4.2.2 Open Bite Malocclusion

Much has been discussed regarding open bite in previous sections. In this section, a closer examination of open bite is presented.

As far as cause-and-effect is concerned, historically it has been a case of which came first, the open bite or the tongue thrust. An up-to-date discussion of these aspects is also presented.

4.2.2.1 Classifications, Characteristics, and Incidence

Before a systematic investigation of open bite began, a number of authors described, from their own observations, only parts of the overall nature of open bite. Hellman (1931) stressed the skeletal growth aspects of open bite. In a study of 43 patients, both treated and untreated, who exhibited anterior open bite, of those who were treated he found that the percentage of successful treatments was equal to the number of spontaneous self-corrections in the untreated group. He suggested that open bite is primarily due to skeletal growth deficiencies, "when growth changes are favourable, the prognosis of open bite cases is excellent and may require no orthodontic treatment; when unfavourable, the outcome of treatment is questionable".

Swinehart (1942) and Straub (1960) on the other hand, concluded that tongue thrust is the primary cause of open bite and that retraining the tongue and eliminating muscle dysfunction would correct the condition. In the first of a collection of articles, Straub (1951) made a number of interesting observations involving tongue thrust and open bite. He blamed tongue thrusting for open bite malocclusions, based on his research into children with tongue thrusts. He found it common to see "a narrow upper arch and in a great many cases a severely contracted maxilla with protruding upper teeth in open bite
relationship". He also noted that posterior teeth as well as anterior teeth may be separated by tongue thrust. Open bites could also be caused, he believed, by unusually large tongues together with the tongue thrust. He made mention, too, of the futility of correcting the open bite malocclusion without correcting the tongue thrust because of the ensuing relapse.

Rix (1953) described more fully the open bite characteristics of young children. He found that they showed varying degrees of anterior open bite, "these children not only exhibit a suckling behaviour of the tongue during swallowing, but the posture of the tongue remains infantile throughout", meaning that there was tongue to lower lip contact. He found that the tongue "did not retreat within the dental arches, it remained forward and over the lower incisors. Lower incisors, he found, erupting under these conditions, were impeded in their eruption. Likewise the upper incisors are also impeded at a later date, but the more forward they are guided by the "suckling tongue action" the less they are impeded. Thus the anterior open bite cases retained an infantile postural pattern of the tongue. However, he noted that this phenomenon tended to lessen with age, with anterior open bites gradually improving.

Winders (1963) attempted to explain the etiological relationship between anterior open bite, perioral muscular contraction, and tongue thrust by using "logic", "If we can accept the basic premises that teeth seek occlusal antagonists, be it teeth or palate - and that in the infant before eruption of teeth there is a normal contraction of perioral musculature associated with a tongue thrust, we can validly argue the tongue thrust is primary or causal in nature. Ontogenetically, we may look at this syndrome as a prolonged infantile swallowing reflex in which the tongue thrust does not allow the anterior teeth to erupt." This was in agreement with Rix (1953). He also
mentioned condylar growth and habits as being, among others, causal influences.

From his lingual/labial pressure studies, Winders found that the buccal and labial musculature does not contract during swallowing unless there is an anterior open bite or lack of anterior overbite with accompanying antero-posterior skeletal dysplasia. Also that a tongue thrust during swallowing can be the cause, not the result, of the anterior opening; however, this was not well explained.

In defining open bite, Brodie (1971) called it "any condition in which the lower incisors do not make contact with the palate or with the upper incisors when the jaws are clenched".

Similarly, Moyers (1973) simply described an open bite as the result of vertical development that is insufficient to permit a tooth or teeth to meet their antagonists in the opposite arch, resulting in "localised absence of occlusion". He listed three main types of causes of open bites:

1. Disturbances in the eruption of the teeth and alveolar growth, e.g., ankylosed primary molars;

2. Mechanical interference with eruption and alveolar growth, e.g., digit sucking habit; and


He described two kinds of incisor relationship in anterior open bite - one where there is open bite without vertical overlap of the incisors, and another where there is vertical overlap (Fig. 149).

Moyers did distinguish between anterior and posterior open bites. For anterior open bites the aspects of "deleterious habits" and "gross osseous dysplasia" as causes, were discussed. He mentioned that the majority of
Fig. 149  Open bite classification. Left, normal incisal relationship. Centre, open bite without vertical overlap of the incisors. Right, open bite with vertical overlap of the incisors. This type of open bite often is not diagnosed. (Moyers, 1973)
anterior open bites were caused by sucking habits - finger, thumb, tongue, pipe, other. The interposition of these objects between the teeth impeded the eruption of the teeth. Control of the habit is a primary treatment aim, he said, and successful retention of a corrected open bite depended on the elimination of the deleterious habit. For skeletal dysplasias such as micrognathia, mandibular hypertrophy, and other severe disorders of the craniofacial skeleton, the open bite caused could be just one aspect of the gross bony discrepancy.

Regarding posterior open bite he discussed tongue thrust and ankylosed primary teeth as causes. For tongue thrust he suggested that "only rarely" is it a primary etiologic agent. He found that frequently "the open bite is formed and afterward the tongue maintains it to effect a seal during swallowing."

Another classification of open bite by Worms, Meskin, and Isaacson (1971) described it in three anatomical phases:

1. Simple open bite - from canine to canine, with 4mm or more in centric relation;
2. Compound open bite - from premolar to premolar; and
3. Infantile open bite - from molar to molar.

Subtelny and Sakuda (1964) discussed the definition of open bite. Drawing attention to differing opinion at that stage, they pointed out that, of course, figures describing the frequency of its occurrence, would also vary. They proposed that open bite must be considered as a deviation in the vertical relationship of the maxillary and mandibular dental arches. In particular there must be a "definite lack of contact, in the vertical direction, between opposing segments of teeth".
These authors drew up tables comparing dento-labial and skeletal relationships in open bite malocclusions and normal occlusions. These have served as an aid in diagnosis of severity of open bite (Tirk, 1965). The main characteristics of skeletal open bite they found to be:

1. Posterior cranial base shorter (sella-basion);
2. SNA smaller;
3. In a few cases, the premaxillary area seemed tipped up towards the skull;
4. Overeruption of maxillary molars and incisors, and alveolar process (not found in the mandible);
5. Excessively steep mandibular plane;
6. Short ramal height;
7. Larger gonial angle;
8. Ramus and body of mandible seemed to be bent away from each other;
9. Mandible more retruded relative to the cranial base; and
10. Excessive vertical dimension of the lower face (nasal floor to lower border of symphysis).

In assessing the role of the tongue in open bite malocclusions an assessment of skeletal deformity could be made and from there an assessment of the influence of tongue function could be made. In the more severe skeletal deformities, they suggested the tongue's influence is more likely to be an adaptation.

Cangialosi (1984) compared cephalometrically open bite patients with subjects with Class I normal occlusions. An attempt was made to clarify the differences between a true skeletal dysplasia and a habitual problem involving the dentoalveolar structures. He suggested that any means of identifying the skeletal pattern of an open bite may be helpful in the possible prevention or early treatment of this condition, and could also be a guide in assuring that
the mechanics used will not aggravate the condition.

He found great differences in skeletal patterns of normal and open bite subjects. He also found "great variation in skeletal morphology" between skeletal open bite and dentoalveolar open bite (Figs. 150, 151). Another distinguishing feature was the amount of dental eruption. In skeletal open bite the anterior teeth are likely to be normally erupted or even overerupted, whereas in habitual open bite they are undererupted because of the presence of "some object". This may be a tongue, thumb, pencil, etc. His findings showed a "great variability as well as an almost infinite number of combinations of dental and skeletal patterns in open bite subjects". Furthermore that most open bites have elements of both skeletal and dental dysplasia, "the phenomenon of anterior open bite is multifactorial, and there is an almost infinite variety to the dentsoskeletal configuration and the magnitude of dysplasia associated with it." He did however, reach some conclusions:

1. **Posterior** face-height is shorter, and overall **anterior** face height is greater in open bite subjects;

2. **Lower** face height is greater in relation to upper anterior face height in persons with open bite;

3. The **mandibular plane** and the **gonial angle** are larger in persons with open bite;

4. The **maxilla to mandible angle** (PP to GoGn) is greater in persons with open bite, and this is due mostly to downward tipping of the mandibular plane;

5. Ratios and angles measured remained relatively constant in both mixed- and permanent-dentition groups, indicating that only size (but not facial proportion) changes with age; and

6. Measurements made on the group designated as having **skeletal** open bites were significantly **different** from those of the subjects with dentoalveolar open bites and from the open bite sample as a whole,
Fig. 150  Skeletal open bite.  (Cangialosi, 1984)

Fig. 151  Habitual dentoalveolar open bite.  (Cangialosi, 1984)
for the cranial base to maxilla (SN-PP) angle.

These findings of Cangialosi were in basic agreement with those of Nahoum (1977) previously. He found that most open bites are deficient in growth of the upper face and in growth of the posterior face. However, some varieties have adequate posterior face height. A compensatory increase in lower anterior face height occurs in both types. As well, Nahoum suggested that the ratio of upper anterior face height to lower anterior face height (UAFH/LAFH) acts as an aid in determining the prognosis for the orthodontic correction of anterior open bite. This ratio was a guide to determining whether the patient was a skeletal or dental open bite. For a "normal dentition and a good face" the ratio was 0.810. The mean for patients with open bite was 0.686. Those with a ratio below 0.650 were considered "poor risks" for conventional orthodontic treatment, and surgery should be considered.

He also found that the rate of success of treatment diminished as open bite extended laterally and posteriorly into the canine, premolar, and molar regions.

A longitudinal 10-year postretention evaluation of orthodontically treated anterior open bite patients by Lopez-Gavito, Wallen, Little, and Joondeph (1985) revealed a number of significant findings:

1. That more than 35% of the treated open-bite patients exhibited a postretention open bite of 3mm or more;

2. The relapse group demonstrated, across time, less mandibular anterior dental height, less upper anterior facial height, greater lower anterior facial height, and less posterior facial height; and

3. Neither the magnitude of the pretreatment open bite, the mandibular plane angle, nor any other single parameter of dentofacial form proved to be a reliable predictor of posttreatment stability.
Interestingly, these authors found that the lower incisor vertical position was depressed in the relapse cases at all time periods studied which may indicate a possible influence of the tongue in the open bite relapse due to a pre-existing forward tongue posture.

Cooke (1980, 1981) classified anterior open bite into the following groups, adding that a single case may fall into more than one category:

1. **Skeletal** open bite (Fig. 152);
2. **Habit (dental)** open bite (Fig. 153);
3. **Abnormal tongue function** (Fig. 154);
4. **Iatrogenic** open bite (Fig. 155); and
5. **Pathological** open bite (Fig. 156).

Cooke described **Skeletal** open bite as having the following "classical features": only the posterior teeth in contact; a wide symmetrical anterior opening, associated with an excessive lower face height, i.e., a high Frankfort mandibular plane angle; a short ramus length; marked antegonial notch; and "a tendency toward a Class III malocclusion and a bilateral posterior crossbite".

The incisor teeth, he mentioned, erupt to their full potential but due to the skeletal imbalance between the dental bases, "even supraeruption is inadequate to achieve inter-incisor contact". Citing the work of Nahoum, he drew attention to the finding that in most cases it was the growth of the **upper face** which was deficient, and that there was a "diagnostic upward cant of the palate, which further increased the imbalance within the anterior part of the intermaxillary space". He mentioned also that these open bites were "essentially hereditary in origin" and "rarely treatable by conventional orthodontic means".

The **Dental** open bite due to habit activity he said, "represents the largest group seen in orthodontic practice". He described the characteristics
Fig. 152  Skeletal open bite.  (Cooke, 1980)

Fig. 153  Dental (habit) open bite.  (Cooke, 1980)
(asymmetrical open bite, localised to a few anterior teeth, and fitting snugly
around the offending agent - usually a thumb) and noted that it was digit
sucking which was responsible on the whole. Some cases were simple
deformations, others were more involved (Fig. 153). Also there may be a
combination of skeletal and habit features involved. Elimination of the habit
would then lead to a more precise diagnosis of the skeletal problems.

"Abnormal Tongue Function" open bites tended to show "seemingly
large tongues 'flopping forward' between the dental arches". As far as
causation goes, Cooke quoted the work of Gershater (1972) who, as mentioned
previously, found a high incidence of anterior open bites among his survey of
intellectually disabled and emotionally disturbed children. "This", Cooke said,
"supports other studies showing the open bite tendency in mongols, cretins, and
children with neuromuscular disabilities, where there are problems 'in
controlling the tongue at rest or in function". He suggested that this evidence
indicated that in some cases "the aberrant tongue is the actual cause of the
open bite". Cooke also cited Tulley's (1969) observations that there were
cases where the tongue played an overwhelming role in causing the open bite.
These were due to "congenital anomalies of tongue behaviour" termed
"endogenous tongue thrusts". The signs of these being: (1) A tendency by the
patient to lisp; (2) An anterior open bite larger than would be expected with a
tongue to lower lip anterior oral seal; (3) Excessive muscular activity around
the lips when swallowing, "the descriptively termed 'bag of wriggling worms'
appearance". Cooke showed a case (Fig. 154) where it was thought that a
"hyperactive tongue" was the cause of the open bite. Due to the resulting
closure of the open bite after a lower fixed lingual tongue crib and lip
exercises were used, he concluded that this had been an open bite "due totally
to tongue activity".
Fig. 154  Open bite due to abnormal tongue activity. (Cooke, 1981)
On the other hand, Cooke made mention of the tongue's "adaptive role" as opposed to its causative one. He pointed to studies involving measurement of lingual vertical forces, incidence of spontaneous correction of open bites, tongue size and age, lip seal incompetence, and disturbed nasal respiration. From these it was intimated that the exact relationship between tongue function as cause and tongue function as part of an overall adaptive pattern was not clear.

In iatrogenic open bite, orthodontic treatment had led to an open bite or increase in open bite due to misapplication of treatment techniques.

Pathological open bites included cleft palate, acromegaly, and bilateral mandibular condylar fractures.

In an incidence study Rogers (1961) found that of a sample of open bite subjects from a normal population, 98% showed tongue thrusting; and of a similar sample from an orthodontic practice, 92% showed tongue thrusting. He found that these figures lent support to the premise that the tongue thrust is not only responsible for the original open bite condition but also may be the most potent factor in its recurrence after correction.

The often quoted study of Worms, Meskin, and Isaacson (1971) found a significant decrease in anterior open bite with age. In a study of 1,408 Navajo children they found that there was a spontaneous correction of 80% of the anterior open bites as they proceeded from the 7-9 year age group to the 10-12 year age group. However it must be noted that throughout the five age groups studied, there was considerable variation in frequency of open bite. These authors found that open bite malocclusions "represent, at least in part, a dynamic condition subject to change". They attributed this self-correction to
Fig. 155 Iatrogenic open bite: a, lower incisor crowding. b, after extraction of lower incisor and lower fixed appliance. (Cooke, 1981)
Fig. 156 Pathologic open bite: bilateral mandibular dislocation of three months duration. (Cooke, 1981)
the maturation of the swallowing reflex, transition from the mixed dentition to the permanent dentition, and skeletal growth. A link between their findings and transitional swallowing patterns was suggested by Stanley and Lundeen (1980). The large decrease in frequency of open bite in the 10-12 year age group they equated with their findings of a decrease in the frequency of tongue thrust at ages 10 and 12 years.

However, Hanson (1975) cautioned against the use of non-longitudinal data in making inferences about a decline in the incidence of open bite. He said "only in a longitudinal study, wherein the same children are seen from year to year, can valid statements be made concerning changes in the malocclusion".

Proffit (1977) drew attention to inherited tendencies in open bite. Citing U.S. Public Health Service statistics (Fig. 157) he pointed out that open bite was largely a problem for the black population, and that these findings were "innate characteristics of these population groups, and not just related to their environment".

4.2.2.2 Lingual Cause/Effect Relationships

(a) Anterior Oral Environment

Frankel and Frankel (1983) found from their clinical observations that an open bite could be closed without using any device which interferes with tongue movement or tongue posture. From this they suggested that tongue thrust alone may not be the primary cause of that malocclusion and that there may be a functional relationship between the postural behaviour of the tongue and lips.
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1. Any open bite
   Total   | 5.7% | 5.2% |
   White   | 4.0% | 3.5% |
   Black   | 16.3%| 16.3%|

2. Open more than 2mm
   Total   | 2.5% | 1.1% |
   White   | 1.4% | 0.5% |
   Black   | 9.6% | 5.7% |

Fig. 157 Table: Incidence of open bite (U.S.A.) (Proffit, 1977)
Ballard (1965) suggested that in an evaluation of habit behaviours and their clinical relevance, more attention should be paid to the size and shape of the soft tissues. He stated that each case must be judged in relation to the disproportions in the facial skeleton and, from his clinical observations, concluded that tongue thrust as the major cause of open bite had been overemphasised. He argued that the faulty interdental posture of the tongue appeared to be a compensatory or adaptive behaviour which established an anterior oral seal when the lips were incapable of so doing.

Kydd, Akamine, Mendel, and Kraus (1963) in a study of lip and tongue forces in anterior open bite subjects (tongue thrusters) and subjects without an anterior open bite, found a basic difference. It was in the duration of lip and tongue pressures applied to the anterior teeth. Anterior open bite subjects, i.e., tongue thrusters, applied both tongue and lip pressure against the anterior teeth for a longer duration than did the subjects without an anterior open bite.

Neff and Kydd (1966) believed that the presence of the tongue between the teeth alone is not enough to induce an open bite, but that there had to be an active force applied around a relatively passive tongue to produce an open bite distortion. The active force, they suggested, came from the muscles of mastication used during swallowing.

Frankel and Frankel (1983) suggested that in the presence of a hyperdivergent skeletal pattern the entire splanchnocranium is affected, "in addition to the deviant vertical proportions below the palatal plane, a characteristic feature present in every case, the middle face is affected as well".

They also suggested that poor postural performance of the orofacial muscles was a characteristic feature of skeletal open bite and placed great
emphasis on the re-establishment of the anterior oral seal in regulating the postural activity of the orofacial musculature. They related this importance to the work of Bosma (1975) who stressed the functional interrelationship of faulty postural performances with spatial disorders in the oronasopharyngeal spaces.

Proffit and Mason (1975) proposed that in open bite cases where it seemed that undereruption of incisors was a feature, the vertically directed forces of the tongue might play a causal part. However, pointing to the work of Wallen (1974), it was found that no increase in vertical tongue force was found during swallowing. They thus proposed that resting tongue posture was responsible.

Lowe (1980) found that undererupted incisors were related to low thresholds in the genioglossus muscle. Thus anterior open bite malocclusions "may be due to the impeded eruption of anterior teeth as a result of a forward tongue posture". He suggested a possible sequence leading to the development of an anterior open bite malocclusion: firstly, an initial factor "probably related to airway maintenance", which involves a mandibular rotation, a resultant forward tongue posture in order to maintain the airway, and the eventual appearance of an open vertical dimension between the incisal edges of the maxillary and mandibular teeth.

Frankel (1980a) suggested that tongue thrust seems more likely to be the effect rather than the cause of an open bite malformation, "the tongue is a very adaptive organ, and its position between the maxillary and mandibular incisors in the presence of an open bite seems to be an adaptive function". He attributes this adaptive function to the necessity of the tongue to seal the anterior gap when the lip musculature is too weak to produce an anterior seal.
His success in treating open bite cases with function regulators is due, he suggested, to the re-establishment of an anterior lip seal and at the same time not trying to directly influence the tongue. This, he suggested, was evidence that the tongue can hardly be the primary factor in causing an open bite. He regarded this as a secondary causal factor "in the sequence of events causing physiological disorders in the orofacial complex".

Again Frankel cited his treatment results achieved in Class II and skeletal open bite to support the concept that when the orbicularis oris is not capable of producing a competent lip seal, other orofacial muscles must operate to compensate for this failure. With reference to the findings of Simpson (1976) he described how the activity of the mentalis and of the suprahypoid muscles increased when, in the presence of an incompetent lip seal, the lips were approximated. This activity of the tongue resulted in a postural change of the tongue anteriorly and was considered to be a tongue thrust. He regarded therefore the suprahypoid activity as being a "'compensatory function' more than a bad habit".

