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Original Article

Impact of orthodontic treatment on masticatory muscle electromyographic patterns in patients with anterior crossbite

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KEYWORDS
Crossbite;
Masticatory muscles;
Orthodontics;
Surface electromyography

Abstract *Background/purpose:* Anterior crossbite affects facial aesthetics and may cause temporomandibular joint disorders and masticatory muscle dysfunction. Prolonged abnormal bite positioning may impact muscle activity and affect treatment stability. Although orthodontic therapy aims to realign teeth and correct bite relationships, the neuromuscular adaptations following treatment remain unclear. This study aims to investigate the changes in masticatory muscle activity before and after orthodontic treatment in patients with anterior crossbite using surface electromyography (sEMG).
Materials and methods: 41 participants were recruited and divided into 3 groups: normal occlusion (n = 18), anterior crossbite without treatment (control, n = 6), and anterior crossbite with orthodontic treatment (experimental, n = 13). Muscle activity of the temporalis, masseter, and anterior digastric muscles was recorded across four stages. Measurements included maximum mouth opening, muscle endurance, and sEMG. Data were analyzed using IBM SPSS (IBM Corp., Armonk, NY, USA) with independent *t*-tests and repeated measures ANOVA.
Results: After orthodontic treatment, the experimental group showed improved mouth opening and biting force. While no significant differences in resting muscle potentials were found between treated patients and the normal group, statistically significant differences were observed between the experimental and control groups in the clenching activity of both left and right temporalis muscles ($P < 0.05$) throughout all stages of the study ($P < 0.05$).
Conclusion: Orthodontic treatment improved muscle function in anterior crossbite patients, but

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muscle activity does not fully normalize. This suggests that post-treatment muscle adaptation may require more time and highlights the importance of monitoring muscle function as part of comprehensive orthodontic care.

Introduction

Anterior crossbite not only affects aesthetics but can also lead to temporomandibular joint disorders and muscle discomfort, negatively impacting quality of life. Advancements in orthodontic treatment have led to improved occlusion and facial aesthetics, yet the role of muscles in treatment outcomes is often overlooked. Research by Kiliaridis et al. (2006) suggests that strong masticatory muscles correlate with specific facial morphologies and influence mandibular and facial growth.[1] As such, muscle function can affect treatment results.

Globally, anterior crossbite affects 5–6 % of the population,[2–5] with a higher prevalence of 13.83 % reported in Taiwan.[6] Common causes include skeletal discrepancies, trauma to deciduous teeth leading to lingual displacement of the permanent tooth bud, crowding in the anterior region, insufficient arch length, the presence of supernumerary teeth or odontoma, habits, and genetic factors.[7–9]

Surface electromyography has become an ideal method for assessing muscle activity due to its safety and ease of use. Previous studies have demonstrated variability in muscle activity during orthodontic treatment, and research findings on the impact of treatment on muscle function remain inconclusive.[10–14] Moreover, malocclusion has been identified as a contributing factor to altered muscle activity patterns, which may confound assessments of post-treatment muscular adaptation.[10–14]

This study focuses specifically on patients with anterior crossbite undergoing fixed orthodontic treatment and compare the changes in muscle activity before and after treatment. This research aims to provide a deeper understanding of how treatment influences muscle dynamics. Furthermore, the results of muscle activity assessments can be used to design more precise treatment plans for addressing muscle-related issues, ultimately helping maintain occlusal stability and enhancing long-term treatment outcomes.

Materials and methods

Study design and participants

This study was conducted in two phases at China Medical University and China Medical University Hospital, Taichung, Taiwan from **September 8, 2022, to July 31, 2024**. Ethical approval was obtained from the Research Ethics Committee (Protocol No. CMUH111-REC3-142), and informed consent was acquired from all participants.

Phase I: included 41 healthy adults who met the following inclusion criteria: age twenty years or older, fully erupted permanent dentition, and no observable history of facial trauma.

Phase II: included 13 adult patients who met the following criteria: aged twenty years or older, diagnosed with anterior crossbite, with fully erupted permanent dentition, no observable history of facial trauma, and receiving orthodontic treatment using ISW (LH) archwires (TOMY Inc., Fuchu-shi, Tokyo, Japan).

Participants were excluded if they met any of the following conditions: unwillingness to cooperate or presence of any systemic disease; current or past diagnosis of temporomandibular joint disorder (TMD) or masticatory muscle pain; history of orthognathic surgery; or undergoing a second round of orthodontic treatment (Fig. 1).

Orthodontic appliances

Patients in Phase II received standardized treatment with Roth Formula-R brackets (TOMY Inc., Fuchu-shi, Tokyo, Japan) and Improved Super-elastic Ti-Ni alloy wires (ISW, LH) (TOMY Inc.).

