R.S. Nazarayn, T.G. Khmiz, V.V. Kuzina

PREVENTIVE AND INTERCEPTIVE ORTHODONTICS

Textbook

МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
Харківський національний медичний університет

R.S. Nazarayn, T.G. Khmiz, V.V. Kuzina

PREVENTIVE AND INTERCEPTIVE ORTHODONTICS

Textbook

Р.С. Назарян, Т.Г. Хмиз, В.В. Кузіна

ПРОФІЛАКТИЧНА ТА ПРЕВЕНТИВНА ОРТОДОНТІЯ

Навчальний посібник для англомовних студентів

Харків
ХНМУ
2023

УДК 616.314-089.23(075.8)
N32

Затверджено
Вченою радою ХНМУ.
Протокол № 1 від 26.01.2023.

Reviewer
L.F.Kaskova – MD, Professor (Poltava State Medical University)
I.V.Kovach – MD, Professor (Dnipro State Medical University).

Nazarayn R.S., Khmiz T.G., Kuzina V.V.
N32 Preventive and interceptive orthodontics: Textbook / R.S. Nazarayn and etc. Kharkov : KNMU, 2023. 152 p.

In the textbook covers the development of dentognatic systems, the characteristics of physiological and pathological types of occlusion, physiological and pathological types of the basic functions of the oral cavity. The changes in the dentognatic system after the impact of adverse factors, including oral habits, pathological functions of the oral cavity, untimely sanitation of the oral cavity are also described. Measures of prevention and treatment of pathological types of malocclusion in different periods of the development of dentognatic systems are described in detale.

The textbook is recommended for students of dental faculty, dentists.

Назарян Р.С., Хмиз Т.Г., Кузіна В.В.
Профілактична та превентивна ортодонтія: навчальний посібник для англомовних студентів / Р.С. Назарян та ін. Харків : ХНМУ, 2023. 152 с.

У навчальному посібнику описано розвиток зубощелепної системи, характеристики фізіологічного та патологічних видів прикусу, фізіологічний та аномалійний варіанти основних функцій порожнини рота. А також описана біомеханіка змін у зубощелепній системі при впливі несприятливих факторів, у тому числі шкідливих звичок, аномалійних функцій порожнини рота, несвоєчасної санації порожнини рота. Докладно викладено заходи профілактики та лікування патологічних видів прикусу у різні періоди формування зубощелепної системи.

Рекомендовано для студентів стоматологічних факультетів, лікарів-стоматологів.

УДК 616.314-089.23(075.8)

Preface

The orthodontics now recognize the importance of growth and associate it with etiology and the purpose of orthodontics to create and maintain a normal environment and proper physiologic activity of the teeth, the soft oral tissues, the facial and masticatory musculature, in order to ensure as far as possible optimum dentofacial development and function. The use of the word prevention before correction symbolizes the need to diagnose a problem as soon as possible.

Preventive orthodontics, as the name implies, is action taken to preserve the integrity of what appears to be the normal occlusion at a specific time. Preventive orthodontics requires the ability to appraise normal dentofacial and general development and growth and the recognition of deviations from the normal. It entails the elimination of deleterious local habits involving dentofacial structures; the correction of general contributory causes, such as incorrect posture and malnutrition; the maintenance of tooth form by proper restoration of individual teeth; timely removal of retained deciduous teeth; use of space maintainers after premature loss of deciduous teeth, if indicated, and reference for treatment of related affections and abnormalities to other specialists.

Interceptive orthodontics is action taken to employ to recognize and eliminate potential irregularities and malpositions in the developing dentofacial complex. This phase specifically concentrates on its efforts towards improving environmental conditions to permit future normal development. The preventive measures envisaged may include caries control, anatomical dental restorations, space maintenance, transitory oral habit correction, genetic and congenital anomalies, and supervising the exfoliation of deciduous teeth. Certain procedures under the preventive and interceptive orthodontic fields may overlap. Hence, at times it may not be possible to segregate the two; however, interception always recognizes the existence of a malocclusion or malformation whereas the prevention is aimed at preventing the malocclusion or malformation from occurring.

The solution of the problem of high prevalence of dentognatic anomalies can be realized only during a skilled prevention of dentognatic anomalies, whose main goal is the timely elimination of the causes and conditions for the emer-gence and development of the disease. That is why orthodontic prophylaxis consists of a set of interrelated organizational, treatment and preventive activities. Knowledge of theoretical and practical framework for prevention of dentognatic anomalies are required for the orthodontist, pediatric dentist and the dentist of general practice.

Chapter 1. Introduction to orthodontics

What does the common man associate with the term orthodontics? It is a special branch of dentistry which deals with the alignment of teeth. What people perceive of this branch of dentistry is what we as dentists project of the capabilities of an orthodontist.

Orthodontics has been defined as 'a branch of science and art of dentistry which deals with the developmental and positional anomalies of the teeth and the jaws as they affect oral health and the physical, esthetic and mental well being of the person". This definition may be over sixty years old yet even at that time the potential of orthodontics was not lost. The emphasis is on maintenance of oral, physical and mental health of the patient and also his/her esthetics.

Yet, this was not the first definition proposed. Definitions proposed earlier had also recognized the importance of the knowledge of growth and the essentiality of correcting aberrations as early as possible. In 1911, Noyes defined orthodontics as "the study of the relation of the teeth to the development of the face, and the correction of arrested and perverted development."

This definition was further modified when in 1922 the British Society of Orthodontists proposed that "Orthodontics includes the study of growth and development of the jaws and face particularly, and the body generally, as influencing the position of the teeth; the study of action and reaction of internal and external influences on the development, and the prevention and correction of arrested and perverted development".

The definitions now recognize the importance of growth but also associate it with etiology and the purpose of orthodontics to create and maintain a normal environment and proper physiologic activity of the teeth, the soft oral tissues, the facial and masticatory musculature, in order to ensure as far as possible optimum dentofacia1 development and function. The use of the word prevention before correction symbolizes the need to diagnose a problem as soon as possible.

The definition of orthodontics proposed by the American Board of Orthodontics (ABO) and later adopted by the American Association of Orthodontists states:

"Orthodontics is that specific area of the dental profession that has as its responsibility the study and supervision of the growth and development of the dentition and its related anatomical structures from birth to dental maturity, including all preventive and corrective procedures of dental irregularities requiring the repositioning of teeth by functional and mechanical means to establish normal occlusion and pleasing facial contours."

The American Board of Orthodontics' definition recognizes the capability of the orthodontists in being able to change the profile by not only moving teeth but also by redirecting growth using functional appliances. Hence, we see that the science has evolved over the years and its scope has been increasing along with our increased knowledge of the underlying biological principles of growth and development.

Branches of orthodontics. The art and science of orthodontics can be divided into three categories based on the nature and time of intervention.

- Preventive orthodontics
- Interceptive orthodontics
- Corrective orthodontics.

Preventive orthodontics. Preventive orthodontics, as the name implies, is action taken to preserve the integrity of what appears to be the normal occlusion at a specific time. Preventive orthodontics requires the ability to appraise normal dentofacial and general development and growth and the recognition of deviations from the normal. It entails the elimination of deleterious local habits involving dentofacial structures; the correction of general contributory causes, such as incorrect posture and malnutrition; the maintenance of tooth form by proper restoration of individual teeth; timely removal of retained deciduous teeth; use of space maintainers after premature loss of deciduous teeth, if indicated, and reference for treatment of related affections and abnormalities to other specialists.

Interceptive orthodontics. According to the definition given in the brochure on orthodontics by the American Association of Orthodontists, Council of Orthodontic Education, is "that phase of the science and art of orthodontics, employed to recognize and eliminate potential irregularities and malpositions in the developing dentofacial complex". This phase specifically concentrates on its efforts towards improving environmental conditions to permit future normal development. The preventive measures envisaged may include caries control, anatomical dental restorations, space maintenance, transitory oral habit correction, genetic and congenital anomalies, and supervising the exfoliation of deciduous teeth. Certain procedures under the preventive and interceptive orthodontic fields may overlap. Hence, at times it may not be possible to segregate the two, however, interception always recognizes the existence of a malocclusion or malformation whereas the prevention is aimed at preventing the malocclusion or malformation from occurring.

Corrective orthodontics. Corrective orthodontics, like interceptive orthodontics, recognizes the existence of a malocclusion and the need for employing certain technical procedures to reduce or eliminate the problem and the attendant sequelae. The procedures employed in correction may be mechanical, functional or surgical in nature.

Aims of orthodontic treatment. The treatment provided should not only satisfy the patient's esthetic desires but also satisfy certain functional and physiologic requirements. Jackson had summarized the aims of orthodontic treatment as:

- Functional efficiency
- Structural balance
- Esthetic harmony

These three are now famous as the Jackson's triad.

Functional efficiency. The teeth along with their surrounding structures are required to perform certain important functions. The orthodontic treatment should increase the efficiency of the functions performed by the stomatognathic system.

Structural balance. The structures affected by the orthodontic treatment include, not only the teeth but also the surrounding soft tissue envelop and the associated skeletal structures. The treatment should maintain a balance between these structures, and the correction of one should not be detrimental to the health of another.

Esthetic harmony. The orthodontic treatment should increase the overall esthetic appeal of the individual. This might just require the alignment of certain teeth or the forward movement of the complete jaw including its basal bone. The aim is to get results which gel with the patient's personality and make him/her to look more esthetic.

Scope of orthodontics. Orthodontic treatment is aimed at moving teeth, orthopedic change and altering the soft tissue envelop.

Moving teeth. The main reason for the existence of this specialty was it's capability of moving teeth. Moving teeth without any deleterious effects into more ideal locations is what everyone always associates this field with. How efficiently this can be undertaken and to what extent, depends upon the nature of the malocclusion and the capability of each individual clinician.

Orthopedic change. Using functional appliances and the latest orthognathic techniques, it is possible to move entire jaws into more favorable positions. It is very much within the capabilities of an orthodontist to use appliances at times in conjunction with other specialists to move the entire jaws along with its basal bone and the soft tissue envelop to achieve the objectives of treatment.

Altering the soft tissue envelop. The functions performed by the soft tissue envelop of the teeth and the oral cavity have a definite impact on the growth and development of the oral and facial structures. The orthodontist can help retain or restrain the soft tissues and or bring about a change in them by altering the position of the teeth or the jaws. The various functional appliances and at times habit breaking appliances may be used along with other treatment procedures.

Chapter 2. Preventive of dentognathic anomalies development during prenatal and postnatal period

2.1. Prenatal growth of cranium, facial and oral structures

Prenatal growth phases. The prenatal life of an individual can be broadly classified into the following three phases:

1. Period of ovum (from fertilization to the 14th day)
2. Period of embryo (from 14th to 56th day)
3. Period of fetus (56th day to birth)

Prenatal growth proper. The growth of the cranial, facial and oral structures begins around the 21st day (period of embryo) after conception. At this stage the embryo is about 3 mm in size and the head begins to take shape. *Figure 2.1* (A-E) explains clearly the sequence of events.

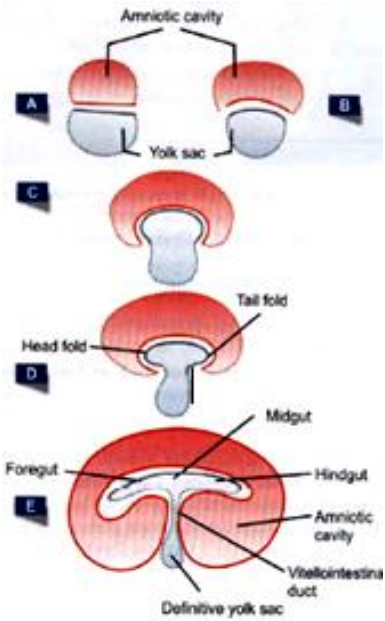

Fig. 2. 1. (A-E). Formation of the head and tail folds

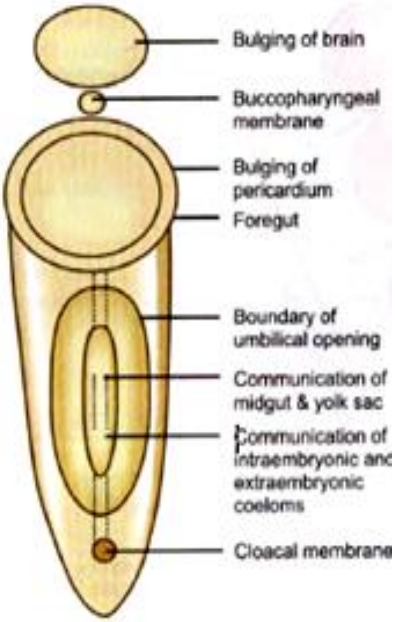

Fig. 2.2. Prominent brain and pericardium buige

Although there is progressive increase in the size of the embryonic disc, yet, the head and tail ends of the disc (x, y) remain relatively close together. This results in the disc to bulge upwards into the amniotic cavity. As the disc enlarges further, the embryonic disc becomes folded on itself at the head and tail ends. These are called the head and tail folds.

After the formation of head fold, the developing brain and the pericardium forms two prominent bulging in the ventral aspect of the embryo *(Fig. 2.2).*

In between them, there is a depression called the stomodaeum, the floor of which is formed by the buccopharyngeal membrane. This membrane separates the stomodaeum from the foregut (*Fig. 2.3).*

Soon the mesoderm covering the developing brain proliferates and forms a downward projection that overlaps the upper part of the stomodaeum. This downward projection is called the frontonasal process *(Fig. 2.4).*

As is evident till now, the neck is not yet present. The neck is formed by the elongation of the region between stomodaeum and the pericardium. This is achieved partly by a descent of the developing heart and mainly due to the appearance of a series of mesodermal thickenings in the wall of the cranial most part of the foregut. These are called the pharyngeal or the branchial arch *(Fig. 2.5).*

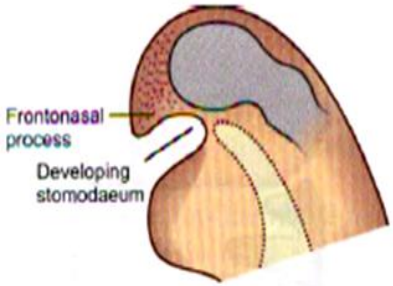

Fig. 2.3. Appearance of stomodaeum

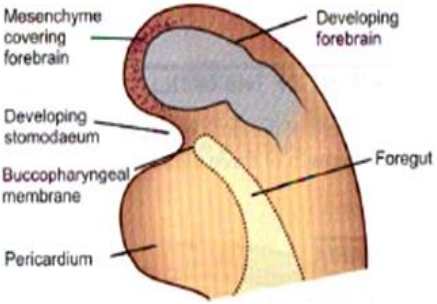

Fig. 2.4. Formation of frontonasal process

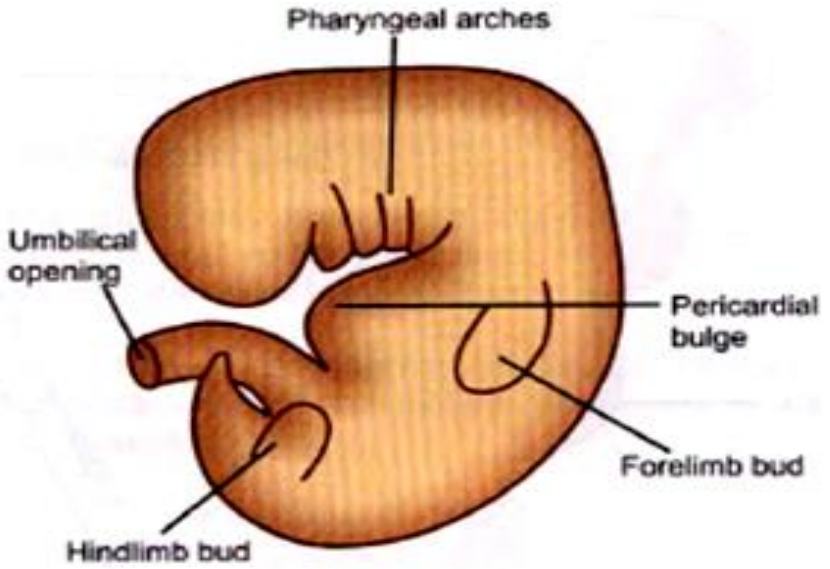

Fig. 2.5. Pharyngeal arches

These are 4 in number. Although there is a 5th arch, it disappears soon after formation *(Fig. 2.6).* Only the first two arches are named; the mandibular arch and the hyoid arch respectively. Each of these arches is separated by a groove and is supplied by a nerve that innervates the striated muscle of the arch along with the overlying ectoderm and endoderm.

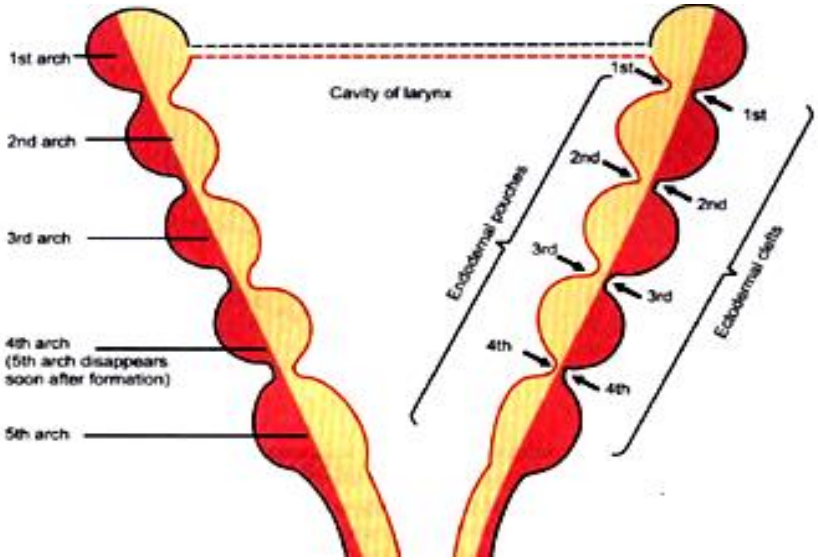

Fig. 2.6. Formation of pharyngeal arches

At this stage, the mid-sagittal section of the embryo looks like *Fig. 2.7.*

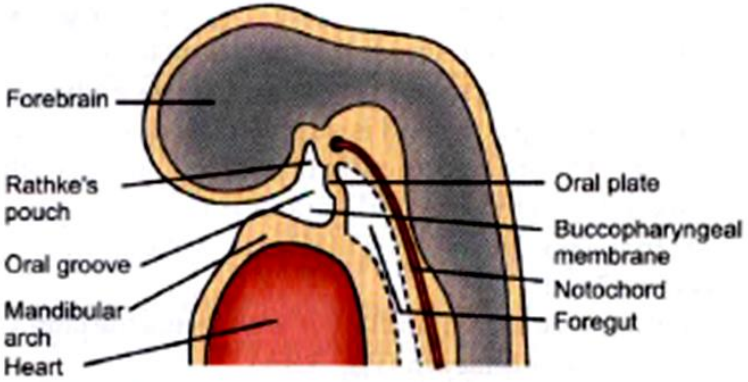

Fig. 2.7. Mid-sagittal section of the embryo (21 days)

At this stage each mandibular arch forms the lateral wall of the stomodaeum *(Fig. 2.8.A).* This arch gives off a bud from its dorsal end and is called the maxillary process *(Fig. 2.8.B).* It grows ventromedially cranial to the main part of the arch which is now called the mandibular process.

