

CHAPTER 5: VOLUMETRIC ASSESSMENT OF BODY SPACES WITH THE AID  
OF COMPUTED TOMOGRAPHY

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Fig. 49. View of mandible, intra-oral, subject 11



## CHAPTER 5: VOLUMETRIC ASSESSMENT OF BODY SPACES WITH THE AID OF COMPUTED TOMOGRAPHY

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### 5.1 PRINCIPLES AND BACKGROUND

Computed Tomography has acquired many names. The common ones are Computerised Transverse Axial Tomography or CTAT (Perry and Bridges 1973), Computerised Axial Tomography or Computer-Aided Tomography or CAT (Scott and New 1974), Reconstructive Tomography or RT (Brooks and DiChiro 1975), Computerised Reconstruction (Cho, Ahn, Bohm and Huth 1974), Computerised Tomography or Computed Tomography or CT (Alfidi et al 1977).

Each of the above names refers to at least one aspect of the procedure. The most limiting term is CTAT because it pertains to the reconstruction by computer of those cross-sectional images that are transverse to the patient's long axis. The most general term is Computerised Reconstruction, which may be applied to non-medical as well as medical situations, and to reconstructions in three, in addition to two, dimensions.

In the conventional radiographic technique, a large proportion of the available information is lost in attempting to portray all the information from a three-dimensional body on a two-dimensional photographic plate, the image superimposing all objects from front to rear. In order that any one internal structure may be seen, it must clearly stand out against the variations of the materials in front and behind it (Hounsfield 1973).

The ideal goal, therefore, is the elimination, by more or less efficient blurring, of layers outside the focused plane and has been partially achieved by conventional tomography. By the use of special motions (transmission) or collimators (emission), unwanted planes are blurred while the desired layer is kept in focus. It is also clear, however, that under no circumstances can elimination be total (Brooks and DiChiro 1975).

A limitation of conventional tomography is that the blurred images

of the unwanted planes reduce the available contrast and the perception of low contrast objects even reduced further.

Computed Tomography is a new type of body-layer visualization, either radiographic or radioisotopic, which allows for complete exclusion of sections not under study. In this methodology the detected radiations pass through or originate from the desired anatomical plane without entering other areas. Projections of the radiations detected at multiple angles are used to reconstruct the anatomical layer, usually with the aid of a computer.

The reconstruction of the internal structures of an object from its projections is neither new nor novel, but has only recently been used in medical applications. Radon (1917) proved that a three-dimensional object could be reconstructed mathematically from an infinite set of projections. Practical techniques were first developed by Bracewell (1956) while identifying microwave radiation from the sun, and propounded by Olendorf (1961) who used a gamma ray source and a collimated scintillation counter to pass a photon beam through an object and measured the attenuation coefficient at the centre of rotation.

Further experimentation along a similar line were done independently by Cormach (1963), Kuhl and Edwards (1968), and Gordon, Bender and Herman (1970). It was, however, the collaboration of Hounsfield (1973) and Ambrose (1973) that made CT scanning of the human body a reality.

A few generations of CT scanners have been produced since, featuring head or body scans, with scan time ranging from 5 seconds to 120 seconds, having detector elements varying from 1 pair to 150 pairs, giving tomogram thickness of 2 to 13 mm, and able to produce computer rearranged sections.

Recently, direct sagittal CT scan of the face and paranasal sinuses have been obtained, and these nonarranged scans have spatial and density resolution nearly equal to that achieved in the standard axial plane, and are obtained with an equivalent patient dose (Osborn and Anderson 1978).

It is not the intent of this study to present the theory of image reconstruction of CT (Brooks and DiChiro 1975), but it is interesting to note that as high as 28,800 readings of transmission taken by a single detector can be stored and processed by an attached computer (Hounsfield 1973). A picture can be reconstructed from the data, given water the density of zero, and the read-outs expressing comparative densities of each segment of tissue within the tomogram, so that results can be displayed as:

- (1) A computer print-out of relative absorption coefficients.
- (2) A cathode ray tube display with the image brightness proportional to the absorption value.
- (3) A polaroid or a DeltaMat Multi-Format picture (Wortzman and Holgate 1976).

An accuracy of 2.5 per cent in CT image has been reported by Hounsfield (1973) who also suggested that this technique may open up a new chapter in x-ray diagnosis. It is therefore hardly surprising that CT has been praised as 'no comparable discovery in this field has been made since Roentgen discovered x-ray in 1895' (General Electric).

## 5.2 APPLICATIONS

The enormous impact of CT on Medicine has been put by Alfidi et al (1977) who said:

*'This extremely rapid development of Computed Tomography has resulted in a new horizon of imaging techniques that has significantly changed the present and future of both diagnosis and treatment' (Alfidi et al 1977, p.vii).*

The advantages of CT over conventional radiography are numerous. In conventional methods, differences in radiation transmission of less than 5 per cent are generally not detectable. This is due to the superimposition of bone and soft tissues and the existence of scattered radiation, even under the favorable conditions of small field size and low x-ray energy. In CT, scattered radiation is minimal and sensitive detectors are used. As a result, contrast differences of 0.5 per cent are resolvable (Abrams and McNeil 1978).

Skin surface irradiation is minimal because of collimation (Montgomery et al 1979), while a serial six tomographic slices covering the whole of the head only gives 1.9R skin exposure (Hounsfield 1973), the single-scan/multiple-scan exposure ratio varies from 1.0R to 1.9R (McCullough and Payne 1978).

The diagnostic effectiveness of CT is undeniable. Wortzman and Holgate (1976) attributed this to the excellent depiction of normal and abnormal anatomy, the non-interventional nature of the technique, and the ability to demonstrate variations in tissue density. Diagnostic accuracy, moreover, can be increased by introduction of a contrast medium such as Urografin (Wishaw 1980).

While pointing out that CT can provide accurate images of areas previously unseen or very poorly seen, Abrams (1978) asserts that in abdominal tumors alone, treatment will be improved in 25 per cent of the cases.

Some of the procedures CT has influenced are:

- (1) Pneumoencephalography. CT can differentiate between atrophy and normal pressure hydrocephalus as well as capable to visualize ventricles and cisterns.
- (2) Nuclear Medicine, CT is superior to radionuclide brain scans, especially in tumor identification.
- (3) Angiography. CT can define well cerebral hematomas and aneurysms thereby decreasing the need for cerebral angiography.
- (4) Neurosurgery. Precise surgical approaches possible from analyses of cross sections of brain in different planes.
- (5) Ophthalmology/ENT surgery. Exact localisation of tumors and foreign bodies are possible with CT. Air-containing structures such as the naso- and oropharynx are also well examined.

(Hounsfield 1973; Abrams and McNeil 1978; Ambrose 1973; Wortzman and Holgate 1976).

### 5.3 ACCURACY IN THREE-DIMENSIONAL ASSESSMENTS

Apart from the obvious purpose of diagnosis and lesion identification, Computed Tomography is also a proven useful tool in quantitative

assessment of irregularly shaped parts of the human body.

Frontal ventricular dimensions (Hahn and Rim 1976) and intracranial tumor volumes (Penn, Walser, Kurtz and Ackerman 1976) have been quantitatively evaluated. The sensitivity of CT in delineating boundaries of body cavities and the known slice thickness and scan minification factor allow measurements to be highly accurate (Miller, Dinismore, Wittenberg, Maturi and Powell 1977).

Such techniques involve the use of serial tomographic images, and the cavity volumes of individual tomographic scans can be totaled. The volume of each scan of given thickness is obtained either by the use of planimetry or from a numerical computer printout.

The application of planimetry to volume studies in Medicine was preceded by Venable (1945) in the calculation of volumes of more than eighty ova and blastocysts using serial sections of known thickness.

The basis of such deduction is derived from the integral equation employing the moving cross-section or slab method (Tierney 1975):

$$V = \int_a^b A(x) dx;$$

where  $V = \text{volume}$

$A(x) = \text{area of the cross section}$

$dx = \text{function of the distance moved}$

*If the thickness of the slab approaches zero, the function approximates total volume (Stein 1967).*

Areas of each CT scan can, therefore, be calculated by planimetric measurement, then corrected for minification and multiplied by scan thickness to give a slice volume. Alternatively, the volume of each pixel in the slice can be derived using the partial volume phenomenon to analyze the numeric printout of the scanner and the respective volumes obtained by simple addition (Miller et al 1977).

The accuracy of the above method has been verified by Miller et al (1977) when they compared CT volumes and actual paraffin cast volumes of arrested canine hearts, and found a statistically high correlation between them.

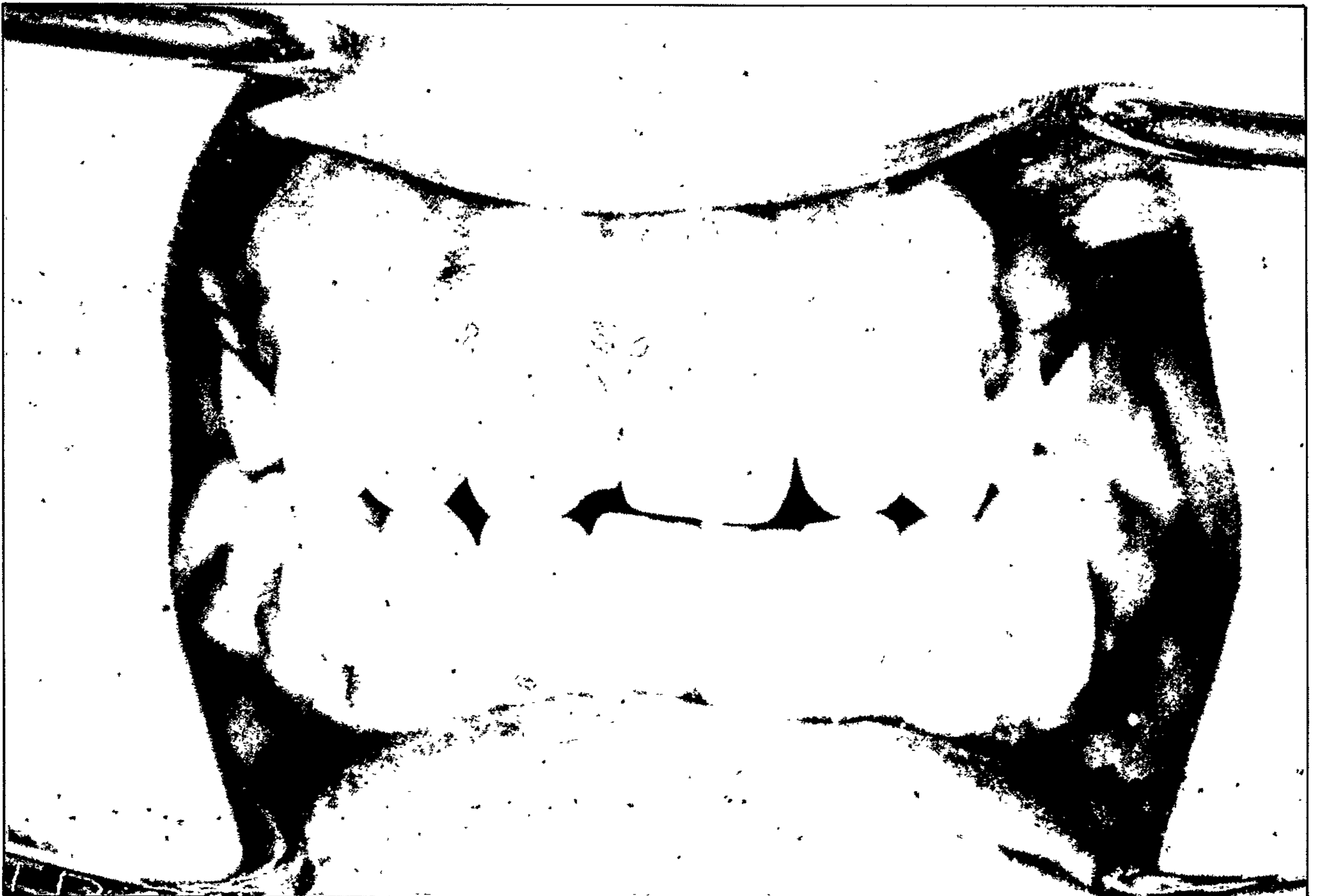
The thickness of the slice offers one limitation because a thick slice could obscure variation in tissue boundaries while the planimetric method does not take into account the potential error caused by an oblique orientation of the cavity wall in the volume calculation for an individual slice. Later investigations, however, showed that accuracy can be improved by making end cuts bisected and that computed tomographic measurements are largely independent of cavity orientation (Lipton, Hayashi, Boyd and Carlsson 1978).

The accuracy of CT-derived volume determination of the nasal airway has been similarly tested by Montgomery et al (1979), who also predicted that future use of CT in assessing amount of adenoid blockage is possible though no literature on such method is published to date.

CHAPTER 6: CLINICAL EVALUATION OF UPPER AIRWAY PATENCY

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Fig. 50. Frontal intra-oral view, subject 11



## CHAPTER 6: CLINICAL EVALUATION OF UPPER AIRWAY PATENCY

### 6.1 BACKGROUND

The smallest constriction of the upper airway, according to Martens (1979), may be found at the nasopharyngeal isthmus, the nasopharynx, the choanae, the nasal cavity or the anterior nasal nares. A constriction present at any of these regions invariably restricts the ability to breathe through the nose.

For this reason, the terms nasal patency and upper airway patency are used synonymously in the literature. A quantitative determination of upper airway patency, or nasal airway resistance data, provides objective values that can substantiate and quantitate the symptoms of nasal obstructions while rhinoscopic evidence of nasal deformity could not (McCaffrey and Kern 1979).

The first recorded means of measuring nasal resistance was probably by Zwaardemaker (1889) when he placed a cool mirror beneath the nares and estimated the respective patency of the two sides by the area and persistence of the condensed moisture on the mirror. Other rough methods include calibrating the pitch of the tone given off by air during unilateral nasal breathing (Zanjanian 1975) and the use of cotton butterfly placed under the nostril to be tested (Moyers 1973, p.332).

### 6.2 RHINOMANOMETRIC METHODS

Rhinomanometry - the measurement of nasal airway pressures for the evaluation of nasal airway patency - is probably the most widely used method.

The nasal airway resistance ( $R_n$ ), which is defined as the quotient between the transnasal pressure difference and the nasal airflow ( $dF/V$ ) and expressed in  $\text{cm H}_2\text{O/L/s}$ , can be measured directly and indirectly by a variety of methods.

Anterior rhinomanometry was initially used by Courtade (1902) and

later modified. This procedure requires the introduction of air, at constant or measurable flow rate, into one nostril while the other nostril is occluded but pressure recorded. Measurements are taken for both nostrils sequentially, at the peak of inspiration. Total nasal airway resistance can then be derived mathematically (McCaffrey 1979).

A limitation of this method is that it cannot be applied to patients with:

- (1) inability to relax soft palate,
  - (2) nasal septal perforation,
  - (3) complete obstruction of one or both nasal airways,
  - (4) inability to establish an adequate seal at the nozzles
- (Foxen, Preston and Lack 1971; McCaffrey 1979).

Posterior rhinomanometry was first introduced by Spiess (1899) and later adopted by Seebohm and Hamilton (1958). It involves the placement of two tubes in the oropharynx via closed lips. One tube allows the entry of air at a constant flow rate and the other connected with a pressure recording device by which the resistance to egress of air through the nose can be assessed.

A combined anterior and posterior rhinomanometric method has been suggested by Stoksted (1953a; 1953b) when he led a tube from one middle meatus to one limb of a U-tube manometer. The nostril on the same side is closed while breathing can be effected through the contra-lateral nostril. Electrodes in the manometer are connected with an electrocardiograph and the pressure readings obtained indicate nasopharyngeal pressures.

Instead of expressing degree of nasal patency by mathematical derivations, Aschan, Drettner and Ronge (1958) used a transducer to measure the difference in pressure between the nasopharynx and a nasal mask and a pneumotachometer to measure the actual amount of air flow. These two values are plotted against each other on an oscilloscope screen and the line obtained has a slope which is representative of the amount of nasal resistance.

Linder-Aronson and Backstrom (1960) employed a variation of this

method by substituting the transducer and pneumotachometer by manometer and venturimeter to measure nasal airflow in mouth breathing 9-year-olds.

A large number of modified versions has evolved since but they all suffer the drawback that the basic calculation is dependent on laminar fluid flow and the nasal airway resistance becomes unreliable at high nasal flow rates when turbulence invariably occurs. It is therefore recommended to use low flow rates of 0.4 litres per second (Taylor, MacNeil and Freed 1973) or 0.5 litres per second (Watson, Warren and Fischer 1968) which are compatible to normal quiet respiratory rate.

Notwithstanding the above obstacle, Watson, Warren and Fischer (1968) identified subjects with a nasal airway resistance above 4.5 cm H<sub>2</sub>O/L/s as clinically observable mouth breathers while McCaffrey (1979) noted the presence of obstruction symptoms in subjects with nasal airway resistance above 3 cm H<sub>2</sub>O/L/s and a high correlation between the two variables.

Hasegawa, Kern and O'Brien (1979), on the other hand, illustrated the dynamic nature of nasal resistance by reporting that the physiologic change in nasal resistance in a normal individual during a 15-minute interval should be less than 53 per cent of the previous observation.

Although cyclical changes in nasal resistance had been demonstrated as due to the state of congestion of nasal turbinates (Principato and Ozenberger 1970), unilateral resistance fluctuates in opposite phase. Total nasal resistance, on the whole, remains fairly constant, and is less than either of the individual unilateral resistances (Stoksted 1953a and 1953b).

### 6.3 EFFECTIVE CROSS-SECTIONAL NASAL AIRWAY

Sophisticated as they may be, most rhinomanometric methods are cumbersome and unreliable for rapid clinical determinations. Connel (1966) suggested a simple method to measure the effective

cross-sectional area of the nasal airway which reflects the degree of upper airway patency.

The patient breathes into a mask which extends from the upper lip to the forehead and across the cheeks laterally. Two openings in the mask system permit air to flow: one is the nasal airway ( $N_a$ ) and the other an opening to the atmosphere ( $M_a$ ), which can be varied. Pharyngeal pressure ( $P_p$ ) and mask pressure ( $M_p$ ) are recorded continuously during normal respiration.  $N_a$  can be calculated by the formula:

$$N_a = 2.5 \frac{(M_a)(M_p)}{P_p} .$$

The advantages of this device includes:

- (1) Patient's nose not physically touched.
- (2) All variables can be measured with existing equipment.
- (3) An airway measurement can be made with each inspiration.
- (4) Physiologic range is possible because patient's effort in breathing need not be constant or strenuous.

#### 6.4 NASAL AIR-FLOW ASSESSMENTS

Simpler methods which measure nasal air flow but correlate closely with rhinomanometric results have also been developed to evaluate the degree of upper airway patency.

As early as 1940 Uddstroemer used an air flowmeter to measure nasal air flow and expressed it as a percentage to the flow through the mouth.

Rasmus and Jacobs (1969) measured oral and nasal air flow velocities by using three bead thermistors, one for oral and one for each nasal respiratory passage. The thermistors are pre-heated to 75°C, and as they are cooled by oral or nasal air flow, the fluctuation of voltage resulted gives an indication of corresponding air velocities. The authors, however, accepted the errors caused by instrument positioning and normal physiologic respiratory fluctuation.

Feeling that pressure is an unsatisfactory modality to measure as

the actual pressures are very small in relation to the proportionately large pressure variations involved in rhinomanometric methods, Morrison (1969) devised a method using a nasal spirometer to measure nasal air flow capacity. It allows a constant supply of air to be passed into a nasal mask on a patient, and the volume of air unused calibrated with a spirometer. The main advantage, of course, is the simplicity.

When Wright and McKerrow (1959) developed the Peak Flow Meter, they were looking for a method to assess ventilatory capacity by a single forced expiration, giving the maximum possible figure but carrying low resistance at high flow rates. The instrument belongs to the class of 'variable area orifice meter' exemplified by the rotameter. The instrument is calibrated to the pressure and amount of air blown into it and the authors found it satisfactory to register maximum forced expiratory flow rate (or peak flow rate).

While thinking along the same line of simplicity and versatility, Taylor, MacNeil and Freed (1973) adopted the use of Wright Peak Flow Meter to assess the degree of nasal patency. Total expiratory effort through the nose (i.e., peak expiratory flow rate  $PEFR_n$ ) and the mouth (i.e.,  $PEFR_o$ ) are measured independently using interchangeable mouth and nose pieces. The function called Blockage Index (BI) is derived from the formula:

$$BI = \frac{(PEFR_o - PEFR_n)}{PEFR_o}$$

The value is independent of total expiratory effort, but correlates closely with nasal airway resistance, and is a reliable indicator of upper airway patency.

A more recent production of the mini Wright Flow Meter has also been proven to be reliable besides being economical, though bearing a less fine calibration.

Similar devices, like the peak-flow gauge, and the airflowmeter have been constructed since for a similar purpose (Klajman and Sitkowski 1961; Van der Lende et al 1974; Poppius and Mattson 1977).

## 6.5 CONCLUSION

While clinical symptoms can identify the mode of breathing (nasal, oral, or oro-nasal) and rhinoscopic examinations can establish the cause of nasal obstruction (nasal or adenoids), only nasal airway resistance data can assess the degree of upper airway patency.

CHAPTER 7: EFFECTS OF NASAL OBSTRUCTION ON GENERAL BODY STATES

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Fig. 51. Mirror image of left dentition, subject 11



## CHAPTER 7: EFFECTS OF NASAL OBSTRUCTION ON GENERAL BODY STATES

### 7.1 INTRODUCTION

A scan of the literature reveals a multitude of terminology allocated to denote states of upper airway patency: nasal obstruction, anterior or posterior obstruction, postnasal obstruction, obstruction of nasal respiration, upper respiratory obstruction, diminution of the airway, nasopharyngeal obstruction, and many more. Each of these terms usually depicts the site or manner whereby the upper airway is partially or completely occluded.

The term 'nasal obstruction' is most widely assigned in this context, and is also used in the present study, indicating a level of difficulty or inability to breathe through the nose. It is very much a clinical judgement implying some form of mechanical obstruction along the upper airway.

### 7.2 DIAGNOSIS AND ETIOLOGY OF NASAL OBSTRUCTION

As early as 1929, Schuller was able to demonstrate cases of nasal obstruction unrelieved by adenoidectomies, but caused by congenital hyperkyphosis of cranial base or by low position of the middle cranial fossa.

Rosenberger (1934) classified nasal obstruction into:

- (1) Anterior obstruction or occlusion of the nasal chambers, and produced by turbinate congestion or enlargement complicated by septal swelling or deviation.
- (2) Posterior obstruction produced by adenoid overgrowth.

Neivert (1939) ascribed chronic sinusitis, allergy or adenoids as causes of nasal obstruction.

The presence of nasal obstruction can be identified simply by holding the subject's mouth shut and see if nasal breathing is possible, and it is often found that the child had always been

too lazy to breathe through the nose (Fairchild 1968).

Thurston, Larson, Shanks, Bennett and Parsons (1980) listed the symptoms and signs of chronic nasal obstruction as:

- (1) Sleep disturbances - chronic daytime fatigue, personality change (depression, hostility, paranoia), morning headaches, hypersomnolence, snoring, nocturnal choking spells, and frequent awakenings.
- (2) Decreased exercise tolerance - shortness of breath with exertion, and mouth breathing.
- (3) Decreased appetite - growth arrest, failure to thrive, lessened olfaction, and difficulty in eating and swallowing.
- (4) Denasal speech.
- (5) Chronic nasal drainage - frequent or persistent 'colds'.
- (6) Cor Pulmonale - systemic hypertension, and congestive heart failure.

Having identified nasal obstruction, the etiology can be evaluated firstly by clinical examination which may include palpation of the adenoid tissue (Fairchild 1968) or a complete rhinoscopic examination (McCaffrey 1979) to assess:

- (1) Structural abnormalities, i.e., of the bony or cartilaginous skeleton of the nose, including the septum, lateral nasal walls, upper lateral cartilage, lobular cartilage, soft tissue scars, and synechiae.
- (2) Mucosal abnormalities, i.e., disorders of the nasal mucosal lining, including allergic rhinitis, vasomotor rhinitis, and rhinitis medicamentosa.
- (3) Mixed abnormalities involving a combination of above conditions.

Brodie (1971), in addition to those described by Rosenberger (1934), suggested that constricted posterior choanae can be a cause of nasal obstruction.

The results of studies done by Emslie, Massler and Zwemer (1952), Reed (1963), Paisner (1972), and Gray (1977) reveal the following accepted causes of nasal obstruction:

- (1) Acute rhinitis, accompanying the common cold or an upper respiratory infection.