Electromyographic measurements

Surface electromyography (sEMG) signals were recorded using the JMAnalyser+ BT system (Zebris Medical GmbH, Isny im Allgäu, Germany) and WINJAW+ software (Zebris Medical GmbH, Isny im Allgäu, Germany). Electrodes were placed bilaterally on temporalis, masseter, and anterior digastric muscles based on anatomical landmarks. Given individual variations in craniofacial development, occlusal habits, and physiological structures, consistent electrode placement is critical to minimize experimental error. Therefore, the electrode positions were standardized and kept constant for each participant.[15] For the temporalis muscle, the sEMG electrodes were placed with one electrode positioned 1.0 cm superior to the zygomatic arch, and the second electrode placed 1.5 cm posterior to the anterior margin of the muscle, following the placement protocols used in previous studies.[16–20] Electrode placement for the masseter and anterior belly of the digastric muscles was relatively straightforward. For the masseter, electrodes were positioned between the angle of the mandible and the zygomatic arch. For the anterior digastric, one electrode was placed at the anterior border of the chin, and the second electrode was positioned approximately 1.0 cm posterior to the first along the muscle fiber direction. Participants were seated upright on a backless chair.

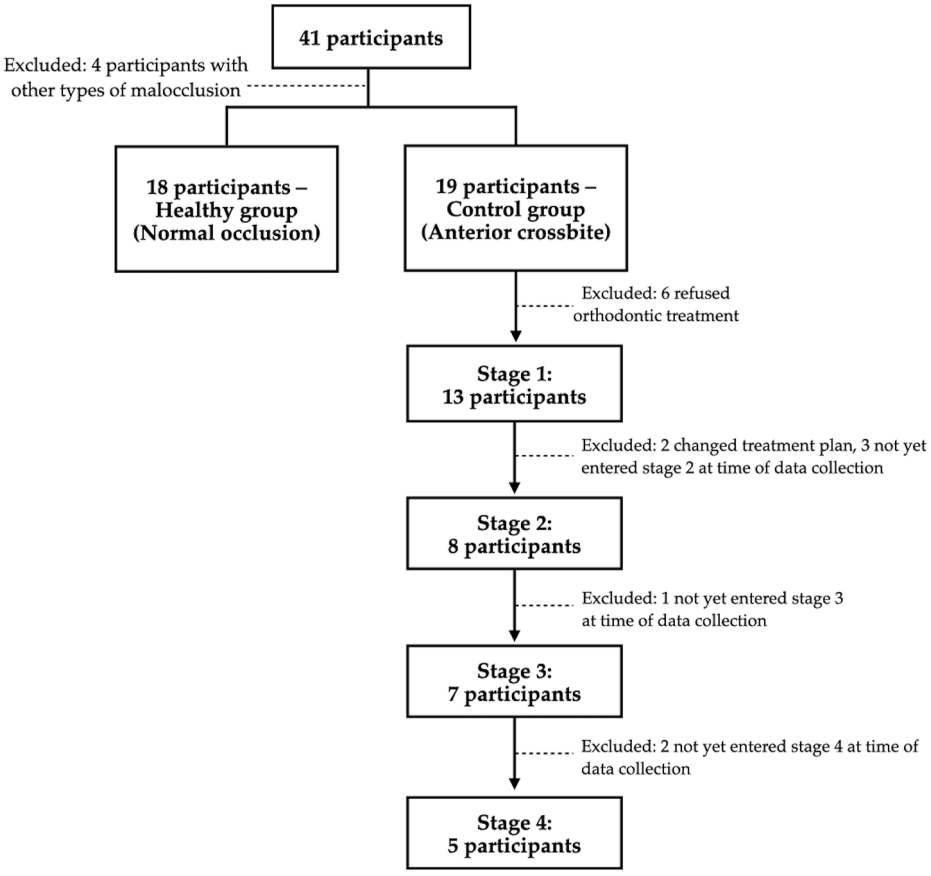

Figure 1 Flow diagram illustrates the inclusion and exclusion of participants in both the healthy and control groups, as well as the number of participants completing each treatment stage (stages 1—4).

Before recording, maximum mouth opening was measured using a caliper (Mitutoyo Measuring Instruments, Kawasaki, Japan), and maximum voluntary bite force was measured using a wireless microFET®2 digital handheld dynamometer (Hoggan Scientific LLC, Salt Lake City, UT, USA).

Testing protocol

Muscle activity was measured during seven tasks:

Group A: Resting
Group B: Clenching
Group C/D: Left deviation
Group D: Right deviation
Group E: Protrusion
Group F: Tapping at 130 BPM
Group G: Tapping at 88 BPM

Data were analyzed using MATLAB R2023a (MathWorks Inc., Natick, MA, USA).

Statistical analysis

Line plots were generated to illustrate trends in maximum mouth opening and bite force across different treatment stages. Statistical analyses were performed using SPSS (IBM Corp., Armonk, NY, USA). Independent samples *t*-tests were used to compare sEMG activity between participants with normal occlusion and those with anterior crossbite. For the anterior crossbite group, repeated-measures ANOVA was conducted to evaluate changes in sEMG activity across four treatment stages: pre-treatment, edge-to-edge correction, post-correction, and three months post-treatment. An independent *t*-test was also applied to compare post-treatment sEMG activity in anterior crossbite patients with that of the control group (anterior crossbite without treatment). A *P*-value of <0.05 was considered statistically significant.

Results

This study investigated the electromyographic (EMG) activity of six key masticatory muscles—bilateral temporalis, masseter, and anterior belly of the digastric—across four stages of orthodontic treatment in patients with anterior crossbite.