In another paper (1980b) Frankel described how with the use of "lip seal training" only, i.e., without appliances, there were "morphological alterations in the dentition and skeleton" in cases of open bite malocclusions. He found that these alterations were improvements. This reinforced his view that the tongue "cannot be a primary factor in causing open bite" and further, that "the postural pattern of the lip and perioral musculature is the major essential factor in the establishment of physiological conditions in the whole of the orofacial area".

Barber and Bonus (1975) studied a group of 31 tongue thrusters with anterior open bite, and 9 with normal occlusion and no tongue thrust. These
children were given programmes of circumoral muscle exercise and the effect on the open bite, and protrusion of the anterior teeth was noted. They found that the children exhibiting tongue patterns, had weaker or more incompetent circumoral muscular strength than did the non-tongue-thrusting (normal swallowing) children before any exercise programme was initiated. Also they found that the circumoral musculature was strengthened through exercise in the tongue-thrusting children, and the amount of muscle strength directly increased in proportion to the number of days of exercise. The increased strength after the periods of exercise remained nearly the same after 18 months. However, they found no change in dental relationships after the increase in muscular abilities throughout the 24 month study period. They concluded that increasing of the muscle strength of the lips does not produce any great effect on the dentition of children with tongue thrust and an anterior open bite with protrusive maxillary incisors. The inference, as discussed, was that the tongue still was too much of an influence on the dentition. Parallels could be drawn between this study and the clinical findings of Frankel (1980b). However, whereas Barber and Bonus' patients received lip strengthening exercises, Frankel's patients received lip seal training, although the method was not stipulated. Frankel, though, has claimed success in changing dento-skeletal relationships.

(b) Posterior Oral Environment

Frankel (see Watson, 1982b) mentioned that many authors believed that a disparate increment of vertical growth at the condyle and the alveolar process is the essential factor in causing mandibular rotation or vertical disproportions in skeletal growth, thus likely to be significant influences in skeletal open bite. However, he suggested that it must be considered that condylar and alveolar growth are not interstitial or expansive, but rather, appositional and adaptive in nature and dependent on the postural performance
pattern of the suspensory musculature of the mandible, dental eruption, and the interrelated feedback mechanism.

Subtelny (1970) identified what he thought to be "inadequate adaptations" where skeletal relationships are poor and possibly beyond the limitations of correction. He noted the features of "skeletal open bites": steep mandibular planes, short posterior vertical ramal heights, divergent gonial angles, tipped-up maxillary palates, and open bite malocclusions, "which may not be readily amenable to conventional orthodontic correction". However, he noted that there may be many "aberrant skeletal relationships" that are not quite so severe. It is here that "abnormal tongue posture and activity" may be associated, and correct diagnosis could better lead to more successful treatment. He described how the tongue could be "the victim of an unfavourable environment"; change of the environment, he believed, would alleviate this problem.

Further to this, Gershater (1972) determined that inherited patterns have a decided influence on the growth and development of the orofacial structures, but adverse environmental factors can markedly exaggerate the open bite deformity.

He was one of the very few authors to discuss the site of open bites, saying that it depended on which forces predominate and the ability of the teeth and supporting structures to resist change. "If, for example, there is an abnormal swallowing pattern and the strong propelling forces of the tongue are directed forward, the chances are that there would be a tendency toward an anterior open bite. Also, the presence of pernicious thumb or finger sucking, lip and mouth breathing habits, and poor labial musculature would greatly influence the severity of the anterior open bite". Conversely if there are no
habits and the forward tongue forces are counteracted by strong labial musculature, and the upper incisors and supporting structures resist changes then "the chances of causing an anterior open bite are greatly diminished". He suggested that the "abnormal tongue forces" are diverted elsewhere as a result. This may lead to lateral open bite or an incisal edge to edge relationship. He also noted, "many of the etiological factors and structural changes are similar in all types of open bite, and practically all cases have some degree of abnormal swallowing behaviour".

Arvystas (1977) reported on a number of skeletal open bite cases and suggested that in one of the cases the cause could be attributed to the patient's tongue thrust behaviour. He felt that the constant forward posturing of the tongue might have increased the overjet and open bite, with the peripheral portions of the tongue no longer lying over the lingual cusps of the buccal segments. The resultant eruption of the posterior teeth could then have eliminated the interocclusal clearance with the open bite worsening.

Mills (1982), in contrast, differentiated between types of tongue thrust. He described the "endogenous" tongue thrust, as opposed to the "normal secondary type of tongue thrust", as being able to produce an anterior open bite "if powerful enough". He also presented a case, in which attempted orthodontic correction of the malocclusion was unsuccessful due to the action of the tongue.

Speidel, Isaacson, and Worms (1972) proposed that tongue thrust was an adaptive influence on an already compromised skeletal pattern in anterior open bite cases. The main feature of the open bite subject was the backward rotating mandible and thus the increased growth of the lower face. Of tongue thrust as a cause they said: "In the rare case of an open bite persisting after
10 to 12 years, and when such open bites are not present on backward rotating skeletal patterns, one may consider the primary etiologic factor to be extrinsic, possibly abnormal tongue activity".

Thurow (1975) proposed that open bite could be a secondary effect due to tongue position as a consequence of a decrease in size of the airway. This could develop at an early age, or may develop with growth changes, or in the course of orthodontic treatment.

In discussing a case study of a patient with open bite complicated by clefts of the maxilla and mandible, Lubit (1976) stressed the importance of establishing normal tongue posture. Posttreatment cephalometric tracings revealed a forward (counterclockwise) rotation of the mandible after treatment which was instrumental in correcting the open bite. This, Lubit said, was the important factor in the correction of the open bite. How was it achieved? According to this author it was a normal growth response to an improved functional matrix. According to Lubit "skeletal open bites are primarily due to a clockwise rotation of the mandible as a result of altered tongue position".

Citing the work of Barrer (1974), Lubit stated that the rest position of the mandible was reasonably stable throughout an individual's growth period. This being the result of the balance established between the forces of gravity and those tissues which support the mandible and maintain a patent airway; "unless this balanced functional matrix system is altered, we cannot permanently change the mandibular rest position by orthodontic means". In this case "establishment of normal tongue position and function by maxillary expansion, change in arch form, surgical release of the tongue and myofunctional therapy created a more normal functional matrix". The change in this patient's facial growth was attributed to "an effective functional
matrix plus the ability to express her full growth potential".

Dubner, Sessle, and Storey (1978) suggested that the extent to which the tongue contributes to the creation of an open bite is probably related to its postural activity, citing the work of Harvold (1968). Harvold found that he could produce an open bite in several months by placing a piece of acrylic permanently in the dorsal palatal vault of monkeys. He concluded that crowding of the tongue resulted in jaw-opening and forward posturing of the tongue. These results were substantiated by the later work of Lowe (1980). Harvold found that the jaw-opening and tongue protrusion were conjoint responses to an infringement on the pharyngeal airway. Dubner et al. pointed out that the reflexes protecting the pharyngeal airway can be initiated by natural causes such as enlarged or inflamed tonsils, large tongue, small maxillary or mandibular dental arches, or by the insertion of bulky appliances, or by appliances restraining the tongue. They stated that appliances that crowd the tongue may be contraindicated where interception further jeopardises the pharyngeal airway.

Another point that these authors have made concerns the nature of jaw, facial, and tongue reflexes in relation to the previous points of discussion. It has been found that different stimulus sites may be determining factors on which tongue muscles are activated. Laryngopharyngeal and temporo-mandibular joint sensory information is primarily associated with genioglossus activity, the main protrusive tongue muscle. These authors relate this association with airway maintenance and mention it "as being of possible etiological significance in certain orthodontic problems such as anterior open bites".

It seems then that by applying the cephalometric criteria of Subtelny
and Sakuda, Nahoum, and Cingialosi, whether an open bite malocclusion with tongue thrust is skeletal or dentoalveolar in nature can be determined. It appears that a dentoalveolar open bite can be influenced primarily or secondarily by tongue thrust. Whether a skeletal open bite can be caused primarily by a tongue thrust remains to be seen. However, there appear to be definite links between airway function and tongue posture which could be directly associated with skeletal open bite due to muscle function principles and growth dynamics.
4.2.3 Nasorespiratory Function

This discussion has made direct and indirect reference to the significance of adequate nasopharyngeal function in the lead-up to this section. As Rubin (1980) has pointed out, articles pertaining to this topic first appeared over a hundred years ago. However, with the advent of cephalometrics and mechanotherapy, the study of airway adequacy and its impact on craniofacial development waned. Only in the last 25 years has renewed interest been generated. The ensuing discussion will deal mainly with the more recent developments in the study of nasopharyngeal function, since there appears to be more and more interest in this field of study.

4.2.3.1 Nasal Airway: Significance and Maintenance

Thompson (1917) was one of the earliest authors to review the biological significance of changing head orientation in relation to the cranial base and the pharynx. Gwynne-Evans (1948, 1951), as well, traced the biological development of the oronasal airways in relation to orthodontics.

Brodie (1971) also stressed the importance of the airway for the purpose of survival. From an evolutionary perspective Brodie traced the development of mammals from the quadruped stage to the erect posture of Man. The flexion of the junction between head and body which changed from a horizontal, straight line to a right angle was stressed. He described the changes that occurred as a result of the flexing of the head to almost a right angle bend to the vertebral column. All neck structures suspended from the mandible in front, i.e., tongue, hyoid bone, and larynx, fell back; the tongue, previously long and flat, was forced into a "ball-like" structure in the shortened muzzle and crowded back into the pharyngeal space. Radical adaptive changes were needed to re-establish the airway relationships. The mandible broadened to provide more room for the throat structures, and the
chin developed. Jaw movements changed to permit the hyoid bone and larynx to remain almost motionless even during mandibular movements, assuring stability for the base of the tongue. As well, the seal allowing nasal airway patency, formed by the soft palate and epiglottis was now replaced by the soft palate to tongue seal, "for the first time the tongue became a threat to the airway". The adaptive response was the development of reflex control of the hyoid bone and strengthening of the protrusive possibilities of the external pterygoid muscle. Brodie, further, discussed a case of a boy with tongue tie who protruded the mandible in order to open the airway. After the lingual frenum was relieved the tongue could position forward, freeing the airway and the mandible dropped back into a Class II relationship. The boy had been protruding the mandible up until then to improve airway patency.

Bosma (1963) had made similar observations and stressed the relationships of pharyngeal airway, head posture, and neck posture. The pharyngeal airway was found to be greater in diameter with the head and neck in the extended position.

Timms (1965) also described the maintenance of airway patency as being of very high biological significance. Neurologically, the airway reflexes will predominate if there is competition among reflexes for the same neural pathways.

Moyers (1973) stressed the necessity for the neonate to maintain the airway from the first day of life. He emphasised the role of the "oro-jaw musculature" for the vital positional relationships maintaining the oropharyngeal airway. The muscles function to maintain the diameter of the airway by: (1) maintaining the mandible anteroposteriorly; and (2) stabilising the tongue base and posterior pharyngeal wall relationships. The cervical
muscles are also involved as "these primitive neotatal protective mechanisms provide the motor background on which are developed, with growth, all of the postural mechanisms of the head and neck region".

Schulhof (1978) extended Moyers' comments by noting that mouth breathing, i.e., upper airway impairment, was significant in being an "obstacle" to successful orthodontic treatment.

As mentioned previously Thurow (1975) emphasised the importance of the nasal airway. He also stressed the significance of the anatomical region of the lower pharynx. He pointed out that in nasal breathing the smallest cross-section of the airway may be found at the lower pharynx, the nasopharynx, in the nasal cavity, or at the anterior nares. The only one of these potentially limiting constrictions which cannot be bypassed by opening the mouth is the lower pharynx. He related this aspect of respiratory function to survival of the individual (as the other authors have done). If there is a threat to respiratory function, he suggested, then various compensatory adaptations would have to come about, since a patent airway is the most essential aspect of an individual's survival. A restriction of the intake of air in this region, the lower pharynx, by enlarged tonsils in particular, was believed to be responsible for a change in position of the mandible to allow for an opening up of the pharyngeal airway. The importance of lessening the functional load on the geniohyoid muscles was also identified, so that the hyoid bone could be held forward thereby keeping the tongue from dropping back into the airway.

4.2.3.2 Nasopharyngeal Airway: Examination, Maturation and Development

Before examining the nasopharyngeal structures it is worthwhile noting the region known as the "adenoid pad" (Lanier and Tremblay, 1979) and in particular Waldeyer's ring. Both the tonsils and the adenoids are part of a
collection of lymphoid tissues known as 'Waldeyer's ring'. This tissue encircles the posterior pharynx and consists of lymphoid tissue at the base of the tongue (lingual tonsil), the two faucial (palatine) tonsils, and the pharyngeal tonsils or adenoids (Fig. 158).

**Examination:** Working from the most superior end of the airway, Rubin (1979) described the methods of examination of nasal and pharyngeal structures. A panoramic head plate can be used to detect and assess a deviation of the nasal septum. A tomograph at the level of the central incisors is used. Direct examination with a nasal speculum is also essential, showing that deviation is invariably accompanied by hyperplasia of the turbinates on the concave side. He described the "lateral head plate" as being very valuable in evaluating tonsillar and adenoid tissue (Figs. 159, 160).

Handelman and Osborne (1976), Schulhof (1978), and more recently McNamara (1984) are among the various authors who have measured and plotted the dimensions of the oronasopharynx from lateral cephalic radiographs.

As well, Holmberg and Linder-Aronson (1979) developed a five-index scale for evaluating, on lateral cephalic radiographs, the size of adenoids. In a study performed on children aged 6-12 years, they demonstrated that lateral and frontal skull radiographs provide a satisfactory means of evaluating the dimensions of the nasopharynx and the capacity of the nasal airway, respectively.

More recently (Tsui, 1980), computed tomography has been utilised to assess pharyngeal airway dimensions. Browning (1982), an otorhinolaryngologist, has described the use of (1) two bimanual spatulae to retract the oral structures and a headlight, with or without the aid of a postnasal
Fig. 158  Waldeyer's Ring is composed of tonsils (T), adenoids (A), and lingual tonsil (L)  (Lanier and Tremblay, 1979)

Fig. 159  Panoramic radiograph showing markedly deviated nasal septum.  (Rubin, 1979)
Fig. 160  Lateral headplate showing extensive adenoid tissue and tonsils. (Rubin, 1979)
mirror, and (2) endoscopy under general or local anaesthesia, to view the pharynx and hypopharynx. Similarly, Mulder (1976) described the use of nasendoscopy or more recently "pharyngoscopy", and electromyography, to view and to study the upper pharyngeal morphology and function.

Another method of airway examination other than by rhinoscopy or endoscopic techniques is by measuring the volume of air passing through the upper airway structures. Mouth breathing is seen as a consequence of nasal airway inadequacy. Via 'ororhinomanometry' authors have attempted to measure the amount of air passing either through the nose and/or the mouth. Vig, Sarver, Hall, and Warren (1981) quite reasonably sought to define 'mouth breathing'. Their studies suggested that most people fall between the two extremes of 'mouth breather' and 'nasal breather', which was no surprise. But, they failed to determine what value of oral to nasal respiration ratio is of etiologic significance to malocclusion.

In an earlier study Watson, Warren, and Fisher (1968) found that the incidence of clinically observable mouth breathing was found to be greater among the subjects with a nasal resistance above 4.5 cm H2O/litre/second. The subjects with a nasal resistance above this level were observed to have had either "a deviated nasal septum", "injection of the turbinates", or "allergic rhinitis". Those subjects exhibiting mouth breathing but with a resistance below the 4.5 level were believed to have been doing so as a result of habit.

Recently Warren (1984), unhappy with the clinical assessment of nasal airway impairment, claimed to have produced a method of quantitatively estimating nasal airway dimensions so that normal and impaired naso-respiratory function could be defined. He suggested that by his technique, which involved sophisticated technological equipment, and which might be seen
as impractical in the clinical situation, one could: (1) estimate the size of the airway during breathing; (2) distinguish between normal and impaired nasal respiratory function; and (3) determine quantitatively the effects of surgical and/or orthodontic treatment for improving nasal respiration. Warren hoped also that by this technique a method of predicting when the breathing mode will change from nasal to predominantly oral would be found.

Linder-Aronson (1983) had just previously asserted that clinical examination establishing the mode of breathing was "still more accurate than any objective measuring technique available today". Whether Warren's (1984) technique has changed this view remains to be seen. According to Principato (1985) physical examination performed in conjunction with several clinical investigatory studies can accurately evaluate the nasal airway; "in many patients with nasal obstructive phenomenon, this evaluation can provide specific etiology and location of the pathophysiological process". Principato stresses the "objective determination of nasal airway flow and resistance", adding that "values reflective of normal airways are being compiled for various ages".

Maturation and Development: Subtelny (1954) studied adenoid tissue cephalometrically. He noted a cyclic nature of growth, relative to the surrounding structures, from birth to maturity, growing from a small mass in infancy to a large mass towards the onset of puberty, then a diminution of the size towards late adolescence. He also noted that the development in size of excessive adenoid tissue can be due to allergic reactions, a reaction to infectious agents resulting in hypertrophy, or abnormal growth.

In a review of tonsil and adenoid development Ardran and Kemp (1972) discussed the growth, and necessity, of epipharyngeal lymphoid tissue from the
newborn stage, the 1-5 years infant stage, to the adult stage. They concluded that due to the varying anatomical influences associated with normal development from birth to adulthood: (1) Adenoid and tonsillar masses were not necessary for adequate tongue, soft palate, and pharyngeal function in the newborn; (2) With the descent of the face in infancy to the pre-pubertal period, these lymphoid masses are necessary as space fillers for normal function; and (3) Towards maturity, as adult anatomical proportions are being reached, the need for space filling for proper function is no longer required and atrophy of adenoid and tonsillar tissues is observed.

Handelman and Osborne (1976) found that during the pre-school and primary grade years the adenoid area increases more than the bony nasopharyngeal area, resulting in a restriction of the air space.

Most recently Linder-Aronson and Leighton (1983) studied adenoid tissue development (epipharyngeal lymphoid tissue) and the airway. It had previously been accepted that epipharyngeal lymphoid tissue followed the growth curve of Scammon (1930) for the lymphatic cycle. These authors found that the growth of the adenoids and of the soft tissues on the nasopharyngeal wall do not follow Scammon's lymphatic growth cycle, i.e. attaining maximum bulk between the ages of 9 and 15 and showing subsequent atrophy. Scammon's curve was developed for lymphoid tissue of the appendix, spleen, and thymus and did not include measurements of the tonsils and adenoids.

Linder-Aronson and Leighton found that:

(1) The thickness of the soft tissue on the posterior nasopharyngeal wall is largest at 5 years and subsequently decreases until 10 years of age. There is a slight increase between 10 and 11 years of age and then the decrease continues; and
(2) The sagittal nasopharyngeal airway is narrowest at 5 years of age, increasing between 5 and 10 years and then again after 11 years of age.

4.2.3.3 Nasopharyngeal Airway Obstruction: Causes

With regard to orthodontics, Ricketts (1968) described the factors that can lead to an obstruction of the normal respiratory airway. He divided them into two main groups: (1) General skeletal characteristics as a structural frame; and (2) Soft tissue and local conditions. Under the first group he included: a naturally small nose (nasal atresia or natural impatency), the choanal opening and piriform aperture being too small to permit air flow; abnormal mandibular growth causing vertical maxillary asymmetries; dysplasia of the cranial base, e.g. cranial dysostosis; and extreme deflection of the cranial base angles. Included in the second group were: upward and forward adenoid tissue, especially when the inferior turbinates are long; chronic rhinitis; recurrent upper respiratory infections; allergies; asthmas; polyps; foreign bodies; deviated septa; unreduced fractures; and ambitious surgical treatment of cleft palate conditions.

Bluestone (1979) has produced a classification of respiratory obstruction due to tonsils and/or adenoids. It attempts to quantify the amount of obstruction encountered in the clinical assessment of a patient. Bluestone identified the site of the obstruction - adenoids in the nasopharynx, tonsils in the oropharynx; whether the obstruction is intermittent or persistent; and the degree of obstruction - mild, moderate, or severe.

Gershater (1972) listed the three indications for the surgical removal of the tonsils and adenoids according to Steele (1968): (1) Repeated attacks of tonsillitis; (2) Hypertrophy of tonsil and adenoid tissue to the extent of destruction to the Eustachian tubes and nasal or oral airways; and (3) Evidence
that the tonsils harbour chronic infection. To these he added: (4) Large hypertrophied tonsils and adenoids "which can adversely influence the growth and development of the orofacial structures which may result in a poor cosmetic facial pattern". He believed that a potential open bite malocclusion could often be avoided by the proper handling of the tonsil and adenoid problem in the early stages of growth and development.