- (2) Chronic rhinitis, may be due to -
- (a) Allergic rhinitis: particularly at the posterior end of the inferior turbinates, gives an intermittent or variable obstruction.
  - (b) Vasomotor rhinitis: reflects an inability of the autonomic nervous system to evoke a proper vasomotor response of the nasal mucosa to variations in temperature and humidity.
  - (c) Atrophic rhinitis: an idiopathic progressive atrophy of the nasal mucosa producing crustings which enlarge and obstruct nasal breathing.
  - (d) Nose-dropitis: due to excessive use of nasal vasoconstrictors.
- (3) Anatomic variations, may be due to -
- (a) Deviated septum: may be congenital or acquired.
  - (b) Flaccid or collapsed alae nasi.
  - (c) Thickening of the columella.
  - (d) Choanal atresia: which is a bony or membranous obstruction at the posterior nares. When it occurs bilaterally in a newborn must be attended to immediately because a newborn does not know how to breathe through the mouth.
  - (e) Trauma.
- (4) Septal hematoma and abscess, can result from acute trauma or extension of a contiguous nasal cellulitis or furuncle.
- (5) Foreign bodies, usually unilateral.
- (6) Tumors, may be benign like polyps, fibromas and papillomas, or malignant like sarcomas.
- (7) Nasopharyngeal obstruction, can be the presence of -
- (a) Adenoids: contrary to common belief, Gray (1977) does not regard it as a common cause.
  - (b) Choanal polyp arising from chronic maxillary sinus infection.
  - (c) Nasopharyngeal tumors.

Paisner (1972) further suggested that bilateral nasal obstruction in a child indicates enlarged adenoids, allergy or deviated septum, in that order, while unilateral obstruction indicates choanal atresia or foreign body. In adults, the nasal passages may be blocked by polyps, a benign or malignant tumor, or a septal hematoma. He also

reported the rare incidence of 'the nose of pregnancy' and 'the nose of new bride', both associated with vasomotor rhinitis.

While Piness and Miller (1940) pointed out that allergy alone may produce chronic nasal obstruction without other symptoms, Hansel and Chang (1940), in an observation involving 200 children indicated for adenoidectomy, established nasal allergy as the cause of 13 per cent of the cases only.

Although Ardran and Kemp (1972) regarded adenoids as 'space fillers', they agreed that infected adenoids could lead to nasal obstruction. Pratt (1974) on the other hand cited cases of nasopharyngeal stenosis or obstruction of the nasopharynx caused by scar tissue after adenoidectomy.

Besides the above local and environmental conditions, Ricketts (1968) considered genetics or heredity as the primary etiologic factor, and regarded anomalies of the skeletal structural frame attributable to nasal obstruction. These anomalies include abnormal growth of the mandible, cranial dysostosis, abnormal cranial base angle and 'micro-rhino-dysplasia'.

### 7.3 POSSIBLE SEQUELAE OF NASAL OBSTRUCTION

Although the nose offers a much higher resistance to air flow than the mouth, nasal respiration is naturally obligatory in the infant (James and Hastings 1932; Paisner 1972), and a preferred mode of breathing in the adult.

Rubin (1979) even considered it life threatening if choanal atresia is not diagnosed promptly after birth because of the inability of the baby to breathe through the mouth. A significant number of infants by age two years are, however, mouth breathers.

The functions of the nose include olfaction and respiration. In serving the respiratory needs, the mucous membrane cleanses, warms and moistens the air (Emslie, Massler and Zwemer 1952).

The ramifications of nasal obstruction can therefore be considered as local responses due to the nature of the obstruction, and as systemic effects due to impediment of nasal respiration.

### 7.3.1 Local

Emslie, Massler and Zwemer (1952) observed that the sense of smell is dulled along with taste and appetite in cases of nasal obstruction. Occasionally, hyperplasia of the adenoids will occlude the opening of the Eustachian tube and results in defective hearing and recurrence of otitis media (Poncher, Schour and Massler 1945).

The associations of ear diseases with adenoidal hyperplasia is also supported by Reid and Donaldson (1970) who reported the presence of serous middle ear effusions, secretory otitis media, recurrent otitis media, or even chronic suppurative otitis with damage to the tympanic membrane, ossicles and tympanic cavity. On top of these, Devgan and Leach (1979) found chronic maxillary sinusitis and rhinolalia clausa at times.

By using contrast radiographic and manometric techniques on pre- and post-adenoidectomised children, Bluestone (1975) was able to assess the ventilatory function of the Eustachian tube. He concluded that the obstruction of Eustachian tube can be mechanical, or functional, exemplified by the effect of pressure on a pliant tube. He also suggested that adenoid size is not necessarily predictive of Eustachian tube function (or malfunction), though most children with large adenoids have retrograde obstruction.

Hindrance of speech production may also occur if the obstructive adenoids occlude the posterior aspect of nasopharynx (Morris 1975), though facilitation of speech is due to the adenoidal pad providing a cushion against which velar contact is achieved in cleft palate subjects (Subtelny 1956).

If the nature of the obstructive lesion is pathologic, adjoining tissues will be affected. Metastases and invasion of intra-cranial contents are invariably expected from nasopharyngeal carcinomas (Harrison 1972; Muir 1972).

There is, of course, the controversial implications of nasal obstruction leading to mouth breathing, thereby affecting head posture and craniofacial morphology (Vig, Showfety and Phillips 1980). This subject will be elaborated in the subsequent chapter.

### 7.3.2 Systemic

Possible systemic sequelae of nasal obstruction were suggested as early as 1939 by Neivert who reasoned that the resultant mouth breathing will lead to irritations in the mouth, pharynx, larynx, and lungs, producing various degrees of inflammatory reactions in all parts of the respiratory tract.

Since nasal airway resistance accounts for 42 per cent of the total respiratory resistance (Craig, Dvork and McIlreath 1965), a rise of nasal airway resistance due to nasal obstruction must have profound implications in the cardiorespiratory system.

In studies involving normal and abnormal nose groups, Ogura (1964), and Ogura, Unno and Nelson (1968) reported an increase in pulmonary resistance during mouth as well as nasal breathing in cases with high nasal obstruction, probably due to changes within the air passages. Further investigations by Unno, Nelson and Ogura (1968) revealed that both components of pulmonary resistance, i.e., airway resistance (friction between the respiratory air and walls of the respiratory tracts) and tissue resistance (friction in displacement of the lung tissues), are increased concomitantly.

It is possible to infer from the above results that in subjects having nasal obstruction, some kind of changes also occur in the broncho-bronchiolar system and in lung tissues, and these functional and reversible changes play a role in the reduction of compliance and the increase in tissue resistance.

Although hypoventilation as a sequelae of nasal obstruction was accounted for by Comroe et al (1955), it was not until 1965 when cor pulmonale secondary to enlarged tonsils and adenoids were first recognised by Menashe, Farrehi and Miller.

The authors noted the narrow nature of the upper airway of an infant, and affirmed that hypertrophy of adenoids can lead to hypoventilation, pulmonary hypertension, right ventricular hypertrophy, and finally right ventricular heart failure.

This cardiorespiratory syndrome due to enlarged adenoids has been further substantiated by Thanopoulos, Ikkos, Milingos and Foutakis (1975) and many others. Talbot, Robertson and Richmond (1973) reported 4 of 36 cases have died, and concluded that early recognition and treatment of progressive upper airway obstruction is curative.

It is also interesting to note that in children having concomitant congenital heart disease, the early management in this regard is crucial (Clairmont, Hart, Rooker and Franch 1975).

#### 7.4 THE T'S AND A'S PROBLEM

Ever since Wise (1867) reported tonsillectomies performed by skilled Asiatic Indians 1,000 years before Christ, the problem of tonsils and adenoids has perplexed clinicians and parents alike. Notwithstanding the plea for fewer operations by Layton (1914), it is believed that adenotonsillectomy is one of the most frequently performed surgical procedures in the United States involving approximately one-third of all children and an additional two million per year (Reid and Donaldson 1970).

Tonsillectomy used to be done concurrently with adenoidectomy for fear of subsequent necessity. This notion, however, has been found unjustified (Young 1962) as tonsillectomy does not resolve problems caused by adenoids, or vice versa. The prevailing trend, therefore, is that the combined procedure is desirable only if indications for each are present (Reid and Donaldson 1970).

While tonsils and adenoids are not regarded as true lymph nodes (Neivert 1939) and they do not follow Scammon's lymphatic growth curve (Pruzansky 1975), their functions have been regarded as immunologic (Fairchild 1968; Morag and Ogra 1975; Gray 1977), as

space fillers (Ardran and Kemp 1972), and to assist velopharyngeal closure in cleft palate subjects (Subtelny 1956), the last two functions pertaining to adenoids only.

The enlargement of adenoids, be it allergic, infectious, idiopathic, or iatrogenic in origin, causes a series of possible but unwanted local and systemic complications as reviewed earlier. It seems that their routine removal would be preventive, and surgery as symptoms arise would be curative. Or is it not?

Numerous benefits have been proclaimed resulting from successful adenoidectomy. Amongst them, a reduction of purulent otorrhea and other ear diseases (Kaiser 1930; Neivert 1939; McKee 1963; Roydhouse 1970), an increase in ventilation and compliance of Eustachian tube (Bluestone 1975), a relief of cardiorespiratory distress (Menashe, Farrehi and Miller 1965; Talbot, Robertson and Richmond 1973), restoration of nasal breathing (Eley 1954; Linder-Aronson 1973), and improvement of dentofacial characteristics (Linder-Aronson 1972).

Despite the above, futility of adenoidectomy has also been related. Mawson, Adlington and Evans (1967) were able to show that adenoids removal has no apparent influence on ear disease, and that after two years there will be very little difference between the operated and control groups.

While complete surgical enucleation of the adenoids is often impossible (Meltzer 1954; Steele 1968), recurrence of adenoid tissue has been observed (Gray 1977), and nasopharyngeal stenosis may revive nasal obstruction (Pratt 1974). Restoration of nasal respiration does not always follow (Yip and Cleall 1971), and allergic symptoms may not be alleviated (Hansel and Chang 1940).

Although Peller (1934) felt that adenoidectomised children grow faster, Hoople (1954) saw fit to remove adenoids from a 3-month old child, and Neivert (1939) could not acquire evidence to condemn adenoidectomy, serious post-operative complications have been reported in 1.5 per cent of all cases (Gibb 1969).

Bearing in mind the inherent risk of surgery and general anesthesia, some post-operative complications associated with adenoidectomies are: death, haemorrhage, speech disability, nasopharyngeal stenosis, granular pharyngitis, loss of immunity, temporomandibular joint dysfunction, and adverse psychological effects on the child (Gray 1977).

The days of routine adenoidectomy and tonsillectomy are rapidly fading, and the current opinion is to perform either operation as indications dictate, and rarely is it done on a preventive basis.

In a study to evaluate efficacy of tonsillectomy and adenoidectomy, Paradise (1976) recommended surgery only for subjects who are definitely threatened, handicapped, or rendered uncomfortable by the tonsil or adenoid related problems. While tonsillectomy is indicated mainly in association with chronic infections, the criteria for adenoidectomy are:

- (1) Persistent nasal obstruction
  - (a) apparently not due to allergy, and
  - (b) accompanied by both clinical and radiographic evidence of adenoid hypertrophy, and
  - (c) manifested by mouth breathing and hyponasal speech. Or
- (2) Recurrent serous or suppurative otitis media, if myringotomy and insertion of tympanostomy tube have been performed at least once previously. Or
- (3) Chronic sinusitis or nasopharyngitis
  - (a) apparently not due to allergy, and
  - (b) accompanied by both clinical and radiographic evidence of adenoid hypertrophy, and
  - (c) persisting despite appropriate antibiotic and other medical therapy.

One therefore should not regard the removal of adenoids and tonsils as a trivial procedure (Badger 1954), must possess a sober judgement (Boies 1954), and be cognizant of the physician's dilemma in the land of belabored lymphoids (Watson 1979).

CHAPTER 8: ASSOCIATIONS OF NASAL OBSTRUCTION, ADENOIDS, AND  
MOUTH BREATHING WITH DENTOFACIAL MORPHOLOGY

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Fig. 52. Mirror image of right side dentition, subject 11



## CHAPTER 8: ASSOCIATIONS OF NASAL OBSTRUCTION, ADENOIDS, AND MOUTH BREATHING WITH DENTOFACIAL MORPHOLOGY

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### 8.1 INTRODUCTION

A glance at the literature shows a number of seemingly interchangeable terms describing the mode of breathing: nasal breathing, oro-nasal breathing, partial mouth breathing, complete mouth breathing, continuous or intermittent mouth breathing, obstructive mouth breathing, habitual mouth breathing, and so on. Each of the above terms designate a state of upper airway patency, i.e., whether there is complete, partial or no nasal obstruction.

Massler and Zwemer (1953) clarified the situation by dividing mouth breathing into two types:

- (1) Habitual: often performed at night, patient unaware, and nose generally patent. May present partial or intermittent nasal obstruction.
- (2) Obstructive: usually a complaint, and patient cannot breathe through the nose even on demand. An obstruction somewhere along the upper airway.

Functionally, mouth breathing can be classified as:

- (1) Total and continuous: when nasal passages are completely occluded and respiratory entirely oral. Habitual mouth breathing can be total and continuous.
- (2) Partial and intermittent: when nasal passages are partially occluded so that nasal breathing is supplemented by intermittent oral breathing. Habitual mouth breathing, again, can be partial and intermittent.

Backlund (1963), however, preferred the term oro-nasal breathing because mouth breathing alone is exceedingly rare. Vig (1980) regarded complete occlusion of the nose as impossible, and there is always some air flow through the nose during respiration rendering the term 'mouth breathing' a misnomer.

For the purpose of this investigation, the term 'mouth breathing' is

used to imply some form of nasal obstruction demanding oral respiration usually, and the term 'habitual mouth breathing' denotes oral respiration due to habits or muscular weakness but the upper airway is patent.

## 8.2 DIAGNOSIS AND ETIOLOGY OF MOUTH BREATHING

The genuine 'mouth breather' can be distinguished from the 'habitual mouth breather' by simply asking the subject to shut the mouth and breathe (Massler and Zwemer 1953). If difficulty is incurred, nasal obstruction is suspected. Otherwise, habits can be the cause.

Although Gwynne-Evans (1958) stated that mouth breathing due solely to gross nasal obstruction is comparatively rare and that due to failure of the orofacial muscles is common, most current clinicians would disagree.

Emslie, Massler and Zwemer (1952) described the etiology of mouth breathing as multiple, and dependent on a combination of anatomic predisposition (facial type or airway) and precipitating factors (local causes of nasal obstruction).

Granting the validity of the above theory, any one of the causes of nasal obstruction listed in the foregoing chapter can therefore attribute to mouth breathing. While enlarged adenoids do not seem to be a major cause of nasal obstruction (Gray 1977), there is controversy in dental literature over the frequency of adenoid hypertrophy (implying a diminished nasopharyngeal space) concurrent with mouth breathing.

While consenting that hypertrophied adenoids could interfere with respiratory passage, Todd (1936) did not agree to an association between that and mouth habits.

On the other hand, James and Hastings (1932) documented that infected adenoids can cause hypertrophy and produces oral breathing. Backlund (1963) even cited a number of conditions and concluded that adenoids as being the most common cause of mouth breathing. Murray and

Anderson (1969) further reported that enlarged adenoids and allergic nasal obstruction are two common causes of mouth breathing.

A coexistence between adenoids and mouth breathing is also believed by Brash (1929), Sillman (1942), Leech (1958), and Subtelny (1975).

The idea that the absolute size of the adenoids is not as important as the relative size of adenoids to nasopharynx was put forward by Emslie, Massler and Zwemer (1952), and elaborated by Lubarth (1960), Ricketts (1968), and Linder-Aronson (1970). They found that nasal obstruction leading to mouth breathing is related to the size of the nasopharynx. The smaller the nasopharynx, the less the amount of adenoidal enlargement needed to obstruct the airway.

The conjecture of nasal allergy causing nasal obstruction, and then leading to mouth breathing was verified by Balyeat and Bowen (1934), Straub (1944), and Marks (1965).

The mechanisms of the two types of mouth breathing were explained by Gwynne-Evans (1958) as:

- (1) the soft palate is elevated from the tongue in order that breaths may be drawn deliberately through the mouth because of nasal obstruction, or
- (2) the tongue lies naturally away from the soft palate whether nasal obstruction is present or not.

Whereas habitual mouth breathers are not uncommon, the open mouth habit should not be confused with actual mouth breathing. Massler and Zwemer (1953) cautioned that many children habitually keep their lips apart, especially with a characteristic familial short upper lip, but they breathe normally through the nose.

The etiology of mouth breathing is undoubtedly multiple. It is summed up by Emslie and Massler and Zwemer (1952) to comprise of a predisposing factor (facial type) governing the airway size, and precipitating factors (local environmental) indentifiable as nasal or nasopharyngeal obstruction, the combined effects of both determine the degree of mouth breathing.

### 8.3 ADENOIDS, MOUTH BREATHING, AND DENTOFACIAL MORPHOLOGY

It is believed that the nasorespiratory area plays an important role in dentofacial development and in the developing facial configuration (Subtelny 1975). On this theme, there evolved the so-called 'adenoid facies', descriptive of a particular facial expression in association with hypertrophied adenoids and mouth breathing. The 'long face syndrome' or the 'open mouth syndrome' also depict the same set of dentofacial characteristics:

*'The mouth stays open, the nose appears flattened, the nostrils look small and underdeveloped, the upper lip short, the lower lip thick and everted, and - especially due to the half-open mouth - the individual has a vacant stupid expression. The dentition is stated to be of a special type, consisting of protruding upper incisors, a narrow V-shaped upper jaw with a high palatal vault, and a post-normal relation between the upper and lower jaws' (Linder-Aronson 1970, p.5).*

It has also been noted that mouth breathing secondary to allergy if occurring at an early age can lead to a similar type of dentofacial anomaly (Bowen and Balyeat 1934; Cohen 1939; Straub 1944; Marks 1965). Such deformity has been named as 'a typical mouth breather', 'allergic face', 'typical gothic arch', or even 'adenoid facies', and classified according to malocclusion, usually Class II division 1 (Straub 1944).

This type of anomaly is thought to be induced by perennial nasal allergy, which interferes with the bony development of the nasal process of the maxilla, the anterior portion of the zygomatic arch and the area over the antra, and causes contraction of the maxillary arch with protraction of the anterior teeth (Todd 1938; Fuchs 1939).

Duke (1930), on the other hand, described a type of allergic face characterised by paranasal depression, prominent nose, depression below the zygomatic arch and prominent cheek bones due to underdevelopment of ethmoid cells and antra. He further disclosed that this type of face differs from that caused by adenoids or mouth breathing in the absence of the pinched appearance of the nose, lack of prominence of upper incisors and the absence of abnormal mouth or abnormal lower jaw.

'Adenoid facies' is therefore a collection of descriptions, some of which are disputed upon, without indication of etiology. Incisor protrusion, for example, has been denied by Murray and Anderson (1969) to be present in children with enlarged adenoids and nasal obstruction. They nevertheless postulated that allergic palatal itching can be a stimulus for increased digital sucking, resulting in more incisor protrusion in some cases only.

Linder-Aronson (1970) even went a step further, and regarded retroclination of upper and lower anterior teeth to be part of this loosely-termed syndrome. He ascribed this to adverse lips pressures concomitant with mouth breathing.

Perhaps it is neither allergy nor adenoids that led to 'adenoid facies', but the persistence of mouth breathing during a rapid growth phase of the face that results in such dentofacial anomalies. The dispute seems to centre on the prevalence of adenoids or allergy in this context.

There are others, however, who do not see any correlation between adenoids and mouth breathing on the one hand, and certain dentofacial characteristics on the other. To further decompose the issue, some researchers contend that the effects of adenoids are manifested only under some hereditary conditions.

### 8.3.1 Significant relationships

Tomes (1872) is the earliest author quoted by Linder-Aronson (1970) to document that V-shaped, contracted jaws are generally found in association with greatly enlarged adenoids which necessitate mouth breathing. He considered that the physical forces at work - namely, the open lips and relaxed tongue - are sufficient to account for all the phenomena observed.

Three theories have been put forward to explain the changes involved:

- (1) Compression theory. The imbalance between pressure from the tongue and the cheek muscles results in the alveolar process in the premolar region being pressed medially by the cheek muscles at the same time as the anterior part of the upper jaw

is pressed forward.

While many authors shared the same conjecture (Angle 1907; Bowen and Balyeat 1934; Neivert 1939; Massler, Poncher and Schour 1945; Subtelny 1954; Negus 1955), justification was provided by Sprawson (1947) who illustrated the formation of 'saddle arch' due to a combination of adenoids and rickets. Also, Emslie, Massler and Zwemer (1952) found this musculature action particularly noticeable during the mixed dentition period.

- (2) Inactivity theory. The height of the palatal vault increases to the same extent as the nasal cavity becomes atrophied as a result of inactivity (Bentzen 1903; Nordlund 1918).
- (3) Excavation theory. The high palate which develops when there is an impediment to nasal breathing is a result of the upper direction of the air stream, which impinges on the palate in mouth breathing (Nordlund 1918).

Kantorowicz (1916) and Wustrow (1917) also expressed the view that the high palate in these cases can be due to the raised negative air pressure in the nasal cavity and the resulting increased difference between the pressure in the mouth and nose.

Although Johnson (1936) supported the idea of negative pressure in the mouth having a molding effect, he observed the presence of mouth breathing in a large range of malocclusion.

Linder-Aronson (1970) reported a relationship between adenoids and subsequent mouth breathing, and a narrow maxillary arch, high incidence of cross-bite or tendency towards cross-bite, retroclined upper and lower incisors, a small sagittal depth of the nasopharynx and an increased anterior facial height.

Further substantiation was provided in later studies (Linder-Aronson 1972, 1973a and 1973b). The normalisation of mode of breathing, depth of nasopharynx and dentition characteristics were noted in children one year after adenoidectomy, explained on the cessation of mouth breathing altering oral muscular forces.

Recent studies, however, were focused on the effects of enlarged adenoids on posture of the head and mandible.

Ricketts (1958a) claimed that a posterior rotation of the mandible occurs in mouth breathers since they frequently maintain a somewhat rotated backward head posture. Similarly, Koski and Lahdemaki (1975) observed that children with obstructive adenoids have dorsal rotation of ramus.

The mechanisms leading to a different posture of the mandible were explained by Thurow (1973), Hanson and Cohen (1973), Martens (1979), and Vig, Showfety and Phillips (1980) on the basis of the need to maintain a patent airway despite blockage somewhere in the upper pharynx. In order to adapt to mouth breathing, the tongue is maintained low and forward. A greater force is also exerted by the geniohyoids, giving maximum mandibular opening effect but predisposing to a steep mandibular plane and distoclusion.

The basis of the above mechanisms was also put forward by Moss and Salentijn (1971) who visualised the translative growth of the jaws as part of the capsular functional matrix comprising of oro-nasopharyngeal functional spaces determined by volumetric demand of the respiratory system.

Some verification was asserted by Harvold, Vargervik and Chierici (1973) who showed an increase in anterior face height in monkeys with experimentally induced mouth breathing. Further evidence was given by Vig, Showfety and Phillips (1980) who observed a progressive change in head posture in experimentally induced mouth breathing human subjects.

The concurrence of diminished upper airway (i.e., large adenoids) and distoclusion of the mandible was also justified by Mergen and Jacobs (1970) who found a larger midsagittal nasopharyngeal dimension in subjects with Class II malocclusion.

On a similar premise, Hanson and Cohen (1973) suggested that large tonsils cause crowding of tongue leading to malocclusion, whereas Ricketts (1958) reported a change of tongue position after removal of tonsils.

Although mouth breathers because of enlarged adenoids will probably

outgrow the habit, a good proportion of craniofacial growth and development will have occurred by the time the adenoids atrophy (Subtelny 1975).

### 8.3.2 Insignificant relationships

The basis for disregarding any causal relationship between adenoids, mouth breathing, and 'adenoid facies' is either frequent observation of all types of dentofacial characteristics in mouth breathers or a mouth breather having a normal face.

In as early as 1888, Kingsley stated that he could not accept the view that muscular contraction of the cheeks could produce a contraction of the palate. He felt that the V-shaped palate is inherited and not acquired through mouth breathing.

While Siebenmann (1897) observed a common occurrence of adenoids in leptoproscopic individuals, McKenzie (1909) reporting on his examination of 222 children with adenoids, noted that over 40 per cent had normal palates.

Whitaker (1911) ascribed thyroid hormone deficiency as a cause of contracted maxilla and found no evidence for a causal relationship between adenoids and dental deformity.