However, this study also aimed to evaluate its potential impact on the maximum mouth opening in patients with anterior crossbite. Based on the trendlines, patients with anterior crossbite exhibited a continuous increase in maximum mouth opening following orthodontic treatment, which may reflect the restoration of oral physiological

Table 1 Maximum mouth opening measurements (in mm) recorded at four stages of orthodontic treatment in anterior crossbite patients.

Maximum mouth opening, unit: mm				
	Stage 1	Stage 2	Stage 3	Stage 4
Patient 1	38.0	37.5	35.5	41.5
Patient 2	38.0	37.0	37.0	39.5
Patient 3	36.5	40.0	39.0	38.0
Patient 4	35.5	45.0	39.0	43.0
Patient 5	47.0	41.0	47.5	50.0

Table 2 Maximum biting force measurements (in N) recorded at four stages of orthodontic treatment in anterior crossbite patients.

Maximum bite force, unit: N				
	Stage 1	Stage 2	Stage 3	Stage 4
Patient 1	55.0	41.0	55.0	67.0
Patient 2	56.0	56.0	61.0	81.0
Patient 3	55.0	61.0	54.0	59.0
Patient 4	89.0	84.0	57.0	79.0
Patient 5	56.0	46.0	96.0	65.0

functions, including improvements in mastication and speech (Table 1and Fig. 2).

Additionally, we assessed the patients' maximum biting force to evaluate how orthodontic treatment affects masticatory muscle function, particularly the capacity to withstand pressure. According to the trend shown in the graphs, patients in the experimental group exhibited an overall increase in biting force during the treatment period, suggesting that orthodontic treatment may have a positive impact on oral muscular strength, thereby enhancing the patients' ability to cope with external functional demands (Table 2) (Fig. 3).

sEMG data were analyzed using independent sample *t*-tests to examine differences in muscle activity between subjects with normal occlusion and those with anterior crossbite during various mandibular tasks: relaxation, left lateral excursion, right lateral excursion, forward protrusion, clenching, and open-close tapping at 130 BPM and 88 BPM. Among these movements, significant differences were observed in the left temporalis muscle during tapping at both 130 BPM ($P = 0.017$) and 88 BPM ($P = 0.007$). Additionally, the left anterior digastric muscle showed a significant difference during 130 BPM tapping ($P < 0.001$). These differences may be related to distinct adaptation patterns of the left temporalis muscle in anterior crossbite patients, individual variability, and habitual chewing side preference, which together could account for the disparities observed between anterior crossbite and normal occlusion groups during tapping (Table 3).

Repeated-measures ANOVA was used to evaluate mean sEMG activity across four treatment stages: pre-treatment, edge-to-edge correction, post-correction, and three months post-treatment. Although differences were noted in mean and standard deviation values across these stages, no statistically significant changes were found in muscle activity (all $P > 0.05$). This suggests that, for these specific movements and muscle activities, orthodontic treatment did not produce significant effects in anterior crossbite patients, or that the muscles may require a longer adaptation period to exhibit measurable changes (Table 4).

An independent *t*-test was also applied to compare post-treatment sEMG activity in anterior crossbite patients with that of the control group (anterior crossbite without treatment). The results indicated that the experimental group achieved muscle activity levels comparable to those of the control group, with no significant differences. In different mandibular movement directions, significant differences were found in specific muscle groups, underscoring the influence of mandibular movement on related muscles. The masseter muscle showed no significant changes before and after orthodontic treatment during left, right, and forward deviations, suggesting limited treatment effects. In contrast, the temporalis and anterior belly of the digastric muscle displayed significant changes in certain directions, indicating a greater treatment impact on these muscles (Table 5).

Discussion

As biting positions were corrected, patients exhibited a progressive increase in maximum mouth opening during orthodontic treatment (Fig. 2). This improvement suggests enhanced mandibular mobility, which may positively influence functions such as mastication and speech. The

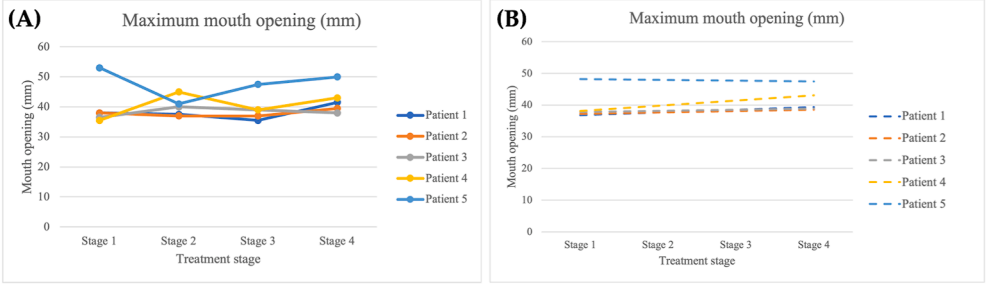

Figure 2 Maximum mouth opening (mm) at different treatment stages in patients with anterior crossbite. (A) raw data showing individual maximum mouth opening at each treatment stage. (B) corresponding trendlines demonstrating an overall increasing trend in maximum mouth opening during orthodontic treatment.

