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RESEARCH ARTICLE

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Uniformity of palatal volume and surface area in various malocclusions

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Abstract

Aims: To assess palatal volume, surface area and linear dimensions across sagittal and vertical components of malocclusion.

Methods: Pre-treatment cephalographs and dental casts of 178 non-growing patients (88 males, 90 females, age 25.81 ± 8.23 years) were used for classification in sagittal malocclusion groups: Class I (n = 48), Class II division 1 (n = 42), Class II division 2 (n = 40) and Class III (n = 48); and in vertical divergence groups based on the MP/SN angle: hypodivergent (n = 35), normodivergent (n = 95) and hyperdivergent (n = 48). Dental casts were scanned and palatal measurements recorded: palatal width, depth and interdental distances; palatal surface area (PSA, mm^2) and volume (PV, mm^3). A 3-way ANOVA was used to compare palatal dimensions across groups. Pearson product-moment correlations were employed to assess associations among variables.

Results: PSA and PV (in both sex groups) were not statistically significantly different across sagittal malocclusions and vertical patterns. Significant differences were depicted more among the vertical divergence groups than sagittal groups. Correlations between PSA and PV and palatal dimensions were low to moderate. Predictability of PSA and PV ranged between 15 and 18%.

Conclusion: The finding of similar palatal volume and surface area, on average, across malocclusions possibly underscores the limitations of environmental influences within the inherited orofacial phenotype. The vertical facial pattern seems to be more interactive with palatal dimensions than sagittal relations. Studies of palatal changes following orthodontic and orthognathic treatments are warranted.

KEYWORDS
palatal surface, palatal volume, three-dimensional

1 | INTRODUCTION

The complex and multifactorial aetiology of malocclusion reflects the interplay between predominantly genetic factors and environmental influences.[1,2] The latter include localized effects in response to sustained impactful insults such as mouth breathing, sucking habits and abnormal swallowing and tongue activity.[3] The more commanding 'genetically determined' functions in the craniofacial complex (e.g. brain growth, respiration and muscle activity) are thought to direct the growth of the craniofacial skeleton.[4]

The literature on the environmental influence is extensive and may be summarized in key findings. The dimensions of the palatal vault have been closely associated with oral functions and habits such as mouth breathing and thumbsucking.[5] A narrow maxillary

arch has been related to airway obstruction, as well as lower tongue position, itself a likely consequence of the obstruction to facilitate breathing that leads to imbalance of the perioral musculature and arch narrowing.[6,7] Alterations in mandibular growth direction have also been reported.[6,7] The Class II division 1 malocclusion particularly has been associated with a narrower palate[8–14] and anterior open bite.[15,16] The constricted palate may conceal a posterior crossbite because the distal position of a retrognathic mandible places a narrower part of the mandibular arch opposite the maxillary teeth.

More variations are found in research on the palatal depth (or height), with greater measures observed in subjects with Class II division 1,[9] open bite,[15] and even Class I malocclusions.[17] However, the greater depth was also reported as merely an impression resulting from narrower arches.[16] In addition, conflicting conclusions have been formulated on the association between palatal depth and width. The findings of moderate-to-strong linear associations between these measurements are countered with disagreement on the direction of the association and the specific measures involved in different malocclusions.[8,17] Low correlations have been reported between palatal depth and other palatal dimensions.[8,15,18] Also, contrasting some results of similar dimensions in men and women,[19,20] most researchers found wider and deeper palates in men than in women across various populations.[21–24] Disparities among studies may be related to sampling methods, population differences and variable two-dimensional measurements from dental casts, three-dimensional digital images of dental casts or oral scans.[25,26]

Considering the shape variation of the maxillary arch and palate among malocclusions, particularly the U-shaped and shallower arch in Class II, division 2, and the elongated and narrower arch in Class II, division 1,[27] we hypothesized that if these characteristics have been moulded by repeated environmental influences during years of growth, palatal volumes and surface areas might be less variable. Studies of palatal volume in specific malocclusions are limited. Smaller palatal volumes were illustrated in children with posterior crossbites compared to those with normal occlusions.[25,28] Pre-pubertal children with Class III malocclusions have been shown to have palatal volumes similar to those with normal occlusion.[29] We then considered that the hypothesis should be tested in subjects with diminished or no growth, whereby long-standing environmental effects would have set irreversibly. Accordingly, the specific aims were to assess palatal volume, surface area and linear palatal dimensions across the various malocclusion types and components, namely sagittal and vertical, also exploring the effects of sex on palatal vault morphology.

