JAW MOVEMENT DURING SLEEP

by

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A thesis submitted to the Department of Physiology
In conformity with the requirements for
the degree of Master of Science

Queen’s University
Kingston, Ontario, Canada
(August, 2008)

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Abstract

Objective: We aim to improve our understanding of sleep physiology by describing the changes in mandibular position during sleep in normal subjects. Methods: We developed a novel method for mapping mandibular position simultaneously in three dimensions (anteroposterior, vertical and lateral) using magneto-resistive sensors strategically placed around 3 different moving joints on an external apparatus attached to the head and mandible. Spherical coordinates derived from these sensors provided information of jaw position in each of the three measurement planes. We assessed changes in jaw position in twelve healthy subjects (6 male, 6 female) aged (mean ± SD) 23 ± 7 years, Body Mass Index 22.5 ± 3.4 kg/m², and with nasal resistance 3.24 ± 0.67 cmH₂O/L/s by recording mandibular position simultaneously with overnight sleep polysomnography. Results: Jaw position was significantly influenced by sleep stage (p<0.001). The transition from wake to light sleep (stage one) was accompanied by significant jaw closure and jaw protrusion (p<0.05). As non-rapid-eye-movement (NREM) sleep deepened from stages 1 through slow wave sleep (SWS), vertical jaw opening (p<0.05) and posterior jaw movement progressively increased (p<0.05). REM sleep was associated with the greatest degree of jaw opening of all sleep stages (p<0.05). Lateral jaw position was not significantly different between sleep stages. Conclusion: This study describes, for the first time, an accurate method of measuring changes in mandibular position during sleep in all three dimensions. The observed changes during sleep in healthy subjects suggest a simultaneous modulation of upper airway muscular tone, which may be important in the understanding of upper airway occlusion in Obstructive Sleep Apnea.
Co-Authorship

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Acknowledgements

I acknowledge the helpful suggestions of Dr. Helen Driver (Department of Psychology, Queen’s University, Kingston, Ontario, Canada; Sleep Disorders Laboratory, Kingston General Hospital, Kingston, Ontario, Canada) as well as the extensive help and support by the Kingston General Hospital Sleep Laboratory Staff. We are grateful for the help of Carl Blouw in the department of physics, Queen’s University for his help in the earlier iterations of the jaw measurement equipment as outlined in Appendix A. We would also like to acknowledge the statistical assistance of Andrew Day (Kingston General Hospital) and the collection assistance of Gillianne Labelle, Stephanie Willings, Kendra Hollidge and Arif Nazir.
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List of Abbreviations

ABDO – Abdominal movement measured during sleep

BMI – Body Mass Index

CSA – Cross Sectional Area

EEG – Electroencephalography

EMG-electromyography

EOG- electrooculography

IEMG-Intercostal Electromyography

LEMG-Leg Electromyography

Eff – Sleep Efficiency

FEVi – Forced Expiratory Volume (L) expired in 1 second

FVC – Forced Vital Capacity

NASAL – Nasal airflow measured at the nares

NR – Nasal Resistance

NREM – Non Rapid Eye Movement (Stages 1, 2, 3 and 4)

ORAL – Oral airflow measured at the lips

OSA – Obstructive Sleep Apnea

REM – Rapid Eye movement

REM-L - REM onset latency

SOL - Sleep Onset Latency

SWS – Slow Wave Sleep (stages 3 and 4 combined)

THOR – Thoracic movement measured during sleep

TTFS – Total Time of First Sleep Cycle

Wake – Portion of time during recording when the subject is awake but relaxed and not moving
Chapter 1

Introduction

Obstructive Sleep Apnea (OSA) is a common sleep disorder that affects both obese and non-obese individuals and has been linked to high mortality and morbidity. Risk factors for OSA include obesity, gender (affects 4% of males and 2% of females), race (more common in Blacks and Asians), age, post-menopausal status, pregnancy, adenotonsillar enlargement, retrognathism, hypertension, heart failure, renal failure, and diabetes mellitis (Young et al., 2002). Understanding such a debilitating disorder is extremely important in order to enhance current medical therapies.

OSA events are caused by physical obstruction of the supraglottic airway during sleep, resulting in recurrent episodes of airflow reduction for 10 or more seconds, often accompanied by hypoxemia and hypercapnia. Recurrent arousals often follow these events with restoration of upper airway muscle tone and airflow (Okabe et al., 1994, Krug, P., 1999). Respiratory events during sleep are defined as apneas if airflow is reduced by more than 50% during the event and hypopneas if airflow is reduced by less than 50% and combined with a 3% fall in oxygen saturation or an arousal. (American Academy of Sleep Medicine Task Force., 1999)

The pathophysiology behind OSA is complex and is still under investigation. The role of mandibular movement in the pathophysiology of OSA has received little attention to date, despite the fact that mandibular advancing oral appliances are commonly used as a treatment for mild and moderate OSA. It is clear that jaw opening narrows the retroglossal airway (Kuna & Remmers, 1985) and increases upper airway collapsibility
but we remain uncertain of its role in the pathogenesis of airway occlusions during sleep. Preliminary published work with few subjects has demonstrated increased jaw opening with deepening sleep (Miyamoto et al., 1998), that patients with OSA have greater jaw opening in all sleep stages than normal subjects (Hollowell & Suratt, 1991), and that jaw opening changes with respiration – being greater on inspiration than expiration (Hollowell & Suratt, 1991). No study to date has measured anteroposterior jaw movement during sleep in combined multidimensional measurements of jaw movement..

The aim of this study is to describe anteroposterior and vertical movement of the mandible during sleep in young, normal adults. We hypothesize that at sleep onset, and as sleep deepens, jaw opening and jaw retraction will increase significantly, possibly as a result of gravity or sleep related muscular hypotonia. In order to investigate our hypothesis, we developed a novel method for accurately and reproducibly measuring mandibular position during sleep to within half a millimeter. The information this method has provided while studying twelve healthy subjects provides an essential baseline to understand the potential role of physiological changes in jaw position during sleep in the pathophysiology of OSA. Our methodology provides a powerful tool to compare jaw position during sleep between patients with OSA and normal controls, and between older subjects and healthy young subjects.
Chapter 2  
Literature Review

2.1 Normal Anatomy of the Upper Airway: An Overview

2.1.1 The Upper Airway

The upper airway is a hollow muscular tube that serves the important functions of speech, swallowing and ventilation. It is conventionally divided into three parts, the nasopharynx, oropharynx and laryngopharynx (the latter is also referred to as the hypopharynx. The nasopharynx extends from the turbinates to the posterior margin of the hard palate. The oropharynx begins at the proximal margin of the soft palate and extends to the base of the epiglottis (Arens & Marcus, 2004). The oropharynx is commonly viewed as having two sections: the retropalatal oropharynx (or the velopharynx), which lies behind the soft palate and extends from the proximal margin of the soft palate to the distal tip of the uvula, and the retroglossal oropharynx, which extends from the distal tip of the uvula to the base of the epiglottis (Arens & Marcus, 2004).

Upper airway diameter and length are defined by a surrounding bony enclosure consisting of the mandible, the maxilla, the hyoid and the cervical vertebrae. Changes in the relative positions of any of these structures can decrease pharyngeal diameter and increase pharyngeal length leading to increased airway compliance and resistance to airflow (Watanabe et al, 2002) (Figure 1).
The mandible, or jaw bone, is an extremely important structure in the anatomy of the upper airway because it serves as the attachment for many of the upper airway muscles, houses the tongue, and mandibular position greatly influences upper airway geometry (Isono et al., 2004). It is a mobile structure and moves in three planes – superoinferiorly, anteroposteriorly, and laterally. This movement is achieved through its articulation with the temporal bone of the skull at the temporomandibular joint. The temporomandibular joint (TMJ) consists of the articular tubercle and the mandibular fossa of the temporal bone, an articular disk, and the condyle of the mandible (Martini et al., 2003). During normal speech, the joint acts as a hinge with the condyle turning on a transverse axis within the articular disk. When the mouth opens more widely, the condyle and the articular disk slide anteriorly onto the articular tubercle, while the condyle turns within

![Diagram of the interaction between soft tissue and the upper airway bony enclosure.](image)

**Figure 1:** A schematic representation of the interaction between soft tissue and the upper airway bony enclosure on pharyngeal diameter. Bony enclosure size and the magnitude of soft tissue of the pharynx play a critical role in upper airway patency. (Watanabe et al., 2002) *Reprinted with permission from reference 87.*

2.1.2 The mandible and the temporomandibular joint

The mandible, or jaw bone, is an extremely important structure in the anatomy of the upper airway because it serves as the attachment for many of the upper airway muscles, houses the tongue, and mandibular position greatly influences upper airway geometry (Isono et al., 2004). It is a mobile structure and moves in three planes – superoinferiorly, anteroposteriorly, and laterally. This movement is achieved through its articulation with the temporal bone of the skull at the temporomandibular joint. The temporomandibular joint (TMJ) consists of the articular tubercle and the mandibular fossa of the temporal bone, an articular disk, and the condyle of the mandible (Martini et al., 2003). During normal speech, the joint acts as a hinge with the condyle turning on a transverse axis within the articular disk. When the mouth opens more widely, the condyle and the articular disk slide anteriorly onto the articular tubercle, while the condyle turns within
the articular disk. Mouth closure is simply the reverse motion. Jaw protrusion and retraction involves a gliding anterior and posterior motion, respectively, of the condyle and articular disk. Side-to-side motion of the jaw involves opposing rotation and gliding motions of both temporomandibular joints (Anatomy of the Human Body, Henry Gray, 1918). The interquartile range for hinge-axis rotation at maximum opening for normal subjects is between 29.09° and 34.87° (Piehslinger et al., 1993), consistent with a 3 – 4cm distance between the upper and lower incisors (Ayuse et al., 2004; Inazawa et al., 2005).