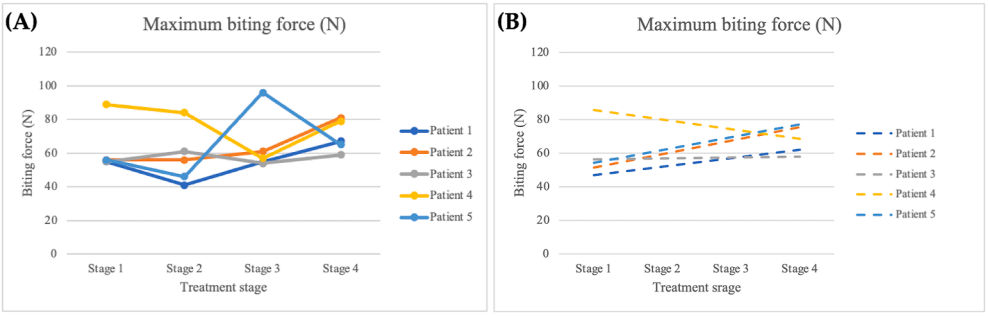

Figure 3 Maximum biting force (N) at different treatment stages in patients with anterior crossbite. (A) raw data showing individual bite force measurements at each treatment stage. (B) corresponding trendlines demonstrating an overall increase in bite force during orthodontic treatment.

Table 3 Surface electromyography (sEMG) activity (mean ± SD) in patients with normal occlusion (N) and anterior crossbite (C + E) during various mandibular tasks.

Task	Muscle	N (n = 18)	C + E (n = 19)	*P* value
Resting	TR	9.11 ± 4.88	8.36 ± 3.68	0.601
	TL	8.52 ± 4.29	6.23 ± 3.56	0.052
	MR	5.46 ± 2.27	6.34 ± 2.71	0.292
	ML	5.09 ± 1.60	6.41 ± 2.60	0.076
	DR	5.54 ± 2.35	6.47 ± 4.18	0.415
	DL	7.09 ± 2.85	7.82 ± 7.62	0.698
Clenching	TR	79.56 ± 50.75	79.2 ± 43.64	0.984
	TL	75.84 ± 48.49	53.60 ± 44.12	0.153
	MR	93.82 ± 58.81	84.91 ± 63.46	0.661
	ML	108.15 ± 70.02	80.00 ± 47.08	0.166
	DR	18.45 ± 9.24	14.38 ± 8.47	0.171
	DL	16.16 ± 10.16	10.92 ± 8.26	0.102
Left deviation	TR	10.60 ± 7.02	13.18 ± 6.34	0.248
	TL	12.50 ± 5.31	12.38 ± 14.72	0.975
	MR	10.72 ± 8.28	17.93 ± 15.89	0.095
	ML	19.92 ± 14.33	15.12 ± 11.14	0.270
	DR	17.02 ± 15.71	23.20 ± 17.91	0.273
	DL	25.50 ± 26.50	22.52 ± 37.10	0.783
Right deviation	TR	15.52 ± 11.88	15.98 ± 12.06	0.907
	TL	9.92 ± 4.43	8.95 ± 4.75	0.525
	MR	18.74 ± 16.76	19.17 ± 12.45	0.931
	ML	12.77 ± 7.79	19.35 ± 14.95	0.105
	DR	23.04 ± 14.64	23.43 ± 12.33	0.933
	DL	19.42 ± 10.05	18.64 ± 21.30	0.890
Protrusion	TR	12.38 ± 7.60	9.16 ± 3.57	0.115
	TL	11.93 ± 8.18	7.62 ± 4.43	0.053
	MR	17.89 ± 13.85	19.94 ± 16.21	0.682
	ML	19.47 ± 14.82	21.78 ± 18.15	0.679
	DR	17.58 ± 8.30	21.30 ± 13.10	0.323
	DL	20.29 ± 16.39	12.11 ± 8.97	0.066
Tapping 130	TR	25.63 ± 19.30	18.93 ± 9.33	0.194
	TL	23.69 ± 16.49	12.61 ± 8.30	0.017
	MR	21.22 ± 19.17	15.92 ± 11.36	0.310
	ML	25.05 ± 24.20	17.03 ± 10.97	0.211
	DR	15.11 ± 14.64	11.69 ± 6.50	0.361
	DL	15.43 ± 7.88	12.58 ± 8.61	<0.001
Tapping 88	TR	22.87 ± 13.42	18.71 ± 9.34	0.279
	TL	21.06 ± 10.53	12.08 ± 8.46	0.007
	MR	15.76 ± 8.30	16.15 ± 13.63	0.917
	ML	18.20 ± 13.02	16.68 ± 13.70	0.731
	DR	10.97 ± 5.27	12.58 ± 8.61	0.500

Table 3 (*continued*)

Task	Muscle	N (n = 18)	C + E (n = 19)	*P* value
	DL	14.09 ± 8.76	9.24 ± 10.71	0.142

Abbreviations: C + E= Control group + experimental group; DL = Left anterior digastric; DR= Right anterior digastric; ML = Left masseter; MR= Right masseter; N= Normal group; TL = Left temporalis; TR= Right temporalis.

Table 4 Surface electromyography (sEMG) activity (mean ± SD) in the experimental group across four treatment stages: pre-treatment (stage 1), edge-to-edge correction (stage 2), post-correction (stage 3), and three months post-treatment (stage 4).