2 | MATERIAL AND METHODS

This retrospective study was approved by the Institutional Review Board of the American University of Beirut (ID#OTO.RH.03).

The minimum sample size required to achieve 80% power to detect a between-subjects difference for a three-way ANOVA with an effect size of 0.28 (palatal surface area: mean 1730.42, SD 130.11 mm^2) and level of significance of 0.05 was 144. This effect size was calculated from a pilot study on 15 patients run prior to the present one. G*Power 3.1.9.3 was used to calculate sample size.

The sample consisted of the pre-treatment orthodontic records of 178 non-growing patients (88 males, 90 females, mean age 25.81, SD 8.23 years) recruited from the orthodontic division at the American University of Beirut over a period of 3 months. Inclusion criteria were as follows: females older than 16 years (mean 25.87, SD 8.31 years) and males older than 18 years (mean 25.74, SD 7.19 years) with a cervical vertebral maturation index of 6 (the most mature stage), determined according to Baccetti et al[30]; dentition with a complete set of fully erupted permanent teeth (excluding third molars); high-quality lateral cephalogram and dental casts; and absence of posterior crossbite. Excluded were patients with congenital anomalies, history of orthodontic treatment and surgical treatment involving the head and neck.

Patients were classified in the sagittal malocclusion groups on the basis of the following criteria that minimize overlap between the malocclusions:

- Class I ($n = 48$): bilateral Class I molar occlusion within one-half cusp, an overjet of 2–3 mm and ANB ranging within ±1 standard deviation (SD) around the average 2 ± 2°. The range was effectively between 0 and 3.5°.
- Class II division 1 (II/1; $n = 42$): maxillary molars occluding in Class II by one-half cusp or more, overjet ≥4.5 mm and ANB ≥5° (beyond +1 SD);
- Class II division 2 (II/2; $n = 40$): Class II molar relationship (one-half cusp or more), overbite ≥80% and overjet of 2–3 mm. The combination of molar distoclusion with severe overbite and reduced overjet has been characteristic of the definition of this malocclusion. A cephalometric measurement was not included because of the wider range of the ANB angle in this malocclusion spanning the domains of Class I and Class II.[31]
- Class III ($n = 48$): mesioclusion by one-half cusp or more, overjet ≤0 mm and ANB <0° (below - 1 SD).

Based on the angle MP/SN (MP being GoGn plane), patients were stratified into 3 groups of vertical divergence within and beyond 1SD around the mean angle: hypodivergent (Hypo; n = 35): MP/SN <27°; normodivergent (Normo; $n = 95$): 27° ≤ MP/SN ≤37°; and hyperdivergent (Hyper; $n = 48$): MP/SN >37°.

Maxillary and mandibular dental casts were de-identified by research support personnel not directly involved in the study. All remaining procedures were carried out by the first author (MS). Dental casts were scanned using the laser scanning system Perceptron (ScanWorks® V5,), and saved data files were processed using IMInspect software from PolyWorks® to generate the polygonal model derived from the point cloud for all anatomical structures present in the model, at point-to-point resolutions up to 12 µm. The same software was used to measure the linear dimensions, area and volume of the palate (Table 1).

TABLE 1 Definitions of palatal dimensional measurements performed on digital dental casts

