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Neuromuscular evaluation of post-orthodontic stability: An experimental protocol

To prevent relapse after orthodontic treatment, retention is often considered indispensable. Soft tissues are thought to have a significant influence on dental movements. To quantify the influence of masticatory muscles on post-treatment relapse, and in an attempt to avoid unnecessary procedures, 2 male orthodontic patients (13 and 30 years old at debonding) were followed up. The patients completed 2 years of fixed orthodontic treatment and received no post-orthodontic retention. After 1 week and again after 6 months, alginate impressions of dental arches and a surface electromyographic (EMG) assessment of the masseter and temporalis muscles during maximum voluntary clenching were performed. The younger patient received surface EMG monitoring once a month for the first 6 months and at the 1-year follow-up appointment. Arch dimensions and the 3-dimensional inclination of the facial axis of the clinical crown (FACC) were measured using a computerized digitizer. Symmetry in muscular contraction was measured by the percentage overlapping coefficient (POC), and potential lateral displacing components were assessed by the torque coefficient (TC). At the 6-month follow-up, no clinical modifications were observed. Quantitative evaluation assessed that arch dimensions had changed slightly (up to 1 mm). While the adolescent patient had no modifications in FACC inclinations, the 30-year-old patient showed significant alterations (up to 18 degrees). In all examinations of the adolescent patient, POC was higher than 86% and TC was lower than 10%. In the adult, POC was inside the normal range, while all TCs were higher than 10.5%. The larger TC measured in the adult may explain the larger modifications in the 3-dimensional position of his dental crowns. In conclusion, a surface EMG assessment may help in the detection of patients who might need post-orthodontic retention. (Int J Adult Orthod Orthognath Surg 2002;17:307–313)

Post-orthodontic stability is still a matter of concern. While a certain amount of post-treatment modifications are considered to be within the normal changes expected with development or aging, some alterations are correctly classified as relapse.[1,2] Indeed, it is assumed that no orthodontic treatment can guarantee absolute stability of the dental arrangement obtained at debonding.[2]

To prevent (or limit) this phenomenon, retention is often considered indispensable, and sometimes lifelong procedures are performed. Unfortunately, no sound scientific bases seem to support the several kinds of retention protocols, and in only a limited number of patients do post-treatment modifications seem predictable.[2–5]

Soft tissues are thought to have a significant influence on dental movements, and the tongue, the lips, and the perioral muscles, as well as the periodontium, have all been claimed to have some bearing on post-treatment modifications.[3] However, no investigations have analyzed in detail the effect of masticatory muscles on post-orthodontic relapse, although significant alterations in the electrical activity of the masseter muscle during treatment have been reported.[6,7]

Orthodontic treatment may provoke an altered equilibrium in oral and dental structures, and relapse may be a "physiologic" attempt to return to an acceptable neuromuscular equilibrium.[1] Also, prolonged retention may provoke some kind of hard and soft tissue damage.[8]

In an attempt to provide a quantitative basis for the influence of masticatory muscles on post-treatment relapse, and to avoid unnecessary procedures, the authors followed up 2 male patients with morphologic analyses of their dental arches and functional evaluations of their masticatory muscles.

Materials and methods

Patients

Two male patients were analyzed immediately after completion of fixed orthodontic treatment.

The first patient (B.D.) had been treated for a skeletal and dental (molar and canine) Class II malocclusion with deep bite, overjet of 10 mm, scissor bite between the maxillary right first premolar and the mandibular right second premolar, and malposition and crowding of the mandibular anterior teeth. Orthodontic treatment consisted of a bimaxillary fixed appliance with 0.17×0.25-inch rectangular stainless steel wire, extraoral traction, and Class II malocclusion elastic threads. Extraoral traction was used for 18 months. At 12 months after the onset of treatment, the Class II malocclusion elastic threads were added; these were maintained for 12 months (until debonding). At debonding, the patient was 13 years old and had a full permanent dentition, including the second molars, with bilateral Angle Class II molar and canine relationships.