Nordlund (1918), Brash (1929), and James and Hastings (1932) all found contracted maxillae in individuals never troubled by adenoids.

Howard (1932) examined 500 patients with histories of enlarged faucial tonsils. He found that 159 were definite mouth breathers. Of these 59.1 per cent had a normal occlusion, 21.3 per cent were Class I, 13.9 per cent were Class II, and 5.7 per cent were Class III. These results clearly show that the occlusions of mouth breathers are distributed among all types of occlusion, rather than being restricted to one particular or classic type.

While attributing nasal allergy as a chief cause of facial deformity, Bowen and Balyeat (1934), and Balyeat and Bowen (1934) did not find sufficient evidence to assign any significant correlation between the

presence of adenoids and the incidence of the dental-palate deformity. They further concluded that heredity must be considered as an important causative factor in all types of malocclusion.

Sillman (1942) conducted a longitudinal study on children from birth to 5 years of age and did not find that adenoids and mouth breathing constituted a primary etiological factor in malocclusions. Hartsook (1946), and Huber and Reynolds (1946) also shared the same opinion.

Miller (1949) denied any connection between allergy and malocclusion while Humphrey and Leighton (1950), in an investigation of 1033 cases, found an approximately equal distribution of normal and post-normal occlusion in both mouth and nose breathers. Of all those who kept their mouths open, almost half breathed through the nose only.

In an article suggesting treatment for mouth breathers, Massler and Zwemer (1953) decided that mouth breathing may accentuate and aggravate but probably does not cause a Class II division 1 malocclusion.

Gardiner (1956), after a study of malocclusion and etiological factors in 1,000 Sheffield school children, found that only 23 per cent of open mouth habiters have open-bite or other dental anomalies.

In studies covering more than 15 years, Ballard (1948), Gwynne-Evans (1948), Gwynne-Evans and Ballard (1957), and Gwynne-Evans and Ballard (1958) concluded that oral and facial morphology remains strikingly constant during growth. They believed that mouth breathing does not cause jaw deformities and malocclusion, and nor does it lead to 'adenoid facies'. They further indicated that 'adenoid facies' is a type of natural physiognomy related to hereditary factors rather than to any structural defects. The authors also found no special morphological type coincidental with mouth breathing.

Whereas Leech (1958) in a study of 500 patients at an upper respiratory clinic pointed out that mouth breathing does not seem to affect the skeletal or occlusal relationship or the width of the jaw, Linder-Aronson and Backstrom (1960) drew the same conviction after

investigating 115 children of an average age of 10 years.

While endorsing an association between low tongue posture, non-contact between tongue and soft palate, and separated lips with mouth breathing, Backlund (1963) found these factors only secondary. He also failed to locate any significant correlation with either type of breather and width of upper dental arch, inclination of upper incisor or overjet. Results were therefore conclusive that mouth breathing could not be a causal factor in persistent malocclusions.

In a study relating nasal airway resistance to 51 children, Watson, Warren and Fischer (1968) found that the magnitude of nasal resistance, the breathing pattern, and the skeletal classifications of the subjects are independent of one another.

Similarly, Yip and Cleall (1971) were unable to prove that enlarged adenoids and tonsils may cause forward positioning of the tongue both at rest and during deglutition.

### 8.3.3 Conditional relationships

The vast amount of literature supporting each of the opinions for or against a causal relationship between adenoids, mouth breathing, and dentofacial morphology only asserts the multi-factorial nature of this controversy.

Perhaps the two opposing points of view can be reconciled on the basis that mouth breathing is injurious only to the narrow-faced individual who already possesses a narrow arch - one which can be further contracted easily by slight abnormalities in muscular forces.

It has been said that even in the absence of mouth breathing, the narrow-faced individual has a strong tendency to a contracted palate and a Class II division 1 type of malocclusion (Herbst 1910). It was also an observation that adenoid facies and the open-mouth habit occur only in a particular type of individual (James and Hastings 1932).

In as early as 1921, Bilderback realised that the removal of adenoids will not cure the mouth breathing unless there is a normal coaptation of the teeth which implies a normal development of the maxillary arch.

The supposition that the effects of adenoids are more pronounced in individuals with a small nasopharynx, and the importance of adequate nasopharyngeal volume have been well-documented (Nordlund 1918; Brash 1929; Schuller 1929; Emslie, Massler and Zwemer 1952; Ricketts 1954; Subtelny 1954; Goldman and Bachman 1958; Lubarth 1960).

In a series of follow-up studies, Linder-Aronson (1972, 1973a and 1973b) showed normalisation of dentofacial characteristics following adenoidectomy on patients with large adenoids relative to their bony nasopharynx ascertaining that adenoids affect the mode of breathing, which then influences the individual's dentition.

Emslie, Massler and Zwemer (1952) believed that mouth breathing does accentuate a Class II division 1 type of malocclusion, but only in the genetically narrow-faced and narrow-arched individual with a strong natural tendency in this direction. Broad-faced individuals are seldom subject to mouth breathing, and suffers less consequences even when it occurs.

Adenoids are therefore not the sole cause of mouth breathing leading to 'adenoid facies'. Other precipitating factors like allergy (Todd 1936; Balyeat and Bowen 1934), and a predisposing factor like dolichoprosopic individuals (Quick and Gundlach 1978) are needed to bring forth a latent genetic tendency to a narrow arch and Class II division 1 malocclusion (Emslie, Massler and Zwemer 1952).

#### 8.4 CONCLUSION

If mouth breathing is to affect dentofacial morphology, it must be prevalent continuously during the rapid growth period of the cranio-facial skeleton. Most of the causes of nasal obstruction can be alleviated by medical or physical means so that mouth breathing so induced is invariably intermittent. On the other hand, hypertrophy of adenoids superimposed by allergy or chronic infection occurs

frequently in early childhood. Once they become sufficiently obstructive, mouth breathing will pursue until ultimate regression of adenoids, often a few years later.

Granting the above possibility, inherent characteristics of 'adenoid facies' in certain individuals may certainly be accentuated.

## **Section III**

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### **Aim of the Present Investigation**

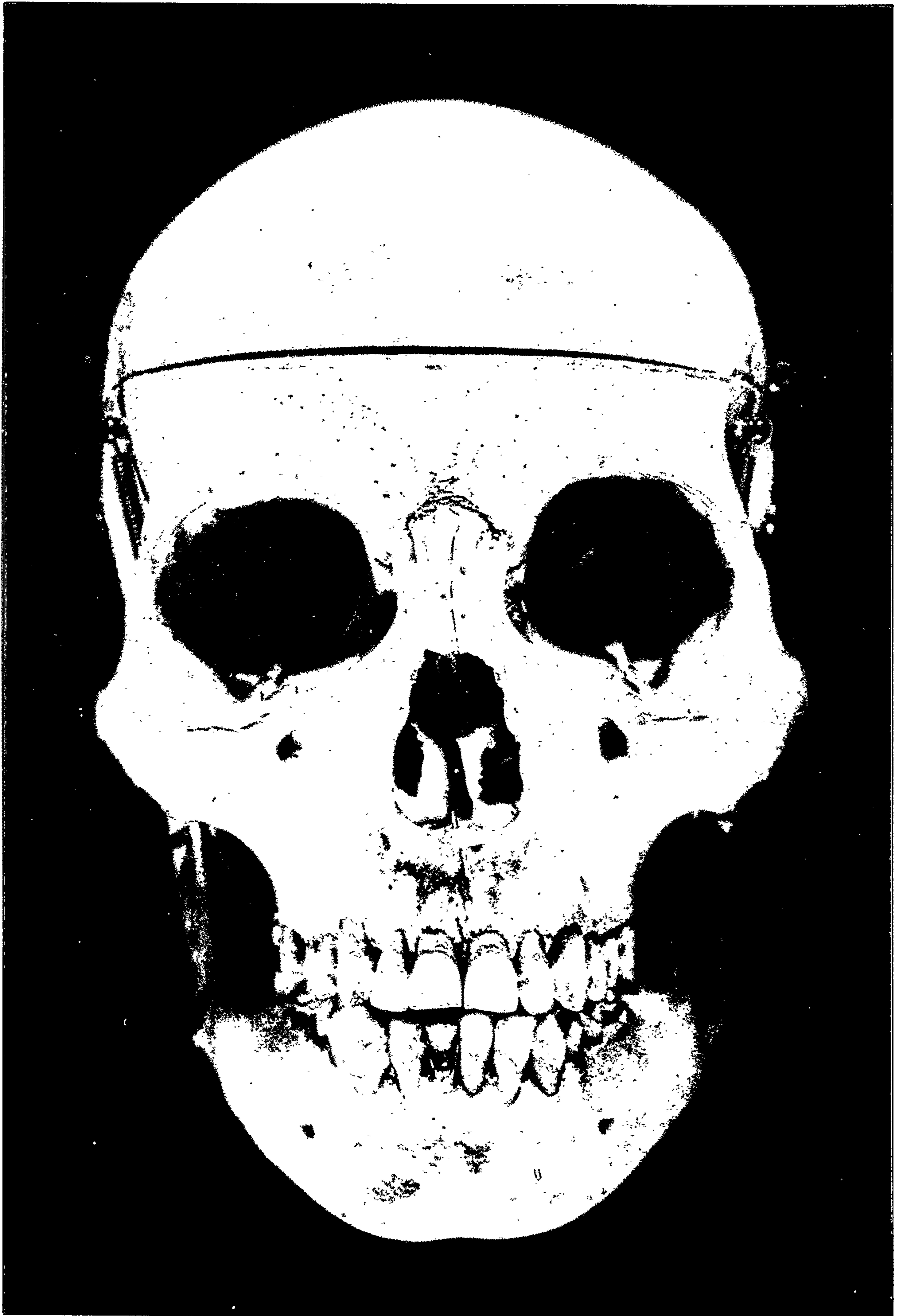


Fig. 53. Front of human skull showing bony nasal openings  
(Author's material)

SECTION III: AIM OF THE PRESENT INVESTIGATION

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It is apparent that a total and comprehensive verification of the hypotheses requires a large number of experimental and control subjects on a longitudinal basis. This is not feasible at present. From the research into literature, however, it can be concluded that:

- (1) As it stands, Computed Tomography offers the most accurate and non-invasive means to evaluate nasopharyngeal dimensions.
- (2) The use of mini Wright Peak Flow Meter is a simple, reliable and economical way to assess the degree of nasal patency.
- (3) There is still much controversy over the relationship of adenoids (size of nasopharyngeal space), nasal obstruction (degree of nasal patency), mode of breathing (mouth or nasal), and special characteristics of dentofacial morphology ('adenoid facies').

On a smaller scale, it is felt that the following tests can assert a direction whether the *actual* nasopharyngeal space, the *degree* of nasal patency, the *mode* of breathing, and *characteristics* of 'adenoid facies' are related:

- (1) To design a standardised and reproducible method in evaluating the actual volume of nasopharyngeal space with the aid of Computed Tomography (scan-based volume determination).
- (2) To establish a reliable cephalometric quantity (AA) representing scan-based volume determinations in normal subjects.
- (3) To test for correlation between AA and clinically assessed nasal patency using the mini Wright Peak Flow Meter (Blockage Index).
- (4) To relate AA and Blockage Index (BI) with the mode of breathing (MB) in selected children.
- (5) To test the extent of associations among AA, BI, MB, and major dentofacial characteristics connected with 'adenoid facies': narrow upper arch, retroclination of upper and lower incisors in relation to the base planes, a short lower arch, high palatal vault, large angles between the occlusal plane and the mandibular plane and the nasion-sella and palatal planes, small index value for face depth to face height, cross bite and distoclusion between upper and lower jaws.

Though data so obtained will be analysed for inference of causal theory, the primary aim of the present investigation is not to substantiate a cause-effect relationship. It is designed to disclose the strength of relationship among components of the 'Adenoid Facies Path', while viewing the concurrence of adenoids, decreased nasal patency, mouth breathing, and dentofacial abnormalities as coexisting and somehow associated with one another.

# **Section IV**

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## **Material and Methods**



Fig. 54. Base of skull (Author's material)

## SECTION IV: MATERIAL AND METHODS

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### 1. MATERIAL

The present investigation was conducted on two separate groups of subjects.

#### 1.1 FIRST GROUP

The first group consists of seventeen subjects selected at a private radiology clinic at Liverpool, Sydney, where the CT scanner is located. Computed tomograms and lateral radiographs were obtained during the period January 1980 to August 1980.

Out of this group, measurements made from two subjects (A and B) were used for a preliminary effort to design a standardised and reproducible method of employing the CT scanner to evaluate actual nasopharyngeal space (i.e., scan-based volume determination). The remaining fifteen provided measurements to correlate scan-based nasopharyngeal volumetric determinations with various selected acclaimed nasopharyngeal quantifications on lateral skull radiographs.

Selection criteria for this group are:

- (1) They were patients normally present at the clinic for multiple CT scans of the head.
- (2) No relevant nasopharyngeal pathology suspected or detected.
- (3) Of sufficient cooperation so that no undue discomfort was experienced by the patients or inconvenience to the operator.

Details of the seventeen subjects are given in Table 1. The same number designated to each subject was used in subsequent correlation analyses. Patients A and B were introduced only for the preliminary task of devising a standardised method to be used later, and so not included in the analyses.

Subject no.	Abbreviation	D.O.B.*	Sex
A	FS	24/12/34	F
B	AH	5/10/37	F
1	MMc	6/ 3/45	F
2	JP	15/11/42	F
3	JW	6/ 3/32	M
4	EF	15/ 4/42	F
5	AE	16/ 5/25	F
6	GMu	4/12/60	M
7	JC	6/ 8/45	M
8	RO	8/ 2/37	F
9	KW	1/ 1/14	F
10	MA	3/ 2/33	F
11	MS	27/12/36	F
12	FB	31/ 8/33	M
13	AMc	16/11/27	M
14	HL	15/ 4/55	F
15	GMa	24/ 5/14	M

\*Date of Birth

**Table 1 Conversion list of subjects chosen for First Group**

## 1.2 SECOND GROUP

The second group consists of 30 patients selected from the University Orthodontic Department of the United Dental Hospital of Sydney. They were children of Orthodontic age referred to the Department of Preventive Dentistry, University of Sydney, for specialist treatment.

Out of this group, 5 were diagnosed mouth breathers, 10 were nasal breathers, and 5 were oro-nasal breathers. All of them satisfied the following criteria:

- (1) No adenoidectomy was ever performed.
- (2) A lateral cephalometric radiograph and study casts were concurrently available.
- (3) No orthodontic treatment was administered at the time of recording.
- (4) Within the orthodontic group of 11 to 18 years of age, with a variety of malocclusions.
- (5) Airflow measurements were taken within 1 year of taking of the initial radiograph and study casts.

All patients were numbered according to Table 2. Again, the same numerical designations were used in the subsequent multiple regression analyses of data obtained from this group.

Since the aim of the present study is to expose the strength of relationship, if any, between nasopharyngeal capacity and other variables (upper airway patency, mode of breathing, and dento-facial characteristics), and not to specifically test the properties of diagnosable nasopharyngeal problems, a control groups was not warranted.

## 2. METHODS AND MEASUREMENTS

### 2.1 SCAN-BASED VOLUME DETERMINATIONS

Since no previous method of apply CT scanning to evaluate the volume of nasopharyngeal space is available, and that the orientation of

Subject	Abbreviation	Age (yrs.)	Sex
1	KT	13	F
2	SC	13	M
3	MSpa	17	F
4	BG	12	F
5	HW	14	F
6	MSpi	16	M
7	LR	11	F
8	GMc	14	M
9	HM	14	F
10	RM	14	M
11	JW	15	M
12	MY	12	F
13	LC	13	F
14	TS	14	F
15	BA	15	F
16	MT	15	F
17	MMc	16	M
18	CB	12	F
19	TH	12	F
20	JH	14	M
21	VK	16	F
22	JN	15	F
23	SM	14	F
24	SB	17	F
25	TD	14	F
26	RP	17	M
27	PW	15	F
28	AP	15	F
29	TP	14	F
30	RW	15	F

Table 2 Conversion list of subjects included in Second Group

subjects for this purpose could deviate from conventional techniques, a method was formulated to be used subsequently.

The method was contrived with the following objectives:

- (1) The exact height of the nasopharynx must be procured.
- (2) The least number of scans should be used to minimise radiation hazard.
- (3) The scans be made perpendicular to the long axis of nasopharyngeal space, thus reducing the potential error arising from oblique placement of cavity walls in the volume calculation for each computed tomogram.
- (4) The minification factor for each tomogram must be readily available.

#### 2.1.1 Height determination

The aim was to use a single radiograph on which the actual nasopharyngeal height can be derived. The radiograph was taken using a combined lateral cervical spine and lateral skull technique, on both subject A and B (Fig. 55).

The x-ray machine used was a Philips Medio 50, with rotating anode tube, 1 mm focal spot, non-bucky method, operating on 20 mAs at 66 kvp, and has a focus-film distance (FFD) of 2 meters.

The following steps were taken:

- (1) Subject was placed erect with mid-sagittal plane of skull perpendicular to the x-ray machine, and the orbitomeatal line (i.e., radiographic base line) parallel to the floor.
- (2) It was ensured that the whole of the pharynx was included (as in lateral cervical spine positioning), and a long FFD of 2 meters (as in lateral skull method to avoid gross magnification) was employed.
- (3) The distance between the mid-sagittal plane of the skull and the film was measured (SFD) by a plastic ruler prior to exposure (Fig. 56).
- (4) At the time of exposure, the subject was instructed to keep dentition at occlusion, with absence of deglutition or phonation, and respiration suspended during nasal inspiration.



Fig. 55. Radiograph of subject AE (no. 5), using the combined lateral skull and lateral cervical spine technique

From the resultant radiograph, the height of the nasopharyngeal space (i.e., distance between the most superior soft tissue point of nasopharyngeal airway and the tip of the soft palate) could be accurately measured, and converted to actual height using the formula:

$$h_2 = F \times h_1 \quad ;$$

where  $h_2$  = actual nasopharyngeal height

$$F = \text{diminution factor} = \frac{200 - \text{SFD}}{200}$$

$h_1$  = radiographic nasopharyngeal height.

### 2.1.2 Tomographic volume determination

The CT scanner used was a DeltaScan 150 of Ohio-Nuclear Inc., with the following specifications designed for neurological scan:

Scan speed: 120 seconds

Slice per scan: 1

Slice thickness: 10 mm

Detectors: 3 BGO Scintillation Crystal/Photomultiplier, plus  
1 reference

Scan circle diameter: 25 cm

Gantry aperture: 30.5 cm

Scan technique: 120 kv, 25 mA

Reconstruction time: less than 30 seconds

Matrix: 256 x 256

Pixel size: 1 mm x 1 mm

Computer: digital PdP 11/04

Image storage: 25 image magnetic disc, 134 image magnetic tape

Photography: DeltaMat Multi-Format camera.

Since the subjects were initially requiring brain scan, a contrast medium of Urografin 30% 250 ml was administered intra-venously using dripping fusion.

Operation of the scanner was effected at a control panel in a room next to the x-ray machine so that the movable table, on which the subject lies, could be remotely controlled and the scanning process monitored (Fig. 57). Fig. 58 shows the computer in action and the cathode ray tube display.

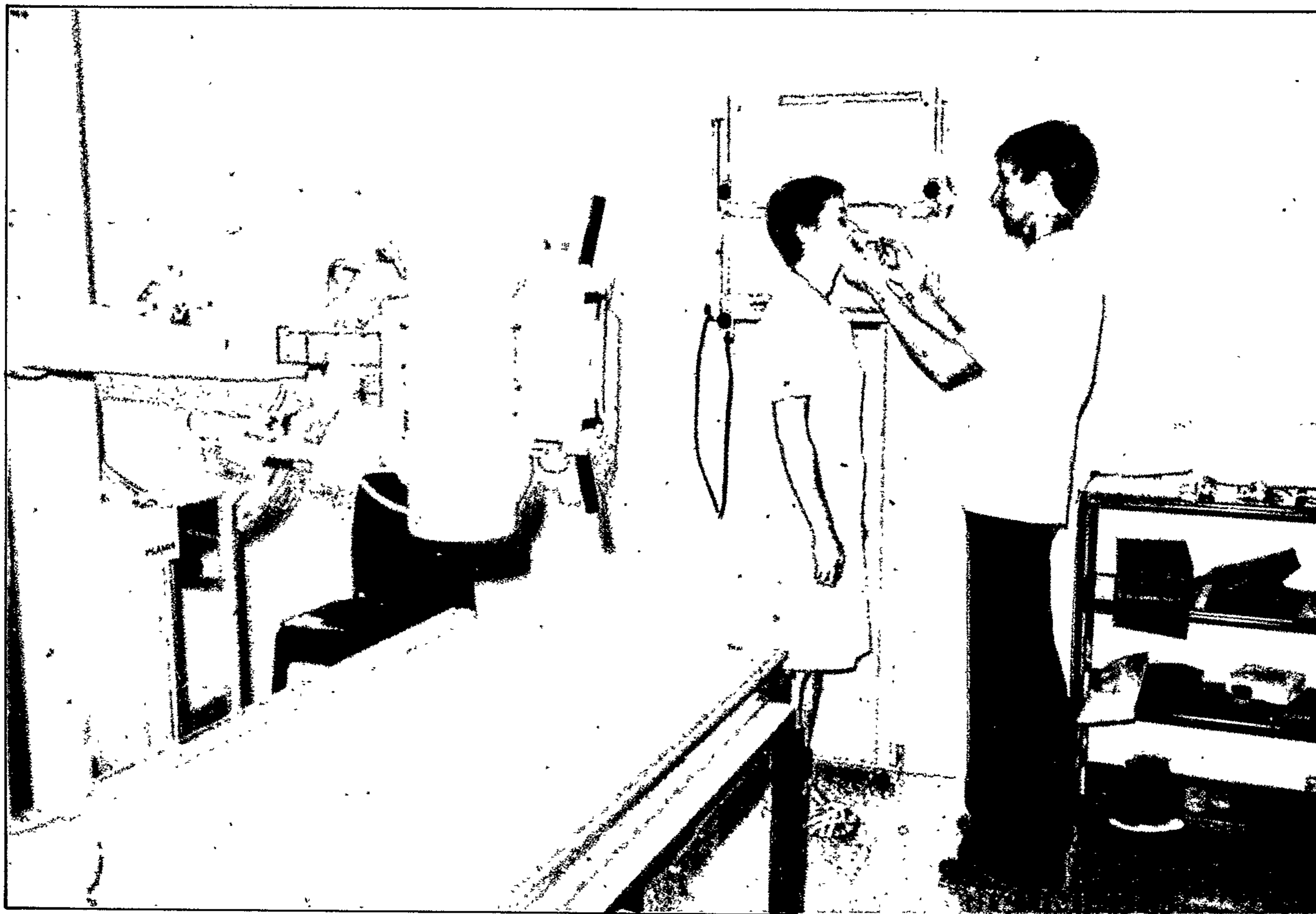


Fig. 56. Method of registering SFD

Fig. 57. Control room monitoring the CT scanner; note the Multi-format camera at the extreme left

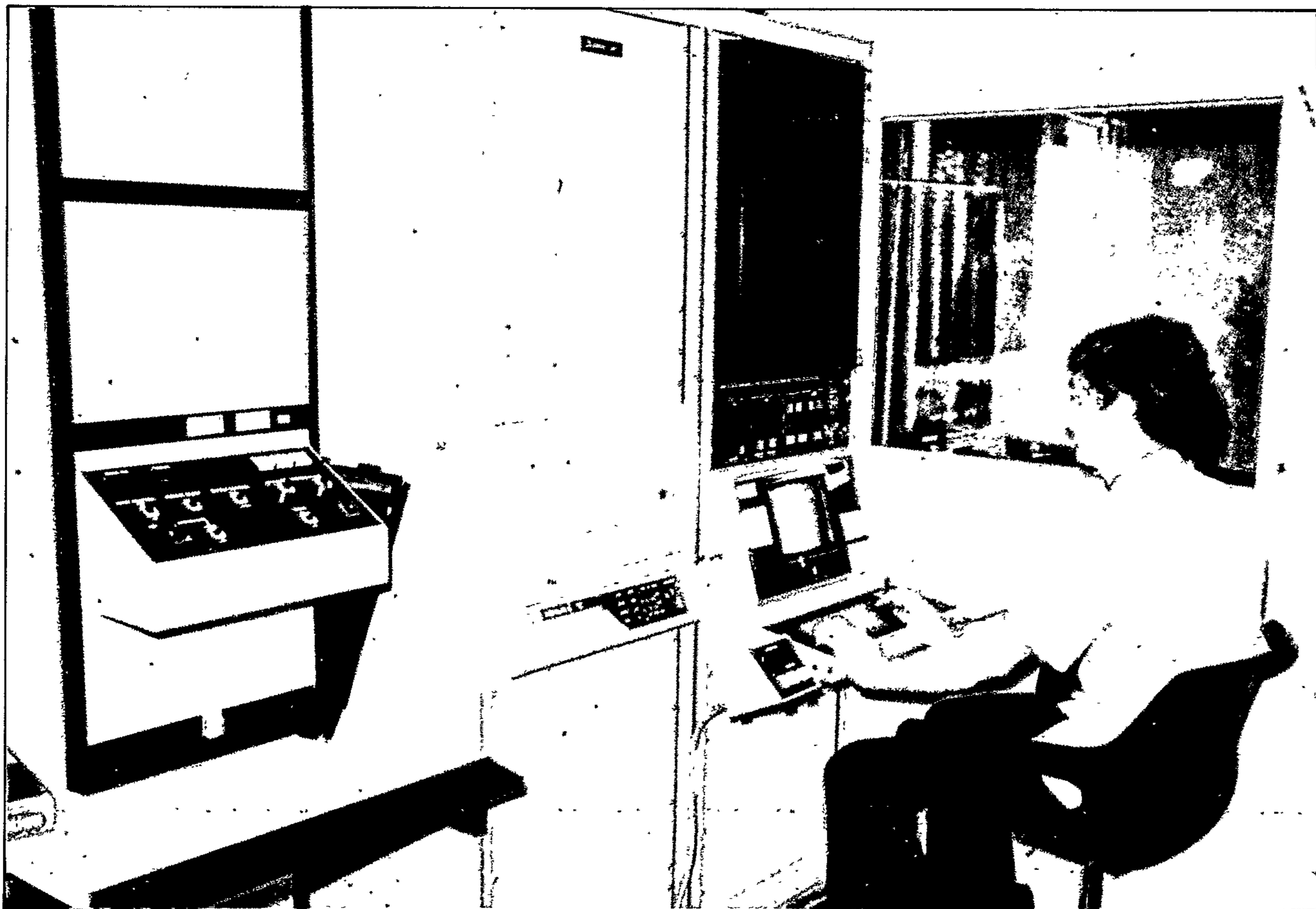




Fig. 58. Computer of scanner, control panel, and cathode ray tube display

The patient was placed face up and the head positioned to allow transverse scans where desirable (Fig. 59). To guide the orientation of the radiographic slice, a green light corresponding to the plane of collimated x-ray could be switched on inside the scanning tube (Fig. 60).