Abbreviation	Measurement and definition
PW-C	Palatal width at the level of the canines: distance between the mid-points of the gingival margins of the right and left permanent canines
PW-PM	Palatal width at the level of the first premolars: distance between the mid-points of the gingival margins of the right and left first premolars
PW-M	Palatal width at the level of the first molars: distance between the mid-points of the gingival margins of the right and left permanent first molars
PD-C	Palatal depth at the level of the canines: perpendicular vertical distance between the gingival plane and an opposing point on the mid-palatal raphe, measured at the level of the mid-points of the gingival margins of the permanent maxillary canines
PD-PM	Palatal depth at the level of the first premolars: perpendicular vertical distance between the gingival plane and an opposing point on the mid-palatal raphe, measured at the level of the mid-points of the gingival margins of the maxillary first premolars
PD-M	Palatal depth at the level of the first molars: perpendicular vertical distance between the gingival plane and an opposing point on the mid-palatal raphe, measured at the level of the mid-points of the gingival margins of the maxillary first permanent molars
ICD	Intercanine distance: distance between the cusp tips of the right and left canines
IPMD	Interpremolar distance: distance between the palatal cusp tips of the right and left first premolars
IMD	Intermolar distance: distance between the mesio-palatal cusp tips of the right and left first molars
PL	Palatal length: perpendicular distance between the most posterior point on the incisive papilla and the line joining the mesial surfaces of the first molars
PV	Palatal volume: volume enclosed between the gingival plane and the distal plane
PSA	Palatal surface area: surface area of the portion of the palate enclosed between the gingival plane and the distal plane

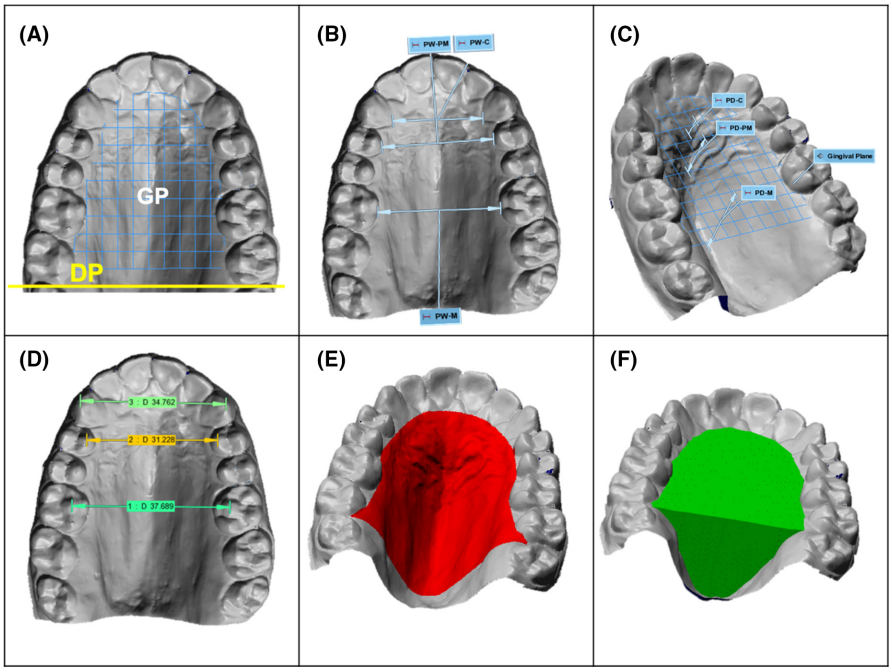

FIGURE 1 A. Construction of gingival (GP) and distal (DP) planes. B. Illustrations of palatal width measurements at three levels: canine, first premolar and molar. C. Measurements of palatal depth from GP to perpendicular opposing point on mid-palatal raphe at three levels: canine, first premolar and molar. D. Interdental spaces: intercanine; interpremolar and Intermolar. E. Software representation of palatal surface area measurement. F. Software display of palatal volume measurement

Two planes were constructed: gingival (GP) fitting through the palatal gingival mid-points of all maxillary teeth (generated by the software as the best approximation of all mid-points), and distal (DP) through the distal surface of the second molars and perpendicular to the gingival plane (Figure 1A). The palatal measurements included palatal width (PW) at the levels of canines, premolars and molars (Figure 1B); palatal depth (PD) at the level of canines, first premolars and first molars (Figure 1C); interdental widths between canines (intercanine distance ICD), first premolars (interpremolar distance IPMD) and first molars (intermolar distance IMD) (Figure 1D); palatal length (PL); palatal surface area (PSA) measured in mm^2 (Figure 1E); and palatal volume (PV) measured in mm^3 (Figure 1F). The boundaries for measuring palatal area and volume were the gingival plane and the distal plane, in line with previous definitions.[25,28]

Taken in the same cephalostat (OP100, Instrumentarium, Tuusula, Finland), the inherent magnification (14%) of the digital lateral cephalograms was automatically corrected for in the CliniView software of the machine. The images were thus imported from CliniView into the Dolphin Imaging software (Dolphin Imaging and Management Solutions, Version 11.5,) without magnification and digitized using a customized setting only to locate the selected landmarks needed for two angular measurements necessary to classify the patients into corresponding sagittal (ANB) and vertical (MP/SN) malocclusions.

