A COMPARATIVE STUDY OF THE RELATIONSHIP

BETWEEN AIRWAY SIZE, TONGUE ACTIVITY

AND BODY POSITION

By

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ABSTRACT

Airway obstruction in Obstructive Sleep Apnea (OSA) patients is believed to occur in the supine position during sleep. In order to investigate the relationship between upper airway size and genioglossus(GG) muscle activity, up-right (in natural head posture) and supine cephalograms were obtained for twenty OSA and ten asymptomatic control subjects. Tongue EMG and pressure recordings were obtained with the surface electrodes and pressure transducers in ten asymptomatic control subjects. The Student's t test and Wilcoxon signed rank test were used to test for differences between the two groups and between body positions.

The OSA group revealed a longer tongue (p< 5%), a larger soft palate (p< 1%), an anteroposteriorly narrower and vertically lengthened upper airway (p< 1%), a inferiorly positioned hyoid bone (p< 1%), a more extended head posture (p< 5%) and a smaller hypopharynx (p< 1%) in the up-right standing position. After changing from the up-right to the supine position, the tongue cross-sectional area increased 4.3% (p< 5%) and oropharyngeal area decreased 36.5% (p< 1%) in the OSA group. When comparing the supine to the up-right control cephalograms, changes in tongue area were not observed, but the thickness of the soft palate increased (p< 1%). Differences in tongue cross-

sectional area between two groups become significant with body positional changes from the up-right to the supine (p< 1%). With body positional changes, the hyoid bone moves superiorly toward the mandibular plane in the control group (p< 1%), but anteriorly toward the mandibular symphysis in the OSA group (p< 5%). The rest EMG activity of the GG muscle increased 33.8% (p< 5%) and the posterior tongue pressure increased 17% (p< 5%) with body positional changes from up-right to supine. Overall, the orophayngeal cross-sectional area collapsed 28.8% (p< 1%) despite a 34% increase (p< 5%) in GG muscle activity in the asymptomatic control group as a result of body positional changes. Furthermore, a 17% increase of tongue pressure on the posterior load cell indicates positional change of the tongue.

In conclusion, it may not be the size of the soft palate alone but also the vertical and anteroposterior position of the tongue which could actively contribute to the development of OSA. Quantification of subtle differences in tongue and oropharyngeal size and position, geometry of the hyoid bone, upper airway muscle activity and tongue pressure accompanied by body positional changes aids in our understanding of the pathogenesis of OSA.

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INTRODUCTION

It is only recently that the field of dentistry has become concerned with the obstructive sleep apnea (OSA) syndrome. However, OSA is no longer an unknown in the field of dental research. Familiarity with this syndrome has increased not only because of its high rate of incidence but also because orthodontists and oral surgeons are beginning to play a more significant role in treating OSA syndrome with the use of intra-oral devices and/or orthognathic surgery. It has been suggested that OSA may be one of the most prevalent diseases in modern society. One epidemiological report suggests a prevalence rate of 0.89% as a lower limit in the adult male population (Lavie, 1983). In spite of the remarkable amount of research that has been done in this field in the last decade, the OSA syndrome is masked by various symptoms and by the intricacy of its pathogenesis.

A. What is sleep apnea ?

An apnea is defined as a cessation of air flow at the nose and mouth for more than 10 seconds (Guilleminault et al., 1975). According to Tassinari et al.(1972), three types of apnea have been defined. The first, central apnea is defined as the complete arrest of respiration for 5-10 seconds. The

second, obstructive apnea is defined as an interruption of the airflow for 10 to 60 seconds while the abdominal pneumogram indicates persistence of ineffective respiratory movement. The third type, complex apnea is defined as an episode of central apnea for 5-10 seconds followed by an episode of obstructive apnea. According to Guilleminault et al.(1975), the reverse order never seems to occur. Guilleminault (1985) defined hypopnea as an incomplete form of apnea which is induced by decreased diaphragmatic effort and partial obstruction of the airway.

There are several ways of describing the severity of sleep apnea. The Apnea Index (AI) is defined as the total number of apnea incidents per total sleep time multiplied by 60. Guilleminault et al.(1987) suggested an Apnea Index of 5 as the upper limit of normality. The RDI (Respiratory Disturbance Index) is another index measuring the severity of sleep apnea and is defined as the Apnea plus Hypopnea Index.

In his book "Posthumous Papers of the Pickwick Club"(1837), Charles Dickens described an extremely fat boy named Joe who suffered from persistent somnolence. In 1918, Sir William Osler coined the term "Pickwickian", to refer to obese and hypersomnolent patients (Burwell, 1956). Several studies reviewed the symptoms of the Pickwickian syndrome. In 1964, Gastaut and his associates reported the presence of repetitive obstructive apnea during sleep in the "Pickwickian" patient. In 1966, the same group evaluated the sleep,

respiration and blood gas chemistry of the patient by means of polygraphic registration. They measured EEG, spirogram and EMG of diaphragmatic and mylohyoid muscle activity, hypothesized the pathogenesis of obstructive sleep apnea and distinguished central apnea from obstructive apnea. The term 'sleep apnea' was first used in 1971 by Kumashiro et al.. In 1972, Guilleminault et al. differentiated obstructive sleep apnea both from the Pickwickian syndrome and from narcolepsy. In the following year(1973), they reported insomnia with Sleep Apnea as a new syndrome and finally named it sleep apnea syndrome.

OSA is predominantly a disease of obese middle aged men, yet is less known in obese women or children. Regardless of the age or sex of the sufferer , snoring is considered the cardinal sign of sleep apnea. Patients may present restless sleep patterns such as turning, tossing, flailing of the extremities and even sleep walking. Some patients may complain of a sensation of choking or gagging. Guilleminalt (1987) observed esophageal reflux in a number of patients and nocturia was also reported as a more common symptom in children. In the early or middle stages of the development of the disease, insomnia may be observed;however, excessive daytime sleepiness and/or fatigue is another hallmark symptom of OSA. In extreme cases, the patient falls asleep while talking, eating and even driving. Deterioration of the memory, disorientation, morning confusion and hypnogogic hallucinations were reported as side-effects of hypersomnolence. Occasionally, a hyponasal voice, noisy

breathing and mouth breathing are noticed.

In 1960, hemodynamic abnormalities in extremely obese patients were studied by Sieker and his colleagues. Burwell et al.(1956) presented a comparative hemodynamic study between the reduced weight state to initial state in a Pickwickian patient. Abnormal alteration of gas exchange and cardiovascular function is well documented by several researchers (Bradley 1988, Bradley and Phillipson 1985). Hypoxia, hypercapnea and acidosis may develop polycythemia, systemic hypertension, cor pulmonale, vagal bradycardia and nocturnal cardiac arrhythmia. Another hemodynamic effect occurs mechanically in the cardiovascular system (Scharf,1984). Decreased intra-thoracic pressure during the obstructive apneic period creates large negative loads in the pleural cavity, which increases the venous return to the right ventricle, and may result in cardiac malfunction.

B. Pathogenesis of OSA

As mentioned previously, OSA is still an unclarified, complex phenomenon. At least three factors are thought to be involved in the pathogenesis of OSA: sleep, upper airway anatomy and upper airway muscle function. Sullivan et al.(1984) reviewed a number of leading theories and has suggested that a balanced force between tissue weight, muscle tone, and airway lumen suction pressure likely governs the pathophysiology of OSA syndrome. No one theory has been accepted, although all of them may contribute in part to the pathogenesis of OSA .

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1] Overview

In 1978, Remmers et al. undertook an elaborately designed study on humans in an attempt to explain the pathogenesis of OSA. They hypothesized that an uncoordinated force balance between genioglossus(GG) muscle force and the oropharyngeal transmural pressure causes pharyngeal occlusion. When the pharyngeal transmural pressure increases more than the GG muscle force, occlusion of the upper airway may occur. Sullivan et al.(1984) summarized that the narrower airway is more vulnerable and dependent on dilator muscle tone for airway patency. They concluded that airway suction pressure was the key force provoking airway occlusion;i.e. the greater the inspiratory muscle force and the smaller the cross-sectional area, the higher the linear velocities of airflow and the greater the transmural pressure gradient favoring upper airway collapse.

Guilleminault (1987) explained that once airway obstruction occurs due to the various predisposing factors (for instance, anatomic abnormalities of the upper airway, decreased airway muscle activity, small pharyngeal cavity, high pharyngeal compliance and increased upstream resistance), Pco_2 tension should rise. Increased Pco_2 tension causes an increase in diaphragmatic effort which may result in more negative intra-thoracic and oropharyngeal pressures. As a counter factor to the negative forces triggering airway collapse, the stabilizing forces of the upper airway dilators are crucial. Whether

determined by a sleep related failure of the upper airway muscle activity and/or defective chemorecepter function, the specific characteristics of the dilator muscles are the most likely underlying dominant factor in the mechanism of OSA. Several studies suggest that upper airway resistance, especially expiratory resistance, is increased in sleep (Orem, 1986). Moreover, the airway resistance in OSA patients is assumed to be higher than normal (Suratt et al., 1985) and many hemodynamic reports of overnight studies in OSA patients show a high Pco_2 level. In 1987, Parisi and associates compared the Pco_2 threshold of the GG and the diaphragm in goats. They found that the Pco_2 threshold of the GG, in comparison to the diaphragm, is disproportional both during NREM(Non-Rapid-Eye-Movement) sleep at low CO_2 tension and during phasic REM(Rapid-Eye-Movement) sleep at any CO_2 tension. They postulated that this imbalance between the GG and the diaphragm may predispose the upper airway to inspiratory occlusion during sleep.

2] Predisposing Factors

a. Sleep

Most of the psychiatric phenomena that occur in OSA can be explained on the basis of sleep derangement (Singh, 1984). By definition, sleep may be considered as recurrent spontaneous episodes of motor quiescence accompanied by raised thresholds of sensory response (McGrinty and Beahm,1984). On the basis of electrophysiological measurements, mammalian sleep is divided

into two distinct types: NREM and REM sleep. REM and NREM sleep alternate throughout the night. NREM sleep, which is also called slow-wave sleep or synchronized sleep, consists of four stages. In infants, REM sleep is also called paradoxical sleep or active sleep, and is characterized by brief, abrupt phasic motor events, eye movements or twitches of the facial and extremity muscles (McGrinty and Beahm, 1984). The waking stage shows a low amplitude, high freqency(13 to 35 Hz) beta wave. Stage 1 NREM sleep normally consists of 5-10% of total sleep time, and is a transitional phase between wakefulness and sleep. It is characterized by mixed-freqency activity in the beta and theta(4-7Hz) waves. Stage 2 is marked by the appearance of EEG sleep spindles (i.e.,burst of activity from 12 to 14 Hz lasting one half to two seconds) and K-complexes(well-delineated, slow, negative EEG deflections). Delta sleep, the deepest stage, is distinguished by slow delta waves greater than $75\mu V$, peak to peak. In REM sleep, the EEG consists of mixed frequency, low-voltage activity resembling stage 1 NREM sleep. REM sleep periods tend to become longer as sleep progresses and constitute about 20% of total sleep time in the healthy adult. REM sleep is characterized by broad physiological changes. While EMG activity maintains its lowest level during REM sleep, the extra-ocular muscles show rapid, intermittant, conjugated eye movements. In about 80% of wakenings from REM sleep, people recall dreams (Hauri and Orr, 1982) which are vivid, active and filled with colorful and complex ideation (Baker, 1985). Phasic

components within REM sleep are characterized by a high autonomic variability such as irregular heart rate and blood pressure which may transiently increase as much as 40 mmHg (Coccagna et al, 1972). Contrarily, during tonic REM sleep, the heart rate and blood pressure are more constant and there is greater relaxation in most muscles. Cerebral blood flow is greater during REM than either NREM or wakefulness. Respiration also seems altered during REM sleep. Many upper airway muscles and intercostal muscles become hypotonic whereas the diaphragm maintains activity and CO_2 sensitivity is lost with frequent phasic events (Sullivan et al, 1979b). Loss of intercostal EMG activity may lead to paradoxical collapse of the chest during inspiration (Henderson-Smart, 1984). Rib cage 'paradox' is exacerbated by increased upper airway resistance (McGrinty, 1984). The tone of the GG and geniohyoid muscles may significantly decrease during REM stage. In other words, REM sleep is associated with a decreased upper airway patency and increased transpharyngeal resistance (Guilleminault, 1988).

b. Anatomical Factors

It is not difficult to assume a respiratory malfunction in patients who have apparent anatomical anomalies, pathoses or neuromuscular defects. Moran (1987) enumerated many of the abnormal physical characteristics which could be found in OSA patients. However, a great number of patients suffering from moderate or less severe OSA symptoms are not likely to have

obvious physical deficiencies, except obesity. OSA is often coincident with obesity and snoring. Wittels (1985) defined obesity as a body weight at least 20% above the ideal. Obesity induces numerous changes in pulmonary and cardiovascular function. According to Wittels, in the case of obesity, the total lung volume, functional residual capacity and tidal volume are diminished. In addition, the accumulation of fat around the chest wall decreases chest compliance. Due to the low lung volume, respiratory resistance is increased. Because the respiratory work of breathing is somewhat greater than normal, the oxygen cost is increased. An increased cardiac output is demanded in the obese patient, hence left ventricular stroke work is increased, which finally leads to an increased left ventricular end-diastolic pressure. An interesting survey was undertaken by Grunstein et al.(1989) in Western Samoa. The ANCOVA(Analysis of Covariance) study revealed a significant correlation between snoring and collar size, BMI(Body Mass Index), blood pressure and smoking, and yet no relation was found with aging. Suratt and co-workers (1987) found in a prospective study that moderate weight loss in obese patients with OSA improves disordered breathing during both sleep and wakefulness. They hypothesized that a narrow collapsible pharyngeal airway in awake subjects is an important factor in the pathophysiology of OSA. A precisely weight-matched investigation by Horner et al.(1988) concluded that there are no systemic differences in fat deposit distribution between OSA

patients and normal patients, and that there are no significant fat deposits immediately posterior to the airway in any of the subjects. Furthermore, Rubinstein and colleagues (1988) postulated that improvement of OSA symptoms after weight loss may be related to improvement in pharyngeal glottic function, in other words, weight loss results in beneficial changes in pharyngeal mechanics. Furthermore, Lugaresi (1988) supposed that the narrower and longer oropharyngeal isthmus in snorers and OSA patients results from the repeated heavy inspiratory efforts associated with a downward stretch of the laryngo-tracheo-bronchial tree. They hypothesized that a loss of even a few kgs of body weight can reverse a typical OSA into simple snoring.

Weight is intimately related to the airway size. Numerous researchers have attempted to measure upper airway size (Jackson et al., 1980; Lowe et al., 1986; Martin et al., 1987). Several visualization techniques are available including cephalometry, CT(Computerized Tomogram)scans, MRI(Magnetic Resonance Imaging), cineradiography, nasopharyngoscopy, acoustic reflectance and flow volume curves. Among them, CT and MRI could be used for three-dimensional(3D) reconstructions (Lowe et al., 1986,1989). Lowe et al. emphasized the obvious limitation of two-dimensional(2D) methods since they may not accurately quantify 3D spatial relationships. In this respect, 3D CT or MRI reconstruction is recommended as a more reliable method. However, such a 3D reconstruction technique is time-consuming

and expensive for routine clinical use. As a non-invasive technique, Fredberg and co-workers(1980) actively adapted the acoustic reflection technique for measuring airway geometry. D'urzo et al.(1987) performed a validity test for the acoustic reponse measurement with respect to CT, and proved its high compatibility. The cephalometric technique is another prevalent method, despite its 2D limitations. Not only linear measurement but also area measurement have become feasible. Pae et al.(1989) attempted to prove a compatibility of 2D lateral cephalometric technique to 3D CT reconstruction, yet failed to demonstrate a high correlation between 2D and 3D of the tongue, soft palate and pharynx.

Several visualization techniques are available to identify the occlusion site. CT has been used as a tool for identification of the occlusion site. Haponik et al.(1983) found the obstruction site mainly at the nasopharynx and oropharynx. Using fluoroscopy, Suratt and associates (1983) observed that the obstruction always begins during inspiration when the soft palate touches the tongue and posterior pharyngeal wall. Chaban et al.(1988) developed a technique based on the analysis of inspiratory airflow pressures. They used a movable catheter pressure transducer localized by cephalometrics and identified two different obstruction sites: the posterior portion of the soft plate and the base of the tongue. By means of cine-CT scans, Crumley and associates (1987) examined the obstruction site and found a diminution in the anteroposterior

diameter after assuming the supine position. They presumed that the tongue musculature appears to settle posteriorly under the pull of gravity in the supine position. Stein et al.(1987) confirmed Crumley and associates' observations and conclusions and illustrated the obstruction sites by means of cine-CT at the uvula and oropharynx mainly. Hoover et al.(1987) reviewed the magnetic resonance imaging technique of the larynx and the base of the tongue and demonstrated the superiority of the MR image of the soft tissue structure and stressed the non-invasiveness of the MRI.

c. Functional Factors

As mentioned earlier, upper airway muscle function may also play a primary role in the pathogenesis of OSA. As far as OSA is concerned, the GG has been the most closely studied upper airway muscle in the last decade. The GG is a virtual protrudor associated with the hypoglossal(XII) nucleus. In adult humans, the GG consists of three parts: an anterior portion attached to the superior tubercle, a middle fan-shaped oblique portion which is attached by a separate tendon lateral to that tubercle, and an inferior, almost horizontal portion attached to the mandible between the superior and inferior tubercles. Some fibres of the inferior portion are attached to the upper aspect of the body of the hyoid bone. A horizontal component inserted into the posterior one-third of the tongue is the main protrudor (Doran and Bagget,1972). None of the GG muscle fibres are inserted

through apex of the tongue tip (Doran and Bagget, 1972; Hellstrand, 1980). Histologically, both type I and type II fibres are identified in the extrinsic tongue muscle in cats. The fibre-type nomenclature of the human skeletal muscle, 'type I' and 'type II' fibres, are based upon fibre identification with the myofibrillar adenosine triphosphatase (ATPase) reaction at PH 9.4. According to Engel(1974), the muscle fibres markedly predominating in the red muscles are type I, whereas the fibres occuring exclusively in white muscles are type II. Compared with white muscle tissue, biochemical assays of tissue homogenates show that red muscle has more myoglobin, succinate dehydrogenase, and cytochrome oxidase, and has less myosine ATPase, lactate dehydrogenase, diphosphopyridine nucleotide-linked alpha glycerophosphate dehydrogenase, phosphorylase and mitochondrial alpha glycerophosphate dehydrogenase. All type II fibres are fast-twitch units, but some of these are fast fatiguing and others are fatigue resistant, whereas type I units are all slow-twitch and fatigue resistant. Hellstrand (1980) found that 75-81% of the tongue extrinsic muscles consist of type II fibres, whereas the digastric muscle consists of 60% type II fibres (Miller and Farias, 1988). In humans, the GG muscle is proportionately larger than in other mammals such as cats, rats and dogs (Doran, 1975).

Sauerland and Mitchell (1970), demonstrated phasic GG muscle activity during inspiration by means of bipolar needle electrodes. In 1975, they observed a markedly increased tonic

activity of the GG in the supine position. They interpreted that the base line activity of the GG increased to counteract the tongue relapse due to the gravitational pull. Sauerland and Harper (1976) recorded the GG EMG activity in humans during the various sleep stages. Remmers et al.(1976) described the role of the GG muscle in upper airway obstruction during sleep. They suggested that atonia of the GG contributed to the inspiratory upper airway obstruction. Guilleminault et al.(1978) also found significantly decreased GG EMG activity during obstructive apnea. However, with the "tent hypothesis", they negated the singular involvement of the GG muscle in the genesis of the OSA syndrome, but suggested the involvement of several muscle groups, primarily the superior constrictors. A number of comparison studies of the GG and the diaphragm soon followed. Brouillette and Thach(1980) postulated that the GG response to chemoreceptor input and non-specific stimuli is qualitatively similar but quantitatively different from diaphragm responses. Önal and colleagues(1981) observed synchronous activity of the GG and the diaphragm in normal human subjects and emphasized the importance of the inspiratory function of the upper airway muscles in maintaining upper airway patency. They postulated that the GG and geniohyoid muscles maintain upper airway patency by pulling the tongue and hyoid bone forward during inspiration. Haxhiu et al.(1987) designed an experiment to compare the response of the diaphragm and upper airway dilating muscle activity in sleeping cats. They found that hypercapnea

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affects the GG differently than the diaphragm, and suggested this might be due to the different threshold characteristics of hypoglossal and phrenic neurons.

From the point of view of the mechanics of respiration, the supra-hyoid muscles have only recently been recognized as important. Doran and Baggett (1972) discuss the relationship between the GG muscle and hyoid apparatus. They observed that some fibres of the inferior portion of the GG are attached to the upper portion of the body of the hyoid bone. This finding agreed with that of Abd-El-Malek (1938), but not with Hellstrand (1980). Doran and Baggett postulated that in the early phylogenetic stage, the GG muscle and the geniohyoid muscle might be the one supra-hyoid group. They rely on Livingston's (1965) criteria of tongue mobility, which suggest that movement of the tongue depends largely on the movement of the hyoid bone. By definition,the supra-hyoid group includes digastric, stylohyoid, mylohyoid and geniohyoid muscles (Kaneko, 1975). Among them, the geniohyoid muscle alone has the airway dilatation function;i.e., it pulls the elevated hyoid bone directly forward which increases the anteroposterior diameter of the pharynx (Romanes, 1979). In contrast, the stylohyoid muscle pulls the hyoid bone posteriorly; the mylohyoid muscle stabilizes either the mandible or hyoid bone. Of the infra-hyoid muscles, the sternohyoid and thyrohyoid muscles pull the hyoid muscle down (Kaneko, 1975). In 1984, van de Graaff et al. explained that the respiratory function of the hyoid muscle

and hyoid arch affect upper airway resistance. They suggested that the strategic location of the hyoid arch may contribute to the patency of upper airway and the decreased or poorly coordinated function of the hyoid muscle may induce upper airway occlusion. This hypothesis was suggested by Mathew (1984), as well as by Roberts et al.(1984).