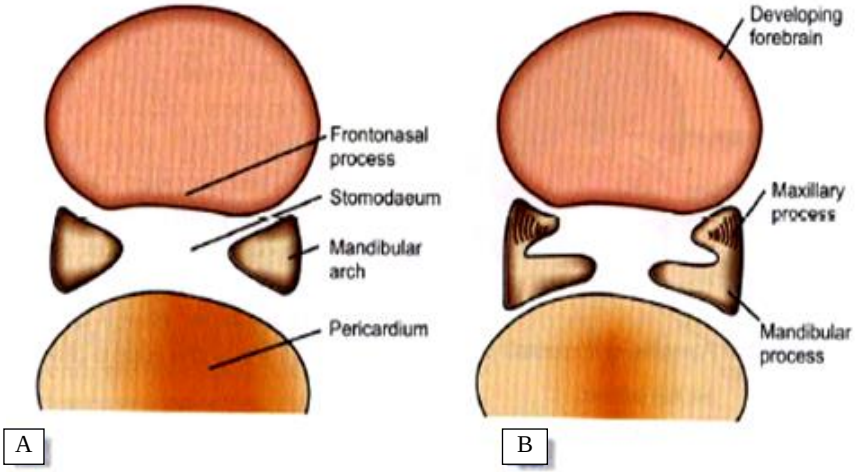

Fig. 2.8. Formation of maxillary process

The ectoderm overlying the frontonasal process soon shows bilateral localized thickenings that are situated a little above the stomodaeum.

These are called nasal placodes and they soon sink below the surface to form nasal pits *(Fig. 2.9. A-B).* The pits are continuous below with the stomodaeum. The edges of each pit are raised above the surface; the medial raised edge is called the medial nasal process and the lateral edge is called the lateral nasal process.

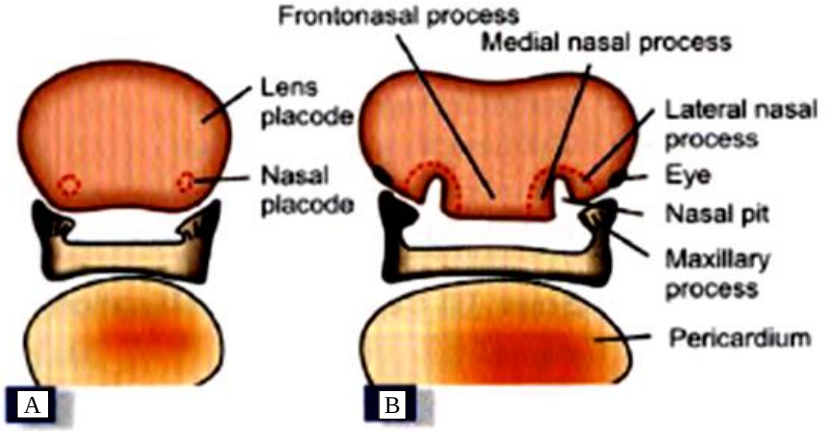

Fig. 2.9. Formation of nasal placodes and nasal processes

The facial skeleton. For the sake of convenience, the face may be divided into following thirds:

1. The upper face (corresponding to frontonasal process, embryologically).
2. The middle face (corresponding to maxillary process, embryologically).
3. The lower face (corresponding to mandibular process, embryologically).

Their boundaries being approximately the horizontal planes passing through the pupils of the eyes and the rima oris.

The upper third of the face, which is primarily of neurocranial composition, initially grows most rapidly in keeping with its neurocrania I association and the growth of the frontal lobes of the brain. In contrast, the growth of middle and lower third is slow and fairly prolonged.

The facial bones develop intramembranously from ossification centers in the neural crest mesenchyme of the embryonic facial processes. The above can be tabulated as follows:

The maxilla. A primary intramembranous ossification center appears for each maxilla in the 8th week IU at the termination of the infraorbital nerve just above the canine tooth dental lamina. Secondary cartilages appear at the end of the 8th week IU in the regions of the zygomatic and alveolar processes that rapidly ossify and fuse with the primary intramembranous center. Two further intramembranous' premaxillary centers' appear anteriorly on each side in the 8th week IU and rapidly fuse with the primary maxillary center.

Single ossification centers appear for each of the zygomatic bones and the squamous portions of the temporal bones in the 8th week IU.

The palate. The growth and development of the palate holds special interest for the orthodontist. The three elements that make up the secondary definitive palate are:

1. Lateral maxillary processes
2. Primary palate of the frontonasal process.

These are initially widely separated due to the vertical orientation of the lateral shelves on either side of the tongue. Later in the 7th week IU (between the 47th and 54th day) a remarkable transformation in position of the lateral shelves takes place, when they alter from vertical to horizontal, as a prelude to their fusion and partitioning the oronasal chamber.

Ossification of the palate proceeds during the 8th week IU from the spread of the bone into the mesenchyme of the fused lateral palatal shelves and from trabeculae appearing in the primary palate as' premaxillary centers'. all derived from the single primary ossification centers of the maxillae.

Posteriorly hard palate is ossified from the trabeculae spreading from the single primary ossification centers of each of the palatine bones. Mid-palatal sutural structure is first evident at around 10th week IU when an upper layer of fiber bundles develops across the midline.

In the most posterior part of the palate, ossification does not occur, giving rise to the region of soft palate.

A cleft of the palate occurs if the palatal shelves fail to fuse together as may happen if the tongue fails to descent due to underdevelopment of the mandible. Incomplete penetration of the mesoderm into the palatal shelves can give rise to a submucous cleft palate. Thus the formation of a cleft lip and alveolus (primary palate) occurs between the 4th and 8th week after conception and clefts of the hard and soft palate (secondary palate) occurs between the 8th and 12th week. A complete cleft of the lip, alveolus and palate would therefore, suggest a continuation of the effects of the etiological factors over all these weeks while clefts of the primary or secondary palate alone would imply its restriction to the appropriate weeks.

It is now well understood that anyone of the following reasons can result in cleft palate.

- Defective growth of the palatine shelves
- Failure of elevation of the shelves
- Failure of fusion of the shelves
- Postfusion rupture of the shelves
- Micrognathia as in Robin Complex

The Mandible. The first structure to develop in the primordium of the lower jaw is the mandibular division of the trigeminal nerve that preceded the mesenchymal condensation forming the first (mandibular) arch *(Fig.2 10).* The prior presence of the nerve has been postulated as being necessary to induce osteogenesis by the production of neurotrophic factors.

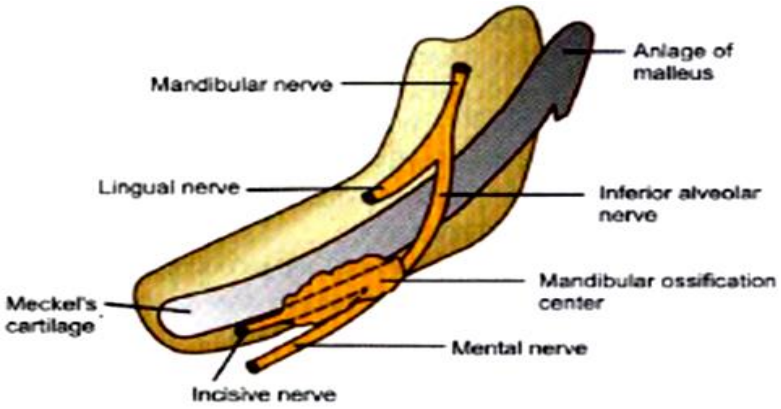

Fig. 2.10. Schematic representation of center of ossification of the mandible lateral to Meckel's cartilage at the bifurcation of the inferior nerve

The mandible is derived from the ossification of an osteogenic membrane formed from ectomesenchymal condensation at around 36 to 38 days TU. The resulting intramembranous bone lies lateral to Meckel's cartilage of the first (mandibular) arch.

A single ossification center for each half of the mandible arises in the 6th week TU, in the region of the bifurcation of the inferior alveolar nerve and artery into the mental and incisive branches. There is marked acceleration of mandibular growth between the 8th and 12th weeks IU. As a result of mandibular length increase, the external auditory meatus appears to move posteriorly.

Bone begins to develop lateral to Meckel's cartilage during the 7th week and continues until the posterior aspect is covered with bone. Ossification stops at the point, which will later become the mandibular lingula, and the remaining part of the Meckel's cartilage continues on its own to form the sphenomandibular ligament and the spinous process of the sphenoid (Meckel's cartilage lacks the enzyme phosphatase found in the ossifying cartilages, thus precluding its ossification. Meckel's cartilage does, however, persists until as long as the 24th week IV, before it disappears).

Secondary accessory cartilages appear between thelOth and 14th weeks IU to form the head of the condyle, part of the coronoid process, and the mental protuberance *(Fig. 2.11).*

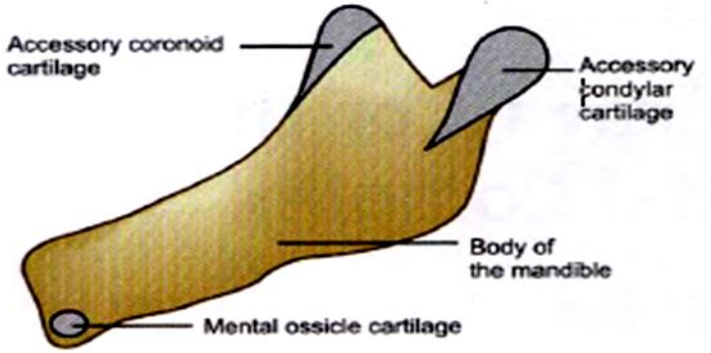

Fig. 2.11. Accessory cartilages of the fetal mandibl

2.2. Postnatal growth of the craniofacial complex

For the basis of simplicity, the growth of the craniofacial complex can be divided into four areas that grow rather differently:

a. The cranial vault the bone that covers the upper and outer surface of the brain.

b. The cranial base the bony floor under the brain, which is also a dividing line between the cranium and the face.

c. The nasomaxillary complex made up of the nose, maxilla, and the associated structures.

d. The mandible.

The cranial vault. The growth in the cranial vault is because of the enlarging brain. The rate of bone growth is more during infancy and by the fifth year of life more than 90 percent of the growth of cranial vault is achieved. It is made

up of a number of flat bones that are formed directly by intramembranous ossification, without cartilaginous precursors. From the time that ossification begins at a number of centers that foreshadow the eventual anatomic bony units, the growth process is entirely the result of periosteal activity at the surfaces of the bones. Some selective resorption occurs early in postnatal life on the inner surfaces of the cranial bones to help flatten them out as they expand. Apposition can be seen on both the internal and external tables of the cranial bones as they become thicker. This increase in thickness which permits the development of the diploe is not uniform. Sicher attributes this to the fact that the inner cranial table is primarily under the influence of the growth of the brain-the brain capsule while the outer plate has certain mechanical influences operating upon it. These mechanical influences contribute to the growth of cranial superstructures. Of particular significance are the supraorbital, otic, and mastoid regions. These structures are usually more marked in the males than females.

Remodeling and growth occur primarily at the periosteum lined contact areas between adjacent skull bone, called the skeletal sutures. At birth, the flat bones of the skull are rather widely separated by relatively loose connective tissues. These open spaces, the fontanelles *(Fig. 2.12–14)*, allow a considerable amount of deformation of the skull at birth-a fact which is important in allowing the relatively large head to pass through the birth canal.

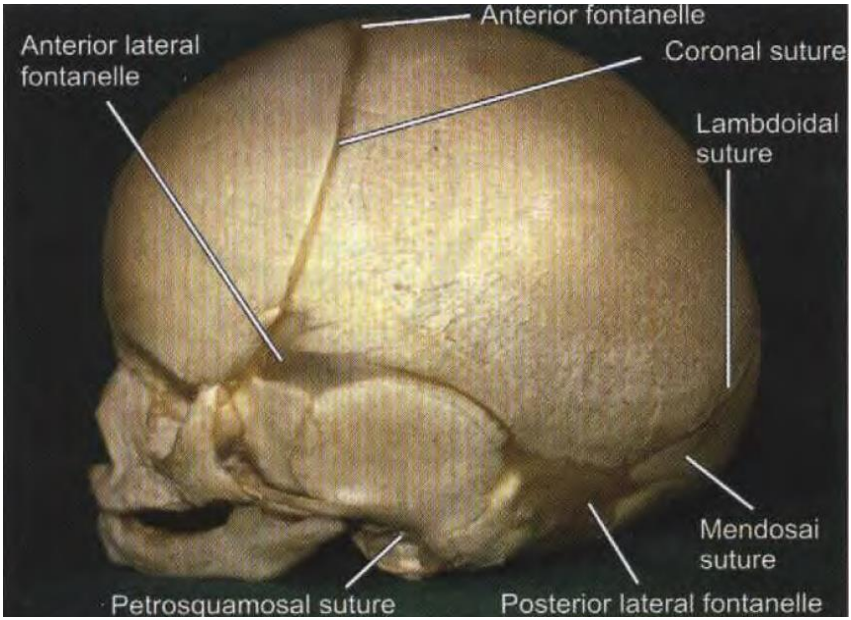

Fig. 2.12: The location of the various fontanelles (lateral view)

After birth, apposition of bone along the edges of the fontanelles eliminates these open spaces fairly quickly, but the bones remain separated by a thin periosteum lined suture for many years, eventually fusing in adult life.

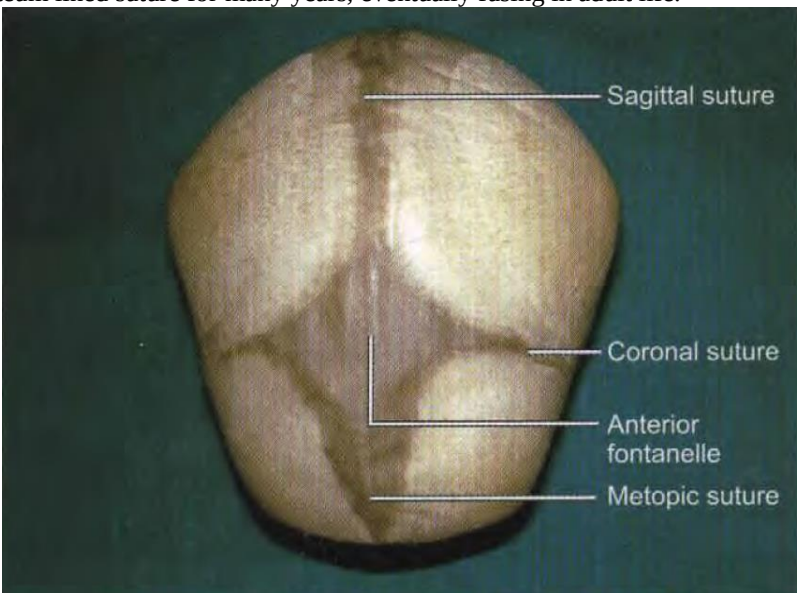

Fig. 2.13: Location of the various fontanelles (superior view)

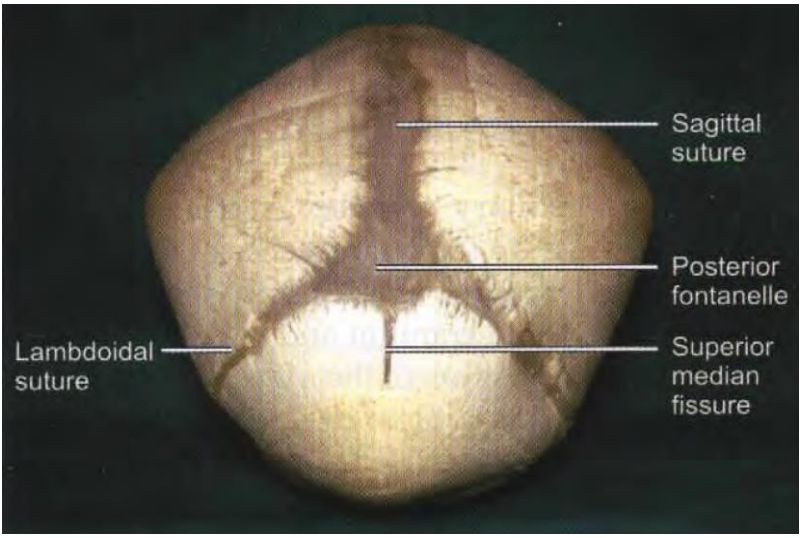

Fig. 2.14. Location of the various fontanelles (posterior view)

The newborn not only has his frontal bone separated by the soon to close metopic suture, but also has no frontal sinuses. Both the inner and outer surfaces are quite parallel and quite close to each other. With the general growth and thickening of the cranial vault there is an increase in the distance between the internal and external plates in the in the supraorbital region. This may be seen on the external surface as a ridge. The spongy bone between the external plates is gradually replaced by the developing frontal sinus.

The cranial vault increases in width primarily through 'fill in' ossification of the proliferating connective tissue in the coronal, lambdoidal, interparietal, parieto-sphenoidal and parietotemporal sutures. It should be realized that there is actual translation as well as remodeling of the individual bones, with the structures being moved outward by the growing brain. Despite early accomplishment of the pattern, the parietal bones do not close until the middle of third decade of life.

Increase in length of the brain case may be primarily due to the growth of the cranial base with active response at the coronal suture.

Height of the brain case is due to the activity of the parietal sutures along with the occipital, temporal, and sphenoidal contiguous osseous structures.

The cranial base. The cranial base, unlike cranial vault, is not completely dependent on brain growth and may have some intrinsic genetic guidance and a pattern that is, similar in some dimensions, to that of the facial skeleton.

In contrast to the cranial vault, the bones of the cranial base are formed initially in the cartilage and are later transformed by endochondral ossification into bone. This is particularly true of the midline structures. As one moves laterally, growth at sutures becomes more important, but the cranial base is essentially a midline structure.