For subject A, three sequential CT scans (10 mm thick) beginning from the level of the orbitomeatal line (i.e., from corner of eye to tragus of ear) were taken. For patient B, the Frankfort plane (i.e., from lower orbital margin to porion) was used instead. These orientations were represented by lines A and B in Fig. 61 and 62, which were drawn on a dry skull and a life subject.

A grid marking 2 cm squares was placed at the same orientation in the scan field to give a minification factor, and is seen on resultant computed tomograms of subject B in Fig. 63a, 63b and 63c.

It was decided that the orientation of Frankfort plane will be chosen as a standard procedure because it gives a most transverse section of the nasopharyngeal airway, besides being reasonable stable (Wei 1968).

Fig. 63a shows the level of Frankfort plane where the serial scans proceeded inferiorly. Since a tomogram of this nature includes all the structures within the 10 mm slice, a section at this level discloses the superior border of nasopharyngeal space and part of sella turcica as well. It was therefore considered appropriate to begin scanning 5 mm below the Frankfort plane, thus excluding the soft tissue present below the cranial base.

Taking all factors into consideration, it was decided to limit the number of scans to three, and the method is diagrammatically represented in Fig. 64.

The total thickness of the three slices (i.e., A, B and C), being 3 cm, is likely to be less than the actual nasopharyngeal height ( $h_2$ ). The thickness of the last slice (D) can be derived by the formula:

$$D = h_2 - 3 \text{ cm}$$

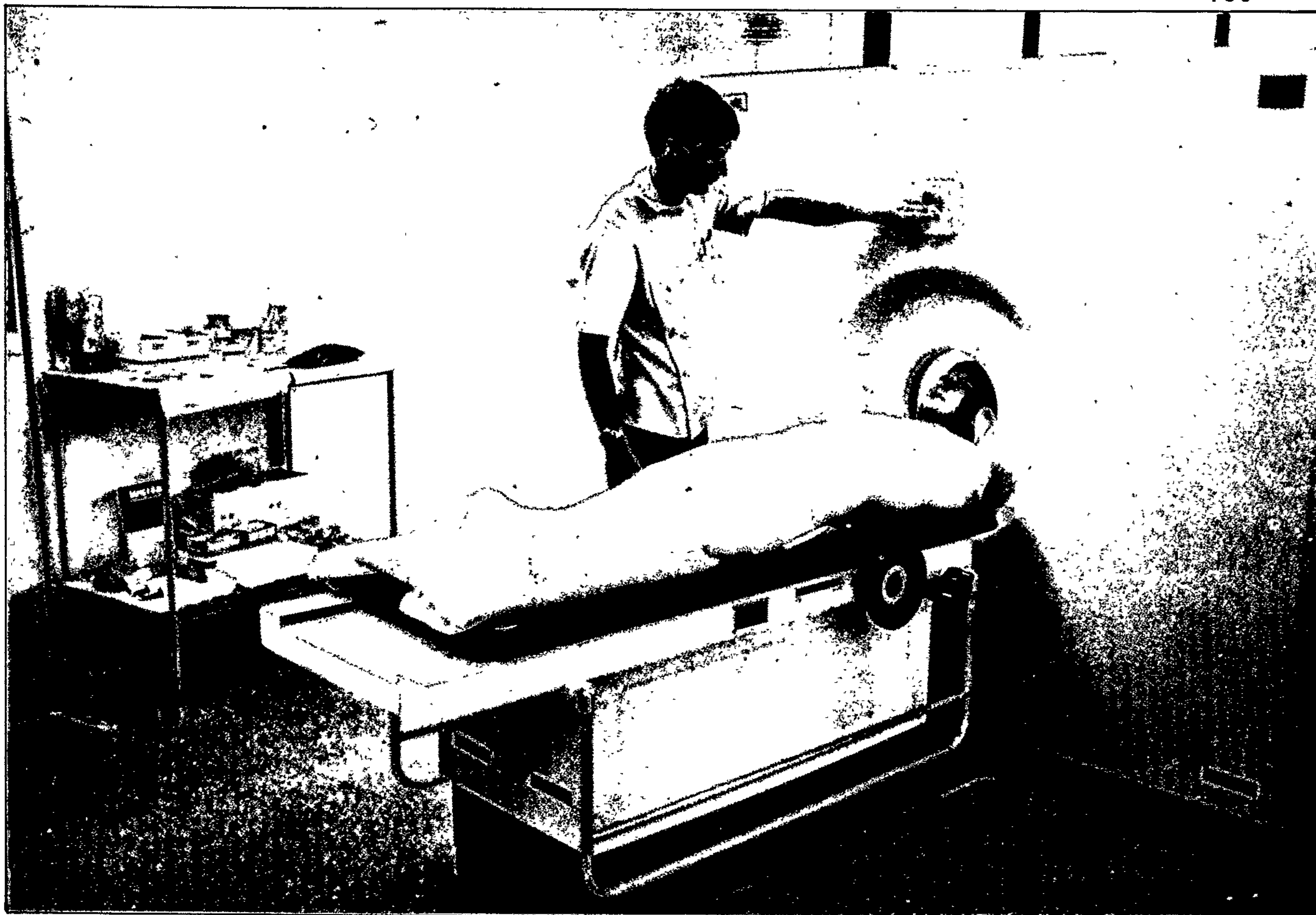


Fig. 59. Placing subject in position for head scan

Fig. 60. Green light guiding orientation of plane



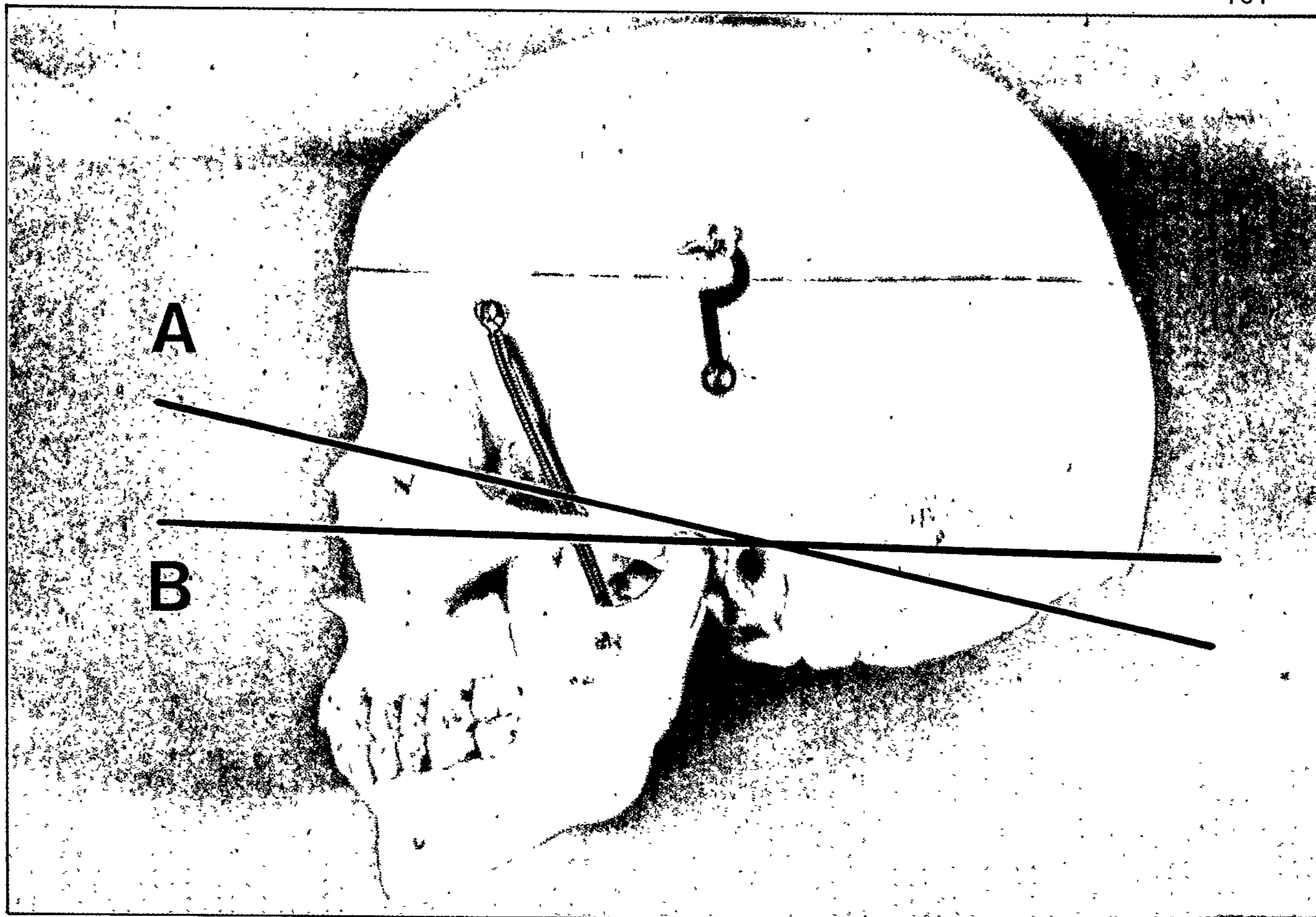


Fig. 61. Dry skull showing orbitomeatal plane (A) and Frankfort plane (B) (Author's material)

Fig. 62. Same orientations shown on a live subject



Fig. 63a.  
Subject B CT  
slice at level  
of Frankfort  
plane and 0  
degree to it

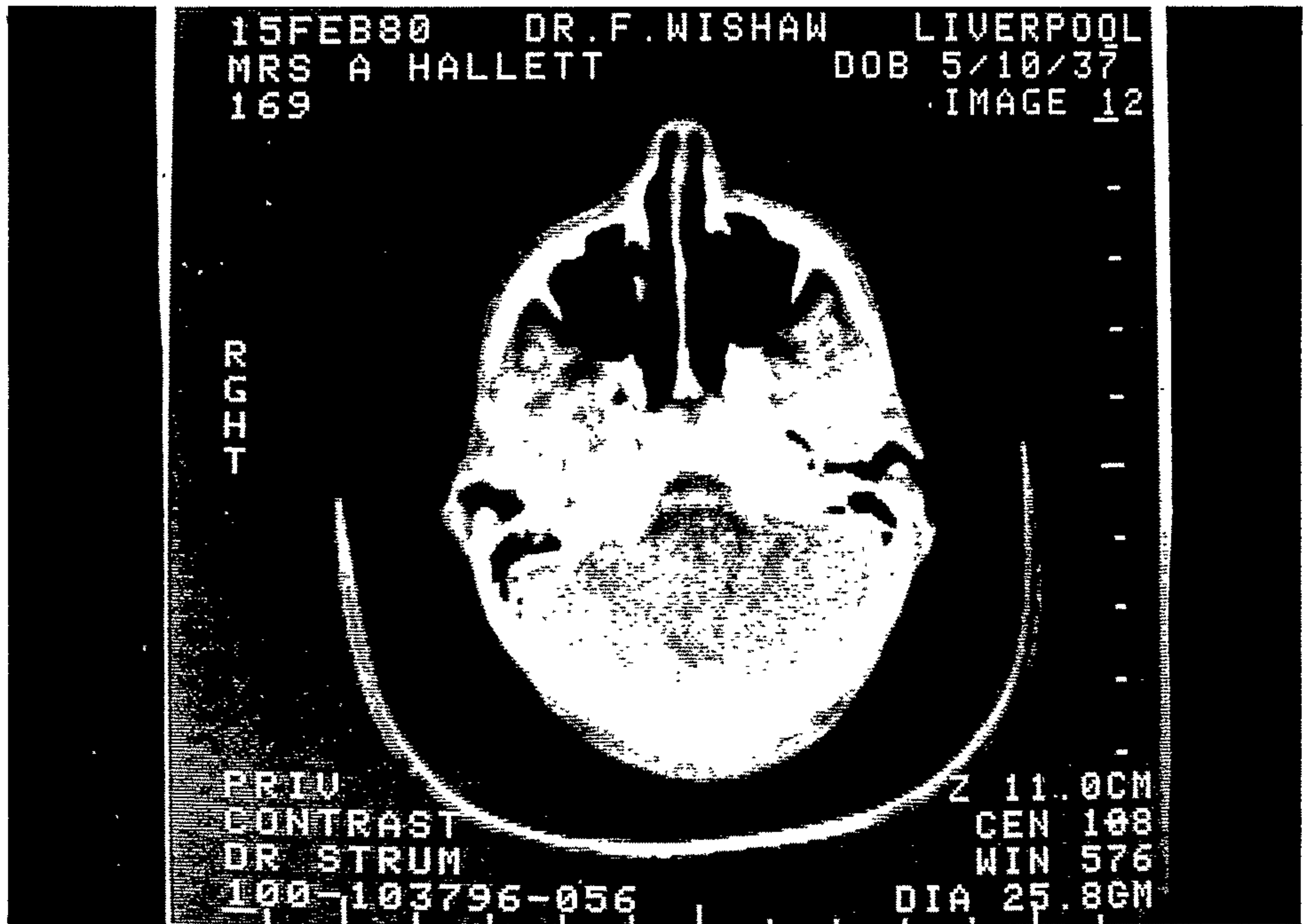


Fig. 63b.  
Same subject  
at 1 cm below  
above fig.

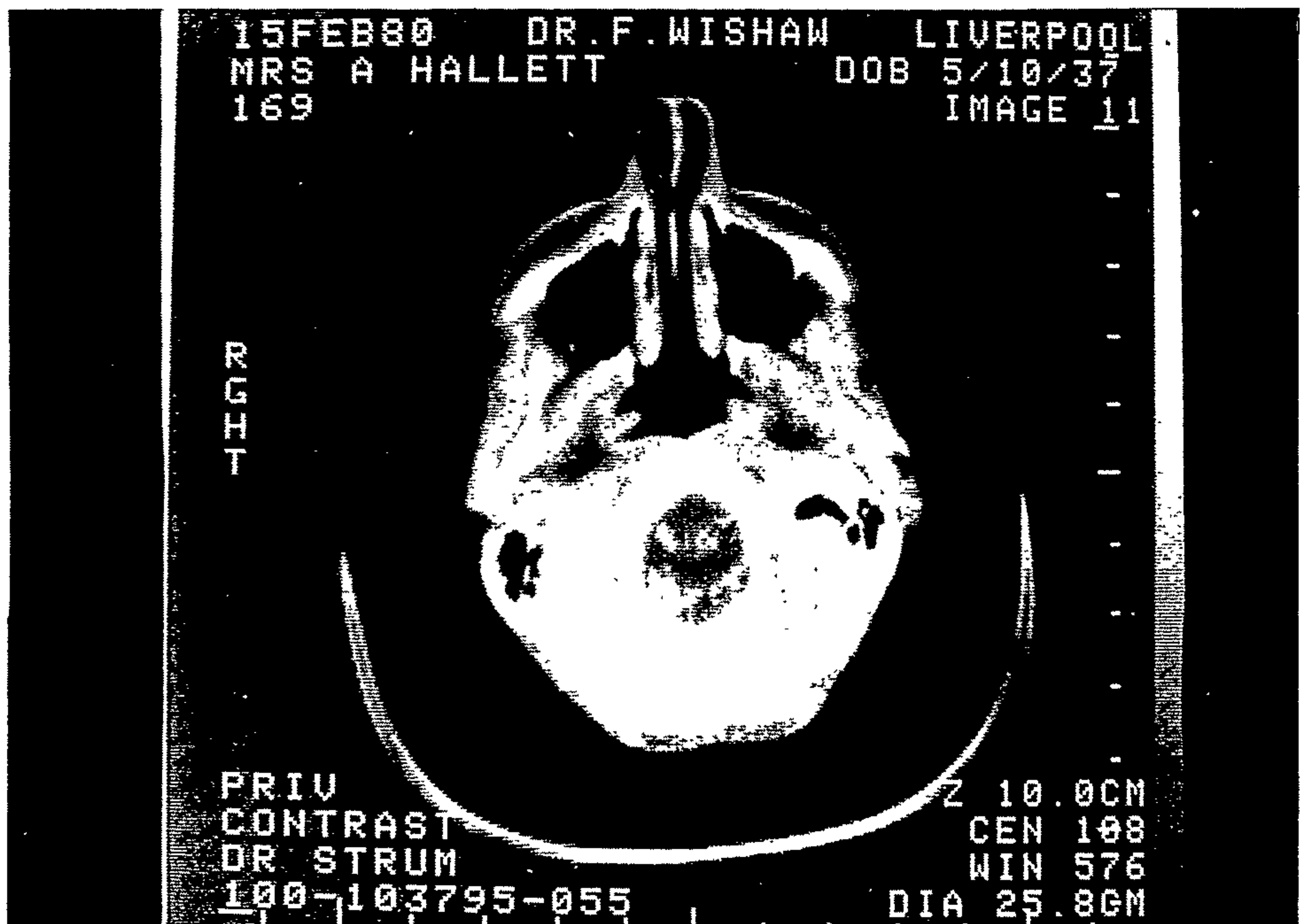
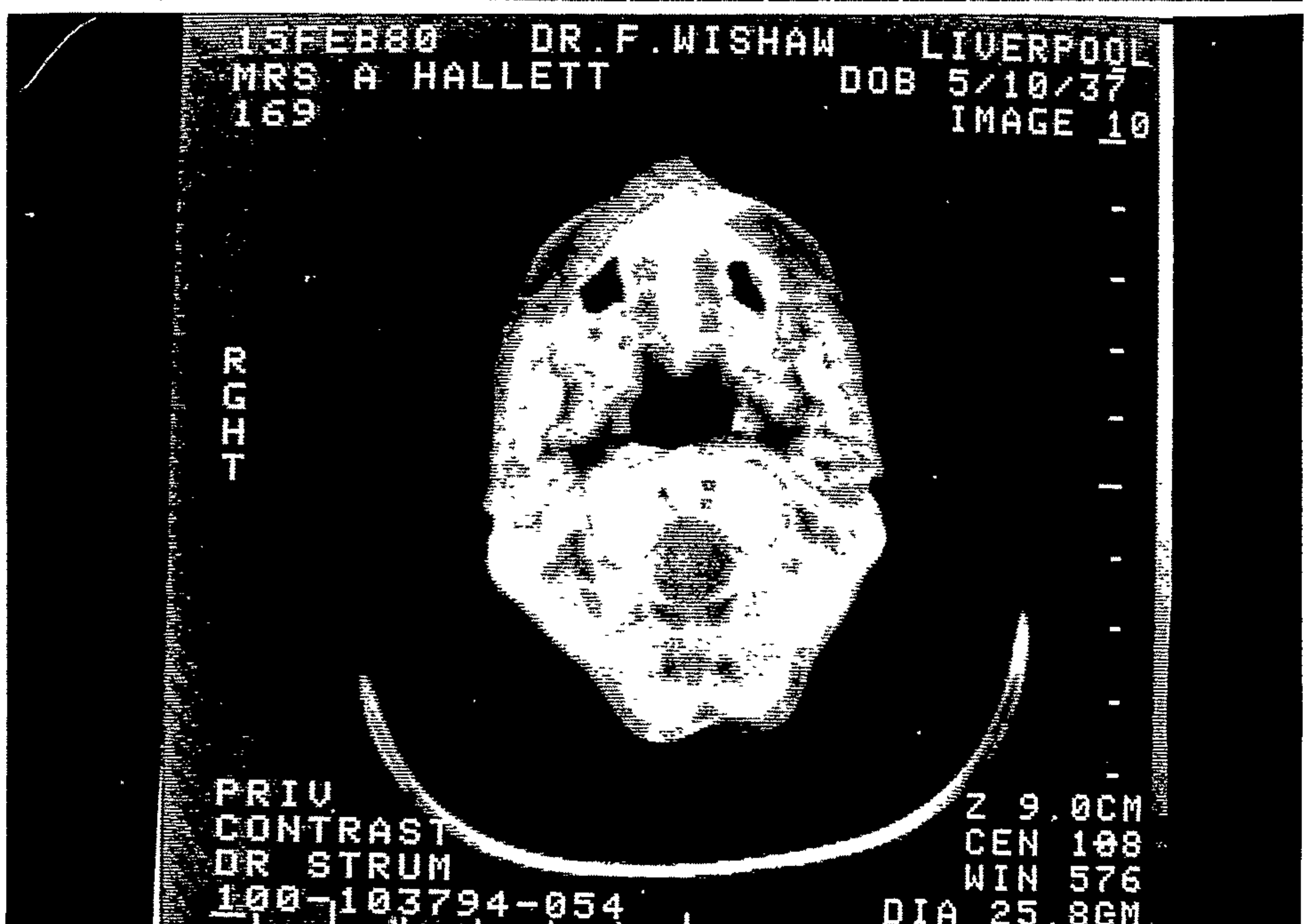


Fig. 63c.  
Same subject  
at 1 cm below  
above fig.,  
just under  
palatal plane



$h_2 = F \times h_1$ ; where

$h_2$  = actual nasopharyngeal height

$F$  = diminution factor =  $\frac{200 - \text{SFD}}{200}$

$h_1$  = radiographic nasopharyngeal height

SFD = mid-sagittal plane to film distance

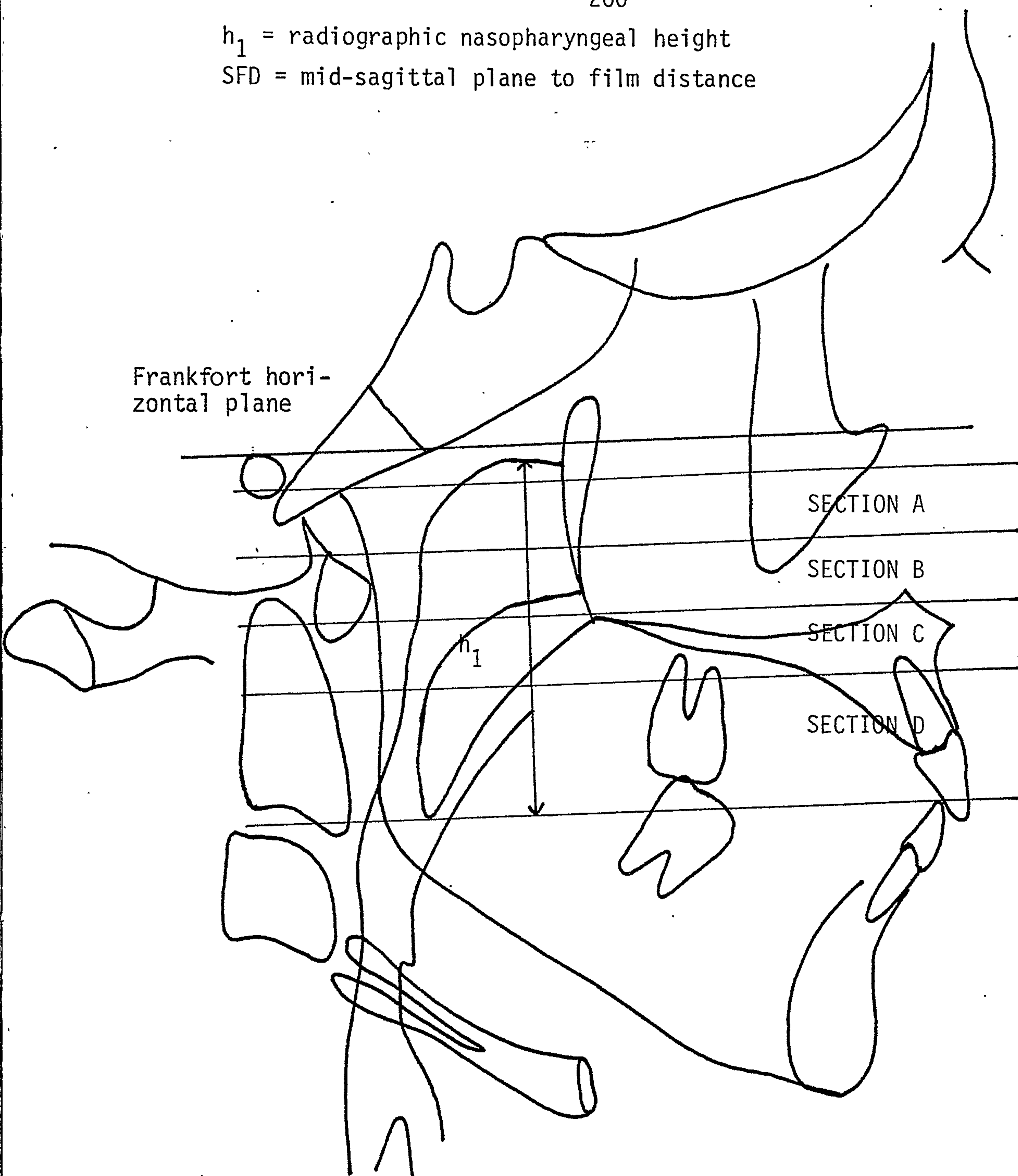


Diagram showing sections of serial scan in a mid-sagittal plane. Note that height of section D varies according to actual nasopharyngeal height.