The second patient (F.M.) had Angle Class I molar and canine relationships on his right hemi-arch and Angle Class II molar and Angle Class I canine relationships on his left hemi-arch. He had crowding of his anterior maxillary teeth; crossbite between maxillary right lateral incisor/mandibular right lateral incisor, mandibular right canine, and maxillary left first molar/mandibular left first molar; asymmetry of the mandibular arch with a left-positioned midline; and rotation of the mandibular left second premolar and first molar. Before orthodontic treatment, the maxillary left first premolar had been extracted. The 2-year orthodontic treatment included 0.16-inch nickel-titanium (Ni-Ti) and 0.17×0.25-inch stainless steel archwire, an elastomeric chain on the maxillary left canine to retract it, springs between the maxillary right central incisor and canine and between the mandibular left first and second premolars, and a bondable lingual button on the mandibular left second premolar. At debonding, the patient was 30 years old and had a full permanent dentition, including the second molars, except for the maxillary left first premolar.

Both patients received no post-orthodontic retention treatment. They both were examined at 1 week and again at 6 months after debonding. On both occasions, alginate impressions of their dental arches were taken and a surface electromyographic (EMG) examination of their masticatory muscles was performed. Additionally, the younger patient (B.D.) received surface EMG examinations once a month for the first 6 months as well as at the 1-year follow-up. He had attended a dental practice for orthodontic control twice a month. Patient F.M. received a surface EMG examination also at the 1-month follow-up.

Dental arches

The dental casts of both subjects were obtained from alginate impressions cast in yellow stone.

Anteroposterior and left-right dental arch dimensions, as well as the 3-dimensional (3-D) inclination of the facial axis of the clinical crown (FACC) and the size of the clinical crowns, were measured starting from a set of standardized dental landmarks[9,10] (Figs 1 and 2). The digital coordinates of the landmarks were obtained by a single operator with a 3-D electromagnetic

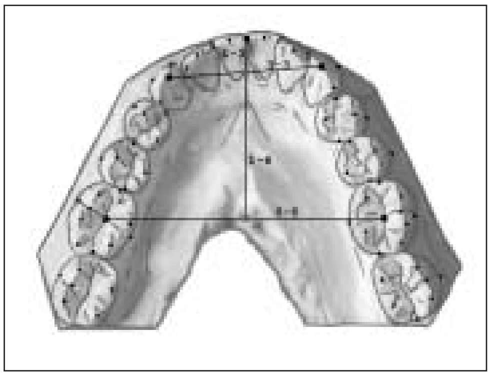

Fig 1 Mandibular cast with the digitized landmarks. The anteroposterior and transverse dental arch dimensions are also indicated. 3-3 = intercanine distance; 6-6 = intermolar distance; 1-3 = mid-incisor to mid-canine distance; 1-6 = mid-incisor to mid-molar distance.

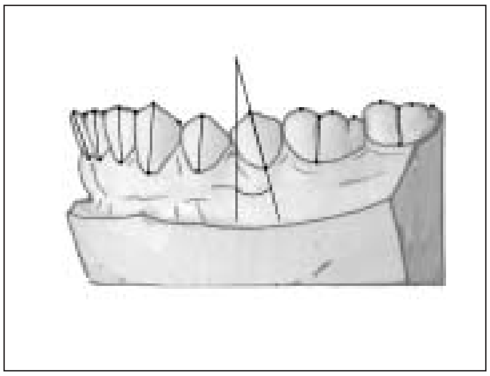

Fig 2 Mandibular cast, lateral view. The FACCs are indicated, as well as the inclination of the left second premolar on the sagittal plane.

digitizer (3Draw; Polhemus, Colchester, VT) interfaced with a computer. A detailed description of the measurement protocol can be found in Ferrario et al.[9,10]

The files of the 3-D coordinates were obtained, and computer programs devised and written by one of the authors were used for all subsequent calculations.