To assess intra-observer reliability, all measurements were repeated (by MS) on 55 randomly selected patients (30% of the sample) at least 14 days after the initial assessment. In addition, measurements on 20 randomly selected casts were repeated by another orthodontist to evaluate inter-rater reliability.

2.1 | Statistical analysis

The Shapiro-Wilk's test of normality confirmed that the data were normally distributed ($P > .05$). A 3-way analysis of variance (ANOVA) was used to compare palatal dimensions between males and females, sagittal classes of malocclusion (I, II/1, II/2 and III) and vertical patterns (hypo, normo and hyperdivergent), as well as the interactions among the 3 variable components. Tukey's post hoc analysis was carried out when ANOVA results were statistically significant. Pearson product-moment correlations were employed to assess the linear correlations among linear palatal dimensions, PSA and PV. Data were processed through the Statistical Package for Social Sciences (SPSS®, version 23.0, IBM®) and Stata/SE ™ 11.1. Statistical significance was set at 0.05.

3 | RESULTS

The intraclass correlations ranged between 0.871 and 0.978 and between 0.867 and 0.969 for intra- and inter-rater reliability, respectively, demonstrating high correspondence. In addition, the paired t-test showed no statistically significant difference between first and second readings ($.082 < P < .973$).

The results of the 3-way ANOVA revealed the absence of three-way and two-way interactions among the evaluated components (sex, sagittal malocclusion and vertical pattern) except between sex and sagittal malocclusion for the PV ($P = .028$). Therefore, main effects are reported for all the comparisons and simple main effects for the PV in the different sagittal malocclusions.

3.1 | Sex dimorphism

Regardless of the sagittal and vertical malocclusion, females presented with significantly smaller palatal widths ($P = .017$) and depth ($P = .001$) at the level of the molars and slightly greater palatal depth at the level of the canines ($P = .029$) (Table 2). Intermolar distance ($P = .004$), palatal length and surface area ($P \leq .031$) were greater in males (Table 2). The PV measurement presented a significant two-way interaction between sex and malocclusion; accordingly, the comparison between males and females was segregated for the 4 groups of sagittal malocclusions and yielded greater volume for males ($P < .03$) except for the Class II division 1 group ($P = .888$).

3.2 | Sagittal malocclusion

PSA and PV (in both males and females) presented no statistically significant differences across sagittal malocclusions (Table 3). Most of the palatal dimensions did not exhibit significant differences among Class I, Class II/1, Class II/2 and Class III when the other factors (sex and divergence) were kept constant. Only palatal depth at the level of the canines was significantly greater for Class II/2 (mean 5.77, SD 0.36 mm) compared with Class I (mean 4.2, SD 0.25 mm; $P = .003$) and Class III (mean 4, SD 0.26 mm; $P = .001$). A marginal statistically significant difference depicted between malocclusions for the intermolar distance ($P = .047$) was lost at the multiple comparison level.

3.3 | Vertical pattern

No statistically significant differences were observed for PSA and PV across vertical malocclusions ($P = .42$ and $.292$, respectively) (Table 4). Statistically significant differences were observed between the hypo and hyperdivergence groups and between the hyper and normodivergence groups for the palatal width, which was greater in the hypodivergence group at the level of the molars ($P = .009$ and $P = .005$) and premolars ($P = .003$ and $P = .018$). Similar findings were noted for the intermolar distance and only between the hypo and hyper groups for the interpremolar distance. Palatal width at the level of the canines was significantly greater in the normodivergent than the hyperdivergent patterns ($P = .006$; Table 4).

3.4 | Correlations among palatal dimensions

Palatal surface area was strongly and significantly correlated with PV ($r = .87$, $P < .001$; Table 5). Among the linear palatal dimensions, PW-M, PD-M, IMD and PL ($r = .50–0.51$, $P < .001$) showed the highest linear correlations with surface area. The linear palatal measures showed similar trends in correlation with PV, although some correlations were smaller in magnitude. However, palatal depth at the level of the molars was correlated more strongly with PV than with PSA ($r = .65$, $P < .001$; Table 5).