Centers of ossification appear early in embryonic life in the chondrocranium, indicating the eventual location of the basioccipital, sphenoid and ethmoid bones that form the cranial base *(Fig. 2.15).*

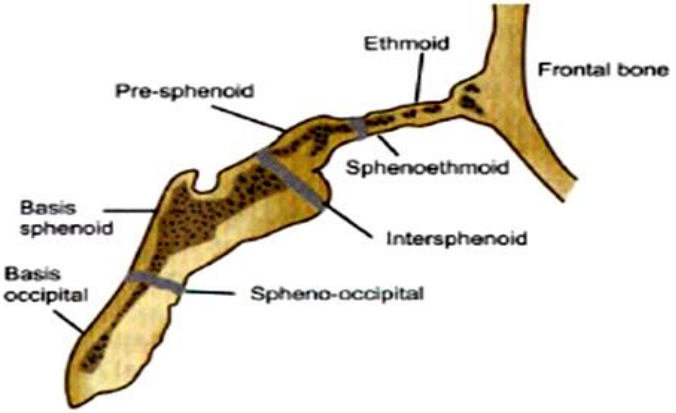

Fig. 2.15. The bones that from the base of the skul

The cranial base grows primarily by cartilage growth in the sphenoethmoidal, intersphenoidal, spheno-occipital and intraoccipital synchondroses, mostly following the neural growth curve *(Fig. 2.16).*

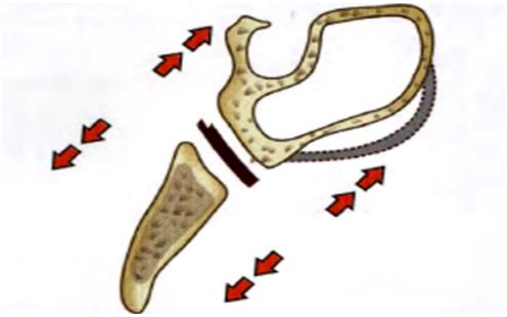

Fig. 2.16. Growth at the inter-sphenoidal synchondrosis

Activity at the intersphenoidal synchondrosis disappears at birth. The intraoccipital synchondrosis closes in the 3rd to 5th years of life. The sphenooccipital synchondrosis is a major contributor as the ossification here extends till the 20th year of life.

The nasomaxillary complex. The growth of the cranium and facial skeleton progress at different rates (Scammon). By differential growth, the face literally emerges from beneath the cranium *(Fig. 2.17).*The upper face, under the influence of cranial base inclination, moves upwards and forwards; the lower face moves downwards and forwards on an 'expanding V".

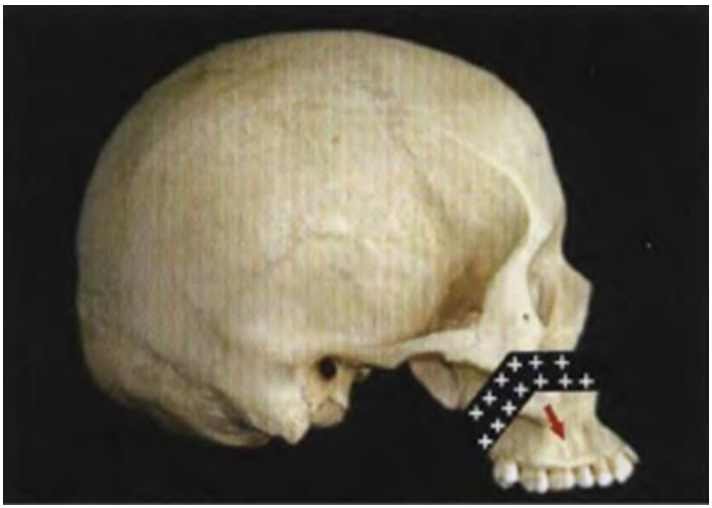

Fig. 2.17. The nasomaxillary complex as it emerges from beneath the cranium

Since the maxillary complex is attached to the cranial base, there is a strong influence of the latter on the former. Although, there is no sharp line of demarcation between cranium and maxillary growth gradients, yet the position of the maxilla is dependent upon the' growth at spheno-occipital and sphenoethmoidal synchondroses. Hence, while discussing the growth of nasomaxillary complex, we have to look into two aspects:

1. The shift in the position of the maxillary complex
2. The enlargement of the complex itself.

Needless to say that both these issues are interrelated and concomitant. Enlow and Bang apply the principle of "area relocation" to the complex and multidirectional growth movements. As the dynamic process continues, "specific local areas come to occupy new actual positions in succession, as the entire bone en larges. These growth shifts and changes involve corresponding and sequential remodeling adjustments in order to maintain the same shape, relative positions and constant proportions of each individual area in the maxilla as a whole". Moss described these as translocation and transposition respectively.

The maxilla develops entirely by intramembranous ossification. Sutural connective tissue proliferations, ossification, surface apposition, resorption and translation are the mechanisms for maxillary growth. The maxilla is related to the cranium at least partially by the frontomaxillary suture, the zygomaticomaxillary suture, zygomaticotemporal suture and pterygopalatine suture. Weinmann and Sicher have pointed out that these sutures are all oblique and more or less parallel with each other *(Fig. 2.18).* Thus, growth in these areas would serve to move the maxilla downward and forward (or the cranium upward and backward) *(Fig. 2.18).*

Moss and Greenberg point out that the basic maxillary skeletal unit is the infraorbital neurovascular triad, where the maxillary basal bone largely serves as a protection mechanism for the trigeminal nerve. 1t is this neurotrophic influence, which maintains the spatial constancy for the infra orbital canal with respect to the anterior cranial base. Thus, indirectly it produces a similar constancy of the basal maxillary skeletal unit relative to the same base.

Moss cites three types of bone growth changes to be observed in the maxilla:

1. Those changes that are associated with compensations for the passive motions of the bone brought about by the primary expansion of the orofacial capsule.
2. There are changes in bone morphology associated with alterations in the absolute volume, size shape or spatial position of any or all of the several relatively independent maxillary functional matrices, such as orbital mass.
3. There are bone changes associated with the maintenance of the form of the bone itself.

All these changes do not occur simultaneously but rather differentially or sequentially.

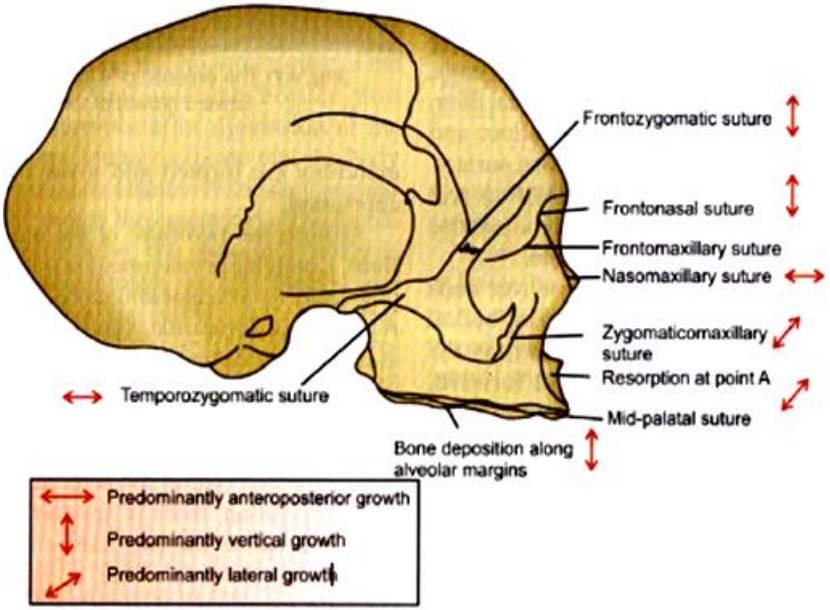

Fig. 2.18. The placement of the various sutures

To analyze the growth of the maxilla better, we must shift our focus to the functional matrices. It has been noted that the growth of the eyeball is essential for the development of the orbital cavity. Experimental evidence suggests that if there is no primordium for the eye, there is no orbit. It is clear that this functional matrix has a direct effect on the contiguous osseous structures. Also, just as the neurocranial bones are enclosed within a neurocrania I capsule, the facial bones are enclosed within the orofacial capsule. Resultantly the facial bones are passively carried outward (downward, forward, and laterally) by the primary expansion of the enclosed orofacial matrices (orbital, nasal, oral matrices). In addition there is an essential growth of the sinuses and spaces themselves, which perform important functions. The resultant maxillary changes would thus be secondary, compensatory and mechanically obligatory. In anteroposterior direction vector, the forward, passive motion of the maxilla is constantly being compensated for by the accretions at the maxillary tuberosity and at the palatal processes of both the maxillary and the palatine bones.

Specifically mentioning, the vertical growth of the maxillary complex is due to the continued apposition of alveolar bone on the free borders of the alveolar process as the teeth erupt. As the maxilla descends, continued bony apposition occurs on the orbital floor, with concomitant resorption on the nasal floor and apposition of the bone on the inferior palatal surface. By the alternate process

of bone deposition and resorption, the orbital and nasal floors and the palatine vault move downward in a parallel fashion.

Transversely, additive growth on the free ends increases the distance between them. The buccal segments move downward and outward, as the maxilla itself is moving downward and forward, following the principle of expanding "V" *(Fig. 2.19).*

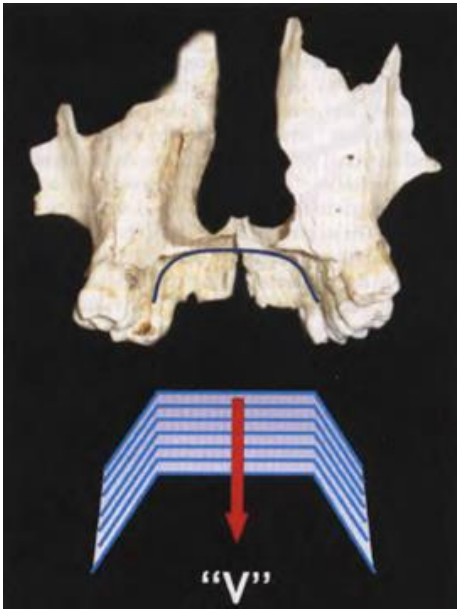

Fig. 2.19. The expanding "V" in the downward and forward growth o the maxilla

The mandible. In contrast to maxilla, both endochondral and periosteal activities are important in growth of the mandible. Cartilage covers the su rface of the mandibular condyle at the temporomandibular joint. All other areas of the mandible are formed and grow by direct surface apposition.

At birth the two rami of the mandible are quite short. Condylar development is minimal and there is practically no articular eminence in the glenoid fossa. A thin line of fibrocartilage and connective tissue exists at the midline of the symphysis to separate right and left mandibular bod ies. Between four months of age and the end of the first year, the symphyseal cartilage is replaced by bone. Although growth is quite general during the first year of life, with all surfaces showing bone apposition, there is apparently no significant growth between the two halves before they unite. During the first year of life, appositional growth is specially active at the alveolar border, at the distal and superior surfaces of the ramus, at the condyle, along the lower border of the mandible and on its lateral surfaces *(Fig. 2.20).*

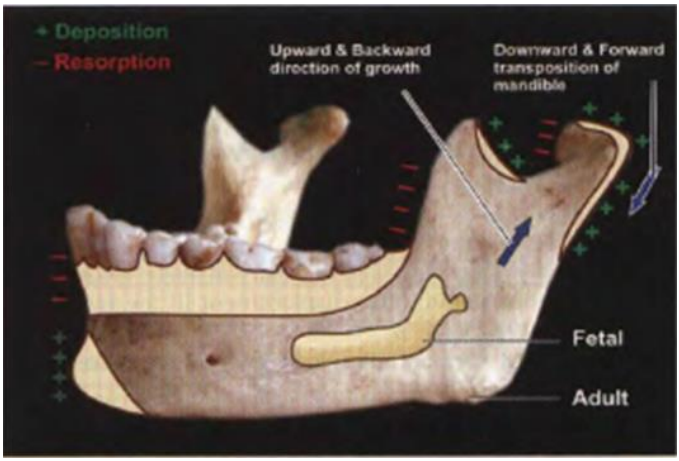

Fig. 2.20. Bone deposition and resorption sites at the mandible

After the first year of life the mandibular growth becomes more selective. The condyle does show considerable activity as the mandible moves and grows downward and forwards. Heavy appositional growth occurs on the posterior border of the ramus and on the alveolar process. Significant increments of growth are still observed at the tip of coronoid process.

Resorption occurs along the anterior border of the ramus lengthening the alveolar border and maintaining the antero-posterior dimension of the ramus. Cephalometric studies indicate that the body of the mandible maintains a relatively constant angular relationship to the ramus throughout life. The gonial angle changes little after the muscle activity is established. Transverse dimensions, after the first year of life are mainly due to the growth at the posterior border in an expanding "V" pattern *(Fig. 2.21).* The two rami also diverge outward from below to above so that additive growth at the coronoid notch, coronoid process and condyle also increases the superior interramus dimension.

The alveolar processes of the mandible grows upward and outward on an expanding arc. This permits the dental arch to accommodate the larger permanent teeth. Relatively little increase in mandibular body width is noted after the cessation of lateral surface appositional growth. Modeling deposition at the canine eminence and along the lateral border is seen.

Scott divides the mandible into three basic types of bone: basal, muscular and alveolar. The basal portion is a tube like central foundation running from the condyle to the symphysis. The muscular portion (gonia I angle and the coronoid process) is under the influence of the masseter, internal pterygoid and temporalis muscle. Alveolar bone exists to hold the teeth and it is gradually resorbed in the event of tooth loss.

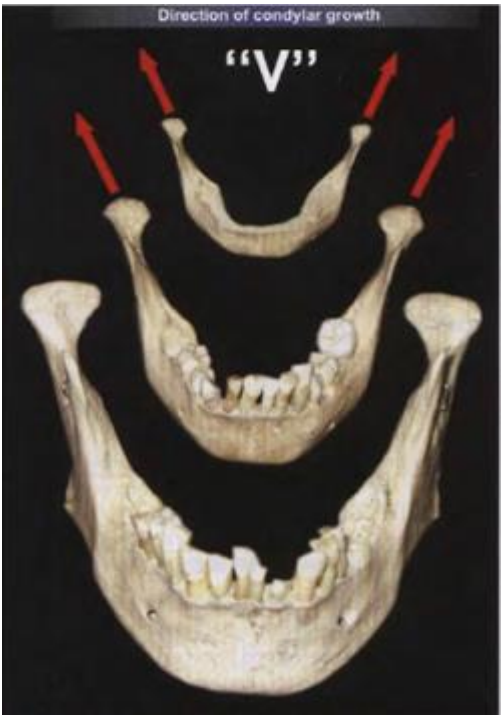

Fig. 2.21. The expanding "V" principle in the development of the mandible

Moss speaks of the mandible as a group of microskeletal units *(Fig. 2.22).*

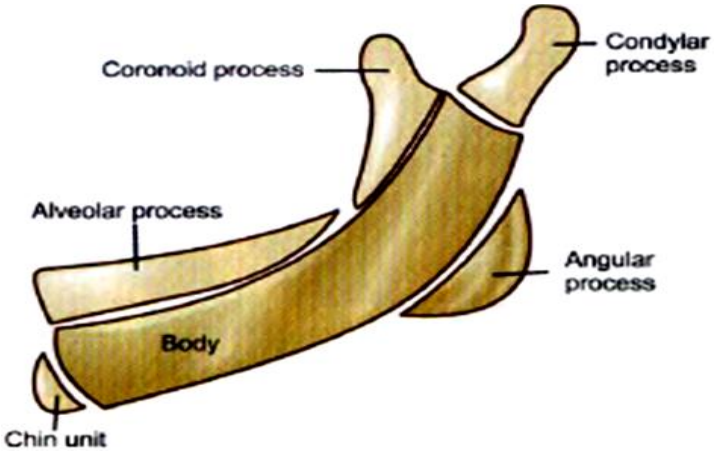

Fig. 2.22. The microskeletal units in the mandible as proposed by Moss

The coronoid process is one skeletal unit under the influence of ternporalis. The gonia! angle is another skeletal unit under the influence of masseter and internal pterygoid muscles. The alveolar process is under the influence of the dentition. The basal tubular portion of the mandible serves as protection for the mandibular canal and apparently follows a logarithmic spiral in its downward and forward movement from beneath the cranium.

The chin. Enlow and Harris feel that chin is associated with a generalized cortical recession in the flattened regions positioned between the canine teeth. The process involves a mechanism of endosteal cortical growth.

On the lingual surface behind the chin, heavy periosteal growth occurs, with the dense lamellar bone merging and overlapping on the labial side of the chin. Particularly in the male, the apposition of the bone at the symphysis seems to be about the last change in shape during the growing period. This means that some time between 16and 25years of age, appositional changes give a new shape to the symphysis in males. This change is much less apparent in females.

2.3. Characteristics of the oral cavity of a newborn

The Gum Pads *(Fig. 2.23).* The alveolar arches of an infant at the time of birth are called Gum Pads. These are greatly thickened oral mucous membrane of the gums, which soon become segmented, and each segment is a developing tooth site. They are pink in color and firm in consistency.

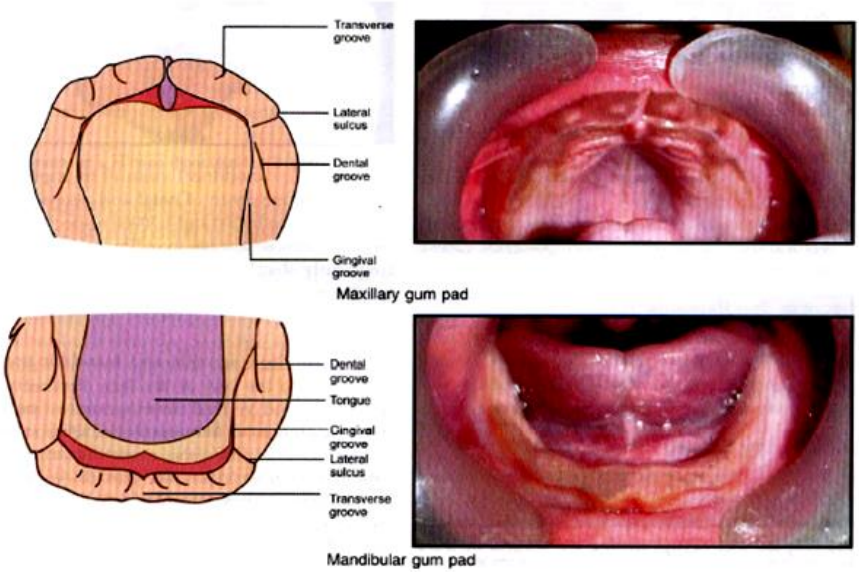

Fig. 2.23. Gum pads of the maxillay and mandible

The pads get divided into a labio/buccal and a lingual portion which differentiates later. Transverse grooves separate the gum pads into 10 segments. The groove between the canine and the first molar region is called the lateral sulcus, which helps to judge the inter-arch relationship.