2.1.3 Muscular control of the mandible.
Inferior movement of the mandible is caused by gravity in the erect position, and is aided by four muscles: the platysma, the digastricus, the mylohyoideus, and the geniohyoideus. The latter three comprise the suprahyoid muscles and connect the hyoid bone to the mandible (figure 2). The hyoid bone serves as the origin for the muscles which are inserted in the mandible (Martini, et al., 2003). Superior movement of the mandible (jaw closure) is carried out by the masseter, pterygoideus internus and temporalis muscles, which also stabilize mandibular position against gravity (figure 3). Jaw protrusion involves both the pterygoidei internus and externus while retraction involves the masseter and the temporalis. Side-to-side motion of the jaw is caused by alternate contractions of the pterygoidei on either side (Martini, et al., 2003).
Figure 2: Anterior view of the neck showing the suprathyroid muscles connecting the hyoid and the mandible. Contractions of these muscles influence upper airway diameter and the relative positions of the mandible and hyoid bone. *Reprinted with permission from reference 23.*

Figure 3: Lateral view of the pterygoidei (left) and the temporalis (right) muscles. Contractions of these muscles influence mandibular position and stabilization. *Reprinted with permission from reference 23.*

2.1.4 Innervation of the muscles controlling mandibular movement

Innervation of the mandibular musculature is supplied by the craniofacial nerves. The masseter, temporalis, pterygoideus externus and pterygoideus internus are all supplied by the mandibular branch of the trigeminal nerve (V). The mylohyoideus and digastricus are supplied by the mylohyoid branch of the inferior alveolar nerve which originates from the
trigeminal nerve (V). The geniohyoideus muscle is supplied by the hypoglossal nerve (XII) (Martini, et al., 2003).

2.1.5 Common Upper Airway Morphology in Obstructive Sleep Apnea Patients

Anatomical factors such as abnormal upper airway morphology and enlargement of the soft tissue structures surrounding the lumen are directly linked to upper airway narrowing often found in Patients with OSA (Lowe et al., 1995). OSA severity is correlated with anthropometric measures (such as BMI and neck circumference) and anatomical anomalies such as a high-arched hard palate, retrognathism, a posterior placed palate, thick soft palate, elongated or thickened uvula, tonsil hypertrophy, septal deviation and turbinate hypertrophy (Zonato et al., 2005). These characteristics can lead to the smaller mouth, narrower oropharyngeal opening and smaller pharyngeal diameter characteristic of many Patients with OSA (White et al., 1995; Schwab et al., 2003). Retrognathism (posterior placement of the mandible) has been identified as a causative factor for OSA in many non-obese patients as it results in a smaller bony enclosure that increases pharyngeal tissue thickness and decreases lumen diameter (Figure 1). Hyoid bone displacement, which is also prevalent in Patients with OSA, lengthens the pharynx leading to increased resistance to air flow (Zonato et al., 2005). Soft tissue anomalies, such as tonsil and uvula hypertrophy, decrease pharyngeal diameter and increase the pharyngeal tissue thickness, thereby increasing the external closing pressures on the upper airway. While low incidences of these characteristics have been found in non-apneic patients, it is believed that the occurrence of OSA is caused by a combination of
three or more of these abnormalities that compromise airflow and decrease oropharyngeal
diameter (Zonato et al., 2005). It is, therefore, not surprising that closing pressures in the
upper airway are less subatmospheric in anesthetized patients with OSA than in age and
BMI-matched controls (Isono et al., 1997).

In obese patients, fat deposition in the neck is the predominant mechanism leading to
upper airway obstruction. Fatty tissue buildup within the bony enclosure of the pharynx
narrowed the passive pharynx leading to increased propensity for collapse (Kyzer &
Charuzi, 1998). The greater tissue thickness of the pharyngeal walls results in greater
extraluminal closing pressures exerted on the airway (Figure 1) (White et al., 1995).

Functional abnormalities of the upper airway dilating muscles have been observed in
OSA patients. The genioglossus, the main pharyngeal dilator muscle, remains an
important marker for upper airway muscular tone (Remmers et al. 1978). Its importance
in the maintenance of upper airway patency is clear; genioglossus activity increases late
during an apneic event (presumably to counter airway occlusion) and delayed
genioglossal activation prolongs the apneic event (Okabe et al., 1994) Previous
comparisons between age and BMI matched apneic patients and normal controls have
shown significantly greater upper airway dilator activity in patients with OSA in response
to negative pressure (Mezzanotte et al., 1996; Fogel et al., 2001) which is thought to be
due, in part, to an enhanced negative-pressure reflex (Eckert et al., 2007). Since both
negative pressure generation during normal inspiration and negative pressure loading
cause an increase in genioglossal activity, it is likely that a pharynx predisposed to
collapse may require higher than normal genioglossus activity to maintain airway patency
(Malhotra et al., 2000; Jordan et al., 2002). Inspiratory threshold loading and hypercapnia are both associated with an increase in both the genioglossus and the masseter suggesting a requirement for the jaw to be stable during airway dilation (Hollowell et al., 1991).

The amplified genioglossus activity while awake significantly decreases in apneic patients during stable sleep (Horner et al., 1994), which removes this compensatory mechanism. This reduction in genioglossus activity is thought to be due, at least in part, to an absence of the wakefulness stimulus (Fogel et al., 2001) and is paralleled by a loss of muscle activity (and likely a diminished negative-pressure reflex) observed at sleep onset (Horner et al., 1994; Mezzanotte et al., 1996). While this wakefulness stimulus is thought to be higher in Patients with OSA (Fogel et al., 2001), the reduction in dilator muscle activity is much greater upon sleep onset than in normal controls (Mezzanotte et al., 1996) indicating a greater reduction in neuro-muscular activity in the upper airway.

Impairment of the tonic and phasic contractions of the dilating muscles causes an imbalance between dilating and collapsing forces of the upper airway and can lead to compromised airway integrity. Unfortunately, repeated hypoxic events alters the muscle fiber composition of pharyngeal dilator muscles toward more fatigable, fast-twitch muscle fibers, reducing muscle endurance and compromising upper airway patency (Salmone & Van, 1991; Johal et al 2007)

### 2.2 Sleep Posture
Gravity plays an important role in the generation of apneas and snoring in normal individuals, leading investigators to postulate that sleep posture is important in determining the severity of OSA (Elliott et al., 2001). Examinations of pharyngeal
diameter in sleep disordered patients have shown a significantly greater pharyngeal narrowing in the supine position compared to the upright position, especially at the level of the velopharynx (Yildirim et al., 1991; Pae et al., 1994; Pracharktam et al., 1994). A similar study with normal subjects, however, showed no significant difference in nasal resistance (an indirect measure of pharyngeal compliance) between these two postures indicating a waking compensatory mechanism that is impaired in Patients with OSA (Okawara et al., 2004). Closing pressures (the cumulative forces promoting airway collapse) are greater in the supine position than in the lateral position, indicating a higher propensity for collapse during supine sleep (Penzel et al., 2001).

2.3 Sleep Stage
Muscular hypotonia accompanies sleep onset and may explain, at least in part, the greater propensity for airway collapse during sleep (Penzel et al., 2001). In addition, genioglossus activity is significantly greater during NREM than in REM sleep, indicating neural inhibition of upper airway dilator muscles in REM and a consequent greater propensity to upper airway collapse during REM sleep (Sullivan & Issa, 1980). Similar muscular hypotonia of masticatory muscles with sleep onset and REM sleep, although not studied to date, could result in increased jaw opening with resulting compromise of the upper airway diameter (see section 2.5).
2.4 Breathing Route and the Upper Airway

2.4.1 Anatomical and physiological differences between oral and nasal breathing.

Humans can breathe nasally, orally, or oronasally. Both oral and nasal breathing routes provide similar resistance to airflow during wakefulness provided that the individual is not breathing through a mouth-piece and panting with the glottis open (in which case the upper airway resistance is minimized) (Amis et al., 1999). At rest most individuals breathe nasally unless the nose is obstructed, but as ventilation increases the demands for increasing airflow force the individual to breathe orally with an upper airway configuration that minimizes upper airway resistance (Proctor, 1964). It is important to differentiate between mouth opening and mouth breathing - the mouth may be open wide and the individual may be breathing exclusively through the nose (Lieberman et al., 1990). The soft palate has been found to play a major role in determining breathing route by shepherding airflow via the oral airway (the soft palate closes off the nasopharynx) or the nasal airway (the soft palate closes off the oropharyngeal inlet) and, although soft palate movement is involuntary, the breathing route can be selected voluntarily while awake (Rodenstein & Stanescu, 1984). During oronasal breathing, the soft palate rises and falls to proportion oral and nasal flow during breathing. The soft palate position is probably governed by gravity, tongue position and soft palatal muscle contraction (Kuna & Remmers, 1985; Isoni et al., 1993). The position of the soft palate is important as it is a potential site of airway obstruction (Rodenstein & Stanescu, 1984).
2.4.2 Oral breathing and airway calibre.

There are several pieces of evidence that support the concept that oral breathing route is an important factor in the pathogenesis of obstructive sleep apnea:

1. Obstructive sleep apnea is more common among subjects who complain of nasal congestion due to allergy (Young et al., 2001).

2. Nasal obstruction is more common among snorers and patients with OSA than in normal subjects (Desfonds et al., 1998; Lofaso et al., 2000; Verine et al., 2002).

3. Obstruction of the nasal airway with gauze, balloons or tape markedly increases the severity of OSA and can induce OSA de novo among normal subjects (Zwillich et al., 1981; Lavie et al., 1983; Suratt et al., 1986; Fitzpatrick et al., 2003b).

4. Naturally occurring nasal obstruction in patients with allergic rhinitis is associated with an increased frequency of obstructive apneas during sleep, and these resolve when the nasal resistance falls back to normal outside the allergy season (McNicholas et al., 1982).

5. During wakefulness, the cross sectional area at the level of the velopharynx decreases significantly with oral compared to nasal breathing (Burger et al., 1998). This is countered by an increase in cross-sectional area at the level of the oropharynx due to compensatory rise in genioglossal activity (Burger et al., 1998).
Oral breathing often occurs in combination with mouth opening. Genioglossal activity increases with jaw opening (Lowe et al., 1977) to compensate the increased propensity for upper airway collapse associated with an infero-posterior movement of the mandible. (Meurice J-C et al., 1996). Any diminution of this reflex during sleep would likely predispose individuals to upper airway obstruction as a result of mouth opening.

2.4.3 Breathing route during sleep
During wakefulness, oral and nasal breathing routes have similar resistance to airflow in both the upright and supine positions (Amis et al., 1999) but during supine sleep the upper airway resistance is substantially higher while breathing orally and there is a marked increase in upper airway collapsibility as compared with nasal breathing in normal subjects (Fitzpatrick et al., 2003b). This likely explains why normal subjects breathe primarily through the nose during sleep (Fitzpatrick et al., 2003a). If mouth breathing during sleep is associated with increased mouth opening, this may be one factor predisposing to upper airway obstruction in mouth breathers.