Blood gas changes, pressure changes, and tissue distortion can elicit respiratory reflex effects (Cherniac and Hudgel,1986). As lung volume increases during respiration, stimulation of pulmonary stretch receptors feeds back to the respiratory center via the vagus nerve. This is the classic Hering-Breuer reflex (Parisi and Neubauer,1986). Van Lunteren et al.(1984) investigated the effects of vagally mediated volume-related feedback on the activity of the upper airway muscles and found that the amount of depression at the end of inspiratory airflow was greater for all of the upper airway muscles than for the diaphragm. They explained this phenomenon as possibly due either to a lower threshold of the upper airway muscles for inspiratory depression or to a vagal depression effort on the upper airway muscle or possibly to both. Agostoni et al.(1986) undertook an examination on the time-coursed effect of the stretch receptor of the bronchi or trachea on GG muscle activity in rabbits and reported that bronchial input facilitates GG activity at the end-expiratory volume and inhibits it at larger volumes. A hypercapnic response has been extensively studied by several groups (Bulow, 1963; Douglas et

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al,1982; Berton-Jones and Sullivan; Weiner et al, 1982).

St. John et al.(1984) reported in cat that activity in the phrenic and hypoglossal nerves increases or decreases in parallel fashion with the hypercapneic condition. This finding concurrs with the results of Önal et al.(1981). However, a number of recent studies show a curvilinear relationship between the GG and the diaphragm in conditions of persisting hypercapnia (Parisi et al., 1987; Hixhiu et al., 1987). Nevertheless, it is obvious that arousal increases the GG activity more than that of the diaphragm. A number of research studies support the idea that it is not hypoxia but hypercapnia or asphyxia which provokes the arousal (Fleetham et al, 1982; Issa and Sullivan, 1986; Mathiot et al, 1986).

Kuna (1987) carried out an experiment concerned with the interaction of hypercapnia and phasic volume feedback on the motor control of the upper airway in decerebrate, paralyzed, and intubated cats. He found that the hypoglossus is more sensitive than the recurrent laryngeal nerve to suppression by phasic volume feedback. Kuna (1987) also concluded that hypercapnia may potentiate the effect of lung volume on the suppression of the upper airway motor neuron activity. This phenomenon implies that the respiratory control mechanism is designed to decrease energy expenditure of the upper airway muscles to suppress their activation when they are not needed, i.e., in the presence of phasic volume feedback, which results in augmented upper airway motorneuron activity. The mechanism

of the cyclic changes in ventilation has been considered a fundamental factor in respiratory function. Several studies (Van Lunteren and Strohl, 1986; Adachi et al., 1989; Hudgel and Harasick, 1989) suggest that the inspiratory activity of the upper airway muscles slightly precedes that of the diaphragm in normal situations. This rhythmic order may be a critical factor of the pathogenesis of OSA. The instability in the rhythmic feedback control of respiration could provoke OSA (Longobardo et al., 1982). Cherniac and Longobardo (1986) assume that this disturbance in feedback rhythm may trigger recurrent apneas.

Sullivan et al.(1981) have developed CPAP(Continuous Positive Airway Pressure) to assist in the maintenance of upper airway patency. The mechanism hypothesized was that the CPAP would act as a pneumatic splint and prevent upper airway occlusion, pushing the soft tissue and tongue forward and away from the posterior pharyngeal wall. However, there has been considerable controversy about the rationale of CPAP use and its immediate relapse tendency after withdraw. On the other hand, a number of negative pressure studies have been reported. The influence of upper airway negative pressure change on the respiratory activity of upper airway muscles was examined in rabbits by Mathew (1982a, 1982b, 1984). He hypothesized the presence of a reflex pathway that regulates GG activity in response to the upper airway pressure loads. In 1984, van Lunteren et al. found in dogs that the duration of inspiration

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and the length of inspiratory activity of all upper airway muscles increased in proportion to the amount of negative pressure applied. They concluded that negative pressure in the upper airway inhibits the activity of the diaphragm and preactivates the upper airway dilating muscles. Recently, Kuna (1988a, 1988b) and his associates carried out two different experiments in normal human subjects. From the investigation to determine the effect of nasal occlusion on GG activity, they concluded that subatmospheric pressure in the upper airway and withdrawal of phasic volume feedback may not play an important role in the regulation of upper airway muscle activity in normal sleeping adults. However, Kuna et al. did not exclude completely the possibility of the critical role of the GG even though this role may be very small. Secondly, by means of CT, they compared size of the upper airway area, while applying the CPAP, to its size without the CPAP. Furthermore, Kuna and his co-workers compared EMG activity of the GG between with and without CPAP during wakefulness. They observed no change in phasic and tonic EMG activity between the two protocols. However, they found a linear increase in airway area proportional to the increment in positive pressure.

Issa et al.(1988) examined how sensory information from the upper airway influences the ventilatory and GG muscle response to airway occlusion during different sleep stages. Interestingly, they conducted a nasal occlusion experiment in dogs in the same manner as Kuna et al.. Nasal occlusion caused

a markedly increased GG EMG activity, while tracheal occlusion created a smaller increase in GG activity. Issa and his associates postulated an upper airway protective load-compensation reflex in NREM sleep. Furthermore, they suggested that damage to afferent receptors in the upper airway, caused by the mechanical trauma of snoring might lead to the reduced effectiveness of such a reflex and thus to OSA. Aronson and colleagues (1989) conducted an experiment to examine the effectiveness of nasal CNAP(Continuous Negative Airway Pressure) in normal human subjects. They found that during wakefulness, both diaphragmatic and GG activities increased immediately in response to CNAP. However, during NREM sleep, despite progressive diaphragmatic and GG responses, airway patency was not reestablished until arousal. They are doubtful of rationale of the CNAP hypothesis and presume that the gradual increment of GG activity probably reflects a response to changing blood gas composition and is not due to the mechanical receptor.

C. Biomechanical relationship between postures and airway
    adequacy

1] Head posture

Control of head position is a complex process that integrates information from a variety of sources to generate functionally appropriate motor activity (Schor et al., 1988). Basically, three feedback subsystems relay information about head position by means of different sensory modalities. The

cervicocollic reflex(CCR) subsystem relays somatosensory information to monitor the position of the head with respect to the body;the vestibulocollic reflex(VCR) and optocollic reflex(OCR) subsystems signal movements of the head in space using vestibular and visual signals. These feedback systems undoubtly connect centrally as well as peripherally, and thus contribute to the central voluntary commands as well as to reflex responses. Since the head control system contains a number of feedback loops, it is difficult to distinguish reflex from voluntary responses. The relationship between head posture and craniofacial morphology has long been a research target for anthropologists (Moss and Young, 1960; Riesenfeld, 1967) and orthodontists (Moorrees and Kean, 1958; Bench, 1963; Carlsoo and Leijon, 1960). Solow et al.(1971, 1979, 1984) hypothesized an intimate relationship between head position, airway adequacy and craniofacial morphology. Their findings are also concurrent with Woodside and Linder-Aronson (1976) and Vig et al.(1980). Solow et al.(1979, 1984) postulated that inadquate patency of the nasopharynx induces an extended neck posture. Recently, Liistro et al.(1988) illustrated that head posture influences upper airway resistance and provided evidence that resistance is decreased when the head is extended.

2] Body posture

Issa and Sullivan (1986) observed that some patients exhibited central apnea while asleep in a supine position, but exhibited obstructed apnea or simple snoring when in the lateral recumbent position. According to Parisi and Neubauer (1986), changes in pulmonary mechanics during sleep would occur primarily when changing body position. In the up-right position, inspiratory muscle activation results in increased lung volume via outward movement of the chest wall. However, in the supine position, due to the gravitational effect, the same pattern of muscle activity produces greater abdominal output than chest wall displacement. In addition, the supine position decreases functional residual capacity (Behrakis et al., 1983). A number of experiments support the hypothesis that changes in lung volume directly influence the upper airway flow patency (van de Graaff,1989; Navajas et al,1988). Strohl (1986) explained the increase in upper airway resistance in the supine position by two mechanisms: first, gravity moves the mandible and hypoglossal structures posteriorly, compromising the size of the airway. Secondly, supraglottic resistance increases due in large measure to increased blood in the nasal mucosa (Anch et al.1982). Several groups(Brown et al,1987;Navajas et al, 1988; Fouke and Strohl, 1987) found a decreased pharyngeal cross-sectional area in the supine position. Notably, Fouke and Strohl (1987) have shown that the decreased pharyngeal cross-

sectional area is a result of the change from the up-right to the supine position, independent from the change in the FRC (Functional Residual Capacity). While the presence of a small airway does not appear to determine OSA (Rodenstein et al., 1989), a high Respiratory Disturbance Index indicates its presence. George et al.(1989) further confirm that the RDI(Respiratory Disturbace Index = AHI) is significantly higher in a supine position, in NREM sleep.

STATEMENT OF PROBLEM

Airway obstruction in OSA patients occurs in the supine position. Previous investigators have shown a smaller airway size, an extended head posture and a reduced GG muscle activity in OSA patients. Fouke et al.(1987) compared pharyngeal size in healthy subjects with acoustic reflection techniques and reported that the size of the pharynx was 23% smaller in the supine position when compared to the up-right position. Contraction of the GG muscle advances the tongue base, dilates the upper airway, and decreases airflow resistance. Increased GG muscle activity in the supine position has been reported by several investigators. However, such activity does not always indicate actual muscle shortening, since the muscle shortens only when the neural output produces enough contractile forces to overcome an external load. Airway size measurements from up-right lateral cephalograms can hardly be matched with supine EMG data which approximates the sleeping body position. Few articles have reported size changes of the upper airway and functional changes of the upper airway muscles in the supine position at the same time.

To investigate the relationship between upper airway size and GG muscle activity, the following questions were presented.

1.   How are airway size and positional relationships of the hyoid bone affected by body positional changes in OSA and control groups ?

2.   What do changes in body position affect GG muscle EMG and tongue pressure ?

METHODS

The current experiment was composed of two major parts; a cephalometric study, and tongue EMG and pressure recordings. The cephalometric study was undertaken in twenty patients with OSA and in ten asymptomatic controls. The tongue EMG and pressure study was carried out in the ten asymptomatic controls.

A. Experimental subjects

A total of thirty subjects were evaluated and selected as study material from a population recruited over a ten month period (1988-1989) at the University Hospital (UBC site) in Vancouver. Twenty subjects with diagnosed OSA as determined by overnight sleep studies were used as the patient group. Ten asymptomatic subjects found not to have OSA on the basis of overnight monitoring and/or medical histories were used as controls. The overnight sleep study was carried out at the Respiratory Sleep Disorder Clinic and all anthropometric, pulmonary function and sleep study data used in this study were provided by the clinic. Edentulous subjects, subjects who had an ongoing respiratory infection or any medication known to affect muscle activity, subjects who needed orthognathic surgery and those unable to give fully informed consent were excluded from the study. All individuals included in the study were adult males (see Table I).

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Table I   Demographic Variables for the Experimental Subjects

DEMOGRAPHIC VARIABLES

	OSA (N=20)		CONTROL (N=10)	
	MEAN	S.D.	MEAN	S.D.
AGE	48.4	13.03	33.5	5.95
WEIGHT	97.78	22.64	78.20	11.72
BMI	31.55	7.88	26.30	3.19
RDI	29.91	29.88		

Legend Table I   Age = years, Weight = kg,
BMI(Body Mass Index) = kg/m^2
RDI(Respiratory Disturbance Index)
= Apnea Index + Hypopnea Index

The average age of the twenty subjects composing the OSA group was 48.4 years and ranged from 20 to 71 years (see Table I). Their weights ranged from 69Kg to 150Kg;the mean was 97.8Kg. Average BMI [Body Mass Index = Weight(Kg)/Height^2 (m^2)] was 31.5 (ranging from 24.76 to 47.48). All of the OSA subjects were snorers. The mean AI was 9.99, and the mean RDI was 29.91. The ages of the control subjects ranged from 23 to 46 years;the mean age was 33.5. The average weight of the control group was 78.2Kg and ranged from 62Kg to 95Kg. The average BMI was 26.3, ranging from 21.45 to 32.18.

B. Experimental Procedures

1] Cephalometric study

A pair of individual cephalograms were obtained with identical equipment (Counterbalanced Cephalometer Model W-105, Wehmer Co.) and by an identical method (165 cm source target distance, 14 cm film target distance; 90 kVp, 15mA, 1.25 sec; Kodak 8x10" films, Kodak Lanex Regular Screens) for each subject in the up-right standing position and supine position. All of the cephalograms were taken at the end of the expiration phase.

a. Up-right versus supine lateral cephalograms

To obtain up-right lateral cephalograms, natural head posture was determined by visual feedback in a mirror and recorded by a modified level device (see Fig. 1) attached to the soft-tissue FH plane(infraorbital notch to tragion).

Fig. 1   **Measurement of Head Posture with the Modified Fluid Level Device**

Lead Marker

Tape

Level Device
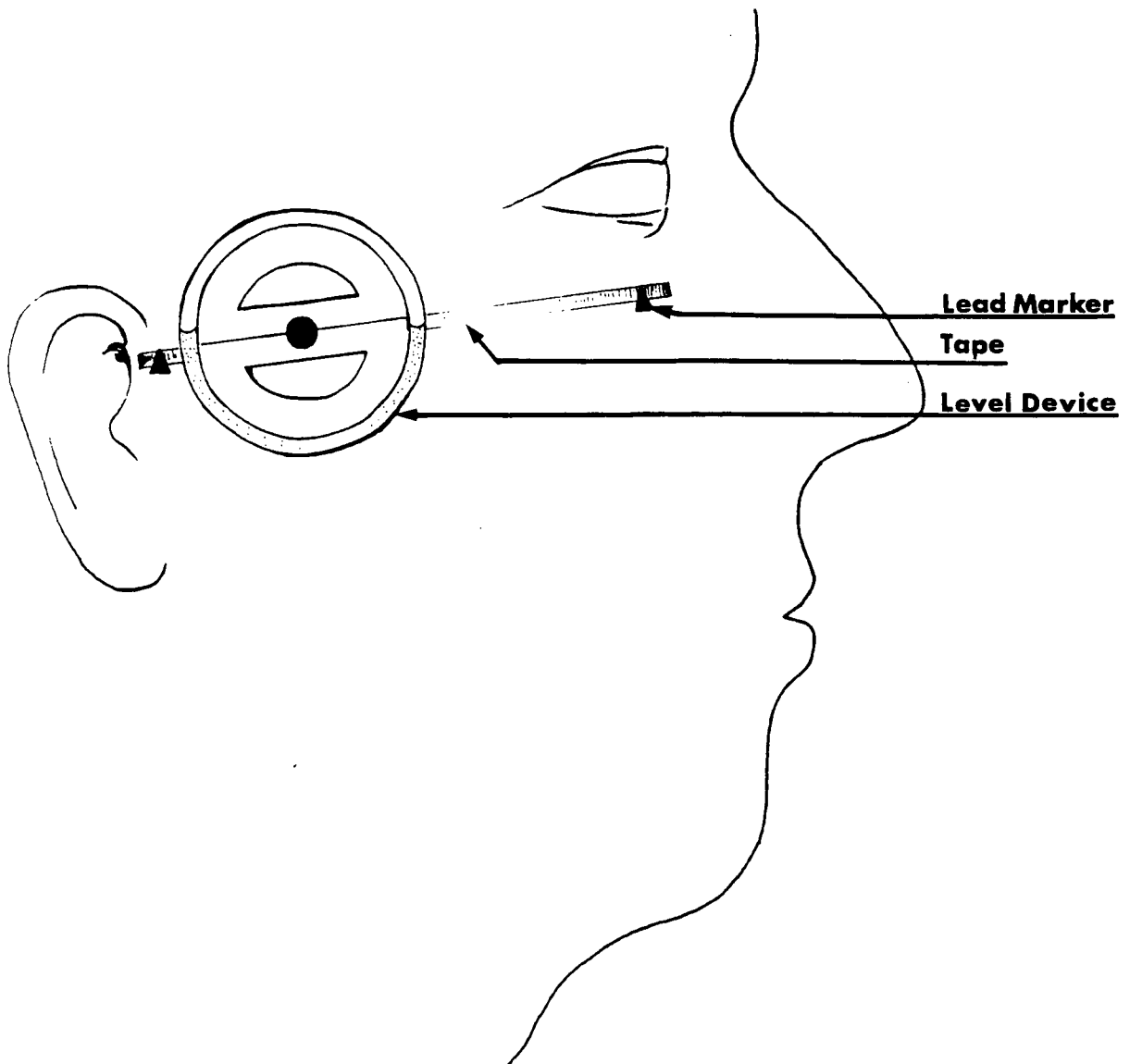
Legend Fig.1   Angle between the upper margin of the tape and the true
horizontal(level of the liquid) represents the natural
head posture.

Natural head posture in the standing position has been considered highly reproducible (Moorrees and Kean, 1958; Siersbæk-Nielson and Solow, 1982; Sandham, 1988). A visual feedback method in standing position was employed for the current experiment. The subject was required to stand 1.5 m away from and in front of the mirror (150 cm long). In a relaxed and natural body posture, the subject was instructed to swing his head back and forth and gradually reduce the magnitude of the swing. Finally, the subject stopped at his own natural head posture determined by his own sense of head balance and sight. This procedure was carried out with the eyes closed first, then the second time with the eyes open and gazing in the mirror (see patient instruction sheet, p30). The degree of the head posture was indicated by the fluid level in the device. This process was repeated 2-3 times, averaged and recorded. A lateral cephalogram was taken of the subject in his own natural posture as previously determined with the modified level device. In order to take the supine cephalogram, the subject was instructed to lie down on a stretcher and to mimick his own usual sleeping position and pillow height. The head angle was recorded after the patient was established in his own comfortable, natural sleeping position. Finally, the supine lateral cephalogram was taken with the jaw in a completely relaxed position.

CEPHALOMETRIC INSTRUCTIONS FOR PATIENTS

You are going to have two head x-rays taken. The first is obtained in a standing position with a neutral head posture, the other is taken in a reclined position.

A. NATURAL HEAD POSTURE

1. Stand on the black line in front of the mirror.

2. Stand in a comfortable position and look directly at your own eyes in the mirror as if you were gazing off into the distance.

3. Close your eyes and nod your head back and forth and gradually reduce the magnitude of the swing. Please stop when you feel you have a natural head posture.

4. Repeat the above task twice with your eyes open.

B. STANDING POSITION

1. Stand on the foot print and reproduce your neutral head posture as instructed.

2. Relax your jaw and bite slightly on your back teeth.

3. After three regular breaths, breathe out, and hold your breath until the x-ray is completed.

C. SUPINE POSITION

1. Lie down on the stretcher and make yourself as comfortable as you can.

2. Please mimick your usual sleeping position. Let your jaw relax as you would when you are asleep. Do not bite on your back teeth.

3. After three regular breaths, breathe out, and hold your breath until the x-ray is completed.

The level device which was employed in the current experiment (see Fig.2) was an improved type modelled on several former level devices (Showfety et al, 1983; Huggare, 1985). This modified level device, developed in consultation with Dr. C. Price of the Divison of Radiology in Faculty of Dentistry at UBC, was composed of three pieces of acrylic plate and an acrylic tube. A microfied circular protractor measuring 360 degrees was attached to the small middle acrylic plate so that an angle could be read up to half a degree in any position. Prescribed saturated radiopaque liquid was held inside the polyvinyl tube to register the true horizontal plane on the film. The liquid was composed approximately of 30% lead nitrate (PbNO3), red dye, surfactant and water.

In order to determine the soft-tissue FH plane, the infra-orbital notch was palpated and tragion was determined. Two lead markers were attached to the infraorbital notch and tragion with an appropriate length of coloured scotch tape. The modified level device was placed on the tape which indicates the soft-tissue FH plane and was adhered to the line with double sided tape (see Fig. 1). After the natural head posture was decided in the manner described above, one tablespoon of the Microtrast Esophageal cream (Esobar, Therapex Inc.) was delivered. The dorsum of the tongue and upper pharyngeal airway were coated with the radiopaque cream to enhance radiopacity of the outline.