Ardran and Kemp (1972) suggested that large tonsils could be responsible for displacing the soft palate upwards, leading to a narrowing of the nasopharyngeal cavity and airway obstruction, especially if adenoids are a permanent feature.

Thurow (1977) suggested various causes of obstruction to incoming air: acute or chronic nasal blockage, adenoid enlargement, faucial tonsil enlargement, or tongue encroachment on the lower pharynx. He described each segment as having its own problems when obstruction occurs. (a) **Nasal** airway obstruction: This may be a temporary inflammatory condition, or it may be a long-term blockage caused by anatomic deviations of the nasal cavity or by adenoid enlargement. He identified mouth breathing as the immediate response to any obstruction of the nasal airway. However, it may have the secondary effect of causing inflammation of oropharyngeal tissues which in turn could lead to a perpetuation of the mouth breathing tendency. He described the chain of events leading to changes of mandibular loading and possible skeletal changes, which are discussed later. (b) **Lower pharyngeal** airway: Here adjustment of geniohyoid function must take place, thereby affecting tongue base positioning. However, he described this kind of obstruction as being of a basic anatomical kind and "beyond the reach of therapy". (c) **Tonsils**: Lying on both sides of the nasal and oral passageways at the top of the lower pharynx, enlargement of these tissues could cause a
variety of responses. Fauclial tonsil enlargement may be acute or chronic, painful or painless. He described the response to simple painless enlargement as being some combination of mouth breathing and advancement of the tongue (Fig. 161).

Rubin (1979) divided the etiology of nasal airway obstruction into: (1) congenital; and (2) acquired or developmental. **Congenital**: including choanal atresia either bony or membranous, and hypoplastic external nares. **Acquired and developmental**: (a) deviated septa, (b) extensive nasopharyngeal lymphoid tissue, (c) perennial allergic rhinitis, (d) neoplasms, (e) polyp formation, (f) trauma, and (g) iatrogenesis. Rubin identified **allergic rhinitis** as being the most common cause of nasal airway obstruction, the treatment for which is medication, removal of allergens, and desensitisation. If a deviated septum is the cause then surgery is indicated, often a partial turbinectomy is necessary. If lymphoid tissue is the cause then adenotonsillectomy is indicated. Rubin also pointed out the use of rapid maxillary expansion as being able to increase the capacity of the nasal airway.

In relation to allergic conditions, cow's milk has been implicated by some (Marks, 1965; Rubin, 1979; Penzer, 1980) as causing adverse reactions at the infant stage. It is claimed to "trigger allergic reactions, edema of the mucous membranes, enlargement of adenoids, and obstruction of respiratory passages" (Penzer, 1980) in some infants introduced to cow's milk too early.

Further, Hannuksela (1983) found atopic disease (seasonal or perennial allergic rhinitis, bronchial asthma and atopic dermatitis) frequently associated with mouth breathing and nasal obstruction.

Sosa, Graber, and Miller (1982) identified some of the multiple causes of mouth breathing: (1) narrow nasal passages and a narrow or occluded
Fig. 161  A, tongue during normal swallowing. B, abnormal swallowing due to hypertrophied tonsils. As the tongue is retracted, it touches inflamed and swollen tonsils. Pain causes dropping of the mandible, so the tongue can thrust forward away from the pharyngeal region. With the mandible lowered, the lips must be closed forcibly to keep the tongue in the oral cavity. (Moyers, 1973)
nasopharynx; (2) swollen nasal membranes; (3) enlarged turbinates; (4) adenoids of the nasopharynx; (5) deviated nasal septum; (6) choanal atresia; and (7) tumors of the nose or nasopharynx. Importantly, again, they identified mouth breathing as being "habitual without the presence of nasal obstruction".

Likewise Jonas, Schlenter, and Mann (1983) claimed that mouth breathing in children can be a habit or a consequence of nasal airway inadequacy. They also listed similar causes of nasal airway inadequacy in children. Their studies of the effectiveness of oral screen treatment revealed that nasal air flow rate can be greatly improved in patients with a large nasal cavity and congestion of the turbinates.

4.2.3.4 Dentoalveolar and Craniofacial Changes Associated with Airway Obstruction

Angle (1907) was one of the early authors to condemn nasal obstruction and mouth breathing due to what he believed as its "evil" and "baneful" effects on the dentition, as well as other facial structures. He described the typical mouth breather as having the mouth held open "almost constantly"; the upper lip as being short and drawn up in the effort to breathe; the mandible drawn back and also failing to develop "being almost always smaller than normal, although usually regular in form": the upper arch, becoming narrower than normal, and usually lengthened, due to unequal muscle pressure; and the tongue, "being made to rest between the lateral halves of the lower arch".

Gwynne-Evans (1948) believed that mouth breathing, and the muscular and dentoalveolar positional associations were not necessarily associated with airway obstruction as such, but with a genetically determined predisposition to these features.
Tulley (1952) was not convinced that there was a causal relationship between upper respiratory obstruction and malocclusion, although he did observe that upper respiratory disease was often associated with a Class II division 2 "type" of malocclusion.

Hovell (1955) found that the "adenoid facies" appearance, basically that which Angle (1907) had described, was necessarily associated with mouth breathing or upper respiratory obstruction. He believed that lip incompetence could be responsible.

Various authors have investigated the 'adenoid facies' associations. Rubin (1979) outlined the cases for and against adenoid facies being of interest to the orthodontist. Ricketts (1968) and McNamara (1981b) were others to address the issue, as well as Marks (1955). He, an allergist, reviewed the arguments on both sides and concluded that nasal obstruction was a significant cause of altered facial growth. Korkhaus (1960) identified nasal obstruction and what he found as the accompanying malocclusion - narrow maxillary arch, high palatal vault, thus underdeveloped jaws and reduced function. Ricketts (1968) observed, on a clinical basis, a high frequency of collateral dento-alveolar and craniofacial findings "in the presence of adenoids and tonsils". He described a condition which was termed the "respiratory obstruction syndrome" which included some of the following characteristics: cross-bite; tonsil and adenoid enlargement; open bite; thrusting of the tongue on swallowing; mouth breathing; and functional cross-bites or functional protrusions.

Following his comprehensive studies, Linder-Aronson (1970, 1974) found that "adenoids and the mouth breathing which they induce are associated with a narrow upper arch, a high incidence of cross-bite or a tendency to cross-bite, retroclined upper and lower incisors, and small sagittal depth of the
nasopharynx. The narrow upper arch and higher frequency of cross-bite among children with large adenoids he attributed as being parallel phenomena to the narrow facial type of these individuals, along with a small nasopharyngeal cavity and narrow nose. He suggested that there may also be some causal relationship between the width of the upper arch and the mouth breathing and low tongue position associated with adenoids. The retroclination of upper and lower incisors in the mouth breathers was interpreted in the same study as a consequence of increased tension in the lip musculature due to the mouth being kept open.

A study by Paul and Nanda (1973) found that the associations of mouth breathing were mainly confined to maxillary arch dimensions. They were: (1) contraction of the maxillary arch; (2) increase in maxillary arch length; (3) increased overjet; (4) deep over bite; (5) the palate appeared high due to the contraction of the maxillary arch; and (6) a high percentage of Class II Division 1 malocclusions were seen.

Bushey (1979) produced a classification of airway obstruction and tongue/tonsil problems in orthodontics. As an aid to diagnosis and to assist treatment planning he grouped pharyngeal abnormalities, and skeletal variations together with soft palate dysfunction and tongue malposturing into six types.

Turning to the mandibular region, Thurow (1975) observed that a restricted airway was most commonly found with the type of facial form wherein the geniohyoid area there is a shorter span from chin to hyoid (Figs. 162, 163). Opening and closing movements of the mandible produce much greater proportional changes in this distance, requiring greater responses by the geniohyoid muscle. He suggested that when this is the case it places even
Fig. 162  Geniohyoid relations: flat mandible.  (Thurow, 1975)

Fig. 163  Geniohyoid relations: steep mandible.  (Thurow, 1975)
greater demands on geniohyoid function. Thus in the course of growth, mandibular opening is produced or enhanced.

Proffit (1978) believed also that respiratory needs influence head, jaw, and tongue posture. Emphasising these postural changes he suggested that if there is difficulty in nasal breathing, then physiological adaptations which facilitate mouth breathing include a forward positioning of the head on the neck, a lowered position of the mandible, and a low and forward tongue posture. Further to this he added; "It is not coincidence that respiratory difficulties are associated with long face syndrome". He cited experimental work with primates and observation of human subjects to support the concept that postural relationships are changed to meet respiratory needs; "Alterations in the growth of the jaws and teeth can be produced experimentally by requiring mouth breathing. Both Harvold et al. (1973) and McNamara (1973) have produced dentofacial deformities in monkeys in this way." Harvold (1972) had actually caused malocclusion in monkeys by artificially blocking off the nasal airway.

Posen (1972) noted changes in lip function and incisor position due to mouth breathing as a result of adenoid, tonsillar, and allergic influences. Unlike Linder-Aronson's (1970) findings on incisor position he found the incisors to be protruded as a result of impairment of normal lip function during mouth breathing leading to weak perioral musculature.

On this point, Rubin (1979) identified several alterations to the force system acting on the facial skeleton due to the mandible being lowered and lips parted to permit mouth breathing. They were:

(1) The tongue assumes a lower position in the oral cavity "denying the palate and maxillary alveolus their support";

(2) The lower lip drops away from the labial surface of the maxillary incisors.
(3) The mandible may fail to contact the maxilla during deglutition permitting unrestrained vertical alveolar development and posterior tooth eruption to occur; and

(4) The elevators of the mandible may undergo atrophy because of underutilisation. This may be compounded by edema and venous stasis if the cause of the airway obstruction is allergy.

The resultant findings may include: (a) long face, (b) anterior open bite, (c) high palatal vault, (d) gaping expression, (e) inflamed anterior gingiva, (f) steep mandibular plane, (g) Class II malocclusion, and (h) distress when lips are held together. The increased incidence of Class II malocclusion in mouth breathers, Rubin believed, is caused by the backward rotation of the mandible secondary to the elongation of the posterior alveolus and tooth eruption following a constant open-mouthed posture.

Bushey (1979a), after studying adenoid obstruction of the nasopharynx, suggested that because naso-respiratory obstruction may be partial or complete, mouth breathing may also be partial or complete, and "thus offers a continuum of physiologic adaptation related to a number of morphologic variations of the craniofacial complex".

Subtelny (1980) also studied adenoid tissue development. He related the association of growth of the nasopharyngeal region with adenoid tissue growth, pointing out that sufficient nasopharyngeal space must be maintained for the enlargement of the adenoid tissue. If there is insufficient growth of the airway structures then the enlarging adenoids could cause mouth breathing. This is significant at the pre-pubertal growth stage, since if mouth breathing is encouraged at this point there are more likely to be masked morphological changes since the jaws are growing at a rapid rate. He
suggested that more vertical facial growth may follow, with continued eruption of the posterior teeth increasing lower anterior facial height and increasing the potential for open bite.

Subtelny hypothesised that a viable nasal respiratory system may be necessary for ideal development of the nasomaxillary complex and mandible. As well as the nasomaxillary complex the maxillary architecture seemed to be affected. Clinically he found that there frequently seems to be some degree of maxillary retrusion, some degree of "anticlockwise" rotation of the palate, and some degree of reduction in maxillary width when nasal respiration is not possible.

McNamara (1981b) pointed out, however, that the features of the typical mouth breather, mentioned above, are not always present. He described a study of his of subjects requiring adenoidectomy. On average he found that these subjects were characterised by excessive anterior facial height and a steep mandibular plane. However, in reviewing various studies he felt that there was no one specific craniofacial pattern which could be directly correlated with mouth breathing. He suggested that the variety of skeletal and dental configurations observed in mouth breathing patients may be presumed to be the secondary results of the neuromuscular adjustments required to maintain adequate respiratory function. (This point is dealt with more fully further on).

Harvold, Tomer, Vargervik, and Chierici (1981) found that rhesus monkeys adapted to mouth breathing in different ways. All experimental animals gradually acquired a facial appearance and dental occlusion different from those of the control animals. They found changes in tongue morphology which were restored to normal subsequent to nasal breathing being restored.
The most common dentitional changes were an incisor cross-bite due to narrowing of the mandibular arch and a decrease in maxillary arch length. Overall they found that oral respiration could induce "mesioclusion, maxillary protrusion with distoclusion, open-bite, and dual bite". They also found that the oral muscle function was changed to allow mouth opening and some advancement of the mandible. The genioglossus and intrinsic tongue muscles were "rhythmically recruited for tongue protrusion and the lip elevator increased the mouth opening". The mouth breathers were found to have increased tonic activity in the tongue and in the upper and lower lips, as well as in the medial and lateral pterygoid muscles. The authors suggested that the increased tonic activity of these muscles and a specific change in jaw positioning may cause corresponding bone remodelling.

Bresolin, Shapiro, Shapiro, Chapko, and Dessel (1983) recently studied the relationship between mouth breathing in children with allergic rhinitis, "the most common cause of chronic airway obstruction in children", and dentofacial development. They found that these children demonstrated: larger upper anterior facial height and larger total anterior facial height; greater angular relationships of the sella-nasion, palatal, and occlusal planes to the mandibular plane; larger gonial angles; more retrognathic maxillae and mandibles; higher palatal height and greater overjet; narrower maxillary intermolar width; and a higher incidence of posterior cross-bite. Overall they found that mouth breathers had longer faces with narrower maxillae and retrognathic jaws. Hannerksela (1983) also found similar observations in atopic children, while Wenzel, Hojensgaard, and Henriksen (1985) in a study of children with bronchial asthma and perennial allergic rhinitis, found that the severely affected asthmatic children tended to develop retrognathic jaws, while altered dentoalveolar relations were seen in the asthma group compared with controls.
Warren, Lehman, and Hinton (1984) predicted that: (1) A nasal airway cross-sectional area of less than 0.4cm² may represent an inadequate airway in adults and some mouth breathing would be expected; (2) The amount of adenoid obstruction must be very large to affect airway resistance. However, if airway resistance in the nose is high, large adenoids would present a serious airway problem and cause predominant mouth breathing; (3) When nasal airway resistance is high, the mouth will open approximately 0.4 to 0.6cm². This shifts a significant amount of air orally and reduces airway resistance to a normal level; and (4) If morphologic changes are caused by airway impairment, other factors such as a large tongue, large tonsils, or a long, draping velum are probably significant contributing factors. They suggested that the reason for possible morphologic changes may be multifactorial and involve not only the nasal cavity and nasopharyngeal airway but, more importantly, the oropharyngeal airway as well.

The postnatal development of certain craniofacial structures seems closely associated with functions which ensure an adequate airway space. Bosma (1963, 1969, 1973, 1975) has described one of these functions; the muscle activities related to head and neck posture.

As mentioned previously, Ricketts (1968) described the tipping back of the head to increase the airway with respiratory obstruction. Recent studies on head posture include one by Vig, Showfety, and Phillips (1980) which showed that total nasal obstruction leads to progressive extension of the head. Removal of the obstruction resulted in a return to previous head posture. It also appeared that elevation of the head contributes considerably to the movement which results in the altered relation of the jaws.

A great deal of work has been done in this field by Solow and
associates (Solow and Tallgren, 1971, 1976, 1977; Solow and Kreiborg, 1977; Solow and Greve, 1979, 1980; Siersbaek-Nielsen and Solow, 1982). Their most recent findings (Solow, Siersbaek-Nielsen, and Greve, 1984) indicate that obstruction or reduced adequacy of the nasopharyngeal airway was associated with a lower craniocervical and cranovertical angulation. This is in agreement with the findings of Woodside and Linder-Aronson (1979), Vig, Showfety, and Phillips (1980), and Ricketts (1968). A large craniocervical angle, in turn, was seen in connection with small linear mandibular dimensions, mandibular retrognathism, and a large inclination of the mandible in relation to the anterior cranial base. These authors consequently contend that there exists a growth co-ordinating mechanism which regulates mandibular development to craniocervical angulation. Solow et al. have found that airway adequacy was related to the size and position of the mandible. Their findings overall then, suggest that mandibular development is related to both airway adequacy and craniocervical angulation. Wenzel, Hojensgaard, and Henriksen (1985) have also shown that the more severe the asthma (in their asthmatic subjects) the greater the extension of the head and the more retrognathic the mandible.

Huggare and Kylamarkula (1985) studied the consequences of altered head posture on the morphology of the first cervical vertebra, the atlas. In children with enlarged tonsils they found: (1) A change in the shape of the first cervical vertebra, i.e. the height of the atlas; (2) A more retrognathic mandible; and (3) An enlarged cranial base angle.

Linder-Aronson (1970, 1975) noted that the infrahyoid and post-vertebral muscles increased tension to maintain a stable posture, often rotating the head upward and backward, in cases of increased nasal resistance.
How close then are these associations of interrupted nasal breathing to craniofacial and dental maldevelopment? Linder-Aronson (1979) believed that only by longitudinal studies of before and after removal of the obstruction could the associations be more readily gauged. He studied a group of mouth breathing patients with hypertrophic adenoid vegetations before, and five years after, their adenoids were removed. He found that after adenoidectomy a wide range of skeletal and dental changes back to normal values occurred spontaneously. His results support the contention that disturbed nasal respiration can and does affect both the facial morphology and the dentition.

A critical review of the literature by O’Ryan, Gallagher, La Banc, and Epker (1982) cast doubt on the findings of those who supported the concept of airway obstruction causing changes in craniofacial and dental structures. A recommendation of theirs was that studies be carried out to quantify the relative amounts of oral versus nasal respiration. However, according to Linder-Aronson (1983) this article misrepresented the facts of previous research and provided a biased viewpoint, thus casting doubt on the reliability of their critical review. Moreover Linder-Aronson doubted the usefulness of quantitative assessments of respiration, saying that the current measuring techniques were not yet accurate enough.

Before turning to neuromuscular aspects, it is worth noting the observations of Sosa, Araber, and Miller (1982), who have revived the functional matrix concept of Moss (1968) in relation to nasorespiratory function. They point out that there is an assumption that the absence of nasal breathing may indirectly affect the form of the maxillary arch through interference with the growth of the upper facial skeleton as a whole and the floor of the nasal cavities locally. Quoting the longitudinal study by Linder-Aronson (1979), again they suggest that there is support for the relationship
between "epipharyngeal tissue excess and orofacial growth".

Miller, Vargervik, and Chierici (1984) found that the response of a longer vertical face height following nasal obstruction was associated with increased active discharge and use of the geniohyoid and digastric muscles, but not of the jaw-elevator muscles. The only other craniofacial muscles to alter their neuromuscular drive significantly were related to the upper lip and tongue.

Frankel and Frankel (1983) felt that postural performances of the orofacial structures should be seen in a functional interrelationship with spatial disorders in the oronasopharyngeal spaces. In particular they placed great emphasis on the influence of lip incompetence on the function of these caudal structures, saying that lip competence was necessary for normal nasopharyngeal function, and therefore for jaw and tongue posture.

In conclusion, Linder-Aronson (1983) has emphasised that the relationship between nasorespiratory function and dentofacial development is not a simple cause-and-effect relationship. Instead his studies have shown that a relationship does exist between mode of breathing and the morphology of the face and dentition and that there is a complex interaction between hereditary and environmental influences.

4.2.3.5 Tongue Position and the Influence of Airway Obstruction

Rix (1946) identified a possible causal relationship between upper respiratory dysfunction and tongue thrust. Again, Rix (1948) suggested that there may be an association between nasorespiratory obstruction, tongue thrust and malocclusion, the emphasis being on the contraction of the upper arch. He felt that children who had nasopharyngeal troubles very early, at a time
when the swallowing mechanism had not become fully specialised, were the ones most prone to continue with their infantile mechanism. He suggested also that "stagnation of nasal secretions in the post nasal space" as a result of inadequate velo-lingual function in tongue thrust, could lead to nasopharyngeal disorders.

Subtelny (1954) found that where excessive adenoid tissue was present the dorsum of the tongue tended to move away from its usual position of contact with the soft palate. It tended to be found more downward and forward away from the soft palate (Fig. 164A) thereby establishing an oral pathway for respiration. The repositioning of the tongue was then surmised to have removed its influence from the lingual surface of the upper posterior teeth against which it normally rested, thus explaining the cross bite tendency. After removal of the adenoid and tonsillar tissue the soft palate was found to rest again in contact with the posterior surface of the tongue (Fig. 164B).