2.4.4 Nasal Resistance during Oral Breathing
Although there are several methods of measuring nasal resistance, it is usually measured as the resistance to airflow between the anterior nares and the posterior nasopharynx by a small catheter placed transnasally 8 cm from the anterior naris – this technique is called posterior active rhinometry because the resistance is measured as the subject is actively
inhaling. Nasal congestion such as allergic rhinitis predisposes individuals to OSA (Young et al., 1997) and, experimental occlusion of the nose induces sleep apnea in normal subjects during sleep. (Lavie et al., 1983; Zwillich et al., 1981) High nasal resistance significantly increases the incidence of snoring, respiratory disturbances and oxygen desaturations during sleep in Patients with OSA, indicating the importance of unobstructed nasal breathing in sleep (Li et al., 2005).

Mechanical, surgical and pharmaceutical therapies to alleviate congestion do not significantly reduce OSA severity in patients with nasal obstruction although they improve the Apnea-Hypopnea Index (the number of apneas and hypopneas per hour) in allergy patients. (Dayal & Phillipson, 1985; Braver & Block, 1994; Todorova et al., 1998; Friedman et al., 2000; Schonhofer et al., 2000; Verse et al., 2002) A subgroup of Patients with OSA with absent pharyngeal narrowing responded favourably to nasal surgery (Series et al., 1993). Pharmaceutical treatment for nasal obstruction in patients with OSA with a normal retroglossal area reduces mouth breathing and apnea severity although it does not cure the disorder (McLean et al., 2005). Even in the presence of severe nasal obstruction, subjects continue to breathe partially through their nose implying a physiological disadvantage to pure oral breathing. While decreasing the amount of time spent breathing orally during sleep, habitual jaw opening may have inhibited the effectiveness of these pharmacological treatments and rendered the upper airway more collapsible (Meurice J-C et al., 1996). This idea is supported by research showing that jaw opening is greater during obstructive events than during normal sleep.
(Miyamoto et al., 1998) and patients with OSA, on average, consistently have greater jaw opening during sleep compared with normal controls (Hollowell & Suratt, 1991).

### 2.5 The effect of mandibular movement on the upper airway calibre

#### 2.5.1 Jaw opening and the airway

Limited information is available on the effect of jaw position on airway calibre. During mouth opening, the mandible travels inferiorly and posteriorly, much like a trap door opening, bringing it closer to the hyoid and the posterior pharyngeal wall respectively. This inferoposterior mandibular movement thus results in anatomic narrowing of the retroglossal oropharynx with a corresponding increase in respiratory resistance in much the same way as neck flexion does (Choi et al., 2000). A compromise in the retroglossal airway size, in association with jaw opening, increases the propensity of upper airway collapse (Meurice J-C et al., 1996).

#### 2.5.2 Jaw protrusion and the effects on the airway

In awake OSA patients, and in controls, tongue and jaw protrusion greatly increase the cross sectional area (CSA) of the pharynx (Ferguson et al., 1997). Tongue protrusion increases the cross sectional area of the upper airway more than jaw protrusion and only tongue protrusion increases the CSA of the airway at the level of the velopharynx (soft palate). As the tongue attaches to the mandible, protruding the jaw pulls the tongue anteriorly away from the posterior wall of the pharynx increasing the CSA of the upper airway (Ferguson et al., 1997).
Several varieties of oral appliances have been developed to treat OSA. These devices hold the mandible forward and prevent inferior-posterior movement during sleep, consequently increasing upper airway diameter and decreasing compliance. (Schmidt-Nowara et al., 1991; Gale et al., 2000; Battagel et al., 2002) Their effect on upper airway geometry has not received detailed attention, but an increase in jaw protrusion by a two-thirds of maximum causes an increase in the CSA of the velopharyngeal without significant change in the CSA of the hypopharynx, resulting in a significant reduction in OSA severity (Lowe et al., 2000). This forward displacement of the mandible pulls the tongue, and with it, the soft palate, forward increasing the anteroposterior distance of the velopharynx and improving nasal airflow dynamics (Okawara et al., 2004). The magnitude of pharyngeal opening after mandibular advancement is variable, although this may be due to differences in experimental procedure and degree of mandibular advancement. Battagel et al., 2002, found only minor improvements to oropharyngeal diameter, however, they found a significant decrease in the hyoid-mandible distance (due to the upward and forward repositioning of the hyoid), which causes a decrease in pharyngeal length and improvement of upper airway resistance to airflow.

The magnitude of jaw protrusion influences the therapeutic effectiveness of oral appliances used to treat OSA; the greater the jaw protrusion, the greater the therapeutic efficacy (Walker-Engstrom et al., 2003). In fact, step-by-step protrusion of the jaw using an oral appliance causes a ‘dose-dependent’ reduction in closing pressures of the passive pharynx and reduction in both nocturnal oxygen desaturations (Kato et al., 2000) and sleep apnea severity (de Almeida et al., 2002). On average, jaw protrusion with an oral
appliance, to two-thirds of the maximum protrusion possible, consistently decreases OSA symptoms, likely due to increased anteroposterior area of the pharynx (Tsuiki et al., 2003).

Oral devices also influence upper airway physiology by increasing muscular tonicity. Jaw protrusion significantly increases the tonic activities of the genioglossus, genhiohyoid and masseters above baseline during wakefulness as well as during sleep (Yoshida, 1998; Tsuiki et al., 2000; Johal et al., 2007). By increasing tonicity of the pharyngeal dilator muscles, oral devices further decrease airway collapsibility. The recruitment of masseter activity upon protrusion aids the stabilization of the mandible following activation of the genioglossus activity, allowing for more effective pharyngeal dilation (Hollowell & Suratt, 1991).

Maximal inspiratory airflow decreases in both normal subjects and patients with OSA on moving from the upright to the recumbent position (Masumi et al., 1996), but it is almost fully restored to normal in the supine position when the jaw is protruded. Inspiratory airflow almost fully recovers when the jaw is protruded in the supine position (Masumi et al., 1996). This recovery may have been due to advancement of the tongue with jaw protrusion, increasing the space between the posterior pharyngeal wall and the soft palate and tongue.

Changes associated with long term use of oral appliances are increased palatal length and anterior face height due to depressed condyle position within the temporomandibular joint (Robertson, 2001). These changes in bony structure, which involve depressing the
angle of the mandible and sliding it anteriorly away from the posterior wall of the pharynx, increase the diameter of the passive airway.

Oral devices are not effective on everyone. Comparison between responders and non-responders to oral protrusion therapy show a correlation between BMI and original pharyngeal diameter with its effectiveness (Otsuka et al., 2006). Pharyngeal fatty tissue buildup in the pharynx as the result of obesity may decrease the effectiveness of jaw protrusion as it remains an even greater problem for upper airway compliance even when the bony enclosure size is returned to normal through therapeutic intervention (Otsuka et al., 2006). A very large airway CSA during wakefulness may indicate enhanced compensatory activity during wakefulness that during sleep, jaw protrusion may not be enough to restore upper airway patency. Non-responders, with an equal amount of mandibular advancement as responders, show no significant forward displacement of the anterior wall, indicating that the primary therapeutic effect of oral devices is enlarging the CSA of the upper airway (Tsuiki et al., 2000).

2.5.3 Jaw abnormalities and airway caliber

Non-obese patients with OSA have, on average, a smaller anteroposterior distance from the mandible to the cervical vertebrae (67.6±6.9mm from the symphysis of the mandible to the anterior surface of the C3, C4 vertebrae) than normal subjects (76.2±8.2mm from the symphysis of the mandible to the anterior surface of the C3,C4 vertebrae), which leads to decreased anteroposterior upper airway diameter (Sakakibara et al., 1999). The severity of sleep apnea is correlated to the degree of airway compromise (Sakakibara et
al., 1999), although it is well known that both anatomic and functional abnormalities play a major role in the pathogenesis of OSA (White, 2005).

2.6 What we know about jaw movement during sleep

2.6.1 Jaw position and sleep stage

Very little is known about jaw movement during sleep as most researchers investigate the effects of jaw position on airway patency in awake subjects. Nevertheless, vertical mandibular position is significantly affected by sleep stage as reported by Miyamoto et al., 1998 (Figure 4). Miyamoto found that jaw opening progressively increases from wake to deep NREM sleep, which may indicate a parallel decrease in tone of the stabilizing muscles. While jaw opening is significantly wider during REM sleep than during quiet wake and stage 1 sleep, it is not significantly different from the jaw position during the deep NREM sleep stages (Miyamoto et al., 1998). This same study shows that jaw position in lateral as compared with supine sleep is not significantly different (Miyamoto et al., 1998).
2.6.2 Jaw position and sleep posture

There has been little investigation into the effect of sleep posture on jaw movement during sleep. Clearly, the use of mandibular advancement devices increases inspiratory airflow and reduces upper airway resistance in patients with OSA and in normal subjects. (Masumi et al., 1996) Gravity may play an important role in jaw position during sleep when the muscles governing normal jaw posture become hypotonic, as one might expect during sleep, and particularly during REM sleep. The marked increase in sleep apnea severity with supine sleep (Cartwright, 1984; Braver et al., 1995), particularly in non-obese patients with OSA, provides circumstantial evidence in support of this concept. The fact that sleeping at 60° upright reduces sleep apnea severity (Braver & Block, 1994) also supports the concept of the gravitational influence on the upper airway. However,
whether this gravitational influence on upper airway collapsibility is mediated through jaw position or through soft tissue abnormalities directly is unclear.

### 2.6.3 Jaw Position in Normals vs Patients with OSA

Patients with OSA demonstrate significantly wider mouth opening than normal subjects at both end-inspiration and end-expiration, with even greater mouth opening during apneas (Figure 5) (Hollowell & Suratt, 1991), that narrows the pharynx and increases its propensity for collapse (Meurice J-C *et al.*, 1996). Patients with OSA are found to have greater masseter and submental activation during apneas suggesting an increased effort to stabilize the mandible and, possibly, to reduce upper airway collapsibility by optimizing the effects of submental muscle contraction (Hollowell & Suratt, 1991).

**Figure 5:** Magnitude of jaw opening (mm) in normal and Patients with OSA at end expiration and end inspiration during apneas. Jaw opening was significantly greater in Patients with OSA at both end inspiration and end-expiration in both stage 2 and REM sleep.* - p<0.05, ** - p < 0.01. (Hollowell & Suratt, 1991). *Reprinted with permission from reference 28*
2.7 Limitations

2.7.1 General Limitations to Studying Natural Sleep

Obtaining accurate information about OSA in humans is difficult due to the varied ability of the subjects to sleep during the invasive procedures and the experimenters’ ability to reproduce natural sleep in experimental conditions. Many of the experiments on upper airway collapsibility, such as those requiring the Muller maneuver to mimic airway collapse) require voluntary actions performed by an awake subject. Selecting for specific age, gender, BMI, cephalometric abnormalities or OSA severity can also be a challenge in studies requiring compliance. Randomization in these instances can be difficult.

