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TITLE PAGE

Magnetic resonance imaging of the upper airway in patients with quadriplegia and obstructive sleep apnoea

SUBTITLE: Upper airway anatomy in quadriplegia

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ABSTRACT

The aim of this study was to investigate upper airway anatomy in quadriplegics with obstructive sleep apnoea (OSA). Fifty subjects were recruited from three hospitals in Australia: People with quadriplegia due to spinal cord injury and OSA (SCI-OSA, n=11), able-bodied people with OSA (AB-OSA, n=18) and healthy, able-bodied controls (AB-CTRL, n=19). All underwent 3-Tesla Magnetic Resonance (MR) imaging of their upper airway. A subgroup (N=34) received a topical vasoconstrictor, phenylephrine, and a post-phenylephrine MRI. Mixed model analysis indicated no significant differences in total airway lumen volume between the three groups (p=0.086). SCI-OSA subjects had significantly larger volume of soft palate (p=0.020) and retroglossal lateral pharyngeal walls (p=0.043) than AB-CTRLs. AB-OSA subjects had smaller mandible volume than SCI-OSA and AB-CTRLs (p=0.036). No differences were seen in airway length between groups when controlling for height (p=0.055). There was a marginal increase in velopharyngeal volume across groups post-phenylephrine (p=0.050), and post-hoc testing indicated the difference was confined to the AB-OSA group (p<0.001). No other upper airway structures showed significant changes with phenylephrine administration. In conclusion, people with OSA and quadriplegia do not have a structurally smaller airway than able-bodied subjects. They did however have greater volumes of soft palate and lateral pharyngeal walls, possibly due to greater neck fat deposition. The acute response to upper airway topical vasoconstriction was not enhanced in those with OSA and quadriplegia. Changes in upper airway anatomy likely contribute to the high incidence in OSA in quadriplegic subjects.

Keywords: Obstructive Sleep Apnoea (OSA); quadriplegia; upper airway; airway anatomy; Magnetic Resonance Imaging (MRI)

INTRODUCTION

Sleep difficulties are extremely prevalent in quadriplegia due to spinal cord injury (SCI), and have a profound impact on daily functioning and quality of life (Berlowitz et al., 2005, Spong et al., 2008, Stockhammer et al., 2002, Sankari et al., 2015, Proserpio et al., 2015). In particular there is the high prevalence of obstructive sleep apnoea (OSA) in this group (Berlowitz et al., 2005, McEvoy et al., 1995, Proserpio et al., 2015, Sankari et al., 2015, Giannoccaro et al., 2013) that may contribute to the high incidence of cardiovascular disease (Myers et al., 2007).

OSA in able bodied subjects is primarily a disorder of upper airway anatomy (Schwab et al., 1995, Schwab et al., 2003) However in many subjects alterations in arousal threshold, impaired effectiveness of upper airway dilator muscles, and unstable ventilatory control (Eckert and Malhotra, 2008, O'Halloran et al., 1998) are involved to varying degrees. OSA pathogenesis in SCI may be different to able-bodied subjects (Proserpio et al., 2015). Immediately after acute quadriplegia, traumatic tissue swelling and surgical fixation are likely to reduce the upper airway size, while later potentially causative factors include an increase in body fat (Crane et al., 2011), neck size (Spong et al., 2008, McEvoy et al., 1995), reduced lung volume (Heinzer et al., 2006), and reduced or absent sympathetic tone leading to upper airway vascular engorgement and mucosal thickening (Berlowitz et al., 2005, Krassioukov, 2009, Wasicko et al., 1990, O'Halloran et al., 1998).

In addition to upper airway narrowing, increased collapsibility of the airway may lead to OSA (Schwartz et al., 1988, Gleadhill et al., 1991), and indeed collapsibility in sleep is increased in sedated SCI patients with OSA compared to healthy control subjects (Sankari et al., 2014). In able-bodied subjects, increases in lung volume reduce airway collapsibility through tracheal traction and reduce OSA severity (Van de Graaff, 1991, Stanchina et al., 2003, Van de Graaff, 1988). SCI patients have a marked reduction in resting lung volume immediately following injury, which only partially improves over time (Anke et al., 1993). Furthermore, MRI studies have demonstrated pharmacological vasodilation of the upper airway mucosa increases collapsibility and decreases airway area in cats, whilst topical vasoconstriction increases cross-sectional area, and reduces nasal and pharyngeal resistance and collapsibility (Parisi et al., 1989, Wasicko et al., 1991, Wasicko et al., 1990). Thus it is plausible that in people with quadriplegia, OSA may develop because of altered anatomy of the upper airway, augmented by mucosal vascular engorgement due to unopposed parasympathetic innervation, leading to increased upper airway resistance and collapsibility. Given the reduced acceptance of conventional OSA treatments in SCI patients, this highlights the need to determine pathophysiological mechanisms in order to guide the

development of novel and effective therapies (Berlowitz et al., 2012, Stockhammer et al., 2002, Berlowitz et al., 2009).