Fig. 64.

The transverse area of slice D, furthermore, was observed to be about 0.7 times that of slice C. This was obtained by applying a further scan to subject 15 (Figs. 65a, 65b, 65c and 65d). The area of the nasopharyngeal space in slice D (i.e., Fig. 65d) was expressed as a percentage of area in slice C (i.e., Fig. 65c), and found to be close to 70%.

The area of each tomogram can thus be measured by planimetry, then corrected for magnification or minification registered on the grid shown in each tomogram, and multiplied by slice thickness to give a slice volume.

Total nasopharyngeal space is the simple addition of all slice volumes, as indicated by the moving cross-section method (Tierney 1975). Details of measurement using planimetry is discussed in the next section (2.2.1).

## 2.2 TO ESTABLISH A RELIABLE CEPHALOMETRIC QUANTITY

This was based on measurements obtained from subjects 1 to 15 of the first group of samples.

### 2.2.1 Scan-based volume determinations

By using the aforesaid technique, scan-based volumes of patients 1 to 15 were obtained. A typical set of tomograms and lateral skull radiographs are shown in Fig. 66, and Figs. 67a, 67b, 67c, taken on subject no. 6.

While the cross-sectional areas of the nasopharyngeal space on each tomogram could have been measured with the aid of the computer, it was decided to trace each airway image on Kodak acetate tracing paper on a view box with a sharp pencil. To reduce instrumental and measurement error, tomograms were enlarged photographically and the minification factors adjusted accordingly.

The area of each tracing of the nasopharyngeal space was measured with a Koizumi model KP-3 polar planimeter with zero setting device

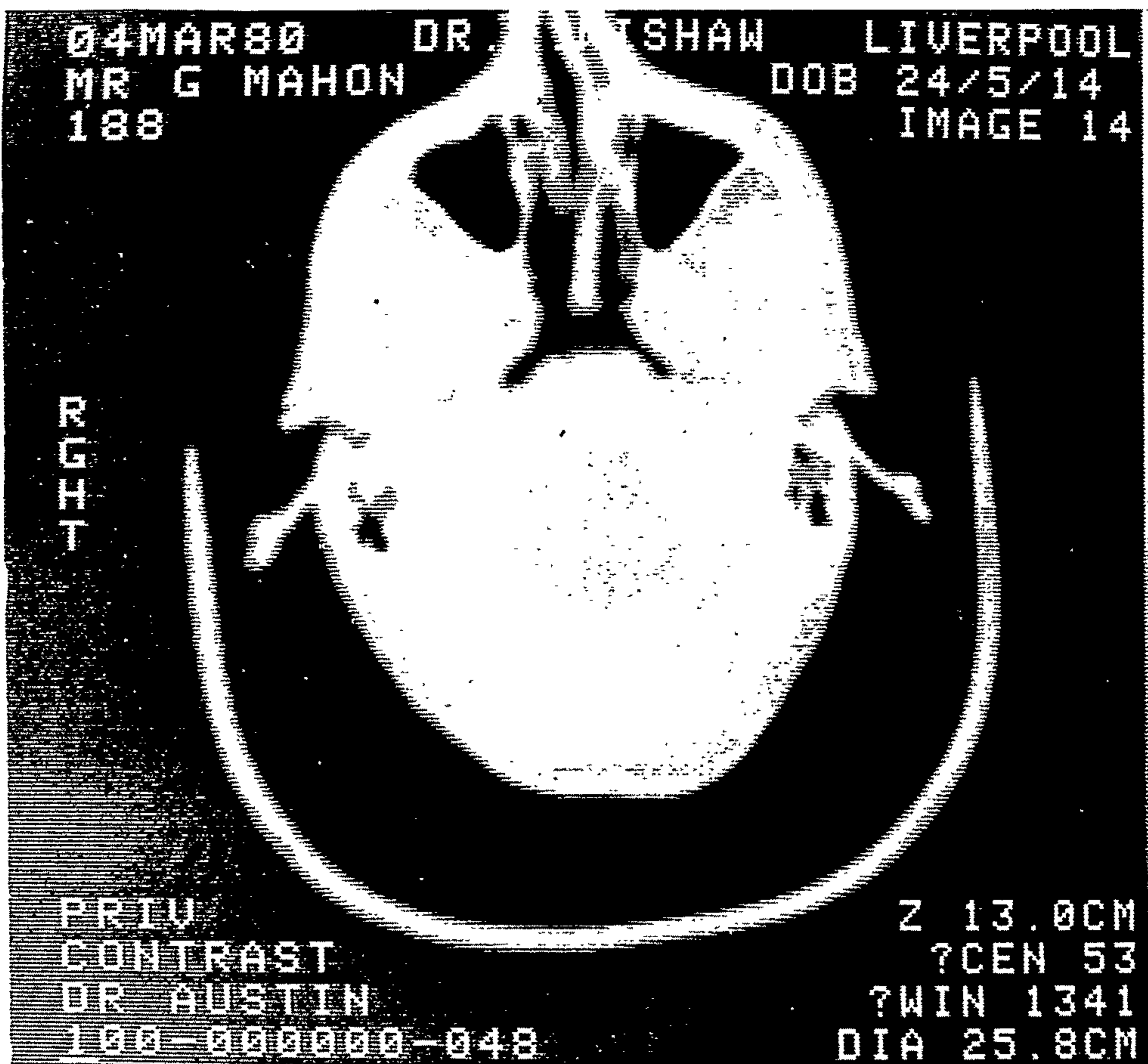


Fig. 65a. CT slice at 0.5 cm below Frankfort Plane level, corresponding to section A, subject 15

Fig. 65b. CT slice 1 cm below above figure, corresponding to section B, same subject



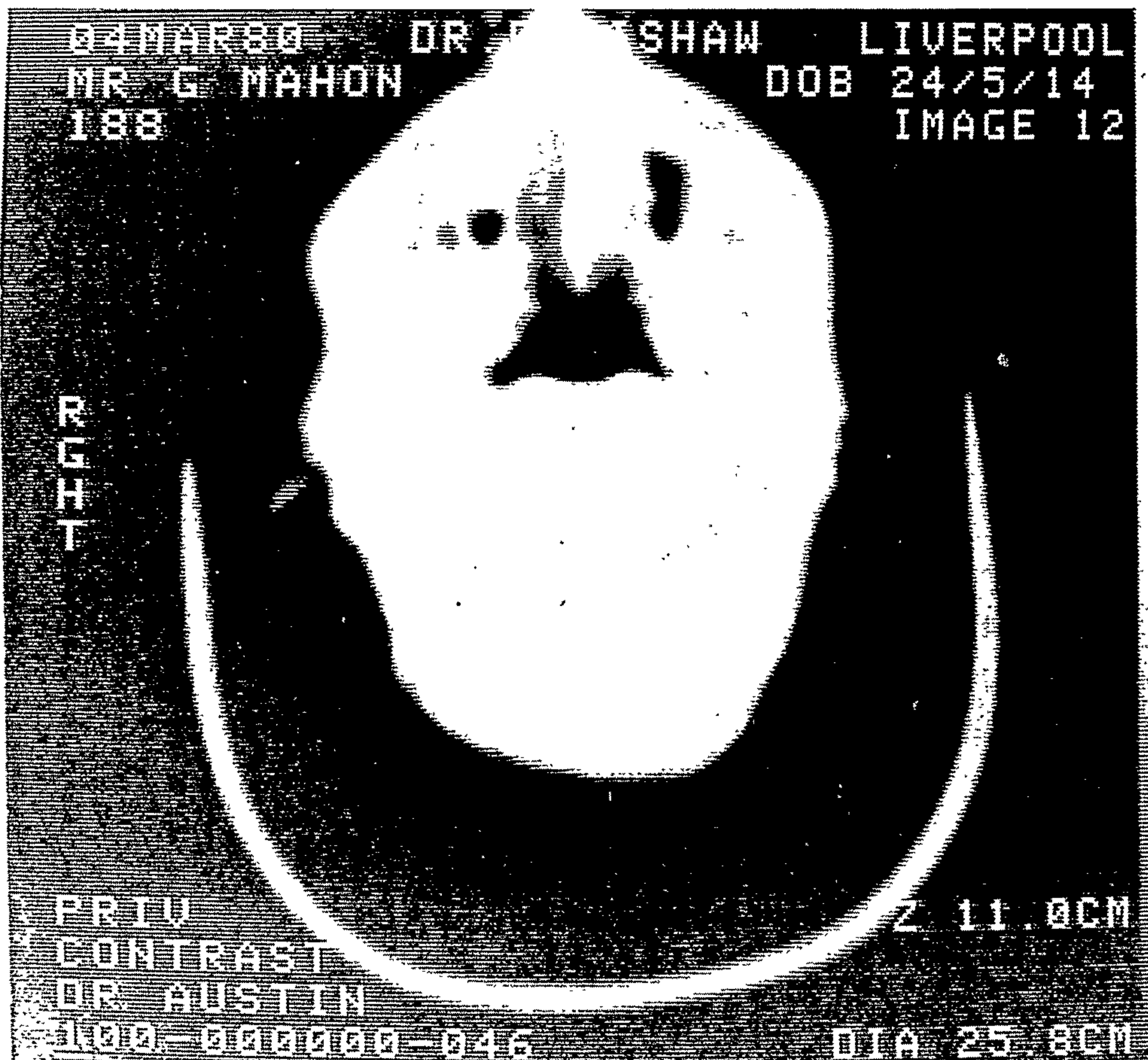


Fig. 65c. CT slice 1 cm below Fig. 65b, corresponding to section C

Fig. 65d. CT slice 1 cm below above figure, corresponding to section D





Fig. 66. A typical lateral radiograph used on the 15 subjects in scan-based nasopharyngeal volume determinations (subject no. 6)

Fig. 67a.  
Section A of  
a typical set  
of three serial  
CT slices  
(subject 6)

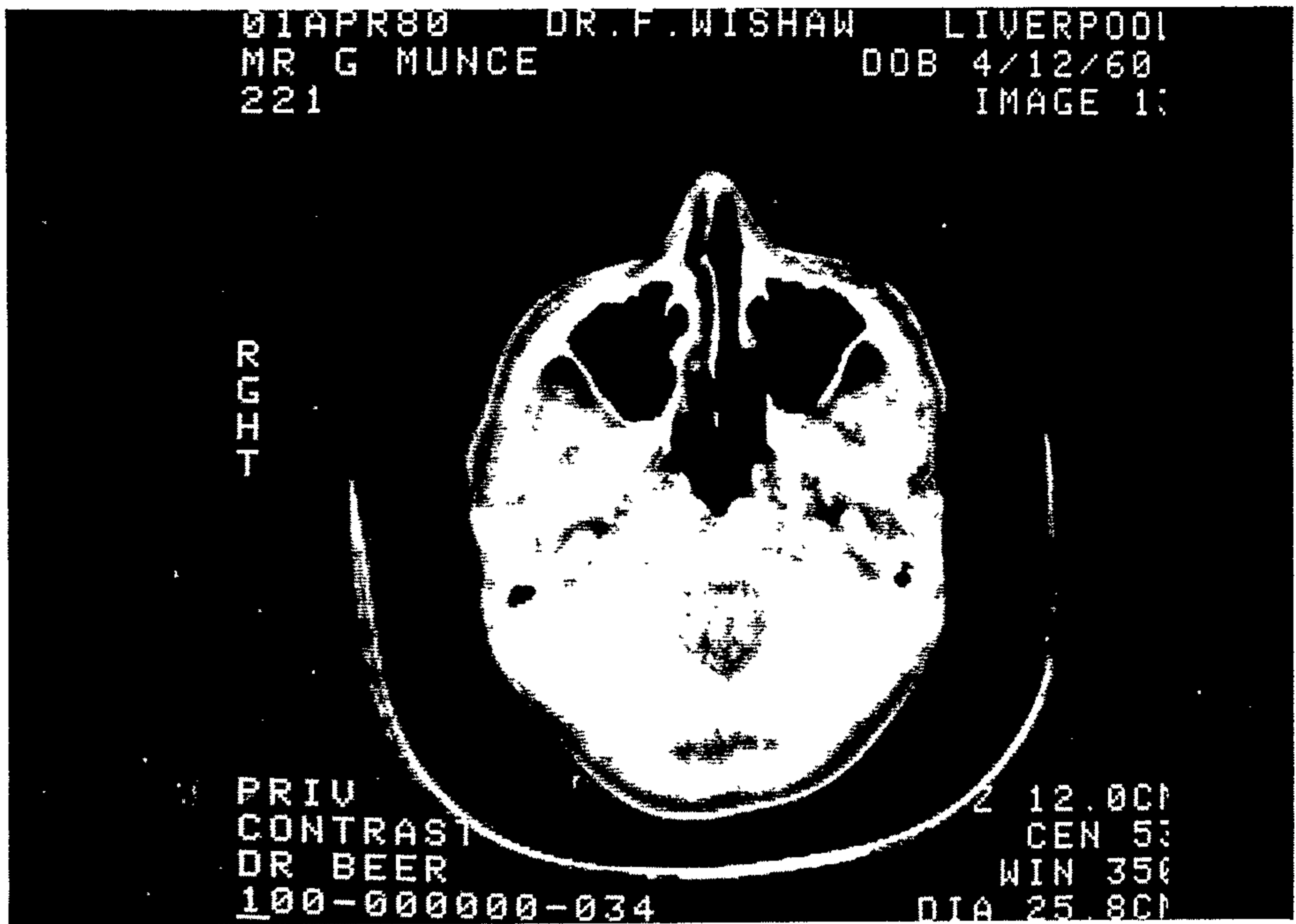


Fig. 67b.  
Section B of  
the same set

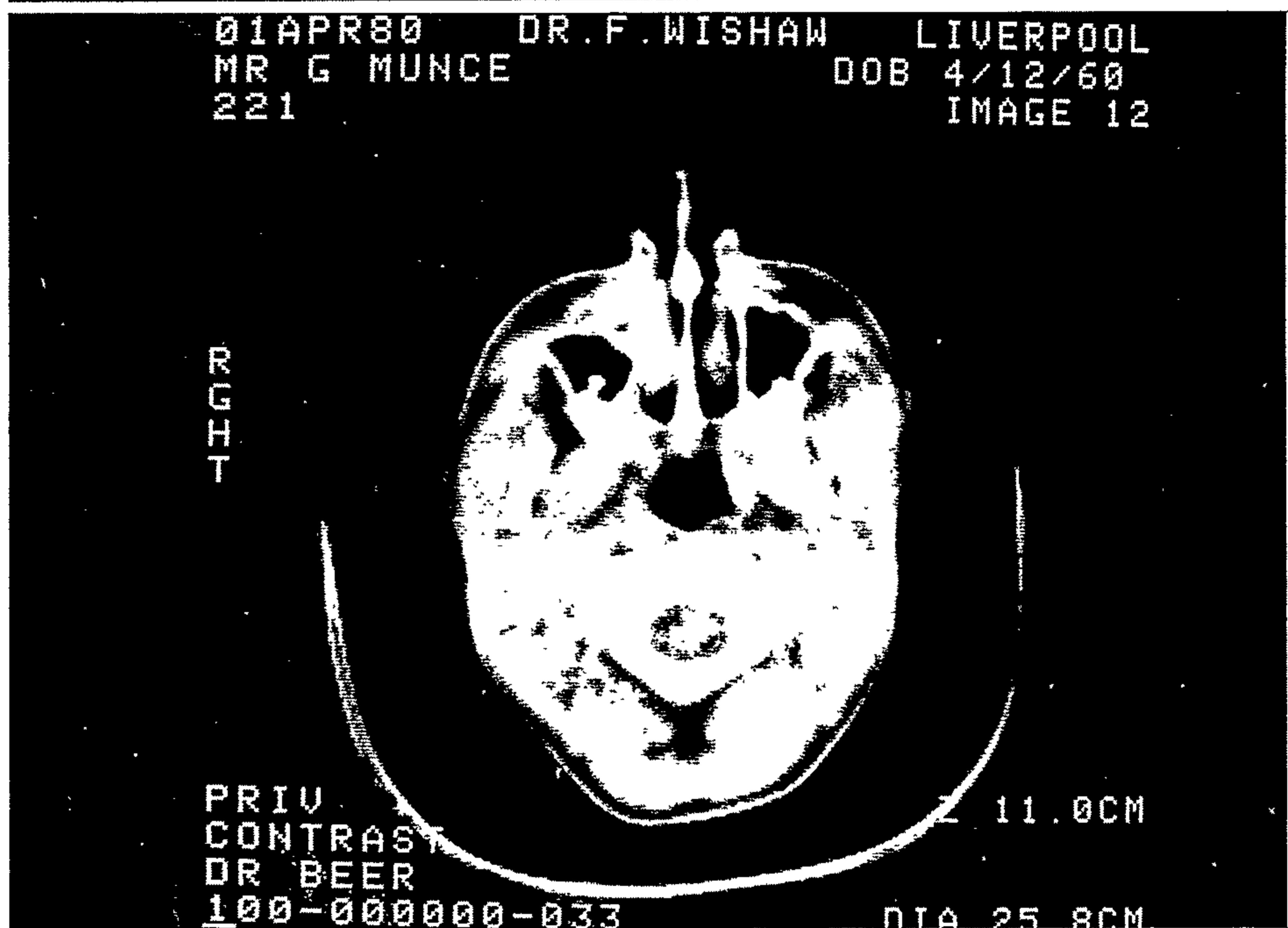
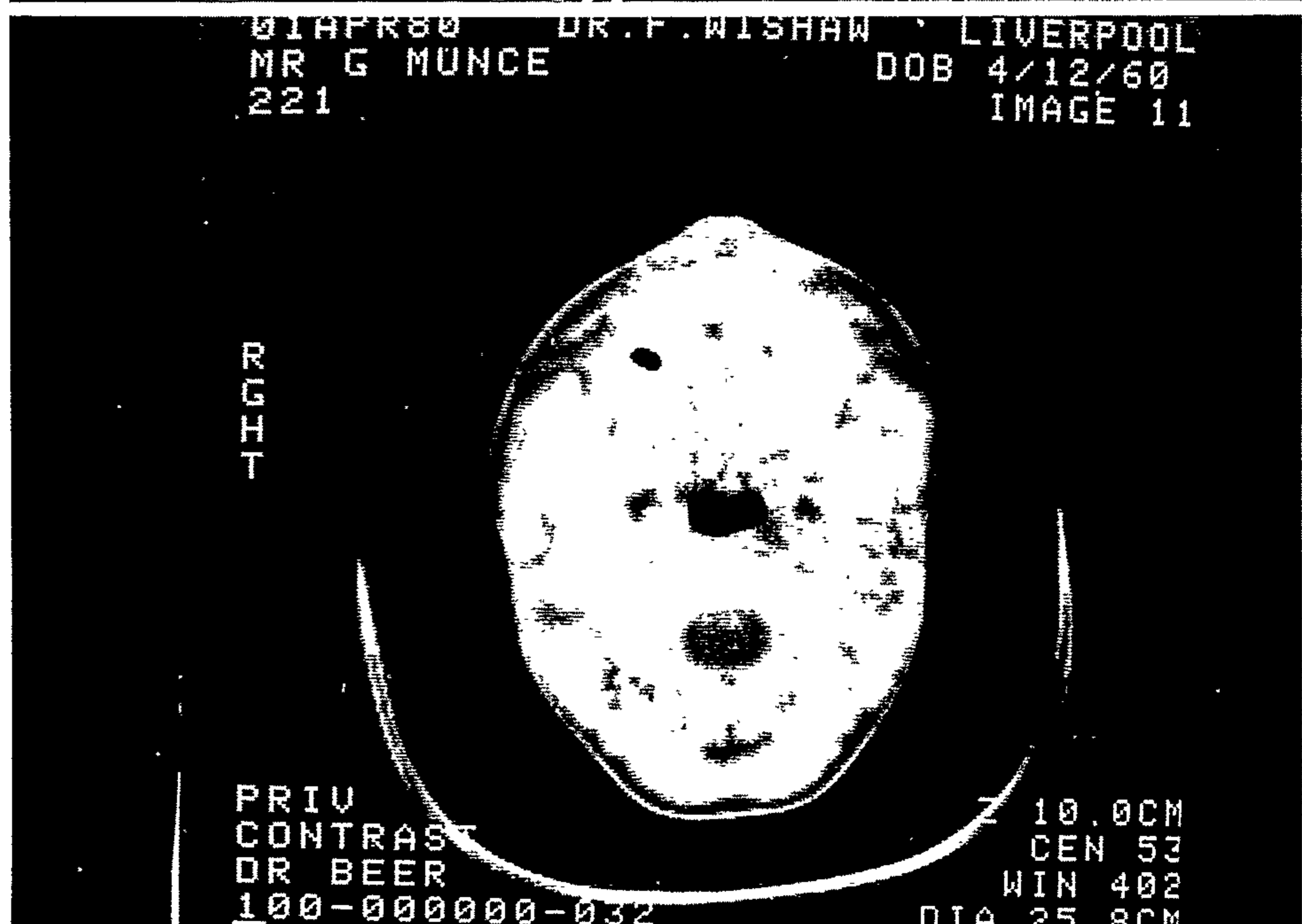


Fig. 67c.  
Section C of  
the same set



and optical tracer to the nearest  $0.1 \text{ cm}^2$  (Fig. 68). When the perimeter of a two-dimensional irregular structure is circumscribed with the hair-line dot on the optical tracer of this instrument, the area contained by the perimeter appears on the measuring wheel (Fig. 69).

Each airway tracing was at first measured three times and averaged. Typically, two of the three area readings were identical and the third  $0.1 \text{ cm}^2$  different. Eventually, only two measurements per airway were made if they were identical.

Volumes were calculated to the nearest  $0.1 \text{ cm}^3$  following the method described in the preceding section. Errors inherent in this technique will be examined in Section V2.2.

### 2.2.2 Lateral radiographic quantifications

Because of the vast number of nasopharyngeal measurements used in the past, it is undesirable to include all of them for testing. A total of 13 measurements devised by various researchers, plus 2 additional ones suggested by the author to represent nasopharyngeal space were tested. Out of these measurements, 10 were linear and 5 were areal. Since the FFD of each radiograph was registered, all measurements were conveniently converted to actual sizes based on the principle of similar triangles.