Sleep scoring uses specific EEG markers to determine sleep stage. While this is reliable, it can be difficult to determine the exact time of sleep onset, the onset of stage 1, the onset of stage 2. This can make conclusive observations based on changes in sleep stage can be difficult. Many researchers must rely on data obtained during stable NREM or REM sleep. It can also be difficult to use natural sleep when investigating mechanical causes for airway occlusions.

2.7.2 Previous Limitations to studying Jaw Movement and our Solutions

In general, previous studies failed to examine the horizontal and vertical components of jaw opening and, therefore, are unable to offer insight into the effect of muscular hypotonia on the magnitude of jaw opening and retraction in the supine position. Development of new methodology to accomplish this is critical if jaw position is to be recorded accurately in all three dimensions during sleep.
One of the purposes of this thesis was to design equipment to measure jaw position that would have minimal influence on jaw opening and not limit jaw movement in any direction. Side-to-side motions of the jaw can potentially influence the recorded magnitude of jaw opening by increasing the distance between the upper and lower incisors without there being a significant increase in mouth opening. Previous studies have defined the magnitude of jaw opening as the distance from a single point on the face (for example the upper incisors) to a single point on the mandible (for example the lower incisors). In these instances, side to side motions of the jaw that move the lower incisors laterally away from the upper incisors may independently be recorded as jaw opening. Our goal was to map the position of the jaw in three dimensional space so that the magnitude of jaw opening is not affected by the magnitude of lateral movement.

2.7.3 Our current study

We developed an innovative new technology that has allowed us to measure jaw opening in its vertical and horizontal components simultaneously with overnight polysomnography in the supine position in 12 male and female subjects. We hypothesize that, as sleep deepens, jaw opening and jaw retraction will increase as a result of gravity and sleep related muscular hypotonia which could increase the propensity for upper airway collapse in OSA. Our data will provide baseline data that will be useful in future studies that focus on understanding the role of jaw position in OSA.
3.1 Subjects

Twelve healthy subjects, 6 males [age 23 ± 7 (mean ± SD) years (range 19-38 years), BMI 24.7 ± 3.9 kg/m² (range 21.8-29.6 kg/m²)] and 6 females [age 22 ± 6 (mean ± SD) years (range 19-35 years), BMI 20 ± 2.0 kg/m² (range 18.1-22.9 kg/m²)] were studied. Each underwent a single overnight polysomnogram in the supine sleeping position at the Kingston General Hospital Sleep Laboratory. Each subject underwent an initial in-person interview to ensure they met the inclusion criteria. The inclusion criteria for the study were: 1) normal circadian sleep cycle; 2) nasal resistance within the normal range (<5cmH₂O/Ls); 3) healthy well-aligned incisors (for attachment of the jaw movement measuring device). The exclusion criteria included: 1) any history of sleep disorders or respiratory diseases; 2) symptomatic nasal obstruction; 3) history of bruxism, 4) use of medications and/or substances known to alter sleep duration or architecture (antidepressants, analgesics, hypnotics, stimulants); 5) temporomandibular joint (TMJ) abnormalities (arthritis, pain, dislocation); 6) the use of removable dentures in the upper or lower jaw. Only those subjects that met these inclusion criteria were selected to participate in our study. One individual was also excluded because the limited elasticity of his lower lip resulted in pressure on the dental platform, making it impossible for the platform to remain fixed to the teeth. No data was collected from this subject.
3.2 Nasal Resistance.

Nasal resistance was measured in the erect seated and supine positions by posterior active rhinometry 2 hours prior to, and immediately following, each overnight sleep study.

The procedure for posterior active rhinometry was conducted as previously described (Fitzpatrick et al., 2003a). An infant nasogastric feeding catheter (6 French diameter - MED-Rx Benlan Inc, Oakville Ontario) was lubricated and inserted through the an unobstructed nostril until it was visible at the pharynx on mouth opening. The distal catheter tip was then retracted 1.5 cm above the free margin of the soft palate. The proximal end of the cannula was attached to a calibrated differential pressure transducer (Ultima Dual Pressure sensor - model 0585- Braebon Medical Corporation, Kanata, Ontario. A CPAP (continuous positive airway pressure) mask was placed over the patient’s nose, taking care to ensure that there was no compression of the nasal airway by the mask (by monitoring the posterior nasal pressure before and after attachment of the nasal mask), and ensuring that there was no air-leak from the mask. A heated pneumotach (3700 series, Hans Rudolph Inc., Kansas City, USA) was placed at the outlet of the CPAP mask, and the patient was instructed to breathe quietly through the nose only, with the lips closed. The pressure at the anterior nares, provided by the measured pressure within the nasal CPAP mask, provided the reference pressure for calculation of the differential pressure across the nasal airway. Each pneumotach was calibrated with a 3 litre syringe to an accuracy of .015L before each study. Nasal resistance was measured as the change in pressure (cm H2O) across the nose for a standardized inspiratory flow rate of 0.3 L/s (18 L/min). (Fitzpatrick et al., 2003a)
3.3 Overnight Polysomnography

Subjects underwent a single overnight sleep study at the Kingston General Hospital Sleep Laboratory, Ontario using the usual clinical polysomnographic montage (Channels: 4 Electroencephalogram (EEG), 2 electrooculogram (EOG), 2 chin electromyogram (EMG), 2 electrokardiogram (EKG) oronasal airflow, thoracoabdominal movement, intercostal EMG, pulse oximetry, right and left anterior-tibialis EMG. Data from the jaw measurement device (see below) were interfaced with the polysomnographic montage (Sandman; Mallinckrodt, Ottawa, Canada).

For measurement of the jaw position in wakefulness, only ‘clean’ epochs of quiet wakefulness (defined for this purpose as the subject appearing relaxed and with no body movement or talking) immediately preceding (within 3 minutes of sleep onset) were included in our analysis. Data from stages 3 and 4 sleep were classified under slow wave sleep (SWS). Epochs of 30 second duration were standardized for sleep stage and those containing arousals, movement, or apnea/hypopnea events were excluded. The data acquisition sampling frequency of jaw movement was 64 Hz and jaw movement data from the first 10 seconds (representing approximately 2 or 3 breaths) of all clean epochs were analyzed.

Subjects were asked to sleep in the supine position only. To ensure the head remained in the supine position (and did not turn laterally during sleep), two rolled towels were positioned at either side of the head for support. This provided a recess in which the head could rest but did not apply any pressure to the head, neck, mandible or the apparatus. Each subject was allowed one pillow.
Simultaneous video recordings were gathered throughout the night to confirm subject and apparatus position.

3.4 Measuring Jaw Position

3.4.1 Equipment design Requirements

We developed and tested several different methods to measure jaw position during sleep, beginning with a device using magnets and hall sensors in an experimental design similar to that of Miyamoto et al, 1998. None of these methods was successful – each of these iterations and the associated shortcomings are outlined in Appendix A.

We defined the requirements for an apparatus to measure jaw position as: 1) that it be securely mounted on teeth and/or the bony parts of the face (the temples and the bridge of the nose) for stable measurements of jaw movement without interference from head, neck or facial movements or stretching of the skin; 2) that sensors have sufficient sensitivity to detect changes in jaw position with a resolution of 0.5mm; 3) that sensors retain calibration throughout an eight hour period with little or no reproducible error from night to night; 4) that it be light-weight to minimize the risk of damage to the teeth and any unwanted influence on jaw position, 5) that the sensors remain accurate through the full normal physiological movement range (from teeth clenched to jaw wide open – inter-incisor distance of 5cm); and 6) that the apparatus be sufficiently comfortable to allow the subject to sleep undisturbed in the supine position.
Specific problems that had to be overcome in designing the apparatus included: 1) electrical interference in the sleep laboratory environment; 2) fixation of the device to the head/face where skin elasticity would have little or no effect on the stability of the measurements; 3) mandibular movement from the clenched position to full jaw opening required attachment of the device directly to the teeth and not the lips; and 4) avoidance of salivary contamination of the sensors (interfere with sensor function). All of these issues were accounted for, and resolved before data collection.

3.4.2 Equipment Design – Final iteration “The Tiara”

Figure 6: Lateral view of the “The Tiara” in situ illustrating its components. Arrows point to adjustment screws on either side of the support extension, which allow for attachment and the modification of the instrument to the features of each subject.
The jaw movement measurement apparatus is divided into 3 separate, yet equally important structures: the support, the sensor brace, and the mandibular attachment.

3.4.3 The Support
The support structure of the apparatus, which holds the sensors, is made from brass rods which extend anteriorly from the ears and pass in front of the face, ending in a 2.5 cm long brass rod extending out from the bridge of the nose. Screws at either end of this extension (see figure 6) act to attach the support to the sensor brace and individualize the apparatus to each subject’s face.

The support, designed to fit like a pair of eye glasses, attaches to the face at 3 points: the bridge of the nose, and both temples. Securing the device on the head with a thick elastic band provides a stable foundation for the apparatus. To prevent any shifting of the apparatus during the night, the platforms are cemented in place using spirit gum and latex tape.

3.4.4 The Sensor Brace
The sensor brace portion of the instrument as shown in Figure 7 is very mobile and very light. The apparatus, in total, weighs approximately 13g but the weight distributed to the mandibular platform could still influence jaw opening during the experiment. To reduce this effect, we added a counterbalance (see Figure 7) to the sensor brace to remove all but 0.3g of weight from the mandible. The sensor brace also includes three light brass rods (A, B, and C), two polystyrene rods (extending to the mouth), and three sensor
complexes. The position of the mandible is defined by the three angles located at the junctions between the rods.

**Figure 7:** Lateral view of the sensor brace. Brass Rods A, B and C support the sensors and the Polystyrene rods connect the sensor brace to the mandibular attachment. The counter balance consists of a series of brass plates attached to the junction between rods B and C which offsets most of the weight of the appliance from the mandible.