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Fig. 2 **Schematic Diagrams to Illustrate Assembly of the Modified Fluid Level Device**

backing for double sided adhesive tape

plastic base for protractor

pivot

plastic tube

radiopaque fluid
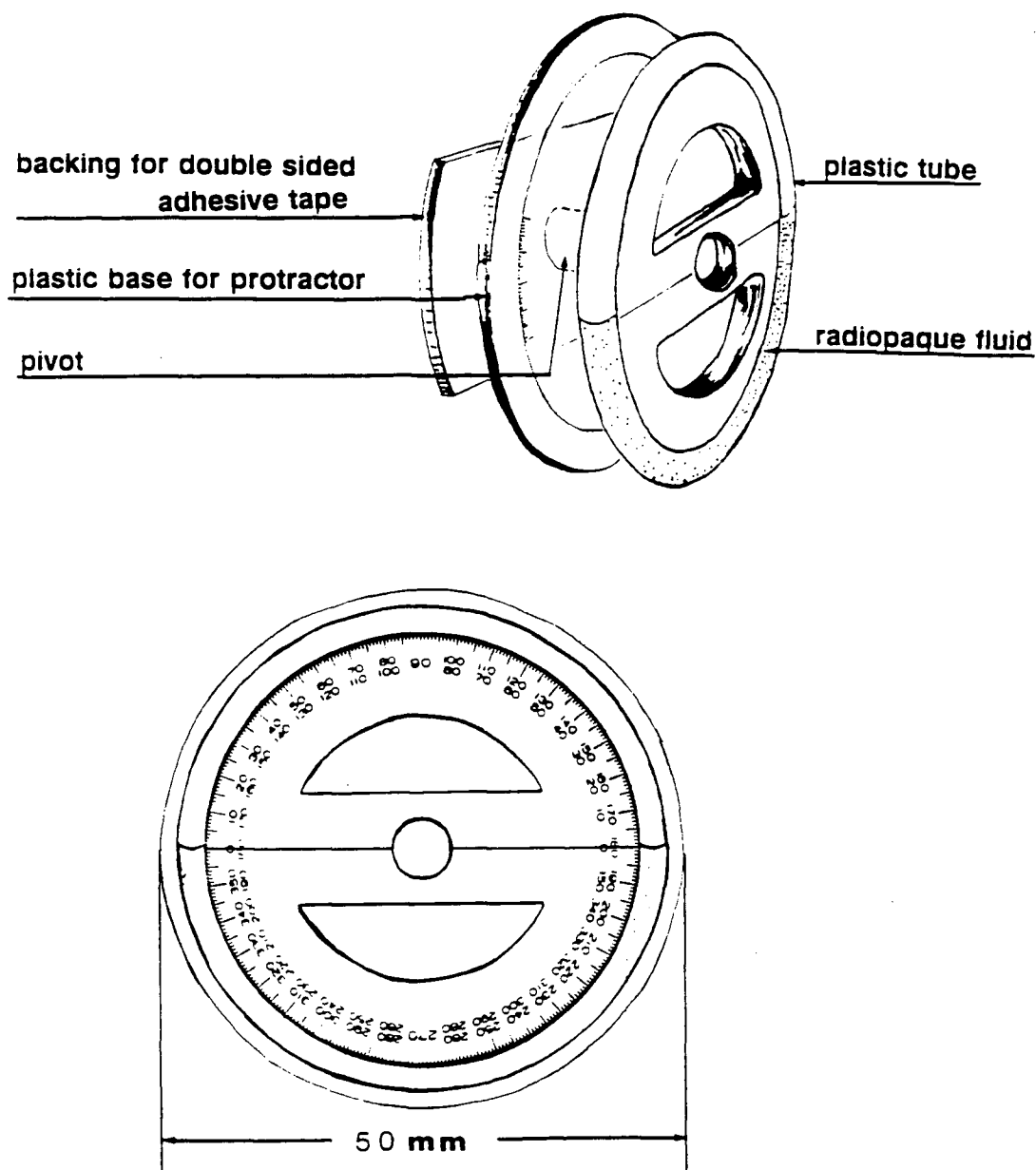
50 mm

Legend Fig.2 The plastic tube(diameter= 3.3 mm) contains $PbNO_3$, red dye, surfactant and water.

Subsequently, a lead marker with a diameter of 5.0 mm was placed on the midpoint of the tongue tip by means of biocompatible adhesives (Iso Dent, Ellman Dental Inc.) in order to register the exact position of the tongue tip. The tongue tip represents the midsagittal junction of the tongue dorsum and the inferior mucous membrane. The subject was instructed to stand on the footprint in the cephalostat and to reproduce his own natural head posture with the aid of the angulation recorded by the modified level device. A metal chain was suspended from the surface of the cassette to confirm and register a true vertical plane on the film. Finally, cephalograms were taken in the instructed position in both the up-right and supine position at end- expiration. Figure 3 illustrates the posture maintained for taking the supine cephalogram.

Fig. 3　　Photograph of the Supine Cephalometric Procedure
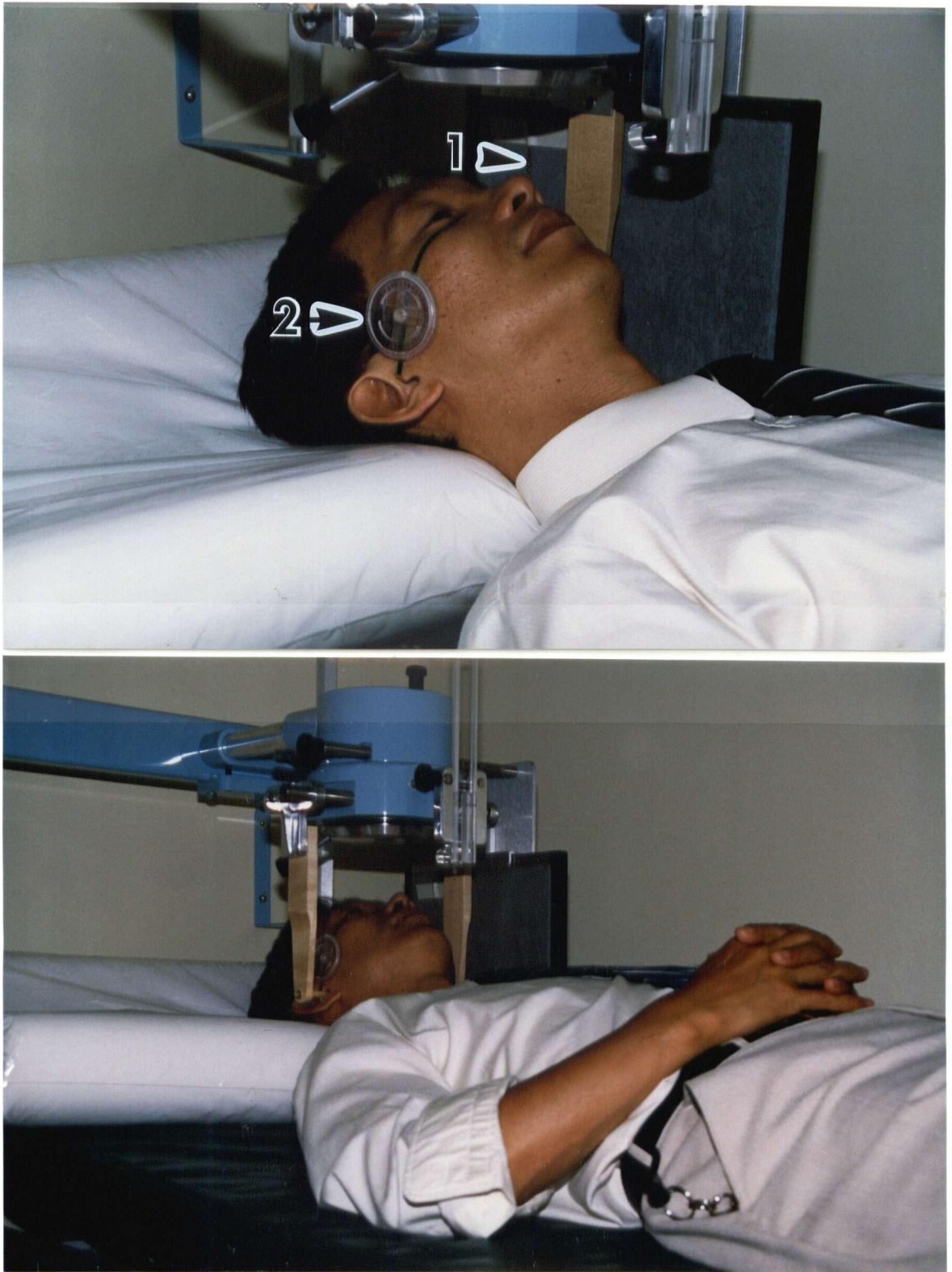
Legend Fig.3　　Arrow 1 indicates the metal chain as a pendulum on a cassette. Arrow 2 indicates the modified fluid level device on the face.

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b. Definitions and rationale of landmarks, planes, lines, angulations, areas and their measurements

In addition to the traditional cephalometric variables, several soft-tissue and vertebral points and lines were used to evaluate the size and location of upper airway structures. Specific landmarks, lines and angles were designed and identified. All lateral cephalometric landmarks were coordinated with X and Y axes, and these two axes were oriented to true horizontal and vertical lines.

1.Hard-tissue landmarks (see Fig.4)

S   Sella - The estimated centre of the sella turcica.

N   Nasion - The most anterior point of the naso-frontal suture.

Or Orbitale - The most inferior point of the infra-orbital margin.

ANS Anterior nasal spine - The apex of the anterior nasal spine.

PNS Posterior nasal spine - The posterior tip of the posterior nasal spine of the palatine bone.

R Roof of the pharynx - The point on the posterior pharyngeal wall constructed by a line PNS to the cross-sectional point of the cranial base and the lateral pterygoid plate.

A point   Subspinale   - The deepest point on the anterior

      surface of the maxillary alveolar bone.

B point   Submentale   - The deepest point on the anterior

      surface of the mandibular symphysis.

Pog   Pogonion   - The most prominent point on the anterior

      surface of the mandibular symphysis in respect to the

      mandibular plane.

Me   Menton   - The most inferior point on the mandibular

      symphysis.

Gn   Gnathion   - The midpoint between Pog and Me on the

      bisecting line of the angle formed by mandibular plane

      and facial plane.

RGN   Retrognathion   - The most posterior point of the

      mandibular symphysis along a line perpendicular to

      the FH (Frnkfort Horizontal) plane.

Go   Gonion   - The most inferior, posterior and outer most

      point of the mandibular angle, determined by a bisector of

      the angle formed by the tangent to the posterior and

      inferior border line of the mandible.

Po   Porion   - The uppermost point of the ear rod.

CV2tg   Second   Vertebra Tangent   - The most posterior and

      superior point on the posterior surface of the second

      vertebral corpus.

CV2ip   Second Vertebra Inferior Posterior   - The most

      posterior and inferior point of the second vertebral

      corpus.

CV4ip   Fourth Vertebra Inferior Posterior   - The most
        posterier point of the fourth vertebral corpus.

C3   Third Vertebra   - The most anterior inferior point of the
     third vertebral corpus.

C4   Fourth Vertebra   - The most anterior inferior point of the
     fourth vertebral corpus.

Fig. 4 Cephalometric Hard Tissue Landmarks
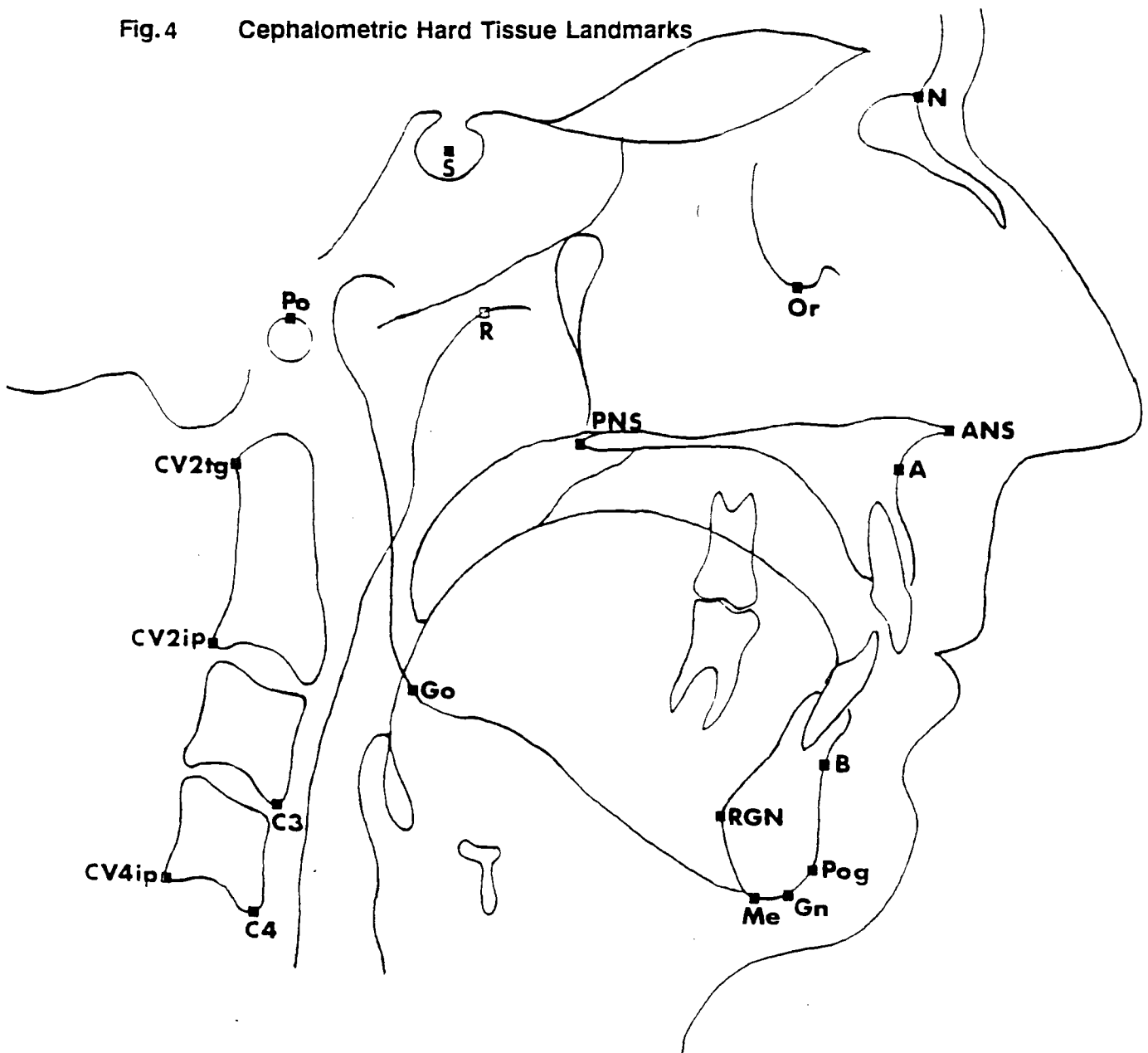
Legend Fig. 4 S(sella), N(Nasion), Or(Orbitale), ANS(Anterior Nasal Spine), PNS(Posterior Nasal Spine), R(Roof of the pharynx), A(Subspinale), B(Submentale), Pog(Pogonion), Me(Menton), Gn(Gnathion), RGN(Retrognathion), Go(Gonion), Po(Porion), CV2tg(Second Vertebra Tangent), CV2ip(Second Vertebra Inferior Posterior), CV4ip(Fourth Vertebra Inferior Posterior), C3(Third Vertebra), C4(Fourth Vertebra)

2. Soft-tissue landmarks (see Fig.5)

a) Tongue

TT   Tongue Tip - The center of the lead disc attached to the border between the ventral and dorsal surfaces of the tongue tip.

TH   Tongue Height - The highest point of the tongue curvature relative to a line from base of the epiglottis to TT.

Eb   Base of Epiglottis - The deepest point of the epiglottis.

Et   Tip of Epiglottis - The most superior point of the epiglottis.

H   Hyoidale - The most anterior and superior point of the hyoid bone.

b) Soft palate

P   Palate Point - The most inferior tip of the soft palate.

AP   Anterior Palate - The anterior point of maximum palatal thickness determined along a line perpendicular to a line from PNS to P.

PP   Posterior Palate - The posterior point of maximum palatal thickness determined along a line perpendicular to PNS-P.

AST   Anterior Superior Tongue - The cross-sectional point on the tongue curvature determined by a line through the mid-point of PNS-P and parallel to Go-B.

ASP   Anterior Superior Palate - The anterior cross-sectional point of the soft palate determined by a line bisecting PNS-P and parallel to Go-B.

PSP   Posterior Superior Palate - The posterior cross-sectional point of the soft palate determined by a line bisecting PNS-P and parallel to Go-B.

PSPh Posterior Superior Pharynx - The cross-sectional point of the posterior pharyngeal wall by a line bisecting PNS-P and palallel to Go-B.

c) Pharyngeal airway

MAA   Middle Anterior Airway - The anterior point on the tongue on a line through point P parallel to Go-B.

MPA   Middle Posterior Airway - The point on the posterior pharyngeal wall on a line through point P and parallel to Go-B.

IAA   Inferior Anterior Airway - The anterior cross-sectional point on the posterior surface of the tongue or soft palate determined by a extended line of Go-B point plane.

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IPA Inferior Posterior Airway - The posterior cross-sectional

point on the posterior pharyngeal wall determined by the

line Go-B.

Fig. 5   Cephalometric Soft Tissue Landmarks
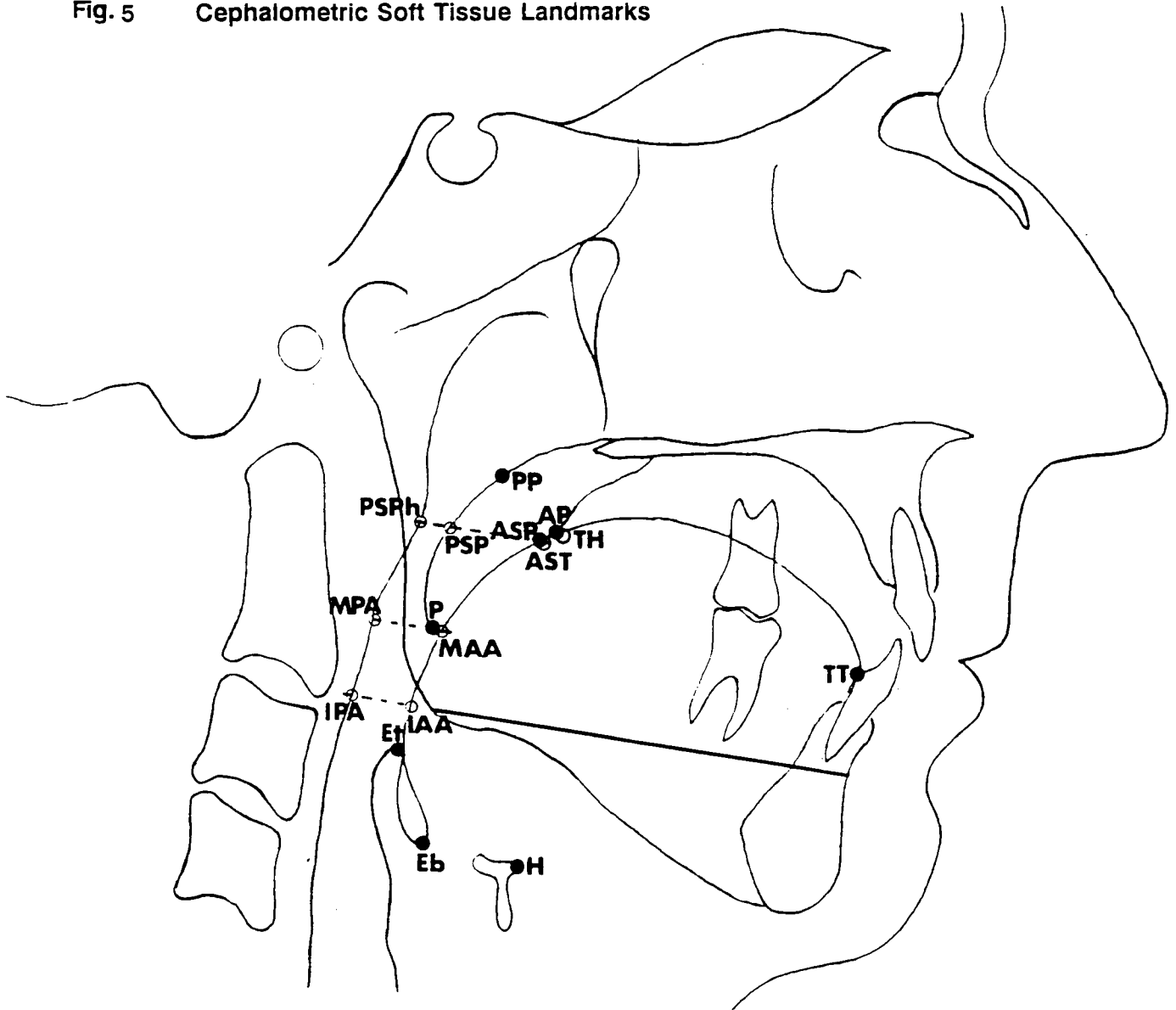
Legend Fig.5   TT(Tongue Tip), TH(Tongue Height), Eb(Base of Epiglottis), Et(Tip of Epiglottis), H(Hyoidale), P(Palate Point), AP(Anterior Palate), PP(Posterior Palate), AST(Anterior Superior Tongue), ASP(Anterior Superior Palate), PSP(Posterior Superior Palate), PSPh(Posterior Superior Pharynx), MMA(Middle Anterior Airway), MPA(Middle Posterior Airway), IAA(Inferior Anterior Airway), IPA(Inferior Posterior Airway)

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Several linear, angular and area variables were determined.