Designations of each quantity (X1 - X15) are tabulated in Table 3. Figs. 71a and 71b depict the corresponding tracings on a radiograph diagrammatically.

Criteria for selection are based on the following:

- (1) Only measurements devised to assess soft tissue nasopharynx were used.
- (2) Those in common use for a long time (X6, X10, X11).
- (3) Those proven statistically correlated to incidence of mouth breathing or nasopharyngeal problems (X1, X2, X5, X12, X14).
- (4) Those including the most extreme delineations of the nasopharyngeal space, i.e., depicting the point of greatest concavity or convexity (X7), or using the most superior soft tissue point (X3, X8).

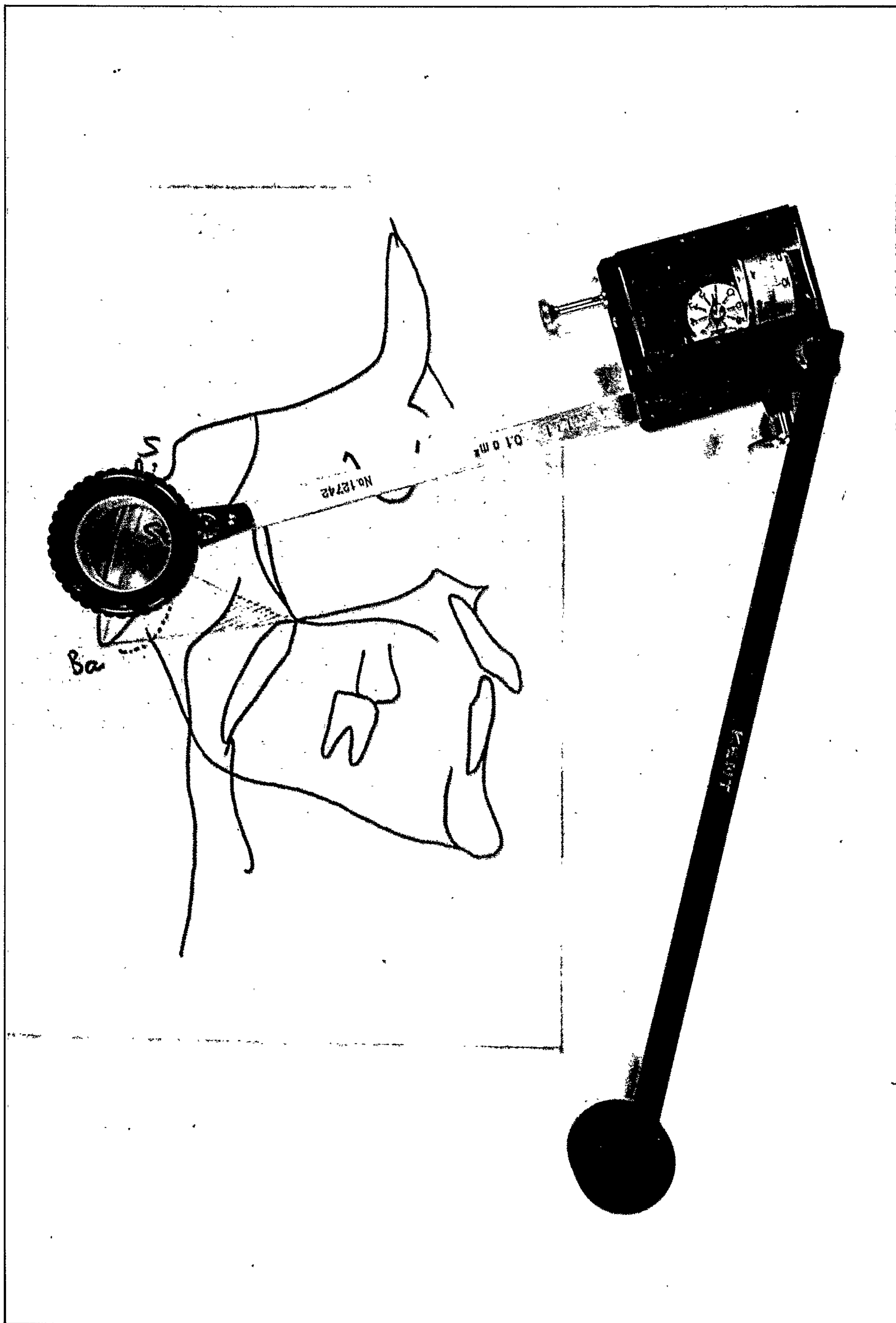


Fig. 68. A Koizumi model KP-3 polar planimeter with zero setting device and optical tracer

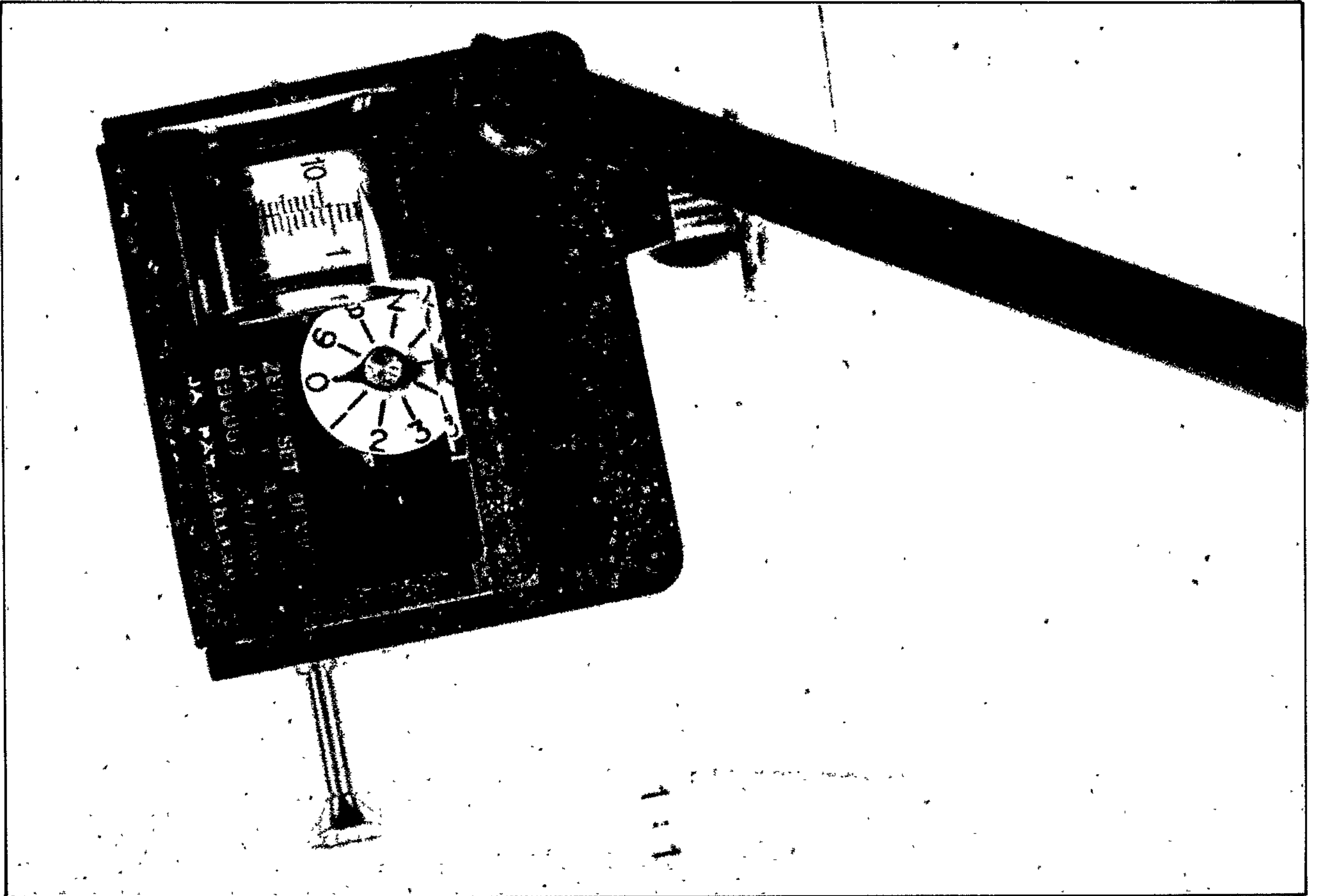
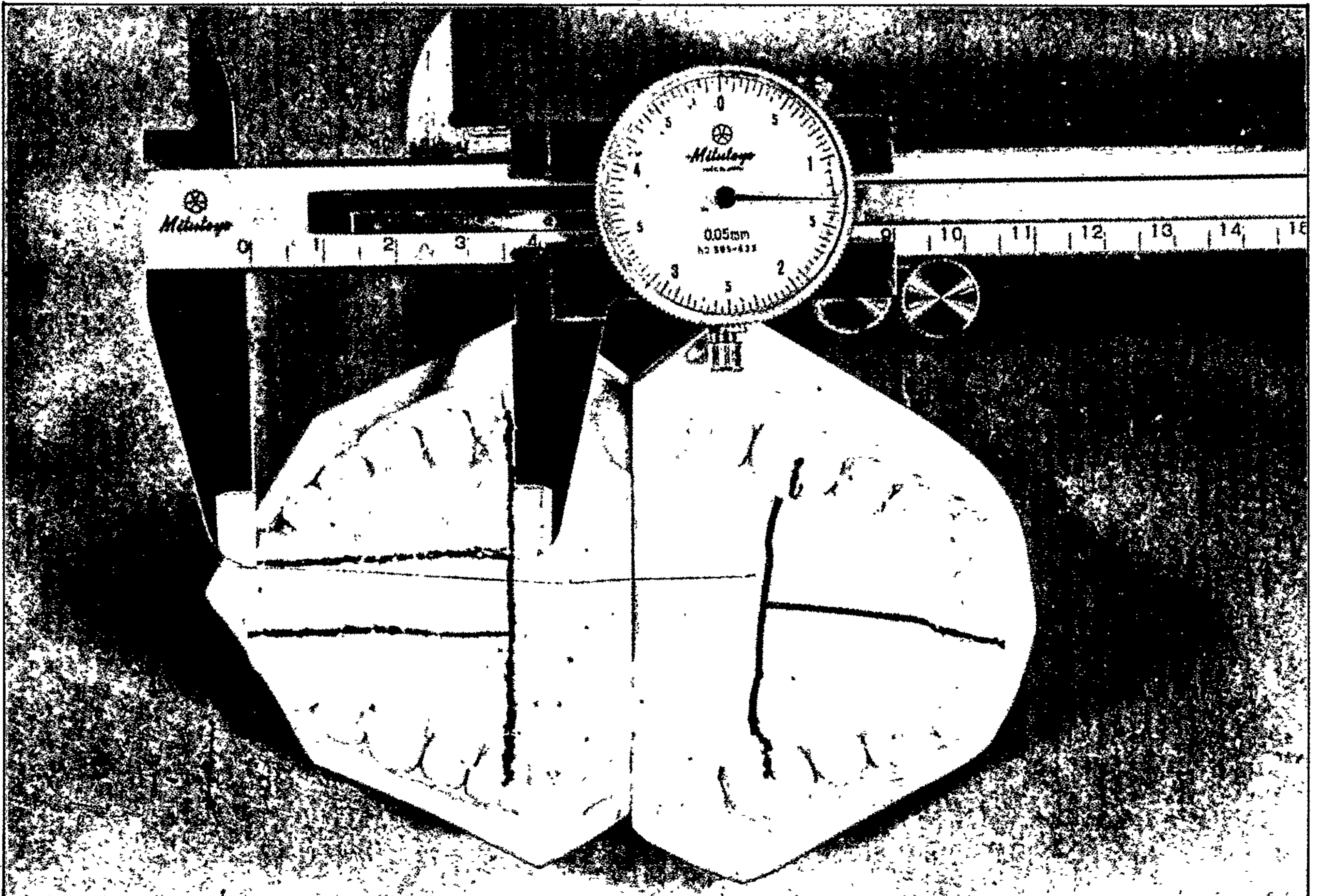


Fig. 69. Measuring wheel of the polar planimeter

Fig. 70. The sliding caliper used in the present study to measure study models



Variables	Definitions	Adapted from
<i>Linear Measurements</i>		
X1	AD1-PMF, distance from pterygomaxillare to nearest adenoid surface along the line PMF-Basion	Brader (1957); Linder-Aronson (1970)
X2	AD2-PMF, distance from PMF to nearest adenoid surface along line PMF-S <sub>0</sub> where S <sub>0</sub> = mid-point on line S-Ba	Linder-Aronson (1973)
X3	PMF-most superior soft tissue point	Castelli, Ramirez and Nasjleti (1973)
X4	PMF-PH, distance from PMF to posterior pharyngeal wall along line PMF-anterior point of atlas (AA)	
X5	PTV-AD, distance from pterygomaxillary vertical to adenoid surface on a line 5 mm above PMF	Ricketts (1954)
X6	PNS-PH, distance from posterior nasal spine to posterior pharyngeal wall on the palatal plane	Schweiger (1966); Subtelny (1957); McCarthy (1979)
X7	PNS-point of greatest concavity or convexity	Mergen and Jacobs (1970)
X8	Perpendicular to palatal plane to most superior soft tissue point	Mazaheri et al (1977)
X9	PNS-PH, distance from PNS to PH on line connecting PNS-anterior socket of central incisor	Maran et al (1971)
X10	Shortest distance between soft palate and posterior pharyngeal wall	Wolfe (1942)
<i>Areal Measurements</i>		
X11	Area of air space within triangle PMF-Sella-Ba	Brader (1957)
X12	Area of air space within triangle PMF-S <sub>0</sub> -Ba	Linder-Aronson and Henrikson (1973)
X13	Area of air space within triangle PNS-S, posterior nasopharyngeal wall, palatal plane	Mergen and Jacobs (1970)
X14	Area of air space within the trapezoid bound by sphenoid plane, palatal plane, pterygomaxillary vertical, and atlas vertical (both verticals being perpendicular to palatal plane)	Handelman and Osborne (1976)
X15	Area of air space bound by the total soft tissue boundary of nasopharyngeal space	

**Table 3** Designations of radiographic nasopharyngeal quantifications

- X1: AD1-PMF (on line PMF-Ba)  
 X2: AD2-PMF (on line PMF-S<sub>0</sub>)  
 X3: PMF-most superior tissue point  
 X4: PMF-PH (on line PMF-AA)  
 X10: shortest distance between soft  
 palate and posterior pharyngeal  
 wall

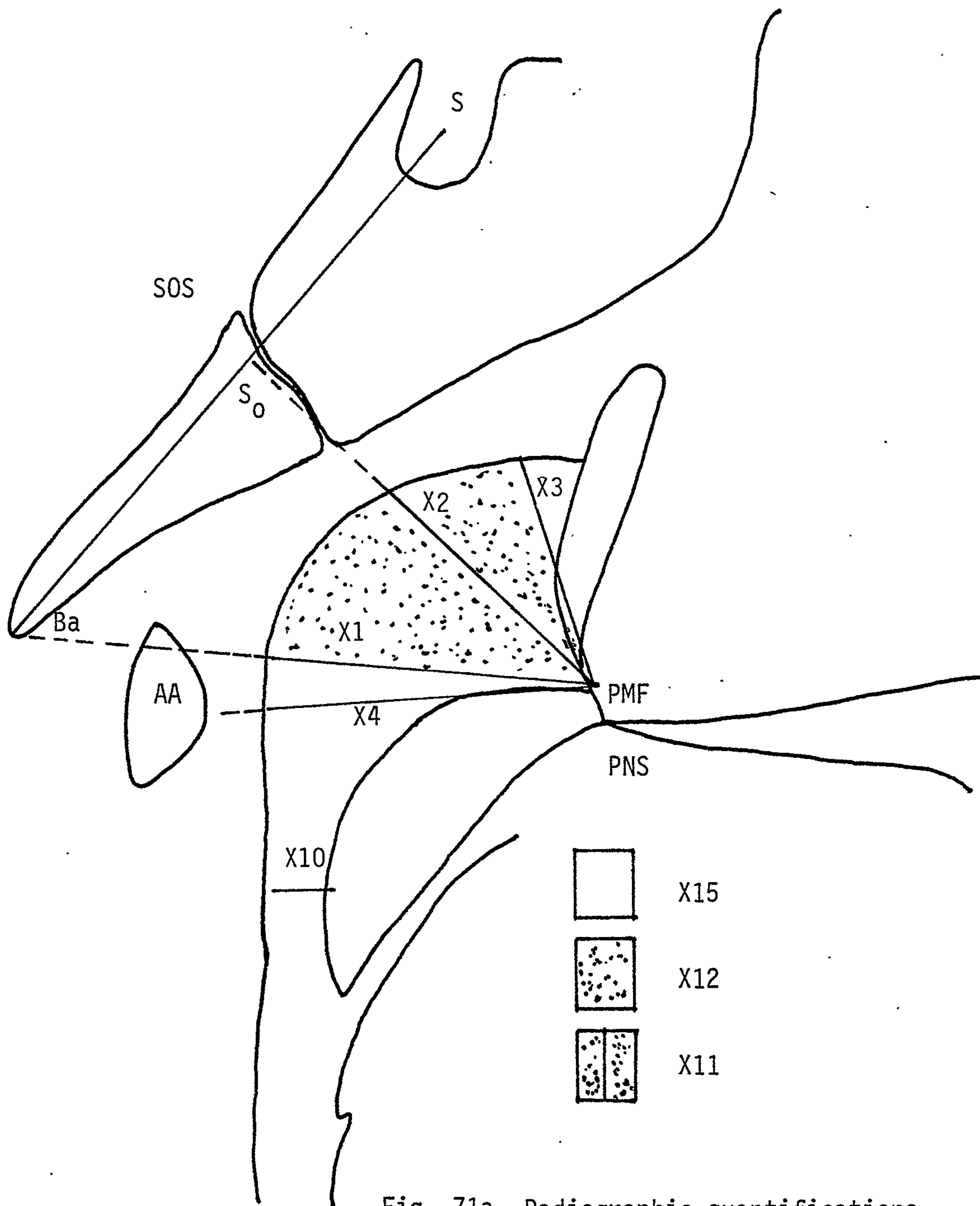


Fig. 71a. Radiographic quantifications  
 of nasopharyngeal space

- X5 : PTV-AD (on line 5mm above PMF)  
 X6 : PNS-PH (on palatal plane)  
 X7 : PNS- point of greatest concavity or convexity  
 X8 : perpendicular to palatal plane to most superior tissue point  
 X9 : PNS-PH (on line PNS-anterior socket of central incisor)

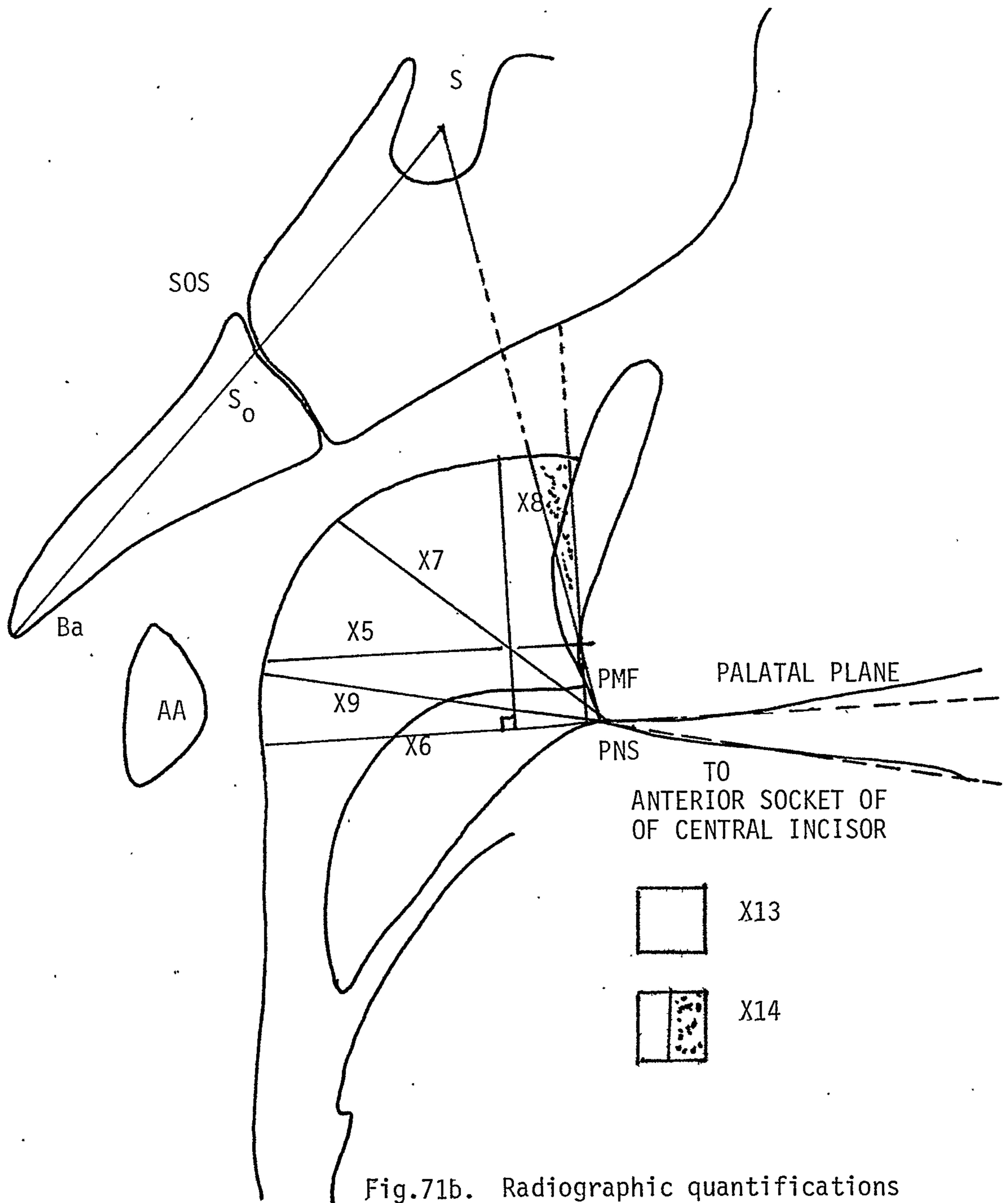


Fig.71b. Radiographic quantifications of nasopharyngeal space

- (5) Those claimed to pass through the bulk of adenoids if present (X5, X9).
- (6) That regarded as related to distoclusion of mandible (X13).
- (7) All of them should be readily reproducible and identified.

In addition, it was felt that the following two denotations, though not previously used, are justified:

- (1) X4: points AA and PMF, apart from being antero-posterior delineation of nasopharynx, are also displaced concurrently with growth of the region. AA (anterior point of atlas) is also functionally related to posterior pharyngeal wall.
- (2) X15: since most measurements were based on the portion of nasopharyngeal space above palatal plane, it was thought that an areal measurement following the outline of the total sagittal nasopharyngeal space would provide a more realistic assessment.

Again, tracings were made on Kodak acetate tracing papers. Linear measurements were recorded to the nearest 0.01 cm using a plastic ruler. Areal measurements made on the polar planimeter used previously to the nearest 0.1 cm<sup>2</sup>, though values to the nearest 0.01 cm<sup>2</sup> were obtained after conversion to actual sizes.

### 2.3 TO TEST THE EXTENT OF ASSOCIATION BETWEEN THE COMPONENTS OF "ADENOID FACIES PATH"

This was performed on the thirty patients in second group. Variables were chosen to represent each of the components in the 'Adenoid Facies Path' portrayed in Section I3, so that their relationships, if any, can be disclosed. To reiterate, these components include

- (1) Adenoids (nasopharyngeal space): scan-based volume determinations.
- (2) Nasal obstruction (upper airway patency): Blockage Index.
- (3) Mouth breathing: clinical assessment.
- (4) Dentofacial anomalies (Adenoid Facies): skeleton variables, and dentition variables.