The goal of the present study was firstly to determine whether upper airway anatomy in patients with quadriplegia and OSA differs from able-bodied subjects with and without OSA. Specifically, we hypothesised that the upper airway of patients with OSA and chronic quadriplegia would demonstrate different anatomic risk factors to able-bodied OSA patients, when compared to able-bodied controls. Secondly we aimed to examine the effects of topical vasoconstriction on the pharyngeal airway of patients with quadriplegia. We hypothesised that upper airway patency would improve post-vasoconstriction as a result of mucosal vasoconstriction and that it would improve more in patients with quadriplegia than in either of the able bodied groups.

METHODS

Subjects

Subjects were recruited from Austin Health in Melbourne, and the Prince of Wales and Royal North Shore Hospitals in Sydney, Australia, and through community advertisements at all sites. Subjects were recruited in three groups: chronic quadriplegia and OSA (SCI-OSA; n=11), able-bodied subjects with OSA (AB-OSA; n=18) and healthy, able-bodied controls (AB-CTRL) (n=19). Only SCI-OSA subjects with stable, chronic (greater than 12 months), injuries resulting in quadriplegia were recruited to the study. AB-OSA and SCI-OSA subjects were recruited from the clinical populations of the sleep and spinal cord injury services at each hospital, and were matched for age, OSA severity, and Body Mass Index (BMI). OSA was defined as an Apnoea-Hypopnea Index (AHI) >10 events/hour on polysomnography (PSG). AB-CTRL subjects were required to have an AHI ≤10 event/hour, and no other sleep disorders. Subjects underwent 3 Tesla (3T) MRI safety screening for the presence of non-compatible implants. Subjects with body weight >125kgs were also excluded due to scanner bore limitations. The project was approved by relevant research ethics committees, and subjects provided informed written or verbal (SCI unable to sign) consent.

Polysomnography

Subjects underwent PSG unless results from a recent (<2 yrs) diagnostic PSG were available. Portable PSG was performed using a Somté (Compumedics Pty, Abbotsford, Australia), or Embletta X50 (Medcare, Iceland) device. Studies were scored according to standardised criteria (Quan et al., 1999) by independent, experienced sleep technologists.

Magnetic Resonance Imaging of the Upper Airway

High resolution 3T MR upper airway images were acquired in Melbourne using a Siemens Magnetom Tim Trio scanner (Siemens, Erlangen, Germany) and in Sydney using a Philips Achieva 3TX scanner (Philips Healthcare, Best, Netherlands). Scanning protocols at both sites were matched for consistency during preliminary testing. Scans were acquired with the subject awake in the supine position with head and neck supported with foam padding to minimize movement with the Frankfort plane as close to vertical as possible (SCI subjects with spinal fixation usually cannot achieve this head alignment). Subjects were asked to keep as still as possible with mouth closed in a relaxed bite, and to refrain from swallowing. Detailed scanning protocols are provided in the online supplement.

In a subset of subjects (n=34; SCI-OSA=6, AB-OSA=16, AB-CTRL=12), the scan sequence was repeated after administration of a atomised solution of the decongestant/vasoconstrictor, phenylephrine hydrocholoride 0.5%, to each nostril, and to the pharynx while the participant remained with head immobilized on the scanner bed. Respiratory-gated sagittal T2 weighted images were also acquired pre- and post-administration of the nasal decongestant/vasoconstrictor using a TSE Blade sequence. Scanning protocols are again provided in detail online.