Ardren and Kemp (1972) studied tongue to tonsil size relationships. They found small tongues associated with large tonsils, and large tongues associated with small tonsils. They also suggested that large tonsils did not lead to swallowing problems except when they were inflamed. They also drew attention to the lymphoid tissue content of the posterior third of the tongue, the bulk of this structure being "dependent on its lymph content". They believed that this tissue is necessary for the space filling requirements of the pharynx in association with the tonsils and adenoids.

Ricketts (1958) studied the changes in posture of the tongue, soft palate, and head before and after adenoidectomy, tonsillectomy, or both. His results showed, in the tongue, an upward and backward retraction. He found that for the removal of large tonsils, large changes in tongue posture were
Fig. 164  A. Excessive adenoid tissue causing an aberrant tongue-soft palate relationship. The posterior dorsum of the tongue appears to be in a downward and forward position away from the soft palate.

Fig. 164  B. Improved tongue position resulting from the provision of an adequate nasorespiratory channel. Note that some residual tissue still remains. (Subtelny, 1954)
noted. For the soft palate, an alteration in the form was noted. For the head, a two degree change in tilt forward from the cervical vertebrae. He concluded that a reduced airway led to a lower tongue posture, leading to dentitional changes. If the airway is opened up by removal of tonsils and/or adenoids a more normal, i.e. upward and backward, tongue posture is likely.

Cleall (1965) said that a highly developed neuromuscular mechanism must be operating to protect the airway (discussed previously). He postulated at least two controlling systems involved in deglutition; one functioning in the anterior part of the mouth and the other in the pharynx. Bosma (1963) had said that the relative anteroposterior position of the dorsal portion of the tongue is principally determined by the requirements of the pharyngeal airway.

Subtelny and Sakuda (1964) felt that enlarged tonsils may force the tongue to "move forward, away from its normal position, while in repose". And if the obstruction was severe enough, the tip of the tongue might "habitually lie between the anterior teeth, preventing their eruption or displacing them during eruption". They advised the removal of the enlarged tissue allowing reposturing of the tongue and subsequent eruption of the incisors.

Gershater (1972) added a cautionary note to this. He conceded that the removal of "the offending lymphoid tissue" permitted the muscles of mastication to function in a more normal environment. Nevertheless, this did not always predispose a spontaneous reversion to a normal pattern of swallowing. The explanation for this being that the normal compulsive functional patterns of deglutition had become deeply entrenched and, as a result, the muscles had to make compensatory adjustments to a new function and
environment. Yip and Cleall (1971) found that the posterior oral seal improved dramatically after removal of enlarged tonsils and adenoids. However, they reported little change in "teeth-apart swallow and tongue thrusting".

It seems then that removal of excessive epipharyngeal lymphoid tissue may lead to resolution of atypical tongue behaviour in some, but not in others. Could the explanation for this lie in the underlying neuromuscular physiology and patterned neural pathways as suggested?

Hanson, Barnard, and Case (1970a) found only 2 factors out of a total of 22 to be functionally associated with tongue thrust with any meaningful consistency. They were enlarged tonsils and lingual cross-bite. They added that the enlarged tonsils possibly contributed to the development or persistence of tongue thrust by encouraging an habitual forward placement of the tongue.

Ricketts (1968) believed that a great many "tongue problems" were in some way related to respiratory problems, either at the present or in the past history of those with the problems. He identified the functional demands of the soft palate, as well. If the soft palate was influenced to be held downward in an effort to maintain the airway due to enlarged adenoids, then the tongue would conditionally acquire a forward or downward posture in the mouth.

There have been numerous studies relating forward tongue posture to airway inadequacy, Subtelny and Subtelny (1973), Linder-Aronson (1970, 1974) to name a few. Proffit, McGlone, and Barrett (1975) also found that forward positioning of the tongue in Australian Aborigines was "a physiological necessity" because of airway considerations. The pharyngeal cavity being
smaller and the oral cavity being larger than white Americans'.

Thurow (1975) described the effect of tonsillar enlargement, pointing out that the enlargement may be acute or chronic, painful or painless. The degree and proportion of the response of the tongue depended on the amount of enlargement and pre-existing relationships of the tongue and related structures, each patient being an individual problem. A tongue thrust posture and/or swallowing pattern could result, he felt, this being the opening wedge in the establishment of a tongue thrust pattern that could "persist long after the tonsils are gone". Tender or painful tonsils, he said, could aggravate the physical responses to enlargement. The tongue would avoid the tonsils on swallowing (or at rest) often involving an anterior thrust combined with opening of the mandible and contraction of the geniohyoids to keep the tongue pressure off the tonsils. He pointed out though, that enlarged tonsils may or may not be associated with forward tongue posture, depending on the space requirements of the pharynx. On that note Proffit (1977) believed that a change in resting posture of the tongue, because of respiratory requirements, would be much more significant than tongue tip position during swallowing.

Proffit and Mason (1975) felt that the tongue thrust associated with airway obstruction was only one of several related factors, and the key to its resolution was correction of the respiratory problem. They also felt that with normal growth and development of the faucial isthmus and pharynx, and the tendency for the pharyngeal lymphoid tissue to atrophy around the time of puberty, tongue thrusters were likely to "evolve a normal adult swallow pattern". However in the light of Linder-Aronson and Leighton's (1983) studies, this notion of waiting for resolution before treatment could be misguided. Since it is likely that if the lymphoid tissue reaches its greatest size at 5 years, then the enlarged lymphoid tissue seen at age 12-13-14 years
is likely to remain enlarged unless medical or surgical treatment is undertaken.

Further to this point, according to the findings of Hanson and Cohen (1973), of the factors which were most likely to be associated with the persistence of a tongue thrust throughout the mixed dentition, i.e. between persistent and temporary tongue thrusters, the main factor was mouth breathing.

Pierce (1978) attempted to define the relationship between mouth breathing and tongue thrust, whether at rest or during swallowing (Fig. 165). Mouth breathing was seen as one of the causes of tongue thrust. She points out, though, that "not all tongue thrusters are mouth breathers; not all mouth breathers are tongue thrusters. For a large number of patients, however, mouth breathing may be the factor primarily responsible for the retention of abnormal swallowing habits".

Barrett and Hanson (1978), interestingly, related the "cause" of mouth breathing to abnormal deglutition. They felt that the "antigravity" muscles of the mandible and tongue provided adequate development only during normal deglutition, and that tongue thrust denied these muscles normal tonicity. As a consequence they felt that in a resting state the mouth falls open in these patients, because of the "sheer weight" of the mandible, tongue, and their associated structures. Mouth breathing then, occurred even though the nasal airway is available and used occasionally. Resting muscle tonus then was responsible for not maintaining normal mandibular elevation and effortless lip closure.

Returning to the work of Linder-Aronson (1979), the improvements seen in skeletal and dental relationships following adenoidectomy were believed
Fig. 165  Airway factors, mouth breathing, and tongue thrust. (Pierce, 1978)
to be the result of a change from mouth to nasal breathing which allowed the
tongue position to be raised and the lips held together. The change in
mandibular inclination seemed to be associated with a change in tongue
position and head posture.

In relation to the tongue and hyoid bone position Adamidis and
Spyropoulos (1983) found the same results as the previous authors for tongue
position and mandibular inclination. They also noted that the hyoid bone
appeared to follow the inclination of the mandibular plane.

Frankel (1980a) also described the postural alterations of the tongue
and jaws resulting from mouth breathing. He suggested that these alterations
were possible as a result of disadvantageous intra-oral space conditions, e.g.
when bulky acrylic appliances are inserted. Thurow (1975) also made mention
of this latter point of orthodontic treatment adversely affecting these space
requirements. Again, according to Frankel as quoted by Watson (1982b) the
anterior and posterior seals of the oral cavity (mentioned previously) function
properly to produce a balance. Further, the postural performance of the
muscles providing the anterior lip seal is of the utmost importance in
establishing the physiologic atmospheric pressure conditions of the oral, nasal,
and pharyngeal spaces to maintain equilibrium of the teeth and supporting
bone. The impact of respiratory function on craniofacial morphogenesis, he
felt could be recognised only when the space conditions are seen in the
context of the postural behaviour of the orofacial musculature.

Returning to the neuromuscular physiology aspects, Miller, Vargervik,
and Chierici (1984) recently found that nasal obstruction can induce neuro-
muscular changes which extended beyond the period of obstruction and
remained after the original stimulus for neuromuscular change had been
removed. This probably explains, they felt, why some patients continue to breathe through the mouth 'habitually' after tonsil and/or adenoid airway obstructions have been removed, whereas most revert to nasal respiration.

Finally on a practical note, according to Lowe (1985) the existence of a reflex protrusion of the tongue in response to mandibular rotations has been confirmed. "Canonical correlations have quantified a tongue tip ahead of the lower incisor and above the occlusal plane, together with a forward posterior pharyngeal wall in subjects with a steep mandibular plane and a large gonial angle". Lowe warned of probable posttreatment relapse if only the dento-alveolar structures were altered, with little regard to pharyngeal, lingual, and mandibular growth rotation factors.

4.2.3.6 Conclusions and Current Medico-Dental Recommendations

Ricketts (1968) was one of the first to advocate closer communication between orthodontist, otorhinolaryngologist (ENT), and pediatrician, in order that more comprehensive management of patients with airway and malocclusion disorders could be achieved. Brodie (1971) was another of the earlier protagonists of more active involvement by orthodontists in airway assessment and appropriate management by and with otorhinolaryngologists. He called for a more biological, rather than a mechanical, appraisal of malocclusion.

Lanier and Tremblay (1979), both allergists, urged that there should be a greater awareness among health care providers, of the possible consequences of mouth breathing and nasal congestion, particularly among dental practitioners. They emphasised the need for early diagnosis and appropriate treatment designed to promote better nasal breathing as part of a "comprehensive medicine program".
Earlier, Linder-Aronson (1970) had recommended that assessment of the indications for adenoidectomy should include an orthodontic evaluation.

Thurow (1975) also advocated the early removal of pathologic tonsillar tissue where indicated so that aberrant functional patterns would not be likely to persist after the cause had been removed.

Proffit (1977) however, did not regard recommending early removal of tonsils and/or adenoids as being advisable, but preferred to leave patients until their late teens before ENT evaluation. He warned of the chances of increased morbidity which could result from early removal, and of the risks of the surgical procedures, although McCurdy (1977) comprehensively disputed this claim. Moreover, it is difficult to see the wisdom of this argument that late removal (post-adolescent) is preferable when, as has been shown, the untoward craniofacial and dentoalveolar consequences of airway obstruction are very likely to have been established well beyond the primary preventive stage. He recommended, in fact, waiting until it was obvious that "continued vertical growth and mandibular rotation, as well as other difficulties from the tonsils and adenoids themselves" became evident, before doing something about them.

Schulhof (1978) recommended the detection of mouth breathing in a child as soon as possible. He also pointed out that having a norm for the size of the adenoids might allow for a strategy of partial adenoidectomy. In this way, he thought, the amount of tissue necessary to attain a normal percentage of airway could be removed and the lymphoid system not compromised.

Hanson (1978, 1979a), a myofunctional therapist, recommended the "elimination or amelioration" of any untoward airway condition through medical treatment or surgery before starting tongue thrust treatment. At age
4 to 5 years, he recommended "important preventive procedures" to be taken primarily concerned with promoting easy nasal breathing.

Likewise, Subtelny (1980) added that anything that orthodontists could undertake "to improve and permit nasal respiration would seem to be desirable and helpful and should be attempted".

Rubin (1979, 1980) too, has gone as far as advocating that orthodontists take a more assertive role in the management of nasal airway dysfunction. He also recommended early assessment of respiration, advising appropriate early medical treatment to prevent problems from developing. He also recommended that our medical and dental colleagues be educated to recognise those conditions that can produce facial deformity.

To conclude this section, and to attempt to draw together these last three controversial subsections into some lucid form, I have drawn upon the work of two eminent researchers, E.P. Harvold and M.L. Moss.

According to Vargervik, Miller, Chierici, Harvold, and Tomer (1984) it has been demonstrated that altered function can affect the development of form. Furthermore, changing the position of a bone relative to its attached and surrounding musculature results in the remodelling of surface areas as well as of the internal architecture of the bone; and, experimentally induced changes in neuromuscular activity can result in altered skeletal morphology.

Vargervik et al. (1984) postulated that the variations in morphologic response seen in their monkey studies are due to differences in muscle recruitment associated with the change in mode of respiration. These variations included malocclusions, and tip and tongue changes. These authors
have shown that the lip and tongue changes disappeared after nasal breathing was restored. They postulated that the neuromuscular patterns that were induced by oral respiration, and that presumably caused the alteration in soft tissue morphology and adaptive motor behaviour, would change after removal of the stimulus. Consequently, it was also expected that the skeletal morphology that resulted from the adaptation to oral respiration would again be affected by the new postexperimental neuromuscular recruitment patterns. If these patterns returned to pre-experimental levels, the skeletal and dental changes might be reversed.

What these authors found was that the extent of the skeletal changes appeared to depend on the degree of soft-tissue alterations, "in the animals with the most marked morphologic tongue changes, the skeletal and dental changes tended to be less than in the other animals". They found that although their animals had similar degrees of nasal obstruction, they demonstrated a wide range in morphologic adaptation. Vargervik et al. believed then, that the degree of morphologic change does not depend on the amount of air that flows through the mouth and nose (O'Ryan et al., 1982; Vig et al., 1981), instead, it depends on the nature of the neuromuscular and soft-tissue adaptations.

The same, they say, is true in human beings, where a great variation in soft-tissue and skeletal morphology can be observed in response to complete nasal airway obstruction (Crouzon's syndrome, Apert's syndrome, choanal atresia) as well as in response to partial nasal airway obstruction. Most importantly, therefore, their data has shown a wide individual variation in response to an identical stimulus. As McNamara (1981b) has noted, and as Vargervik et al. put it, "in assessing the contribution of oral respiration to the development of certain dental malocclusions and facial morphologies, the
clinician should be aware of the great variation in individual response to similar stimuli".

It would seem then that vital respiratory needs strongly affect jaw and tongue, as well as dentoalveolar positions. To bring this discussion finally to a conceptual level, Moss and Salentijn (1971) pointed out how the orofacial capsular matrices, i.e. the volumes of the oronasopharyngeal functioning spaces, act as a mould for the positioning of the jaws. They have gone so far as to assert that the growth of the oral functioning space, epithelially produced and neurotrophically regulated, is the primary morphogenetic event in mandibular growth (Moss, 1964). They have found that mandibular and orofacial growth can be described by a single logarithmic spiral indicating that such growth is gnomonic. It is known, they pointed out, that following the closure of the embryonic palatal processes, the shape of the oral, nasal, and pharyngeal functioning spaces remains constant and only their size increases. Why then do anterior open bite and deep bite develop? As seen in an earlier section, anterior open bite and deep bite have been considered as either 'skeletal' or 'dental' growth disharmonies or, most usually, as some combination of both factors, "in any case, the etiologic problem was thought by most to reside in calcified tissues as such" (Moss and Salentijn, 1971). According to these authors, it is apparent that the root of the problem logically may be sought in the capsular functional matrices, with their related processes of passive translatively growth: "In anterior open-bite, for example, the possibility exists that either the form (the size, the shape, or both) of the oral functioning space might be characteristically abnormal or the location of this functioning space, relative to the nasal and pharyngeal functioning spaces, might be abnormal".

It is likely then, that extraneous influences on normal function are
likely to be the cause of an imbalance in the growth regulating mechanisms. The most likely influences being interruption to normal airway operation and/or an impairment of the neurologic regulating system (Gershater, 1972; Arvystas, 1977; Rubin, 1980; Miller et al., 1984). From either or both of these influences it seems that stable positions of dental equilibrium and normal overbites are very likely to be upset, depending on the individual's neuro-motor response, which regulates posture and movement of the jaw. These aspects of adaptive growth having been extensively reviewed by Cachel (1979, 1985) and Goss (1978). It is not the intention of this discussion to dwell on these aspects of adaptive growth in detail.
4.3 Speech and Tongue Thrust

As mentioned in previous sections, the assessment of tongue function in speech is an important part of diagnosing tongue thrust behaviour. A very concise overview of speech production, speech pathology, and their relationships to tongue thrust is presented.

4.3.1 Speech Production and Speech Defects

Speech may be described briefly (Jenkins, 1978) as the production of sound by the larynx (phonation) and its modification by the resonance of the air in various spaces between the larynx and the lips (articulation). Both processes are under voluntary control and both contribute to the considerable variety of sounds which go to make up speech.

(A) Speech Production:

In common with many other complex oral motor functions, speech utilises numerous muscle groups (Roth and Calmes, 1981). These include muscles of the larynx, pharynx, palate, tongue, jaws, lips, and cheeks; the respiratory muscles of the chest wall and diaphragm are also used.

A brief overview given by Scott and Symons (1971) states that in the production of speech the muscles of the tongue, lips, and soft palate cooperate with those of the larynx. The sounds produced by the larynx are modified according to whether the expired air passes through the nasal cavity, the oral cavity, or through both, and this depends on whether the lips are open or closed. Temporary obstruction to the escape of air is produced by the palate, lips, teeth, or tongue, and is responsible for the majority of consonant sounds, whereas in the production of vowel sounds there is little if any obstruction but various modifications in the form of the oral passage produced by the action of the muscles of the jaws, cheeks, lips, and tongue. An extremely useful account of speech production with particular emphasis on
normal tongue placement in producing the various speech sounds was given by Mitchell (1957).

As Bashir and Schultz (1978) have pointed out, speech and speech disorders fit into the overall heading of "communication disorders", which they have separated into 5 categories:

(1) **Articulation Disorders** - the major group of problems in speech sound production. These deficits differ from language disorders in that they are not symbolic deficits;

(2) **Resonance Disorders** - deficits arising from a disruption in normal oral-nasal sound balance. These are most commonly heard as hypernasality or hyponasality (dealt with later);

(3) **Fluency Disorders** - disruption in the natural flow of connected speech; the most common form being stuttering;

(4) **Language Disorders** - disruption in the ability to comprehend and/or manipulate the symbol system of the language community. The causes of such disabilities are diverse; and

(5) **Voice Disorders** - deviations in the quality, pitch, or loudness of the voice. The basis of these deficits may be psychologic and/or physiologic in nature.

It is not intended that this discussion should examine in detail all the mechanisms of speech production. Various authors (Travis, 1971; Espir and Rose, 1976; Winitz, 1969; Minifie, Hixon, and Williams, 1973; Weiss, Lillywhite, and Gordon, 1980) have already explored these areas in detail. Nor will the aspects of fluency, language, and voice disorders be discussed. Here, briefly, is an account of some of the main elements in speech production relating tongue posture to positions of the teeth, in particular.
(i) **Anatomy of Speech** (Warwick and Williams, 1980)

Speech has several components, as mentioned above: voice, articulation and language, according to Vig (1973), as well as fluency or rhythm, according to Bond and Lawson (1968) and Greulich (1972). The larynx and vocal tract coordinate to produce voice and to facilitate articulation. The larynx is the primary source of the complicated and endlessly varying chains of sounds which are the basis of speech. A full description is covered by Warwick and Williams (1980). The vocal tract is made up of the associated respiratory spaces of the pharynx, mouth, and nasal cavities. These structures act together in the process of 'phonation', the production of sounds and their articulation into 'phonemes', the basic units of speech. The acoustics of speech, in terms of the structure of the vocal tract is compared to an organ pipe (Warwick and Williams, 1980). There is: (1) a source of energy - the velocity of the stream of expired air; (2) structures capable of periodic and also aperiodic oscillations - the vocal folds; and (3) a resonator - the multi-form 'column' of air from the vocal folds to the lips and nostrils.

**Phonation.** The vibratory movements of the vocal folds (vocal cords) play a critical role in phonation (Roth and Calmes, 1981). Changes in the frequency of these vibrations bring about changes in the pitch or tone of the voice. Four factors act to change the pitch: (1) contraction of the muscles within the cords, bringing about an increase in tension; (2) changes in the shape of the cords; (3) increases in the strength of contraction of the external laryngeal muscles; and (4) air pressure (Jenkins, 1978). Expired air pressure is also the chief factor which increases the loudness of speech.