Task	Muscle	E (n = 13)				
		Stage 1 (n = 13)	Stage 2 (n = 8)	Stage 3 (n = 7)	Stage 4 (n = 5)	*P* value
Resting	TR	8.60 ± 3.73	11.36 ± 2.16	10.52 ± 2.70	9.95 ± 6.77	1.000
	TL	5.81 ± 3.81	7.27 ± 6.46	4.33 ± 2.57	5.37 ± 5.09	0.734
	MR	6.40 ± 3.00	6.79 ± 2.05	6.18 ± 2.58	6.01 ± 2.21	0.631
	ML	14.38 ± 6.42	6.54 ± 1.58	5.02 ± 2.19	6.50 ± 4.89	0.346
	DR	6.46 ± 4.11	6.46 ± 1.69	9.04 ± 6.49	9.19 ± 5.82	0.180
	DL	7.47 ± 8.24	4.96 ± 2.66	3.92 ± 1.97	4.02 ± 1.42	0.154
Clenching	TR	73.63 ± 50.85	31.11 ± 16.35	56.11 ± 31.27	59.27 ± 34.22	0.396
	TL	39.00 ± 44.68	12.68 ± 18.10	20.27 ± 12.97	45.59 ± 51.71	0.281
	MR	55.18 ± 40.89	14.85 ± 10.15	40.69 ± 32.77	46.96 ± 46.85	0.446
	ML	63.86 ± 42.97	13.97 ± 11.91	34.73 ± 30.13	29.82 ± 29.71	0.151
	DR	14.45 ± 10.03	12.38 ± 5.85	15.29 ± 7.68	20.26 ± 11.23	0.195
	DL	30.28 ± 77.79	5.67 ± 4.78	7.81 ± 3.36	20.35 ± 32.14	0.455
Left deviation	TR	11.31 ± 4.51	12.28 ± 8.26	14.91 ± 13.73	15.08 ± 9.90	0.737
	TL	6.91 ± 5.11	5.85 ± 4.19	11.82 ± 5.51	16.42 ± 20.85	0.456
	MR	15.39 ± 9.21	11.87 ± 8.00	14.32 ± 8.81	12.60 ± 8.20	0.908
	ML	11.51 ± 5.96	24.31 ± 20.43	20.80 ± 24.62	11.61 ± 5.54	0.381
	DR	17.31 ± 8.51	17.09 ± 10.02	15.18 ± 4.42	15.77 ± 4.21	0.583
	DL	7.38 ± 8.48	13.74 ± 20.53	8.88 ± 7.19	17.37 ± 30.99	0.711
Right deviation	TR	11.31 ± 4.51	12.28 ± 8.26	14.91 ± 13.73	15.08 ± 9.90	0.737
	TL	6.91 ± 5.11	5.85 ± 4.19	11.82 ± 5.51	16.42 ± 20.85	0.456
	MR	15.39 ± 9.21	11.87 ± 8.00	14.32 ± 8.81	12.60 ± 8.20	0.908
	ML	11.51 ± 5.96	24.31 ± 20.43	20.80 ± 24.62	11.61 ± 5.54	0.381
	DR	17.31 ± 8.51	17.09 ± 10.02	15.18 ± 4.42	15.77 ± 4.21	0.583
	DL	7.38 ± 8.48	13.74 ± 20.53	8.88 ± 7.19	17.37 ± 30.99	0.711
Protrusion	TR	8.76 ± 3.25	11.28 ± 4.37	8.17 ± 2.78	15.83 ± 10.48	0.182
	TL	6.30 ± 3.89	5.65 ± 3.55	7.54 ± 5.28	5.50 ± 5.17	0.558
	MR	16.70 ± 9.22	14.26 ± 8.15	14.42 ± 6.31	13.00 ± 5.28	0.811
	ML	17.55 ± 11.26	16.96 ± 12.03	19.39 ± 10.55	11.38 ± 4.41	0.840
	DR	21.73 ± 14.17	18.58 ± 12.24	16.67 ± 9.25	21.71 ± 10.25	0.497
	DL	7.52 ± 4.65	9.02 ± 9.22	8.93 ± 8.80	3.85 ± 0.61	0.765
Tapping 130	TR	19.76 ± 10.19	19.50 ± 14.93	18.31 ± 10.60	16.48 ± 7.82	0.736
	TL	10.26 ± 7.83	6.07 ± 5.53	9.62 ± 7.90	12.20 ± 11.80	0.410
	MR	16.29 ± 12.51	10.02 ± 4.76	13.63 ± 8.24	14.75 ± 10.16	0.606
	ML	17.35 ± 11.51	10.56 ± 5.65	14.08 ± 8.72	10.11 ± 5.49	0.365
	DR	14.04 ± 6.57	15.40 ± 7.67	14.75 ± 8.85	13.61 ± 7.06	0.222
	DL	4.79 ± 1.95	5.13 ± 4.47	5.47 ± 1.61	10.90 ± 11.73	0.317
Tapping 88	TR	19.80 ± 10.23	17.57 ± 9.21	18.60 ± 11.99	16.98 ± 8.36	0.867
	TL	9.98 ± 8.12	5.83 ± 5.74	9.16 ± 7.65	14.03 ± 14.68	0.419
	MR	12.07 ± 6.10	9.56 ± 4.16	14.40 ± 11.46	16.94 ± 14.07	0.610
	ML	17.43 ± 15.19	9.65 ± 5.04	13.65 ± 11.66	11.02 ± 6.63	0.686
	DR	13.19 ± 8.06	13.45 ± 7.03	13.07 ± 9.25	14.62 ± 5.38	0.937
	DL	5.65 ± 3.94	6.18 ± 5.05	6.20 ± 2.49	14.08 ± 19.90	0.365

Abbreviations: DL = Left anterior digastric; DR= Right anterior digastric; E: Experimental group; ML = Left masseter; MR= Right masseter; TL = Left temporalis; TR= Right temporalis.