Anatomic Definitions and Image Analysis

A single trained assessor, blinded to subject group, analyzed images in random order. Segmented volumes of airway lumen, soft palate, parapharyngeal fat pads, tongue, mandible, and lateral pharyngeal walls (LPW) (Figure 1) were determined using Amira software (v5.5; FEI Visualization Sciences Group, Hillsboro, OR, USA) as previously utilised by ourselves (Sutherland et al., 2011, Chan et al., 2010) and others (Schwab et al., 1995, Welch et al., 2002, Schwab et al., 1996, Schwab et al., 2003). The airway lumen was segmented into three regions: velopharynx (hard palate to tip of uvula), oropharynx (tip of uvula to base of epiglottis), and hypopharynx (base of epiglottis to vocal cords). The tongue region was segmented into the tongue (genioglossus and intrinsic tongue muscles) and lower tongue (geniohyoid and mylohyoid) muscles. The LPWs were subdivided into retropalatal LPW and retroglossal LPW and the tonsils were segmented separately. For those who had pre/post vasoconstrictor sagittal scans, cross sectional area of the upper airway and lateral and antero-posterior diameters of the velopharynx were measured. This latter measurement was complicated by the curved and folded nature of the anterior wall of the velopharynx. Therefore a line was drawn joining the anterior extremes of the folds and

another joining their posterior extremes, and finally a line midway between. This latter line was then used to make the measurement in the median sagittal plane. The lateral dimension measurement was taken in the coronal plane at the widest point of the velopharynx. Airway length was calculated by multiplying the number of contiguous slice segments of the three airway regions by the slice thickness, and was corrected for height. To investigate the effect of phenylephrine, airway lumen and LPW volumes were compared pre- and post-phenylephrine, as well as minimum cross-sectional area (CSA), and minimum lateral and antero-posterior measurements of the airway.

Measurement Reliability

Volumetric analyses were repeated in a blinded fashion by the same assessor on five randomly sampled scans. Intra-class correlation coefficients (ICCs) were calculated for all structures.

Statistical analysis

Statistical analyses were performed using SPSS Version 16.0 (IBM, Chicago, IL, USA). To identify differences in clinical characteristics, one-way ANOVA was used for normally distributed data, with Mann-Whitney and Chi-Square tests for non-normally distributed or categorical variables. Linear mixed models were utilized to investigate the differences in upper airway tissue/lumen volumes, with group (fixed effects) and site (random effects) as factors. Post-hoc Least Significant Difference (LSD) t-tests were used to further investigate group differences. In a secondary analysis, neck circumference was added as a covariate to investigate the relationship between neck size and upper airway lumen and tissue volumes. One-way ANOVA was used to compare the changes pre- to post-phenylephrine across groups, followed by post-hoc LSD t-tests to investigate differences between groups. Repeated measures ANOVA was used to investigate differences in minimum airway CSA, AP and lateral dimensions, and length, from pre- to post-phenylephrine. Results are presented as mean ± SD unless otherwise specified. Statistical significance was defined as $p<0.05$.

RESULTS

Clinical characteristics

Fifty subjects underwent upper airway imaging across the two sites. Despite inherent difficulties in image acquisition in subjects with quadriplegia (Allouni et al., 2013), scans from 49 subjects were of adequate quality for analysis. In addition, one AB-CTRL participant who

provided false personal demographic details was excluded from the analysis. Spinal injury levels ranged from C3 to C7, and American Spinal Injury Association (ASIA) impairment grading ranged from 'A' to 'C', with median (range) time post injury 18 (2.5 – 52) years. Participant characteristics are presented in Table 1.

No differences were observed in BMI, gender mix, height or study site between the groups. Control subjects were younger than both SCI-OSA (p=0.008) and AB-OSA groups (p<0.001). SCI-OSA subjects had larger neck circumference than both the AB-OSA (p=0.009) and the AB-CTRL groups (p<0.001). There was no significant difference in sleep apnoea severity as measured by AHI between OSA groups (p=0.56).

Measurement Reliability

The ICCs for the upper airway structure measurements ranged from 0.80 (lower tongue) to 0.997 (soft palate) with a mean ICC of 0.96, suggesting intra-rater reliability was good.

Upper airway volumetric measurements

Airway luminal volumes, total or regional, did not differ significantly between groups (Table 2; Figure 2). However, differences in soft palate, retroglossal LPW and mandibular volumes were observed between the groups (Table 2). Post-hoc tests indicated that SCI-OSA subjects had a larger volume of soft palate tissue than AB-CTRLs (p = 0.007) but not significantly larger than AB-OSA (see Table 2). Similarly for the retroglossal LPW, SCI-OSA subjects were found to have a larger tissue volume than AB-CTRLs (p=0.014). In addition, AB-OSA subjects had smaller mandible volumes than both SCI-OSA (p=0.019) and AB-CTRLs (p=0.038). Please refer to the online supplement for images depicting the differences in airway lumen, soft-palate and retroglossal LPW volumes between groups.