**Articulation.** Articulation is the shaping of sound into vowels and consonants. Other oral structures as well as the larynx come into play to produce the vowel sounds and consonants that make up speech patterns. The possible structures and resonance chambers or spaces include:
(1) The vestibule between the (true) vocal cords and the ventricular (false) cords;
(2) The nasal passages and the spaces between the larynx and the base of the tongue;
(3) The posterior pharyngeal wall and soft palate;
(4) The dorsal surface of the tongue and hard palate;
(5) The tongue tip and teeth; and
(6) The teeth and lips.

**Vowel Sounds:**

For vowel sounds, the air passages remain uninterrupted but the oral resonating spaces change in size and shape by the movements of the tongue, lips and pharynx. The differences between vowel sounds are essentially differences in pitch and pressure (Fig. 166).

**Consonants:**

In contrast to vowel sounds, consonants are produced by the interruption of the air current above the larynx by the soft palate, teeth, and lips (Fig. 167). Consonants, like vowels, are produced by the resonance of different shaped chambers and their sounds can also be analysed usually into two main pitches (Jenkins, 1978). The resonances for the consonants may vary with the vowel associated with it. The probable reason for this variation is that the tongue will take up, not a constant position every time the same consonant is uttered, but that position most easily reached after the preceding vowel has been produced, which still gives a sound which the ear can identify correctly. For some vowels and consonants (e.g., b, z), the vocal folds also contribute to the sound produced. Consonants vary from each other depending upon whether they are accompanied by phonation (voiced) or not (unvoiced).

The consonants may be classified in several ways, Mitchell (1957),
Fig. 166  Approximate tongue positions for the vowels.
A. Front vowels.
B. Back vowels.
C. Central vowels.  (Weiss, Lillywhite, and Gordon, 1980)

Fig. 167  Place of articulation of the consonant sounds:
A. Bilabial;   B. Labiodental;
C. Linguadental; D. Lingua-alveolar;
E. Linguapalatal; F. Linguavelar.
(Weiss, Lillywhite, and Gordon, 1980)
Jenkins (1978), Bashir and Schultz (1978), one being according to the structures which cause the interruption of the current of expired air (sites), thus: bilabials, labiodentals, interdentals, alveolars, alveolopalatals, palatals, velars, and glottals (Fig. 168). Sites have two similarities; (a) a partial obstruction or constriction at some level of the vocal tract, and (b) production of aperiodic vibration (noise) which is superimposed or interrupts the flow of laryngeal tones.

Another similar classification is:

- **Plosives** (stop consonants): p, b, t, d, g, and k which require a complete stoppage of air,
- **Fricatives**: f, v, and th which require only a partial stoppage,
- **Affricatives**: ch and j which, although involving only a partial stoppage of the air, do require a rapid release of this air,
- **Nasals**: m and n which require obstruction of the mouth with the nasal passages open,
- **Lateral**: l air forced to leave side of mouth,
- **Rolled**: r

A full description is found in the accounts of Mitchell (1957), Bashir and Schultz (1978) and Jenkins (1978). Of note is that **sibilant sounds**, /s/, sh, and z/ depend upon the passage of expired air through a very narrow space between the tip of the tongue and the anterior part of the hard palate with or without phonation of /z/ and s/ respectively. Consonants are also differentiated as either **oral** or **nasal** consonants (Mulder, 1976).

(ii) **The role of the soft palate**

During the production of most of the speech sounds (except m, n, and ng) the soft palate closes off the naso-pharynx from the oro-pharynx. The soft palate rises least for the 'ah' sound and touches the pharyngeal wall lightly or not at all. The soft palate rises progressively and therefore touches with
### The Consonants of the English Language

<table>
<thead>
<tr>
<th>Place of Production</th>
<th>Plosives</th>
<th>Glides</th>
<th>Fricatives</th>
<th>Affricates</th>
<th>Nasion</th>
<th>Semi-vowels</th>
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<tr>
<td>Bilabial</td>
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<td>p</td>
<td>ʋ</td>
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<td>Labiodental</td>
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<td>Interdental</td>
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<td>Alveolar</td>
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<td>Alveopalatal</td>
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<td>Glottal</td>
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</table>

All consonants are listed by sound value as though the consonant could be produced without accompanying intra-yabby vowel (see text for discussion).

V = voiced, produced with accompanying vocal fold vibration.

Ṿ = voiceless, produced without accompanying vocal fold vibration.

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**Fig. 168** Table: The consonants of the English language. (Bashir and Schultz, 1978)

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**Fig. 169** Movement of the soft palate in a low vowel A (/a/) to a high vowel U (/y/). (Mulder, 1976)
increasing force as the vowels 'oh', 'a' (as in late), 'ee', and 'oo' are pronounced (Jenkins, 1978; Mulder, 1976), (Fig. 169).

According to Weiss, Lillywhite, and Gordon (1980), if the soft palate is too short, does not move far enough, or does not move as quickly or consistently as it should, articulation and resonance usually are disordered because of inadequate velopharyngeal closure.

Browning (1982) and others (Bashir and Schultz, 1978) have described the two different types of speech defect caused by palatal dysfunction: (1) Hyponasality, occurring when the nose is unable to function as a resonant chamber because the postnasal space is full of adenoid tissue and the palate cannot relax any further to allow air into the nose. Those with hyponasal speech sound as if they have a constant cold; and (2) Hypernasality, occurring when the soft palate is unable to close off the postnasal space. The consequence is an inability to say consonants clearly, and speech sounds as it does in a cleft palate person. Adenoid hypertrophy does not itself cause hypernasality but it can occur after adenoidectomy if there is a relative shortness of the soft palate.

(iii) Neural mechanisms and reflex control

The neural mechanisms that underlie the activities of the various muscle groups during speech are still poorly understood (Roth and Calmes, 1981). The relative importance of sensory versus central control is not clear. Previously the importance of the cerebral cortex in speech production and control was stressed. A unilateral cortical dominance was found, with the so-called cortical 'speech areas' or 'centres' restricted to the left cortical hemisphere in most people. These speech centres were localised particularly to the lateral frontal and temporal regions of the cortex (Fig. 170). Speech is currently viewed as an integrated function involving these cortical areas and
Fig. 170 The superolateral surface of the left cerebral hemisphere showing the motor speech areas of Broca (44, 45) and Wernicke. The latter is variously depicted by different authorities, and is tentatively indicated by the large parieto-temporal area enclosed in an interrupted outline, which itself includes areas 39 and 40. Areas 22 and 37 are considered by some to be respectively auditory and visuo-auditory areas associated with speech and language. (Williams and Warwick, 1980)
other cortical regions of the brain; superimposed on this central substrate is sensory feedback, which adds refinement and control. Espir and Rose (1976) have discussed these matters in detail.

Sensory feedback stems from auditory and oral receptors. The importance of auditory feedback is exemplified by the stuttering that may ensue if the normal short delay between speaking and hearing one’s spoken words is experimentally prolonged. The oral receptors have not yet been precisely defined, but those most likely involved in providing sensory messages used for speech production and control are tongue mechanoreceptors, laryngeal joint receptors, and laryngeal and possibly tongue muscle receptors. Some studies reported by Jenkins (1978) tend to suggest that tongue receptors are more important than hearing receptors. The anterior part of the tongue, the region responsible for the complex articulation of sibilant sounds (s and sh) is much more richly supplied with tactile receptors, muscle spindles, and tendon organs than the posterior part which carries out relatively coarse movements. In English, it is these sounds involving the anterior part in which slips and errors are most likely to occur. Also these sounds are among the last to be mastered by children when learning to talk.

**Development of speech**

According to Bond and Lawson (1968) and Vig (1973), children have voice from birth. The necessary skills for articulation and language, however, develop considerably later. Eight distinguishable sounds are usually heard during the first few days of life. This represents about a fifth of the sound elements used by adults. An infant's sound repertoire consists mostly of 'front' vowels and 'back' consonants. As the child grows older, non-crying sounds begin to dominate the cries. The child achieves increasing control of the oropharyngeal structures, and back vowels and front consonants appear. At
two and a half years, only two-thirds of the sound complement is developed, although meaningful words appear towards the end of the first year. The sounds /s/, /th/, and /r/ are the last to be learned and are physiologically the most difficult (Subtelny and Subtelny, 1962). These sounds are generally not acquired until about 6½ years for girls and 7½ years for boys (Fig. 171). Speech learning is thought to be related to growth of the jaws to produce a suitable space for the tongue. As a result of differential growth during maturation, the tongue comes to take up relatively less space in the oral cavity at maturity than at birth (Brodie, 1952). There seems to be an increase in available tongue space from the age of 9 years to 16 years. According to Vig (1973) the rate of speech development in children is likely to be affected by the differential growth of the tongue and its rigid confines.

Children tend to learn first those sounds which require the fewest muscles, the largest muscles, and the least degree of coordination between muscles. Progress is made in a physiological manner from production of gross sounds to more sophisticated sounds (Barrett and Hanson, 1978). Of note is that due to the serial order of mastery, severely distorted sounds may be considered perfectly normal speech at one age but be classified as speech defects at a later date. If several of the late-arriving sounds are mis-articulated at 4 years of age, it is not thought to constitute a speech defect, whereas the same speech at 6 years of age would be difficult to judge as normal.

(B) **Speech Defects:**

Speech is not an automatic process. It has to be learned. A child will learn to speak by means of hearing, sight, and touch (Bond and Lawson, 1968). Provided there is no impairment to these faculties the child will learn from the speech of others. The child will then reproduce what is heard as nearly as
Table □ Age at which selected consonants are usually produced correctly.

<table>
<thead>
<tr>
<th>Age (in years)</th>
<th>Consonants</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>p, m, ng, f, h, w</td>
</tr>
<tr>
<td>3.5</td>
<td>b</td>
</tr>
<tr>
<td>4</td>
<td>d, g, k, r</td>
</tr>
<tr>
<td>5</td>
<td>t, s, sh, ch, l, v</td>
</tr>
<tr>
<td>6</td>
<td>th (unvoiced), z</td>
</tr>
<tr>
<td>7</td>
<td>th (voiced), s and r blends, sm, gr</td>
</tr>
</tbody>
</table>

Fig. 171 Table: Age at which selected consonants are usually produced correctly. (Warman, Weidman, and Kolman, 1981)

<table>
<thead>
<tr>
<th>Type of disorder</th>
<th>Individuals per 1000 population</th>
<th>Percent of total population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Functional articulation</td>
<td>35</td>
<td>3.5</td>
</tr>
<tr>
<td>Stuttering</td>
<td>7</td>
<td>0.7</td>
</tr>
<tr>
<td>Voice disorders</td>
<td>5</td>
<td>0.5</td>
</tr>
<tr>
<td>Delayed speech</td>
<td>5</td>
<td>0.5</td>
</tr>
<tr>
<td>Impaired hearing (with speech defects)</td>
<td>5</td>
<td>0.5</td>
</tr>
<tr>
<td>Cerebral disorders (brain injury)</td>
<td>2</td>
<td>0.2</td>
</tr>
<tr>
<td>Cleft-palate speech</td>
<td>1</td>
<td>0.1</td>
</tr>
<tr>
<td>Total</td>
<td>60</td>
<td>6.0</td>
</tr>
</tbody>
</table>

Fig. 172 Table: Estimate of incidence of speech defects (Van Riper, 1963). (Barrett and Hanson, 1978)
possible. A speech defect can be defined as an absence, loss, or disturbance of language, voice, articulation, rhythm, or any combination of the four.

Speech defects may be developmental or acquired subsequently, and many factors may be responsible, including:

(1) Lack of, or delay in, the development of speech caused by:

- Mental deficiency
- Severe or partial deafness
- Cerebral lesions
- Late neurological maturation
- Severe congenital abnormalities of the organs of speech
- Auditory imperception
- Emotional factors
- Lack of stimulation - little or no speech in the child's environment.

(2) Loss or impairment of speech resulting from:

- Severe or partial acquired deafness
- Cerebral lesions
- Incoordination of the neuromuscular systems
- Acquired abnormalities of the organs of speech
- Hysteria.

Each type of speech defect - absence, loss, or disturbance of voice, language, articulation, or disturbance of rhythm, may result from different causes.

According to Van Riper (1963) functional articulatory problems are the most common the speech defects (Fig. 172). Such factors as malocclusion and abnormal swallowing behaviour play a significant role in many of these cases.
4.3.2 Speech Defects and Malocclusion

Graber (1973) wrote that the positions of the teeth and the relationship of the supporting tissues are basic in speech physiology. The outgoing airstream and vocal tone are inflected by the relative positional changes of these hard and soft tissues. Dental malocclusions therefore serve as a factor in speech pathology. Most of the time adaptive, adaptive, or compensatory activity largely overcomes the malocclusion handicap, but not always. "The articulation of consonants and vowels may be attributed to a functional maladaptation to the dental malocclusion."

Bloomer (1971) notes that the effects on speech may be direct or indirect. Direct ones are by mechanical difficulties imposed as the person tries to obtain the proper position and movement of the articulators of speech; and indirect ones by deformities influencing the physical and mental health of the individual. Graber (1973) cites the example of a Class II, Division I malocclusion as a direct influence. If there is a protruding maxillary segment it makes the normal production of bilabial consonants difficult. If there is an anterior open bite, anterior escapage is permitted interfering with the normal production of sibilants.

In general there are a number of speech defects "of special interest to the dentist" (Barrett and Hanson, 1978). The list includes:

(1) Articulation: (a) Lolling,
    (b) Lisping - frontal lisp
        - lateral lisp
        - nasal lisp
        - occluded lisp,
    (c) Dentalisation of linguoalveolar sounds.
(2) Delayed Speech.
(3) Hypernasality and Hyponasality.
(4) Hearing Loss.

A full description accompanies this list.

Graber (1973) again, points out that most speech sounds can show the effects of malocclusion, since the teeth, supporting structures, tongue, and lips are directly involved in the production of consonants requiring pneumatic control for fricative and plosive characteristics. The oral structures also modify the air column by blocking it - widening, narrowing, or otherwise altering the passageway. The accurate formation of vowels and diphthongs may also suffer, though this is less critical than the enunciation of consonants.

Most of the defects are dyslalic phenomena, that is, defective articulation due to faulty learning or abnormalities of the external speech organs and not caused by lesions of the central nervous system (Fig. 173). However the problem is not a simple cause-effect relationship because of the excellent adjustable mechanism. Some of the most severe malocclusions show no speech pathology. Some people consciously or unconsciously have taught themselves how to compensate for their abnormality (Jenkins, 1978). According to Greulich (1972), on this important point, articulation disorders often occur when there is damage to, or absence of, oral structures and their associated muscles. However the oral deformities do not always produce articulation disorders. "The patient is often able to compensate for the absence or deformity of structures, and the disturbances in function. His intelligence and motivation will be the deciding factors in the way he handles his problems". Fawcus (1975) adds that the flexibility of the speech system is such that the presence of structural, or even some physiological, deviations is not of itself reason for deterioration in the quality of the acoustic output.
<table>
<thead>
<tr>
<th>Causes</th>
<th>Diagnostic terms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Idiopathic articulatory dyscoordinations</td>
<td>Articulatory disorder</td>
</tr>
<tr>
<td>2. Orofacial abnormalities</td>
<td>Dysarthria</td>
</tr>
<tr>
<td>3. Emotional disorders (neurotic or psychotic)</td>
<td>Articulatory disorder</td>
</tr>
<tr>
<td>4. Defective learning environment</td>
<td>Articulatory disorder</td>
</tr>
<tr>
<td>5. Neural dysfunctions</td>
<td></td>
</tr>
<tr>
<td>(a) Affecting the corticospinal motor system</td>
<td>Dysarthria</td>
</tr>
<tr>
<td>(b) Affecting the primary motor and association areas of</td>
<td>Articulatory dyspraxia</td>
</tr>
<tr>
<td>the cerebral cortex</td>
<td></td>
</tr>
<tr>
<td>(c) Affecting the primary sensory and association areas</td>
<td>Articyspecific articulatory</td>
</tr>
<tr>
<td>of the cerebral cortex</td>
<td>dyspraxia</td>
</tr>
<tr>
<td>(d) Affecting the special senses (as in peripheral</td>
<td>Dysaudia</td>
</tr>
<tr>
<td>deafness)</td>
<td></td>
</tr>
<tr>
<td>(e) Affecting the coordinating functions of the cere-</td>
<td>Articulatory cerebellar dys-</td>
</tr>
<tr>
<td>bellum</td>
<td>praxia</td>
</tr>
</tbody>
</table>

Fig. 173 Table: Speech defects. (Bloomer, 1963)
Pierce (1978) also notes the compensatory ability of the oral peripheral mechanism. She says that the speech problems which could be considered to result from malocclusion are primarily cosmetic rather than acoustic, and might not be noticed by the untrained observer. Sounds are referred to as being acoustically correct or cosmetically incorrect when the phonemes sound right even though the placement of the articulators is wrong. However, there are patients with moderate to severe malocclusions who have not learned to compensate and who exhibit faulty articulation. It would be difficult to state that the malocclusion caused the articulation problem, although perhaps this is the case with certain patients. "Speech therapy techniques, orthodontic treatment, or a combination of the two should be successful".

Bloomer (1971) summarises relationships between structure and function as follows:

1. Normal structure + normal movements = normal speech.
2. Abnormal structures + maladaptive movements = defective speech.
3. Normal structures + maladaptive movements = defective speech.
4. Abnormal structures + adaptive movements = normal (compensated) speech.

Vig (1973) draws the conclusion, from these findings and his, that speech therapy is not indicated merely for the correction of a specific pattern of behaviour if the sounds are acceptable phonetically.

Certain malocclusions are, however, more difficult to compensate for and more likely to cause speech anomalies. Graber (1972) lists the following:

Class I:
- anterior open bite
- missing anterior teeth
Class II, Division 1:
- excessive overbite and overjet
- abnormal perioral muscle function

Class III:
- absence of incisal contact
- redundant lower lip
- deviant tongue function.

Full analyses of these malocclusions and their speech correlations are covered by numerous authors (Bloomer, 1971; Barrett and Hanson, 1978; Reaney, 1976; Graber, 1972; Jenkins, 1978; Pierce, 1978). Essentially there are some main features worthy of note:

1) **Open bite**: if severe it is liable to interfere with speech. Acoustically acceptable sibilants are hard to produce and bilabial consonants may suffer. /f/ and /v/ will be defective if the upper teeth cannot touch the lower lip and /p, b, and m/ are impossible to pronounce correctly if the lips cannot be brought back into contact. Pronouncing /s/ and /z/ is difficult because the amount of air escaping between the hard palate and the tip of the tongue will be larger than usual. Suzuki, Sakuma, Michi, and Veno (1981) studied maximal palato-lingual contacts in patients with anterior open bite using dynamic palatography. They identified the abnormal speech sounds produced and the method by which the palate and tongue combined to compensate for the lack of incisor positioning. Correction is usually achieved by closing the bite.

2) **Missing anterior teeth**: extraction of the upper incisors will affect /f/ and /v/. If the upper incisors protrude and normally rest on the lower lip, the lips may be made to meet only with difficulty which will affect the pronunciation of the labials /p, b, and m/.

3) **Class II, Division 1**: /p, b, m, s, and z and th/ are affected - all
sounds depending on the correct position of lips, incisors, and tongue. Subtelny and Subtelny (1962) identified the adaptive nature of strong lower lip action in extreme Class II, Division I cases during speech. Reducing the protrusion and then instituting a program of lip exercises to restore function to the upper lip usually eliminates the speech defect.

(4) Class III: /l/ and /v/ are likely to be defective because the upper incisors cannot readily touch the lower lips. These sounds then are said to be "inverted". Orthodontic or surgical correction of the structures would normally be the first step, followed by speech therapy, if still necessary.

Lisping (sigtamism) is the substitution of /s/ and /z/ by /th/. A full coverage of this topic is found in Vig (1973), Barrett and Hanson (1978), Weiss, Lillywhite, and Gordon (1980) and Jenkins (1978). Lisping may result from any of the above defects, particularly a recessive mandible in which the tongue is too far forward and protrudes between the upper and lower incisors instead of being behind them in attempting to pronounce /s/ or /z/. Another case is failure of the sides of the tongue to meet the molar teeth which allows some of the sound to escape laterally giving an impure /l/ sound. Lisping may be caused in denture wearers if the denture is too thick in the rugae area (Lawson and Bond, 1968). Of particular note to orthodontists is the fact that many removable appliances such as simple maxillary retainers can have a similar effect, producing a sigtamism.