3. Linear measurements (see Fig.6)

a) Tongue

TGL   Tongue Length - The linear distance between TT and Eb.

TGH   Tongue Height - The linear distance between a point on the most superior curvature of the tongue dorsum and the base of a line drawn perpendicular to the TT-Eb line.

b) Soft Palate

PNS-P   Soft Palate Length - The linear distance between PNS and P.

MPT   Maximum Palate Thickness - The maximum thickness of the soft palate measured on a line perpendicular to the PNS-P.

c) Upper Airway

SPAS   Superior Posterior Airway Space - The thickness of the airway behind the soft palate along a line parallel to the Go-B point plane.

MAS   Middle Airway Space - The thickness of the airway along a line parallel to the Go-B point Plane through P.

IAS   Inferior Airway Space - The thickness of the airway along a line extended through the Go-B point plane.

VAL   Vertical Airway Length - The linear distance between
           PNS and Eb.

d) Hyoid Bone
  MPH   Mandibular Plane to Hyoid - The linear distance along
           a perpendicular from H to the mandibular plane.
  HH1   Vertical Hyoid - The linear distance between H and a
           perpendicular to the C3-RGN plane.
  HRGN   Horizontal Hyoid - The linear distance between H and
           RGN.
  C3H   Vertebral Hyoid - The linear distance between C3 and
           H.

Fig. 6   Cephalometric Linear Measurements
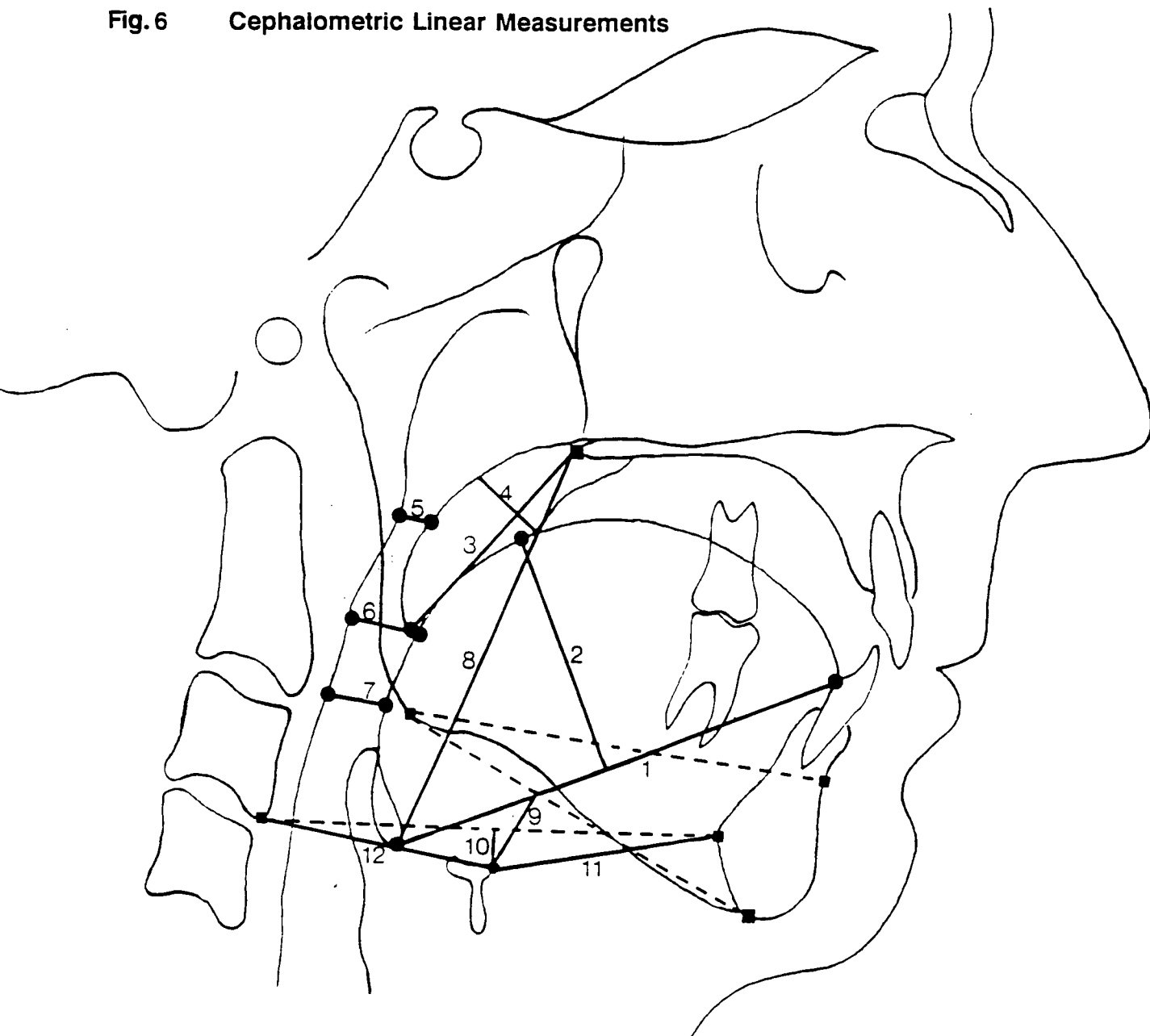
Legend Fig. 6   1:TGL, 2:TGH, 3:PNS-P, 4:MPT, 5:SPAS, 6:MAS, 7:IAS
8:VAL, 9:MPH, 10:H-H1, 11:HRGN, 12:C3H

4. Angular measurements (see Fig.7)

E-TT Tongue Angle - The angle constructed by an extension of the Eb-TT plane and the true horizontal.

CVTPP Vertebrae to Palatal plane - The angle constructed by an extension line of CV2tg-CV4ip plane and palatal plane .

CVTSN Vertebrae to SN plane - The angle constructed by an extension line of CV2tg-CV4ip plane and SN plane.

OPTPP Odontoid to Palatal plane - The angle constructed by an extension line of CV2tg-CV2ip plane and Palatal plane.

OPTSN Odontoid to SN plane - The angle constructed by an extension line of CV2tg-CV2ip plane and SN plane.

5. Area measurements (see Fig.7)

a) Tongue The area outlined by the dorsal configuration of the tongue surface and lines which connect TT, RGN, H and Eb.

b) Soft Palate The area confined by the outline of the soft palate which starts and ends at PNS through P.

c) Nasopharynx The area outlined by a line between R and PNS, an extension of the Palatal plane to the posterior pharyngeal wall, and the posterior pharyngeal wall.

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d) Oropharynx   The area outlined by the inferior border of the nasopharynx, posterior surface of the soft palate, a line from P to the dorsal surface of the tongue parallel to the palatal plane, the posterior inferior surface of the tongue, a line parallel to the palatal plane through the point Et, and the posterior pharyngeal wall.

e) Hypopharynx The area outlined by the inferior border of the oropharynx, the posterior surface of the epiglottis, a line parallel to the palatal plane through the point C4, and the posterior pharyngeal wall.

Fig. 7   Cephalometric Angular and Area Measurements
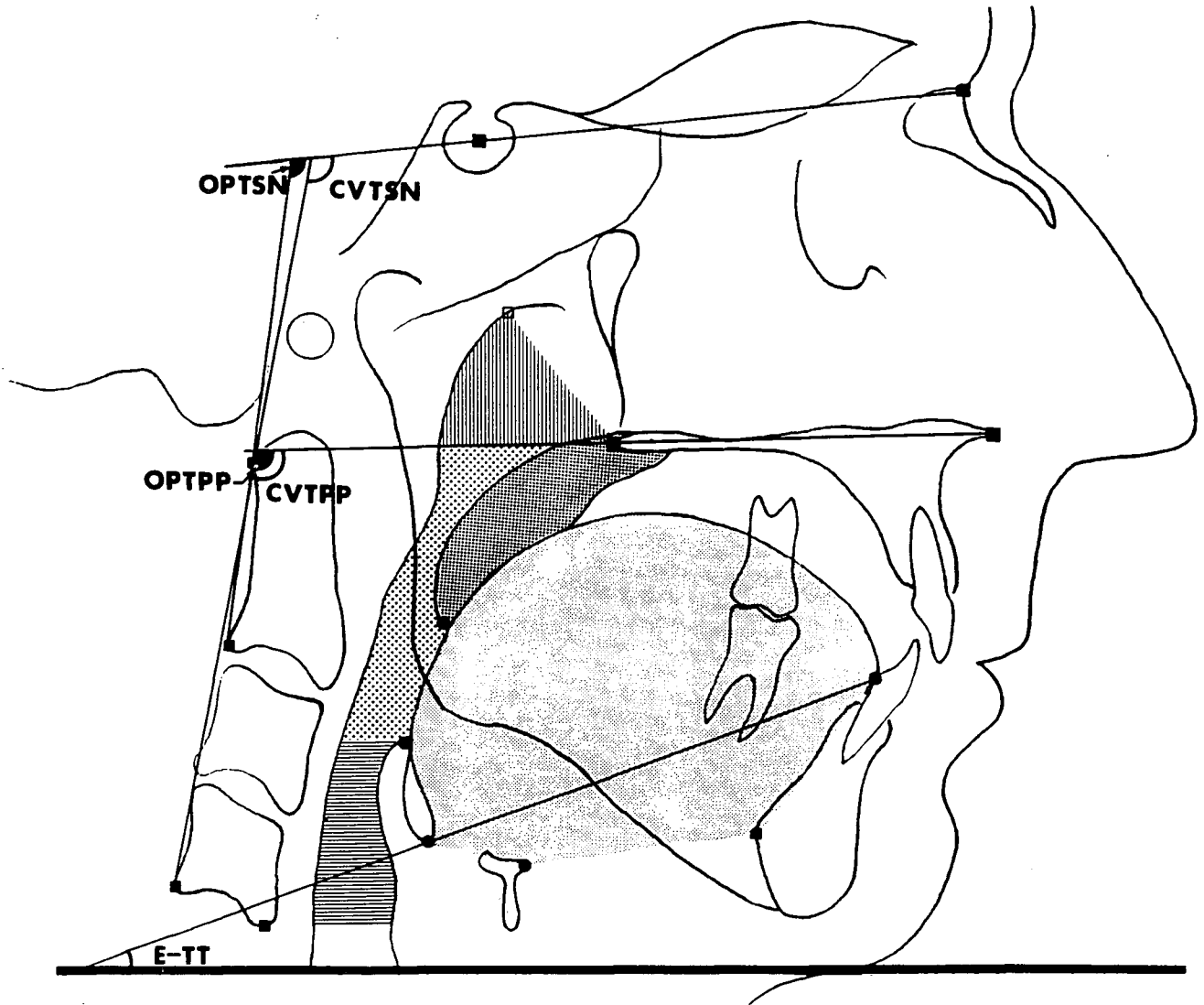
Legend Fig.7   E-TT(Tongue Angle), CVTPP(Vertebrae to Palatal plane),
CVTSN(Vertebrae to SN plane), OPTPP(Odontoid to Palatal
plane), OPTSN(Odontoid to SN plane),
Tongue,
Soft Palate,
Nasopharynx,
Oropharynx,
Hypopharynx

C. Tracing and digitization

Tracings were made on acetate paper with a .5mm pencil for each of the points, planes and outlines for the tongue, soft palate and upper airway structures by one investigator. Boundaries were outlined in the middle of tissue transition zones to take into account averaging. A data entry program was written to permit digitization of cephalograms by means of a digitizer (HP Model 9874). A cross-hair cursor was used to enter the points and contours of each structure into the computer (HP 1000E series). Analysis programs were written to determine the length, angulation and cross-sectional area of specific structures. All of the digitization procedures were fulfilled by one investigator.

2] EMG and pressure study

EMG and pressure differences of the tongue and perioral musculature in different body positions were investigated. Each of three tasks (i.e. rest, maximum protrusion, maximum opening) was performed on 10 asymptomatic controls in up-right and supine body positions. The computer stored signals every millisecond during the two seconds of the actual sampling time, after all 2000 signals were detected. The signals were integrated and averaged by existing computer software.

a. Data aquisition system and recording technique

A total of six channels were used in order to obtain EMG and pressure signals: four channels for the GG, masseter, orbicularis oris and supra-hyoid muscle group and two channels for the anterior and posterior pressure recordings of the tongue (see Fig.8).

channel 0 :EMG activity of the GG was taken intra-orally with a ball-type bipolar surface electrode.

channel 1 :EMG activity from the right-side masseter was taken with conventional surface electrodes.

channel 2 :EMG signals from the right-side inferior orbicularis oris muscle were detected by conventional surface electrodes.

channel 3 :EMG activity from the right-side supra-hyoid muscle group was taken with conventional surface electrodes.

channel 10:Pressure signals from the anterior portion of the tongue were taken with a strain gauge type load cell positioned in acrylic appliance at the midpoint of the central incisors.

channel 11:Pressure signals from the posterior portion of the right side of the tongue were taken with the load cell positioned in acrylic appliance opposite the right mandibular first molar.

Fig. 8 Schematic Illustations of the Surface Ellectrodes for the Masseter, Supra-hyoid and Orbicularis Oris Muscles, and the Intraoral Appliance

pressure transducers

acrylic resin portion of the appliance

EMG electrodes

rubber base portion of the appliance
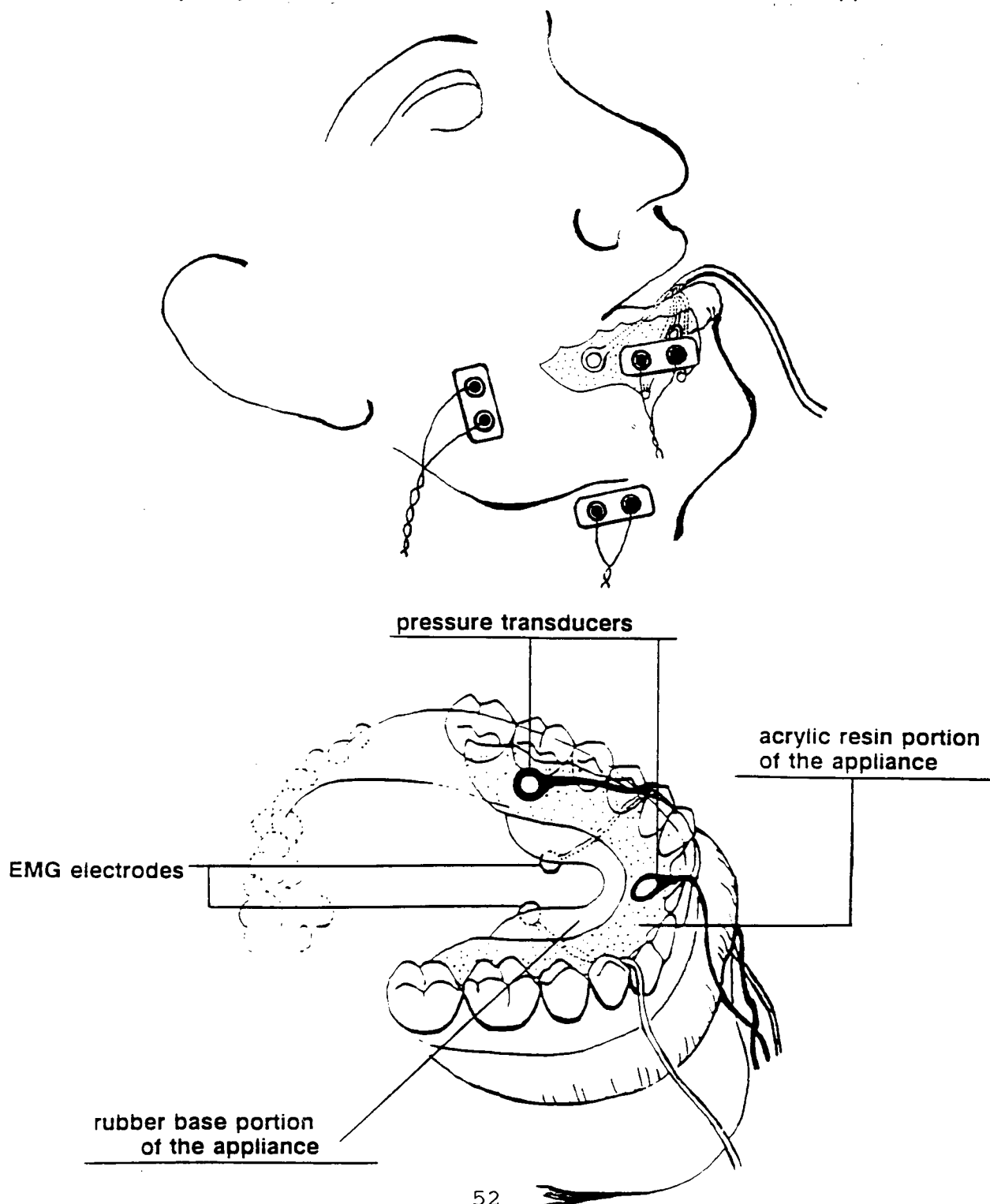
A customized acrylic and rubber base appliance was constructed as a carrier on the individual mandibular cast and adjusted in the mouth (see Fig.8). After a determined thickness(2 mm) of resin plate in the dough stage was applied on the lingual side of the mandibular teeth and gingiva on the cast, a small amount of rubber base material (Reprosil putty 1500, Densply) was added to the resin, adjusted and molded. Two custom made ball-type electrodes were embedded on each side of lingual flange of the appliance to record directly from the GG muscle (Doble et al., 1985; Milidonis et al., 1988). The pressure transducers were mounted at the midpoint of the mandibular central incisors and lingual to the right mandibular first molar. The right mandibular molar region was thickened in order to exaggerate differences in tongue pressure in accordance with body positional change. The thickness of the appliance and the location of the transducers were carefully standardized and the sensitivity of each transducer was determined and calibrated at proper gain. A diaphragm type of load cell, manufactured by Kyowa electronic instruments Co., which contains four electrically connected strain gauges (i.e. a wheatstone bridge) was used to record tongue pressure. The miniature pressure transducer was a PS-A type, with a $10Kgf/cm^2$ of capacity, 6 mm in diameter and 0.6 mm thickness. Specification details are provided in Figure 9.

Fig. 9   Pressure Transducer and Its Specifications

Glossy surface / Pressure

$\phi 6$

0.6 ± 0.1

(mm)
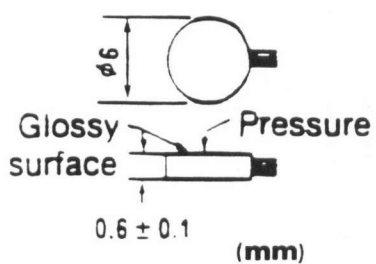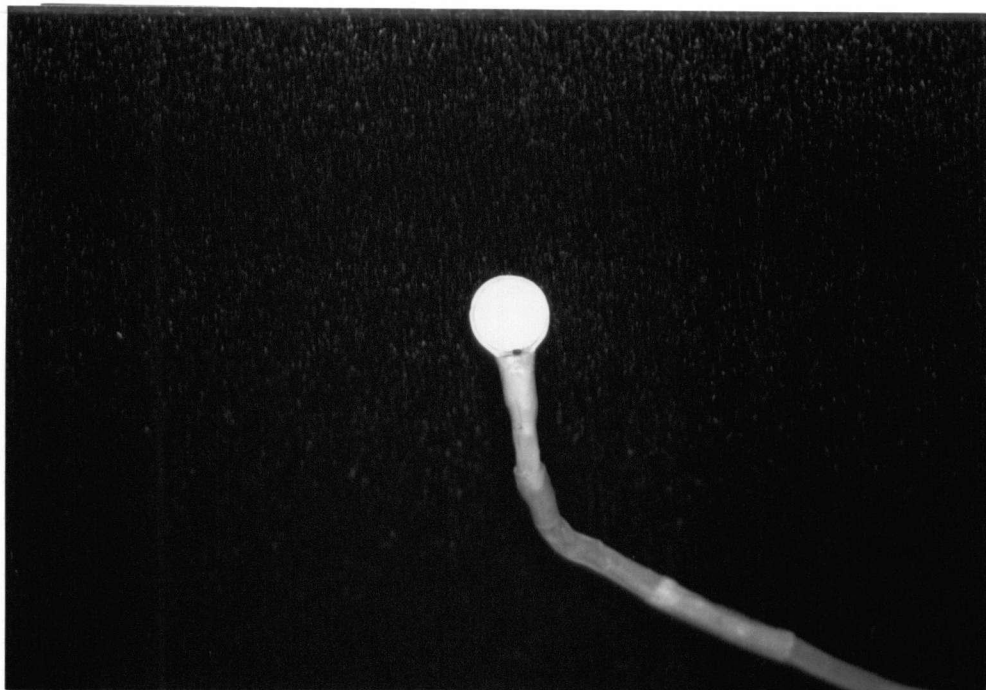
(Type PS-A)

Specifications

Type	**PS-A**
Safe excitation	3V
Input/output resistance	120Ω
Compensated temperature range	0-+50·C
Safe temperature range	-20-+70·C
Temperature effect on output	±0.2%/·C
Safe overload rating	150%

A static calibration was undertaken with a calibration system for each of the transducers in vitro before and after the experimental session (see Fig.10). The calibration system was set up with a dental plaster housing and a rubber ballon containing water with no bubbles at a temperature approximately 36°C. Weights were set at 100 g intervals on the calibration system. The noise levels were less than 5mV per min. The adjusted sensitivity of both transducers was approximately 1.5g/cm^2 in both channels and a linearity was established (see Fig.10). The linearity of sensitivity in load cells was represented by a correlation coefficient (anterior side r=0.9878; posterior side r=0.9954). Both of the linearities were calculated from 0 to 400g in force changes on the given surface area. Based upon the regression line of the scattergram (see Fig.10), 1mV of electrical change in the load cell turned out to be equivalent to approximately 1.5g/cm^2 of pressure change as an average for both transducers.