• The upper gum pad is horse shoe shaped and shows:

– Gingival groove: Separates gum pad from the palate.

– Dental groove: Starts at the incisive papilla, extends backward to touch the gingival groove in the canine region and then moves laterally to end in the molar region.

– Lateral sulcus.

• The lower gum pad is U-shaped and rectangular, characterized by:

– Gingival groove: Lingual extension of the gum pads.

– Dental groove: Joins gingival groove in the canine region.

– Lateral sulcus.

Relationship of gum pads *(Fig. 2.24)*:

• Anterior open bite is seen at rest with contact only in the molar region. Tongue protrudes anteriorly through this space. The intermaxillary space closure, occurs with eruption of primary teeth, thus it is a self-correcting anomaly of the developing dentition.

• Complete overjet

• Class II pattern with the maxillary gum pad being more prominent.

• Mandibular lateral sulci posterior to maxillary lateral sulci.

• Mandibular functional movements are mainly vertical and to a little extent anteroposterior. Lateral movements are absent.

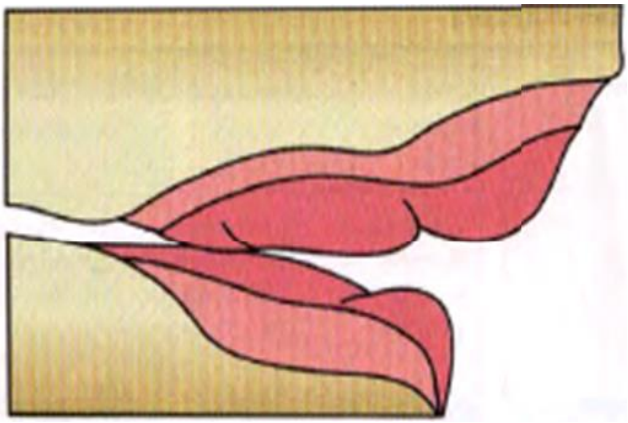

Fig. 2.24. Relation between upper and lower gum pads at birth

Neonatal jaw relationships. A precise "bite" or jaw relationship is not yet seen. Therefore, neonatal jaw relationship cannot be used as a diagnostic criterion for reliable prediction of subsequent occlusion in the primary dentition.

Precociously erupted primary teeth. Occasionally a child is born with teeth already present in the mouth. Natal teeth *(Fig. 2,25)* are present at birth whereas neonatal teeth erupt during the first month. Pre-erupted teeth erupt during the second or third month. The incidence of natal and neonatal teeth is estimated to be 1 : 1000 and 1 : 30000 respectively. These teeth are almost always mandibular incisors, which frequently display enamel hypoplasia. There are familial tendencies for such teeth. They should not be removed if normal but removed if supernumerary or mobile.

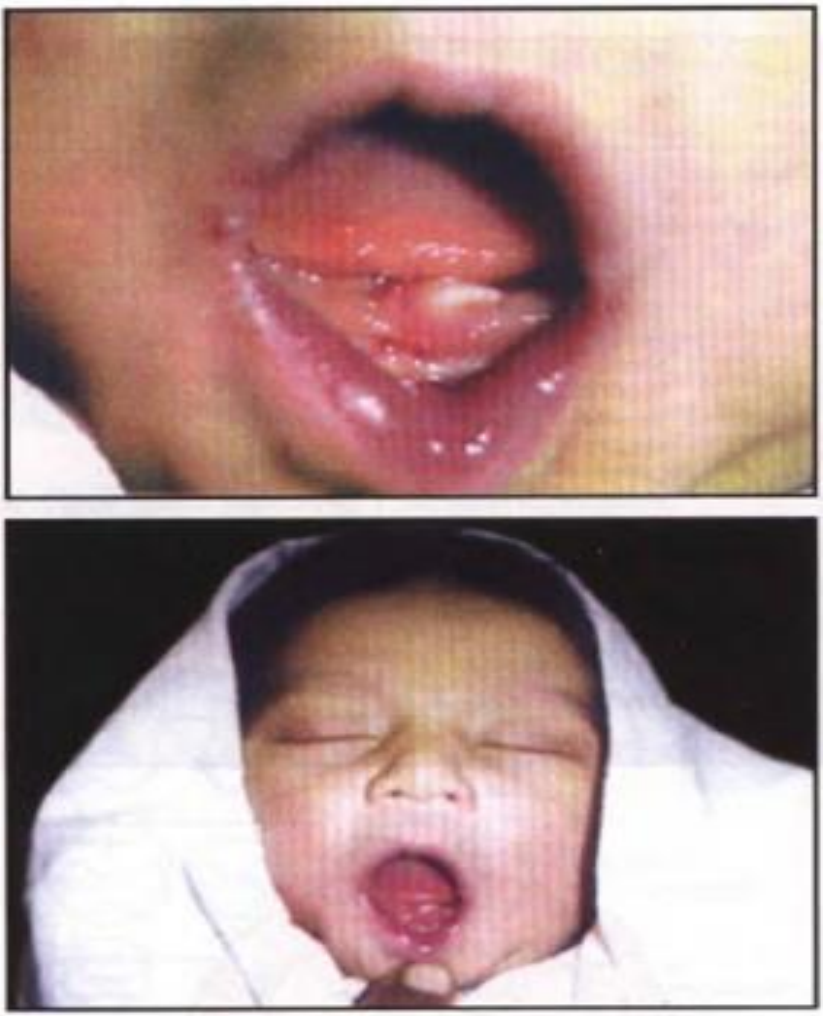

Fig. 2.25. Natal tooth seen in a newborn

At birth, both maxilla and mandible are small compared to the rest of the face. Extensive early transverse and ventral development of both jaws occurs leading to an anteroposterior relation between the jaws. Overjet diminishes markedly during the first 6 months. Increase in jaw size provides enough space for harmonious arrangement of deciduous teeth. Thus crowding seen in the pads disappears when the teeth erupt. Eruption of deciduous teeth commences at about 6 months of age. Occlusion starts developing posteriorly when deciduous first molars attain contact. By the time the first molars have settled, occlusion in the posterior region is established.

Peculiarities of the infant oral cavity. The infant oral cavity and all elements of its masticatory apparatus are fully adaptated to the act of sucking. They include:

- transversally striated lips (Pfaundler–Liushke's proboscidiform cushions) with the evident orbicular muscle of mouth;
- 4–5 pairs of cross folds, due to which asperity forms in the anterior part of the hard palate, which promotes holding of the nipple;
- elastic gingival membrane (Roben–Mazhito's fold) in the form of mucous tunic duplicaton with a lot of elastic fibers;
- comparatively big tongue;
- fatty interlayer of cheeks and fatty balls of Bichat, which provide negative pressure in the oral cavity during sucking;
- high position of the aperture of larynx (above the level of the inferoposterior margin of the palate veil) and its junction only with the nasal cavity allow the child to breath, suck, and swallow simultaneously;
- articular tubercle absence and occipital inclination of the underdeveloped branch *(Fig. 2.26)*;
- distal position of the lower jaw, physiological retrognathia;
- wide flat glenoid fossa;
- undeveloped intra-articular disk and glenoid fossa create favorable conditions for the unhampered movement of the lower jaw in the sagittal plane during sucking.

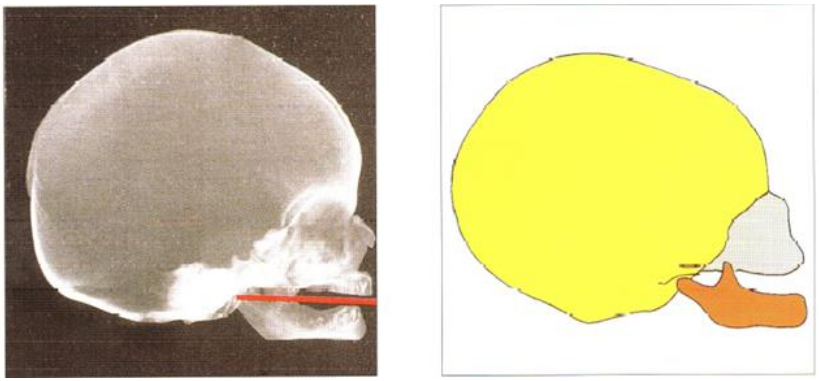

Fig. 2.26. The peculiarities of the newborn's temporomandibular joint

2.4. The Etiology of Orthodontic Problems

Malocclusion is a developmental condition. In most instances, malocclusion and dentofacial deformity are caused, not by some pathologic process, but by moderate distortions of normal development. Occasionally a single specific cause is apparent, as for example, mandibular deficiency secondary to a childhood fracture of the jaw or the characteristic malocclusion that accompanies some genetic syndromes. More often these problems result from a complex interaction among multiple factors that influence growth and development, and it is impossible to describe a specific etiologic factor.

Although it is difficult to know the precise cause of most malocclusions, we do know in general what the possibilities are, and these must be considered when treatment is considered. In this chapter, we examine etiologic factors for malocclusion under three major headings: specific causes, hereditary influences, and environmental influences. The chapter concludes with a perspective on the interaction of hereditary and environmental influences in the development of the major types of malocclusion.

Disturbances in embryologic development. Defects in embryologic development usually result in death of the embryo. As many as 20 % of early pregnancies terminate because of lethal embryologic defects, often so early that the mother is not even aware of conception. Only a relatively small number of recognizable conditions that produce orthodontic problems are compatible with long-term survival.

A variety of causes exist for embryologic defects, ranging from genetic disturbances to specific environmental insults. Chemical and other agents capable of producing embryologic defects if given at the critical time are called *teratogens.* Most drugs do not interfere with normal development or, at high doses, kill the embryo without producing defects, and therefore are not teratogenic. Teratogens typically cause specific defects if present at low levels but if given in higher doses, do have lethal effects. Problems that can be traced to embryologic defects, though devastating to the affected individual, fortunately are relatively rare. The best estimate is that fewer than 1 % of children who need orthodontics had a disturbance in embryologic development as a major contributing cause.

Skeletal growth disturbances. ***Fetal molding and birth injuries.*** Injuries apparent at birth fall into two major categories: (1) intrauterine molding and (2) trauma to the mandible during the birth process, particularly from the use of forceps in delivery.

Intrauterine molding. Pressure against the developing face prenatally can lead to distortion of rapidly growing areas. Strictly speaking, this is not a birth injury, but because the effects are noted at birth, it is considered in that category. On rare occasions an arm is pressed across the face in utero, resulting in severe maxillary deficiency at birth. Occasionally a fetus' head is flexed tightly against the chest in utero, preventing the mandible from growing forward normally. This is related to a decreased volume of amniotic fluid, which can occur for any of several reasons. The result is an extremely small mandible at birth, usually accompanied by a cleft palate because the restriction on displacement of the mandible forces the tongue upward and prevents normal closure of the palatal shelves.

This extreme mandibular deficiency at birth is the Pierre Robin syndrome. The reduced volume of the oral cavity can lead to respiratory difficulty at birth, and it may be necessary to suture the tongue forward temporarily or even perform a tracheostomy so the infant can breathe.

Because the pressure against the face that caused the growth problem would not be present after birth, one would predict normal growth thereafter and perhaps eventually a complete recovery. Some children with Pierre Robin syndrome at birth do have favorable mandibular growth thereafter. For that reason, early aggressive treatment to lengthen the mandible should be avoided. Others never make up the deficit and surgical intervention is needed. It has been estimated that about one-third of the Pierre Robin patients have a defect in cartilage formation and can be said to have Stickler syndrome. Not surprisingly, this group have limited growth potential. Catch-up growth is most likely when the original problem was mechanical growth restriction that no longer existed after birth.

Birth trauma to the mandible. Many deformity patterns now known to result from other causes once were blamed on injuries during birth. Many parents, despite explanations from their doctors, will refer to their child's facial deformity as being caused by a birth injury even if a congenital syndrome pattern is evident. No matter what the parents say later, Treacher Collins syndrome or Crouzon's syndrome obviously did not arise because of birth trauma.

In some difficult births, however, the use of forceps to the head to assist in delivery might damage either or both the temporomandibular joints. At least in theory, heavy pressure in the area of the temporomandibular joints could cause internal hemorrhage, loss of tissue, and a subsequent underdevelopment of the mandible. At one time this was a common explanation for mandibular deficiency. If the cartilage of the mandibular condyle were an important growth center, of course, the risk from damage to a presumably critical area would seem much greater. In light of the contemporary understanding that the condylar cartilage is not critical for proper growth of the mandible, it is not as easy to blame underdevelopment of the mandible on birth injuries. It is interesting to note that although the use of forceps in deliveries has decreased considerably over the last 50 years, the prevalence of Class II malocclusion as a result of mandibular deficiency has not decreased. In short, injury to the mandible during a traumatic delivery appears to be a rare and unusual cause of facial deformity. Children with deformities involving the mandible are much more likely to have a congenital syndrome.

Childhood fractures of the Jaw. The falls and impacts of childhood can fracture jaws just like other parts of the body. The condylar neck of the mandible is particularly vulnerable, and fractures of this area in childhood are relatively common. Fortunately, the condylar process tends to regenerate well after early fractures. The best human data suggest that about 75 % of children with early fractures of the mandibular condylar process have normal mandibular growth, and therefore do not develop malocclusions that they would not have had in the absence of such trauma. Interestingly, the prognosis is better the earlier the

condylar fracture occurs, perhaps because the growth potential is greater early in life. From the number of children with later growth problems whose original fracture was not diagnosed, it appears that many early fractures of the condylar process go completely unnoticed.

It seems to be relatively common for a child to crash the bicycle, chip a tooth and fracture a condyle, cry a bit, and then continue to develop normally, complete with total regeneration of the condyle. When a problem does arise following condylar fracture, it usually is asymmetric growth, with the previously injured side lagging behind. This suggests that childhood jaw fractures, though potentially a cause of severe orthodontic problems, do not make a large contribution to the total pool of patients with malocclusion.

It is important to understand the mechanism by which trauma can produce a distortion in subsequent growth. The maxilla normally grows downward and forward because of a combination of push from behind by the lengthening cranial base (which is largely complete at an early age) and pull from in front by anteriorly positioned tissue elements (probably including but not limited to the cartilaginous nasal septum). The mandible seems to be almost entirely pulled forward by the soft tissue matrix in which it is embedded. After an injury, growth problems arise when there is enough scarring in the area to restrict the normal growth movements, so that the maxilla or, more frequently, the mandible cannot be pulled forward along with the rest of the growing face. If there is more scarring and restriction on one side, subsequent growth will be asymmetric.

This concept is highly relevant to the management of condylar fractures in children. It suggests, and clinical experience confirms, that there would be little if any advantage from surgical open reduction of a condylar fracture in a child. The additional scarring produced by surgery could make things worse. The best therapy therefore is conservative management at the time of injury and early mobilization of the jaw to minimize any restriction on movement. An old condylar fracture is the most likely cause of asymmetric mandibular deficiency in a child, but other destructive processes that involve the temporomandibular joint such as rheumatoid arthritis, or a congenital absence of tissue as in hemifacial microsomia, also can produce this problem.

Muscle dysfunction. The facial muscles can affect jaw growth in two ways. First, the formation of bone at the point of muscle attachments depends on the activity of the muscle; second, the musculature is an important part of the total soft tissue matrix whose growth normally carries the jaws downward and forward. Loss of part of the musculature can occur from unknown causes in utero or as a result of a birth injury, but is most likely to result from damage to the motor nerve (the muscle atrophies when its motor nerve supply is lost). The result would be underdevelopment of that part of the face.

Excessive muscle contraction can restrict growth in much the same way as scarring after an injury. This effect is seen most clearly in torticollis, a twisting

of the head caused by excessive tonic contraction of the neck muscles on one side (primarily the sternocleidomastoid). The result is a facial asymmetry because of growth restriction on the affected side, which can be quite severe unless the contracted neck muscles are surgically detached at an early age. Conversely, the decrease in tonic muscle activity that occurs in muscular dystrophy, some forms of cerebral palsy, and various muscle weakness syndromes allow the mandible to drop downward away from the rest of the facial skeleton. T h e result is increased anterior face height, distortion of facial proportions and mandibular form, excessive eruption of the posterior teeth, narrowing of the maxillary arch and anterior open bite.

Acromegaly and hemimandibular hypertrophy. In acromegaly, which is caused by an anterior pituitary tumor that secretes excessive amounts of growth hormone, excessive growth of the mandible may occur, creating a skeletal Class III malocclusion in adult life. Often (but not always–sometimes the mandible is unaffected) mandibular growth accelerates again to the levels seen in the adolescent growth spurt, years after adolescent growth was completed. The condylar cartilage proliferates, but it is difficult to be sure whether this is the cause of the mandibular growth or merely accompanies it. Although the excessive growth stops when the tumor is removed or irradiated, the skeletal deformity persists and orthognathic surgery to reposition the mandible is likely to be necessary.

Occasionally, unilateral excessive growth of the mandible occurs in individuals who seem metabolically normal. Why this occurs is entirely unknown. It is most likely in girls between the ages of 15 and 20, but may occur as early as age 10 or as late as the early 30s in either sex. The condition formerly was called *condylar hyperplasia*, and proliferation of the condylar cartilage is a prominent aspect; however, because the body of the mandible also is affected, *hemimandibular hypertrophy* now is considered a more accurate descriptive term. The excessive growth may stop spontaneously, but in severe cases removal of the affected condyle and reconstruction of the area is necessary.

Disturbances of dental development. Disturbances of dental development may accompany major congenital defects but are most significant as contributors to isolated Class I malocclusion. Significant disturbances include:

Congenitally missing teeth. Congenital absence of teeth results from disturbances during the initial stages of formation of a tooth—initiation and proliferation. *Anodontia,* the total absence of teeth, is the extreme form. The term *oligodontia* refers to congenital absence of many but not all teeth, whereas the rarely used term *hypodontia* implies the absence of only a few teeth. Since the primary teeth give rise to the permanent tooth buds, there will be no permanent tooth if its primary predecessor was missing. It is possible, however, for the primary teeth to be present and for some or all the permanent teeth to be absent.