In particular, the intercanine, intermolar, midincisor to midcanine, and midincisor to midmolar distances, as well as the height of clinical crowns, were measured in millimeters, while the inclination of the FACCs in the frontal and sagittal planes for each tooth were computed in degrees.[9,10] For all measurements, the same reference planes were used and mathematically set horizontally. The planes (maxillary and mandibular) were computed between the incisive papilla and the intersections of the palatal or lingual sulci of the first permanent molars with the gingival margin.[10]

The method error has been reported before, with mean random errors for FACC inclination between 2.3 and 2.5 degrees.[10] Crown height had a mean random error of 0.19 mm,[10] while the dental arch dimensions had coefficients of variation up to 1.23%.[9]

EMG recordings and measurements

The masseter and anterior temporalis muscles of both sides (left and right) were examined as detailed by Ferrario et al.[11–13] During testing, disposable silver/silver chloride bipolar electrodes with a diameter of 10 mm and an interelectrode distance of 21 ± 1 mm (Duo-Trode; Myo-Tronics, Seattle, WA) were used, while a disposable reference electrode was applied to the forehead.

EMG activity was recorded using 4 of the 8 channels of a computerized instrument (De Götzen; Legnano, Milano, Italy). The analogic EMG signal was amplified (gain 150, bandwidth 0 to 10 Khz, peak-to-peak input range from 0 to 2,000 μV) using a differential amplifier with a high common mode rejection ratio (CMRR = 105 dB in the range 0 to 60 Hz, input impedance 10 GΩ), digitized (12 b resolution, 2,230 Hz A/D sampling frequency), and digitally filtered (high-pass set at 30 Hz, low-pass set at 400 Hz, band-stop for common 50- to 60-Hz noise).

The signals were averaged over 25 ms, with muscle activity of the 4 tested muscles assessed as the root-mean-square (rms) of the amplitude (unit: μV). EMG signals were then recorded for further analysis.[11–13]

A first recording for the standardization of EMG potentials was made as detailed by Ferrario et al.[13] In brief, two 10-mm-thick cotton rolls were positioned on the mandibular second premolars and molars of each subject, and a 3-second maximum voluntary clench (MVC) was recorded. For each muscle, the mean EMG potential was set at 100%, and all further EMG potentials were expressed as a percentage of this value (unit: μV/μV × 100).

EMG activity was then recorded during a maximum voluntary clench test in intercuspal position lasting 5 seconds. The subject

Table 1 Six-month changes in arch size (in mm)

Distance	Patient B.D. (13 y) Maxilla	Patient B.D. (13 y) Mandible	Patient F.M. (30 y) Maxilla	Patient F.M. (30 y) Mandible
Intercanine	–0.36	–1.13	0.03	0.18
Intermolar	–0.23	–0.70	–0.11	0.57
Mid-incisor to mid-canine	–0.09	0.94	0.12	0.17
Mid-incisor to mid-molar	–0.28	0.43	–0.05	0.33

A positive value indicates an increment in the distance between debonding and the 6-month follow-up.

was invited to clench as hard as possible and to maintain the same level of contraction for the entire test. During recording, the subjects sat with their head unsupported and were asked to maintain a natural erect position.[13] Reproducibility of surface EMG measurements of the same muscles has already been tested in our laboratory and was found to be good.[13]

For each subject, the central 3 seconds of the maximum voluntary clench test were then analyzed, and the EMG potentials were standardized as detailed before. Subsequently, the EMG waves of paired muscles were compared by computing the percentage overlapping coefficient (POC; unit: %),[12,13] an index of symmetric muscular contraction. The index ranges between 0% and 100%, ie, when 2 paired muscles contract with perfect symmetry, a POC of 100% is obtained. A masseter as well as a temporalis POC was obtained for each subject and for each assessment.

Because an unbalanced contractile activity of contralateral masseter and temporalis muscles, such as that of right temporalis and left masseter, might give rise to a potential lateral displacing component, the torque coefficient (TC; unit: %)[12,13] was assessed. TC ranges between 0% (complete absence of lateral displacing force) to 100% (complete presence of lateral displacing force). TC is 0% when both the differences between the left and right temporalis and between the left and right masseter are zero and POC = 100% (complete symmetry of paired masseter and temporalis waves).

Results

Modifications in the anteroposterior and left-right distances computed in the dental arches of the 2 patients between debonding and the 6-month follow-up are reported in Table 1. Both patients had larger modifications in their mandibular arch. In particular, the mandibular intercanine and intermolar distances of patient B.D. decreased by more than 0.5 mm, while the mandibular mid-incisor to mid-canine distance increased by nearly 1 mm. The mandibular intermolar distance of patient F.M. increased by more than 0.5 mm. In both patients, no clinically detectable crowding was observed.