Tulley (1964) found a statistical link between skeletal patterns and speech disorders. He found that 2.7% of the children in his study with a Class I skeletal pattern displayed a deviant articulatory pattern for the /s/ phoneme. Some evidence for the hypothesis that there is a relationship between structure and performance was observed: 20% of the children with a gross Class II pattern and 50% of those with a gross Class III pattern displayed a marked articulatory deviation.
Bond and Lawson (1968) have also commented on this association of skeletal pattern and speech. They point out that the tongue starts to move for speech from its resting position which is different in different individuals, "but appears to be related to the individual's skeletal pattern". Some individuals will have to move the tongue further than others to produce the same sounds, and consequently must move it more rapidly to speak at the same rate.

Another aspect of malocclusion cited by Jenkins (1978) was that of the abnormal position of any tooth, usually an incisor. It may interfere with the position of the tongue and cause abnormal speech sounds. For example, a lingual displacement of one incisor may cause whistles or hissing sounds when closed sounds, such as 'd' and 't' are intended.

Cleft Lip or Palate:

Graber (1972) outlined the speech problems associated with cleft lip and palate. The features which may be profoundly disturbed are:
- phonation,
- resonance, and
- articulation.

The factors contributing to loss of normal speech sounds are:
- velopharyngeal incompetence,
- naso-oral communication,
- abnormal palatal morphology,
- severe dental malocclusion,
- abnormal tongue posture and function, and
- pathologic lip involvement.

There is a limited range for success in speech therapy for these cases
despite surgical and orthodontic treatment. Much depends on the type of cleft deformity, the technique and timing of surgical assistance, the growth and developmental pattern, hearing involvement, and assistance from other services such as prosthetics and speech therapy. Cooper (1963) described programmes of rehabilitation and physical restoration for such patients. The whole defective speech pattern of cleft palate speech or "uranoschilalia" is covered by Kent and Schaaf (1982), who also identify specifically those speech sounds found to be affected by cleft lip and cleft palate separately. Weiss et al. (1980) pointed out that surgical repair of cleft palates at an early age (around 18 months) could cause difficulties in articulation-resonation. This is thought to be due to the lowering of the palatal vault associated with such surgery and consequent limitation of tongue movements for speech purposes.

Comprehensive reviews of cleft lip and/or palate speech can be found in the works of Morley (1970), Ross and Johnston (1978), Bzoch (1979), and McWilliams, Morris, and Shelton (1984).
4.3.3 Speech Defects and Tongue Thrust

Barrett and Hanson (1978) suggest that since normal articulation of speech is based, at least in part, on normal swallowing behaviour, it must follow that any gross malfunction of the musculature in basic function, as in tongue thrust swallowing, would be a strong influence toward misuse of these muscles in their secondary role. They also point out that if there is malocclusion accompanied by a speech defect, there is a strong possibility of tongue thrust. If present, the abnormal swallowing behaviour may be the basis for both of the other anomalies, or it may be an adaptive pattern. A careful judgment as to the order of importance of each item is necessary.

Fletcher, Casteel, and Bradley (1961) found that the subjects in their study, with a tongue thrust swallow were much more likely to have associated sibilant distortion than were the subjects without this pattern of swallowing. The range in incidence of articulation distortions with tongue thrust varies from 23.4% in this study, to even higher - 55.5% in the study of Ward, Malone, Jann, and Jann (1961). The variations in incidence are related to the ages of the subjects, criteria for determination of the tongue thrust, and the subjective criteria employed by the examiner in speech assessment. Fletcher, Casteel, and Bradley (1961) discussed the findings that tongue thrusting seemed to decrease with age, and that if there was no tongue thrust, sibilant distortion also decreased with age. Regardless of age, however, sibilant distortion appeared in a highly consistent manner in subjects with a tongue thrust. "This suggests that the tongue thrust pattern of oral activity, though perhaps adequate for vegetative functions, may present a barrier in acquisition and adequate production of certain consonants. It further suggests that spontaneous resolution of speech problems involving consonants that depend upon accurate placement is unlikely to occur in association with a prominent and consistent tongue-thrust swallowing pattern."
According to Goda (1968) while there can be a high percentage of agreement concerning the existence of an articulation disorder in a child with a severe speech disturbance, judgments will differ in the evaluation of the child with a mild speech disturbance particularly when judgement concerns adequacy of production of /s/ and /l/, two sounds affected principally by the thrust. This author also discusses 'tongue tie' position in tongue thrusters and non-tongue thrusters, and advocates a method by which he feels both articulation disorders and tongue thrust can be corrected together.

'Fronting' of the tongue in the production of the /s/ sound appears to be the major topic of discussion with authors when discussing tongue misplacement in speech associated with tongue thrust. The study of Subtelny and Subtelny (1962) described the increased fronting of the tongue in the "maladaptive" group of Class II, Division I subjects. This happened both at rest and during function. In the defective speakers the tie of the tongue slightly overlays the lower incisors at rest and protruded beyond the lower incisors during /s/ sound production. Here too the tongue could be seen often contacting the maxillary incisal edges or extending beyond the upper incisal edges, during /s/ sound production. This behaviour was deemed to be excessive fronting of the tongue tip (Fig. 174). Again Subtelny (1965) reviewed these findings, together with new data. He found that in subjects with Class II, Division I malocclusions those who had tongue thrust were much more likely to have an associated lisp. The incidence of lisping was twice as high among the tongue thrusters as it was among the non-thrusters. However 17% of the tongue thrusters had normal speech, and 17% of the non-thrusters had defective speech (Fig. 175). "On the basis of these findings, the relative adequacy of speech cannot be predicted on the basis of tongue-thrust swallowing and associated malocclusion of the Class II, Division I variety. A positive relationship between the two factors (tongue thrust and signatism) is
Fig. 174  Class II: tongue position at rest and with /s/ sound. (Subtelny and Subtelny, 1962)

Fig. 175  Speech in Class II, Division 1: Non-thrusters, thrusters. (Subtelny, 1965)
recognised. However, it seems hazardous to assume that the tongue thrust caused the defective speech.

According to Pierce (1978) some authorities consider tongue thrust to be a leading cause of speech problems. She mentions the "triad of oral anomalies": tongue thrust, malocclusion, and lisp, as being viewed as an entity. According to her, the sounds most likely to be misarticulated by tongue thrusters are the linguo-alveolar phonemes, /t, d, l, n, s, z, sh, ch, z, and dz/. These are the very sounds which should be produced by placing the anterior tongue against the alveolar ridge. The tongue thruster is likely to place the tongue too far forward for production of these sounds.

Barrett and Hanson (1978) cite different articulation problems and their relation to tongue thrust:

(1) **Lalling:** this is the distortion of sounds that require vertical lifting of the tongue tip /r, l, t, d/. The establishment of a normal rest posture, based on normal swallowing behaviour in which the tongue functions with some force in the anterior palatal region, would be indicated prior to any efforts made toward correction of the speech sounds.

(2) **Lisping:** frontal lisp and lateral lisp. **Frontal lisping** is made with excessive protrusion of the tongue; substitution of /th/ instead of /s/ and /z/. These authors stress a significant point regarding cause and effect in speech. The forces exerted, they say, during speech are too light in action, and too rapidly executed and released to have any bearing on the posture of oral structures. In those cases in which teeth are being "pushed around", it is probable that the abusive force is embodied in an abnormal pattern of swallowing accompanying the lisp.
The lateral lisp has a distinctive sound. The tip of the tongue pushes against the alveolar ridge or palate forcing the airstream over the sides of the tongue in the buccal region. The /s/ sound should be produced with the tongue tip in close proximity to the alveolar ridge, with the airstream directed through a small longitudinal groove down the midline of the tongue. A unilateral tongue thrust during swallowing is most often found together with the lateral lisp. These authors recommend establishing normal swallowing habits first which then corrects the lisp (which is normally regarded as being very difficult to correct).

Dentalisation of linguoalveolar sounds, /t, d, l, n/ and sibilant sounds: Many children with tongue thrust distort these sounds by placing the tip of the tongue against the upper incisors of the alveolar ridge, appearing to have "too much tongue". This forward positioning of the tongue usually becomes a positional problem leading to a thrusting swallow. Hanson (1979a) stressed the need to watch the tongue thruster speaking rather than to listen, since the fricative sounds /s/ and /z/ sound like /th/ but the linguo-alveolar consonants /t, d, n, and l/ may not be affected to any appreciable degree to the listener, yet by watching, the tongue can be seen protruding well forward interdentally.

Vig (1973) described the variations in the mode of articulation of dental fricatives /s and th/. He found that for /s/ sounds there could be two positions of the tongue, a high tongue position or a low tongue position. For /th/ there can be a dental position or an interdental position (Fig. 176). The findings of his study suggest that a high tongue position is more common than a low tongue position for /s/, and that a dental position is more common than an interdental position for /th/. In the same paper Vig found evidence to suggest that the rate of speech development is likely to be affected by the differential growth of the tongue and its rigid confines. Individual variation between children in acquiring a more favourable relation between tongue size
Fig. 176 Variations in articulation of /s/ and /th/. (Vig, 1973)
and intermaxillary space can thus influence their rate of speech development.

Suzuki, Sakuma, Michi, and Veno (1981) also found that the tongue protrudes interdentally in a manner different from that of healthy subjects, when the patient has an anterior open bite. They found that the abnormal articulatory movements were primarily the result of failure of contact between the upper and lower incisor teeth, and secondarily related to the mandibular prognathic deformity or malfunctions of the tongue in the subjects they studied.

Bloomer (1963) found that there could be a link between tongue thrust swallowing and speech defects. He related it to a neural dysfunction involving the cerebellum and related pathways. Discordinations of a neurogenic type could be detected using tests for oral disdiadokokinesia (lack of ability to perform consecutive movements in a controlled way). He drew a distinction between speech defects associated with either a delay in learning or maturation, and adaptations to malocclusions.

Other factors which could be held responsible for tongue thrusting in speech (Tulley, 1964) include: a receptor defect, poor auditory perception, high frequency loss; various defects in central control; minor variations in cortical control which could be inherited; and psychological disorders. Tongue thrusting with lisp, according to Tulley (1964) is usually due to one or more of the following: (1) Pure habits; (2) Built-in behaviour patterns of genetic origin (endogenous); (3) Adaptive behaviour patterns.

According to Goldberger (1978) tongue thrusting becomes a speech problem if, after a child can consciously place and move the tongue in the correct position to improve the speech, the correction of the sound cannot be
maintained due to the tongue thrust.

Hopkin and McEwen (1957) drew attention to the activity of the tongue when one is under emotional stress, saying that the forward protruding of the tongue under these circumstances can be responsible for speech defects such as lisping.

Proffit and Mason (1975) believed that a tongue thrust may or may not be associated with speech problems. This is in accord with the concept of compensation and adaptation previously mentioned. In order to find out the relation between the tongue thrust and the speech problem in a child, Rampp and Panbacker (1979) advocate the use of their "Tongue Thrust Articulation Test". This test, among other things, is claimed to be able to evaluate the spontaneous production of speech sounds that are more frequently defective in tongue thrust.

It is not clear whether tongue thrust causes malocclusion which in turn is responsible for a particular speech defect, or whether the malocclusion causes the tongue thrust which causes the speech defect. In this respect, Palmer (1948) was one of the earlier authors to advocate tongue swallowing exercises to help correct the speech problem in association with the malocclusion. Supporting this view, Whitman and Rankow (1961) strongly recommended correction of the swallowing behaviour before proceeding with the correction of the speech habit.

Weiss and van Houten (1972) recommended the incorporation of articulation therapy into the myofunctional therapy program for those patients exhibiting a tongue thrust and who have a frontal or interdental lisp or dentalisation of linguo-alveolar sounds.
In relation to age and maturation, Greulich (1972) points out that it is not always possible to predict whether a patient with speech problems and tongue thrust will grow out of them. "It is also probably dangerous to think of maturation in swallow patterns and think that changes in speech patterns will automatically follow."

Proffit and Mason (1975) recommend speech therapy using articulation techniques, for the correction of tongue thrust with associated speech problems - notably lisping. However they do not advocate specific tongue thrust therapy for these corrections. Dworkin and Culatta (1980) suggest on the basis of their studies on tongue strength, that tongue strengthening exercises may be superfluous to the correction of tongue thrusting or associated frontal lisping. However the other consonants such as /l, t, d, and n/ have been found (McGlone, Proffit, and Christiansen, 1967) to be produced with greater lingual pressures than the /s/ sound in lisping, and may differ in their response to training. Hanson (1976) disagrees with the assessment of Proffit and Mason (1975), and suggests that tongue thrust training is of benefit in cases of abnormal speech with tongue thrust. It is possible to correct frontal lisp when tongue thrusting is present without attending to the tongue thrust. However, the more subtle speech distortions - dentalisation of /t, d, l, n/ - apart from lisping distortions may need correction as they are often present. This can be achieved with tongue thrust therapy.

Pierce (1980), on this point, argues that these traditional articulation techniques advocated by Proffit and Mason (1975) do not always work. Quoting the work of Overstake (1975, 1976) she suggests that the correction of the swallowing pattern and of the habitual resting posture of the tongue may be sufficient to stimulate self-correction of articulation in many patients. "Simple" articulation cases may not be quite so easy to deal with if there is an
underlying tongue thrust problem. Recognition of this and appropriate therapy which takes into account the effectiveness of tongue retraining aspects is more likely to be successful. A study by Christensen and Hanson (1981) comparing the effectiveness of articulation services, to myofunctional therapy in the correction of articulation disorders, revealed that both methods were equally successful. The children in the sample exhibited both tongue thrust behaviours and articulation errors. Only those receiving myofunctional therapy, however, improved tongue thrust behaviours and articulation errors.

When a malocclusion is included in the association of tongue thrust and speech problems, Proffit and Mason (1975) do recommend modification of the resting posture of the tongue. "The tongue positioning exercises used in classic myofunctional therapy may be helpful." They also recommend articulation therapy techniques involving adaptive phonetic placements for repositioning the tongue tip posteriorly in these cases. In addition, they recommend early speech therapy before orthodontic treatment is begun. Furthermore, early orthodontic treatment may be indicated to make it easier for the child to achieve proper tongue placement for speech.
4.4 Relationships of Other Deviant Oral Behaviours and Disorders to Tongue Thrust

This part of the discussion is presented to complete the spectrum of associated factors involved in the relationship of tongue thrust and malocclusion. A brief review is presented which is aimed at including most of the other significant factors.

These 'behaviours and disorders' are presented in groups. The first two are related under the category of 'habits'; the next, related to disorders of occlusal equilibrium; the following three, to functional anatomical disorders; while the last is directly related to neurophysiological disorders.

4.4.1 Digit, Pacifier, and Non-nutritive Sucking

Digital sucking, as defined by Gellin (1978), is the placement of the thumb or one or more fingers to varying depths inside the mouth.

Pacifier sucking is defined in a similar way, while "non-nutritive sucking", in the terms of Larsson et al. (1985), is non-productive suckling of an infant at the breast.

Although the terms 'thumb', 'finger', 'digit', 'pacifier', and 'dummy' have all been used in the literature, here the term 'digit' is used to represent both thumb and any other finger used in such sucking; while 'pacifier' is used in preference to 'dummy' since its use in pacification of the infant by parent or guardian is its principal purpose.

Historically, authors such as Fluhrer (1950) and Klein (1952) have pointed out that bone, as a living tissue, is subject to change by environmental influences. The extent to which it can be changed is still controversial. These authors studied "abnormal pressure habits" and their part in the etiology
of malocclusion. Klein cited examples of intentional or planned pressures aimed at modifying an existing bony pattern for a particular reason:

1. Reshaping horns of cattle, e.g. downturned horns of Hereford cattle;
2. Chinese foot binding customs;
3. Giraffe-necks of Padaung women;
4. Intentional head deformation - North American Indian infants;
5. Myofunctional therapy; and

To these could be added the intentional pressure on soft tissues, e.g. increasingly larger wooden disc placement in the lower lips of South American ("Chukahoy") natives.

Klein also listed three classes of unintentional or "abnormal" orofacial pressure habits, some of which may be relevant to tongue thrust:

1. Intrinsic pressure habits (within the mouth);
   - Thumb-sucking
   - Finger-sucking
   - Tongue-sucking
   - Lip-sucking
   - Cheek-sucking
   - Blanket-sucking
   - Nail biting
   - Lip biting
   - Tongue biting
   - Mouth breathing.

2. Extrinsic pressure habits (face);
   - Chin propping
   - Face leaning on hand
- Abnormal pillow positions, leaning on forearm or hand
- Habitually sleeping on one side - deviated nose and possible deflected septum.

(3) **Functional** pressures, such as: the "bowlegged cowboy"; flattening of infants' skulls by lying on one side; and musical instrument playing and malocclusion. All in all Klein emphasised the orthopedic effects of abnormal pressure influences, pointing out that change is possible under the right circumstances. Moss (1962) was later to enhance and further extend this concept with his theories on growth and the "functional matrix".

One of the main factors associated with tongue thrust, throughout the literature, is thumb sucking. This has been covered to some extent previously in this discussion. Along with nasorespiratory obstruction, thumb sucking has been found to be one of the factors associated with the persistence of tongue thrusting throughout the mixed dentition stage (Hanson and Cohen, 1973). How are thumb or digit sucking related to tongue thrust, and how are these pressure influences related to changes in the dental occlusion? They must be viewed in context with other sucking activity.

As has been discussed previously, the newborn child exhibits a well-developed circumoral and intraoral muscular activity. Especially noticeable is the infant's tongue activity, which is manifested primarily in attempts to swallow and suck. During the first days of life, apart from sucking at feeding times, the child attempts to suck his/her fingers or a pacifier, if available. However, a sucking habit can hardly be considered established until it has continued for some time. An initial sucking habit is considered (Larsson and Dahlin, 1985) the daily habit when it is evident in the small child after several months. These authors identify three essential forms of the sucking habit;
(1) Digit-sucking, (2) Pacifier-sucking, and (3) Non-nutritive sucking.

Digit sucking has been viewed separately by some authors (Moyers, 1973; Subtelny and Subtelny, 1973). They attempted to classify digit sucking behaviour and the relative degree of associated oral malformation.

Larsson and Dahlin (1985), examining the overall relationship of digit and pacifier sucking behaviour to sucking in general, studied the initial pacifier- and digit-sucking prevalence in populations with geographic, cultural, and chronologic differences. They found that pacifier- and finger-sucking habits rarely occurred among the African children studied, except as symptoms of other disturbances in normal feeding and/or care. Their investigation of medieval skull material supported a similar finding among the Scandinavian children studied. However, they found that in the Western industrialised countries, most infants were pacifier- or digit-suckers. "This remarkable difference in the total prevalence of initial dummy- and finger-sucking habits among different cultures and epochs suggests that the modern western society contains factors of essential significance for the genesis of dummy- and finger-sucking habits."

They identified the "surplus sucking urge" which, they said, often remains after the child has first taken cereal or mother's milk. The extent of this surplus is dependent on the extent of the original urge and how much of it has been spent on the intake of nourishment. The surplus sucking urge, they suggested, may be either frustrated or re-channelled (Fig. 178). For the child, the most attractive method is unrestricted, sometimes non-nutritive, sucking at the breast. If this possibility is not available, the child must choose between pacifier- and digit-sucking to obtain satisfaction. If the surplus sucking urge is not so strong, it can probably be diverted and the child can
Fig. 178  Etiology of the different sucking habits.  (Larsson and Dahlin, 1985)

Fig. 179  Thumb sucking and tongue thrusting (82%).  (Subtelny and Subtelny, 1973)
find satisfaction through physical closeness and cuddling/stimulation.

Some time ago, and further to the work of Ballard (1951, 1952), Hovell (1955) reiterated the belief that continuous sucking habits, persisting beyond the age of 4 to 5 years, were always associated with a tongue thrust, "and it is this action which is responsible for the major part of the dental deformity". He said it was thought to be the main reason why digit sucking produced such widely varying dental deformities. He described how intermittent digit sucking would produce little effect on the teeth if normal muscle function and normal posture at rest was tending to place them in their normal position. "If, however, digit sucking is accompanied by an atypical swallowing action, then the one reinforces the other, and malocclusion will result, proportional in degree to the amount of abnormality of the swallowing action." He then emphasised the significance of night digit-sucking which is more intense and long-lasting.