Effect of neck circumference

The Mixed Model Analysis was repeated for soft tissue structures while including neck circumference as a covariate, due to the significantly larger neck size of SCI-OSA subjects. In this analysis there was no longer a difference in either soft palate (p=0.701) or retroglossal pharyngeal wall volumes (p=0.577) between SCI-OSA and AB-CTRL groups.

Airway length and Cross-Sectional Area

An overall significant difference in total airway lumen length across groups was observed (p = 0.021) (see Table 3). However there was no significant difference (p=.055) when corrected for height (premorbid height in the case of SCI subjects)(Table 3). No differences were seen in hypopharyngeal, oropharyngeal or nasopharyngeal length (Table 3). Luminal minimal cross-sectional area (CSA) did not differ across the groups (Table 4).

Phenylephrine and the upper airway

Analyses of the magnitude of volume change across groups before and after atomised phenylephrine administration (n=34 subjects) indicated there was a marginally significant difference in velopharyngeal change post-phenylephrine (p=0.050), see Table 5 and Figure 3. LSD testing indicated the change was significantly larger in the AB-OSA than in the other two groups (p<0.001). Individual participant data for velopharyngeal volume change is displayed in Figure 3. There were no differences in changes in any other volumes between groups.

No statistically significant changes were found between groups in minimum CSA, (p =0.216), lateral, (p=.259) or antero-posterior diameter, (p=.548), after phenylephrine administration (see online supplement). In addition, no changes in velopharyngeal length were observed after phenylephrine administration (p=0.08) (see online supplement).

DISCUSSION

This is the first study to examine upper airway anatomy in subjects with quadriplegia and OSA using MRI. The major findings of this study were 1) there was no difference in total or regional airway luminal volume between SCI subjects with OSA and able bodied subjects with or without OSA; 2) similarly, neither minimal airway cross-sectional area, nor airway length differed between groups, once account was taken of height differences; 3) both soft palate and retroglossal LPW volume were greater in SCI-OSA patients than controls; 4) able-bodied OSA patients had smaller mandibular volumes than both SCI-OSA and able-bodied control subjects and 5) after phenylephrine administration there was a marginal increase in velopharyngeal luminal volume in the able-bodied OSA group.

While there are multiple potential causes for the increased prevalence of OSA in SCI subjects (Fuller et al., 2013) only two previous studies have sought to elucidate the mechanisms. Our group observed a higher nasal resistance but no difference in pharyngeal or total upper airway resistance in subjects with OSA and quadriplegia compared with able-

bodied controls matched for OSA severity, age and gender (Gainche et al., 2016). Sankari et al. (2014) examined upper airway mechanics following oxymetolazine decongestion (confirmed by A. Sankari, personal communication) during sleep in both thoracic and cervical SCI patients compared to healthy control subjects and observed no difference in total upper airway resistance between groups. The results of the current study are in broad agreement with both these previous reports, in that airways of similar size and cross-sectional area would be expected to have similar upper airway resistance.

Longer airways are thought to be intrinsically more collapsible, and this has previously been proposed as a factor underlying the higher prevalence of OSA in men compared to women (Malhotra et al., 2002). The current study found that both OSA groups had slightly longer airways than the able-bodied control subjects; however we found no difference in airway length between groups after correction had been made for height.

We observed that able-bodied OSA subjects had smaller mandibles than SCI-OSA and able-bodied control subjects. Altered craniofacial anatomy is a known risk factor for OSA, particularly in the absence of obesity (Sutherland et al., 2012). However SCI patients, in the absence of pre-injury OSA, would not be expected to demonstrate craniofacial differences.

It is well recognised that there are significant changes in body habitus following quadriplegia (Spungen et al., 2003). There is wasting of the limb and axial skeletal musculature, and an increase in percentage body fat. Neck circumference is often increased (Frisbie and Brown, 1994), as in the current study (Table 1), and neck circumference is correlated with both total body fat and total abdominal fat (Ravensbergen et al., 2014). The observation that soft palate and retroglossal LPW volume were higher in the SCI group than control subjects, but that this difference was no longer significant after adjusting for neck circumference in the statistical model, suggests that this difference also may be due to increased fat deposition. Similar changes have been described in able-bodied subjects when compared to healthy control subjects (Schwab et al., 1995).