Tables 2 and 3 list the 6-month modifications in FACC inclinations computed in the 2 patients. In the younger patient, the modifications were limited, and only 17 inclinations of 56 had modifications larger than 2.5 degrees (the method error). Indeed, even in these teeth the changes in FACC inclinations in the frontal and sagittal planes were minor, and only 4 inclinations changed more than 6 degrees (up to 8.8 degrees). In the older patient, 38 inclinations (of 54 altogether) changed by more than 2.5 degrees. In this patient, the FACC extensively changed their frontal and sagittal plane inclinations; 13 of these modifications were larger than 10 degrees (up to 18 degrees), indicating some degree of relapse.

Table 4 reports the EMG indices computed in both patients at their follow-up examinations. On all occasions, patient B.D. had POC indices higher than 86% and TC indices lower than 10%. In patient F.M., the POC indices ranged between 83.9% and 86.8%, while the TC indices were all around 11%.

Table 2 Six-month changes in FACC inclinations (in deg) computed in patient B.D. (13 years old at debonding)

	Right side		Left side	
Tooth	Frontal	Sagittal	Frontal	Sagittal
Maxilla				
Central incisor	−2.4	−2.9	+4.5	+3.2
Lateral incisor	−1.1	+0.2	−0.6	+5.6
Canine	−1.8	+0.9	+0.7	+0.9
First premolar	−1.7	−1.6	−1.6	+2.2
Second premolar	+0.6	−2.1	+6.5	+5.1
First molar	+7.1	+2.5	−1.0	+0.2
Second molar	+0.7	−0.1	−0.4	−0.9
Mandible				
Central incisor	+2.7	+3.6	−5.6	−1.5
Lateral incisor	+4.2	+3.6	−1.9	−5.3
Canine	−1.5	−2.2	−0.0	+1.9
First premolar	+5.4	−0.7	−0.5	−0.7
Second premolar	−2.4	−2.4	+1.6	−2.2
First molar	+4.4	+2.2	+8.0	+0.8
Second molar	+8.8	+5.1	−1.4	−3.7

A positive value indicates an increment in FACC inclination between debonding and the 6-month follow-up.

Table 3 Six-month changes in FACC inclinations (in deg) computed in patient F.M. (30 years old at debonding)

	Right side		Left side	
Tooth	Frontal	Sagittal	Frontal	Sagittal
Maxilla				
Central incisor	+2.4	−3.3	−2.1	−0.8
Lateral incisor	−0.5	−0.3	−11.3	−0.3
Canine	−3.6	−3.7	−6.4	−3.6
First premolar	−9.9	−12.5	—	—
Second premolar	−5.8	−8.1	−7.2	−1.1
First molar	−10.4	−17.7	−8.2	−5.6
Second molar	−8.3	−18.1	+2.0	−8.9
Mandible				
Central incisor	−0.4	+2.1	+1.5	−3.1
Lateral incisor	−5.3	+2.5	−3.7	−4.5
Canine	−6.8	−0.9	−11.0	−8.6
First premolar	−2.5	−3.7	−10.4	−6.7
Second premolar	−3.5	−2.6	−14.7	−15.3
First molar	−7.9	−15.6	+1.7	−5.3
Second molar	−10.5	−14.3	+12.1	−7.3

A positive value indicates an increment in FACC inclination between debonding and the 6-month follow-up.

Table 4 EMG indices computed in patients B.D. and F.M.

	Patient B.D. (13 y)			Patient F.M. (30 y)		
Time	POC T	POC M	TC	POC T	POC M	TC
Debonding	88.56	89.64	7.30	86.79	83.88	11.91
1 month	88.50	89.97	7.52	85.96	84.63	11.78
2 months	88.44	86.94	7.77	—	—	—
3 months	88.26	86.94	7.22	—	—	—
4 months	87.34	88.31	8.78	—	—	—
5 months	89.53	85.58	7.63	—	—	—
6 months	88.36	86.64	8.55	86.30	85.92	10.93
1 year	89.00	86.92	9.63	—	—	—

All values are percentages.
POC = percentage overlapping coefficient for the temporalis (T) and masseter (M) muscles; TC = torque index.