Authors such as Jann, Ward, and Jann (1964) found no relationship between abnormal swallowing and digit sucking in children, while others such as Andersen (1963) found a significant relationship. Barrett and Hanson (1978) believed that digit sucking could be regarded as a possible, but relatively infrequent contributor to the development of tongue thrust, "clinically a small percentage of the tongue thrusters we see have a history of thumb or finger sucking". Hanson (1979a) mentioned that digit sucking tended to narrow the palatal arch, and "since the digit occupies the space needed by the tongue for normal swallowing, normal function is impossible while the sucking is occurring". Harden and Rydell (1984) in a recent study, found that digit sucking was more significantly associated with tongue thrust. Of their sample of tongue thrusters, 43-44% were digit suckers. Bowden (1966) found that after digit sucking had ceased, the increased overjet and reduced overbite,
seen previously, reverted toward more normal relationships. However, the overjet reverted rapidly, whereas the overbite reversion took between 3 and 5 years. He believed that from this latter finding, other factors may be involved, such as tongue thrust.

Further to this and regarding the association of tongue function, a few authors have presented their observations. Brenner (1974) described the detrimental effect of thumb sucking on the dental occlusion, and related structures. He also described the effect on the tongue, "if the upper arch is deprived of the support of the tongue by the downward pressure of the thumb, Class II, Division 1 malocclusion can develop".

Subtelny and Subtelny (1973), reporting on the work of Agin and Blankfort (1971) and Armbrecht (1972) on thumb sucking subjects, described the various patterns of thumb sucking and the prevalence of each type. More importantly, it was found in 82% of these cases, that "during the thumbsucking procedure", the tongue was found to be under and fully contacting the thumb, with the tongue tip approximating and pressing against the lingual aspect of the lower incisors (Fig. 179). In addition, "protrusive tongue activity was found to predominate" in these children. Dental malrelations were noted. These "seemed to be specific to the anterior region of the dental arch", with anterior open bite being most frequently observed, together with upper and lower incisor proclination. The tongue, they believed, "could be exerting a forward pressure on the lower incisors". However, they surmised that the protrusive tongue activity was the result, rather than the cause, of anterior malocclusion associated with thumb sucking. This was based on observations that some anterior open bite malocclusions have been corrected by either voluntary discontinuance, or after the placement of appliances, to eliminate the thumb sucking habit.
Proffit (1977) believed that the tongue thrust associated with digit sucking was really a reflection of the delay in the child's functional lingual development. He proposed that children who have sucking habits, i.e. digit sucking, move more slowly through the transitional stage of swallowing than children who do not have sucking habits. The tongue thrust, he said, was "merely an indication of some immaturity" in the child's swallow pattern.

However, prior to this statement, he had said (Proffit and Mason, 1975) that "a child who sucks his thumb apparently delays his transition toward adult swallowing". Furthermore, "a change in the swallow pattern will not occur until the sucking habit ceases", and that "some therapy to teach the child swallowing behaviour he has not yet learned, may be indicated". Unfortunately Proffit has yet to clarify these conflicting points of view.

In an earlier electromyographic study, Baril and Moyers (1960) found that malocclusion caused by digit sucking is more a matter of individual response to stimuli than a highly classified cause-and-effect syndrome. On a different note, Fletcher (1981) has proposed that since children who are digit suckers tend to have certain characteristics (such as a Class II, Division 1 malocclusion, an increased lower face height, a large overjet, a decreased overbite, together with a degree of lip incompetence and difficulty in swallowing "in the typical manner") these children then, adapt to this environment by digit sucking in an attempt to affect anterior oral seal, and to assist swallowing "in adverse anatomical circumstances". With regard to neural mechanisms involved in digit sucking, Dubner, Sessle, and Storey (1978) noted that this sucking behaviour does not conform to the conventional models of learning theory. Furthermore, while "the literature on human digit sucking is voluminous", very little is known of the underlying mechanisms.
Melsen, Stensgaard, and Pedersen (1979) seem to have produced the most comprehensive study to date on sucking habits and their influence on swallowing pattern and prevalence of malocclusion. Reviewing the literature, they noted that most studies had concentrated on the effects of prolonged sucking habits and indicated that irreversible malocclusions were produced if the sucking persisted beyond the age of four. The study of Melsen et al. indicated that previous sucking habits had a significant influence on the type of swallow. Finger- or thumb-sucking, and pacifier sucking all resulted in an increased tendency to tongue thrust, especially in the finger- or thumb-sucking group.

They also noted the role of pacifiers, some authors having recommended pacifier sucking in place of digit-sucking, believing that digit-sucking habits are more difficult to break. They pointed out though, that it has been found that "providing a child with a pacifier before any finger-sucking habit has started does create a non-nutritive sucking habit". Furthermore, "as a result, the frequency of sucking habits in the countries where pacifiers are commonly used is increasing significantly". Melsen et al. concluded that sucking habits, even of a short duration, may have an indirect effect through abnormal lingual behaviour in swallowing or position, as well as a direct effect on the developing occlusion. They found that using pacifiers instead of digits to lessen the likelihood of malocclusion was a misconception, since pacifiers, although less harmful in the short term, increased the total frequency of sucking habits. Melsen et al. recommended the use of mittens for a few months post-natally, "at times when the rooting and placing reflexes are maximal", i.e. when the baby is hungry. Larsson, however, would probably prefer longer periods of non-nutritive sucking at the breast or increased "cuddling/stimulation". The findings of Pearson (1948) and Golden (1978) tend to support this concept.
At least one other recent author has observed similar findings for pacifier usage. Schloemer (1984) found that in children aged between 3 and 6 years, a pacifier proved comparatively more advantageous in overcoming finger-sucking habits. However, he warned that pacifier use produced more frequent posterior crossbite with a narrower maxillary arch and a tendency to open bite, whereas thumb sucking alone resulted mainly in an increased overjet.

Overall, it seems reasonable to conclude that it would be better to eliminate or prevent these habitual sucking behaviours from becoming inground patterns of behaviour. If tongue thrust is enhanced or promoted by digit and pacifier sucking, then it would seem advisable to promote non-nutritive sucking and other forms of emotional support for the child at the earlier ages.
4.4.2 Other Oral Habits

Massengill, Quinn, Hall, and Boyd (1974) identified the following as often being present with tongue thrusters: nail-biting, lip-biting, frequent lip-licking, gum chewing, and the placement of foreign objects in the mouth. The following discussion will cover:

1. Lip habits
   a. Lip malposture,
   b. Lip and cheek biting,
   c. Lip licking, and
   d. Lip sucking;
2. Nail biting;
3. Foreign objects;
4. Parafunction; and
5. Others.

1. Lip habits
   a. Lip malposture. Barrett and Hanson (1978) believed this was caused by lip nonfunction in mouth breathing (dealt with previously), and lip malfunction in deglutation, as well as skeletal discrepancies such as extreme upper or lower protrusion, "which impel the lips in improper directions". Graber (1958) suggested that after digit habits have ceased, compensatory lip habits could take over, and could be "more potent than the strongest of digit habits". Barrett et al. believed that the most harmful of the lip habits were those of the lower lip, which is associated with mentalis muscle activity. The upper lip plays a significant role when it is short, making habitual lip closure difficult. This may contribute to functional and nonfunctional contacts between the lower lip and upper teeth. The lower lip could then push lingually on the upper anteriors causing them to tip labially (Warren, 1959). Graber (1958) identified the tongue as being a contributing factor as well, reposturing into the open bite or increased overjet relationship established previously with
digit sucking. He stressed that with the lip and tongue "constantly nestled" in the excessive overjet, even when there is no active function, "the teeth yield".

(b) **Lip and cheek biting.** Described by Straub (1962) and Barrett et al. (1978) as "infrequent" habits, they also point out that they are, periodically, a common reaction to stress.

(c) **Lip licking.** This habit also seems to be related to mouth breathing and drying of the lips (Barrett et al., 1978), or sometimes the result of "chronic nervousness". The action of the tongue in reaching the dry lips is protrusive, which may lead to a forward tongue posture, especially if it has to reach further out to the vermilion border of the lip to master it.

(d) **Lip sucking.** This is often regarded as an advanced stage of lip licking (Barrett et al., 1978). "Once the tongue has reached a comfortable limit of protrusion, the alternative of sucking the lip into the mouth is discovered." Hovell (1955) has also described the "tongue-lip" sucking habit. He believed that it replaced the sleeping digit sucking habit during the day, "which produces equally harmful results upon the occlusion". Moyers (1973) believed that lip sucking could appear with or without digit sucking. He found that the lower lip was involved "almost in all cases". This led to malocclusion because the lower lip was held beneath the maxillary anterior teeth bringing about their labioversion, together with an open bite, and sometimes linguo-version of the mandibular incisors.

(2) **Nail biting.** Fischer (1969) believed that nail biting was a variation of the digit sucking habit and may cause protruding upper anterior teeth, retruded lower anterior teeth, and an open bite. Moyers (1973) believed a more localised malocclusion was likely to be associated with nail biting, compared with other pressure habits. Barrett et al. (1978) suggested that it was possible that nail biting was the "chronological successor" to digit sucking as a sign of insecurity or nervous tension.
(3) Foreign objects. Authors like Straub (1962) identified the sucking and/or oral stimulation of pencil biting and foreign objects as possible contributors to malocclusion. In like manner to nail biting, the chewing of objects such as pencils, pens, pipe stems, and bobby pins, and the pressing of the fingers on the teeth, are regarded as having a psychogenic background, serving as outlets for emotional stress (Ramfjord and Ash, 1983).

(4) Parafuction - jaw clenching and bruxism. Klineberg (1983) describes parafuction as occurring consciously or unconsciously. Jaw clenching usually occurs unconsciously, being more common at night, but can occur for long hours during the day (Ramfjord and Ash, 1983). Bruxism is generally regarded as a nocturnal phenomenon, occurring primarily during deep sleep (Ramfjord et al., 1983). Both these entities are believed to be related to daytime stress and anxiety, and not evoked by tooth contact interferences. If interferences are present, an increase in muscle tonicity may be an indirect effect (Klineberg, 1983; Ramfjord et al., 1983). It is not yet known whether this increased muscle tonicity is related to tongue thrust behaviour. However, the incidence of bruxism appears to be elevated in children with brain damage (Lindquist, 1971). Tongue thrust incidence is also related to brain damage (discussed later) and there may be an association between these three factors.

(5) Others. Briefly, Straub (1962) mentioned tongue sucking during sleep. Nahoum (1977) cited a case of a patient with an open bite malocclusion who engaged in the activity of fist biting, presumably an extension of digit sucking. Wildman, Fletcher, and Cox (1979) described another disorder, food gulping. They found that most tongue thrusters exhibited this behaviour, unable to adequately control the bolus of food. Moyers (1973) related faulty body posture to faulty mandibular posture, suggesting that both may be expressions of poor general health. Posture, he believed, was "the summated expression of muscle reflexes". Moreover, a person with good erect vertebral posture will "almost reflexly hold his chin forward in a preferred position". The inference
is that there is more likely to be better oro-lingual muscle posture with better mandibular posture. Another habit mentioned in the literature (Fluhrer, 1950; Straub, 1961) believed to influence tooth position is chin leaning. Straub actually suggested that chin leaning could nullify the detrimental effects of tongue thrust behaviour. Finally, "rubbing and thrusting of the tongue against the teeth" can occur as a manifestation of an anxiety state in the "tense, apprehensive, pent-up individual" (Zucker, 1972). This response may become habitual and even compulsive in the more chronic states, particularly if the person is subjected to great emotional stress.

It would seem, in conclusion, that the oral habits and disorders mentioned could be regarded as possible contributing factors in the overall relationship between malocclusion and the persistence of tongue thrust behaviour. Therefore, they must be identified and evaluated in order that an accurate diagnosis can be made and appropriate treatment prescribed (Begg and Kesling, 1977). As Gianelly and Goldman (1971) point out, the stability of the result of orthodontic treatment is under threat if deviant behaviours are not eliminated, or at least modified to be as insignificant as possible.
4.4.3 Occlusal Disorders

Disorders of the dental occlusion include:

- Anterior open bite,
- Posterior open bite,
- Primary failure of eruption,
- Diastemata,
- Oligodontia, and
- Early loss of the deciduous teeth.

These disorders, by allowing spaces to appear, either interocclusally or intraocclusally, allow the tongue access to spaces, from which an abnormal lingual posturing or functioning behaviour can occur (Gingold, 1974; Pierce, 1978).

Another disorder is occlusal disharmony caused by severe occlusal prematurities in centric relation (Ramfjord and Ash, 1983). People with these prematurities avoid bringing their teeth together during swallowing, "they brace their jaw for the act of swallowing by placing their tongue between the teeth". Klineberg (1985) has also suggested that posterior premature contacts can exacerbate a tongue thrust swallowing pattern. Only by "bilateral and synchronous" contacts, all the way around the arch, can this artificial bracing of the mandible be resolved. Ramfjord (1961) also noted that by eliminating these prematurities, patients may assume a "tooth together swallow".
4.4.4 Oral, Pharyngeal, and Oesophageal Abnormalities

This group, which includes:

- Drooling,
- Pharyngeal pouches,
- Achalasias,
- Gagging,
- Retching,
- Vomiting, and
- Anaesthetic throat;

is included not only to add to other disorders, but also to expand on the range of oral and pharyngeal disorders previously discussed, and to cover some of the abnormalities associated with impairment of neural mechanisms, which are dealt with more fully in a later section. Most of the following information is taken from Dubner, Sessle, and Storey (1978).

**Drooling:** This disorder, especially in brain-damaged children, may be due to swallowing dysfunction. Salivary secretion rates should be determined before salivary duct ligation is contemplated. Dubner et al. stress that reducing salivary flow in cases of drooling due to swallowing dysfunction is contraindicated since normal swallowing is dependent on an adequate salivary stimulus.

**Pharyngeal Pouches:** The formation of these pharyngeal mucosal herniations may be associated with swallowing disorders, which are of interest because of the implication that the rigid patterning of the obligate muscles of swallowing (discussed previously) is subject to alteration. One theory regarding the etiology of pharyngeal pouches has been the delay or failure of relaxation of the hypopharyngeal sphincter in swallowing, with cricopharyngeal myotomy the corrective procedure. Although delayed relaxation seems not to be the cause, spasm resulting from oesophagitis frequently is. Swallowing is not a problem after total laryngectomy because the airway protective reflex is no
longer necessary. Under these conditions, there is evidence for learning in swallowing and the development of a new pattern in obligate swallowing muscles.

**Achalasias:** Failures of relaxation of the hypopharyngeal sphincter, gastro-oesophageal sphincter, and oesophagus may also lead to problems in swallowing. The first disorder has already been discussed, the second and third are beyond the scope of this discussion.

**Gagging, Retching, and Vomiting:** All three entities have been mentioned in the literature as being associated with swallowing activity. Gagging, a reflex initiated from the oropharynx, is usually thought to be a protective reflex of the alimentary tract and possibly of the upper airway. Barrett and Hanson (1978) have observed a decreased incidence of gagging in tongue thrusters. Dubner et al. suggest that the sensory input in the Trigeminal nerve during mastication and swallowing accounts for the suppression of gagging. Whereas gagging is conditioned, retching and vomiting are initiated by chemical stimulation of receptor sites in the medulla and the nodose ganglion. According to Dubner et al., although retching is sometimes used synonymously with gagging, its use is better restricted to non-productive vomiting. Gagging and vomiting are similar in their patterns of muscular activity and "show an orderly reversal of the activity seen in swallowing".

**Anaesthetic Throat:** "Anesthesia throat" (Dillon, 1945) described a throat which has no sensory nerve supply and is "immune to sensation" (Klein, 1952). There is no normal impulse to swallow and no gagging upon touching the soft palate, uvula, or any of the usual trigger points in the throat. Klein described how the patient, in order to swallow, must "seize the tongue between the anterior teeth and, by dint of muscular contortions involving even the face, perform the act of deglutition". The maintenance of an open bite was mentioned as a consequence of this behaviour. Barrett and Hanson (1978)
point out that, although the concept of this entity is losing adherents, there are still some who maintain that a "congenital physiological discrepancy, manifested in hyposensitivity of the velum, brings about abnormal handling of the bolus of food and thus tongue thrust". Furthermore, they point out that some believe that lack of sensation in some way produces the reaction of posterior tongue thrust upward against the teeth, to force the bolus into the pharynx. Describing anaesthetic throat as being a possible side effect of mouth breathing, "especially when combined with the undesirable oropharyngeal muscle patterns found in abnormal deglutition", they believe that it may or may not be a diagnostic indicator of tongue thrust.
4.4.5 Ankyloglossia

Ankyloglossia, or Tongue-Tie, is the condition in which a congenitally short lingual frenulum or frenum unites the tongue to the lingual mucosa of the mandible and floor of the mouth (Mathewson, Siegel, and McCanna, 1966). The frenum, which may be fibrous or muscular, often involves the underlying genioglossus muscle, the main muscle controlling the antero-posterior positioning of the tongue (as mentioned previously). In some patients, fibres of the genioglossus muscle are too short or they are attached in such a way that the tongue is held too low in the mouth, giving the appearance of macroglossia (Whitman and Rankow, 1961).

Diagnosis is sometimes difficult, and usually involves one or more of three methods (Robinson, 1983):

(1) Clinical observation of lingual function as the tongue is moved through its maximum range. On elevation, either actively, or passively for infants (Horton, Crawford, Adamson, and Ashbell, 1969), the dorsum is flat and dimpled with greater elevation on the lateral margins than on the tip. On protrusion there is notching of the tongue tip, and the range of movement is reduced. Wallace (1963) believed that in true ankyloglossia the tongue could not protrude beyond the lower incisors;

(2) Palpation of a tight genioglossus muscle on the undersurface of the tongue. This will help confirm the diagnosis; and

(3) Measurement of the length of the lingual frenum. This is discussed by Fletcher and Meldrum (1968).

Authors vary in their assessment of the incidence of ankyloglossia, and the significance of this to its early infantile development. Cullum (1959) and Nash (1963) believed that ankyloglossia interfered with suckling in early infancy.
There have been varying theories regarding the effect of ankyloglossia on speech production (Robinson, 1983). These aspects are still not resolved.

With regard to orthodontics, various authors (Tuerk and Lubit, 1959; Whitman et al., 1961; Horton et al., 1969; Goldberger, 1978a) have suggested that the inability to raise the tongue to the roof of the mouth may prevent the development of the mature swallowing pattern, and encourage the maintenance of the infantile or tongue thrust pattern. This, they believed, resulted in an open bite malocclusion. Whitman et al. suggested that in many cases the upper arch is underdeveloped and the lower arch is overdeveloped because of the "abnormally low" position of the tongue, "if the tongue is held a little higher in the mouth, a lateral tongue-thrust may be present, thus causing an open-bite in the buccal segments on one or both sides".

Frenectomy with or without genioglossotomotomy have been the usual methods of surgically correcting ankyloglossia (Douglas and Kresberg, 1954; Whitman et al., 1961; Catlin and De Haan, 1971). In addition, speech therapy and muscle retraining are believed to be necessary postoperatively (Block, 1968; Ketty and Sciullo, 1974; Ayers and Hilton, 1977; Goldberger, 1978a). The purpose of this therapy is firstly, to improve the range and control of lingual movement for correct articulation, and secondly, to correct the tongue thrust.
4.4.6 Macroglossia

Incidence and significance. Cole and Cole (1980) describe macroglossia as "a rather logical and obvious cause" of tongue thrust, but add that it does not appear to be a common condition. Straub (1951) suggested that tongue thrust may be "aided by unusually large tongues causing severe open bite cases".

There seems to be general agreement that macroglossia is a rare condition. Tulley (1964a) believed that tongue size was "not an important factor in most cases". He suggested that the tongue, "is never so large that it completely fills the oral cavity". However, a large tongue "may affect its posture and ease of manipulation". He believed that difficulties arise when the maxilla is small and a large tongue develops with a large mandible in a Skeletal III type. Tulley (1969) also believed that true macroglossia is "extremely rare".

Subtelny and Sakuda (1964) stressed the importance of tongue activity relative to tongue size, indicating that the activity was the determining factor in whether or not macroglossia would cause malocclusion, such as open bite. In many of these cases, they pointed out, evidence of some degree of neurologic damage is found, e.g. tongue activity in cases of cerebral palsy. Ballard (1953) had suggested previously that the tongue activity was the principal determinant of whether the tongue would cause a malocclusion or not. He believed that the actual size of the tongue "is of little importance", saying that a large tongue would not produce a malocclusion if it did not exhibit abnormal functional activity.