Increased nasal congestion or nasal resistance is commonly seen clinically in people with quadriplegia, due to unopposed parasympathetic outflow to the nasal mucosa causing engorgement (Lewis et al., 2016, Gainche et al., 2016). The pharyngeal mucosa is exposed to the same unopposed parasympathetic output, in theory causing mucosal engorgement in this region also. Our group recently reported that phenylephrine reduced nasal resistance but not pharyngeal resistance in SCI patients, and had no significant effect in able bodied

OSA patients (Gainche et al., 2016) . In the current study we hypothesised that the effect of topical sympathomimetics administered to the nose and pharynx would be greater in SCI patients than in either of the other 2 groups. We found that phenylephrine led to a significantly greater increase in velopharyngeal volume in AB-OSA patients than in either SCI-OSA or AB-CTRL groups. While it is unsurprising that there would be some upper airway mucosal swelling in able–bodied OSA patients (Ryan et al., 1991), the reasons why this should be less in SCI OSA patients are unclear.

Limitations of the study included the relatively small sample size, particularly of SCI-OSA subjects. This was primarily due to the fact that most spinal implants do not have 3T MR safety clearance. In addition, by design we did not recruit a group of SCI subjects without OSA due to these safety challenges but also due to the very high OSA prevalence in the SCI population, which would make it extremely challenging to find SCI subjects free of OSA. Swallowing, phonation and respiratory compromise, as well as autonomic instability, are all commonly reported in those with higher SCI. While we made no direct assessments of these issues in our study, participants reported none of these clinical problems during screening for study inclusion.

In order to increase recruitment, scanning was conducted at two different sites, presenting challenges in matching data acquisition. Scanning protocols for the Melbourne and Sydney sites were matched as closely as possible between the two MRI scanners however it is impossible to guarantee data acquisition was identical at both sites.

Assessment of skeletal structures was limited to volume of mandible. Given the known interaction between craniofacial and soft tissues structures in OSA pathogenesis (Tsuiki et al., 2008) we do not know whether an anatomical imbalance may have existed in our SCI subjects. On the other hand, there is no reason to believe premorbid skeletal changes would have existed in these patients, though the effects of injury and subsequent spinal stabilization surgery are difficult to estimate. Future research may help clarify these issues.

Direct assessment of the pharyngeal mucosal thickness before and after phenylephrine was not possible because MRI image quality did not allow direct segmentation of mucosal thickness. Furthermore collection of fluid droplets at the back of the throat after phenylephrine administration was seen in some subjects, which could not be distinguished from a change in mucosal water content. Therefore we used a surrogate

measure of the effect of phenylephrine by assessing lumen and LPW volumes from pre-to post-phenylephrine.

Finally, there could be a potential angulation bias influencing the airway length measurement, as SCI subjects may not have had the same pharyngeal angulation as able-bodied subjects

Conclusion

This is the first study to investigate the upper airway anatomy of patients with OSA and quadriplegia using MRI. The results indicate that quadriplegic OSA patients do not have a structurally smaller airway than able-bodied subjects. However they did have greater volumes of soft palate and lateral pharyngeal walls, possibly due to greater neck fat deposition. These differences may contribute to the higher rate of OSA in SCI subjects, in addition to the increased nasal resistance. Due to the low adherence rates to conventional OSA treatment for people with quadriplegia, it is extremely important that we continue to investigate the causes of OSA in this group, and to develop novel treatments that may be able to improve sleep quality and ultimately, quality of life, for people living with quadriplegia.

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Table 1 – Participant characteristics

GROUP	SCI-OSA (*n* = 11)	AB-OSA (*n* = 18)	AB-CTRL (*n* = 19)	*p*
Age (yrs)	49.2 ± 13.9	53.8 ± 11.7	36.8 ± 10.6[#]	.001*
BMI ($kg.m^{-2}$)	27.9 ± 5.1	30.1 ± 5.2	26.5 ± 5.3	.153
Height (cm)	178.2 ± 11.8	172.5 ± 8.5	173.1 ± 9.1	.279
Neck circumference (cm)	44.8 ± 4.1[#]	41.0 ± 4.2	38.6 ± 2.8	.001*
AHI (hr^{-1})	36.7 ± 29.6	30.8 ± 20.2	4.0 ± 3.2[#]	.56 ^
Sex (% Male)	81	72	74	.833
Site (% Melbourne)	54	89	63	.091