Anodontia or oligodontia, the absence of all or most of the permanent teeth, is usually associated with an unusual but mild systemic abnormality, *ectodermal dysplasia*. Individuals with ectodermal dysplasia have thin, sparse hair and an absence of sweat glands in addition to their characteristically missing teeth. Occasionally, oligodontia occurs in a patient with no apparent systemic problem or congenital syndrome. In these children, it appears as if there is a random pattern to the missing teeth.

Anodontia and oligodontia are rare, but hypodontia is a relatively common finding. As a general rule, if only one or a few teeth are missing, the absent tooth will be the most distal tooth of any given type. If a molar tooth is congenitally missing, it is almost always the third molar; if an incisor is missing, it is nearly always the lateral; if a premolar is missing, it almost always is the second rather than the first. Rarely is a canine the only missing tooth.

Malformed and supernumerary teeth. Abnormalities in tooth size and shape result from disturbances during the morphodifferentiation stage of development, perhaps with some carryover from the histodifferentiation stage. The most common abnormality is a variation in size, particularly of maxillary lateral incisors and second premolars. About 5 % of the total population have a significant "tooth size discrepancy" because of disproportionate sizes of the upper and lower teeth. Unless the teeth are matched for size, normal occlusion is impossible. As might be expected, the most variable teeth, the maxillary lateral incisors, are the major culprits. The diagnosis of tooth size discrepancy is based on comparison of the widths of teeth to published tables of expected tooth sizes.

Occasionally, tooth buds may fuse or geminate (partially split) during their development. Fusion results in teeth with separate pulp chambers joined at the dentin, whereas gemination results in teeth with a common pulp chamber. The differentiation between gemination and fusion can be difficult and is usually confirmed by counting the number of teeth in an area. If the other central and both lateral incisors are present, a bifurcated central incisor is the result of either gemination or, less probably, fusion with a supernumerary incisor. On the other hand, if the lateral incisor on the affected side is missing, the problem probably is fusion of the central and lateral incisor buds. Normal occlusion, of course, is all but impossible in the presence of geminated, fused, or otherwise malformed teeth.

Supernumerary or extra teeth also result from disturbances during the initiation and proliferation stages of dental development. The most common supernumerary tooth appears in the maxillary midline and is called a *mesiodens*. Supernumerary lateral incisors also occur; extra premolars occasionally appear; a few patients have fourth as well as third molars. The presence of an extra tooth obviously has great potential to disrupt normal occlusal development, and early intervention to remove it is usually required to obtain reasonable alignment and occlusal relationships. Multiple supernumerary teeth are most often seen in the congenital syndrome of cleidocranial dysplasia, which is characterized by missing clavicles (collar bones), many supernumerary and unerupted teeth, and failure of the succedaneous teeth to erupt.

Interference with eruption. For a permanent tooth to erupt, the overlying bone as well as the primary tooth roots must resorb, and the tooth must make its way through the gingiva. Supernumerary teeth, sclerotic bone, and heavy fibrous gingiva can obstruct eruption.

All of these interferences are present in cleidocranial dysplasia. The multiple supernumerary teeth contribute an element of mechanical interference. More seriously, children with this condition have a defect in bone resorption, and the gingiva is quite heavy and fibrous. If the eruption path can be cleared, the permanent teeth will erupt. To accomplish this, it is necessary not only to extract any supernumerary teeth that may be in the way but also to remove the bone overlying the permanent teeth and reflect the gingiva so that the teeth can break through into the mouth.

In patients with less severe interferences with eruption, delayed eruption of some permanent teeth contributes to malocclusion only when other teeth drift to improper positions in the arch. Although this delays eruption of its permanent successor, there is usually no lasting effect.

Ectopic eruption. Occasionally, malposition of a permanent tooth bud can lead to eruption in the wrong place. This condition is called *ectopic eruption* and is most likely to occur in the eruption of maxillary first molars.

If the eruption path of the maxillary first molar carries it too far mesially at an early stage, the permanent- molar is unable to erupt, and the root of the second primary molar may be damaged. The mesial position of the permanent molar means that the arch will be crowded unless the child receives treatment.

Ectopic eruption of other teeth is rare but can result in transposition of teeth or bizarre eruption positions. Mandibular second premolars sometimes erupt distally and can end up in the ramus. A poor eruption direction of other teeth, especially maxillary canines, usually is due to the eruption path being altered by a lack of space.

Early loss of primary teeth. When a unit within the dental arch is lost, the arch tends to contract and the space to close. At one time, this space closure was attributed entirely to mesial drift of posterior teeth, which in turn was confidently ascribed to forces from occlusion. Although a mesially-directed force can accompany occlusion, it probably is not a major factor in closure of spaces within the dental arches.

The contemporary view is that mesial drift is a phenomenon of the permanent molars only. The major reason these teeth move mesially when a space opens up is their mesial inclination, so that they erupt mesially as well as occlusally. Experimental data suggest that, rather than causing mesial drift, forces from occlusion actually retard it. In other words, a permanent molar is likely to drift mesially more rapidly in the absence of occlusal contacts than if they are present.

Mesial drift of the permanent first molar after a primary second molar is lost prematurely can significantly contribute to the development of crowding in the posterior part of the dental arch. This has been a significant cause of crowding and malalignment of premolars in the past. For this reason, maintenance of the space after a primary second molar has been lost is indicated.

When a primary first molar or canine is lost prematurely, there is also a tendency for the space to close. This occurs primarily by distal drift of incisors, not by mesial drift of posterior teeth. The impetus for distal drift appears to have two sources: force from active contraction of transseptal fibers in the gingiva, and pressures from the lips and cheeks. The pull from transseptal fibers probably is the more consistent contributor to this space closure tendency, whereas lip pressure adds a variable component. If a primary canine or first molar is lost prematurely on only one side, the permanent teeth drift distally only on that side, leading to an asymmetry in the occlusion as well as a tendency toward crowding.

From this description, it is apparent that early loss of primary teeth can cause crowding and malalignment within the dental arches. Is this a major cause of Class I crowding problems? The impact of fluoridation and other cariespreventive treatment on the prevalence of malocclusion indicates that it is not. Even without fluoridation, in other words, most crowding problems are not caused by early loss of primary teeth.

Traumatic displacement of teeth. Almost all children fall and hit their teeth during their formative years. Occasionally, the impact is intense enough to knock out or severely displace a primary or permanent tooth. Dental trauma can lead to the development of malocclusion in three ways: (1) damage to permanent tooth buds from an injury to primary teeth, (2) drift of permanent teeth after premature loss of primary teeth, and (3) direct injury to permanent teeth.

Trauma to a primary tooth can displace the permanent tooth bud underlying it. There are two possible results. First, if the trauma occurs while the crown of the permanent tooth is forming, enamel formation will be disturbed and there will be a defect in the crown of the permanent tooth.

Second, if the trauma occurs after the crown is complete, the crown may be displaced relative to the root. Root formation may stop, leaving a permanently shortened root. More frequently, root formation continues, but the remaining portion of the root then forms at an angle to the traumatically displaced crown. This distortion of root form is called *dilaceration,* defined as a distorted root form. Dilaceration may result from mechanical interference with eruption (as from an ankylosed primary tooth that does not resorb), but its usual cause, particularly in permanent incisor teeth? is trauma to primary teeth that also displaced the permanent buds.

If distortion of root position is severe enough, it is almost impossible for the crown to assume its proper position — that might require the root to extend out through the alveolar bone. For this reason, it may 'be necessary to extract a severely dilacerated tooth. Traumatically displaced tooth buds in children should be repositioned as early as possible, so that when root formation does resume, distortion of the root position will be minimized.

Permanent teeth are often displaced by trauma. If the tooth is knocked labially or lingually, the root of the tooth is sometimes damaged, but there always is a fracture of the alveolar process. Immediately after the accident, an intact tooth usually can be moved back to its original position rapidly and easily, so early

treatment is indicated. After healing (which takes 2 to 3 weeks), it is difficult to reposition the tooth, and ankylosis may develop that makes it impossible.

Genetic influences. A strong influence of inheritance on facial features is obvious at a glance—it is easy to recognize familial tendencies in the tilt of the nose, the shape of the jaw, and the look of the smile. It is apparent that certain types of malocclusion run in families. The Hapsburg jaw, the prognathic mandible of the German royal family, is the best known example, but dentists see repeated instances of similar malocclusions in parents and their offspring. The pertinent question for the etiologic process of malocclusion is not whether there are inherited influences on the jaws and teeth, because obviously there are, but whether malocclusion is often caused by inherited characteristics.

Malocclusion could be produced by inherited characteristics in two major ways. The first would be an inherited disproportion between the size of the teeth and the size of the jaws, which would produce crowding or spacing. The second would be an inherited disproportion between size or shape of the upper and lower jaws, which would cause improper occlusal relationships. The more independently these characteristics are determined, the more likely that disproportions could be inherited. Could a child inherit large teeth but a small jaw, for instance, or a large upper jaw and a small lower one? That would be quite possible if jaw and tooth sizes were inherited independently, but if dentofacial characteristics tended to be linked, an inherited mismatch of this type would be unlikely.

Primitive human populations in which malocclusion is less frequent than in modern groups are characterized by genetic isolation and uniformity. If everyone in a group carried the same genetic information for tooth size and jaw size, there would be no possibility of a child inheriting discordant characteristics. In the absence of processed food, one would expect strong selection pressure for traits that produced good masticatory function. Genes that introduced disturbances into the masticatory system would tend to be eliminated from the population (unless they conferred some other advantage). The result should be exactly what is seen in primitive populations: individuals in whom tooth size-jaw size discrepancies are infrequent, and groups in which everyone tends to have the same jaw relationship. Different human groups have developed impressive variations in facial proportions and jaw relationships. What happens, then, when there is outbreeding between originally distinct human population groups?

One of the characteristics of civilization is the collection of large groups of people into urban centers, where the opportunities for mating outside one's own small population group are greatly magnified. If inherited disproportions of the functional components of the face and jaws were frequent, one would predict that modern urban populations would have a high prevalence of malocclusion and a great variety of orthodontic problems. The United States, reflecting its role as a "genetic melting pot," should have one of the world's highest rates of malocclusion—which it does. In the 1930s and 1940s, as knowledge of the new science of genetics developed, it was tempting to conclude that the great increase in

outbreeding that occurred as human populations grew and became more mobile was the major explanation for the increase in malocclusion in recent centuries.

This view of malocclusion as primarily a genetic problem was greatly strengthened by breeding experiments with animals carried out in the 1930s. By far the most influential individual in this regard was Professor Stockard, who methodically crossbred dogs and recorded the interesting effects on body structure.17 Present-day dogs, of course, come in a tremendous variety of breeds and sizes. What would happen if one crossed a Boston terrier with a collie? Might the offspring have the collie's long, pointed lower jaw and the terrier's diminutive upper jaw? Could unusual crowding or spacing result because the teeth of one breed were combined in the offspring with the jaw of the other? Stockard's experiments indicated that dramatic malocclusions did occur in his crossbred dogs, more from jaw discrepancies than from tooth size-jaw size imbalances. These experiments seemed to confirm that independent inheritance of facial characteristics could be a major cause of malocclusion and that the rapid increase in malocclusion accompanying urbanization was probably the result of increased outbreeding.

These dog experiments turned out to be misleading, however, because many breeds of small dogs carry the gene for achondroplasia. Animals or humans affected by this condition have deficient growth of cartilage. The result is extremely short extremities and an underdeveloped midface. T h e dachshund is the classic achondroplastic dog, but most terriers and bulldogs also carry this gene. Achondroplasia is an autosomal dominant trait. Like many dominant genes, the gene for achondroplasia sometimes has only partial penetrance, meaning simply that the trait will be expressed more dramatically in some individuals than in others. Most of the unusual malocclusions produced in Stockard's breeding experiments can be explained not on the basis of inherited jaw size but by the extent to which achondroplasia was expressed in that animal.

Achondroplasia is rare in humans, but it does occur, and it produces the expected changes. In addition to short limbs, the cranial base does not lengthen normally because of the deficient growth at the synchondroses, the maxilla is not translated forward to the normal extent, and a relative midface deficiency occurs. In a number of relatively rare genetic syndromes like achondroplasia, influences on the form of the face, jaws, and teeth can be discerned,1 but those cause only a small percentage of orthodontic problems.

A careful examination of the results of outbreeding in human populations also casts doubt on the hypothesis that independently inherited tooth and jaw characteristics are a major cause of malocclusion. The best data are from investigations carried out in Hawaii by Chung et al. Before its discovery by the European explorers of the eighteenth century, Hawaii had a homogeneous Polynesian population. Large scale migration to the islands by European, Chinese and Japanese groups, as well as the arrival of smaller numbers of other racial and ethnic groups, resulted in an exceptionally heterogeneous modern population. Tooth size, jaw size, and jaw proportions were all rather different for the Poly-

nesian, Oriental, and European contributors to the Hawaiian melting pot. Therefore if tooth and jaw characteristics were inherited independently, a high prevalence of severe malocclusion would be expected in this population. The prevalence and types of malocclusion in the current Hawaiian population, though greater than the prevalence of malocclusion in the original population, do not support this concept. The effects of interracial crosses appear to be more additive than multiplicative. For example, about 10 % of the Chinese who migrated to Hawaii had Class III malocclusion, whereas about 10 % of the Polynesians had crowded teeth. The offspring of this cross seem to have about a 10 % prevalence of each characteristic, but there is no evidence of dramatic facial deformities like those seen in the crossbred dogs. In other words, if malocclusion or a tendency to malocclusion is inherited, the mechanism is not the independent inheritance of discrete morphologic characteristics like tooth and jaw sizes.

The classic way to determine to what extent a characteristic is determined by inheritance is to compare monozygotic (identical) with dizygotic (fraternal) twins. Monozygotic twins occur because of the early division of a fertilized egg, so each individual has the same chromosomal deoxyribonucleic acid (DNA) and the two are genetically identical. Any differences between them should be solely the result of environmental influences. Twins also occur when two eggs are released at the same time and fertilized by different spermatozoa. These dizygotic twins are not more similar than ordinary siblings except that they have shared the same intrauterine and family environment.

By comparing identical twins, fraternal twins, and ordinary siblings, an estimate of the heritability of any characteristic can be determined. That is, the proportion of the variability in that characteristic due to heredity can be estimated. studies of this type are limited in several ways not only because it is difficult to obtain the twin pairs for study but also because it can be difficult to establish zygosity and confirm that the environments were in fact the same for both members of a twin pair.

The other classic method of estimating the influence of heredity is to study family members, observing similarities and differences between mother-child, father-child, and sibling pairs. For most measurements of facial skeletal dimensions (i.e., length of the mandible), correlation coefficients for parent-child pairs are about 0.5, which is the theoretical upper limit of the genetic contribution for a first-degree relative. For dental characteristics, the parentchild correlations are lower, ranging from a maximum of nearly 0.5 for overjet to a minimum of 0.15 for overbite. When parent-child correlations are used to assist in predicting facial growth, errors are reduced, which in itself strongly indicates the hereditary influence on these dimensions.

From an examination of longitudinal cephalometric radiographs and dental casts of siblings who participated in the Bolton-Brush growth study (carried out between the late 1930s and the early 1970s), Harris and Johnson concluded that the heritability of craniofacial (skeletal) characteristics was relatively high, but that of dental (occlusal) characteristics was low. For skeletal characteristics, the

heritability estimates increased with increasing age; for dental characteristics, the heritability estimates decreased, indicating an increasing environmental contribution to the dental variation. To the extent that the facial skeleton determines the characteristics of a malocclusion, therefore, a hereditary component is likely to be present, but purely dental variation seems to be much more environmentally determined.

The influence of inherited tendencies is particularly strong for mandibular prognathism. In one study, one third of a group of children who presented with severe Class III malocclusion had a parent with the same problem, and one sixth had an affected sibling.

The environmental influences during the growth and development of the face, jaws, and teeth will see in chapter 3.

2.5. Prevention methods during prenatal and postnatal period

Famous clinician S.P. Botkin said: "It is easier to prevent a disease than to treat it".

Prevention of dentognathic anomalies and deformations is possible only by means of timely elimination of the reasons causing or promoting them and is conducted with the help of general and local measures.

All the prophylactic methods of treating dentognathic anomalies may be divided into such periods:

- the 1st period – before the conception of the child (creation of medicogenetic cabinets);
- the 2nd period – intrauterine;
- the 3rd period – lactational (the first half a year of the child's life);
- the 4Ih period – from 6 months till the end of the temporary occlusion period (2–2.5 years);
- the 5Ih period – transitional dentition occlusion (6–13 years);
- the 6th period – permanent occlusion (from 13 years).

Medicogenetic consultations. During the last decades much attention has been paid to the issues of studying anomalies of teeth, jaws, facial and cerebral cranium from genetic positions. In this connection there have been opened medicogenetic cabinets.

It is expedient for young people to be consulted before marriage or right after it but before the conception of a child. This will allow avoiding an accident or decreasing its possibility.

In the process of ovum fertilization with a spermatozoon the fusion of sex cells takes place and a zygote forms with a double number of chromosomes. Genetic information is transferred from parents to the child. Homozygous organism develops from a pair of identical genes with the investigated sign, for instance, healthy genes of parents, heterozygous – from a pair of non-identical genes from a healthy parent and a sick one.

There exist dominant and recessive types of inheritance. At the dominant type of inheriting a sign it is transmitted from generation to generation, at the

recessive type the examination of siblings – relatives of the proband (brothers, sisters, cousins) – is needed. Besides, proband's uncles, aunts, nephews, and nieces are subject to examination. It is important to obtain thorough genealogic anamnesis. The risk of disease recurrence, found by means of medical statistics, makes 25 % at every pregnancy. It is difficult to determine the inheritance of signs, as parents of ill children are not infrequently healthy but are the carriers of a pathologic gene. Most disorders, included into this category, are connected with biochemical (metabolic) disturbances.

The inheritance of anomalies of size, form, structure of teeth, size of jaws, congenital nonunions in the gnathic-facial area are noted by many scientists.

Special attention is paid to the microsigns of anomalies, in particular at adentia of individual or all teeth in a proband, to the decrease of teeth sizes in comparison with the data of average norm in siblings and other relatives with the estimation of face form.

Still, it should be taken into consideration that analogical disorders in the dentognathic-facial area may develop in children and parents without genetic base. Their clinical presentation may be analogical and the reasons – various, polyetiologic.

The aim of medicogenetic consultation is information exchange between the doctor and future parents. It is important to inform future parents about all the possible decisions they have, to help the interested members of the family by pieces of advice how to solve their problems, overcome possible sufferings, find the ways, which would help to carry out the made decision.