Barrett et al. (1978) also believed that true macroglossia is "a relatively rare condition, although many deviant swallowers give the impression
of having an oversized tongue". They suggested that the abnormally forward position of the tongue at rest and in function gives the impression that it is unusually large. They also believed that once "muscles have been retrained, particularly those of the posterior tongue and the velar region" the tongue assumes a much more normal appearance. Pierce (1978) also agrees with this, believing that "proper positioning" of the tongue posteriorly during swallowing, speech and at rest was the reason for the normal appearance. Goldberger (1978) pointed out that whether a true macroglossia is present or not is doubtful, since the tongue "being a muscle, can change with exercise".

Subtelny and Subtelny (1973) suggested that if the tongue is too large to be confined within the surrounding oral cavity, "the undesirable lingual posture and movement may result in an open bite deformity and defective speech". They stressed the point that excessive tongue dimension can preclude orthodontic correction or, in contrast to the preceding authors, myotherapy. "Without sufficient growth to accommodate lingual mass, it may be impossible to correct disproportionate lingual size via orthodontic or myotherapeutic procedures."

Brodie (1971) found that macroglossia was rarely the result of muscle-fibre hypertrophy, "more frequently, the cause seems to lie in the lymphatic tissue of the tongue", i.e. an abnormally large amount, in size and number, of lymphoid tissue - lymphangioma. These, he said, reduced in size with time, bringing the tongue back to a normal size relationship with the jaws. Brodie discussed at length the importance of an adequate pharyngeal airway, any encroachment on this space leading to forward tongue posture and lowering of the mandible. The association of lingual lymphatic tissue excess with macroglossia would tend to suggest a link with epipharyngeal lymph tissue excess related to nasorespiratory irritation or obstruction (discussed previously).
Associated malocclusions

Hovell (1955) described macroglossia as causing "double proclination" of the incisors, often with spacing. The buccal segments tend to drift forward, he believed, resulting in a "bimaxillary dento-alveolar prognathism". Graber (1972) showed an example of a dentition exhibiting the above-mentioned features as a result of macroglossia (Fig. 124).

Subtelny and Sakuda (1964) and Subtelny and Subtelny (1973) described the entities: (1) open bite,

(2) broadening of the arches,

(3) spacing, and

(4) proclination of the anterior teeth, resulting from macroglossia.

In particular, the open bite was said to result from the large tongue producing an open mandibular posture, thereby permitting continued eruption of the molars.

Bandy and Hunter (1969) found a "statistically significant correlation of 0.4" existing between measurable tongue volume and arch perimeter. However, using their method of measuring tongue volume, they found that the volume and length of the tongue had little, if any, influence on the width and length of the lower dental arch, on the degree of interincisal relationship, or on the angle of the lower incisor teeth to the mandibular plane.

Methods of identification and evaluation

Subtelny and Subtelny (1970, 1973) suggested three methods to determine disproportionate tongue size, (1) cineradiographic techniques, (2) cephalometry, and (3) by other clinical examinations "based on experience" such as observing the tongue at rest and in function, relative to the surrounding oral structures.
Previously, Subtelny and Sakuda (1964, 1966) had stressed the need for adequate diagnosis of macroglossia. They believed that the tongue did not always necessarily adapt to a smaller oral environment after orthodontic treatment. This, they said, often led to protrusive tongue activity following "excessive reduction in the tongue's environment". Furthermore, "thrusts of the tongue during swallowing can be caused or perpetuated rather than corrected if orthodontic therapy attempts to encroach on space that must be occupied by a comparatively large tongue".

Timms (1965) related the difficulty in treating these cases to the interaction of morphology and adaptation. He believed that in cases where the malocclusion cannot be corrected orthodontically, citing the examples of Subtelny and Sakuda (1964) and Ricketts (1962), there is a "morphological imbalance to an extent beyond the limits of adaptation".

Using their own method of measuring tongue volume, Bandy and Hunter (1969) found that the volume of the anterior portion of the tongue can be measured with an average accuracy of "2.3 c.c. ± 1.4 c.c.". Coincidentally, they found that the manifestation of scalloped lingual edges (mentioned by some authors) was not a reliable indicator of macroglossia.

Later, Subtelny (1970) stressed that macroglossia was a problem of proportionality, and suggested that proportionate sizes differ from person to person, "making it virtually impossible to develop a universal concept of tongue size that will fit all individuals".

Proffit and Mason (1975) described the growth of the tongue as found by Scammon et al. (1930). They pointed out that it grows "fairly steadily and approaches maximum size at or near age 8 years" (mentioned previously). The
mandible, meanwhile, takes longer to reach its maximum size, usually in the post-pubertal years.

Also on the subject of proportionality, Vig (1974), in a cross-sectional study of subjects at age 10 years and adult, found that while both the tongue and the intermaxillary space undergo considerable growth changes between the age of 10 years and adult, the tongue becomes relatively smaller when compared to the intermaxillary space.

Ardran and Kemp (1972b) studied tongue size using cinefluorography. They found that when the tongue was "large" (as opposed to "normal" or "small"):

1. The oral cavity is filled and the tongue may protrude over the gingivae or teeth;
2. There may be "a little air under the dome of the palate" but this is expelled or considerably reduced if the jaws are closed;
3. The pharyngeal airway may be considerably reduced in size and the vallecular fossae may be obliterated;
4. The body of the hyoid bone may be situated in a low position;
5. The epiglottis may be displaced posteriorly to significantly narrow the laryngeal vestibular airway; and
6. When swallowing, the hyoid bone is not elevated to the lower border of the mandible "as in the normal manner". They also found, clinically, that tongue protrusion at rest may or may not be a predominant feature.

Corrective measures

Briefly, surgical reduction of the tongue is required in some extreme cases (Subtelny and Subtelny, 1973), "if the large tongue presents anatomic and physiologic features considered insurmountable from an orthodontic viewpoint",
i.e. if sufficient jaw growth could not be anticipated to permit correction of the malocclusion. These authors describe two requirements of surgery:

(1) adequate removal of lingual tissue, and

(2) "generous growth potential [of the jaws] remaining".

If the observations of Brodie (1971) are reliable, reduction of tongue mass anteriorly will still allow for adequate functioning of the overlarge lymph tissue at the base of the tongue.

Subtelny (1970) also recommended, where further growth "cannot be hoped for" or "if adequate increase in oral cavity dimensions cannot be anticipated"; (1) **Tongue size reduction**, and/or (2) **Enlargement** of the oral cavity.

Subtelny and Sakuda (1964) believed that it may be "virtually impossible" to retrude teeth against a large tongue (Fig. 180). The author has witnessed such a case (Abbott, 1975) where this was found to be so. Reduction of the tongue mass eventually permitted the retraction of the anterior teeth.
Fig. 180  A and B,
Fig. 180  C and D. Case JWS: Bimaxillary dentoalveolar protrusion, with macroglossia and tongue thrust. Distinct incisor indentations can be seen in the tongue tip region. (Abbott, 1975)
4.4.7 Neurologic Impairment

A detailed discussion of the neurologic processes surrounding tongue function is found in a previous section. This last part is included to cover some of the dysfunctional aspects of neural regulating and controlling influences over lingual behaviour. Very little research appears to have been carried out on the direct links between neurologic impairment and tongue thrust. Much of what follows, therefore, could be regarded as speculative and/or conjectural. However, it would seem that there is adequate reason, in some cases, for authors to regard tongue thrust behaviour as being an unrecognised form of neurologic dysfunction or impairment caused either by traumatic, developmental, or hereditary influences.

Fletcher (1970) identified oroneuromotor deficiency, delayed maturation of infantile suckle-swallow tongue movements, and regression to a less mature phase of physiologic function, as some of the associated factors of tongue thrust behaviour. Goldberger (1978) suggested that the fine motor control and coordination necessary for "proper swallowing" was lacking in some people. Proffit (1972) suggested that the retention of a "completely infantile swallow" into childhood would probably indicate neurologic damage. Arvystas (1977) was another who identified "defective brain function", and a deficient oral sensory mechanism as being contributing factors in open bite, in connection with inadequate oro-linguo-facial muscle function. Rubin (1979, 1980) also identified damage to the central nervous system as being a major contributor to the "long face syndrome". Cerebral palsy patients, he noted, have "a characteristic long face and open bite possibly resulting from the reduced nerve supply to their muscles". Further, Cole and Cole (1980) identified "neuromotor incoordination" as an associated causal factor of tongue protrusion. This was related to "atypical motor patterning of the tongue".
The integrated action of the respiratory centre and about six cranial nerve nuclei (Figs. 72, 67) are required to carry a swallow through to completion (Doty, 1951; Hoffman and Hoffman, 1965). Neural mechanisms which initiate and control lingual behaviour in swallowing and in resting posture have been discussed, as well as occasional references to patients with neurologic impairment. What is the association between neurologic impairment and tongue thrust? Guerin (1984) stated that, with tongue thrusting in an older child, usually other primitive reflexes are also present, frequently caused by minimal cerebral palsy. Hoffman and Hoffman (1965) had made this point earlier. Quoting from Tulley (1962), they discussed "endogenous tongue thrust patterns", which were tongue thrust patterns associated with central nervous system dysfunction. Furthermore, in rare cases the orthodontist may be dealing with "subclinical cerebral palsy".

Scott (1969), commenting on Moss's functional matrix theory (1962), believed that it is the neural integrating system that creates and regulates the various functional components of the oral cavity. In particular, it is the neural component that determines the contribution of the tongue to oral function such as swallowing. He emphasised the role of neural connections: they "not only determine the patterns of muscle activity but probably also their growth, which, in turn, influences the development and growth of various parts of the craniofacial skeleton".

Gwynne-Evans (1948, 1951, 1954, 1956) appears to be the first of the modern authors to examine the nature of neural controlling mechanisms of orofacial muscle behaviour. He believed that oral muscle behaviour is predetermined, patterned, and dominated by the central nervous system. That is, there is an innate plan by which groups of muscles are progressively selected, coordinated, and controlled for the service of the person's future
activities. Moreover, he believed that there are "frequent delays in normal adjustments made in the progress toward higher levels of behaviour. Tongue thrust behaviour was believed to be such a delay, with some "stimulus" required to allow the central nervous system to resume a natural sequence of behaviour patterns. He was the first to suggest that in addition to hereditary factors of skeletal growth, there may exist inherited disturbances of muscle behaviour. While on the subject of hereditary influences, Jorgenson (1983) noted that the body of the tongue is composed of several muscles, "any one of which may be affected by neuromuscular disorders of genetic origin". Also, Warrer (1959), quoting Cauhepe (1955), suggested that "not only the individual anatomic configuration, but also the neuromuscular system which affects it, is inherited".

Returning to Gwynne-Evans, after failing to find this "stimulus" using the Andresen appliance, he advanced the idea of "visceral" as distinct from "somatic" swallowing. As discussed previously, he believed that the oral musculature occupied a position between the overriding influences of these two neural systems; that speech and normal swallowing came under cortical control, but that abnormal swallowing came under the control of lower centres. He decided that the latter or "visceral" tendencies could not be inhibited, and that an "atypical gradient of development" was projected into adult years, hence the concept of the "delayed infantile swallow".

Hopkin and McEwen (1957) quoting Ingram (1956), pointed out that it was "unusual to be able to produce true tongue protrusion after the age of nine months, apart from cerebral palsy cases where immature reflexes tend to persist because of slow development or in cases of mental defect". Furthermore, tongue thrust swallowing (the infantile form of swallowing) may persist for a very long time - "for life in cerebral palsy cases". It was suggested that
mental defect, cerebral palsy, and emotional retardation may be causes of tongue thrust.

Bosma (1963) related tongue function to oropharyngeal airway disturbances resulting from impairment of mechanisms of this area due to motor hypoplasias, as in the Robin syndrome, or in generalised coordinative disturbances, such as cerebral palsy.

Storey (1970) reported that swallowing is usually normal in lateral-facial dysplasia, but abnormal swallowing "may be seen in children following cerebro-vascular accidents". Tongue function, he said, appears to be affected when there is damage to the Hypoglossus motoneurones or cerebellum as a result of cerebral anoxia or kernicterus at birth and trauma later in life.

Likewise, Gershater (1972) identified "malfunction of the muscles due to poor neuromuscular control as a result of central nervous system disorders because of injury, disease, maldevelopment of the brain or inherited congenital causes". He found a high incidence of open bite malocclusion among the mentally retarded and emotionally disturbed children he studied. He mainly attributed the open bites to their "generally poor neuromuscular patterns and pernicious oral habits".

Subtelny and Subtelny (1973) suggested also that problems related to central nervous system control may influence tongue posture and function. They pointed out that in cerebral palsy, poor coordination of tongue, lip, and mandibular movements are observed. In addition, protrusive and erratic tongue movements are "frequently associated with marked deformities of the dental arches" such as open bite. More importantly to orthodontics is the aspect of "minimal central nervous system impairment" that they mentioned. This may
be expressed by reduced control of muscular movements within the oral and pharyngeal cavities. "Although previously unrecognised and undiagnosed, minimal neurologic damage can adversely affect tongue activity and may preclude any positive change in the surrounding environment attempted by the orthodontist". The clinical indications of minimal damage include: difficulty in movements and noticeable reduction in the rate of tongue movements (diadochokinetic impairment), e.g. pseudobulbar palsy; and observable lip incompetence and tongue control due to what appears to be hypotonicity of the muscles involved with poor control of mandibular posture and erratic tongue movements (with or without "adequate clearance of saliva").

Proffit and Mason (1975) considered neuromotor problems, severe enough to prevent normal adult swallowing, to be rare, "but this possibility should be considered in the evaluation of a patient". They suggested that poor muscular coordination in other movements, with or without mental retardation, increases the likelihood that the tongue will be affected neurologically. Furthermore, they stated that a "neurologic abnormality" may cause tongue thrusting. They related neuromotor patterning that has its development in the individual's interpretation of feedback experiences, to tongue thrust behaviour. Finally, they cautioned that the prognosis for any type of therapy in such children is poor, but they did not indicate the evidence used to support this suggestion.

Frankel (1980b) related muscle posture to central control when he suggested that the lack of anterior oral seal, often a characteristic of tongue thrusters, was a symptom of "behavioural immaturity" which pointed to the psychosocial aspects of poor postural control of the orofacial musculature. He believed that the acquisition of new muscular performance patterns was intimately related to central neurologic maturation. Further to this concept,
Frankel and Frankel (1983) pointed out that there are differences in the maturation rates of the tongue compared to the lip musculature, and that therapy may be indicated where the discrepancy of maturation became obvious. Drawing on the observations of Gershater (1972), they related the observations of orthopedists, who have argued that aberrant muscle tone does not constitute a physical problem alone, but "must also be seen in the context of the accompanying neural and psychic disturbances". Frankel and Frankel stressed (as has been mentioned) that the extensive representation of the oral region in the brain explains why emotional and nervous stresses are particularly manifested in the muscular environment around the mouth. From this neurological fact they suggested that the poor postural performances of the oro-linguo-facial muscles may be attributed to "an adverse psychosocial milieu of the affected child".

With regard to Down syndrome (Trisomy 21), Oster (1953), as quoted by Rozner (1983), found that over 60% of these individuals had fissured, large tongues. Later work by Ardran, Harker, and Kemp (1973) found tongue size in Down syndrome children to be not significantly different from that of "normal" children. However, and more relevantly, they do have generalised hypotonia and flaccidity of the oral musculature which could "give the illusion of macro-glossia, due to the sagging jaw and protruding tongue" (Dodd and Leahy, 1984).

According to Bazelon et al. (1968) as quoted by Ardran and Kemp (1972b), children with Down syndrome were grouped into five categories according to tongue protrusion. Following treatment with 5-hydroxytryptophan, "hypotonia was reversed and tongue protrusion was suppressed". They suggested that the impression of the large tongue described in Down syndrome may be the result of oral hypotonia with buccolingual dyskinesia, rather than an actual large size. Ardran and Kemp (1972b) also described the
appearance of large tongues occurring in patients with muscular dystrophies. In some of these patients "the tongue is truly relatively enlarged".

Malkin (1985) related dyslexia to impaired craniofacial growth. Although he did not specifically mention tongue function, his references to general muscle function might be applied to the tongue. Those with dyslexia, he pointed out, frequently are very awkward in their body movements, especially those movements involving fine motor coordination. In addition, a typical dyslexic person, along with other characteristics, will exhibit the following: (1) perceptual motor impairment(s), (2) general coordination deficits, (3) disorders of speech and hearing, (4) poor spatial orientation, and (5) impaired left-right discrimination. Vestibular and cerebral dysfunction are regarded as explanations for these characteristics and others. Malkin also described temporal lesions that have been found and believed to produce a subclinical or borderline alexic aphasia. This is exacerbated by vestibular induced cerebellar dysfunction. Further investigation would be required to substantiate a connection between tongue thrust behaviour and neurologic disorders such as dyslexia. However, there does seem to be a possible association.

Finally, from a study, using ultrasound, of tongue movements in speech, Shawker and Sonies (1984) found that patients with neurological disease and dysarthria showed "varying but significant" differences in tongue movements during articulation of certain sounds. Considering the relation of tongue thrust and speech defects (mentioned previously) this finding could support the concept of neurologic impairment being associated directly with tongue thrust.
CHAPTER 5 CONCLUSIONS

5.1 Existence of Tongue Thrust as an Entity
5.2 Diagnostic Significance of Tongue Thrust
5.3 Malocclusion Cause-and-Effect Relationships
5.4 Indications for Effective Therapy
5.5 Evaluation of Hypothesis and Recommendations
CHAPTER 5

CONCLUSIONS

5.1 Existence of Tongue Thrust as an Entity

It appears from the preceding review that there can be no doubt about the existence of the tongue thrust entity. What is still unclear is its causal nature. Tongue thrust behaviour can still be regarded as:

(a) The persistence of normal suckling behaviour into later life, especially in regard to the relationship between the tongue and perioral musculature at the time of the eruption of the permanent incisors;

(b) Inherent and inherited abnormal patterns of behaviour;

(c) The development of an abnormal habit;

(d) The failure of maturation, particularly in a neurological sense.

From this study it appears that tongue thrust is either:

(1) An organic entity with a major factor being a neurological disturbance or imbalance; or

(2) An adaptive behaviour particularly related to a significant inherent skeletal aberration or to an airway deficiency.
5.2 Diagnostic Significance of Tongue Thrust

Tulley (1964a, 1964b, 1969, 1973, 1980) has cited varying figures for the prevalence of tongue thrust. The form of tongue thrust which he has described as "endogenous", corresponding (it seems) to the 'organic' variety previously mentioned, appears to be the rarest kind. Tulley's incidence figures for those requiring orthodontic treatment also vary from article to article. However, if the most conservative figure is chosen - 1.35% of the general population (Tulley, 1980) - it appears that this may not be as insignificant as Tulley has suggested. For, when this figure is compared to other clinically detectable entities with a "high" incidence rate, for example - schizophrenia (1% of the general population) (Devesen, 1985), Tulley's interpretation may be questioned. Depending on the population size, an incidence of 1.35% would suggest to me a need for increased awareness in order that more effective methods of treatment may be delivered. This is in contrast to the belief of those such as Tulley who has suggested that half of these patients (approximately 0.7%) were beyond the scope of currently available methods of orthodontic treatment, and "would not respond to any kind of treatment". This attitude could be interpreted as one of professional neglect.

From my personal experiences of dealing with these patients, I have found that the rate of 1.35% is indeed a conservative estimate. The prevalence appears to be more common, especially in the less severe cases and if the range in manifestation that one may encounter is considered. I am sure that those speech pathologists and myofunctional therapists with whom I have worked and dealt (Riedl, 1980-82; Stough, 1980; Greene, 1980; Washington, 1982-83; Taylor, 1983-84; Kerr, 1984-85; and Robinson, 1984-85) would agree.

It is interesting to note that some of the more prominent authors
advocating myofunctional therapy procedures for tongue thrust correction have admitted to having been skeptical of the significance of tongue thrust. Hanson (1979b) mentioned that he had been opposed to, and skeptical of, treatment for tongue thrust, for over twelve years. Pierce (1978) and Pannbacker (Rampp and Pannbacker, 1979) were others. It may be likely that others will also change their opinions after gaining a greater awareness of aberrant lingual behaviour and the results of appropriate corrective procedures.