Explanation of abbreviations: AHI = Apnoea Hypopnea Index; AB= able-bodied; BMI = Body mass index; OSA = Obstructive Sleep Apnoea; SCI = Spinal Cord Injured; * $p < 0.05$ on one way ANOVA or nonparametric tests for comparison of all 3 groups. ^ = One way ANOVA only between SCI-OSA and AB-OSA group. [#] $p < .05$ indicating that this group was significantly different from the other two groups (post-hoc LSD t-test)

Table 2 – Upper airway lumen and soft tissue mean (±SD) volumes (cm^3) by participant group

GROUP	SCI-OSA	AB-OSA	AB-CTRL	*p*-value
Total airway lumen	16.4±5.6	12.1±4.9	11.8±4.5	0.086
Velopharynx	3.4±1.0	3.2±1.5	3.1±1.0	0.789
Oropharynx	7.2±3.7	4.7±3.3	4.7±2.5	0.090
Hypopharynx	5.6±1.7	4.7±2.2	4.3±1.8	0.256
Soft palate	14.1±2.3#	12.7±3.2	11.1±2.4#	0.020*
Parapharyngeal fat pads	7.6±2.2	7.0±2.4	6.6±2.0	0.472
Mandible	61.1±10.4	48.3±12.8^	57.9±10.6	0.036*
Total tongue	123.8±19.6	117.3±22.8	108.9±19.2	0.160
Tongue	100.6±14.7	96.0±19.1	88.1±15.3	0.125
Lower tongue	23.2±6.5	21.4±5.1	20.9±5.4	0.568
Total LPW	25.5±5.3	21.1±5.9	23.6±7.2	0.566
Retropalatal LPW	10.3±1.6	10.1±2.6	9.5±1.7	0.466
Retroglossal LPW	10.1±2.90#	7.1±3.0	7.4±2.8#	0.043*
Tonsils	5.1±3.6	3.9±2.9	6.7±4.8	0.143
Total upper airway soft tissue	170.9±23.8	158.2±30.7	150.2±25.4	0.146

Note: * $p<0.05$ for comparison of all 3 groups in Mixed Model Analysis, # $p < .05$ for post-hoc LSD t-test, between groups designated, ^ $p < .05$ for post-hoc LSD t-test between group designated and both other groups, LPW = Lateral pharyngeal walls

Table 3 – Mean (±SD) airway length (cm) by participant group

Airway region	**SCI-OSA**	**AB-OSA**	**AB-CTRL**	***p*-value[β]**	***p*-value [£]**
Total Airway	10.2±0.4	9.7±1.1	9.2±0.9[#]	0.021*	.055
Velopharynx	3.6±0.3	3.6±0.6	3.3±0.5	0.178	.167
Oropharynx	4.0±0.5	3.5±0.8	3.4±0.7	0.105	.266
Hypopharynx	2.7±0.4	2.6±0.5	2.5±0.6	0.347	.466

Note: [β] *p*-value for the effect of group from a Mixed Model Analysis; [£] ***p*-value corrected for height in mixed model analysis** [#] $p < .05$ for post-hoc LSD t-test between group designated and both other groups, without height correction.