Table 5 Surface electromyography (sEMG) activity comparison between treated anterior crossbite patients (experimental group, stage 4) and the normal occlusion group during six functional tasks.

Task	Muscle	N (n = 18)	E Stage 4 (n = 5)	*P* value
Resting	TR	9.11 ± 4.88	9.95 ± 6.77	0.755
	TL	8.52 ± 4.29	5.37 ± 5.09	0.190
	MR	5.46 ± 2.27	6.01 ± 2.21	0.633
	ML	5.09 ± 1.60	6.50 ± 4.89	0.559
	DR	5.54 ± 2.35	9.19 ± 5.82	0.040
	DL	7.09 ± 2.85	4.02 ± 1.42	0.032
Clenching	TR	79.56 ± 50.75	59.27 ± 34.22	0.413
	TL	75.84 ± 48.49	45.59 ± 51.71	0.237
	MR	93.82 ± 58.81	46.96 ± 46.85	0.117
	ML	108.15 ± 70.02	29.82 ± 29.71	0.025
	DR	18.45 ± 9.24	20.26 ± 11.23	0.715
	DL	16.16 ± 10.16	20.35 ± 32.14	0.787
Left deviation	TR	10.60 ± 7.02	15.08 ± 9.90	0.260
	TL	12.50 ± 5.31	16.42 ± 20.85	0.697
	MR	10.72 ± 8.28	12.60 ± 8.20	0.659
	ML	19.92 ± 14.33	11.61 ± 5.54	0.063
	DR	17.02 ± 15.71	15.77 ± 4.21	0.859
	DL	25.50 ± 26.50	17.37 ± 30.99	0.564
Right deviation	TR	15.52 ± 11.88	15.39 ± 9.64	0.982
	TL	9.92 ± 4.43	10.94 ± 12.63	0.867
	MR	18.74 ± 16.76	17.44 ± 6.42	0.868
	ML	12.77 ± 7.79	11.46 ± 4.18	0.725
	DR	23.04 ± 14.64	22.33 ± 11.84	0.922
	DL	19.42 ± 10.05	17.40 ± 30.51	0.891
Protrusion	TR	12.38 ± 7.60	15.83 ± 10.48	0.417
	TL	11.93 ± 8.18	5.50 ± 5.17	0.152
	MR	17.89 ± 13.85	13.00 ± 5.28	0.241
	ML	19.47 ± 14.82	11.38 ± 4.41	0.248
	DR	17.58 ± 8.30	21.71 ± 10.25	0.363
	DL	20.29 ± 16.39	3.85 ± 0.61	0.063
Tapping 130	TR	25.63 ± 19.30	16.48 ± 7.82	0.129
	TL	23.69 ± 16.49	12.20 ± 11.80	0.162
	MR	21.22 ± 19.17	14.75 ± 10.16	0.480
	ML	25.05 ± 24.20	10.11 ± 5.49	0.026
	DR	15.11 ± 14.64	13.61 ± 7.06	0.829
	DL	15.43 ± 7.88	10.90 ± 11.73	0.317
Tapping 88	TR	22.87 ± 13.42	16.98 ± 8.36	0.366
	TL	21.06 ± 10.53	14.03 ± 14.68	0.237
	MR	15.76 ± 8.30	16.94 ± 14.07	0.812
	ML	18.20 ± 13.02	11.02 ± 6.63	0.115
	DR	10.97 ± 5.27	14.62 ± 5.38	0.188
	DL	14.09 ± 8.76	14.08 ± 19.90	0.999

Abbreviations: DL = Left anterior digastric; DR= Right anterior digastric; E = Experimental group stage 4; ML = Left masseter; MR= Right masseter; N= Normal group; TL = Left temporalis; TR= Right temporalis.

observed trend highlights the potential of orthodontic therapy not only in correcting occlusion but also in restoring physiological oral functions. Maximum mouth opening may serve as a dynamic indicator of treatment efficacy and contribute to overall quality of life improvement.

Similarly, the gradual increase in biting force suggests adaptive changes in masticatory muscles during treatment (Fig. 3). Improved occlusion likely enhances muscle efficiency, supporting better oral performance in daily activities. The upward trend in bite force implies that orthodontic treatment may strengthen oral muscles and improve resistance to external loads, offering functional as well as structural benefits.

An interesting finding of this study was the significant difference in left temporalis muscle activity between the anterior crossbite and normal groups during open-close tapping at both 130 BPM and 88 BPM ($P = 0.017$ and 0.007, respectively). This suggests that under specific rhythmic conditions, the left temporalis in anterior crossbite patients may exhibit altered neuromuscular coordination.

