2	T3	T1	T2	T3
Mouth opening	44.5±6.9	42.1±6.3	44.6±6.6	32.7±7.7*	39.3±7.4**	42.1 ± 6.3**
Protrusion	7.3±2.3	7.0±1.8	7.9±1.8	6.7±2.3	6.6±1.8	7.4 ± 2.9
Lateral excursion to affected side	9.4±2.9	8.9±1.9	8.6±2.7	8.1±2.0	8.1±2.7	7.8 ± 2.8
Lateral excursion to contralateral side	8.9±2.3	8.3±2.1	8.0±3.1	6.5±2.4*†	7.0±2.5*†	7.2 ± 2.8
Mouth opening 35 mm or more (%)	92.0	97.0	93.9	42.9	74.0*	92.2*

*$P < .05$ RDD vs PDD.
**$P < .05$ T1 vs T2, T2 vs T3.
†$P < .05$ Lateral excursion: affected side vs contralateral side.

Between T1 and T2, the number of joints with clicking and pain had decreased significantly in the RDD group ($P < .05$). The number of joints with pain and restriction of movement had decreased significantly in the PDD group ($P < .05$). The number of joints with crepitus had increased in both groups ($P < .05$).

At T2, clicking had ceased in 17 of 41 joints of the RDD group (41%). Between joints of this group in which the clicking had ceased and those in which it had not, no significant differences were found with regard to crepitus, pain, or deviation on protrusion. No association was found between cessation of clicking, decrease of mouth opening, and increase of crepitus.

Between T2 and T3, the number of joints with pain had decreased significantly in the RDD group ($P < .05$). The number of joints with restriction of movement had decreased significantly in the PDD group ($P < .05$) (Table 1).

At T3, clicking was found in 23 of 51 joints of the RDD group (45%). Clicking was accompanied by crepitus in six of these joints. Again, no significant differences were found with regard to crepitus, pain or deviation on protrusion between joints in which the clicking had ceased and those in which it had not. No association was found between cessation of clicking, decrease of mouth opening, and increase of crepitus.

Mean maximal mouth opening did not change significantly in the RDD group between the measuring moments ($P > .05$). In the PDD group mean maximal mouth opening and the number of patients who could attain a mouth opening of 35 mm or more increased significantly from T1 through T2 to T3 ($P < .05$) (Table 2). In none of the groups did the mean lateral and protrusive movement ranges gain or lose more than 1 mm between the measuring moments. In the PDD group, the mean movement range to the contralat-

Table 3 Clinical Findings (%) in Joints With ID and in Joints of a Control Group at T3

	RDD (N = 51)	PDD (N = 63)	Control (N = 70)
Free of joint noises	33.3*	44.4*	77.1
Clicking	33.3*,**	11.1	14.3
Crepitus	21.6*	34.9*	8.6
Both clicking and crepitus	11.8	9.5	0
Deviation on protrusion	25.0	30.2*	11.4
Restricted joint mobility	8.0*	12.7*	0
Joint tender to palpation	23.5*	23.8*	8.6
Masticatory muscles tender to palpation	21.6*,**	6.3	4.3

*$P < .05$ RDD, PDD vs Control.
**$P < .05$ RDD vs PDD.

Table 4 Mean Maximal Vertical and Horizontal Movement Ranges and Standard Deviations (mm) in Patients and Controls at T3

	RDD (N = 39)	PDD (N = 60)	Control (N = 35)
Opening	44.5 ± 6.5*	42.1 ± 6.8*	47.9 ± 7.2
Protrusion	7.8 ± 2.0	7.3 ± 2.9	8.0 ± 2.7
Lateral excursion to affected side	8.5 ± 2.8	8.0 ± 2.8	9.1 ± 3.6
Lateral excursion to unaffected side	8.0 ± 3.0	7.6 ± 3.1	8.6 ± 3.9

*$P < .05$ RDD, PDD vs Control.

eral side was significantly lower than that to the affected side at T1 and T2 ($P < .05$). This difference was neutralized at T3.

Comparison With Controls

At T3, clicking was found more often in joints of the RDD group than in joints of the PDD and control group ($P < .05$). Crepitus was found more often in joints of the RDD and PDD group than in joints of the control group ($P < .05$). Mandibular movement capacity was more often restricted in joints of the RDD and PDD group than in joints of the control group ($P < .05$). Joints of the RDD and PDD group were more often tender to palpation than were the joints of the control group ($P < .05$). In addition, masticatory muscles were more often tender to palpation in the RDD group than in the PDD and control group ($P < .05$) (Table 3). The mean maximal mouth opening was significantly larger in controls than in patients ($P < .05$). With regard to horizontal movement ranges, no differences between patients and controls were found ($P < .05$) (Table 4).