In the process of medicogenetic consultation there are determined:

1) if the person, who has appealed for consultation, is ill with any heritable disease, if the person is a carrier of the disease;

2) if there is a risk of having a child with heritable malformation in the dentofacial and other areas;

3) what complications, caused by a heritable disease, are in one of the partners going to get married, or in a couple going to have a baby; what the risk prognosis of such disorders recurrence in the descendant is;

4) what help the parents can obtain in taking a decision after putting prenatal diagnosis;

5) what prophylactic and medical help may be rendered to the newborn child at the presence of genetically conditioned malformation; where the necessary help may be rendered, in what consequence and in what age periods.

Modern medicogenetic consultation is characterised by the accessibility of different means of investigation, including prenatal diagnostics. Its aim is not to solve problems instead of the married couple but to elucidate the situation, prevent the cases of serious heritable diseases.

Medicogenetic consultation allows determining the measure of the risk of a child being born with a congenital pathology analogical to the one present in parents or relatives. This information is important for detecting the possibility

of anomalies prevention in children and siblings, and also the peculiarities of complex treatment, which includes conducting prophylactic orthodontic, myotherapeutic, surgical and other measures in certain age periods and in certain sequence to prevent permanent disorders of occlusion and face deformations.

Intrauterine period. As etiologic factors may affect at different age, prophylactic measures are to be carried out over the time of the child's growth and development.

There are differentiated general and special types of dentognathic anomalies prophylaxis. General prophylaxis consists in creating optimal conditions for the growth of the whole organism beginning from the intrauterine period. In this period measures should be taken to prevent injuries, various diseases, and dysbolism of the pregnant woman. The prospective mother is to be protected from harmful psychic, physical or chemical influences; favorable conditions of work and life are to be created for her.

Health-improving measures are to be taken: general gymnastics, tempering of the organism, hydrotherapeutic procedures, etc. Emotional equilibrium of the pregnant woman, considerate, gentle, and sensitive attitude of relatives and acquaintances towards her, normal way of life, lasting stay in the open air, favorable living conditions, rational nutrition (eating food rich in vitamins and mineral salts in optimal proportion with other necessary nutrients important for the normal development of the child's organism and its mastication apparatus) are of great importance.

The influence on the health of the pregnant woman or a feeding mother is important because for the development of a normal child's organism in general and dentognathic apparatus in particular work and life hygiene is of great significance; the labor is to be physiologic; different obstetric procedures (forceps, vacuum, etc.) should be avoided; feeding of the child should be natural (breast).

Lactational Period. After the child's birth favorable environment is necessary for its development and general diseases prevention. Regimen and character of feeding are of special concern. Correct position of the child's head during sleep should be watched. Lactational period is characterised by moderate influence on the organism of the mother and formation of the child's organism. This period is also only prophylactic. Except for the influence on the organism of the wet nurse with the рифове of increasing the quantity and quality of milk, there is also exerted the direct influence on the child's organism, which consists in improving the conditions of its existence.

S.O. Dulytskyi recommends beginning first walks with the child since 3–4 weeks' age in cold season (temperature being – 15 °C). In summer at temperature not lower than +18 °C (in the shadow) with no wind the child since 3-months' age is to be habituated to air-baths. In other seasons air-baths are to be conducted indoors, beginning from a month's age.

Correct child-minding is also important. The child should not be overheated by excessive wrapping: cloths and bed linen should not cause sweating, the child must get everyday hygienic baths, and beginning from 2-months' age – sponged down with warm (37 °C) salty water.

The child is to be fed with breast milk, which is useful in the view of physiology as more valuable than feeding formulae; it is the healthiest kind of food.

I.P. Pavlov writes: "Secretory work of the stomach for milk assimilation is significantly less in comparison with any other food". For breast milk digestion little fermentative capability of the gastric and pancreatic juices is enough, and in a 3–4-month-old child the secretory activity of digestive glands is qualitatively and quantitatively weaker and milk substitutes are poorly digested. The more favorable ratio of calcium and phosphorus (1 : 1) in the woman's milk than in the animals' milk also plays some role.

Artificially fed children have been found to fall ill more often and more severely than naturally fed children. The former also put on weight slowly.

Therefore, mothers are to be convinced to feed children with breast milk longer; of course, the mother's state of health should be taken into account. In cases of artificial feeding measures should be taken to liken it to the natural one. For this purpose, the nipple of the milk bottle should be made of rubber reminding mamilla by its form and elasticity. The bottle should be held not vertically but horizontally so that the milk is not poured into the child's mouth by itself, the child must make efforts to suck milk from the bottle. It should also be attended that the bottle with milk does not press on the lower jaw in order to avoid its deformation. Finally, the mother ought not put the bottle with milk into the child's mouth and leave the child lying as it is often done, but the child should be hold in hands during feeding. All this creates such conditions, which are at least somehow close to natural.

By the end of the first or second month of life some children suck their fists for a long time, therefore the prophylaxis of sucking fingers and fists should begin from the first days of the child's life.

The child should also be habituated to the correct position during sleep: not to put a fist under the cheek, not to sleep with the head thrown back or bent down.

Chapter 3. Development of the dentition and dental occlusion. Occlusion – basic concept. Anatomy and function of temporomandibular joint in different age periods

3.1. The deciduous dentition stage

The deciduous dentition stage starts from the eruption of the first deciduous tooth, usually the deciduous mandibular central incisors and ends with the eruption of the first permanent molar, i.e. from 6 months to 6 years of postnatal life. By 2.5 years of age, deciduous dentition is usually complete and in full function. Root formation of all deciduous teeth is complete by 3 years of age.

Normal signs of primary dentition:

a. Spaced anteriors: Spacing is usually seen in the deciduous dentition to accommodate larger permanent teeth in the jaws *(Fig. 3.1).*

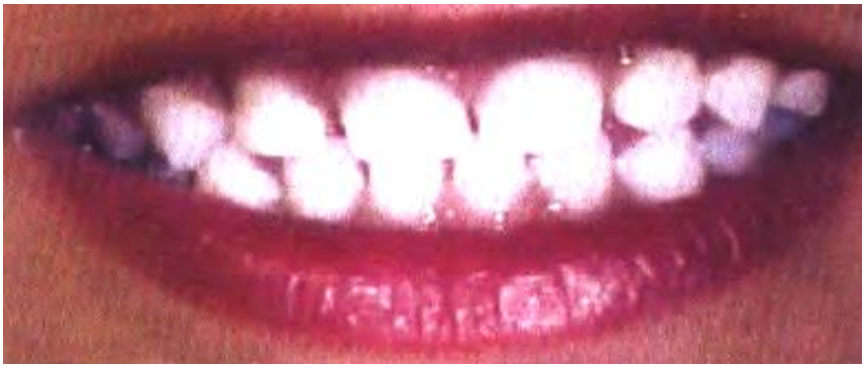

Fig. 3.1. Normal signs of primary dentition: spaced anteriors

b. Primate/ simian/ anthropoid space: This space is present mesial to the maxillary canine and distal to the mandibular canine. Most subhuman primates have it throughout life and use it for interdigitation of opposing canines. This space is used for early mesial shift *(Fig. 3.2, 3.3).*

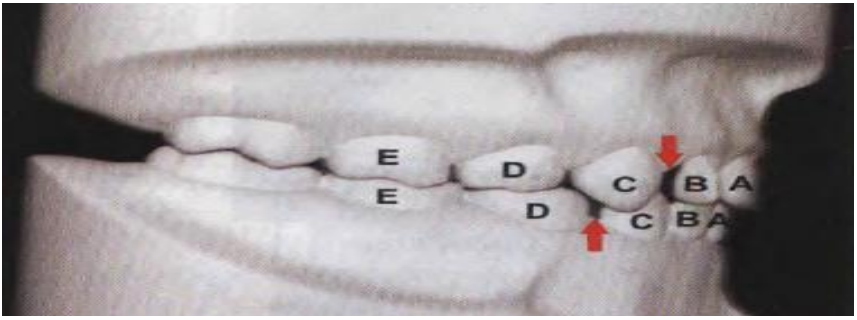

Fig. 3.2. Normal signs of primary dentition: primate/ simian/ anthropoid space

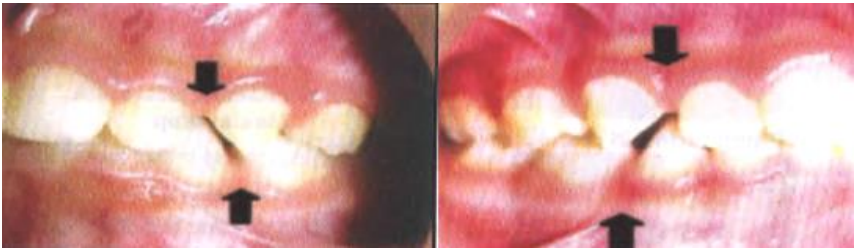

Fig. 3.3. Normal signs of primary dentition: primate/ simian/ anthropoid space

c. Shallow overjet and overbite *(Fig. 3.4).*

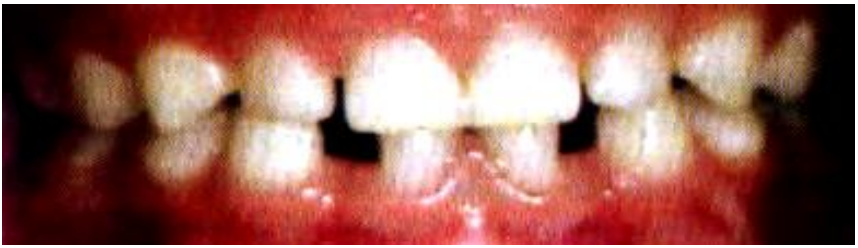

Fig. 3.4. Normal signs of primary dentition: shallow overjet and overbite

d. Almost vertical inclination of the anteriors.

e. Ovoid arch form.

f. Straight/flush terminal plane relation: The molar relationship in the primary dentition can be classified into 3 types *(Fig. 3.5).*

• *Flush terminal plane. When the distal surfaces of* maxillary and mandibular deciduous second molars are in the same vertical plane; this is the normal molar relationship in the primary dentition because the mesiodistal width of the mandibular molar is greater than the mesiodistal width of the maxillary molar.

• *Mesial step. Distal surface of mandibular* deciduous second molar is mesial to the distal surface of maxillary deciduous second molar.

• *Disial step. Distal surface of mandibular* deciduous second molar is more distal than the distal surface of maxillary deciduous second molar, i.e. the upper second molar occludes with two opposite teeth.

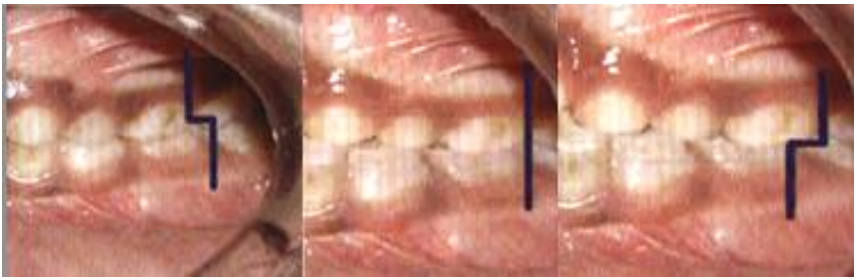

Fig. 3.5. Straight/flush terminal plane relation

Between 5 and 6 years, just before shedding of the deciduous incisors, there are more teeth in the jaws than at any other time.

3.2. The mixed dentition stage

This is the period where teeth of both deciduous and permanent dentition are seen. It extends from 6–12 years of age. Most malocclusions make their appearance during this stage. This stage can be divided into two transitional periods-first and second.

First transitional priod. Emergence of the first permanent molars and transition of incisors. The following events take place during this period.

Eruption of permanent first molars. The first permanent molars erupt at 6 years. They play an important role in the establishing and in the functioning of occlusion, in the permanent dentition. Antero-posterior positioning of the permanent molars is influenced by *(Fig. 3.6)*:

1. Terminal plane relationship

• When the deciduous second molars are in a flush terminal plane, the permanent first molar erupts initially into a cusp-to-cusp relationship, which later transforms into a Class I molar relation using the primate spaces. Later, cusp-to-cusp relationship of the permanent first molar can be converted to a Class I relationship by the mesial shift of the permanent first molar following exfoliation of the primary molar and thus making use of the Leeway space (late mesial shift).

• When the deciduous second molars are in a distal step, the permanent first molar will erupt into a Class II relation. This molar configuration is not self-correcting and will cause a Class II malocclusion despite Leeway space and differential growth.

• Primary second molars in mesial step relationship lead to a Class I molar relation in mixed dentition. This may remain or progress to a half or full cusp Class III with continued mandibular growth.

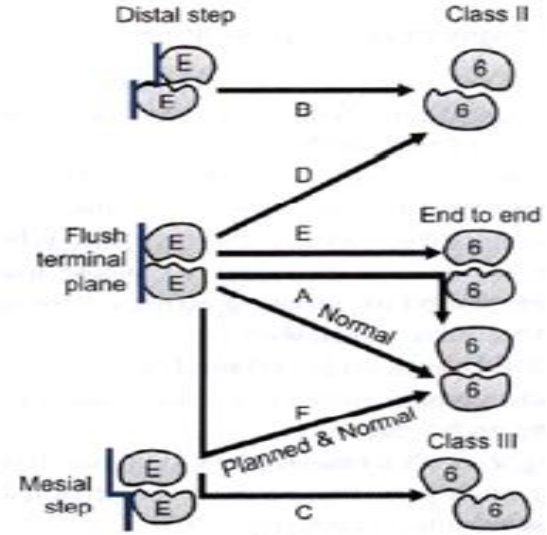

Fig. 3.6.
Occlusal relations of primary and permanent molars

2. Early mesial shift in arch with physiologic spacing: In a spaced arch, eruptive force of the permanent molars causes closing of any spaces between the primary molars or primate spaces, thus allowing molars to shift mesially *(Fig. 3.7.a).*

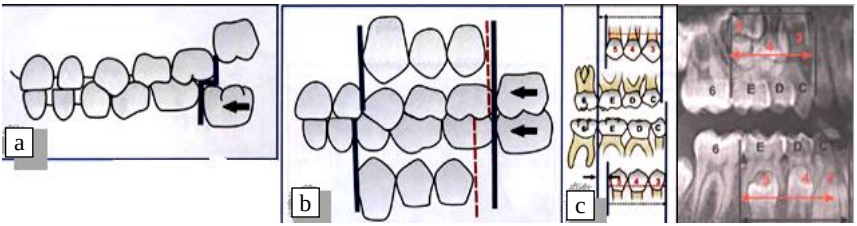

Fig. 3.7. a – Early shift of the erupting first permanent molars moving utilizing the primate spaces. b – Late shift by utilization of the Leeway space. c – Leeway space of Nance

3. Differential growth of maxilla and mandible.

Leeway space of Nance. The combined mesiodistal widths of deciduous canine, first and second molars is more than that of the combined mesiodistal width of permanent canine, first and second premolar *(Fig. 3.7. b. and 3.7. c).* The difference between the two is called the Leeway space (Maxilla 0.9 mm/segment = = 1.8 mm total. Mandible 1.7 mm/segment = 3.4 mm total).

Incisor eruption. Permanent incisors develop lingual to the primary incisors. For incisors to erupt in normal alignment, there is an obligate space requirement in the anterior part of both the arches which is termed as incisor liability, i.e. the total sum of the mesiodistal width of four permanent incisors is larger than that of primary incisors by 7.6 mm in maxilla and 6 mm in mandible (Wayne). This obligate space is provided by:

• Interdental physiologic spacing in the primary incisor region: 4 mm in maxillary arch, 3 mm in mandibular arch.

• Increase in the inter-canine arch width: Significant amount of growth occurs with the eruption of incisors and canines.

• Increase in the anterior length of the dental arches: Permanent incisors erupte labial to the primary incisors to obtain an added space of 2–3 mm.

Change in inclination of permanent incisors: Primary teeth are upright but permanent teeth incline to the labial surface thus decreasing the inter-incisal angle from about 150° in the deciduous dentition to *123° in the permanent dentition (Fig. 3.8)*. This increases the arch perimeter.

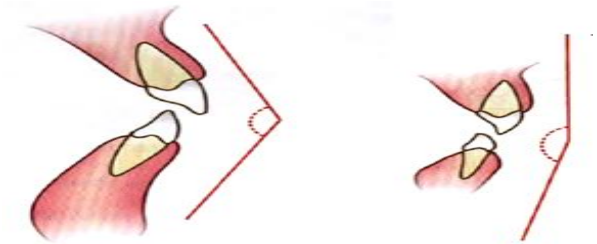

Fig. 3.8. Comparison of the angulation of the permanent and primary teeth

The Broadbent's phenomenon-ugly duckling stage (7–14 years). Around the age of 8 years, a midline diastema is commonly seen in the upper arch, which is usually misinterpreted by the parents as a malocclusion. Crowns of canines in young jaws impinge on developing lateral incisor roots, thus driving the roots medially and causing the crowns to flare laterally. The roots of the central incisors are also forced together thus causing a maxillary midline diastema. The period from the eruption of lateral incisor to canine is termed as the Ugly Duckling stage *(Fig. 3.9).* It is an unaesthetic metamorphosis, which eventually leads to an aesthetic result.

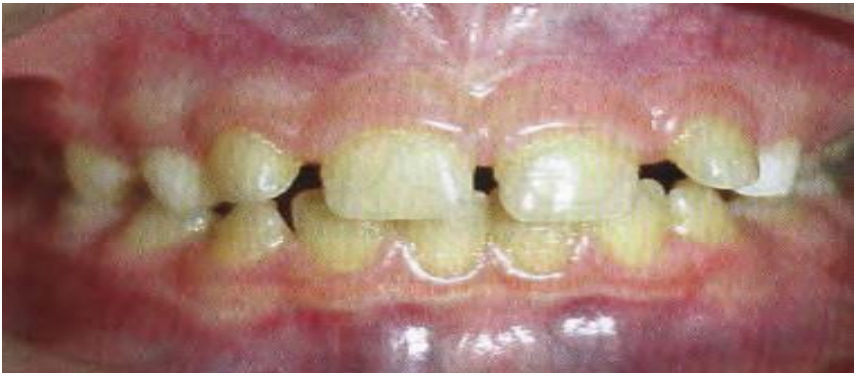

Fig. 3.9. Ugly Duckling stage

With eruption of canines, the impingement from the roots shifts incisally thus driving the incisor crowns medially, resulting in closure of the diastema *(Fig. 3.10).*

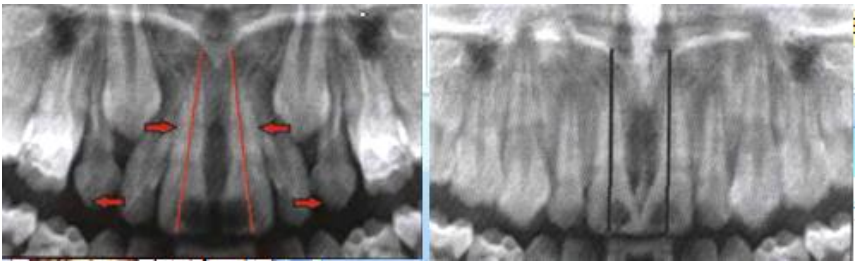

Fig. 3.10. Ugly Duckling stage. Canine crowns impinging of roots of lateral incisors with resultant distal flaring of incisor crowns.
Continued eruption of canines results in closure of midline space

The inter-transitional period (1.5 years). This is a stable phase where little changes take place in the dentition. The teeth present are the permanent incisors and first molar along with the deciduous canines and molars. Some of the features of this stage are:

1. Any asymmetry in emergence and corresponding differences in height levels or crown lengths between the right and left side teeth are made up.