The rods are positioned, using the screws indicated in Figure 7, on all subjects so that rod B is parallel to an imaginary line running from the tip of the nose to the tip of the chin, and rod A is directly above rod C. This positions the apparatus on the face where the effect of the counterbalance is optimized, and the angles created by rods A,B, and C, do not exceed the angular range of the sensor complexes even with large jaw movements (further explanation below). When positioned correctly, the subject feels no weight on
the mandible, no resistance against jaw closure and no propensity towards jaw opening. The subject can move his or her jaw freely in the lateral, anteroposterior and vertical directions.

Each sensor complex, located at the angular junctions between the brass rods, contains two round Wondermagnets® nickel plated Neodymium Iron Boron disk magnets (¼” diameter, 1/8” thick) fixed with epoxy on either side of a Honeywell© Angular Displacement Sensor (Model HMC 1501). Each sensor can resolve the angular direction of the magnetic field across the sensor to 0.07°, as long as the displacement of the magnetic field does not exceed 45° in either direction, and convert this distance into a voltage. Careful positioning of the sensor brace using specific landmarks on the face (as described above) positions the magnets close to 0° of displacement around the sensor at each angle. When the angle at each brass joint changes with mandibular movement, the magnets rotate proportionately causing a corresponding change in the direction of the magnetic field and thus a change in its output voltage. Please note that the sensors do not measure magnetic field strength but rather the magnetic field direction.

3.4.5 The Mandibular Attachment

The attachment as seen in Figure 8 consists of a carbon rod embedded in a rectangular plexiglass platform. The plexiglass platform is attached to the teeth with zinc phosphate cement (Shofu Hy-Bond®), a hard, brittle substance that permits no movement of the platform on the teeth. A detachable magnetic connection on the anterior surface of the carbon rod (made from 2 Wondermagnets® nickel plated Neodymium Iron Boron disk
magnet (1/8” diameter, 1/16” thick) allows the mandibular attachment to be disconnected from the sensor brace during setup and removal.

![Figure 8](image.png)

**Figure 8:** Lateral view of the mandibular attachment. The plexiglass platform is cemented to the lower incisors with zinc phosphate paste and a carbon rod extends out of the mouth to attach (with magnets, indicated by the black arrow) to the polystyrene end of the sensor brace.

### 3.4.6 Measuring Jaw Position

Using the sensor voltage output from each angle and a sampling frequency of 64 Hz, we can collect a complete map of jaw movement in spherical coordinates (Figure 9, Figure 24) throughout an entire sleep study.

The voltage output from each of the three angular magnetic sensors is converted into degrees for the corresponding angle. As the maximum voltage output for each sensor changes with temperature (a 0.32% decrease for each 1°C increase in temperature), this was corrected for the ambient study conditions. These angular measurements were used to find the radius of each spherical coordinate. Using the cosine law, spherical coordinates can be converted into conventional Cartesian coordinates to separately
determine the precise anteroposterior, vertical and lateral jaw position with less than 0.5mm error. The calculation of jaw position in each of the three dimensions from the angular voltages provided by the sensors is outlined in Appendix B.

Figure 9: Angular measurements by the jaw movement apparatus. Lateral view of the mandibular attachment showing the direction of angle movement with jaw movement. \( \phi \) – The angle measured by sensor 1; \( \delta \) – The angle measured by sensor 2; \( \varepsilon \) – The angle measured by sensor 3. These angles describe jaw position in spherical coordinates.

3.5 Biocalibrations

All equipment was tested prior to each overnight sleep study using the calibration and trouble-shooting guidelines required for each device. The jaw position measurement
apparatus was calibrated each night by obtaining the clenched position (used as the origin for all ensuing jaw movements), 1 cm jaw opening and 2 cm jaw opening (Figure 10). The latter two measurements were taken using 1 cm high Lego® blocks placed at the incisors.

![Device Biocalibrations](image)

**Figure 10:** Device Biocalibrations. Raw data demonstrating the biocalibration of the measurement apparatus. The subject was asked to decrease jaw opening by 1 cm increments using Lego® blocks, then protrude/retract his or her jaw, and finally to move their jaw laterally from side-to-side. Channels shown: 4 electroencephalograms (EEG); 2 electrooculograms (EOG); electromyogram (EMG) positioned over the genioglossus; intercostal EMG (IEMG); leg EMG (LEMG) positioned above the left and right anterior tibialis; oral flow (ORAL); Nasal Flow (NASAL); abdominal movement (ABDO); thoracic movement (THOR). Jaw position was mapped with angle 1 (ANGLE Ø), angle 2 (ANGLE δ), and angle 3 (ANGLE ε).
3.6 Statistical Analysis

All jaw movement was referenced to the clenched position, where we assumed the magnitude of jaw opening and jaw retraction was zero. Jaw position for each individual was collected from all ‘clean’ epochs of each sleep stage and a mean lateral, anteroposterior and vertical jaw position was calculated (See Appendix B). Due to non-normal distribution of data, a global test for overall difference in vertical, anteroposterior and lateral position between sleep stages was conducted using Friedman’s one-way ANOVA for paired samples blocked by subject. Significant differences in vertical, anteroposterior, or lateral jaw positions between sleep stages, a pair-wise comparison using the Wilcoxon signed-rank test, was used to compare jaw position between each sleep stage. All tests were two-tailed and are reported without adjustment for multiple comparisons. The tests were performed using SAS version 9.1 (SAS institute Inc, Cary, NC).
Chapter 4

Results

4.1 Subject and Sleep Information

All subjects had nasal resistances within normal limits immediately prior to overnight polysomnography and at 0600 following study termination (mean 3.24 ± 0.67 cmH$_2$O/L/s). Table 1 displays the demographic and lung function data collected from each subject.

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<th>Weight (kg)</th>
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<th>FEV$_1$ %Pred</th>
<th>FEV$_1$ (L)</th>
<th>FVC (L)</th>
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Table 1: Description of subject demographics and respiratory function in means and standard deviations (St.Dev). Body Mass Index (BMI); Forced Expiratory Volume at 1 second (FEV$_1$); Forced Vital Capacity (FVC). All pulmonary function data were obtained using a spirometer and expressed as a percent of the predicted value calculated by the spirometer for the individual’s size, weight, age and sex.
Data from the first sleep cycle were used to examine changes in mandibular position with sleep stage. Few subjects achieved more than one full sleep cycle during the sleep study – possibly because of the experimental conditions requiring supine only sleep and with the recording apparatus in place.

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Table 2: Distribution of sleep variables during the overnight study. Sleep Onset Latency (SOL) – The duration of time between commencement of sleep recording to the first epoch of stage 1 sleep; REM Latency (REM-L) - The time from sleep onset to first epoch of REM sleep; Total Time of First Sleep Cycle (TTFS) – The duration of the first sleep cycle and, for most, the total duration of sleep; Sleep Efficiency (Eff) – The percent of time the subject was asleep for the duration of the sleep study. The amount of time spent awake (Wake) in stage 1, stage 2, SWS or REM sleep is described as a percent of the total sleep time.
All subjects were asked to refrain from any caffeine intake the day before the study, alcohol intake 2 days before the study, and the use of any non-prescription medication (such as nasal decongestants) before the study, which may affect the nasal resistance tests or interfere with natural sleep.

4.2 Vertical Jaw Position vs Sleep Stage
The changes in jaw position with sleep stage are outlined in Table 3 and Figures 12. Average vertical jaw opening from the clenched position was calculated for each sleep stage. At sleep onset (defined as transition from wake to stage 1 sleep), jaw closure was observed in 83% of subjects, jaw opening was observed in a female subject, and no change in jaw position was observed in a male subject. As NREM sleep deepened (progression from stage 1 to stage 2 and then to slow wave sleep), jaw opening increased in all 12 subjects. Mean jaw opening increased from SWS to REM sleep in 10 subjects, and decreased in 2 subjects. These inconsistencies were not observed in the same individuals. Friedman’s one-way ANOVA blocked on subject demonstrated significant jaw opening with sleep stage (p<0.001).
Table 3: Average vertical distance from clenched in each sleep stage for all 12 subjects. This was calculated by averaging the positions measured in all clean epochs of each sleep stage. For further details, see Section 3.6.

The median (range) values for vertical jaw opening were; wake [3.1mm (0.8-14.2mm)], stage 1 [2.2mm (0.3-12.8mm)], stage 2 [5.3mm (0.9-13.7mm)], SWS [5.9mm (0.9-14.4mm)], and REM [8.9mm (1.0-20.4mm)]. Inter-individual variability increased as NREM sleep deepened. Pair-wise comparisons using the Wilcoxon signed-rank test between each sleep stage showed a significant difference (p<0.05) in jaw position between all sleep stages (Figure 11).
Figure 11: Box-plot showing the distribution of vertical jaw position for all subjects during each sleep stage. The horizontal line within each box represents the median value, and the + represents the mean. All pair-wise comparisons showed a significant change in jaw position of at least $p<0.05$ between all sleep stages. Wake vs Stage one ($p=0.0025$); Stage one vs Stage 2 ($p=0.0147$); Stage two vs SWS ($p=0.0042$); SWS vs REM ($p=0.0250$)

The magnitude of jaw retraction from the baseline clenched jaw position was calculated for each sleep stage. At sleep onset, jaw protrusion was observed in 11 of the 12 subjects, while jaw retraction was observed in the same female subject who exhibited jaw opening during this period. As NREM sleep deepened, jaw retraction increased in all but one male subject whose mean anteroposterior jaw movement range during sleep was minimal - less than half a millimeter. Jaw retraction was greatest during REM sleep in 10 of the 12 subjects (consistent with their greater jaw opening during this stage), while one subject demonstrated greater jaw protrusion in REM than in SWS. A global
A statistical comparison of anteroposterior jaw position across sleep stage using Friedman’s test blocked on subject showed significant jaw retraction with sleep stage (p<0.001) (Figure 12).

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Table 4: Average Anteroposterior distance from clenched in each sleep stage for all 12 subjects. This was calculated by averaging the positions measured in all clean epochs of each sleep stage. For further details, please see Section 3.6.