Fig. 10   Calibration System and Calibration Results

Weight
Syringe
Gypsum
Balloon
Water
Pressure Transducer

● **Anterior Transducer**   ■ **Posterior Transducer**
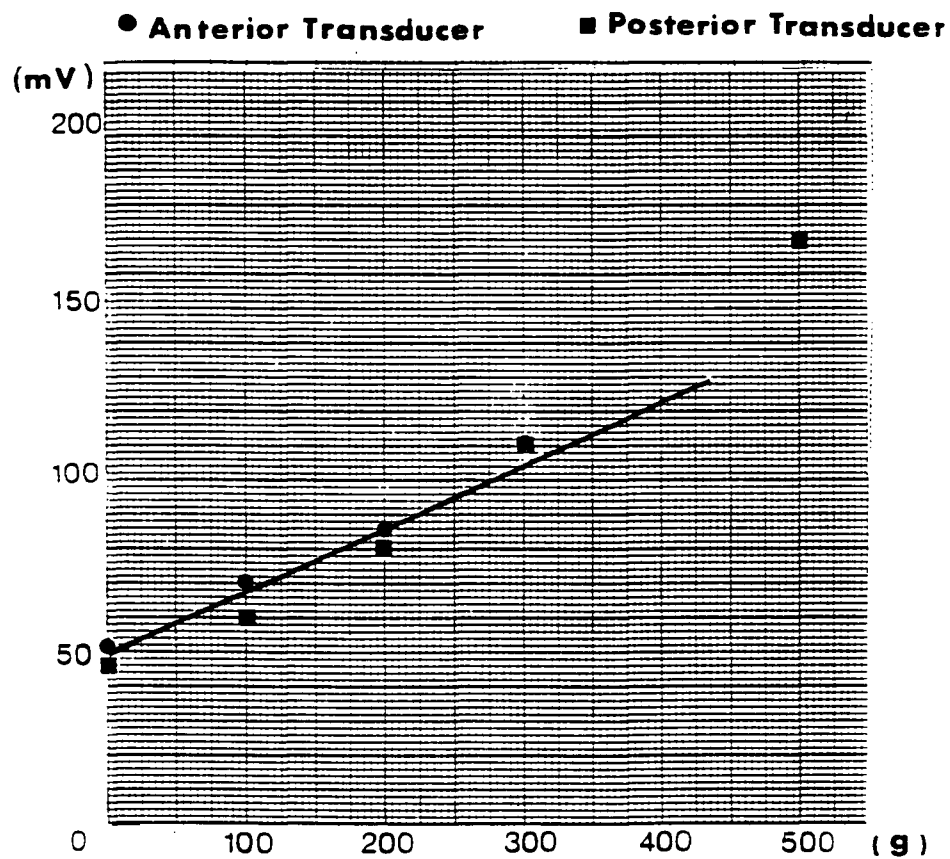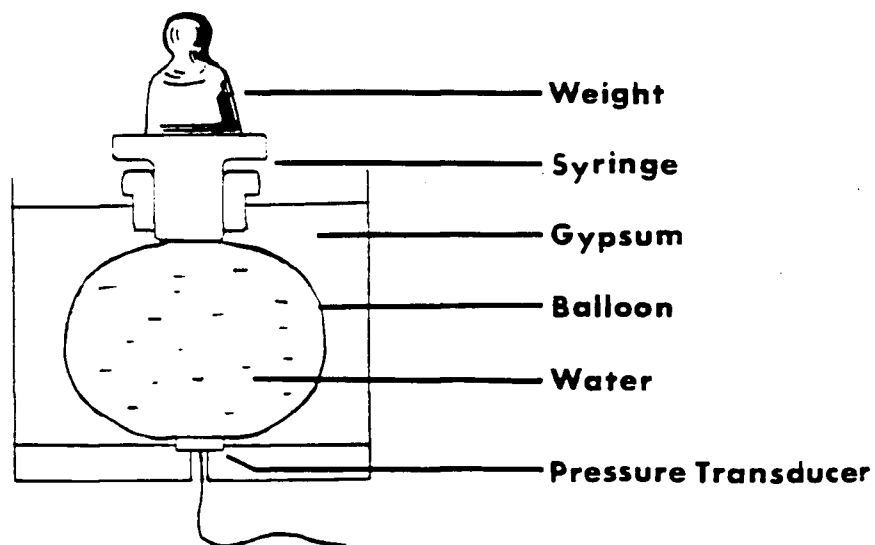
Legend Fig.10   The obtained pressure signals from the pressure
transducer are delivered to Digital Multimeter(Hewlett
Packard) and displayed as numbers(mV).

b. Equipment and data processing (see Fig.11)

EMG data from the GG, masseter, inferior orbicularis oris and supra-hyoid muscles were passed through a differential amplifier (AI 2010 Axon). Each of signals was amplified by dual amplification(mainly 20K or 40K was used) and low(30 Hz) and high(1KHz) filterings were conducted in order to reduce the noise from movement artifacts and electrical high frequency coming from other equipment. The amplified signals were delivered to an eight channel multiplexor(4701 Tektronix inc.) and a control rack. Through a trigger system, the signals were passed into an A/D converter(3852A DATA ACQUISITION/CONTROL UNIT). After conversion of the signals, they were integrated and averaged by means of the pre-programed computer software and stored in the computer. The signals from the pressure transducers were first passed into a custom-made carrier preamplifier through a bridge box. The signals were amplified by pre-adjusted and calibrated gain and were sent to the storage monitor (2221 Tektronik) to verify the tongue forces. The signals were sent to the A/D converter through the same trigger and the rack. The data transformed to digital form was averaged and stored in the same manner as the EMG signals.

Fig. 11   Flow-chart for Data Aquisition

Pressure Transducer

EMG Electrodes

Carrier pre-amplifier

Differential
Amplifier

Storage
Oscilloscope

8-channel
Multiplexor

Trigger

A/D Converter

Computer
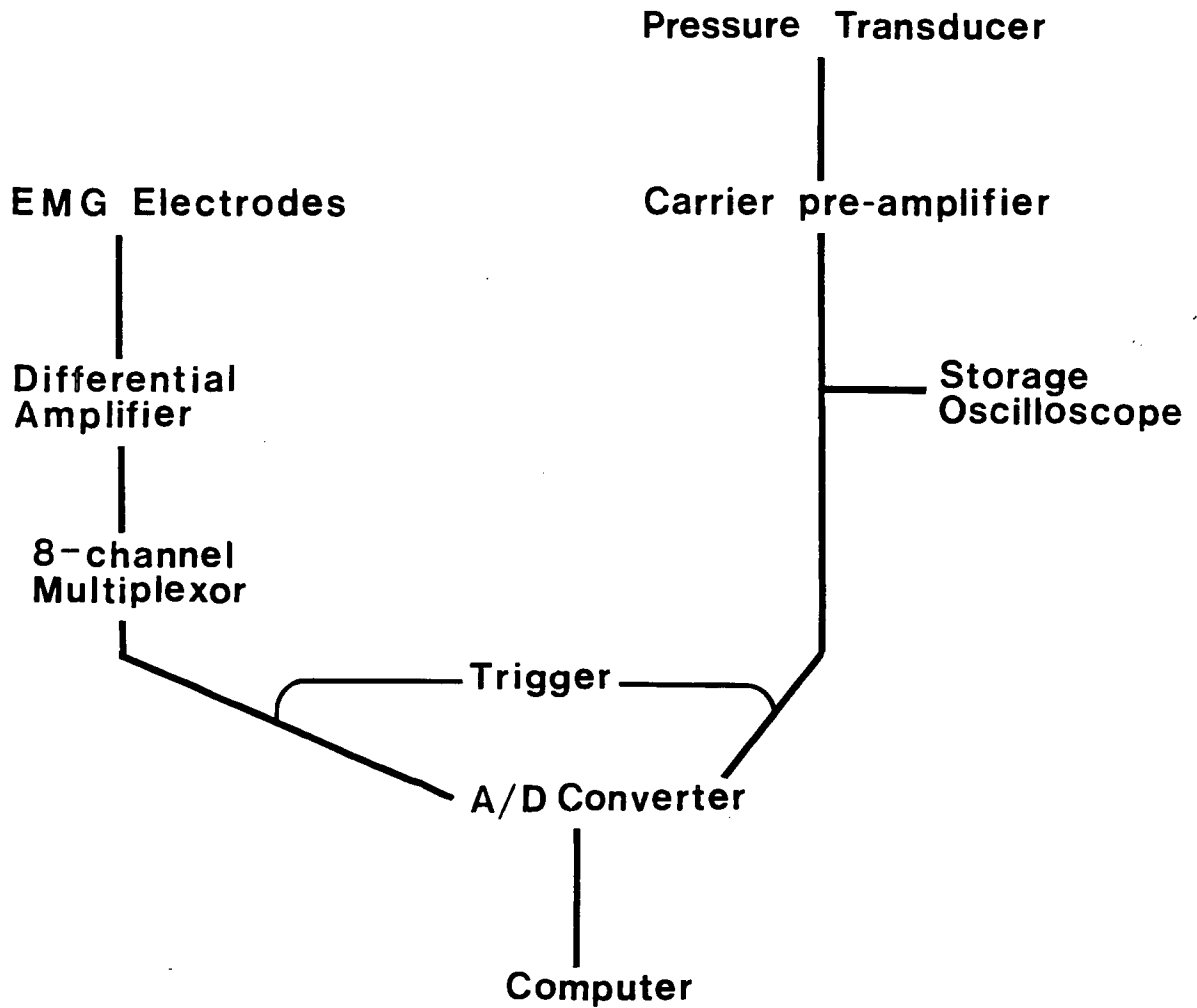
Legend Fig.11   All signals are displayed on the 8-channel multiplexor
and the storage oscilloscope.

c. Experimental procedure

To sample the EMG and pressure data for the rest task, the patient was required to maintain a relaxed and resting mandibular position to monitor resting potentials in the GG, masseter, inferior orbicularis oris and supra-hyoid muscles. To record the protrusion task, the subjects were instructed to perform a maximum protrusion of the tongue against the pressure transducer on the anterior lingual side of the appliance. To record maximum opening, the subjects were requested to open their mouth as much as possible. To sample the tongue baseline pressure, a specially constructed acrylic tray was introduced intra-orally (see Fig.12). The subjects were instructed to sit in a dental chair in an up-right position with natural head posture which could be reproduced by the modified level device. The tray was delivered intra-orally in order to take the base line recording of the tongue pressure without any contact of the tongue on the transducer surface. After the tray was removed from the mouth, EMG and pressure were recorded at the absolute rest position of the mandible. Maximum protrusion and opening tasks were recorded immediately in a consecutive manner. Each of the tasks was performed in not less than three seconds. Four recording sessions of base line, rest, maximum protrusion and maximum opening were repeated five times. Body movement, head movement and swallowing were monitored throughout the session. After the up-right position session, the subjects were requested

to recline on a stretcher in a position identical to that used for the supine cephalograms. All tasks were repeated in the same manner as in the up-right position.

Fig. 12   Tray Used to Measure the Base-line Pressure of the Tongue
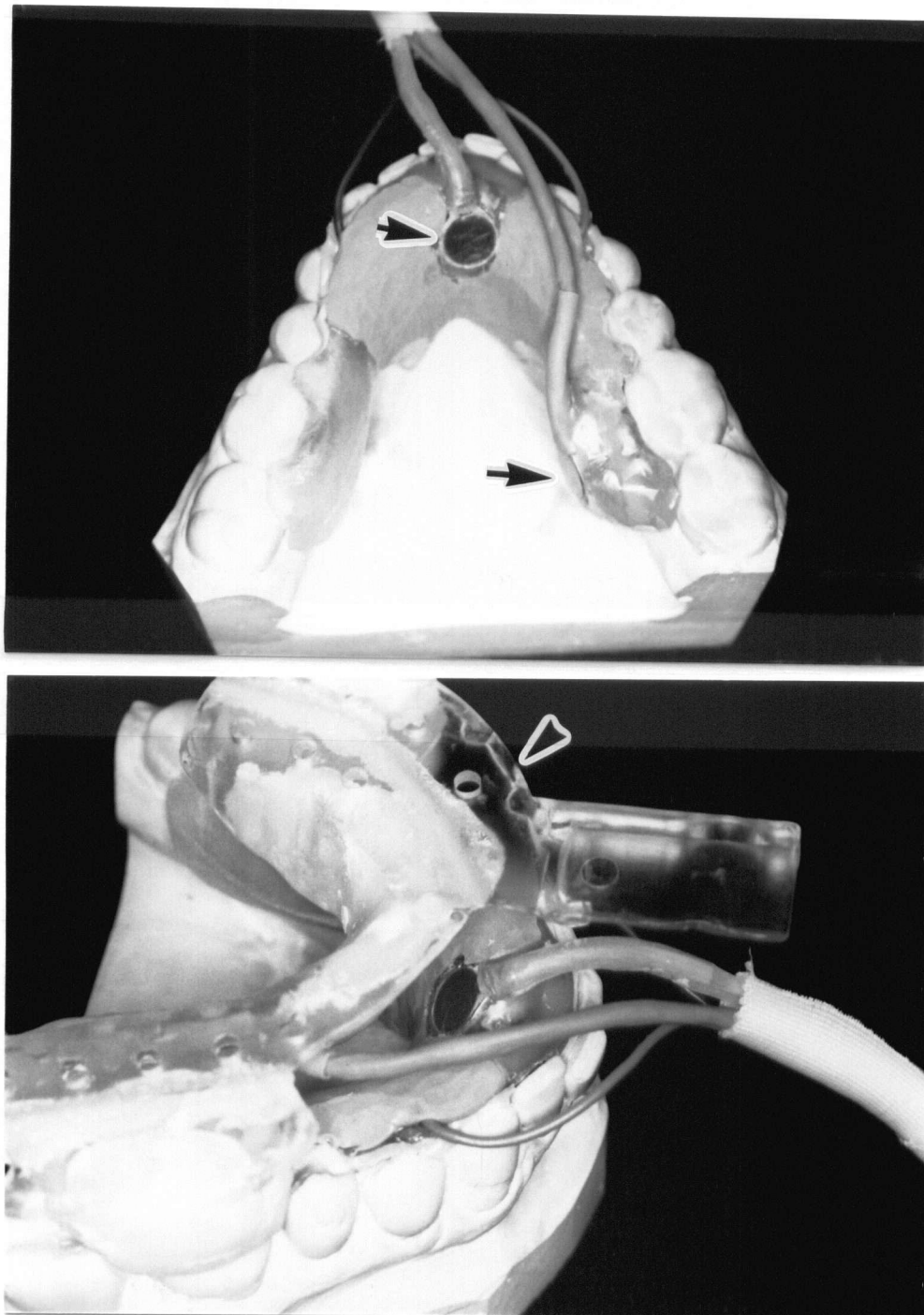
Legend Fig.12   Arrows in the upper picture indicate the pressure transducers, the arrow in the lower picture indicates the acrylic tray. Notice the free space between the tray and the appliance.

C. Statistical method

All the statistical tests were conducted by means of the computer statistical package called SYSTAT. A two-tail test and 5% significant level was used for detecting false positives(Type I error). The Student's t test and Wilcoxon signed rank test were employed for the comparison between the OSA group and the asymptomatic controls, and also for the comparison between the up-right and supine body position. Pearson's simple correlation coefficient, Dahlberg's method error, Houston's reliability test and two-way ANOVA were used to calculate reproducibility and experimental error. Additionally, ANCOVA was conducted in order to test the effects of age on the variables.

The index of reliability, used to test the reliability of the cephalometric variables, was introduced by Houston in 1979. He classified types of error into systemic error (or bias) and random error. The simplest approach to detect systemic errors is the one sample t test for each pair of replicates. In general, at least 25 cases should be replicated for detection of systemic errors arising in obtaining lateral cephalograms (Houston, 1979). On the contrary, random errors can arise as a result of variation in positioning of the patient in the cephalostat, faulty identification of the landmarks and inaccuracy of digitization. Houston emphasizes that Dahlberg's formula ($s = \sqrt{\Sigma d^2 / 2(n-1)}$) which has been traditionally used for

the calculation of the method error, could not discriminate systemic errors from random errors. Therefore, he suggests a new coefficient of reliability and index of reliability(τ) which represent pure random error. The formula is; $\tau = \sqrt{1 - Sd^2/Sg^2}$, where τ is index of reliability, Sd^2 is the variance of differences between replicates, and Sg^2 is the greater variance between two of the replicate variances.

The Student's t test has traditionally been accepted as a robust parametric method. The t test examines variance first and determines the formula to be used later on (Armitage and Berry, 1987). In cases of equal variance, the formula of the t test is $t = X_1 - X_2/SE(X_1 - X_2)$, following t-distribution on $n_1 + n_2 - 2$ degree of freedom. In cases of unequal variance and if the original means of the two samples are not too different, it would be difficult to find a proper transformation form which substantially reduces this parity between the variances. Therefore, a pooled estimate of variances was utilized.
$d = X_1 - X_2/\sqrt{(S_1^2/n_1 + S_2^2/n_2)}$ could be used, which is approximately a standardized normal deviate if n1 and n2 are resonably large.

The Wilcoxon signed rank test was employed for the comparison study (EMG and pressure), since the sample size was rather small. The Wilcoxon signed rank test is a distribution free method (Armitage and Berry, 1987). The observations are put in ascending order of magnitude, ignoring the sign, and give the ranks 1 to n'. Let T+ be the sum of the ranks of the positive values and T- that of the negative. On the null

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hypothesis T+ and T- would not be expected to differ greatly; their sum T+ and T- is 1/2n'(n'+1), so an appropriate test would consist in evaluating the probability of a value of T+ equal to or more extreme than that observed. For large values of n', T+ and T- are approximately normally distributed with variance n'(n'+1)(2n'+1)/24 with continuity correction which is given by [T+ - 1/4n'(n'+1)]-1/2 / $\sqrt{}${n'(n'+1)(2n'+1)/24}.

If there are numerically tied variables, they are given tied ranks and reduced variance of T+ by $t(t^2+1)/48$, where t= number of ties. However, Armitage and Berry (1987) commented that the sign test loses something by ignoring all information about the numerical magnitudes of the observations other than their signs. If a high proportion of large observations were positive this would strengthen the evidence that the distribution was asymmetrically above zero.

Two-way analysis of variance(Two-way ANOVA) was employed to investigate the repeatability of natural head posture measurement with the fluid level device. Armitage and Berry (1987) stated that the purpose of the analysis of covariance(ANCOVA) is to correct bias and to reduce random variation. In the present study, the ANCOVA was used, given that a comparison at the same age would have been more desirable.

RESULTS

A statistical analysis was carried out on the results of the comparative study of the up-right and supine positions in the OSA and asymptomatic control groups. Results were obtained from cephalometric, EMG and pressure measurements. Particularly, the cephalometric comparison study of the up-right and supine positions provided several significant pieces of information regarding the anatomical changes that occur in conjunction with body positional changes. The EMG and pressure results also yielded interesting results in agreement with those of the cephalometric study. The results are reported as non-transformed data(neither log nor square root) and are summarized as measurements which are statistically significant at $p<$ .05 level denoted by one asterisk or $p<.001$ by two asterisks. All of the linear cephalometric measurements were enlarged by 8.5% and no correction of the enlargement effect was attempted.

A. Reliability tests

Reliability and reproducibility tests for the digitizing procedure, soft-tissue tracing and digitizing, measurement of the natural head posture, and validity test for the fluid level device were performed.

The random error of the digitizing procedure was evaluated. Two points on the same cephalogram were digitized twice with enough of a time interval to avoid digitizing fatigue. For representation of the digitizing random error, Pearson's simple correlation coefficient (r) and Houston's reliabilty index were employed. An example of the calculation for the intra-examiner random error is as follows:

	1st time	2nd time	Difference
MP-H	14.4	14.5	0.1
H-RGN	41.0	41.0	0.0
PNS-P	56.5	56.5	0.0
TGL	86.6	86.4	0.2
MEAN	49.62	49.60	0.08
SD	30.16	30.04	0.10
Variance	909.63	902.40	0.09
r	1.000		

Index of reliability $\tau = 0.9985$

As a result of the intra-examiner error, r=1.000 and $\tau=0.9985$ were obtained; as a result of the extra-examiner error, r=0.9670 and $\tau=0.9614$ were observed. From these results, the digitizing method, as used in the current study, was found to be reliable and the intra-examiner error was also acceptable.