These results are in line with findings by Wieczorek et al. (2015), who reported that asymmetry in temporalis muscle activity may be linked to mandibular deviation and uneven occlusal contacts.[11] Notably, all other muscles and tasks in this study showed no significant group differences ($P > 0.05$), indicating that overall masticatory function remains comparable. However, the distinct response of the left temporalis under tempo constraints highlights a potentially sensitive marker of functional adaptation in anterior crossbite cases.

Contrary to our initial hypothesis that orthodontic correction would result in significant changes in muscle activity over time, statistical analysis revealed no significant differences ($P > 0.05$) across the four treatment stages. These findings suggest that orthodontic treatment alone may not have an immediate or substantial impact on masticatory muscle activity in specific functional movements. These results align with previous studies. Kaya et al. (2013) reported no significant changes in masticatory muscle activity following orthodontic treatment.[13] Similarly, Wieczorek et al. (2015) found no significant differences in EMG readings of the temporalis and masseter muscles between treated and untreated individuals.[11]

One possible explanation for the limited EMG variation is the minimal skeletal change observed in our subjects. Cephalometric analysis of participants who completed all four stages of data collection revealed modest changes in mandibular position: the mandibular plane angle changed by only 0.7°–2.1°, ramus inclination by 0°–1.7°, and lower anterior facial height (ANS-Me) by 0–8.1 mm (Fig. 4). These relatively small skeletal shifts suggest that most of the orthodontic changes occurred at the dental rather than skeletal level, which may not have been sufficient to trigger significant neuromuscular adaptation.

Although overall sEMG results showed no significant differences between anterior crossbite patients and individuals with normal occlusion, we expect that muscle activity in the crossbite group would gradually normalize following orthodontic treatment. For example, the left anterior digastric at rest ($P = 0.032$), right anterior digastric at rest ($P = 0.040$), left masseter during clenching ($P = 0.025$), and left masseter during tapping at 130 BPM ($P = 0.026$) all showed significant deviations compared to the control group.

Several factors may explain the limited overall differences. Orthodontic treatment often involves minor mandibular repositioning, primarily through tooth movement, which may not substantially impact muscle activity. The exclusion of surgical cases may also have resulted in a sample with milder malocclusions and smaller treatment effects. Previous findings by Grünheid et al. (2009) indicate that more pronounced changes in muscle activity tend to occur following orthognathic surgery rather than with orthodontic treatment alone.[21]

Additionally, the relatively short treatment duration—often progressing through phases in less than three months—may not have allowed sufficient time for full neuromuscular adaptation. Muscle response is known to depend on the type, intensity, and duration of functional stimulation, and individual variability may further influence adaptive patterns. These factors likely contributed to the limited observable changes in sEMG results post-treatment.

This study faced several limitations. Electrode placement was challenging in participants with smaller facial structures, potentially leading to signal interference from adjacent muscles. The absence of data on chewing preference and handedness limited interpretation of muscle asymmetry findings. Moreover, the short treatment

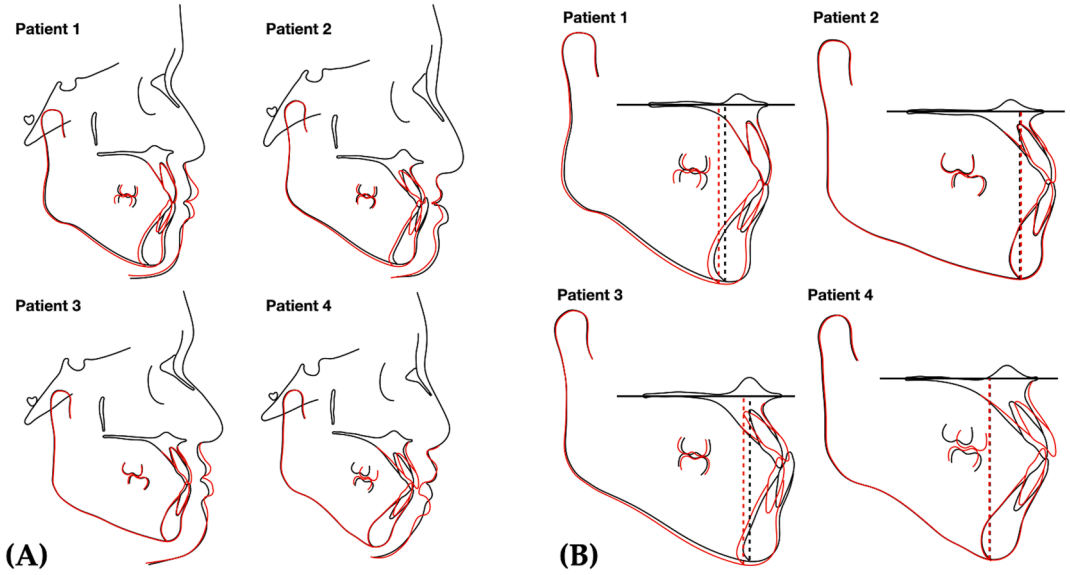

Figure 4 (A) Superimposed lateral cephalometric profiles of four representative participants (patient 1–4) before (black lines) and after (red lines) orthodontic treatment. (B) corresponding skeletal tracings aligned to the anterior cranial base showing minimal changes in mandibular plane angle, ramus inclination (0°–1.7°), and lower anterior facial height (ANS-Me).

duration may not have allowed sufficient time for full neuromuscular adaptation. Our focus on static tasks restricted analysis of functional, dynamic jaw movements. Lastly, the small sample size, with only five participants completing all stages, reduces the generalizability of the results. Future studies should include larger samples, longer follow-up, and more dynamic testing to better understand masticatory muscle adaptation following orthodontic treatment.