Discussion

This study showed that a significant decrease in signs of ID and OA can be obtained with nonsurgical treatment. In the present material, the disorder had reached a stage of quiescence or consolidation 2 to 4 years after treatment. In the next three decades, only minor, but nevertheless noteworthy, changes occurred. At the recall visit, joint mobility assessed by palpation revealed normal to hypermobile joints in more than 90% of the patients. The prevalence of patients with pain on movement had declined to less than 5%. After a significant decrease during and shortly after treatment, the number of joints with joint noises remained at approximately the same level. Not all signs of the disorder had disappeared, but the persisting signs appeared milder and less bothersome to the patients. This was expressed by the fact that hardly anyone had asked for treatment because of recurrent or persisting signs in the intervening years.[11] Improvement of masticatory function, reduction of pain, and feeling secure about the nature of the disorder apparently are more important factors of "recuperation" to the patient than treating the joint noises per se.

Before discussing the results of this clinical study in detail, some general methodologic difficulties inherent with the nature of the investigation must be addressed. A retrospective longitudinal study is subject to methodologic problems such as loss of patients or incomplete records. In the 30-year-old records, for instance, part of the data on movement ranges was not assessed quantitatively. However, in most of these cases qualitative information was available. Furthermore, because there were no reasons or indications for systematic drop-out of the quantitative data, the available data were considered representative for the total group.

The clinical examinations at the time of treatment and 2 to 4 years after treatment were performed by one author, and the examination at the recall visit was performed by another author. Although it is known that longitudinal studies usually benefit by examination by the same observer at all occasions,[14,15] this could not be accomplished in the present study. However, considering the

number of years between both occasions, the benefit of examination by the same observer can be doubted. In all likelihood, the observer will have systematically adapted his methods of examination during these years according to increasing knowledge about temporomandibular disorders as well as changes in opinions and methods in general.[14]

In the present study, the classification of the disorder was based on anamnestic, clinical, and radiographic data. However, an accurate diagnosis of ID requires imaging studies, such as arthrography or magnetic resonance imaging. In a recent publication, the material of this study has been related with ID in a comparative study performed by Nickerson and Boering.[3] This study made it likely that the majority of the patients under study, who were described by Boering[1] first, indeed had ID. Paesani[16] reported that 78% of the consecutive TMJ patients studied had different stages of ID. In an MRI study, Stegenga[12] stated that reproducible reciprocal clicking and restricted movement, among other factors, were highly indicative for different stages of ID. In the present study, records were strictly scored according to predefined criteria for which total agreement between the observers was required.[10,12,17] Therefore, the authors are convinced that this procedure must have resulted in a fairly accurate grouping of patients suffering from ID.

Unfortunately, the material of this study is not suitable to evaluate the natural clinical course of ID and OA in detail. However, looking back at what happened in the past 30 years with this material, the authors were able to see how their results corresponded with the different stages of the suggested natural course of OA and ID. The nonsurgical treatment, which the patients had received 30 years ago, mainly consisted of reassurance, hinge movement exercises, and superficial heat.[1,11] None of these treatment modalities were primarily directed toward correction of disc position. In fact, the hinge movement exercises were even directed toward the opposite way, that is, toward the natural course of the disorder. Therefore, this material might provide a valuable impression of the natural course of the disorder.

Analyzing the data, evidence was found to confirm the suggested clinical course. In the PDD group 2 to 4 years after nonsurgical treatment, the number of joints with restriction of movement had decreased, whereas the number of joints with crepitus had increased. In the RDD group, clicking had ceased in 41% of the joints, while crepitus developed in 23%. However, restriction of movement was hardly found in this group at that time. No significant association could be demonstrated between cessation of clicking, restriction of movement, and increase of crepitus.

In the PDD group, mouth opening had increased in the majority of patients 2 to 4 years after treatment. During the same time, the mobility of joints in the RDD group, in which the clicking had ceased, may have increased again to an acceptable level due to stretching of the disc attachments. This might explain why restriction of movement was not related with cessation of clicking. Cessation of clicking was achieved with help of hinge movement exercises in many patients. In this way, the disc attachments might have been stretched slowly and gently, thus allowing the retrodiscal tissue to adapt to the altered joint mechanics. This gradual adaptation may prevent mechanical irritation, which could in turn result in less inflammation and abrasive wear. In other words, as long as the joint loads do not exceed functional limits and the adaptive capacity is not compromised, tissue damage will be limited.[18] This might explain why crepitus, commonly associated with abrasive wear and perforated disc attachments, was not related with cessation of clicking in these patients.

In 59% of the joints of the RDD group, clicking was still present 2 to 4 years after treatment. Approximately three decades later, clicking was still present in 45% of this group. This finding indicates that a reducing disc displacement may persist for several decades without progression. Clicking was found in the presence of crepitus in 7.5% of the patients. In these cases, clicking was probably caused by some other process than a reducing disc. It has been suggested that adhesions, reduced lubrication, surface irregularities, or structural deformities may be responsible for clicking.[17,19] From this point of view, the presence of clicking and crepitus in one joint is not contradictory.