We determined the median of the mean anteroposterior jaw position [median (range)] for wake [1.4 (0.3-10.6)], stage 1 [1.0 (0.0-9.1)], Stage 2 [3.0 (0.5-9.5)], SWS [4.1 (0.5-11.5)], and REM [5.0 (0.6-16.8)]. All medians were smaller than the mean jaw position, indicating that the data were skewed to the more retracted position. Inter-individual variability increased as sleep deepened and individuals who exhibited the greatest amount of jaw opening tended to exhibit the greatest amount of jaw retraction, as expected. Pairwise comparisons using the Wilcoxon signed-rank test showed a significant difference (p<0.05) in jaw retraction between all sleep stages except between SWS and REM sleep (p > 0.05).
Figure 12: Box-plot showing the distribution of anteroposterior jaw position (mm) for all subjects during each sleep stage (higher values indicate a greater degree of jaw retraction). The horizontal line within each box represents the median value, and the + represents the mean. All pair-wise comparisons showed a significant change in jaw position of at least p<0.05 between all sleep stages. Wake vs Stage one (p= 0.024); Stage one vs Stage 2 (p= 0.01); Stage two vs SWS (p= 0.011); SWS vs REM (p= 0.077)

4.3 Lateral jaw movement vs Sleep stage

Friedman’s test showed no significant difference in lateral jaw position across any sleep stages. (p = 0.41). This was confirmed with video monitoring, which showed that changes in lateral jaw position coincided only with head movement. Small lateral
displacements in jaw position were in the direction of gravitation pull. However, there was no net difference in lateral jaw position with sleep stage (Figure 13).

**Figure 13:** Box-plot showing the distribution of anteroposterior jaw position for all subjects during each sleep stage. The line across the middle of each box represents the median value, and the + represents the mean. All pair-wise comparisons showed a significant change in jaw position of at least p<0.05 between all sleep stages. Wake vs Stage one (p= 0.26); Stage one vs Stage 2 (p= 0.66); Stage two vs SWS (p= 0.60); SWS vs REM (p= 0.74)
Chapter 5
Discussion

5.1 Discussion Summary

We developed and tested a novel method for measurement of jaw position during sleep. The method had an accuracy of 1.5 mm during sleep in humans. To our knowledge, this is the first time simultaneous measurements of jaw position in 3 movement planes have been reported during sleep. We discovered that vertical and anteroposterior (but not lateral) jaw position were significantly affected by sleep stage in the supine position. The transition from wake to sleep was associated with significant jaw closure and protrusion. As NREM sleep progressed to deeper stages, jaw opening increased and, as would be expected, a greater degree of jaw retraction was also observed. Maximum jaw opening and retraction was observed during REM sleep.

These findings may be important physiologically and clinically. The greater jaw opening and retraction observed during SWS and REM could explain why snoring tends to be maximal in those sleep stages (Hoffstein et al., 1991; Hill et al., 2000; Choi et al., 2001). Similarly, the maximal jaw opening and retraction observed during REM sleep would result in upper airway (retroglossal) narrowing and thus accentuate upper airway collapsibility – a finding consistent with the known increase in propensity to upper airway collapse during REM sleep. I did not measure upper airway muscle activity and, therefore, cannot address the mechanism responsible for change in jaw position with sleep. However, these findings would be consistent with the anticipated impact of progressive hypotonia of the postural muscles during sleep, which is maximal in REM,
with an associated gravitational change in jaw position. The literature on upper airway muscle activity during sleep is not entirely consistent, but several authors have demonstrated reduced upper airway muscle tone with NREM sleep (Mezzanotte et al., 1996; Fogel et al., 2001).

Our findings provide a more accurate picture of jaw position during sleep than previously available, and are consistent with the social and clinical observations of greater snoring and OSA with slow wave and REM sleep, respectively. Three-dimensional mapping of jaw position during sleep could have important clinical implications: its use may lead to improved understanding of the impact that jaw movement has on snoring and upper airway collapsibility during sleep. Mapping jaw movement during sleep could potentially also be helpful in determining which patients benefit most from the use of mandibular advancement devices for snoring and sleep apnea. The jaw position measuring device could also have potential utility in detecting bruxism (tooth grinding) or other temporomandibular problems during sleep.

5.2 Sleep Onset

Previous studies have shown a variable decrease in upper airway muscle activity at sleep onset (Mezzanotte et al., 1996) and our results show jaw protrusion and jaw closure towards the clenched position. This suggests that the muscles responsible for closing the jaw may behave differently than the genioglossus during light sleep. Alternatively, awareness of the appliance may have caused the subject to actively hold his or her jaw in a more open position prior to sleep onset; sleep onset-related hypotonia in the jaw-
opening muscles could have resulted in the observed positional change. The mechanism responsible for the observed change in jaw position during the wake-sleep transition certainly merits further study, including measurements of masseter EMG activity during sleep.

5.3 Lateral Jaw Movement During Sleep

Lateral shifts in jaw position were independent of sleep stage. This conclusion was supported by video observations that showed lateral jaw movements coinciding only with the side-to-side shifting of the head and neck. While lateral changes in jaw position may be different in the lateral sleep posture, it seems unlikely, based on current understanding of the pathogenesis of OSA, that lateral movement of the jaw plays a major role in sleep disordered breathing. An important strength of the current measurement apparatus, however, is that these changes in jaw position in the lateral plane do not confound simultaneous measurement of anteroposterior and vertical jaw position. Further, we found jaw positions recorded by the Miyamoto-type magnetic field strength-based apparatus was unable to accurately and reproducibly measure vertical jaw movement due to lateral movement. Indeed, we had assumed (erroneously) that jaw movement during sleep would be largely two-dimensional – vertical and anteroposterior. Only by considering the 3-dimensional range of mandibular motion were we able to accurately measure vertical and anteroposterior jaw position to an accuracy of less than half a millimeter.
5.4 Jaw Opening and Retraction During Sleep

Vertical jaw opening steadily increased with sleep depth (Figure 11), supporting previous work by Miyamoto et al. (1998) and Hollowell & Suratt, 1991. However, I also found that jaw retraction increased with sleep depth indicating greater mandibular movement towards the posterior wall of the pharynx (Figure 12). This unique finding is not a surprising one given the hinge-like motion of the jaw. As the jaw opens, the mandible also swings posteriorly. In fact, Kuna & Remmers, 1985 reported that a 1.5 cm opening of the jaw is associated with a 1cm retraction of the angle of the mandible.

This posterior movement of the jaw with opening may well be the critical factor underlying the increased propensity to upper airway collapse with jaw opening. The pharynx is a compliant hollow muscular tube, and subject to collapse under the external pressure from the surrounding tissues (White, 2005). The anterior fibers of the genioglossus muscle are inserted into the posterior surface of the anterior aspect of the mandible. As the mandible moves inferiorly and posteriorly during sleep, the tongue position would also be expected to move posteriorly, and thus compromise the upper airway lumen. In fact, this posterior movement of the tongue during sleep has been described in imaging studies of the upper airway (Trudo et al., 1998; Welch et al., 2002). Hence, jaw retraction during sleep should increase the propensity for upper airway occlusions (Mitchinson & Yoffey, 1947; Kuna & Remmers, 1985). Indeed, both upper airway resistance and collapsibility increase with jaw opening, unlike the closed position (Meurice J-C et al., 1996).
We did not measure EMG activity in the postural muscles of the jaw, and extrapolations from our findings to changes in upper airway / jaw postural muscle tone are speculative. Jaw opening and retraction during sleep are consistent with the decrease in tonic activity of the masseter as NREM sleep deepens (Tangel et al., 1992). As the tonic activity of the genioglossus and geniohyoideus is maintained during NREM sleep, (Wiegand et al., 1990; Tangel et al., 1992) this results in unbalanced muscular activity on the mandible, which may serve to open the jaw as sleep deepens (Hollowell & Suratt, 1991). The greater jaw opening and retraction observed during REM sleep may explain the propensity for greater severity and frequency of occlusion events during this sleep stage.

Increased genioglossus activity in sleep has been reported in patients with OSA compared with normal subjects, and is usually taken to indicate a compensatory reflex against upper airway collapse (Van et al., 1987). However, genioglossal contraction in the absence of the stabilizing effect of masseter activity during sleep, would result in jaw opening and retraction and thus compromise upper airway integrity (Lowe et al., 1977; White et al., 1995) Indeed, there is a significant linear relationship between submental EMG activity and the force of jaw opening during sleep (Hollowell & Suratt, 1991). Thus, masseter hypotonia during sleep may not only result in upper airway compromise through jaw opening, but may also reduce the efficacy of the reflex mechanisms designed to preserve upper airway patency in response to luminal narrowing. Finally, the mechanical efficiency of the pharyngeal dilator muscles located between the hyoid and the mandible may be significantly compromised by jaw opening, which would serve to
shorten the distance between hyoid and the mandible (Sauerland & Harper, 1976). Hence, jaw opening during sleep might compromise the efficiency of compensatory upper airway dilator muscle contraction, rendering the upper airway more vulnerable to collapse.

5.5 Sleep Posture and Jaw Position

The amount and duration of jaw opening during sleep is significantly greater in the supine position as compared to the lateral recumbent and upright positions (Shikata et al., 2004). The resulting pharyngeal compromise associated with jaw opening may very well be, at least partially, responsible for the greater frequency and duration of obstructive apneic events observed in the supine position (Cartwright, 1984).

Figure 14: Diagram of the head, neck and mandible and their relative positions during bite opening and closing. As the jaw opens, the angle of the mandible retracts towards the cervical vertebrae, narrowing the bony enclosure of the pharynx. (Isono et al., 2004) Image reprinted with permission from reference 33
5.6 Jaw Position and patients with OSA

On average, patients with OSA have greater jaw opening than normal controls at both end expiration and end inspiration during sleep (Hollowell & Suratt, 1991). As described above, the observed increase in jaw opening among the sleep apneics could be caused by either greater sleep-related laxity in the mandibular stabilizing muscles or greater sleep-related activation of the mandibular opening muscles.

Mouth breathers are predisposed to upper airway collapse due to the increased pharyngeal resistance associated with the oral breathing route during sleep (Fitzpatrick et al., 2003b). While mouth opening does not necessarily indicate mouth breathing (Lieberman A et al., 1990), mouth breathers may have a greater degree of jaw opening during sleep. This could be one mechanism responsible for the observation that mouth breathing in promoting upper airway collapse during sleep. Accordingly, individuals with nasal obstruction, who are forced to mouth-breathe, should have greater jaw opening during sleep. To our knowledge, this has not been investigated. Similarly, the impact of stabilizing the jaw position (in the normal relaxed awake position) on upper airway collapsibility has not been described in the literature.