Reproducibility of the hard tissue measurement has been reviewed by numerous investigations (Baumrind et al., 1971,1976; Houston, 1979,1983; Discussion section). However, a reproducibility test for the soft-tissue measurements was

undertaken on four selected variables (H-RGN, PNS-P, IAS and Soft Palate area). Ten cephalograms were randomly selected and the second tracings were completed two weeks after the first tracings. Differences between each of the pairs and variances of each series of measurements were calculated. Pearson's simple correlation coefficient (r) which represents the linearity of the randomness, together with Dahlberg's method error and Houston's reliability index (τ) were also provided (Table II). Minimum mean difference was determined from the measurements of the TGL(mean difference between the first and second measurement=0.5 mm; min=0.01mm; max=1.43mm). Pearson's simple correlation coefficient(r) between the two sets of measurements was .9991; Dahlberg's S.E. was .49; Houston's reliability index was .9995. The variable showing the largest mean difference among the linear soft-tissue variables was PNS-P (mean=.67mm; min=.14mm; max=1.61mm). The correlation coefficient of the P-PNS measurement was .9966; Dahlberg's S.E. was .60; the reliability index was .9986.

The reproducibility of the soft palate cross-sectional area showed a mean difference of 27.47 mm^2 , an r-value of .9875 and a reliability index of .9959. All of the measurements in Table II showed high reliabilities(τ > 0.9900) and negligible method errors i.e. not greater than 1mm in length and 30mm^2 in area.

Table II   **Reliability of the Soft-tissue Measuring Techniques and an Example of the Calculation of the Houston's Reliability Index**

		Difference between 1st measurement and 2nd measurement						
		Min.	Max.	Mean	s2	r	Dahlberg's SE	Houston's Index
H-RGN	(mm)	0.06	1.30	0.64	0.26	.9970	0.60	.9980
PNS-P	(mm)	0.14	1.61	0.67	0.22	.9966	0.60	.9986
IAS	(mm)	0.12	1.51	0.57	0.16	.9707	0.52	.9906
TGL	(mm)	0.01	1.43	0.50	0.20	.9991	0.49	.9995
Soft Palate	(mm^2)	3.82	63.81	27.47	335.08	.9875	24.22	.9959

Legend Table II   Mean value indicates average differences between 1st and 2nd measurement on the same variables. s^2 reveals variance between 10 pairs of measurements. r represents the correlation coefficient between 1st and 2nd measurements. The equation of Dahlberg's SE is $\sqrt{(X1-X2)^2/2(n-1)}$ An example of the calculation of Houston's reliability is as follow:

Subjects	TGL (Tongue Length) 1st	2nd	Difference
1	83.82	83.00	0.82
2	94.16	93.94	0.22
3	98.99	98.98	0.01
4	78.99	78.77	0.22
5	69.91	69.45	0.46
6	82.98	81.55	1.43
7	97.01	96.98	0.03
8	96.50	95.66	0.84
9	76.77	76.08	0.69
10	90.58	90.29	0.29
MEAN	86.47	86.97	0.50
s^2	101.66	98.38	0.20
SD	10.08	9.92	0.45
r	0.9991		
Σd^2			4.29

Houston's reliability index $(r) = \sqrt{1 - S^2d / S^2g}$

S^2d :variance of the differences

S^2g :the greater variance

Reproducibility of the natural head position has been studied by several researchers (Moorrees and Kean, 1958; Solow and Tallgren, 1976; Siersbæk-Nielsen and Solow, 1982; Showfety et al.; Sandham, 1988). The reproducibility of repositioning and measurement of natural head posture was investigated by repeated measurements at three different times on four subjects (see Table III). The subjects were positioned in the natural body and head posture in standing position with the modified fluid level device on the head. The degree on the level device was recorded twice per each time. The second and third measurements were carried out at a minimum interval of a week in the same manner as for the up-right standing position. A two-way ANOVA was used for the test of repeatability and the results are represented in Table III. The results indicate that the angulation of the head posture was significantly different from subject to subject(p=0.000), yet was not different at different times(0.192) for the same subjects. A significant probability(p=.009) in the Subject*Measurement (interaction) term indicates a variation within the same cell.

Table III   Reproducibility Studies of Natural Head Position and Measurement

	Measurements		
Subjects	1st	2nd	3rd
1	15/15	15/15	13/12
2	7/5	6/7	6/7
3	-2/-2	-2/-3	-1/-1
4	2/3	1/0	0/2

unit = degree

TWO-WAY ANALYSIS OF VARIANCE

SOURCE	SUM-OF SQUARES	DF	MEAN-SQUARE	F-RATIO	P
SUBJECTS	890.833	3	296.944	712.667	0.000
MEASUREMENT	1.583	2	0.792	1.900	0.192
SUBJECT * MEASUREMENT	12.471	6	2.069	4.967	0.009
ERROR	5.000	12	0.417		

Legend Table III   A pair of numbers in each cell in top table reveals the degree of head posture from the fluid level device. The probability(0.009) of the interaction term(SUBJECT*MEASUREMENT column) in the lower table suggests a significant variation within the same cell.

For the validity test of the fluid level device, the overall correlation between the head posture angle from the modified level device and the angle from the FH plane and true horizontal line was investigated by twenty randomly selected upright cephalograms and represented graphically in Figure 13. Pearson's correlation coefficient(r) was 0.9505; slope 0.9388; intercept -0.1089 at P less than 0.001 level. The 95% confidence interval suggests a deviation of not greater than 1.5° of the head posture angle in between the level device and the measured angle on the cephalograms (1.097 as a upper limit, 0.7869 as a lower limit). Therefore, 95% of the values measured by the modified level device are identical to the values measured on the cephalograms with 1.5° of deviation.

Fig. 13   Relationship between Level Device Angle and the FH/True Horizontal Cephalometric Angle

ANGLE BETWEEN FH PLANE AND TRUE HORIZONTAL
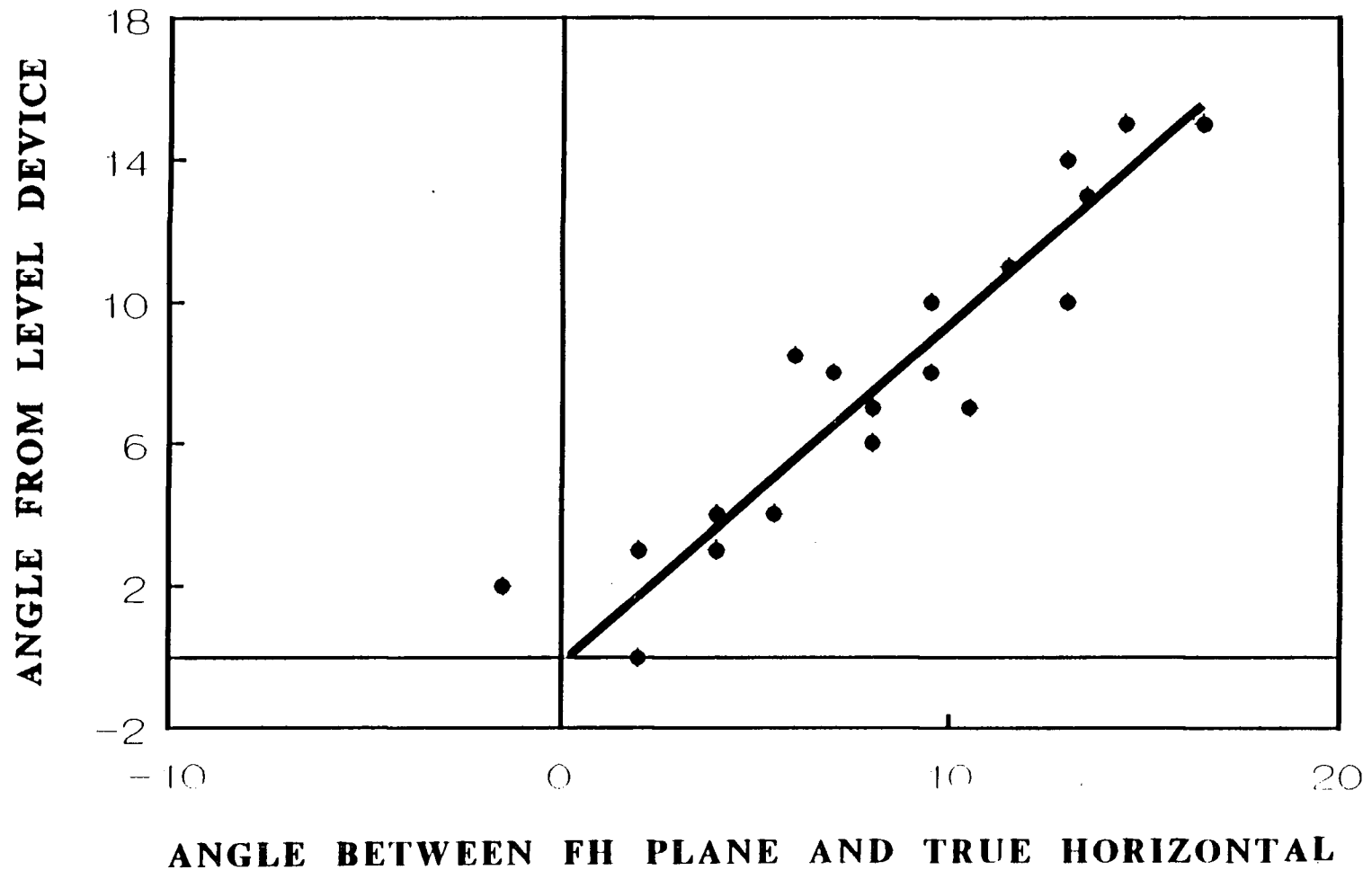
Legend Fig.13   The graphical distribution of the points suggests
a strong linear tendancy (Pearson's r=0.9505).

B. Cephalometric study

The results of the lateral cephalometric comparison study of OSA and asymptomatic controls in different body positions (see Table IV) show that, except for the TGH(p=.331), most of the linear measurements were significantly different in the up-right cephalograms. In up-right cephalograms, the OSA group showed a longer tongue, a longer and thicker soft palate, an anteroposteriorly narrower and superoinferiorly lengthened airway, and a more inferiorly positioned hyoid bone than the asymptomatic controls. For the angulation measurements, the OSA group was significantly different from the asymptomatic control group; the former registered an E-TT value of p=.003 level and also shown significantly larger CVTPP(p=.008), CVTSN(p=.012), OPTPP(p=.013) and OPTSN(p=.014) angulations, which indicates a strongly extended head and neck relationship in the up-right cephalograms. On the other hand, the supine cephalograms could not provide appropriate angular comparisons of the head posture of the OSA and the asymptomatic control group due to significant variations in pillow height. In the comparison of the cross-sectional area measurements, there were no significant differences between the two groups in the tongue(p=.060), nasopharynx(p=.239) and oropharynx(p=.214) areas in the up-right cephalograms. Yet, the OSA group did reveal a larger soft palate(p=.005) and a smaller hypopharyngeal area(p=.004) in the up-right cephalograms.

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Table IV   Comparison of OSA and Asymptomatic Controls in Up-right and Supine Cephalometric Positions

| | | Up-Right | | | | | Supine | | | | |
| | | OSA | | Control | | | OSA | | Control | | |
		Mean	SD	Mean	SD	P less than	Mean	SD	Mean	SD	P less than
Linear (mm)	Tongue										
	TGL	90.76	8.09	83.25	5.21	.013 *	87.41	6.22	79.74	6.84	.005 **
	TGH	40.79	4.49	39.19	3.34	.331	42.90	3.10	38.54	2.89	.001 **
	Soft Palate										
	PNS-P	49.01	10.54	40.37	3.90	.019 *	50.36	9.98	41.55	2.97	.011 *
	MPT	14.99	4.61	11.54	1.64	.030 *	14.44	3.91	12.70	1.15	.100
	Airway										
	SPAS	5.33	2.99	9.24	2.68	.002 **	2.97	2.53	10.37	11.30	.008 **
	MAS	9.96	3.14	13.57	3.34	.007 **	9.63	8.54	12.49	3.50	.323
	IAS	7.52	3.99	11.27	3.28	.016 *	5.37	3.89	9.90	2.45	.002 **
	VAL	87.30	7.14	73.01	3.57	.000 **	84.45	5.88	68.17	6.07	.000 **
	Hyoid bone										
	MP-H	28.30	8.64	17.09	3.87	.001 **	24.93	7.52	12.06	4.96	.000 **
	H-H1	18.90	7.37	12.21	4.54	.014 *	16.44	7.29	6.72	4.57	.001 **
	H-RGN	44.65	7.10	38.88	2.88	.021 *	40.76	7.96	36.29	5.74	.126
Angular (degree)	Tongue										
	ETT	38.05	6.82	28.95	7.92	.003 **					
	Vertebrae										
	CVTPP	104.20	6.97	96.73	6.04	.008 **					
	CVTSN	113.12	7.87	105.76	5.11	.012 *					
	OPTPP	98.83	7.75	89.82	10.22	.013 *					
	OPTSN	107.78	8.24	98.84	9.96	.014 *					
Area (mm²)	Tongue	3826.68	573.45	3437.05	356.73	.060	3992.80	475.74	3436.51	510.99	.006 **
	Soft Palate	519.12	154.34	364.99	53.25	.005 **	554.74	132.86	431.90	48.70	.009 **
	Nasopharynx	205.29	87.88	242.96	63.31	.239	203.84	88.11	230.35	63.60	.406
	Oropharynx	497.70	185.76	579.86	117.12	.214	315.95	127.13	412.42	123.09	.058
	Hypopharynx	161.08	87.63	292.57	141.12	.004 **	171.02	98.99	330.81	102.50	.000 **

Legend Table IV   The comparison is performed by the Student t test.
Significant levels of $p < .05$ are denoted by one
asterisk and $p < .01$ by two asterisks.

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In contrast, TGH(p=.001) and tongue cross-sectional area(p=.006) showed significant differences between OSA and control subjects in the supine position, but not in the up-right cephalograms. MPT(p=.100), MAS(p=.323) and H-RGN(p=.126) did not prove to be significantly different between the OSA and the asymptomatic control groups in the supine cephalograms but were significantly different in the up-right position. The cross-sectional area of the oropharynx failed to show a difference(p=.058) between the two groups in the supine cephalograms.

The results of the lateral cephalometric comparisons between the up-right and supine positions for each of the two different groups are provided in Table V. Of the linear variables, TGH, PNS-P, MAS did not yield differences corresponding to positional changes in both groups. The tongue length(TGL) was reduced significantly by approximately the same amount in the supine position in both groups. The tongue height(TGH) increased in the OSA group, but decreased in the control group in the supine position;however, statistically, there were no significant differences. The soft palate thickness (MPT) revealed no significant change in the OSA group, but in the control group(p=.002) showed significant thickening corresponding to the positional changes. The cross-sectional size of the upper airway was reduced in the supine position. The SPAS was significantly reduced in the OSA group(p=.018), but not in the control group(p=.195).

Table V   Comparison of Up-right and Supine Cephalograms in OSA and Asymptomatic Controls

		OSA					Control				
		Up-right		Supine			Up-right		Supine		
		Mean	SD	Mean	SD	P less than	Mean	SD	Mean	SD	P less than
Linear (mm)	Tongue										
	TGL	90.76	8.09	87.41	6.22	.021 *	83.25	5.21	79.74	6.84	.021 *
	TGH	40.79	4.49	42.90	3.10	.062	39.19	3.34	38.54	2.89	.544
	Soft Palate										
	PNS-P	49.01	10.54	50.36	9.98	.074	40.37	3.90	41.55	2.97	.225
	MPT	14.99	4.61	14.44	3.91	.323	11.54	1.64	12.70	1.15	.002 **
	Airway										
	SPAS	5.33	2.99	2.97	2.53	.000 **	9.24	2.68	10.37	11.30	.772
	MAS	9.96	3.14	9.63	8.54	.870	13.47	3.34	12.49	3.50	.161
	IAS	7.52	3.99	5.37	3.89	.018 *	11.27	3.28	9.90	2.45	.195
	VAL	87.30	7.14	84.45	5.88	.078	73.01	3.57	68.17	6.07	.007 **
	Hyoid bone										
	MP-H	28.30	8.64	24.93	7.52	.074	17.09	3.87	12.06	4.96	.000 **
	H-HI	18.90	7.37	16.44	7.29	.115	12.21	4.54	6.72	4.57	.003 **
	H-RGN	44.65	7.10	40.76	7.96	.041 *	38.88	2.88	36.29	5.74	.109
Area (mm²)	Tongue	3826.68	573.45	3992.80	475.74	.014 *	3437.05	356.73	3436.51	510.99	.996
	Soft Palate	519.12	154.34	554.74	132.86	.043 *	364.99	53.25	431.90	48.70	.000 **
	Nasopharynx	205.29	87.88	303.84	88.11	.918	242.96	63.31	230.35	63.60	.354
	Oropharynx	497.70	185.76	315.95	127.13	.000 **	579.86	117.12	412.42	123.09	.000 **
	Hypopharynx	161.08	87.63	171.02	98.99	.660	292.57	141.12	330.81	102.50	.142

Again, IAS was shown to be shorter(p=.018) in the supine position in the OSA group, but not in the asymptomatic control group(p=.195). The vertical length of the airway did not show a significant change (VAL) following positional change in the OSA group, but did in the asymptomatic control at the p=.007 level. MP-H was not significantly different in a comparison of the up-right and supine(p=.074) positions in the OSA group, but differed in the control group at the p=.000 level. H-H1 was shortened significantly in the supine position in the control group, but not in the OSA group. The distance between the hyoid bone and the retrognathion (H-RGN) was significantly shortened in the OSA group(p=.041) after a reclining position was assumed; however, no significant change was seen in the control group(p=.109). The tongue cross-sectional area was shown to be significantly larger(4.7% increased) in the supine position than in the up-right position in the OSA group(p=.014);however, no difference was observed in the control group(p=.996). Although the soft palate became larger in both groups after a reclining position was assumed, the increase in size was more significant in the asymptomatic control group. In both groups, a large degree of collapse was shown in the oropharyngeal area, after the body positional change, but this was not seen in the nasopharynx and hypopharynx. While the oropharyngeal area decreased by 36.5% in the OSA group, it decreased by 29% in the asymptomatic controls. However, the difference between the two

groups was hardly noticeable in both positions used for the cephalograms(p=.214 in the up-right cephalograms; .058 in the supine cephalograms).

An ANCOVA test was used to investigate the age effect on the hyoid variables, yet the results indicate that none of variables (MP-H p=.353, H-H1 p=.135, H-RGN .228) were affected by an age (see Table VI).

Table VI   ANCOVA test of the Age Effect on the Hyoid Variables

DEP VAR: **MP-H**   N: 29   MULTIPLE R: .621   SQUARED MULTIPLE R:386

ANALYSIS OF VARIANCE

SOURCE	SUM-OF-SQUARES	DF	MEAN-SQUARE	F-RATIO	P
SUBJECTS	432.326	1	432.326	7.667	0.010
AGE	50.414	1	50.414	0.894	0.353
ERROR	1466.130	26	56.390		

DEP VAR: **H-H1**   N: 29   MULTIPLE R: .512   SQUARED MULTIPLE R:262

ANALYSIS OF VARIANCE

SOURCE	SUM-OF-SQUARES	DF	MEAN-SQUARE	F-RATIO	P
SUBJECTS	77.515	1	77.515	1.806	0.191
AGE	102.117	1	102.117	2.379	0.135
ERROR	1116.176	26	42.930		

DEP VAR: **H-RGN**   N: 29   MULTIPLE R: .492   SQUARED MULTIPLE R:242

ANALYSIS OF VARIANCE

SOURCE	SUM-OF-SQUARES	DF	MEAN-SQUARE	F-RATIO	P
SUBJECTS	293.305	1	293.305	8.151	0.008
AGE	54.859	1	54.859	1.524	0.228
ERROR	935.621	26	35.985		

Legend Table VI   The probability of the age reveals the comparisons of the hyoid variables are not significantly affected by age.

Summary of the cephalometric study:

1. The OSA group showed a longer tongue, a larger soft palate, an anteroposteriorly narrower and superoinferiorly lengthened upper airway, a inferiorly positioned hyoid bone, a more up-right tongue, a more extended head posture and a smaller hypopharynx in the up-right standing position.

2. The OSA group showed greater tongue height, a larger tongue and a smaller hypopharyngeal area in the supine position.

3. With the positional changes from up-right to supine, the thickness of the soft palate increased more in the control group, whereas the size of the upper airway was more decreased in the OSA group.

4. With the positional changes from up-right to supine, the hyoid bone was moved up toward the mandibular plane more in the control subjects, and toward the mandibular symphysis more in the OSA group.

5. The tongue cross-sectional area increased significantly by 4.3% in the supine position and oropharyngeal area decreased by 36.5% in the OSA group.

C. EMG and pressure study

A comparison of EMG and Pressure variables obtained in the up-right and the supine positions are provided in Table VII. All of the comparisons were performed by means of Wilcoxon Signed Rank test. The resting activity of the GG muscle increased approximately 34 % (from 2.58 to 3.33 g/cm^2) at the p=.037 level with the body positional change from up-right to supine. Activity of the inferior orbicularis oris decreased significantly in the supine position at the p=.008 level. For the protrusion task, the supra-hyoid muscle group revealed a significantly increased activity (p=.014). The GG muscle revealed significantly larger values on maximum opening task in the up-right and the supine positions at the p=.028 level.