Discussion

An age-related reduction in intercanine width and arch length and an increase in dental crowding have been found in the permanent dentitions of both normal subjects and post-treatment orthodontic patients.[14–17] Masticatory forces, as well as periodontal components, producing buccal and mesial dental drifts, have been cited to explain these modifications.[18,19] Post-orthodontic relapse combines with these physiologic phenomena, and clear differentiation between the 2 processes cannot be made.[1,2] Nevertheless, a post-orthodontic occlusion might possibly be more unstable than a natural occlusion, and thus be subject to larger modifications. While the roles of periodontium and of perioral and tongue muscles seem sufficiently well defined, at least in their general components,[3,18,20] no investigation has analyzed in detail the effect of masticatory muscles.

The neuromuscular control of masticatory muscles is influenced by periodontal afferents: for instance, in healthy subjects a 250-μm-thick experimental occlusal interference provoked an altered pattern of contraction of masseter and temporal muscles.[12] Also, altered occlusal relationships, such as a crossbite, modify jaw muscle activity[11,21] and reduce muscular efficiency, which returns to the norm after orthodontic correction.[6,22] Moreover, during orthodontic treatment the activity of the masseter muscle changes.[6,7] Pain, discomfort, and changes in occlusal relationships all may influence muscular activity.[6,7]

Orthodontic treatment itself provokes alterations in dental positions, and the new periodontal afferents may negatively influence the neuromuscular equilibrium.[7] Further investigations of the relationship between masticatory muscle imbalances during clenching and orthodontic relapse may give a deeper insight into the phenomena controlling craniofacial modeling and remodeling. Moreover, they may provide the clinician with a simple and noninvasive test for the identification of patients with a (potentially) larger risk. Retention procedures may thus be reduced and overtreatment limited.

No definitive consensus on the duration of retention exists, and lifelong procedures are often performed.[1,2,5,8] Indeed, adult patients have a lower cellular and molecular turnover than child and adolescent patients, as well as a lower tooth mobility; thus, longer retention treatments have been proposed.[2,5,23]

In the present preliminary study, 2 orthodontic male patients were followed up after the successful completion of their treatment. All the procedures used in the follow-up examinations were noninvasive and low-cost. Specifically, surface EMG analysis was performed with minimal disturbance to the patients and provided immediate quantitative data. While in the adolescent patient (B.D., 13 years at debonding), all EMG indices calculated during maximum voluntary teeth clenching were well inside normal ranges, in the adult patient (F.M., 30 years at debonding) most indices were different from reference values collected in healthy individuals.[13] In particular, the POC index of the masseter muscle was lower than reference values, thus indicating a certain degree of muscular asymmetry during clenching. The torque coefficient was higher than reference values, showing some imbalance between the right- and left-side rotating muscular couples (Table 4).

This functional evaluation was combined with a quantitative analysis of dental arches. In particular, while no modifications in the dimensions of dental arches were found (Table 1), some alterations in the 3-D arrangement of the dental crowns were measured. In patient B.D., the largest modification in FACC inclination was measured in the frontal plane inclination of the mandibular right second molar (Table 2). Indeed, at debonding this tooth was not completely erupted, and during the subsequent 6 months it continued its eruption with a minor modification in its angulation.

In patient F.M., the mandibular left first and second premolars had large 6-month post-treatment modifications in the inclinations of their FACCs (Table 3): more than 10 degrees in the frontal plane and up to 15 degrees in the sagittal plane. Both teeth had been derotated during orthodontic treatment, and relapse in this occurrence is well known.[8] Before treatment, the maxillary right lateral incisor was in crossbite, and at the 6-month follow-up it showed the fewest modifications in the inclinations of its FACC.

These preliminary data seem to suggest that, after debonding, the patient with the larger modifications in FACC angulations also had the larger alterations in the functional coordination of his main masticatory muscles, at least during clenching. In contrast, the other patient had negligible FACC modifications and normal muscular activity. It must be emphasized that the 2 patients were of different ages and received different orthodontic treatment; both factors may influence post-orthodontic relapse.

Within the limitations of the present study, a surface EMG examination of the main masticatory muscles may provide the orthodontist with a fast, low-cost, and noninvasive quantitative test to predict postorthodontic stability.

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