2. Occlusal and interproximal wear of deciduous teeth causes occlusal morphology to approach that of a plane.

3. Ugly duckling stage.

4. Root formation of emerged incisors, canines and molars continues, along with concomitant increase in alveolar process height.

5. Resorption of roots of deciduous molars.

This phase prepares for the second transitional phase.

The second transitional period. The following events take place:

1. Exfoliation of primary molars and canines. At around 10 years of age, the first deciduous tooth in the posterior region, usually the mandibular canine sheds and marks the beginning of the second transitional period. Usually no crowding is seen before emergence except maybe between the maxillary first premolar and canine.

2. Eruption of permanent canines and premolars. These teeth erupt after a pause of 1–2 years following incisor eruption. The first posterior teeth to erupt are the mandibular canine and first premolar (9–10 years) followed by maxillary premolars and canine around 11–12 years. Most common eruption sequence is 4–5–3 in the maxilla and 3–4–5 in the mandible. Favorable occlusion in this region is largely dependent on:

• Favorable eruption sequence.

• Satisfactory tooth size- available space ratio.

• Attainment of normal molar relation with minimum diminution of space available for bicuspids.

3. Eruption of permanent second molars. Before emergence second molars are oriented in a mesial and lingual direction. These teeth are formed palatally and are guided into occlusion by the Cone Funnel mechanism (the upper palatal cusp/cone slides into the lower occlusal fossa/funnel). The arch length is reduced prior to second molar eruption by the mesial eruptive forces. Therefore, crowding if present is accentuated.

4. Establishment of occlusion.

3.3. The permanent dentition stage

The permanent dentition forms within the jaws soon after birth. Calcification begins at birth with the calcification of the cusps of the first permanent molar and extends as late as the 25th year of life. Complete calcification of incisor crowns takes place by 4 to 5 years and of the other permanent teeth by 6 to 8 years except for the third molars. Therefore the total calcification period is about 10 years. The permanent incisors develop lingual to the deciduous incisors and move labially as they erupt. The premolars develop below the diverging roots of the deciduous molars.

Nolla (1960) gave stages of tooth development to make a meaningful assessment of eruption from panoramic/posteroanterior radiographs which is given in *table 3.1*.

Table 3.1

Chronology of tooth development

Tooth		Tooth germ fully developed	Dentine formation begins	Calcification begins	Crown formation complete	applearance in oral cavity	Root complete
Decidudus	Incisors	3–4 mths i.u.l.	4–6 mths i.u.l.		2–3 mths	6–9 mths	1–1,5 yrs after appearance in the mouth
	Canines				9 mths	16–18 mths	
	1st Molars				6 mths	12–14 mths	
	2nd Molars				12 mths	20–30 mths	
Permanent	Maxillary centrals	30th week i.u.l.	3–4 mths	3–4 mths	4–5 yrs	7–9 yrs	2–3 yrs after appearance in the mouth
	Mandibular centrals			3–4 mths		6–8 yrs	
	Maxillary laterals		10–12 mths	10–12 mths		7–9 yrs	
	Mandibular laterals		3–4 mths	3–4 mths		6–8 yrs	
	Maxillary canines	30th week i.u.l.	4–5 mths	4–5 mths	6–7 yrs	11–12 yrs	2–3 yrs after appearance in the mouth
	Mandibular canines					9–10 yrs	
	Maxillary 1st premolar	30th week i.u.l.	1,5–2,5 yrs	1,5–1,75 yrs	5–6 yrs	10–11 yrs	2–3 yrs after appearance in the mouth
	Mandibular 1st premolar			1,75–2 yrs		10–12 yrs	
	Maxillary 2nd premolar			2–2,25 yrs	6–7 yrs	10–12 yrs	
	Mandibular 2nd premolar			2,25–2,5 yrs		11–12 yrs	
	1st molar	24th week i.u.l.	Before birth	At or shortly after birth	2,5–3 yrs	6–7 yrs	2–3 yrs after appearance in the mouth
	2nd molars	6th mths	2,5–3 yrs	2,5–3 yrs	7–8 yrs	11–13 yrs	
	3rd molars	6th yrs	7–10 yrs	7–9 yrs	12–16 yrs	17–21 yrs	
i.u.l. = intra uterine life							

At approximately 13 years of age all permanent teeth except third molars are fully erupted. Before the deciduous incisors are shed, there are 48 teeth/parts of teeth present in the jaws.

Features of the permanent dentition:

• Coinciding midline.

• Class 1 molar relationship of the permanent first molar.

• Vertical overbite of about one-third the clinical crown height of the mandibular central incisors.

• Overjet: Overjet and over bite decreases throughout the second decade of life due to greater forward growth of the mandible.

• Curve of Spee: Develops during transition and stabilizes in adulthood.

Eruption. Eruption is the developmental process that moves a tooth from its crypt position through the alveolar process into the oral cavity and to occlusion with its antagonist. During eruption of succedaneous teeth:

• Primary tooth resorbs

• Roots of the permanent teeth lengthen

• Increase in the alveolar process height

• Permanent teeth move through the bone.

Teeth do not begin to move occlusally until crown formation is complete. It takes 2–5 years for posterior teeth to reach the alveolar crest following crown completion and 12–20 months to reach occlusion after reaching alveolar margin.

Process of tooth eruption. Tencate divided tooth eruption into the following 3 stages:

1. Pre-eruptive tooth movement: Movement of tooth germs within the jaws before they begin to erupt.

2. Eruptive tooth movement: Tooth movement from its position within the jaws to its functional position.

3. Post-eruptive tooth movement: Maintaining the position of erupted tooth in occlusion while the jaws continue to grow.

Tooth eruption can also be divided into:

1. Pre-emergent eruption during the stage of crown formation, there is very slow labial or buccal drift of the tooth follicle within the bone. However, there is no eruptive movement.

Two processes are necessary for a tooth to erupt intra-osseously:

• Resorption of bone and primary tooth roots overlying the crown of the erupting tooth.

• Eruption mechanism itself must move the tooth in the direction where the path has been cleared.

Resorption is the rate-limiting factor in preemergent eruption.

2. Post-emergent eruption Once a tooth emerges into the mouth, it erupts rapidly (about 4 mm in 14 weeks) until it approaches the occlusal level and is subjected to the forces of mastication. This stage of relatively rapid eruption is called as the postemergent spurt. This is followed by a phase of Juvenile Occlusal Equilibrium, i.e. after teeth reach the occlusal level; eruption becomes almost imperceptibly slow although it definitely continues. After the teeth are in function, they erupt at the same rate as the vertical growth of the mandibular ramus unless there is occlusal wear or the antagonist tooth is lost at any age, in which case additional eruption occurs. When the pubertal growth ends, a final phase of tooth eruption takes place known as Adult Occlusal Equilibrium.

Factors determining tooth position during eruption. Tooth passes through four distinct stages of development:

1. Pre-eruptive initially position of tooth germ is dependent on heredity.

2. Intra-alveolar tooth position is affected by-

• Presence or absence of adjacent teeth

• Rate of resorption of primary teeth

• Early loss of primary teeth

• Localized pathologic conditions.

3. Intraoral stage Tooth can be moved by lip, cheek, tongue muscles or external objects and drift into spaces.

4. Occlusal stage. Muscles of mastication exert influence through interdigitation of cusps. The periodontal ligament disseminates the strong forces of chewing to the alveolar bone.

*Sequence of eruption***.** There is wide variability in the sequence of arrival of teeth in the mouth.

Maxilla 6-1-2-4-3-5-7 or 6-1-2-4-5-3-7 (most common)

Mandible 6-1-2-4-5-3-7or 6-1-2-3-4-5-7(most common)

Dental age 6: First stage of eruption.

• Eruption of mandibular central incisor and permanent first molar.

• Mandibular molar eruption precedes maxillary molar.

Dental age 7

• Eruption of maxillary central and mandibular lateral incisor.

• Root formation of maxillary lateral incisor well advanced.

• Crown completion of canines and premolars.

Dental age 8

• Eruption of maxillary lateral incisor.

• Delay of 2-3 years before any further teeth erupt.

Dental age 9

• One-third root formation of mandibular canine and first premolar is complete.

• Root development of mandibular second premolar begins.

Dental age 10

• One-half root formation of mandibular canine and first premolar is complete.

• Significant root development of maxillary and mandibular second premolar as well as maxillary canine.

• Root completion of mandibular incisors and near completion of maxillary laterals.

• According to Movers, mandibular canine eru pts between 9 and 10 years.

Dental age 11

• Eruption of mandibular canine (according to Proffit), mandibular first premolar and maxillary first premolar.

• Maxillary first premolar erupts ahead of canine and second premolar.

Dental age 12

• Remaining succedaneous teeth erupt.

• Second permanent molars nearing eruption

• Early beginnings of third molar

Dental age 13, 14, 15

• Completion of roots of permanent teeth

• Third molars apparent on the radiograph

Change in eruption sequence is a reliable sign of disturbance in normal development of the dentition.

Certain normal variations with important clinical significance:

• Eruption of second molars ahead of premolars in the mandibular arch. This decreases the space for second premolars, which get partially blocked out.

• Eruption of maxillary canines ahead of premolars will cause the canines to be forced out labially.

• Asymmetries in eruption between the right and left sides occurs when there is lack of space to accommodate erupting teeth due to different pattern of mechanical obstruction, decreased space on one side compared to the other.

***Dimensional changes in the dental arches*.** The usual arch dimensions measured are:

1. Widths of the canines, primary molars (premolars) and first permanent molars:

a. Dimensional increase in width involves alveolar process growth almost totally, since there is little skeletal width increase at this time.

b. Clinically significant differences exist in the manner and magnitude of width changes in the maxilla and mandible. Width increase correlates highly with vertical alveolar process growth. Maxillary alveolar processes diverge while mandibular alveolar processes are more parallel. Thus, maxillary width increases more and can be easily altered in treatment.

c. The only significant increase in mandibular inter-canine width occurs during eruption of incisors when primary cuspids are moved distally into primate spaces and does not increase significantly thereafter. d. Maxillary arch width increase is timed with periods of active eruption of teeth. Eruption of maxillary permanent canines is an important factor in widening of the arch.

e. Maxillary premolar width increase is coincidental with vertical growth whereas mandibular premolar width increase occurs because of further buccal placement of premolar crown**s.**

2. Length or depth: Arch length or depth is measured at the midline from a point midway between central incisors to a tangent touching distal surfaces of second primary molars or premolars. Any changes in arch length are coarse reflections of changes in perimeter.

3. Arch circumference or perimeter: Measured from distal surface of second primary molar or mesial surface of first permanent molar around the arch over contact points and incisal edges in a smoothened curve to the distal of second primary molar or mesial surface of first permanent molar of the opposite side. The reduction in mandibular arch circumference during transitional and early adolescent dentition is a result of:

a. Late mesial shift of first permanent molar as "Leeway space" is pre-empted.

b. Mesial drifting tendency of posterior teeth throughout life.

c. Slight interproximal wear of teeth.

d. Lingual positioning of incisors.

e. Original tipped position of incisors and molars.

***The stages of physiological occlusion height elevation*.** There are singled out 4 stages of the physiological elevation of occlusion height:

• the 1st falls on 2–2.5 years, i.e. the moment of all temporary teeth eruption completion;

• the 2nd is marked at the age of 6 years, i.e. the time of 1st permanent molars eruption;

• the 3rd – 12–13 years, after the complete replacement of temporary teeth with permanent, due to the vertical growth of the alveolar process, full value eruption and regular reciprocal arrangement of other permanent molars;

• the 4lh – 18–25 years, i.e. as a result of the eruption and regular articulation of the 3rd molars; if they are absent, occlusion height elevation occurs at the expense of dentoalveolar lengthening. At all stages, as occlusion height increases dynamically the frontal overbite depth decreases, and dentitions correlation becomes orthognathic.

3.4. Occlusion-basic concept

In the clinical picture of orthodontics there are distinguished four forms of physiological occlusion: orthognathic, straight, biprognathic, and opisthognathic. All these occlusions have identical signs of closure in the region of molar and premolar teeth, and different signs – in the region of incisors and canine teeth *(Fig. 3.11)*.

The sign of the correct sagittal occlusion of molar teeth is the position of the anterior buccal tubercle of the Ist upper molar in the transverse sulcus of the similar lower tooth.

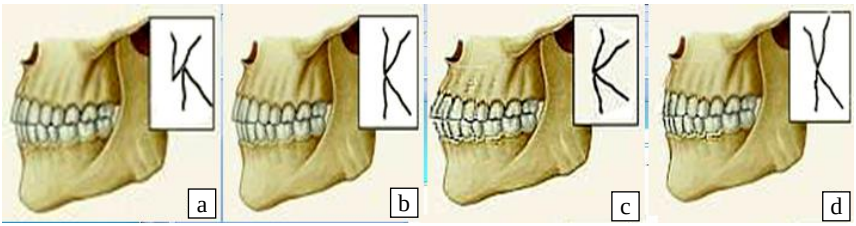

Fig. 3.11. Physiological types of occlusion: a – orthognathic occlusion, b – straight occlusion, c. physiological biprognathism, d. opisthognathic occlusion

Orthognathic bite of permanent teeth in central occlusion is characterised by the following *(Fig. 3.11. a)*:

• the upper frontal teeth cover the lower ones by 1/3 length of the lower teeth crowns;

• the tubercle of the upper canine tooth crown is located between the lower canine tooth and the 1st premolar;

• the centerline between the central incisors of the upper and lower jaws coincides;

• the mesial-buccal tubercle of the upper Ia molar is located in the transverse sulcus of the similar lower tooth;

• every tooth of the upper jaw has two antagonists – similar and standing behind (except for the lower central incisors and upper wisdom teeth);

• the buccal tubercles of the upper lateral teeth cover the buccal tubercles of the lower ones, and the palatine tubercles of the upper teeth are located between the buccal and lingual tubercles of the lower teeth;

• the upper dental arch is semielliptical, the lower – parabolic; in temporary occlusion – a semicircle on both jaws;

• the dental arches of the upper and lower jaws are symmetrical;

• in the state of central occlusion there is a full occlusive contact between all teeth (except for unerupted ones);

• in the state of physiological rest an interocclusive space varying within 2 mm arises between dental arches.

The highest esthetic optimum, the highest indexes of mastication function, the best conditions for the formation of somatic swallowing, and full value tongue function are characteristic of this occlusion.

• At *straight occlusion* all the listed above correlations are kept, except for the occlusion character of frontal teeth, which contact with the lower sculpri *(Fig. 3.11. b).*

• *Physiological biprognathism* – all the listed above correlations are kept, except for the character of frontal teeth occlusion — they have a vestibular inclination of the upper and lower incisors and canine teeth at minor covering of th**e** lower teeth by the upper ones *(Fig. 3.11. c).*

At *opisthognathic occlusion* the canine teeth and incisors on both jaws are inclined into the oral cavity, contacting with each other by means of covering the lower teeth by the upper ones at the level of teeth tubercles or by means of marginal occlusion; all the listed above occlusive correlations are preserved *(Fig. 3.11. d).*

Andrews six keys to normal occlusion. Lawrence F Andrews studied 120 casts of nonorthodontic patients with normal occlusion for four years (1960–1964). He identified 6 key characteristics. He was of the opinion, that for normal occlusion to exist these six characteristics had to be present. According to Andrews, the 6 keys to normal occlusion contributed individually and collectively to the total scheme of occlusion and, were therefore essential for an orthodontic treatment to be considered successful. The six keys were:

Key I (Fig. 3.12. a). Molar relationship . The molar relationship should be such that the distal surface of the distal marginal ridge of the upper first permanent molar contacts and occludes with the mesial surface of the mesial marginal ridge of the lower second molar. Secondly, the mesiobuccal cusp of the upper first permanent molar falls within the groove between the mesial and middle cusps of the lower first permanent molar. Also, the mesiolingual cusp of the upper first molar seats in the central fossa of the lower first molar.

Key 2 (Fig. 3.12. b). Crown angulation, the mesiodistal "tip". In normally occluded teeth, the gingival portion of the long axis (the line bisecting the clinical crown mesiodistally or the line passing through the most prominent part of the labial or buccal surface of a tooth) of each crown is distal to the occlusal portion of that axis. The degree of tip varies with each tooth type.

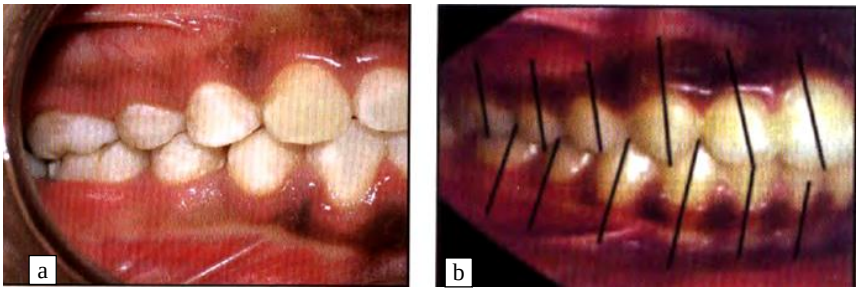

Fig. 3.12. a – The Andrews first key – molar relationship.
b – The Andrews second key – crown angulation, the mesiodistal "tip"

Key III. (Fig. 3.13. a). Crown inclination, the labiolingual or buccolingual, "torque". Crown inclination is the angle between a line 90 degrees to the occlusal plane, and a line tangent to the middle of the labial or buccal surface of the clinical crown. The crowns of the maxillary incisors are so placed that the incisal portion of the labial surface is labial to the gingival portion of the clinical crown. In all other crowns, the occlusal portion of the labial or buccal surface is lingual to the gingival portion. In the maxillary molars the lingual crown inclination is slightly more pronounced as compared to the cuspids and bicuspids. In the mandibular posterior teeth the lingual inclination progressively increases.