Definitions of each of the variables are shown in Table 4. Apart

Variable Notation	Variables	Unit etc.	Definition	Obtained From
AA	<u>Scan-based volume determinations</u> Airway ceph. area	cm <sup>2</sup>		*R
BI	<u>Airflow variable</u> $\frac{PRFR_0 - PRFR_n}{PRFR_0} \times 100$	%	Blockage Index (Nasal patency)	Flow meter
MB	<u>Clinical Status</u> Mode of Breathing	Nose: 10 Oral: 01 Oro-nasal: 00		C
S1 S2 S3 S4 S5 S6	<u>Skeleton variables</u> angle SNA angle SNB angle ANB MP : SN MP : PP SN/NGn x 100	deg. } deg. } deg. } deg. } deg. } %	Measurement of antero-posterior jaw relationship  Steepness of mandible  Face Index ( $\frac{\text{face depth}}{\text{face ht.}}$ )	R R R R R R
S7	Ht. at M <sub>1</sub> - M <sub>1</sub>	mm	Height of palatal vault at upper 1st molars	SM
D1 D2 D3 D4 D5 D6 D7 D8	<u>Dentition variables</u> $\frac{1}{1}$ : SN $\frac{1}{1}$ : MP OP : SN OP : MP Arch width M <sub>1</sub> - M <sub>1</sub> , upper i : M <sub>1</sub> - M <sub>1</sub> , lower Cross-bite Angle's classification of molar relationship	deg. } deg. } deg. } deg. } mm mm no: 0 yes: 1 Class I: 10 Class II: 01 Class III: 00	Measurement of retroclination of incisors to base planes  Angle between occlusal plane and base planes  Maxillary intermolar width  Mandibular Arch length	R  R R SM SM SM SM

\* Notes: R = radiographs  
SM = study models  
C = clinical evaluation

Table 4 Definitions of variables used in study of 'Adenoid Facies Path'

from the one variable assigned to each of nasopharyngeal space, upper airway patency and mouth breathing (i.e., AA, BI, MB), there are 7 skeleton variables (S1 to S7) and 8 dentition variables (D1 to D8).

For each subject, the following steps were taken:

- (1) Complete clinical history taken, with special attention to the mode of breathing and previous adenoidectomies.
- (2) Plaster study models made from impressions taken at the time of examination.
- (3) A lateral cephalometric radiograph taken on the same date in the manner to be described.
- (4) Airflow measurements (Blockage Index) to be obtained within 12 months of date of first examination.

The lateral cephalometric radiographs were all taken in the Radiology Department of the United Dental Hospital of Sydney. They were taken from one machine as a regular feature for the orthodontic records of the patients.

The machine used was 'Selenix Alfa' of Siemens, West Germany, with the following specifications:

Mains: 40 mAs, 70 kv

Exposure time: 0.64 seconds

Focus Film Distance: 6 feet

Only radiographs showing dentition in occlusion and soft palate resting in a pendant position were measured, to ensure a standardised procedure throughout.

### 2.3.1 Nasopharyngeal variable

The variable AA represents the actual nasopharyngeal space volume, and is obtained by planimetric areal measurement on tracings of the entire nasopharyngeal space on the lateral cephalometric radiograph. This variable corresponds to notation X15 in the preceding test, and was found to be the most reliable cephalometric quantity representing scan-based volume determinations of nasopharyngeal space.

### 2.3.2 Airflow variable

This variable (Blockage Index, or BI) was obtained according to the method prescribed by Taylor (1973). This is an evaluation for upper airway or nasal patency, and was obtained from all of the thirty patients within 12 months of history taking.

A mini Wright Peak Flow Meter fitted with an interchangeable mouth and nose piece was used. Total expiratory effort through the mouth was determined using the standard mouth piece (Fig. 72). The best of six readings was regarded as the true oral peak expiratory flow rate ( $PEFR_o$ ). Similarly,  $PEFR_n$  through the nose was measured by substituting an airtight face mask (Fig. 73).

Instructions were given to the subjects to make a strong, quick blow. Typically, the highest readings were at the third or fourth attempts when the subject became used to the instrument. Subsequent exhaustion often rendered a lower reading at the fifth or sixth time.

Blockage Index (BI), which is independent of total expiratory effort but which correlates closely with nasal airways resistance was evaluated from the formula:

$$BI = \frac{PEFR_o - PEFR_n}{PEFR_o}$$

The instrument was produced by Airmed, Clement Clark International Ltd., England. The nose piece was a BP mask with a flexible rim allowing airtight engagement, and the connection between the nozzle and the Flow Meter was sealed with 'Bathtub Caulk Silicone rubber'. The mouth piece was a standard item together with the Flow Meter.

### 2.3.3 Mode of breathing variable

The mode of breathing was identified clinically. It was deduced by simply observing the subject to see whether the mouth was kept open or closed when in relaxed state. Mouth or nose breathing was then diagnosed by holding a cold dental mirror alternately in front of the nose and mouth. The subject was placed in a relaxed position



Fig. 72. Mini Wright Peak Flow Meter with mouth piece attached to register oral flow rate

Fig. 73. Mini Wright Peak Flow Meter with nose piece attached to register nasal flow rate



with the head upright. The ability to breathe through the nose was tested separately, by seeing whether the subject could breathe calmly for 30 seconds when one nostril was closed by pressure and the mouth shut.

Grading of nasal resistance thus observed was made as suggested by Linder-Aronson and Backstrom (1960) as:

- (1) Very low - test managed without difficulty.
- (2) Moderate - test managed with difficulty and with signs of dyspnoea.
- (3) High - test could not be managed, the child had to breathe through the mouth.

From the above observations, three gradings were entered into the variable of MB:

- (1) Oral breathing (01): mouth open when relaxed, breathing wholly or partly through the mouth, resistance in one nostril high, in the other moderate.
- (2) Nasal breathing (10): mouth closed when relaxed, resistance in both nostrils very low.
- (3) Oro-nasal breathing (00): or habitual mouth breather, mouth open when relaxed, breathing wholly or partly through the mouth, resistance in one nostril moderate, in the other very low.

Error in this method is, again, described in ensuing section (V2.4).

#### 2.3.4 Dentofacial variables

These are evaluated from the tracings from lateral cephalometric radiographs and plaster study models. They were divided into skeleton and dentition variables, all of which are generally accepted as specific characteristics of 'Adenoid facies' (Linder-Aronson 1970).

Out of the thirty subjects, three showed classic appearance of 'adenoid facies'. Fig. 74 shows the facial appearance of one (subject no. 11) while Figs. 21, 49, 50, 51 and 52 show intra-oral conditions of the same patient. As in most other patients, only some of the 'adenoid facies' characteristics are present even though relatively large adenoids were observed.



Fig. 74. Subject 11, showing a typical 'adenoid face'

#### 2.3.4.1 Radiographic measurements

These were made on Kodak acetate tracing film on which the reference points or planes were marked. Variables obtained include: skeleton variables S1, S2, S3, S4, S5 and S6; dentition variables D1, D2, D3 and D4.

While cephalometric definitions of these variables are expressed in Table 4, the above measurements are also shown as they were traced by Fig. 75, which is obtained from subject no. 13.

The usual cephalometric notations were used.

#### 2.3.4.2 Study models measurements

These were measured directly on plaster study models made from impressions taken at the time of examination. Measuring points were marked according to Lundstrom (1948), and linear measurements obtained by a caliper to the nearest 0.1 mm (Fig. 70).

Variable D5: the width of the dental arch in the maxilla was taken as the inter-first molar distance registered at mesial fossa centre.

Variable D6: the length of the lower arch measured as the distance between the inter-first molar line and the incisal edges.

Variable D7: lingual cross-bite of the upper first molars was denoted as 1, and normal buccal-lingal relationship was designated as 0.

Variable D8: Angle's classification of molars was designated, for convenience in computer programming, as Class I (01), Class II (10), and Class III (00).

The height of the palatal vault (S7), i.e., to its highest point, was measured at right angles to the plane of occlusion, in the line joining the measuring points on the first molars (Fig. 76). A Vitrex profile gauge, commonly used by carpenters was deemed useful for this purpose (Klami and Horowitz 1979). This instrument was placed over the model so that the pins could be pressed to the palatal surface of the cast and locked into position. The resultant outline of the array of pins were superimposed on a plastic grid (Fig. 77) from which the palatal height could be measured to the nearest 1.0 mm.

SKELETON VARIABLES

S1 Angle SNA  
 S2 Angle SNB  
 S3 Angle ANB  
 S4 MP : SN  
 S5 MP : PP  
 S6 SN/NGn x 100

DENTITION VARIABLES

D1  $\underline{1}$  : SN  
 D2  $\bar{1}$  : MP  
 D3 OP : SN  
 D4 OP : MP

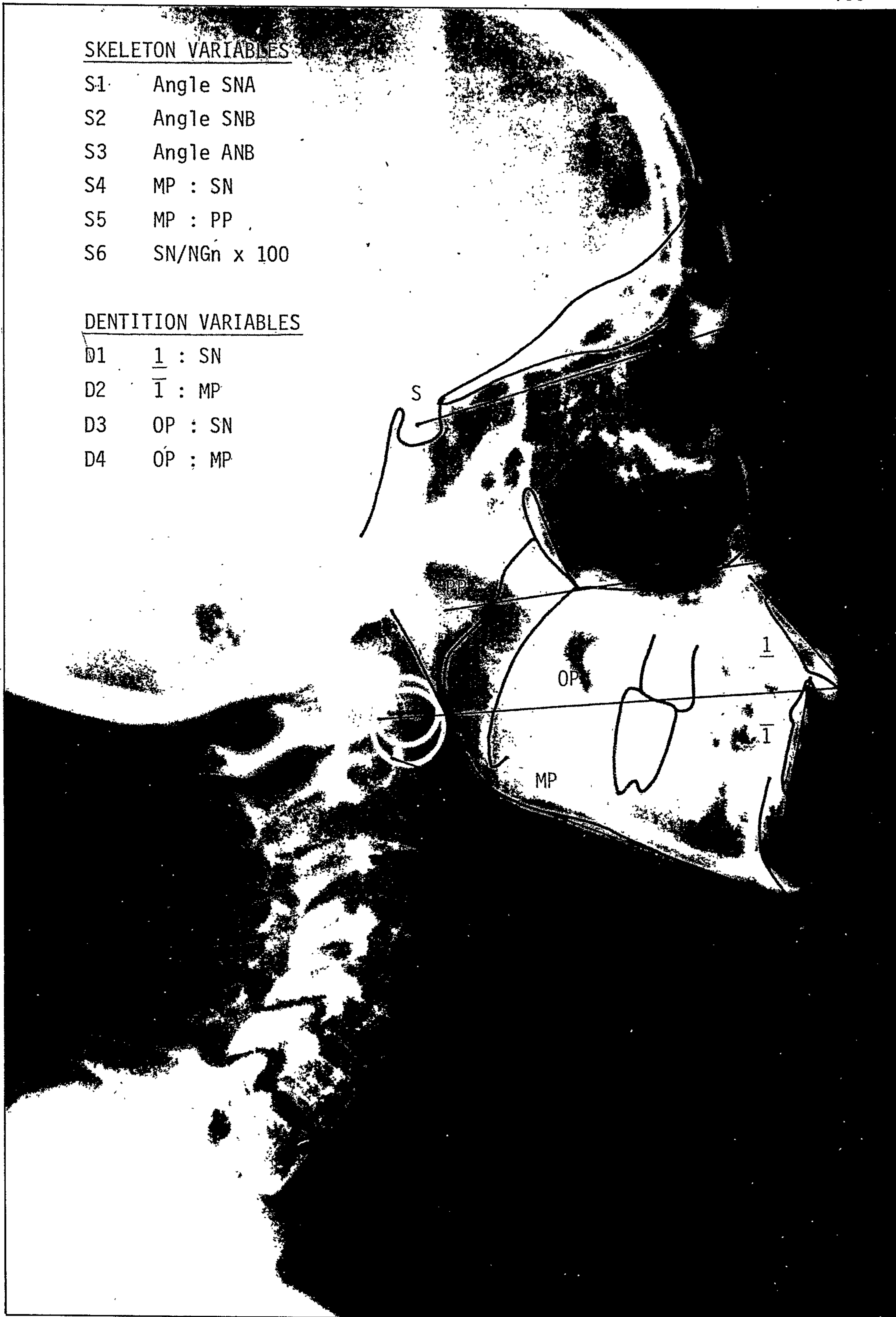


Fig. 75. , Cephalometric sketeton and dentition variables (subject 13)

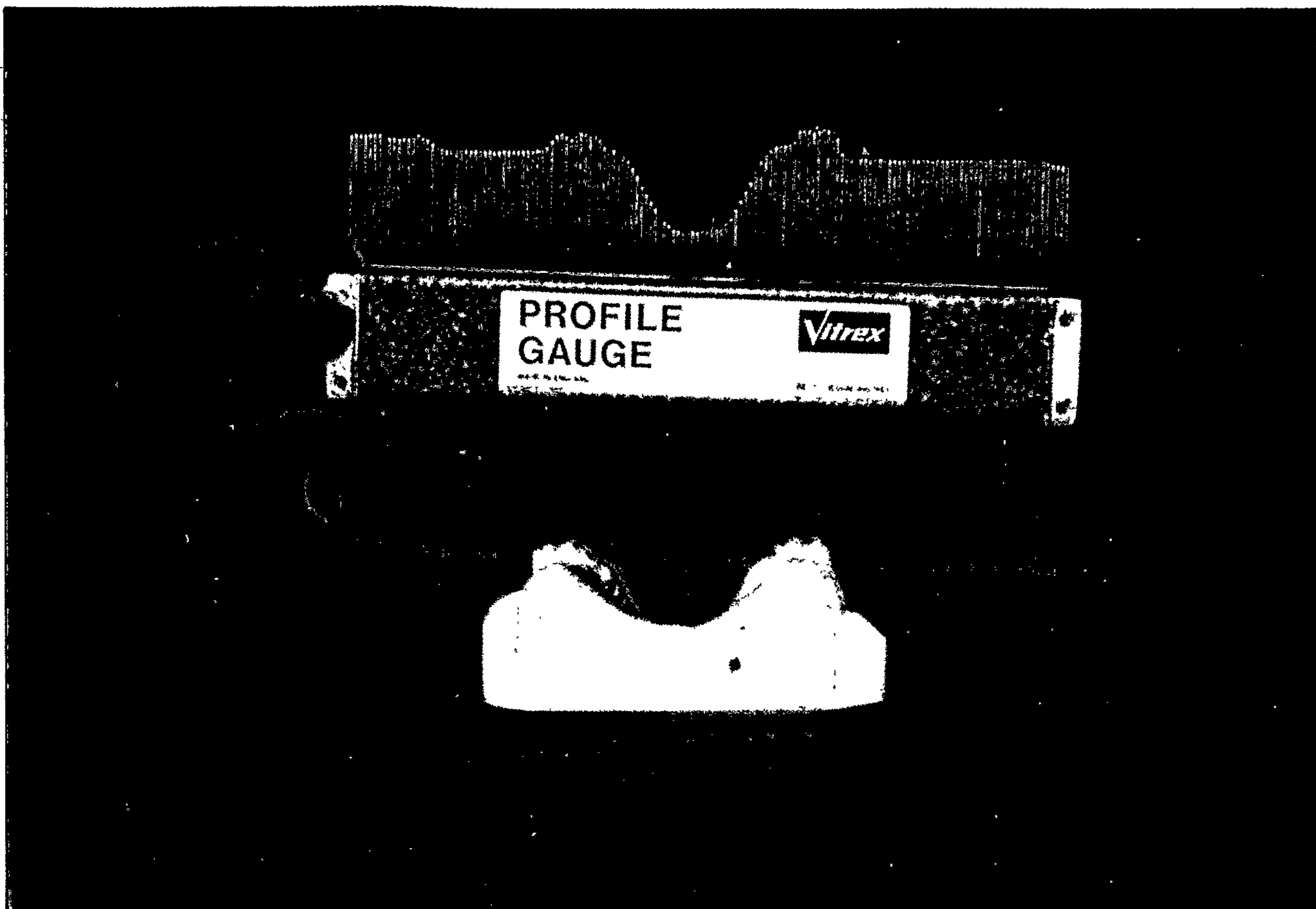
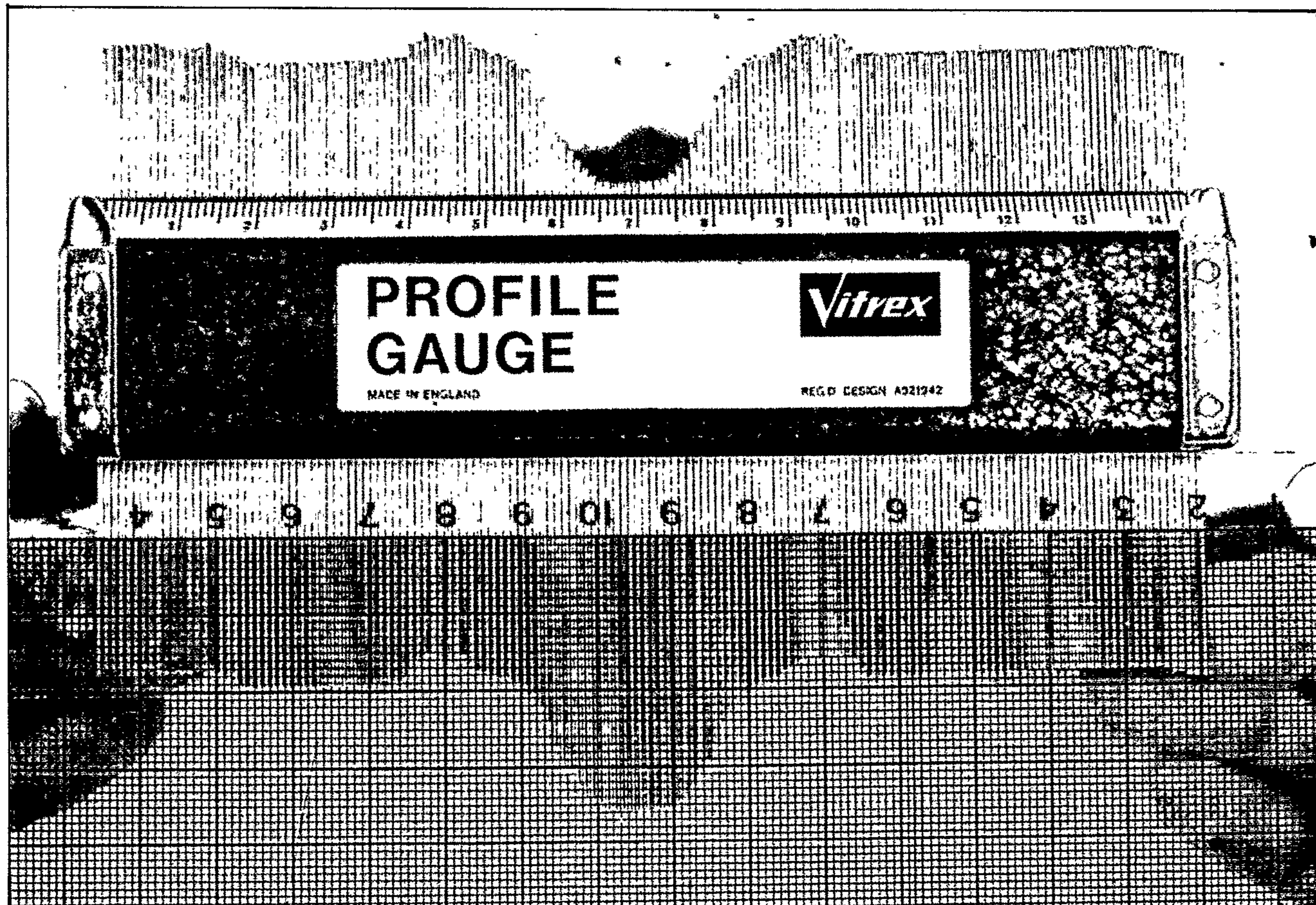


Fig. 76. Vitrex profile gauge used in this study to measure height of palatal vault on study models

Fig. 77. Placement of clear grid over locked pins to obtain measurements



# **Section V**

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## **Error of Methods**

## SECTION V: ERROR OF METHODS

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### 1. CEPHALOMETRIC RELIABILITY

Out of the vast amount of literature examining cephalometric reliability, only those relevant to the present study are mentioned.

The three general types of error in head film measurements identified by Baumrind and Frantz (1971a, 1971b), i.e., errors of projection, landmark identification and tracing, were also elaborated by Thurow (1951), Hallet (1959), and Graveley and Benzies (1974).

While Richardson (1966) regarded error in different dimensions as due to a curved anatomic outline, Bjork and Solow (1962) recommended measurements used in correlation analyses be marked directly on the radiographs.

Whereas Hixon (1956), and Broadway, Healy and Poyton (1962) found that inter-observer error was greater than intra-observer error, Moyers and Bookstein (1979) recently took into the consideration of growth changes as a potential error.

In the light of the above, the following steps were taken to reduce the error of cephalometric technique:

- (1) If possible, landmarks lying in the midsagittal plane were used.
- (2) An average was taken if there were two images of two sides of a region.
- (3) The shadow of the two ear rods were monitored to coincide so that the x-rays passed right angles to midsagittal plane.
- (4) Radiographs were taken with the patient's teeth in occlusion and with respiration suspended during nasal respiration. If the soft palate was not seen suspended in a relaxed manner, or was opposing the posterior pharyngeal wall, the radiograph was either discarded or retaken.
- (5) Only landmarks that were readily discernible and reproducible were favoured.

## 2. INTRA-OPERATOR VARIABILITIES

### 2.1 Tracings

To assess the inherent error of tracing technique, ten lateral cephalometric radiographs were selected at random from the sample. Four measurements were chosen and traced, and then retraced 4 weeks later. The two sets of observations are listed on Table 5.

The mean difference ( $\bar{d}$ ), the standard deviation (SD) using Bessel's correction for small samples, and 't' values for the double observations were calculated and set out on Table 6. The 't' values were entered at N-1 degrees of freedom at distribution of 't' table.

All values were found to be insignificant at 0.05 level.

### 2.2 Scan-based volume determinations

A similar test was performed on double determinations of 10 pairs of scan-based volume determinations as listed on Tables 7 and 8, and values were found to be insignificant.

### 2.3 Study models measurements

Similarly, double measurements of palatal heights as measured on study casts were made as listed on Tables 9 and 10, and again, values were found to be insignificant.

### 2.4 Mode of breathing recordings

An orthodontist and the author made double determinations in every subject observed, and values are shown in Table 11. The results displayed good agreement between the orthodontist and author.

SUBJECT	∠SNA			∠SNB			MP : SN			OP : MP		
	1st	2nd	d	d <sup>2</sup>	1st	2nd	d	d <sup>2</sup>	1st	2nd	d	d <sup>2</sup>
16	80.0	80.5	-0.5	0.25	78.0	78.0	0	0	33.5	33.0	+0.5	0.25
10	75.0	75.0	0	0	73.0	73.0	0	0	40.0	40.0	0	0
2	80.5	80.0	+0.5	0.25	80.5	80.0	+0.5	0.25	37.5	37.0	+0.5	0.25
5	82.0	82.0	0	0	80.0	79.5	+0.5	0.25	31.5	31.0	+0.5	0.25
8	79.0	79.0	0	0	79.0	79.0	0	0	37.0	37.0	0	0
18	83.5	84.0	-0.5	0.25	81.5	81.0	+0.5	0.25	34.0	34.0	0	0
29	80.0	79.0	+1	1.00	78.0	78.0	0	0	39.0	39.5	-0.5	0.25
4	83.0	83.0	0	0	76.0	76.0	0	0	43.0	43.0	0	0
19	85.0	85.0	0	0	82.0	82.0	0	0	28.0	29.0	-1	1.00
3	74.0	74.0	0	0	79.0	79.5	-0.5	0.25	43.0	42.0	+1	1.00
			+0.5	0.75			+1	1.00			+1	1.00
											0	2.00

Table 5 Double determinations of measurements on lateral cephalometric radiographic tracings (N = 10)

Measurement	$\bar{d}$	SD	$SE_{\bar{d}}$	t	at 0.1 level sig/not sig.
$\angle$ SNA	+0.05	0.282	0.089	0.56	ns
$\angle$ SNB	0.10	1.040	0.330	0.30	ns
MP : SN	0.10	1.040	0.330	0.30	ns
OP : MP	0	0.469	0.148	0	ns

Table 6 't' test of tracing error of 4 measurements on lateral cephalometric radiograph

Subject no.	NP vol.		Difference (1-2) d	d <sup>2</sup>
	1st	2nd		
1	11.0	10.3	+0.7	0.49
2	12.1	12.0	+0.1	0.01
3	13.9	14.5	-0.6	0.36
4	15.3	15.1	+0.2	0.04
5	15.4	15.4	0	0
6	16.2	15.9	+0.3	0.09
7	16.3	16.3	0	0
8	16.9	16.8	+0.1	0.01
9	20.7	20.9	-0.2	0.04
10	21.2	21.0	+0.2	0.04

**Table 7 Double determinations of 10 pairs of scan-based nasopharyngeal volume determinations**

$\bar{d}$	SD	$SE_{\bar{d}}$	t	sig/n sig
0.08	1.05	0.33	0.24	ns

**Table 8 't' test of error of scan-based NP volume determinations**

Subject No.	S7		Difference (1-2) d	d <sup>2</sup>
	1st	2nd		
10	21.8	22.0	-0.2	0.04
2	18.8	18.9	-0.1	0.01
5	17.9	18.0	-0.1	0.01
8	18.6	18.3	+0.3	0.09
18	18.7	18.7	0	0
29	19.7	19.7	0	0
4	20.8	20.0	+0.8	0.64
19	16.9	16.9	0	0
3	22.2	22.0	+0.2	0.04
21	19.9	19.9	0	0

**Table 9 Double determination of 10 pairs of study model palatal vault height measurements (S7)**

$\bar{d}$	SD	$SE_{\bar{d}}$	t	sig/n sig
0.09	1.04	0.33	0.24	ns

**Table 10 't' test of error of 10 pairs of study model palatal vault height measurements (S7)**

Variable	assessment by orthodontist and author				
	N	same		different	
		n	%	n	%
Mode of breathing.(MB)	30	28	93.3	2	6.6

**Table 11 Double determination for mode of breathing**

### 3. INTRA-INDIVIDUAL VARIABILITIES

#### 3.1 Due to Growth

Changes due to growth in this study are minimal because all measurements except airflow were taken concurrently. Size of nasopharyngeal space was proven to be stable during a 12-month period after pubertal years (Henrikson, Linder-Aronson and Westborg 1975), implying a constant relationship between airway patency and nasopharyngeal volume during the same period.