Table 4 – Minimum cross-sectional area (cm^2) by participant group

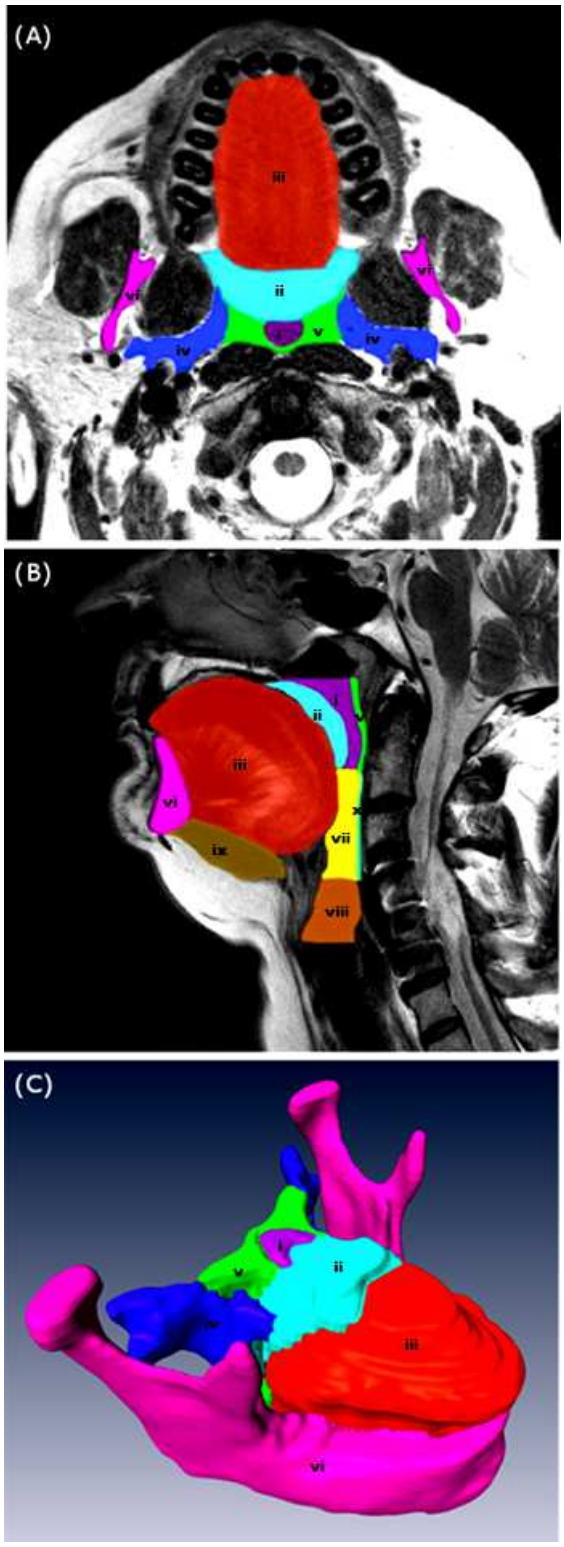
Airway region	SCI-OSA	AB-OSA	AB-CTRL	*p*-value[β]
Total Airway	0.3±0.2	0.3±0.2	0.3±0.1	0.854
Velopharynx	0.4±0.2	0.4±0.3	0.4±0.2	0.880
Oropharynx	0.9±0.6	0.6±0.4	0.7±0.5	0.410
Hypopharynx	1.3±0.5	1.1±0.6	0.9±0.4	0.183

Note: [β] *p*-value for the effect of group from a Mixed Model Analysis

Table 5 - Mean change (±SE) (cm^3) in upper airway volumes from pre-to post-phenylephrine for Melbourne sample.

GROUP	SCI-OSA (*n* = 6)		AB-OSA (*n* = 16)		AB-CTRL (*n* = 12)		
Structure	**Mean±SE**	**95% CI**	**Mean±SE**	**95% CI**	**Mean±SE**	**95% CI**	***p*-value[β]**
Velopharynx	0.3±0.3	-0.3, 1.0	0.9±0.2[#]	0.5, 1.3	0.1±0.2	-0.4,0 .6	0.050
Oropharynx	-0.5±0.5	-1.4, 0.5	0.2±0.3	-0.4, 0.8	-0.7±0.3	-1.3,1.0	0.130
Hypopharynx	-0.4±0.4	-1.3, 0 .4	-0.2±0.2	-0.7, 0.3	-0.3±0.3	-0.8, 0.3	0.892
Soft Palate	0.3±0.7	-1.2, 1.8	1.0±0.4	0.2, 1.9	0.3±0.5	-0.8, 1.3	0.450
Retropalatal LPWs	-0.0±0.5	-1.0, 0.9	0.4±0.3	-0.1, 1.0	-0.1±0.3	-0.7, 0.6	0.471
Retroglossal LPWs	0.4±0.5	-0.6, 1.4	0.6±0.3	-0.0, 1.1	-0.4±0.3	-1.0, 0.3	0.126
Palatine Tonsils	-0.5±0.4	-1.3, 0.2	0.2±0.2	-0.3, 0.6	-0.1±0.2	-0.5, 0.4	0.293

Note: SE = Standard Error; [β] *p*-value for the effect of group from a one-way ANOVA; [#] $p < .05$ for post-hoc LSD t-test and indicates that this group was significantly different from the other two groups; LPWs = Lateral Pharyngeal Walls



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