TABLE 2 Distribution of palatal dimensions by sex ($n = 178$)

	Males ($n = 73$)		Females ($n = 82$)		Difference (M-F)		Univariate ANOVA	
	Mean	SE	Mean	SE	Mean	SE	F	p
PW-M	35.53	0.37	34.11	0.44	1.42	0.57	5.88	0.017*
PW-PM	28.02	0.3	27.03	0.35	0.99	0.46	3.789	0.054
PW-C	24.65	0.27	24.14	0.32	0.51	0.42	1.019	0.315
PD-M	16.71	0.29	15.29	0.34	1.42	0.44	10.600	0.001**
PD-PM	10.10	0.26	10.21	0.30	−0.10	0.39	0.035	0.851
PD-C	4.22	0.19	4.91	0.22	−0.69	0.29	4.852	0.029*
IMD	40.24	0.38	38.47	0.45	1.77	0.59	8.786	0.004**
IPMD	31.45	0.33	30.37	0.39	1.08	0.51	3.386	0.068
ICD	34.44	0.32	33.52	0.37	0.91	0.49	2.631	0.107
PL	35.95	0.32	34.87	0.38	1.07	0.50	4.782	0.031*
PSA	1877.44	24.43	1741.28	28.66	136.16	37.66	13.068	<0.001**
PV[$]								
I	11011.78	437.02	9804.43	385.41	1207.34	582.69	4.82	0.030*
II/1	10366.57	314.68	10284.14	300.04	82.43	434.796	0.02	0.888
II/2	11763.72	498.92	10427.84	498.92	1335.88	705.57	4.995	0.027*
III	11548.36	376.70	9370.44	427.14	2177.92	569.52	15.683	<0.001**

Note: All measurements recorded in (mm) except PSA (mm^2) and PV (mm^3).

[$]Simple main effects reported because interaction between sex and malocclusion was significant.

*Statistically significant at $p < 0.05$.

**Statistically significant at $p < 0.01$.

4 | DISCUSSION

The major finding of this study was that, *on average*, palatal surface area and volume are nearly equal across malocclusions, despite significant variations in linear dimensions. Yet, individual variations include outliers on both sides of this average tendency. The moderate correlations between PSA or PV and linear palatal dimensions, along with virtually non-existent linear correlations between palatal depth and width measures, suggest that the relationship between 3-dimensional palatal morphology and linear palatal dimensions is not proportional. Many palatal dimensions exhibited sexual dimorphism regardless of malocclusions, males showing, on average, greater linear palatal dimensions, surface area and volume.

The vertical facial pattern seems to impact the palatal dimensions more than the sagittal malocclusion. Palatal vault morphology differed significantly across the vertical patterns notably smaller palatal breadth and interdental widths and higher palatal depth in hyperdivergence (Table 4). In contrast, only palatal depths at the level of the canines differed statistically in Class II/2 versus the other sagittal malocclusions.

Comparisons between our results and those from previous studies are limited because prior measurements have not been recorded digitally on 3D images, and methods of measurement varied, palatal width and depth being measured at the level of the cusps of teeth[8,17,32] or at the gingival margin.[12,13] In addition, in the few studies in which the four malocclusions were compared, ANOVA comparisons were reported without *p* values for the multiple post hoc assessments to pinpoint more specific differences. Our findings corroborate other reports of wider palatal width in the Class II/2 phenotype compared with other malocclusions[8,10,17,31] but reflect more statistically significant differences specifically at the level of the canines and compared with Class III and Class I malocclusions. Studies assessing the relationship between palatal dimensions and the skeletal vertical pattern are not available for comparison, but the observed patterns suggest that the heterogeneity of the vertical pattern may be implicated, at least in part, in the inconsistency of previous results in relation to sagittal malocclusions.