Orthodontic treatment in anterior crossbite patients led to increased mouth opening and muscle strength, with limited changes in EMG activity during static tasks. Significant differences in temporalis and masseter activity were observed compared to controls, especially during clenching. These findings suggest functional improvement following treatment and highlight the importance of neuromuscular evaluation during orthodontic care.

Declaration of competing interest

The authors have no conflicts of interest relevant to this article.

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References

1. Kiliaridis S. The importance of masticatory muscle function in dentofacial growth. *Semin Orthod* 2006;12:110—9.
2. Parthasarathy P, Kumar A, Dharman S. Prevalence and gender distribution of dental crossbite and its association with malocclusion: an institution based study. *Ann Med Health Sci Res* 2021;11:447—52.
3. Sá De Lira AL, Alves da Fonseca GH. Anterior crossbite malocclusion: prevalence and treatment with a fixed inclined plane orthodontic appliance. *Braz J Oral Sci* 2019;18:1—8.
4. Cenzato N, Nobili A, Maspero C. Prevalence of dental malocclusions in different geographical areas: scoping review. *Dent J (Basel)* 2021;9:1—10.
5. De Ridder L, Aleksieva A, Willems G, Declerck D, Cadenas de Llano-Pérula M. Prevalence of orthodontic malocclusions in healthy children and adolescents: a systematic review. *Int J Environ Res Publ Health* 2022;19:1—31.
6. Mok CW, Wong RW. Self correction of anterior crossbite: a case report. *Cases J* 2009;2:6967.
7. Bayraka S, Tunc ES. Treatment of anterior dental crossbite using bonded resin-composite slopes: case reports. *Eur J Dent* 2008;2:303—6.
8. Beard GB. Anterior crossbite: interceptive orthodontics for prevention, Maryland bridges for correction. *J Vet Dent* 1989;6:14—7.
9. Almarhoumi A, Alwafi MM. Early interceptive correction for anterior crossbite using a removable appliance: a pediatric case study. *Cureus* 2024;3:e56072.
10. Rodríguez Castañeda CI, Cruz Hervert LP, Llamosas Hernández E, et al. Changes in electromyographical activity during different phases of orthodontic treatment: pilot study results. *Rev Mex Ortod* 2017;5:e233—9.
11. Wieczorek A, Loster JE. Activity of the masticatory muscles and occlusal contacts in young adults with and without orthodontic treatment. *BMC Oral Health* 2015;15:116.
12. Nishi SE, Basri R, Alam AK, et al. Evaluation of masticatory muscles function in different malocclusion cases using surface electromyography. *J Hard Tissue Biol* 2017;26:23—8.
13. Kaya D, Kocadereli İ. Electromyographic activities of masticatory muscles in Class III patients treated by facemask with miniplate anchorage. *Turk J Orthod* 2013;26:65—71.
14. Kubota T, Yagi T, Tomonari H, Ikemori T, Miyawaki S. Influence of surgical orthodontic treatment on masticatory function in skeletal Class III patients. *J Oral Rehabil* 2015;42:733—41.
15. Piancino MG, Isola G, Merlo A, Dalessandri D, Debernardi CL, Bracco P. Chewing pattern and muscular activation in open bite patients. *J Electromyogr Kinesiol* 2012;22:273—9.
16. Tecco S, Tetè S, D'Attilio A, Festa F. Relation between facial morphology on lateral skull radiographs and sEMG activity of head, neck, and trunk muscles in Caucasian adult females. *J Electromyogr Kinesiol* 2011;21:298—310.
17. Takeuchi-Sato T, Arima T, Mew M, Svensson P. Relationships between craniofacial morphology and masticatory muscle activity during isometric contraction at different interocclusal distances. *Arch Oral Biol* 2019;98:52—60.
18. Lindauer SJ, Gay T, Rendell J. Effect of jaw opening on masticatory muscle EMG-force characteristics. *J Dent Res* 1993;72:51—5.
19. Sandhu SS, Utreja A, Prabhakar S, Sandhu N, Kashyap R. A study of electromyographic activity of masseter and temporalis muscles and maximum bite force in patients with various malocclusions. *J Indian Orthod Soc* 2013;47:53—61.
20. Cha BK, Kim CH, Baek SH. Skeletal sagittal and vertical facial types and electromyographic activity of the masticatory muscle. *Angle Orthod* 2007;77:463—70.
21. Grünheid T, Langenbach GEJ, Korfage JAM, Zentner A, van Eijden TMGJ. The adaptive response of jaw muscles to varying functional demands. *Eur J Orthod* 2009;31:596—612.