5.7 The Effectiveness of Mandibular Advancement Devices

Mandibular advancement devices prevent the normal inferoposterior movement of the mandible during sleep (Schmidt-Nowara et al., 1995). Increased masseter tone has been associated with oral appliance use, indicating greater mandibular stabilization against suprahyoid muscle contraction. Without the inferoposterior movement of the jaw, the
tongue and the soft palate are less likely to fall back against the posterior wall of the pharynx even in the supine position (Gao et al., 1999). While preventing significant jaw opening and retraction during sleep should protect against upper airway collapse during sleep, these devices also protrude the mandible thus increasing the anterior tension on the genioglossus, in particular. Therefore, it is not surprising that several studies describe reduced upper airway resistance and collapsibility with the use of oral appliances. These devices are clinically effective for the treatment of mild-moderate OSA (Randerath et al., 2002).

5.8 Jaw Movement during Apneas and Arousals

During spontaneous arousals and arousals associated with termination of apneic events during sleep, jaw closure towards the clenched position was frequently observed (Miyamoto et al., 1998; Griffiths, 1975), a movement which could re-open or increase patency in the compromised upper airway. Jaw closure requires masseter activation, which stabilizes the mandibular position during arousals against increasing contractions of the genioglossus. Following arousals, we found that the jaw quickly relaxed again to the pre-apnea or arousal position (see Figure 15). Hence, the stabilizing effect of the arousal on mandibular position and masseter tone is quickly lost again as sleep resumes, consistent with the simultaneous recurrence of the obstructive apneas and hypopneas observed in these studies.
Figure 15: Sleep polysomnographic data showing a stage 2 sleep arousal. The beginning of the arousal coincided with the beginning of jaw movement which, after conversion into Cartesian coordinates, was found to be a movement towards a more clenched position. EMG activity was also increased during this time. As the arousal ended, jaw began to relax to the pre-arousal position. Channels shown: 4 electroencephalograms (EEG); 2 electrooculograms (EOG); electromyogram (EMG) positioned over the genioglossus; intercostal EMG (IEMG); leg EMG (LEMG) positioned above the left and right anterior tibialis; oral flow (ORAL); Nasal Flow (NASAL); abdominal movement (ABDO); thoracic movement (THOR). Jaw position was mapped with angle 1 (ANGLE Ø), angle 2 (ANGLE δ), and angle 3 (ANGLE ε).

5.9 Comparisons the Previous Literature

The results of my study are consistent with the findings of Miyamoto et al, 1998 and Hollowell & Suratt, 1991; although our methodology was quite different and geared specifically to mapping jaw position in all three dimensions. The use of strain gauges in
Hollowell & Suratt, 1991 was effective in correlating the force of jaw opening in OSA patients with the activation of pharyngeal dilators. Their methodology, the mechanical resistance of the strain gauge, could have influenced jaw position and hampered accurate mapping of jaw movement in sleep. Miyamoto et al, 1998 appeared to alleviate this difficulty by using hall sensors in an oral appliance. However, we were unable to replicate their findings using very similar methodology. In our hands, the hall sensors were prone to several important sources of error – in particular, the loss of biocalibrations and in vitro measurements caused by even minor lateral change in mandibular position. Using the current device, we were able to accurately measure jaw position within half a millimeter, with no reproducible error and could map all three dimensions of jaw position. By accurately measuring jaw position in all three planes of movement, we have increased our understanding of the normal physiology of sleep, which will provide the basis for future sleep disorder studies.

5.10 Advantages of this study

While the current study confirms previous findings (Miyamoto et al., 1998), we were unable to replicate their data while using a modified version of their equipment (see Appendix A). Through trial and error, we developed a measurement instrument which takes into account all three dimensions of movement. We used a greater number of subjects than previous studies to increase the power of our findings, and we investigated jaw movement in both genders for the first time.
5.11 Limitations of this study

While this study did show that jaw opening and jaw retraction increases as sleep deepens, this study was descriptive. It did not examine the mechanisms responsible for change in jaw position during sleep – indeed, we experienced enough difficulty just in developing the apparatus to measure jaw position alone. The addition of EMG hook electrodes into the pterygoid, masseter and suprathyroid muscles would provide very interesting information about corresponding muscle activity in relation to observed jaw movement. However, it would likely require a longer study with several nights per subject in order to record a full sleep cycle because of the discomfort of the recording apparatus.

My study was limited to subjects sleeping in the supine position and I could not examine the interaction between sleep stage and lateral body position on jaw position during sleep. The study was limited to young adults – jaw movement may be different in older subjects. One could speculate that muscular hypotonia in older subjects could accentuate the changes with sleep stage observed in younger subjects. The current study was also limited to healthy individuals – it is possible that jaw movement might be different among patients with OSA than normal adults.

The apparatus that we used to measure jaw movement could interfere with jaw position. While we used a counterbalancing weight to offset any weight applied to the lower jaw, just having an apparatus attached to the lower incisors itself could, potentially, interfere with normal jaw posture.
5.12 Interpretations

This study has advanced our current knowledge of on aspect of normal human physiology during sleep, and may allow us to better understand one of the important elements accounting for such sleep-related breathing disorders as snoring, and OSA. Our findings may serve as a reference point to identify abnormal jaw movement during sleep in afflicted individuals and, through correction of that, to alleviate some of the symptoms behind breathing disorders.

Many factors alter jaw movement during sleep, and increase the propensity for snoring and upper airway collapse. Age-related or hormone-related muscular hypotonia or ligamentous laxity in elderly individuals and post-menopausal women could potentially lead to mandibular instability during sleep and therefore increased propensity for collapse.

Disorders that cause jaw position abnormalities (such as nasal obstruction, temporomandibular ankylosis, and rheumatoid arthritis - induced acquired retrognathism) will likely also induce abnormal jaw movement during sleep. During times of nasal obstruction, the need for oral breathing may increase the propensity for mouth opening but at the expense of increased upper airway collapsibility.

Temporomandibular disorders that limit jaw movement or decrease the size of the pharyngeal bony enclosure will likely be associated with abnormal jaw position during sleep, and may increase upper airway collapsibility.

The greater jaw opening and retraction of the jaw observed during normal sleep explains the previously documented maximal propensity for snoring during the SWS and
REM sleep stages. Greater jaw opening and retraction during REM sleep, as compared with all other sleep stages, may explain the greater number of REM-related obstructive events observed in individuals with OSA than other sleep stages.

5.13 Future Directions
Future studies are required to determine if jaw opening and retraction observed in normal subjects is augmented in patients with OSA, elderly individuals and post-menopausal women. We can now accurately identify jaw movement abnormalities during sleep and may be able to identify specific mechanical influences of jaw position on upper airway collapsibility. We can improve our understanding of sleep physiology, even further, by studying jaw movement in sleep in demographic groups more prone to OSA. We can also expand our current knowledge of mechanical OSA therapies and provide new diagnostic and therapeutic means for alleviating OSA severity.

5.14 Conclusion
This study developed a novel apparatus to measure jaw position during sleep in normal adults. We report simultaneous measurements of jaw position in the vertical, anteroposterior and lateral planes during supine sleep in 6 men and 6 women. We have demonstrated increased jaw closure and protrusion on transition from wake to stage 1 sleep and, from stage 1 sleep, progressive jaw opening and retraction with deepening NREM sleep. Jaw opening and retraction were maximal in REM sleep. These findings in
normal subjects are consistent with previous observations of maximal snoring and upper airway collapsibility in slow wave and REM sleep, respectively.
Bibliography


Appendix A
Equipment Designs

Attempt #1: Jaw movement measurement apparatus using a Hall sensor setup.
(Developed with the assistance of Carl Blouw, Queen’s Engineering, 2005)

The jaw positioning equipment was originally modified from Myamoto and colleagues 1998 study to detect two planes of jaw movement; anteroposterior and vertical. The sensors used during this experiment were created using two Allegro A1321EUA hall sensors bonded with super glue to a plexiglass platform and separated by 10 mm (active element to active element). Five enamel coated copper wires extended from the hall sensors to a male 5 pin plug. A female 5 pin plug connected the sensor platform to a DC power supply set to 7.00V. The copper wires and the sensor platform were all coated with Plasti-dip with exception to the outer surface of the plexiglass; this assembly was coated in a thin layer of clear nail polish to seal the interface between the Plexiglas and the Plasti Dip.

Figure 16: Illustration of the jaw position sensor without the Plasti Dip coating.
Using zinc phosphate cement (Shofu Hy-Bond®), the sensor was bonded to the upper left canine and first premolar teeth. When clenched, the teeth align so that the lower first premolar lies under the upper canine. A nickel-plated Neodymium Iron Boron disk magnet (6.35 mm diameter, 3.25 mm thick) supplied by Wondermagnets® was cemented...
onto the teeth with zinc phosphate so that it was directly under (or close to) the 2\textsuperscript{nd} hall sensor. Both the hall sensors and the magnet had to be in the same plane and between 2 and 5 mm apart.

The two hall sensors create an output voltage proportional to the magnitude of the magnetic field from the disk magnet. This voltage was then used to determine the distance of separation between the hall sensors and the magnet using an series of calculations created from the calibration data collected between the 2 sensors and the magnet. As the distance between the hall sensors was known, trigonometry could be used to calculate the distance of sensor 1 and sensor 2 from the magnet center and determine the angle of interest between sensor 1 and 2. Data was collected every 4\textsuperscript{th} of a second.

\textbf{Trigonometry:}

With the hall sensors placed a known distance (d) from each other within the platform and the magnet attached below, we theorized that basic trigonometric calculations could be used to determine the distance between the magnet and each hall sensor. We assumed the magnet and platform remained in the same plane throughout the night.
The distances $d_1$ and $d_2$ are determined from the voltages from Hall sensors 1 and 2 and the calibration data with the equation:

$$d_1 = -t_1 \ln \left( \frac{V_1}{A_1} - y_1 \right)$$
$$d_2 = -t_2 \ln \left( \frac{V_2}{A_2} - y_2 \right)$$

Where: $d_1$, $d_2$ are the distances between sensor 1, 2 and the center of the magnet, $V_1$, $V_2$ are the voltage outputs from sensor 1 and 2 respectively, and $t_1$, $t_2$, $A_1$, $A_2$, $y_1$, $y_2$ are the calibration parameters for each hall sensor.