This perspective invites the consideration of the functional matrix theory, which stipulates that the oropharyngeal capsular matrix, a volumetric space connected with respiratory function,[4] is genetically determined, and the soft tissues (e.g. muscles and ligaments) surrounding it help determine the shape of the skeletal units associated with their function. If true and applied to the present findings, the shape of the palatal vault might differ in width, height and length, modelling around the matrix because of individually varying environmental conditions. This hypothesis requires advanced research accounting for mandibular morphologic variations, which contribute to defining the individual size of the oropharyngeal matrix. In a similar perspective, Petrovic's cybernetic model may be considered, whereby the environmental influence on the maxilla would in turn affect mandibular growth and steer towards the development of specific malocclusions (e.g. Class II or Class III).[33]

TABLE 3 Distribution of palatal dimensions by sagittal malocclusion ($n = 178$)

	Class I ($n = 48$)		Class II/1 ($n = 42$)		Class II/2 ($n = 40$)		Class III ($n = 48$)		3-way ANOVA	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	F	*p*
PW-M	34.38	0.50	33.92	0.59	35.40	0.71	35.76	0.52	2.075	0.107
PW-PM	27.61	0.41	26.82	0.47	27.68	0.57	28.04	0.42	1.447	0.232
PW-C	24.68	0.37	24.11	0.43	25.11	0.51	23.90	0.38	0.987	0.401
PD-M	16.22	0.39	16.27	0.46	15.05	0.55	16.16	0.41	0.590	0.623
PD-PM	10.01	0.35	10.37	0.41	10.96	0.49	9.54	0.36	2.662	0.051
PD-C	4.20[a]	0.25	4.71[a,b]	0.30	5.77[b]	0.36	4.00[a]	0.26	5.384	0.002**
IMD	38.97	0.52	38.35	0.60	39.69	0.72	40.51	0.54	2.724	0.047*
IPMD	30.86	0.45	30.14	0.53	31.06	0.63	31.61	0.47	1.568	0.200
ICD	34.25	0.43	33.76	0.50	33.97	0.60	33.93	0.45	0.358	0.783
PL	35.77	0.44	35.87	0.51	35.60	0.61	34.47	0.45	1.914	0.130
PSA	1831.55	33.21	1809.28	38.85	1785.09	46.58	1803.43	34.50	0.399	0.754
PV[$]										
M	11011.78	462.60	10366.57	474.02	11763.72	474.02	11548.36	407.97	2.264	0.084
F	9804.43	329.20	10284.14	364.70	10427.84	382.50	9370.44	373.28	1.366	0.256

All measurements were recorded in mm except PSA (mm^2) and PV (mm^3).

Alphabetic superscripts denote the results of Tukey's post hoc analysis: significantly different means at $p < 0.05$.

[$]Simple main effects reported because interaction between sex and malocclusion was significant.

*Statistically significant at $p < 0.05$.

**Statistically significant at $p < 0.01$.

TABLE 4 Distribution of palatal dimensions by vertical malocclusion ($n = 178$)

	Hypo (1) ($n = 35$)		Normo (2) ($n = 95$)		Hyper (3) ($n = 48$)		3-way ANOVA		Tukey's Post hoc p value		
	Mean	SE	Mean	SE	Mean	SE	F	p	1vs.2	1vs.3	2vs.3
PW-M	35.65	0.63	35.11	0.33	33.32	0.45	5.790	0.004**	1	0.009**	0.005**
PW-PM	28.38	0.50	27.57	0.27	26.32	0.36	6.429	0.002**	0.471	0.003**	0.018*
PW-C	24.79	0.46	24.69	0.24	23.46	0.33	3.706	0.027*	1	0.059	0.009**
PD-M	15.56	0.49	15.81	0.26	16.85	0.35	2.066	0.131	–	–	–
PD-PM	9.90	0.43	10.13	0.23	10.51	0.31	1.886	0.156	–	–	–
PD-C	4.99	0.32	4.44	0.17	4.18	0.23	1.193	0.306	–	–	–
IMD	40.15	0.64	39.61	0.34	37.93	0.46	5.328	0.006**	1	0.017*	0.012*
IPMD	31.71	0.56	30.87	0.30	29.89	0.41	3.676	0.028*	0.572	0.03*	0.161
ICD	34.45	0.54	34.15	0.28	33.11	0.39	3.204	0.044*	1	0.134	0.095
PL	35.88	0.54	35.32	0.29	34.90	0.39	0.967	0.383	–	–	–
PSA	1827.64	41.34	1813.35	21.73	1779.66	29.82	0.873	0.42	–	–	–
PV	10914.13	435.2	10776.3	228.81	10198.01	313.9	1.244	0.292	–	–	–

All measurements recorded in mm except PSA (mm^2) and PV (mm^3).