Table VII   Comparison of the EMG and Pressure of the Tongue between Up-right and Supine Positions in Asymptomatic Controls

EMG

		Up- Right		Supine		
		Mean	SD	Mean	SD	P less than
Rest	Genioglossus	2.58	1.03	3.33	1.58	.037 *
	Masseter	2.22	1.78	2.47	1.91	.260
	Orbicularis Oris	3.69	3.50	2.49	1.56	.008 **
	Supra-hyoid	1.87	0.67	2.02	0.77	.066
Protrusion	Genioglossus	93.41	48.89	93.10	56.93	.959
	Masseter	12.78	12.04	12.30	11.27	.575
	Orbicularis Oris	11.13	8.22	12.07	11.60	.767
	Supra-hyoid	27.11	50.36	35.45	69.97	.014 *
Max.	Genioglossus	16.87	9.89	21.79	10.42	.028 *
Opening	Masseter	12.03	14.70	12.69	14.32	.878
	Orbicularis Oris	49.93	46.17	62.76	63.75	.086
	Supra-hyoid	49.64	28.60	57.58	37.08	.285

unit= μV

Pressure

		Up- Right		Supine		
Rest	Anterior	10.33	9.10	7.83	6.88	.333
	Posterior	12.01	5.76	14.01	5.46	.017 *
Protrusion	Anterior	799.79	293.80	808.08	300.77	.878
	Posterior	136.49	93.47	116.54	73.26	.314
Max.	Anterior	11.29	10.81	11.85	13.65	.859
Opening	Posterior	28.25	16.98	32.02	18.74	.241

unit= g/cm^2

Legend Table VII   Each EMG and pressure value was converted and calibrated to μV and g/cm^2.

For the pressure data, the posterior tongue pressure at rest revealed an increased value of 17% ($2g/cm^2$) at the .017 level (where, two extremely increased or decreased values were precluded). The resting EMG activity of the supra-hyoid muscle group showed an almost significant value (p=.066); i.e., only two subjects out of ten revealed decreased EMG values in the supine position, and in turn, showed a strong tendency to increase the supra-hyoid muscle activity in the supine position (see Fig.14). The EMG activity of the supra-hyoid muscle at maximum protrusion was significantly greater in the supine position than in the up-right position, and the GG EMG activity at maximum opening also showed a significantly high value in the supine position. The EMG activity of the masseter muscle did not reveal any significant differences in any of the tasks.

Fig 14   Tongue EMG and Pressure Changes at Rest in Up-right and Supine Positions
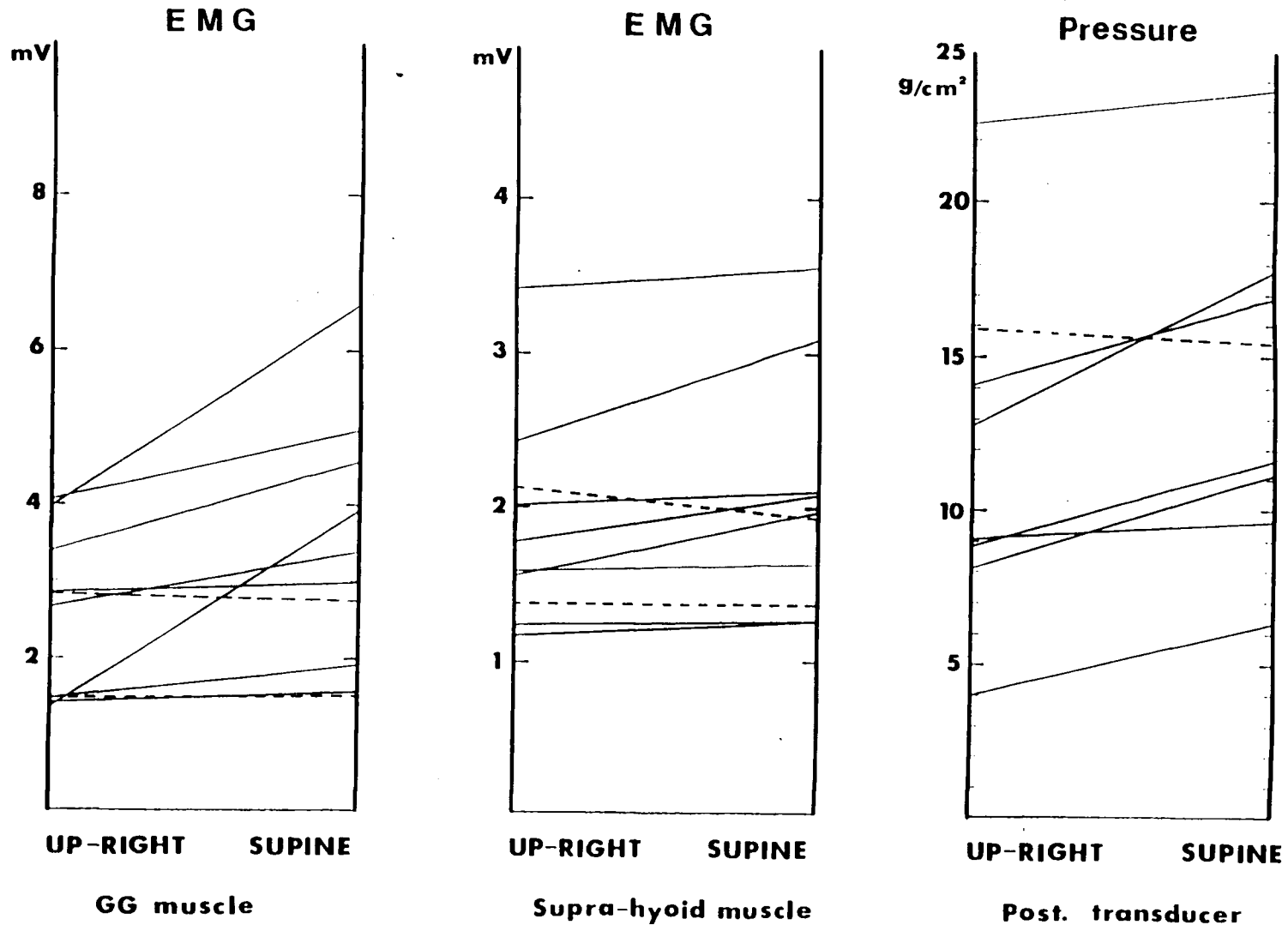
GG muscle   Supra-hyoid muscle   Post. transducer

Legend Fig.14   The solid lines of each graph represent the increasing
change of EMG and pressure due to the change in body
position, whereas the broken lines show a decrease
change.

As a summary of the rest EMG and pressure study, with the positional changes from up-right to supine, the asymptomatic control group showed:

1. The EMG activity of the GG muscle increased by 33.8%.

2. The pressure of the posterior portion of the tongue increased by 17%.

3. The resting potential activity of the inferior orbicularis oris decreased by 32.5%.

4. The EMG activity of the supra-hyoid group increased by 8%, yet the change was not statistically significant.

In conclusion, the oropharyngeal cross-sectional area collapsed by approximately 30% despite a 34% increase in GG muscle activity in the asymptomatic control group as a result of body positional changes. No size change in the tongue was observed in the supine position in the non-apneic group. In addition, 17% of the tongue pressure increment was recorded on the posterior load cell. With the positional changes, the size of the soft palate increased by 18.3% in the control group. All the hypopharynx changes are not statistically significant. The cross-sectional area of the hypopharynx increased by 13% which was accompanied by an 8% increase in the supra-hyoid muscle activity and a 6% decrease in the H-RGN value.

DISCUSSION

The current study was proposed to evaluate the relationship between GG muscle activity, airway size and body posture. For comparisons of airway size, cephalometric measurements were employed; for comparisons of tongue and other muscle activity, EMG and pressure studies were undertaken. The supine cephalometric technique was utilized in the current study and a comparison between the up-right and supine cephalograms was made. It appeared that the OSA patients have larger tongues and smaller oropharyngeal airways in the supine cephalograms. In addition, the locational change of the tongue in the supine position was quantified by a change in posterior tongue pressure.

Previous investigators have postulated a small airway size and reduced GG muscle activity in OSA patients. Recently, Lowe et al.(1986,1989) visualized the constriction area and measured the size of the upper airway by means of computer aided three-dimensional CT reconstruction. In spite of the judicious combined study of the CT and the pressure transducer, Stauffer et al.(1987), could not find any significant differences in airway size between patients with OSA and asymptomatic controls. They concluded that airway resistance seems more likely to be related to the AHI (Apnea Hypopnea Index) than to the cross-sectional size of the airway. Adequate

GG muscle activity may be one of the most decisive factors in the genesis of OSA. Remmers et al.(1978) monitored the EMG of the GG muscle using fine wire bipolar electrodes and elucidated the association of the muscle with OSA symptoms. Subsequently, several publications have addressed the inter-relationship between diaphragmatic and GG EMG activity. Another group working with the hyoid apparatus suggested that the position of the hyoid arch plays an important role in the maintainence of upper airway patency (Van de Graaff et al., 1984; Van Lunteren et al., 1987). They postulated that decreased or poorly coordinated hyoid muscle activity could be a crucial factor in hypopharyngeal obstruction. However, most information acquired to date from this sort of biomechanical study has been retrieved from animal experiments. The coupling action of the hyoid and GG muscles in dogs and cats may be different in humans; yet few studies have been done on human subjects. Recently, Lowe and co-workers (personal communication) presented a facial profilogram of OSA patients in which a typical appearance of the chin was shown. In addition, a inferiorly positioned hyoid bone was reported in the OSA patient group as one of the significant results of the current study in agreement with previous data from several earlier studies. Tonic activity of the GG or hyoid muscle group may be more important than phasic activity, for the tonic activity of the upper airway muscles is likely to be directly related to the size of the airway. In an age-matched study by Brown and associates (1987), it was emphasized that

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the OSA group is distinguished not by the cross-sectional area but by the distensibility of the pharynx. Similarily, in the current study, the size of the pharyngeal cross-sectional area did not differentiate patients with OSA symptoms from non-patients. However, the current study did show that oropharyngeal area was more collapsed in the OSA group in the supine position even though it was not statistically significant(P=.058) (see Table IV). Consequently, it may be an over simplification to consider OSA as a disease which may occur when the airway is small. We should therefore consider not only morphological and functional factors but also the inter-relationship of both factors.

A. Cephalometric study

Numerous investigators have measured the anatomical structures of the upper airway (Jackson and Olson, 1980; Hoponik et al., 1983; Lowe et al., 1986; and Martin et al., 1987). Cephalometric analyses have long been used for the investigation of facial growth and development in the field of orthodontics. Cephalometric techniques have more recently become a routine diagnostic tool to evaluate the size of the tongue and airway (Riley et al., 1983; Lowe et al., 1986; Jamieson et al., 1986; Strelzow et al., 1988; deBerry-Borowiecki et al., 1988; and Lyberg et al., 1989). In spite of obvious limitations as a two-dimensional method of analysis, cephalometrics is still considered a convenient, less invasive, and less expensive technique to evaluate upper airway size. In 1983, Riley and co-

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workers actively introduced cephalometric analysis to the OSA research field and showed its compatibility to the flow volume curve. Lowe and associates (1986) reviewed the soft and hard-tissue characteristics of OSA patients by means of cephalometric analysis and extracted significant variables using a principal component analysis. The results of the current study agree with most of the soft-tissue measurements that they analyzed. As a result of the development of computer equipment and software, several measurement studies on soft-tissue areas and airway space have been produced. Strelzow et al.(1988) reported results from a cephalometric study which concentrated more on the size and positional changes of the tongue, soft palate, pharyngeal space and the location of the hyoid bone. Not surprisingly, not only the linear measurements but also the measurements of the cross-sectional area of the tongue and soft palate approximate the results of the current study (see Table VIII). Another study (deBerry-Borowiecki et al., 1988) which adopted virtually the same definitions of linear measurements and tongue and soft palate cross-sectional areas as the former study, provided similar results but a shorter soft palate and smaller tongue area (see Table VIII). Recently, Lyberg and associates (1989) undertook a cephalometric study that is highly compatible with the present project in terms of the definitions of the linear, angular and area measurement variables. They obtained cephalograms in natural head posture, although it is not clear whether the subjects were standing or sitting.

TABLE VIII   Linear and Area Variable Comparisons Between Current Report and Other Studies

A. Linear Comparisons   unit=mm

	Current study				Lyberg et al.			
	OSA(20)		CONTROL(10)		OSA(25)		CONTROL(10)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
PNS-P	49.01	3.9	40.47	3.9	48.0	4.3	35.3	4.6
MPT	14.99	4.6	11.54	1.6	14.0	2.0	11.2	1.5
MAS	9.96	3.1	13.57	3.3	8.4	3.5	13.7	3.8
IAS	7.52	3.9	11.27	3.3	10.3	4.0	13.8	3.8

	deBerry-Borowiecki et al.				Strelzow et al.			
	OSA(30)		CONTROL(12)		OSA(90)		CONTROL(12)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
PNS-P	41.6	6.0	34.7	7.0	46.0	7.0	36.0	8.0
MPT	14.0	6.0	10.0	2.0	13.0	2.0	11.0	2.0
IAS								

	Jamieson et al.			
	OSA(138)		CONTROL(7)	
	Mean	SD	Mean	SD
PNS-P	46.7	3.0	36.4	4.4
MPT				
IAS	5.3	3.0	12.4	3.5

B. Area Comparisons   unit=mm²

	Current study				Lyberg et al.			
	OSA(20)		CONTROL(10)		OSA(25)		CONTROL(10)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Tongue	3826	573	3437	356	3597	459	3486	344
Soft Palate	519	154	364	53	485	80	288	62

	deBerry-Borowiecki et al.				Strelzow et al.			
	OSA(30)		CONTROL(12)		OSA(90)		CONTROL(12)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Tongue	3790	375	3215	471	3884	404	3374	293
Soft Palate	459	134	251	63	457	105	289	88

Legend Table VIII   Numbers in a parenthesis indicate sample size.

Lyberg et al. provided values for the PNS-P, MPT, MAS, IAS, tongue and soft palate area that are remarkably similar to the results of the current study (see Table VIII). The studies by Jamieson and associates(1986) and deBerry-Borowiecki et al. (1988) provided age-matched results which included females. The controls used in the study by Lyberg et al. were generally younger individuals(mean age =23.6). The tongue area and soft palate of the control group in the study by deBerry-Borowiecki et al. were very small even though the subjects were older compared to those in the other studies. The present study revealed the longest and largest soft palate of all the several research projects in Table VIII; but in these studies the maximum thickness of the soft palate was very similar. This is possibly due to the fact that most of the asymptomatic control subjects employed in the current study were snorers, relatively obese (mean BMI=26.3) and were adult males (mean age = 33.5).

Interestingly, Lyberg et al. failed to find a significant difference between the OSA and the control group for tongue area, as the current study did. However, the cross-sectional area of the soft palate was significantly different in the two studies. A supine cephalogram may provide more physiological information than the up-right cephalogram.

The tongue area was significantly different in the two groups at a probability level of less than 1% in the supine cephalograms (see Table V). In the OSA group, the tongue cross-

sectional area became significantly broadened in the supine position (see Table V). However, the tongue area of asymptomatic controls did not show significant change (see Table V). Therefore, differences became more apparent in the supine cephalograms. In addition, the airway behind the soft palate and tongue(SPAS and IAS) was reduced in the supine position in the OSA group (Table V), but not in the group without apneic symptoms (Table V). These findings explain that not only the cross-sectional area of the tongue is changed but also that the tongue mass settles inferiorly in OSA patients in the supine position, probably due to gravitational pull (Crumley et al,1987). On the other hand, in the non-apneic group, the tongue fell back into a 'bunched up' position, therefore the actual tongue area was reduced and the SPAS was lengthened to keep the airway open. This disparity may be surmised from the significant difference in the posterior tongue pressure values between the up-right and supine position in the asymptomatic controls. Weak GG muscle activity in OSA patients has been presumed by several researchers. On this basis, the tongue sank down more in the symptomatic group due to lack of GG muscle contraction. Among the hyoid variables, vertical measurements of the hyoid bone such as MP-H or H-H1 did not change with the positional change in the symptomatic patient group (see Table V);however, they changed significantly in the asymptomatic group (Table V). This phenomenon also implies a disparity in the GG muscle efficiency between the two groups. In contrast, the horizontal variables

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among the hyoid measurements (H-RGN) did not show significant change in the non-apneic group (Table V). On the other hand, the length was significantly shortened in the OSA group (Table V). This finding suggests that the size of the hypopharnygeal airway is large enough to permit breathing in asymptomatic controls where the supra-hyoid muscle group does not need to contract. In contrast, according to the results of the current study, the hypopharyngeal airway in the OSA group is approximately half the size of that of the controls in terms of cross-sectional area in the supine position; thus, the supra-hyoid muscle group must contract actively to keep the airway open. Moreover, the maximum palatal thickness of the soft palate was 'actively' increased in subjects without symptoms in the supine position; no change was shown in the OSA group. It may therefore be the tongue and its related muscles, not the soft palate, which actively generates an apneic condition.

One of the important factors to consider in the measurement of airway size is head posture. For comparison of head posture between OSA and controls, some head posture angulations that are believed to be most sensitive were employed. Of the variables representing head posture, the CVTPP may be the variable which most strongly supports the hypothesis that the patient group has a more extended head posture than the controls. The angulation variables between the vertebrae and palatal plane (CVTPP and OPTPP, suggested by Dr. Diewert), while newly designed and employed for the first time, were

nonetheless, very significant variables. The relationship between head posture and airway adequacy has been studied by numerous researchers. Span and Hyatt(1971) measured upper airway resistance in conscious men and found that resistance of the upper airway may be affected by the head posture. Solow and Tallgren (1971) hypothesized that each individual has his/her own head posture which is precisely reproducible only in the standing position. Following studies of Solow et al.(1984), Woodside and Linder-Aronson(1979) and Vig et al.(1980) demonstrated significantly different head postures in accordance with airway adequacy. They concluded that a reduced nasopharyngeal airway is associated with a larger craniocervical angulation. When the results are compared, a small difference can be noted in the measurement of the CVTSN between Solow and Tallgren's result (97.72°) and the value from the controls in the current study (105.76°). This difference may derive from the composition of the subject group, i.e. relatively heavy and older subjects were recruited for the current study. Such a disparity was also seen when comparisons to other studies by Kylämarkula and Huggare (1985) and Sandham (1988) were made. Measurements of head angulation in OSA patients were completed by Kalbfleisch in 1988, and regrettably, there is no measurement variable that coincides with the current study;however, the extended head posture as a typical characterestic of the OSA patient is demonstrated in his report. The importance of cervicocranial angulation is also due to the

specific relationship to airway size. As the airway size changes, the head posture changes and vice versa. Kalbfleisch (1988) demonstrated a decrease in airway size with neck flexion by means of lateral cephalometry. Greene et al.(1961), using cinefluorography, demonstrated an increase in upper airway size upon hyperextension of the neck in the supine position. Therefore, it is extremely important to measure the upper airway size in a standardized head posture.

Another variable to be accounted for before measuring upper airway size, is body position. Body position alters not only muscular activity but also airway size. Anch and his co-worker (1982) found that supra-glottic airway resistance is significantly larger in the supine than in the sitting position in both normal and OSA groups. They hypothesize supra-glottic airway narrowing in OSA patients. However, Navajas et al.(1988) explained through their pressure transducer study that this phenomenon is due to the reduction of the FRC(Functional Residual Capacity) in the supine position. Nevertheless, Fouke and Strohl (1987) nullified the effectiveness of the reduced FRC in the supine position by means of an Emerson cuirass♣ and

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♣ **Cuirass**

A shell like casing which is closely fitted around the thorax in treatment of weakness of the respiratory muscles, so that evacuation of air from within the cuirass causes expansion of the thorax and hence inhalation (Wiley et al.,1986).

identified the degree of collapse due to the body positional change. They found that the pharyngeal cross-sectional area was 23% smaller in the supine than in the up-right position.

This information is pertinent to the interpretation of the current results even though Fouke and Strohl used a different method of measurement. The current results show a 12.7% reduction in the entire pharyngeal airway (and a 28.9% reduction in the oropharynx) in the asymptomatic control group; and a 20.1% reduction in the pharyngeal airway (36.6% in the oropharynx) in the OSA patient group. The discrepancy between 23% and 13% could be explained by the effect of a reduced FRC in the supine position in the case of the current study; the result from Brown et al.(1987) was nearly identical (21% in the OSA group, 15% in the normal group) to the results of the current study.

Ultimately, one should take into account head posture, body position, glottic closure and swallowing for a comparative study of the airway size. In the present study, most of the conditions mentioned here were controlled and standardized;however, certain limitations of the cephalometric technique could not be overcome. The cephalogram provides a midsagittal cross-sectional area, so that it is impossible to get full information about the volume. In other words, we may not even be able to read the immediately adjacent para-sagittal area correctly. Furthermore, even CT scans may not be able to

give correct information when it is used as a two-dimensional technique. For instance, Stauffer and associates (1987) were unable to demonstrate that mean pharyngeal size is significantly different between patients and control subjects. However, only the minimal area showed a significant correlation with the AHI($r=0.53$, $P<.01$). In the same sense as mentioned earlier, discrimination of the tongue size difference by means of the upright cephalogram was impossible in the present study. The supine cephalometric technique provides new information which is possibly hidden by the two-dimensional limitation of the technique.

Review of reproducibility tests for hard tissue structures

The reproducibility test in cephalometrics has a long history and has been widely investigated by several statistical methods. Solow (1970) introduced a computer-aided cephalometric analysis based on digitizing principles. In 1979, Houston reviewed the robustness, accuracy, and convenience of digitizing methods. In addition, he enumerates the source of errors that can arise in the digitizing method. According to his review, errors can arise from:

-incorrect identification of a landmark,

-an incorrect sequence of digitization,

-movement of the recording during digitization,

-environmental variation affecting a sensitive digitizer,

-intermittent mechanical faults in the apparatus.