#### 3.2 Associate nasal problems

Before an attempt to obtain airflow variable with the mini Wright Peak Flow Meter, it was ensured that the subject had been in the same room for at least one hour to avoid allergic oedema of nasal membranes. It was also ascertained that the subject had no structural deformities of the nose, suffering from a cold, or having a blocked nose due to obscure causes.

#### 3.3 Nasal cycles

Although alternate cyclic changes in each nostril have been proven (Principato and Ozenberger 1970), unilateral (right and left) resistance fluctuates in opposite phase. Total nasal resistance therefore remains constant and should not affect airflow measurements.

Furthermore, mode of breathing tests involved alternate testing of each nostril, and should nullify error due to nasal cyclic changes.

### 4. APPARATUS ERROR

#### 4.1 CT Scanner

Because of the undesirability of excessive irradiation, inter-scan

differences were not evaluated. However, it has been proven that such error is minimal on measurements of nasal airways in cadavers (Montgomery et al 1979) and is also taken for granted in this study.

#### 4.2 Polar Planimeter

This instrument has been used in many previous studies and found to be satisfactory. Its accuracy was tested in this study by measuring  $1.0 \text{ cm}^2$  and  $2.0 \text{ cm}^2$  each five times. The planimeter reading was reproducible and accurate to better than  $0.1 \text{ cm}^2$  for each square.

#### 4.3 Mini Wright Peak Flow Meter

The reliability of this instrument and strong correlation with the standard Wright Peak Flow Meter have been proven in a report prepared by the Medical Research Council, Pneumoconiosis Unit, Landough, as described in the pamphlet 'notes for the doctor' accompanying the purchase of the instrument.

It is possible, however, that leakage between the nose mask and the face might occur, and was minimised by using a flexible mask with the rim held firmly against the face during usage.

#### 4.4 Error of application and instruction

This occurs mainly during airflow measurements. It was envisaged that some subjects might not respond fully to the command of a 'quick, forceful expiration' to give the peak expiratory rate. The best of six readings usually allowed the subject to accustom to the instrument.

As noted before, the highest reading is often during the third or fourth blow when the subjects were suitably accustomed to the Flow Meter, but before the ultimate 'exhaustion' of breath!

# **Section VI**

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## **Statistical Procedures used in Data Analyses**

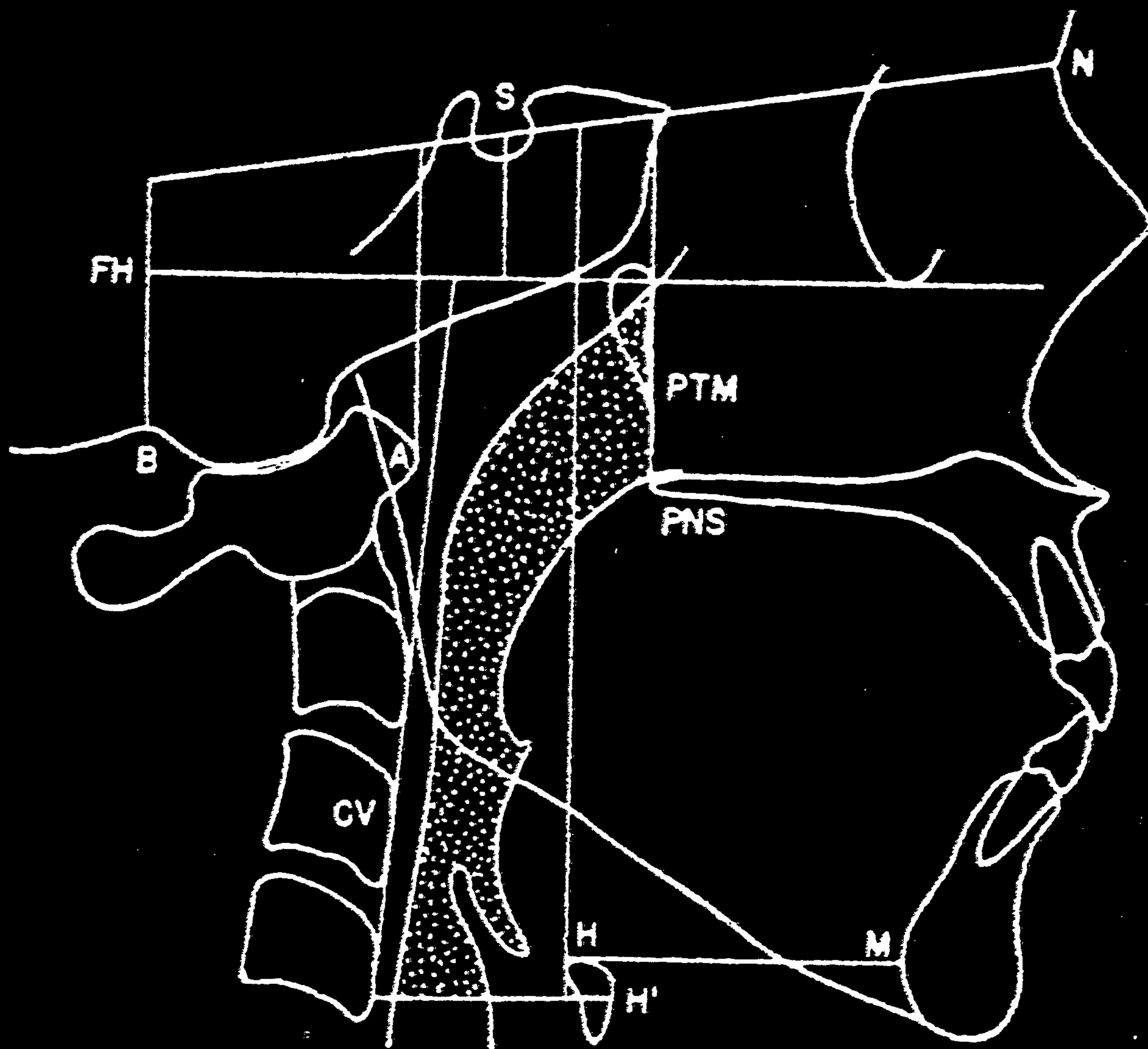


Fig. 78. King's radiographic measurements of velopharyngeal region (From King 1952)

## SECTION VI: STATISTICAL PROCEDURES USED IN DATA ANALYSES

The methods used in this study to analyse relationships among the variables obtained relied on a complex application of simple correlation analysis and multiple regression analysis. While the statistical notations are listed on Appendix 2, it is desirable at this juncture to relate the rationale of the methods so employed.

The basis of the statistical design is adapted from principles propounded by Linder-Aronson (1970), Nie et al (1975), McClymont (1975), and Swinscow (1977).

1. SIMPLE CORRELATION ANALYSIS

This method gives an indication of the relationship between two sets of variables without taking other variables into account. A correlation coefficient ( $r$ ) is a single number which summarizes the relationship between two variables. It indicates the degree to which variation (or change) in one variable is related to variation in another. Besides its role as an indicator of the goodness of fit of the linear regression, it also provides an easy means for comparing the strength of relationship between one pair of variables and a different pair.

Mathematically,  $r$  is defined as the ratio of covariation to square root of the product of the variation in  $X$  and the variation in  $Y$ , where  $X$  and  $Y$  symbolize the two variables. This corresponds to the formula:

$$r = \frac{\sum_{i=1}^N (X_i - \bar{X})(Y_i - \bar{Y})}{\left[ \sum_{i=1}^N (X_i - \bar{X})^2 \right] \left[ \sum_{i=1}^N (Y_i - \bar{Y})^2 \right]}^{\frac{1}{2}}$$

where  $X_i$  =  $i$ th observation of variable  $X$   
 $Y_i$  =  $i$ th observation of variable  $Y$   
 $N$  = number of observations  
 $\bar{X}$  = mean of variable  $X$   
 $\bar{Y}$  = mean of variable  $Y$ .

When there is a perfect fit,  $r$  takes on the value of +1.0 or -1.0. A negative  $r$  means an inverse relationship, and the value of zero denotes the absence of a linear relationship.

There is no set scale of values for  $r$ , but the following verbal description is adopted (McClymont 1975):

- $r < 0.20$  : Slight correlation; almost negligible relationship.
- $r = 0.20 - 0.40$ : Low correlation; definite but small relationship.
- $r = 0.40 - 0.60$ : Moderate correlation; substantial relationship.
- $r = 0.60 - 0.90$ : High correlation; marked relationship.
- $r = 0.90 - 1.00$ : Very high correlation; very dependable relationship.

The reliability of  $r$ , however, depends on the sample size, and is measured by calculating a value for Student's  $t$ , using the formula (Nie et al 1975):

$$t = \frac{r \sqrt{N - 2}}{\sqrt{1 - r^2}}$$

and the distribution of  $t$  is entered at  $N - 2$  degrees of freedom.

A scattergram can be very helpful though in displaying the relationships, and from the display, the researcher can decide whether a polynomial regression is warranted. It is a graph of data points based on two variables, where one variable defines the horizontal axis and the other defines the vertical axis.

In this investigation, simple correlation analyses in the form of scattergrams are applied to the test of correlation between scan-based nasopharyngeal volume determinations ( $Y$ ), and chosen radiographic quantification measurements ( $X_1$  to  $X_{15}$ ), as obtained from the first group of subjects.

Values of simple correlation coefficients are also computed for each pair of all the variables used in the testing of 'Adenoid Facies Path' (i.e., AA, BI, MB, S1 to S7, and D1 to D8), as obtained from the second group of subjects.

## 2. MULTIPLE REGRESSION ANALYSIS

This is a general statistical technique through which one can analyse the relationship between a dependent or criterion variable

and a set of independent or predictor variables. Multiple regression analysis can be used as a descriptive tool by which the linear dependence of one variable on others is summarised and decomposed.

An important property of this technique is to control for other confounding factors in order to evaluate the contribution of a specific variable or set of variables. A variety of partial coefficients can be obtained using multiple regression analysis of a finite number of variables. Therefore, the researcher can examine the effect of say, AA on S1, while controlling for variation in other variables.

Although it is not the intention of this study to substantiate a cause-effect relationship in the 'Adenoid Facies Path', multiple regression analysis is nevertheless a useful tool to assert a direction of causal theory. The emphasis on such an application is neither on the overall dependence of one variable on another nor the relationship between any particular pair of variables. Rather, multiple regression is used to describe the entire structure of linkages between independent and dependent variables, and to assess the logical consequences of a structural model that is posited *a priori* from some causal theory.

The 'Adenoid Facies Path' can therefore be constructed to specify an 'ordering' among the variables that reflects a presumed structure of cause-effect linkages (Fig. 79):

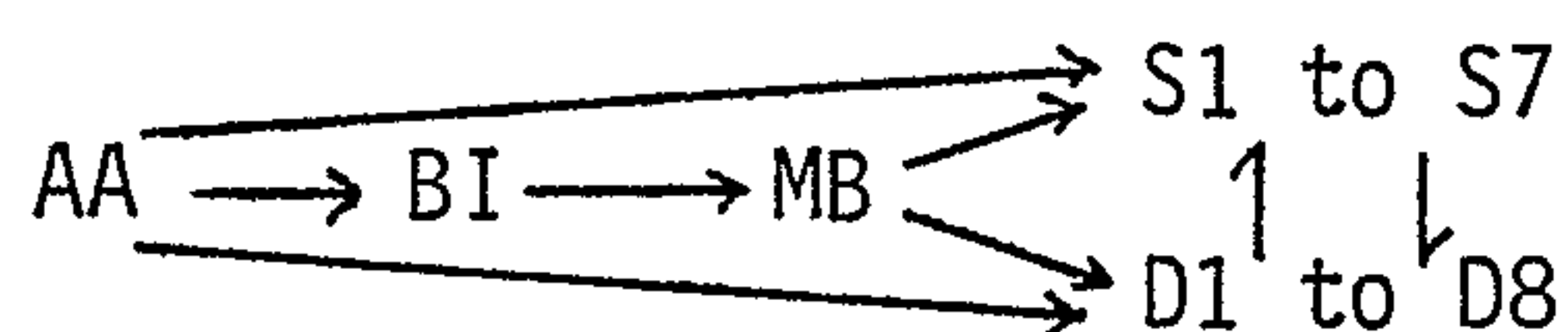


Fig. 79 Causal theory of the 'Adenoid Facies Path'

Multiple regression techniques are then used to determine the magnitude of direct and indirect influence that each variable has on other variables that follow it, in the presumed order and in an exploratory manner. Each arrow in Fig. 79 represents a presumed causal linkage or path of causal influence. Through regression techniques, the strength of each separate path is estimated, involving a large number of regression equations.

## 2.1 REGRESSION COEFFICIENT

A more useful quantity, however, is the partial regression coefficient (B). A partial coefficient, say  $B_1$ , in the equation

$$Y' = A + B_1X_1 + B_2X_2 + \dots + B_kX_k$$

represents the expected change in  $Y$  with a change of one unit in  $X_1$  when  $X_2, \dots, X_k$  are held constant or otherwise controlled for. It can also be looked at as a simple regression coefficient between  $Y$  and the residuals of  $X_1$  from which the effects of  $X_2, \dots, X_k$  are taken out.

In the above equation,  $Y'$  represents the estimated value for  $Y$ ,  $A$  is the  $Y$  intercept, and the  $B_i$  are regression coefficients.

The actual calculation of  $A$  and  $B_i$  requires a set of simultaneous equations derived by differentiating  $\Sigma(Y-Y')^2$  and equating the partial derivatives to zero. A standard form of such equations for two predictor variables is:

$$A + B_1\bar{X}_1 + B_2\bar{X}_2 = \bar{Y}$$

$$B_1(SS_1) + B_2(SP_{12}) = SP_{y1}$$

$$B_1(SP_{12}) + B_2(SS_2) = SP_{y2}$$

where  $SS$  and  $SP$  stand for sum of squares and sum of products, or variation and covariation, respectively.

It is worth noting at this point that the relative magnitude of the partial regression coefficient of an independent variable can be quite different from its bivariate regression coefficient with the dependent variable, since the bivariate coefficient is confounded with the effects of other correlated independent variables.

## 2.2 COEFFICIENT OF DETERMINATION

As in the bivariate case, the proportion of the variance of a dependent variable explained can be evaluated by the square of the multiple correlation coefficient, or coefficient of determination ( $R^2$ ).

It should be noted that the term 'variance' used is a measure of the variability, or lack of homogeneity, in a variable. When the cases

cluster close to the mean, variance will be small; as the cases becomes more spread out, variance increases. The objective of the correlation analysis is to determine the extent to which variation in one variable is linked to variation in the other.

$$R^2 = \frac{\text{variation in } Y \text{ explained by the combined linear influence of the independent variables}}{\text{total variation in } Y}$$

$$= \frac{SS_y - SS_{res}}{SS_y} = \frac{SS_{reg}}{SS_y}$$

where  $SS_{\Sigma(Y-\bar{Y})^2} = SS_{reg} + SS_{res}$  ; or

$$\Sigma(Y-\bar{Y})^2 = \Sigma(Y'-\bar{Y})^2 + \Sigma(Y-Y')^2$$

and  $SS_y$  = total sum of squares in Y (which is the variability of the dependent variable Y),

$SS_{reg}$  = the sum of squares explained by the regression line or the entire equation,

$SS_{res}$  = the residual (unexplained) sum of squares,

Y = actual Y value,

Y' = predicted Y value,

$\bar{Y}$  = mean of values of Y,

Residual = a deviation of an observed Y score from an estimated Y' value.

If  $R^2 = 0$ , nothing of the variance can be attributed to the independent variables. If  $R^2 = 1$ , then all the variability is attributed to the independent variables in the regression equation. In this study, the following verbal description is adopted (Linder-Aronson 1970):

$R^2 < 0.30$  = weak,

$R^2 > 0.50$  = satisfactory.

### 2.3 STEPWISE REGRESSION

Stepwise introduction in an hierarchical fashion is used in the present analyses. Variables are added to the regression in an order pre-determined by the researcher. Variables are added in single steps, and the increment in  $R^2$  (or in the explained sum of squares) at each step is taken as the component of variation attributable to the particular variable added on that step.

This hierarchical procedure involves adjustments for only those variables that precede a given variable in the hierarchical order,

and therefore reflects the *total influence* of each variable.

Consider a regression of a dependent variable  $Y$ , and three independent variables  $X_1$ ,  $X_2$ , and  $X_3$ , the independent contribution of each variable adds up to the total variation explained by the three variables is expressed by the equation:

$$R^2 = \begin{matrix} R^2 \text{ increment} \\ \text{due to } X_1 \end{matrix} + \begin{matrix} R^2 \text{ increment} \\ \text{due to } X_2 \end{matrix} + \begin{matrix} R^2 \text{ increment} \\ \text{due to } X_3 \end{matrix}$$

## 2.4 CONFIDENCE LIMITS

Confidence limits for estimates are evaluated by tests designed for goodness of fit of the regression equation ( $R^2$ ), or for a specific regression coefficient ( $B$ ).

### 2.4.1 Overall F for $R^2$

The overall goodness of fit of the regression equation, as in an analysis of variance, can be assessed by the F ratio as:

$$F = \frac{SS_{\text{reg}}/k}{SS_{\text{res}}/(N-k-1)} = \frac{MS_{\text{reg}}}{MS_{\text{res}}} = \frac{R^2/k}{(1-R^2)/(N-k-1)} ; \text{ where}$$

$SS_{\text{reg}}$  = the sum of squares explained by the entire equation  
 $= \sum (Y' - \bar{Y})^2$ ,

$SS_{\text{res}}$  = the residual (unexplained) sum of squares  
 $= \sum (Y - Y')^2$ ,

$k$  = number of independent variables in the equation,

$N$  = sample size,

$MS_{\text{reg}}$  = mean square explained by the entire equation,

$MS_{\text{res}}$  = the residual mean square,

$R^2$  = squared multiple correlation coefficient.

F ratios are compared to the tabled F distribution with degrees of freedom  $k$  and  $(N-k-1)$ , which gives the probability of the sample being drawn from a population in which  $R^2$  is equal to zero.

### 2.4.2 Standard error of B

If  $B$  is estimated from a sample, the values of  $B$  will vary from

sample to sample. In the long run, however, the mean of B's will coincide with the population value  $B$ , and we can estimate the standard deviation of the sampling variability of  $B$  if certain assumptions are met. The estimate of the standard error of  $B$  is given by:

$$\sqrt{\text{Var}(B)} = \sqrt{\frac{(Y-Y')^2 / (N-2)}{(X-\bar{X})^2}} = \sqrt{\frac{SS_{\text{res}} / (N-2)}{SS_x}}$$

where  $Y'$  = predicted  $Y$  value,  
 $Y$  = actual  $Y$  value,  
 $N$  = sample size,  
 $X$  = actual  $X$  value,  
 $\bar{X}$  = mean of values of  $X$ ,  
 $SS_{\text{res}}$  = the residual sum of squares,  
 $SS_x$  = the sum of squares of  $X$ .

If the sample size is relatively small, the  $B$  estimates follow the  $t$  distribution with  $(N-2)$  degrees of freedom. Therefore, the 95 per cent confidence interval for  $B$  given the sample size of 30, estimated  $B = 2.3$ , and the standard error of  $B = 0.4$ , is given by:

$$2.3 - 2.048(0.4) < B < 2.3 + 2.048(0.4),$$

the value of (2.048) is obtained from the table of Student's  $t$  distribution with degrees of freedom equal to 28.

#### 2.4.3 F tests for B

For the variable tested first,

$$F = \frac{r_{y1}^2 / 1}{(1 - R_{y.12, \dots, k}^2) / (N-k-1)},$$

where  $r^2$  = squared part correlation coefficient.

For the second regression coefficient,

$$F = \frac{r_{y(2.1)}^2 / 1}{(1 - R_{y.12, \dots, k}^2) / (N-k-1)},$$

$$= \frac{\text{incremental SS due to } X_2 / 1}{SS_{\text{res}} / (N-k-1)}$$

where a squared part correlation or incremental sum of squares appears in the numerator.

For the third variable tested, the test statistic is:

$$F = \frac{r_{y(3.12)}^2 / 1}{(1 - R_{y.12, \dots, k}^2) / (N-k-1)}$$

$$= \frac{\text{incremental SS due to } X_3 / 1}{SS_{\text{res}} / (N-k-1)}$$

Each successive variable to be considered in the hierarchical fashion as employed in this investigation will be tested by an F ratio of the latter form.

All F ratios are, again, compared to the tabled F distribution with degrees of freedom 1 and (N-k-1), which gives the probability of the sample being drawn from a population in which B equals to zero.

### 2.5 ORDER OF INCLUSION

The order of inclusion whereby variables of the 'Adenoid Facies Path' were introduced are listed on Table 12. In cases of skeleton and dentition variables, the one with the strongest positive or negative correlation with the dependent variable was introduced first, after which one introduced the variable that showed the strongest partial correlation to the dependent variable with allowance for the first dependent variable, and so on.

### 3. DATA PROCESSING

Manual calculations were performed on a Sharp EL-8131 Elsi Mate electronic calculator.

Scattergrams, simple correlation matrices, and multiple regression analyses in a stepwise fashion were processed by Sydney University Computing Centre employing programmes in the Statistical Package for the Social Sciences (Nie et al 1975).

Dependent Variable	Step one	Step two	Step three	Step four
AA	S1-S7**	D1-D8	S1-S7** + D1-D8	
AA	MB + BI	+ S1-S7**	+ D1-D8	
BI	AA + MB	+ S1-S7*	+ D1-D8	
MB	AA + BI	+ S1-S7*	+ D1-D8	
S1	AA + BI	+ MB	+ S2-S7***	+ D1-D8
S2	AA + BI	+ MB	+ S1-S7***	+ D1-D8
S3	AA + BI	+ MB	+ S1-S7*	+ D1-D8
S4	AA + BI	+ MB	+ S1-S7**	+ D1-D8
S5	AA + BI	+ MB	+ S1-S7**	+ D1-D8
S6	AA + BI	+ MB	+ S1-S7*	+ D1-D8
S7	AA + BI	+ MB	+ S1-S6**	+ D1-D8
D1	AA + BI	+ MB	+ D2-D8	+ S1-S7**
D2	AA + BI	+ MB	+ D1-D8	+ S1-S7*
D3	AA + BI	+ MB	+ D1-D8	+ S1-S7**
D4	AA + BI	+ MB	+ D1-D8	+ S1-S7**
D5	AA + BI	+ MB	+ D1-D8	+ S1-S7*
D6	AA + BI	+ MB	+ D1-D8	+ S1-S7**
D7	AA + BI	+ MB	+ D1-D8	+ S1-S7*
D8	AA + BI	+ MB	+ D1-D7	+ S1-S7*

*Notes:*

- 1) By definition, S1, S2 and S3 exhibit a perfect relationship (angle SNA - angle SNB = angle ANB). These quantities were therefore excluded from the regression equation. \*S1 excluded; \*\*S2 excluded; \*\*\*S3 excluded.
- 2) Definitions of variables are listed on Table 4.
- 3) Where variables were entered as a block (i.e., skeleton and dentition variables) as independent variables to a dependent variable from the same block, the latter was obviously also excluded from the regression equation.

**Table 12 Order of Stepwise Multiple Regression**