*Statistically significant at $p < 0.05$.

**Statistically significant at $p < 0.01$.

TABLE 5 Correlations among linear palatal dimensions, palatal surface area and palatal volume ($n = 178$)

	PW-M	PW-PM	PW-C	PD-M	PD-PM	PD-C	IMD	IPMD	ICD	PL	PSA
PW-PM	0.67**										
PW-C	0.48**	0.75**									
PD-M	0.11	0.02	−0.08								
PD-PM	−0.11	−0.09	−0.05	0.36**							
PD-C	−0.01	0.02	0.04	−0.02	0.67**						
IMD	0.92**	0.63**	0.49**	0.07	−0.16*	−0.02					
IPMD	0.61**	0.92**	0.72**	0.04	−0.15*	−0.07	0.61**				
ICD	0.36**	0.62**	0.78**	0.06	−0.08	−0.06	0.41**	0.64**			
PL	0.04	0.12	0.28**	0.07	0.25**	0.36**	0.15*	0.13	0.39**		
PSA	0.50**	0.45**	0.45**	0.50**	0.28**	0.16*	0.51**	0.41**	0.46**	0.51**	
PV	0.51**	0.44**	0.37**	0.65**	0.37**	0.22**	0.49**	0.40**	0.35**	0.42**	0.87**

Correlation coefficients are Pearson product–moment correlation coefficients *r*.

*Statistically significant at $p < 0.05$.

**Statistically significant at $p < 0.01$.

Sexual differences were observed for all measurements except for PV in Class II/1, possibly because the elongated and narrow maxillary arches in this malocclusion[34,35] reach similar constrictions and depths in both sexes. Huth et al[32] reported that maxillary arch width in Class II adult patients was narrower in males than females. In our study, the statistically significant linear (length, width and depth) sexual differences were less than 2 mm on average (Table 2) and possibly not clinically significant. Nonetheless, more focused research is warranted with larger samples within the Class II and other malocclusions.

The significance of the findings relates to the potential of earlier treatment in childhood during the development of malocclusion to control environmental contributors impacting proper growth and the stability associated with such control. The translation of this tenet in clinical practice would include the control of habits, the interaction with medical specialists regarding mouth breathing to help restore nasal respiration, and accordingly promote proper tongue position. Earlier intervention in Class II/2 would prevent the development of a deep bite that would restrain the forward displacement of the dentition along with the mandible, thus leading to the growth of the chin away from the teeth and enhancing the characteristic 'chin button'.[36] These premises require research in longitudinal samples to sort out the influences of environmental components on the genetic make-up. Whereas the partition of genetic and environmental contributions to organ and system development is complex, the average similarity of volumes across malocclusions underscores the role but also the limitations of environmental influences, including orthodontic treatment.[37]

Several research limitations are noted. Although they were part of the environmental factors that contributed to present phenotype, the influences of prior function and habits such as mouth breathing are unknown. Subjects with posterior crossbite were excluded; however, maxillary transverse hypoplasia may have been camouflaged by lingoversion of the mandibular posterior teeth. Accordingly, skeletal assessment of transverse discrepancies should be included in future studies. While the findings indicate important trends, the results are particular to the population studied and larger samples are needed for generalizable conclusions.

Also, dental casts might have carried minor errors related to impression taking and model pouring, but they provided better access to the powerful laser scanner in a specialized engineering facility. Future research could be based on direct mouth scans that would also be carried in a larger sample. In addition, the investigated outcome variables could be explored in twins and different ethnic groups, and in a more clinical perspective, gauging the short and long-term effect of orthodontic and orthognathic treatments on palatal volume and dimensions.

5 | CONCLUSION

Palatal volume and surface area were similar, on average, across malocclusions, despite differences in palatal shape and linear dimensions (breadth, depth and interdental widths), which differed significantly across the vertical patterns. These findings underscore the role of sustained environmental dynamics in shaping phenotypical variations of malocclusions.

AUTHOR CONTRIBUTIONS

Maria Saadeh contributed to methodology, data collection, investigation, statistical analysis and writing – original draft, in addition to review and editing. **Joseph Ghafari** contributed to conceptualization, methodology, project administration and writing – review and editing.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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