With $d$, $d_1$, $d_2$ known ($d$ is fixed at 10 mm), the interior angles of the triangle formed by the magnet and two Hall sensors can be determined with the cosine law.
\[
A = \arccos \left( \frac{d_1^2 - d_2^2 - d_2^2}{-2dd_2} \right) \\
B = \arccos \left( \frac{d_2^2 - d_1^2 - d_2^2}{-2d_1d} \right) \\
C = \arccos \left( \frac{d_2^2 - d_1^2 - d_2^2}{-2d_1d_2} \right)
\]

Finally, the anteroposterior (x) and vertical (y) distances are calculated as follows:

\[x = d_1 \sin B\]
\[y = d_1 \cos B\]

**Calibration:**

Each jaw motion sensor was calibrated with a specific magnet to correlate distance directly with voltage and define specific calibration parameters that will influence these calculations. Calibration was performed after the jaw motion sensor was constructed coated, and the tooth-facing side of the magnet was roughened. The two Hall sensors in the jaw motion sensor were calibrated independently.

The jaw motion sensor was fixed in place on a 1mm division grid sheet. The grid with attached sensor was fixed to a flat surface. The output of Hall sensor 1 was connected to a digital multimeter. The magnet was placed directly perpendicular from Hall sensor 1 such that 30mm separated the edge of Hall sensor 1 and the edge of the magnet. The output voltage was recorded. The magnet was moved closer to the edge of Hall sensor 1 in 1mm increments; the output voltage was recorded at each separation. The above was repeated for Hall sensor 2.
Software (Origin Lab OriginPro 7.5) was used to plot and fit 1st order exponential decays to the calibration data (output voltage vs. center of magnet to center of Hall sensor separation) for hall sensor 1 and hall sensor 2 using the 1st Order Exponential Decay Equation:

\[ V = y_0 + A e^{-\frac{d}{t_1}} \]

Where \( V \) is the output voltage in volts, \( d \) is the distance between the center of the magnet and the center of the hall sensor, and \( y_0, A, t_1 \) are the fitted parameters. These comprise the calibration for a single jaw motion sensor.

**Figure 20** A sample plot of calibration data from a Hall sensor fitted to an exponential decay.
Limitations of this device

We encountered three problems with this experimental set-up: (i) Our initial assumption that the magnet and platform remained in the same plane throughout the night was incorrect as slight lateral movements of the jaw were common, resulting in a marked alteration of the measured magnetic field strength and complete invalidation of the measured jaw positional change in the x and y planes. (ii) Despite the plasti-dip coating around the Hall sensors, saliva penetrated the sensor apparatus at times, creating unwanted electrical bridges within the sensor, rendering them useless; and (iii) Even in the absence of the problems outlined above, the device did not appear to hold its calibration beyond one hour, after which significant sensor drift occurred.

ATTEMPT #2 External piezo-electric transducer setup
(Developed with the assistance of John Christensen, Clinical Engineering, Kingston General Hospital)

Finding the magnetic route to be unsatisfactory in measuring 2-dimensional jaw position, we turned to an ultrasonic acoustic distance measuring system using piezo electric transducers adapted to an ultrasonic frequency of 44khz for sound generation and reception. The principle of this system was to measure the time required for a pulse of sound, emitted from a transmitter, to travel to two receivers of known distance apart. With all three sides of the triangle known in the clenched and subsequent jaw positions, the relative movements of the jaw in the vertical and anteroposterior directions could be calculated using simple algebraic methods. This system exploited the fact that the
distance traveled by a sound wave in air is a linear function of time, related by the constant speed of sound at a given temperature. Unfortunately the transducers were unable to withstand the environment within the mouth and so a new, external platform setup had to be created.

The platform setup

Two separate platforms were constructed for attachment to the upper and lower incisors. Each platform consisted of a light, polystyrene rod embedded in a plexiglass platform, which extended out of the mouth and supported the piezo-electric transducers. Like the previous setup, the plexiglass platform was attached to the teeth using zinc phosphate cement (Shofu Hy-Bond®). The platform on the upper incisors supported the transmitting transducer while the platform on the lower incisors supported the two receiving transducers. In the clenched position, the transmitter was equidistant from each receiver. As the jaw moved vertically or anteroposteriorly, the dimensions of the triangle formed by the three units changed accordingly. The width of the transducers allowed up to 1 cm lateral jaw movement without affecting the anteroposterior or vertical jaw movement data.
The Measurement System

After some early experimentation with an acoustic phase shift measuring system, we decided to use the amount of time a pulse of sound emitted from piezo-electric crystal transmitters to reach the receivers as the method for measuring distance. In this system, the receivers each generated an analog voltage output proportional to the time delay between emission of an ultrasonic pulse and its reception.

The circuit system responsible for this consisted of timing and control logic. The timing and control logic contained a master clock which determined the sampling rate of 15 measurements / second. At the beginning of each 182 μs sampling period, the controller did two things: initiate a burst of the 44 kHz driving signal for application to the transmit transducer, and initiate a voltage pulse applied to a voltage integrator in the time-to-voltage circuit.

Figure 21: A schematic diagram of the lateral view of the transducer setup. Like the hall sensor setup, the distance of the transmitter from each receiver would be measured allowing us to use basic trigonometry to determine x and y coordinates of mandibular position.
When a sound pulse arrived at the receiving transducer, it excited the generation of a small voltage by that transducer which was amplified and applied to a threshold detector circuit. When the amplified voltage exceeded the threshold level, a signal from the threshold detector caused the control circuit to terminate the voltage pulse to the integrator. The voltage pulse applied to the pulse integrator was thus proportional to the time of flight of the sound wave. The voltage integrator response produced a corresponding voltage output proportional to the width of this pulse. An output amplifier scaled the output from the integrator and the filter removed the 15 Hz ripple inherent in such a system.

**Modifications:**

Problems with the basic mechanical setup of the equipment had to be overcome before continuing with our experiments. The two platforms made the system uncomfortable and difficult to mount. An exact alignment of the receivers directly over the transducers was critical for reliable and linear signal outputs, but this was extremely difficult to do without error. The signal threshold detector could not fully compensate for shifts in signal amplitude due to shifts in transducer alignment, causing small but significant and unpredictable errors in the distance measurements. This made the system vulnerable to even small amounts of lateral movement that would otherwise have not been detected given the size of the transducers.

This mechanism consisted of a rod which attached to the upper incisors by a hinged attachment appliance (to allow for lateral jaw movement). The support rod supported a
carriage which held two transmitters. One pointed along the length of the rod and the other angled downward at 90° in the same direction as a second rod attached to the bottom of the carriage. Also supported by the top rod was a fixed receiving transducer to detected movement of the carriage along the axis of the top rod. (protrusion-retraction). The lower rod supported a second carriage attached by a hinged extension to a platform attached to lower incisors. This carriage supported a receiving transducer to detect vertical jaw position. Each carriage was free to slide along the support rod to which it was attached.

Figure 22: Schematic of the lateral view of the modified system. This system maintained control over transducer alignment to keep transducer separations in a region where good linearity was possible to achieve. The carriages slid along the support rods (path shown with arrows) dividing all jaw movements into vertical and anteroposterior directions. This entire apparatus moved from side to side with lateral jaw movement removing the influence of lateral movement on the measured vertical and anteroposterior distances.
Limitations of this device:

This system was very cumbersome, heavy and difficult to use in vivo. The carriages added load to the jaw. The wiring attachments to the transducers were fragile and broke frequently, necessitating transducer replacement. Manufacturing variations between transducers resulted in varying transducer sensitivity, affecting signal detection thresholds, and these were subsequently reflected as distance variation.

The transducers responded to various types of electromagnetic interference from sources that could not be satisfactorily removed despite acoustic sensitivity to only a small band around 44 kHz. These disturbances repeatedly interrupted the recording process and caused non-linear variation in the electro-mechanical behavior of the transducers, resulting in ambiguities in the distance measurements.
Appendix B
Calculations to Determine Jaw Position

Collection Data:

Voltage outputs are collected (in Volts) from each of the three angles measured by:

Sensor 1 : V1
Sensor 2:  V2
Sensor 3:  V3

Angular distance is determined by referencing the measured voltage to the maximum voltage detected by the sensors. Sensor sensitivity changes with temperature. The maximum voltage decreases at a rate of -0.32 % for every 1°C increase in temperature; we used the maximum voltage at 23.8 ºC, and monitored ambient temperature throughout the study to correct for the discrepancy when calculating the angle.

Reference Temperature: 23.8 ºC
Maximum voltage for all sensors at 23.8 ºC: 4.965 Volts

Calculating Jaw Position at a single recorded data point:

Step 1: Correct for temperature difference

Temperature difference = (23.8 – actual temperature)

New maximum voltage (Vmax) = 4.965 + [0.0032(Temperature difference)]
Step 2: Convert each voltage output into an angular distance (in degrees) and correct for temperature by referencing it to the new maximum voltage.

Angle 1 (\(\phi\)): \(\frac{1}{2} \arcsin \left( \frac{V_1}{V_{\text{max}}} \right)\)

Angle 2 (\(\delta\)): \(\frac{1}{2} \arcsin \left( \frac{V_2}{V_{\text{max}}} \right) + 118\)

Angle 3 (\(\epsilon\)): \(90 - \left[ -\frac{1}{2} \arcsin \left( \frac{V_3}{V_{\text{max}}} \right) \right] + 6.98\)

Step 3: Calculate the length of the radius vector (r) from the origin (the radius of the sphere defined by the measured spherical coordinates). This is found by using the cosine law to solve for the missing side in the triangle formed by the links a and b (6.77 cm and 6.32 cm in length respectively) of the sensor brace.

\[ r \, (\text{cm}) = \sqrt{85.7 - 85.57 \cos (\text{Angle 2})} \]

Step 4a: Calculate the angles made between the radius and link a

\[ \beta = \arcsin \left[ \frac{6.32 \sin \text{Angle 2}}{r} \right] \]

Step 4b: Calculate the angle made between the radius and the y axis

\[ \theta = 90 - (\text{Angle 3} - \beta) \]

Step 5b: Calculate the rotation around the Y-axis

\[ \phi = \text{Angle 1} \]

Step 5: Convert Spherical Coordinates (r, \(\theta\), \(\phi\)) to Cartesian Coordinates (x,y,z) in centimeters
\[x = r \sin \theta \sin \phi\]
\[y = r \cos \theta\]
\[z = r \sin \theta \cos \phi\]

Step 6: Convert x, y, z distances from centimeters into millimeters

**Figure 23:** Schematic diagram of the spherical coordinates system and how it relates to the Cartesian coordinate system.

Calculating Actual Jaw position as referenced to clenched

Step 7: All collected jaw position coordinates are referenced to clenched coordinates

- Vertical Jaw Position: \((y - \text{clenched}_y)\)
- Anteroposterior Jaw Position: \((x - \text{clenched}_x)\)
- Lateral Jaw Position: \((z - \text{clenched}_z)\)