Baumrind and Miller (1980) reviewed a computer-aided head film analysis and illustrated a typical feature of distribution of the errors from several representative anatomic landmarks. They suggest a method of using corner fiducials which provides considerable protection against the impact of rotational errors and increased reliability. Recently, Sandham(1988) extensively reviewed reproducibility tests of cephalometric measurements for hard tissues. He proposes a standard deviation ranging from 0.43 to 2.11 mm for differences between repeated linear measurements. In his study, all of the kurtosis values were acceptable at a level less than 0.1%. The reliability of the current study in soft tissue measurement showed relatively high values compared to previous work. This might be explained by the memorization effect even though the retracing was undertaken with a time lapse of at least one month.   Houston (1976) clarified that immediate redigitization was significantly more reproducible due to a memory effect.

Repeatability to reproduce natural head posture

Moorrees and Kean (1958) investigated the hypothesis of natural head posture and tested the accuracy of reproducing the natural head position by means of lateral head films. They reported 2.05 ° Standard Deviation in repositioning of the natural head posture and clarified that the true vertical line for reference purposes is more reliable than the routinely used FH line. Siersbæk-Nielsen and Solow (1982) studied variability

in head posture in a young age group. They applied Mølhave's most reproducible natural standing position as a standardized body position, and self-balanced position as a natural head posture. They ultimately reported 2.3 - 3.4 ° of method error for positioning of the head, which was statistically acceptable. Another recent work by Sandham (1988) confirmed the hypothesis of reproducible natural head posture. He displayed 5.44 ° of SD as a maximumly deviated value for the OPT/HOR angle (angulation between odontoid process and true horizontal line).

Validity of the modified level device

For future study, it is worthwhile to discuss the validity of the modified level device. Greenfield and associates (1989) evaluated the influence of the ear rod on cephalometric analysis. They concluded that taking a cephalogram without the ear rod provides better reproducibility in head positioning. They used a photogragh as a reference method for the cephalometric technique and suggested that the photogragh method is a 'quick and dirty' method to measure head posture. However, the modified level device technique may be superior to other techniques particularly when comparing two body positions with different analyses. Prior to the validity test for the modified level device, a carefully designed reproducibility test was undertaken. In 1985, Tsuchiya et al. examined the reproducibility of identification of the soft-tissue infra-orbital notch by means of palpation. They concluded

that on average the soft-tissue orbitale was palpated consistantly at 1mm superior to the bony orbitale on a film. Another soft-tissue landmark employed in the current study was the tragion which is the notch on the upper margin of the tragus. The modified level device was attached on the line which was assumed to be the soft-tissue FH plane between soft-tissue orbitale and tragion. The significance level which is shown in Table III encompasses reproducibility of the entire measuring procedure.

B. EMG and pressure study

Contraction of skeletal muscle is triggered by a conducted action potential. When the threshold is reached at a motor end plate, a conducted action potential is elicited. Myoplasms are fairly good electrical conductors, therefore it is possible to record the electrical changes (the sum of the number of depolarizations) in the muscle by means of electrodes. Surface electrodes used in the current study cannot be used for a single motor unit study ;however, they are non-invasive and easy to apply. The intra-oral surface electrode for the GG muscle was first developed and evaluated by Doble and co-workers (1985). In 1988, Milidonis et al. compared the recordings made with these custom fitted intra-oral surface electrodes to the bi-polar fine wire recordings of the GG muscle and proved that the two methods are sufficiently compatible.

Due to calibration problems, recording of the tongue pressure is not a simple task. Proffit et al.(1969) employed a pressure chamber for calibration of the mercury strain gauge and presented high reproducibility for tongue pressure. However, as with EMG, it is difficult to produce high repeatability in pressure recordings. Recently, Hellsing and L'Estrange (1987) investigated pressure change on the lip following head posture change and change in mode of breathing. They used a strain gauge type transducer developed by Proffit which has a transducer head with a area of 0.043 cm^2. The transducer was calibrated directly in the subject's mouth by means of a pen with a spring load system. However, it is so hard to reproduce the exact mode of tongue contact that there are still large variations in terms of absolute tongue pressure from study to study. The current study employed a rubber sac imitating the tongue musculature. No calibration system is perfect and the current project was carried out merely as a comparison study.

Even though this study was carried out only with asymptomatic controls, the current results provide several significant pieces of information and suggest several implications for the relationship between size of the airway, body positional change and upper airway muscle activity. The GG activity increased by approximately 34% in the supine position in comparison to the up-right positon in the subjects without symptoms. The EMG activity of the supra-hyoid muscle group increased by 8%, but it was not statistically significant.

A more curious phenomenon was an increment in posterior tongue pressure despite augmentation of the GG and supra-hyoid muscle activity as a result of positional changes of the body. EMG activity changes in the supine position are well documented and it is reasonable to assume an increment in supra-hyoid muscle activity after changing the body position. Airway patency is related to the changes in muscle tone of the entire upper airway in the supine position. However, the muscle can shorten only when the neural activity overcomes its external load, for example, the weight of the tongue itself. The current study revealed an enlarged tongue cross-sectional area(4%) in the supine position in the OSA group, yet the same tongue area in the non-apneic group. The cross-sectional area of the oropharynx was reduced by nearly the same amount in both groups, but slightly(7%) more in the OSA group. This subtle size difference(4%+7%) in the waking state in controls could considerably induce serious problems during sleep in the OSA group. The oropharynx collapsed approximately 30% in spite of a 34% increment in the GG muscle activity in asymptomatic controls. In addition, the posterior tongue pressure implied the actual tongue location, which was lower down. Cherniack and Hudgel (1985) agreed that electrical activity is not necessarily a good index of the mechanical action of a muscle. For instance, the sternohyoid muscle can lengthen in inspiration even though there is a considerable increase in electrical activity (Luntern et al.,1987). Brown et al.(1987) concluded from their age

and weight-matched study that changes in posture alone are not sufficient to convert a snorer into a patient with OSA. The current study hypothesizes that less tonic muscle activity or a heavier tongue in respect to the GG activity accompanied by a narrowed airway may play a primary role in the pathogenesis of OSA.

From a functional standpoint the muscle spindle acts as a length monitoring system. It constantly feeds back information to the CNS regarding the state of elongation or contraction of muscle stretch. When a muscle is passively stretched, the muscle spindle informs the CNS of a feed-back activity. Active muscle contraction is monitored by both the Golgi tendon organ and the muscle spindle. The mandibular rest position is an example of the neuromuscular mechanism (Okeson, 1989). The gravitational pull of the mandible body prompts the passive stretch of the elevator muscles. This passive stretching also sensitizes the muscle spindle. Through the afferent fibres(Ia or II) firing in the muscle spindle sac, this information ascends to the higher center. The CNS stimulates α motor efferent neurons. This evokes contraction of the extrafusal fibres. Automatically, the length of the muscle spindle is shortened. This shortening brings about a decrese in afferent output of the muscle spindle. The same principle could be applied for the GG muscle in supine position. Normal muscle tonus does not create fatigue. The overload stimulates the GG muscle to overwork and this creates a chronic fatigue. Scardella et al(1989) recently reported that due to the

high percentage of type II fibres(white) in the GG muscle, relatively small increases in its activity would predispose to fatigue. They conducted a fatigue study by means of a lingual force transducer and an intra-oral electrode and concluded that the GG is a readily fatigable muscle.

Another plausible factor inducing the apneic condition is a malarranged hyoid apparatus. The hyoid bone is a suspended structure located between the chin and cervical vertebrae. Brodie(1963) analyzed the hyoid bone as the posture apparatus of the head and jaws which balances the supra- and infra-hyoid musculatures. From the early developmental stage, the hyoid bone is closely allied with the tongue. The posterior portion of the tongue is derived from the second and third branchial arches, so the floor of the mouth is formed by the geniohyoid and mylohyoid muscles. According to Bench(1963), in an average person at the age of three, the hyoid bone is maintained at a level between the third and fourth cervical vertebrae and gradually descends to a level of the fourth vertebra by full adulthood. The hyoid bone is a unique structure floating among the muscles without any bony articulation. The hyoid apparatus includes at least eleven pairs of muscles and three pairs of ligaments, in total twenty-eight related soft-tissue structures. Bibby (1981) conducted a study on the hyoid triangle and emphasized that the hyoid bone reflects the relative tensions of the muscles, ligaments and fascia attached to it.

The hyoid bone serves two basic functions; deglutition and respiration. Pruzansky (1960) demonstrated a low hyoid position caused by excessive infra-hyoid activity in a patient with ankylosis of the TMJ.   In 1963, Bosma observed a reflective reaction of the hyoid bone to keep the airway open in infants and explained that anteflexion of the head and neck in order to stabilize the hyoid and larynx is a part of the reaction of the maintenance of pharyngeal airway. Recently, Miki et al.(1988) tested a new treatment for OSA patients, appling electrical stimulation to the submental region. The overnight polysomnography study supported their postulation that submental stimulation might ameliorate the symptoms of OSA patients.

Why do the OSA patients have a lower positioned hyoid apparatus? First, age may accelerate the inferior migration of the hyoid bone. Again, in Bench's(1963) growth study, he found that the tongue is higher in the younger samples. He postulated that hyoid bone lowering tends to continue after facial growth. Recently, Tallgren and Solow (1987) investigated the relationship between hyoid bone position, facial morphology and head posture in relation to age. They demonstrated a more inferior (2.55mm against mandibular plane) and anterior (1.64mm against vertebrae) positioned hyoid bone in an older group. Hoffstein et al.(1989) found that there was a negative correlation of pharyngeal area with age only in males. The decrement in the airway size with age is seemed to be related

to the decrement in the expiratory reserve volume (Leblanc et al,1970).

A second postulate for an inferiorly positioned hyoid bone is that it is related to a smaller upper airway in the patients with OSA. A smaller airway than the optimal size induces a extended head posture for better airway adequacy. An extended head posture elicites a passive stretch of the supra-hyoid muscle. A recent extensive study by Winnberg and associates(1988) reported a series of precisely conducted studies on hyoid biomechanics. The researchers synchronized EMG and videofluorography and investigated the dynamic and static relationships between the hyoid bone location and head posture. They illustrated a more inferior(22mm) and anterior(14mm) positioned hyoid bone with a 15-25 degree extended head posture. A more extended head posture drops the hyoid apparatus inferiorly and anteriorly and stretches the supra-hyoid muscles. This passive stretch may sensitize the muscle spindles. This information feeds back to the CNS and reacts to the contraction. However, as long as better airway adequacy is required, the extended head positioning may be actively continued because contraction at low muscle tension has no effect on muscle stiffness (Bressler,1974). Consequently, the supra-hyoid muscles, not only the GG and geniohyoid, but also the omohyoid and anterior digastric muscles, fatigue and are less effective. As was mentioned earlier, due to the relatively excessive infra-hyoid behavior, similar to the case of a TMJ ankylosed patient,

the hyoid bone migrates inferiorly.

Mixed type skeletal muscles fail when the force continuously imposed on them exerts 15% to 20% of their maximal force(Grassino et al., 1988). An important determining factor in muscle failure(i.e. fatigue) is the availability of blood circulation to the muscle. Blood washes out catabolites generated during contraction and it provides nutrients and oxygen to the muscle. Fatigue is a reversible physiological situation, and it shows a particular time constant of recovery. Recovery of maximal force is progressive and is completed in 10-15 minutes. However, the capacity to reproduce the initial endurance time is recovered slowly, perhaps over in 18-24 hours (Grassino et al., 1988). Particularly, the fatigue generated by low frequency stimulation may take several hours to recover. The development of fatigue reduces the maximal force a muscle can generate and impairs its contractility and the muscle contractility is directly related to muscle compliance. A muscle forced to contract chronically against heavy loads may develop chronic fatigue or weakness. Recently, Couser and Berman(1989) reported that respiratory muscle fatigue might generate a functional upper airway obstruction. Not only because of fatigue but also because of muscle orientation, the GG and supra-hyoid muscle may not be able to work efficiently. Robert et al.(1984) discussed the pharyngeal airway stabilizing function of the hyoid muscle group in rabbits. In agreement with Brodie's(1950) understanding they found the stabilizing function of the

sternohyoid and sternothyroid muscles to resist pharyngeal airway collapse due to negative intra-luminal pressure. Through a series of investigations Van Lunteren and his associates (1987 a,b) reinforced this hypothesis of hyoid muscle activity in breathing. The coordinated activation of both of the supra- and infra-hyoid muscles may produce a vector of forces, which displaces the hyoid arch in a outward direction, resulting in a dilated upper airway. Furthermore, several investigators reported that the head posture alters not only the resting length of the hyoid muscles but also the geometrical arrangements of these muscles, and in turn, airway size. Van Lunteren and co-workers (1987b) confirmed this hypothesis by means of sonomicrometry in cats. They found that an increment in upper airway volume lengthens the resting lengths of the geniohyoids in cats, but causes variable changes in sternohyoid length. Extension of the neck increases the length of both the geniohyoid and sternohyoid muscles. With flexion of the head, they observed opposite results. Therefore, they concluded that with a given upper airway geometry there is an inverse reaction between upper airway volume and hyoid muscle length, which is especially strong for the geniohyoid muscle. They did not perform the experiment on the upper airway volume change induced by extension and flexion of the head and neck. However, much research done by different groups has supported the idea that patency of the upper airway is enhanced with an extension of the head posture. In addition, Van Lunteren et al.(1987) postulated

that it is the geniohyoid muscle, not the sternohyoid, which plays an important role as a hypopharyngeal dilator in cats. They added that the sternohyoid muscle acts in an accessory fashion to prevent excessive movement to the cranial side due to geniohyoid contraction, and that it functions synergistically to move the hyoid bone in a forward direction. However, as described in the ankylosed TMJ case of Pruzansky's observation, the infra-hyoid muscle greatly affects the hyoid bone location. From the biomechanical point of view, an inferiorly positioned hyoid bone may not yield a sufficient forward movement. This might be due to muscular malfunction and/or to the inefficiency of the direction of the vector sum. In other words, the angulation constructed by the geniohyoid muscle and sternohyoid muscle is altered to become obtuse, and therefore, may not be able to produce a vector sum sufficient to allow an adequate airway size.

Helling(1986) described the supra-hyoid muscle as a group of muscles including the anterior digastric, the geniohyoid and the GG. Van de Graaff and associates(1984) commented that in humans, GG has some fibres inserting into the hyoid bone and may be another important determinant of hyoid bone position. Winnberg(1987) included the anterior belly of the digastric muscle, the mylohyoid muscle and the geniohyoid muscle in his definition of the supra-hyoid muscle group. While the mandible is in the rest position with 2-4mm of inter-occlusal space in an up-right sitting position, among the supra-hyoid muscles only

the geniohyoid muscle may be firing in phase with respiration. Based on the results of the present study, an 8% increase in supra-hyoid muscle activity is presumed in the supine position compared to the up-right position. This amount of increased muscle activity in the supine position may represent augmented EMG activity of the supra-hyoid muscle to keep the airway open. In contrast, significant decrease in EMG activity in the inferior orbicular oris muscle, may represent a relaxation after reclining of the body. Harper(1988) postulated that the posterior movement of the mandible may occur in the supine position during sleep due to relaxation of the lateral pterygoid and masseter muscles. In addition, Hollowell et al.(1989) observed a significant decline in masseter activation in the OSA patients during sleep and the same group(1989) showed a significant increase of masseter muscle activity in accordance with the level of hypercapnea. However, no change in the masseter activity was observed with the positional changes during the present study. Brodie(1950) observed that the mouth opening accompanies the backward movement of the hyoid bone. He assumed a shortening of the supra-hyoid muscles to keep the airway open during opening of the mouth. During maximum opening, EMG activities from the digastric muscle obscure geniohyoid activity regardless of body position. However, more GG activity is still required in the supine than the up-right position due to the gravitational pull. This explanation agrees with the result of the current study that GG activity during

maximum opening is increased in the supine position.

As was discussed earlier, measuring of absolute value of the tongue pressure is extremely difficult. Lear and his co-workers(1965) discussed this matter quite skeptically. Pressure is traditionally expressed in force per square centimeter. This term implies that muscular activity and surface configuration must distribute force evenly over the entire sensing platform of the transducer. However, there is no evidence that oral tissues invariably act in a manner analogous to an air filled ballon(Proffit et al 1964), a mass of sponge rubber, or a water filled ballon(present study). Furthermore, where this contact occured on the transducer surface is unknown. Even when pressure transducers are calibrated in an in-vitro situation, one must pay attention to the inconsistency of the transducer response to a given load according to the distribution of the load. On this point, Lear et al.(1965) recommended that the use of pressure transducers with smaller sensing surfaces may overcome these objections. In the current study, to minimize this problem, a small transducer(0.28 cm^2) was employed and the detected signals were integrated and averaged by means of computer software. The linearity in-vitro of the transducers employed for the present study was shown to be acceptable for a comparison study (see Fig. 10).

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C. Overview

Recently, Suratt and co-workers (1988) presented a paradoxical report. They postulated that the upper airway muscle activity may be more augmented in OSA patients than in normal controls. They found that OSA patients have more phasic GG group activity during NREM sleep, which could be explained as a precarious compensatory mechanism. Strohl (1986) emphasized the importance of the tonic activity of the muscles. He explained that changes in tonic activity could affect airway size as well as muscle length; thus tonic activity of the GG and hyoid muscles overcomes surface tension of the blocked airway, and finally promotes the airway occlusion.

The role of age in OSA is not clear. However, Chaban et al.(1988) recently observed that airway obstructions at the level of the tongue base mainly happen in older age groups, whereas obstructions at the level of the soft palate occur in younger groups. Lugaresi et al.(1980) found that the frequency of snorers in the population increased with age. The current study failed to match age and BMI. However, the results from the ANCOVA test in the current study illustrated that age does not affect the comparisons of the hyoid variables between the OSA and asymptomatic control groups (see Table VI). Nevertheless, as described earlier, the resistance of airflow increases with age, and in addition the hyoid bone migrates inferiorly and anteriorly. These findings imply that age may

be another factor exacerbating OSA symptoms. Anatomically, upper airways are lined by mucous membranes with the musculature underneath, and are long and convoluted as well. However, the small size of the upper airway may not alone be able to induce the apnea. Furthermore, the stability of the upper airway does not seem to be totally determined either by muscular activity or by pulmonary function alone. The functional roles of feedback control of respiration or chemico- or mechanoreceptors are significant. Moreover, from a biomechanical point of view, the coordinated linkage of the airway size, tongue activity, hyoid bone location, as well as the supine position of the body, may contribute to the pathogenesis of the OSA symptoms.

D. Pitfalls and Future Studies

The present study was designed to compare anatomical and physiological relationships between airway structures in accordance with body positional changes in OSA and asymptomatic controls. Therefore, the cephalometric study required a reproducible and standardized recording position for accurate data collection. However, the experiment failed to exclude several inevitable problems inherent to this procedure. For instance, the patients were told to hold the mandible slightly closed in an up-right standing position, but to relax the mandible in the supine position. Obviously, airway size is affected by this major change in mandibular position. This discrepancy was obvious from the onset but to compare our data

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base with all previous OSA reports, the maximum intercuspation position in the up-right position was used. Head position in the supine position was carried out without a fixed reference plane. Subjects were instructed only to mimick their normal sleep position. There was no standardized protocol to confirm whether the relationship between the head position on a pillow and the body posture on a stretcher actually represents a natural sleep position.

The present pilot study did not include EMG or pressure experiments in the OSA symptomatic group. An EMG and pressure study on the OSA group may well be the next logical step for future investigations. Overnight tongue pressure recording, synchronized with EMG, could be a final objective for this comparative study. If, by means of the current method, we could segregate the 'active' GG EMG from the 'passive' GG EMG (that EMG activity with no change in the muscle length during sleep), we may be able to develop a better understanding of the pathogenesis of OSA.

SUMMARY

The cephalometric study differentiated OSA patients from asymptomatic controls as follows:

1. The OSA group revealed a longer tongue, a larger soft palate, an anteroposteriorly narrower and superoinferiorly lengthened upper airway, a inferiorly positioned hyoid bone, a more up-right tongue, a more extended head posture and a smaller hypopharynx in the up-right standing position.

2. The OSA group showed greater tongue height, a larger tongue and a smaller hypopharyngeal area in the supine position.

3. With the positional changes from up-right to supine, the thickness of the soft palate increased more in the control group, whereas the size of the upper airway was decreased more in the OSA group.

4. With the positional changes from up-right to supine, the hyoid bone was moved up toward the mandibular plane more in the control subjects, but more toward the mandibular symphysis in the OSA group.

5. The tongue cross-sectional area increased significantly by 4.3% in the supine position and oropharyngeal area decreased by 36.5% in the OSA group.

As a summary of the rest EMG and pressure study, with the positional changes from up-right to supine, the asymptomatic control group showed:

1. The EMG activity of the genioglossus muscle was increased by 33.8%.

2. The pressure of the posterior portion of the tongue increased by 17%.

3. The resting potential activity of the inferior orbicularis oris decreased by 32.5%.

4. The EMG activity of the supra-hyoid group increased by 8%, yet the change was not statistically significant.

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