Although the overall signs of OA and ID had reduced significantly 30 years after treatment, the patients who had suffered from OA and ID more often exhibited joint noises and interferences during mandibular movements than did control subjects of the same age. In addition, affected joints were still more often tender to palpation than contralateral or control joints. Even though more than 90% of the patients could attain a mouth opening of 35 mm or more, the mean maximal mouth opening of the patients was smaller than that of control subjects, as was the joint mobility.

Despite the differences with the control group, the impression was gained that the impact of the persisting signs on normal functioning was minimal. As described in an earlier report concerning

subjective aspects of the same population, masticatory function was not impaired when compared with the control group.[10] Furthermore, the number of joint noises reported by the patients in the subjective study was significantly less than the number of joint noises found during the clinical examination, although both examinations were performed at the same event. It has been stated that self-reporting of TMJ sounds is not a reliable indicator for the clinical absence or presence of joint noises.[20] Furthermore, it is believed that people who report sounds form a psychologically specific subgroup within the group of people who produce sounds.[21] This knowledge might help to explain why joint noises were found in some of the control subjects, while they all had confirmed to be free from TMJ symptoms including joint noises. It may also explain why a disparity between subjective and objective findings concerning joint noises was found in these patients. Alternatively, the impact of persisting joint noises may be of too little importance for the patient to mention or notice them. During the long course of the disorder, these lasting interferences even may have become part of the patient's life and, consequently, are no longer recognized as disturbing. Lately, it has been suggested that the elderly report fewer signs or symptoms than are found during clinical examinations because they are thought to accept a gradual impairment of bodily functions over time, including the masticatory system.[22,23]

Conclusion

Broadly speaking, the TMJ disorder based on OA and ID evaluated in this study follows the expected course, although the development of this course per person does not seem to be predictable. If progressive, the disorder will reach a stage of quiescence within 2 to 4 years. After that, only minor changes are seen. If clicking does not respond to treatment and is still present 2 to 4 years afterward, it is likely to persist for several decades. It appears that in these cases, reassurance and relief of pain are of more importance than treating joint noises per se. The integrity and mechanics of the TMJ, in particular when the disc becomes permanently displaced, are disturbed. Yet, the TMJ seems to adapt adequately to this altered situation in most cases. In only a small part of the patients does the affected TMJ remain a vulnerable, although not bothersome, part of the masticatory system. Therefore, the use of nonsurgical treatment modalities that are directed toward the natural course of the disorder should be encouraged. However, when the disorder does not respond to various nonsurgical therapies, the time has come for surgical intervention.

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Resumen

Signos clínicos de osteoartrosis y malfuncionamiento interno en la articulación temporomandibular después de 20 años de tratamiento no quirúrgico.

Se reevaluaron 30 años después del tratamiento original a 99 pacientes que habían recibido tratamiento no quirúrgico para resolver problemas temporomandibulares. Tal examen fue realizado para evaluar el curso clínico de la osteoartrosis y el malfuncionamiento interno de la articulación temporomandibular (ATM). Los resultados fueron comparados con la información obtenida de los exámenes clínicos de los pacientes antes y unos pocos años después del tratamiento original, y la información del examen clínico de 35 pacientes de control. Durante los primeros años después del tratamiento, los principales signos de osteoartrosis y malfuncionamiento interno de la ATM disminuyeron significativamente. Se notaron muy pocos cambios en las tres décadas siguientes. Los signos remanentes más comunes fueron los sonidos de clic y la crepitación. Estos signos, sin embargo, no parecieron incomodar a los pacientes. Se concluyó que los desórdenes discutidos, eventualmente alcanzan un estado de calma.

Zusammenfassung

Klinische Zeichen von Arthrose und "internal derangement" des Kiefergelenkes 30 Jahre nach nicht-chirurgischer Therapie.

Um den klinischen Verlauf von Arthrose und "internal derangement" des Kiefergelenkes zu verfolgen, wurden 99 Patienten, die eine nicht-chirurgische Therapie erhalten hatten, 30 Jahre nach Behandlung erneut untersucht. Die Resultate wurden verglichen mit den klinischen Befunden vor und wenige Jahre nach Therapiebeginn, schliesslich auch mit denjenigen von 35 Kontrollpatienten. Während den ersten Jahren nach der Therapie nahmen die Hauptsymptome der Arthrose und des "internal derangement" deutlich ab. In den nächsten drei Jahrzehnten wurden nur wenige Veränderungen beobachtet. Knacken und Reiben waren die häufigsten verbliebenen Symptome, schienen die Patienten aber in der Regel nicht zu stören. Es wurde daraus geschlossen, dass die fraglichen Störungen oft einen Zustand der Ruhe erlangen.