Key IV (Fig. 3.13. b). Absence of Rotations. Teeth should be free of undesirable rotations. If rotated, a molar or bicuspid occupies more space than it would normally. A rotated incisor can occupy less space than normal.

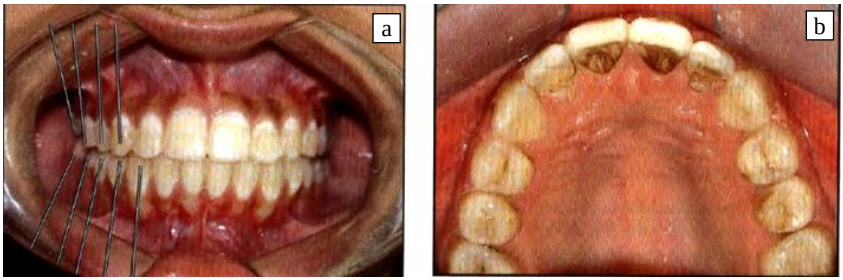

Fig. 3.13. a – The Andrews key III – crown inclination, the labiolingual or buccolingual, "torque". b – The Andrews key IV – absence of rotations

Key V (Fig. 3.14. a). Tight contacts. In the absence of such abnormalities as genuine tooth-size discrepancies, contact points should be tight.

Key VI (Fig. 3.14. b). Flat curve of Spee. A flat occlusal plane is a must for stability of occlusion. It is measured from the most prominent cusp of the lower second molar to the lower central incisor, no curve deeper than 1.5 mm is acceptable from a stand point of stability.

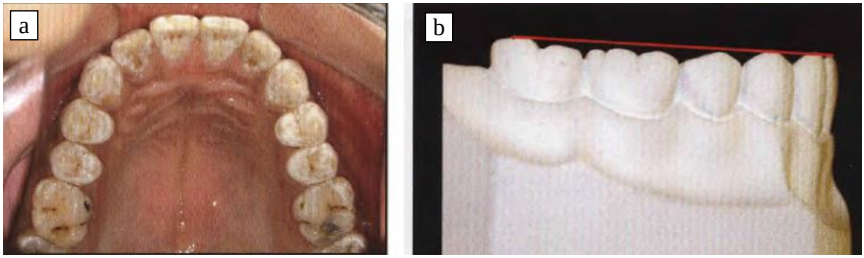

Fig. 3.14. a – The Andrews key V – tight contacts.
b – The Andrews key VI – flat curve of Spee

Based on relationship of 1st permanent molar. Depending on the anteroposterior jaw relationship, Edward H Angle classified occlusion into 3 types. *Class I* (also known as neutro-occlusion) *(Fig. 3.15)*: Dental relationship in which there is normal anteroposterior relationship, as indicated by the correct interdigitation of maxillary and mandibular molars (crowding, rotation or other individual tooth malrelations may be present elsewhere in the arch).

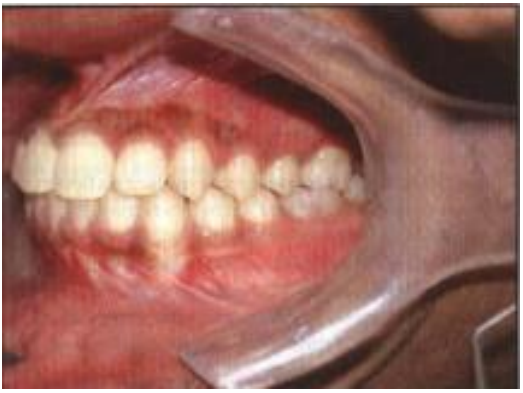

Fig. 3.15. The neutro-occlusion – molar relation Angle's class I

Compensatory curvatures. The occlusal surfaces of dental arches do not generally conform to a flat plane.

a. According to Wilson the mandibular arch appears concave and that of maxillary arch convex.

b. According to Bonwill, the maxillary and mandibular arches adapt themselves in part to an equilateral triangle of similar sides.

c. According to Von Spee, cusps and the incisal ridges of the teeth display a curved alignment when the arches are observed from a point opposite the 1st molar. The curve of Spee, as it is frequently called, is seen from the sagittal plane.

d. Monson connected the curvature in the sagittal plane with compensatory curvatures in the vertical plane and suggested that the mandibular arch adapts itself to the curved segment of a sphere of similar radius.

Here, the maxillary canine guides the mandible, so that the posterior teeth come into occlusion with a minimum of horizontal forces.

*Curve of Spee***.** lt refers to the anteroposterior curvature of the occlusal surfaces, beginning at the tip of the lower cuspid and following cusp tip of the bicuspids and molars continuing as an arc through to the condyle *(Fig. 3.16. a).* If the curve were extended, it would form a circle of about 4 inches diameter.

Curve of Wilson. It is a curve that contacts the buccal and lingual cusp tips of the mandibular posterior teeth. The curve of Wilson is mediolateral on each side of arch. It results from the inward inclination of the lower posterior teeth *(Fig 3.16. b and c).*

The curve helps in two ways

1. Teeth aLigned parallel to the direction of medial pterygoid for optimum resistance to masticatory forces.

2. The elevated buccal cusps prevent food from going 'past the occlusal table.

Fig. 3.16. a – The curve of Spee: A linefromthe tip of the canine touching the tips of the buccal cusps of the posterior teeth (drawnon jaws of a skull). b – A curve drawn on the third molars (of a skull). c – Curves on the first and the second molars of the mandible. Note the flattening of the curve as it progresses distally (the curves have been exaggerated for easy understanding)

3.5. Anatomy and function of temporomandibular joint in different age periods

The temporomandibular joint (TMJ) is a complex joint not only concerning its anatomic structure but also concerning its function. It belongs to binate, combined, incongruent joints.

The TMJ on both sides (left and right) represent a closed circuit, because movement in one joint brings on movement in the other one. The joint is diaxonic, movements in it happen in two directions: horizontal and vertical.

The joint consists of the lower jaw articular head, temporal bone glenoid fossa, temporal bone articular tubercle, articular disk, articular capsule (articular bursa), and articular ligaments *(Fig. 3.17. a–d):*

A newborn child has such peculiarities of the TMJ *(Fig. 3.17. c and d)*:

- The articular process head is of round shape, has almost equal dimensions (diametrical and anteroposterior).
- Forward inclination is not yet evident.
- The head is covered with a thick layer of fibrous connective tissue.
- The glenoid fossa, which is a receptacle for the mandible heads, is flat and round; it does not have an articular tubercle, and from behind – a well-marked

articular cone, which limits the motions of the lower jaw to the side of the middle ear and prevents the pressure of the head onto the middle ear tympanic part.

- The mandibular fossa functions fully as the lower jaw is dislocated distally (the state of physiological childhood retrognathia).
- The articular head is located in the posterior part of the mandibular fossa.
- Fossa fornix bone thickness considerably exceeds 2 mm.
- Mandibular fossa depth is a little bigger than 2 mm.
- The intra-articular disk is a soft intcrlayer of round shape, concave from below and convex from above, with slightly noticeable thickening from the front and from the back; the disk consists of collagenous fibers for the most part.
- The articular capsule synovial membrane villi are absent;.
- the absence of the articular tubercle, occipital inclination of the underdeveloped mandible branch, physiological retrognathia, wide flat fossa, not formed intrauterine disk and articular cone create favorable conditions for the lower jaw movements in the sagittal plane, which are needed for the adequate progress of the sucking function.
- With age the articular head inclines forward relative to the articular process neck. In infancy the lower jaw takes the distal position (physiological retrognathia). With temporary teeth eruption and occlusion height increase further forward shift of the articular head takes place. Articular surface in the anterosuperior part of the articular head is covered with cartilage, and in a newborn – with fibrous connective tissue, in adults – with fibrous cartilage, which becomes thinner with age.

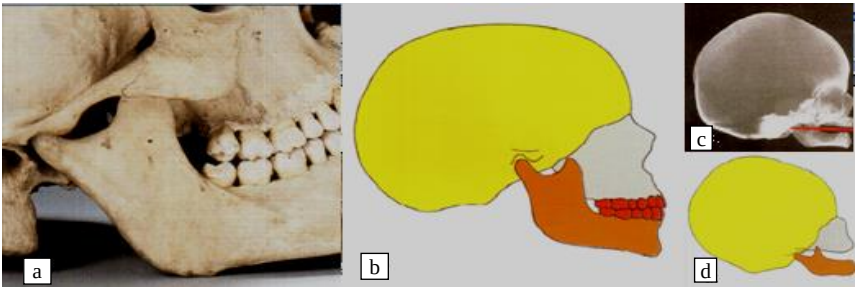

Fig. 3.17. a and b – Temporomandibular joint of adult;
c and d – Temporomandibular joint of newborn

- The mandibular neck is narrowed, there is a pterygoid fossa on its anterior surface, where the most part of the upper head of the lateral pterygoid muscle attaches. Wing fossa formation is observed at the age of 5 years, it looks like a narrow, shallow transversal sulcus.
- Normally, the articular head transfers pressure through the central part of the intra-articular disk onto the articular tubercle posterior clivus.
- Later on, the mandibular fossa depth also increases. It is connected with the growth of the temporal bone zygomatic process, which forms the articular

tubercle and provides deepening of the glenoid fossa and separation of the articular surface from the squama temporal surface. With age the glenoid fossa enlarges, predominantly in the transversal direction, and deepens, which corresponds to the changes of the lower jaw head, and acquires ellipsoidal shape. Articular surface is covered with fibrous cartilage.

- Fossa dimensions are bigger than head dimensions by 2–3 times, therefore incongruence takes place (inadequacy of head and fossa dimensions). Joint surfaces incongruence evens out due to the narrowing of fossa dimensions at the expense of joint capsule attachment from within close to the anterior margin of the temporal bone petrotympanic fissure, and is also compensated by the articular disk, which separates the articular cavity into two chambers, providing the high congruence of articular surfaces.
- The articular disk joins articular surfaces and is a counterpart of the lower jaw head and the articular tubercle posterior clivus, increasing the area of articular surfaces contact.
- The articular tubercle is absent in newborns, it only begins to show from the front of the mandibular fossa. With temporal bone zygomatic process growth and temporary teeth eruption its dimensions increase gradually. At the age of 6–7 years it is well-marked. The articular tubercle in adults is an ellipsoidal opening.
- As joints bony masses are forming, the disk is forming simultaneously. Disk form changes are connected with the securing of articular surfaces congruence. Intra-articular disk gradually acquires anterior and posterior thickening and a thin central part. The superior temporal surface of the disk is convex from behind and saddleshaped from the front, and the inferior surface is concave – repeats the form of the lower jaw head and creates a kind of an additional movable fossa.
- The articular capsule defines anatomic and physiological limits of the TMJ. It is an elastic connective tissue "saccule" containing articular surfaces of bones, which are parts of the joint, and joins the disk along its perimeter. It has the form of a watering-pot converging from top to bottom. The attachment of the capsule to the temporal bone is kind of shifted forward relative to the mandibular fossa. From behind it attaches along the anterior margin of the petrotympanic fissure and divides the mandibular fossa into the anterior intracapsular and posterior extracapsular parts. The capsule also surrounds the surface of the lower jaw head. It is characterised by high strength and elasticity, does not tear at complete joint dislocation.

The synovial membrane has outgrowths, the so-called villi, which are interoreception regions. Depending on age their number and location are different. Newborns do not have the villi. There are few of them at the age of 1–2 years, more – at the age of 3–6 years. At the age of 16–18 years there are a lot of them. As the organism ages, villi involution takes place. In children aged 1–3 years the joint space size in the anterior part fluctuates from 2 to 3 mm, in the superior – from 3 to 4 mm, in the posterior – from 3.5 to 5 mm. TMJ elements are fully formed by the age of 15–17 years.

Chapter 4. Functions of oral cavity. Effects on the dentition, jaw size and shape

Environmental influences during the growth and development of the face, jaws, and teeth consist largely of pressures and forces related to physiologic activity. Function must adapt to the environment. For example, how you chew and swallow will be determined in part by what you have to eat; pressures against the jaws and teeth will occur during both activities and could affect how jaws grow and teeth erupt.

A relationship between anatomic form and physiologic function is apparent in all animals. Over evolutionary time, adaptations in the jaws and dental apparatus are prominent in the fossil record. Form-function relationships at this level are controlled genetically and, though important for a general understanding of the human condition, have little to do with any individual's deviation from the current norm.

On the other hand, there is every reason to suspect that form-function relationships during the lifetime of an individual may be significant in the development of malocclusion. Although the changes in body form are minimal, an individual who does heavy physical work as an adolescent has both heavier and stronger muscles and a sturther skeletal system than one who is sedentary. If function could affect the growth of the jaws, altered function would be a major cause of malocclusion, and it would be logical for chewing exercises and other forms of physical therapy to be an important part of orthodontic treatment. But if function makes little or no difference in the individual's pattern of development, altering his or her jaw function would have little if any impact, etiologically or therapeutically. Because of its importance in contemporary orthodontics, particular emphasis is placed here on evaluating potential functional contributions to the etiology of malocclusion and to possible relapse after treatment.

Equilibrium theory and development of the dental occlusion. Equilibrium theory, as applied in engineering, states that an object subjected to unequal forces will be accelerated and thereby will move to a different position in space. It follows that if any object is subjected to a set of forces but remains in the same position, those forces must be in balance or equilibrium. From this perspective, the dentition is obviously in equilibrium, since the teeth are subjected to a variety of forces but do not move to a new location under usual circumstances. Even when teeth are moving, the movements are so slow that a static equilibrium can be presumed to exist at any instant in time.

The effectiveness of orthodontic treatment is itself a demonstration that forces on the dentition are normally in equilibrium. Teeth normally experience forces from masticatory effort, swallowing, and speaking but do not move. If a tooth is subjected to a continuous force from an orthodontic appliance, it does move. From an engineering point of view, the force applied by the orthodontist has altered the previous equilibrium, resulting in tooth movement.

Equilibrium considerations also apply to the skeleton, including the facial skeleton. Skeletal alterations occur all the time in response to functional demands and are magnified under unusual experimental situations. The bony processes to

which muscles attach are especially influenced by the muscles and the location of the attachments. The form of the mandible, because it is largely dictated by the shape of its functional processes, is particularly prone to alteration. The density of the facial bones, like the skeleton as a whole, increases when heavy work is done and decreases in its absence.

Equilibrium effects on the dentition. Equilibrium effects on the dentition can be understood best by observing the effect of various types of pressures. Although one might think that force multiplied by duration would explain the effects, this is not the case. The duration of a force, because of the biologic response, is more important than its magnitude.

This important point is made clearer by examining the response to the forces applied during chewing. When heavy masticatory forces are applied to the teeth, the fluid-filled periodontal ligament acts as a shock absorber, stabilizing the tooth for an instant while alveolar bone bends and the tooth is displaced for a short distance along with the bone. If the heavy pressure is maintained for more than a few seconds, increasingly severe pain is felt, and so the biting force is released quickly. This type of heavy intermittent pressure has no impact on the long-term position of a tooth. A number of pathologic responses to heavy intermittent occlusal contacts on a tooth may occur, including increased mobility and pain, but as long as the periodontal apparatus is intact, forces from occlusion are rarely prolonged enough to move the tooth to a new position in which the occlusal trauma is lessened.

A second possible contributor to the equilibrium that governs tooth position is pressure from the lips, cheeks, and tongue. These pressures are much lighter than those from masticatory function, but are also much greater in duration. Experiments suggest that even very light forces are successful in moving teeth, if the force is of long enough duration. The duration threshold seems to be approximately 6 hours in humans. Since the light pressures from lips, cheeks, and tongue at rest are maintained most of the time, one would expect these pressures to affect tooth position.

It is easy to demonstrate that this is indeed the case. For example, if an injury to the soft tissue of the lip results in scarring and contracture, the incisors in this vicinity will be moved lingually as the lip tightens against them. On the other hand, if restraining pressure by the lip or cheek is removed, the teeth move outward in response to unopposed pressure from the tongue. Pressure from the tongue, whether from an enlargement of the tongue from a tumor or other source or because its posture has changed, will result in labial displacement of the teeth even though the lips and cheeks are intact, because the equilibrium is altered.

These observations make it plain that, in contrast to forces from mastication, light sustained pressures from lips, cheeks, and tongue at rest are important determinants of tooth position. It seems unlikely, however, that the intermittent short-duration pressures created when the tongue and lips contact the teeth during swallowing or speaking would have any significant impact on tooth

position.' As with masticatory forces, the pressure magnitudes would be great enough to move a tooth, but the duration is inadequate.

Another possible contributor to the equilibrium could be pressures from external sources, of which various habits and orthodontic appliances would be most prominent. As an example, an orthodontic appliance that created light pressure on the inside of the dental arch might be used to expand the teeth laterally and anteriorly, creating enough space to bring all teeth into alignment. After a certain amount of arch expansion, cheek and lip pressure begins to increase. One could expect that as long as the appliance remained in place, even though it no longer exerted any active force, it would serve as a retainer to counter these increased forces. When the appliance was removed, however, the equilibrium would again be unbalanced, and the teeth would collapse lingually until a new position of balance was reached.

Whether a habit can serve in the same way as an orthodontic appliance to change the position of the teeth has been the subject of controversy since at least the first century AD, when Celsus recommended that a child with a crooked tooth be instructed to apply finger pressure against it so that it would be moved to its proper position. From our present understanding of equilibrium, we would expect that this would work, if the child kept the finger pressure against the tooth for 6 hours or more per day.

The same reasoning can be applied to other habits: if a habit like thumbsucking created pressure against the teeth for more than the threshold duration (6 hours or more per day), it certainly could move teeth. On the other hand, if the habit had a shorter duration little or no effect would be expected, no matter how heavy the pressure. Whether a behavior pattern is essential or nonessential, innate or learned, its effect on the position of the teeth is determined not by the force that it applies to the teeth but by how long that force is sustained.

This concept also makes it easier to understand how playing a musical instrument might relate to the development of a malocclusion. In the past, many clinicians have suspected that playing a wind instrument might affect the position of the anterior teeth, and some have prescribed instruments as part of orthodontic therapy. Playing a clarinet, for instance, might lead to increased overjet because of the way the reeds are placed between the incisors, and this instrument could be considered both a potential cause of a Class II malocclusion and a therapeutic device for treatment of Class III. String instruments like the violin and viola require a specific head and jaw posture that affects tongue vs lip/cheek pressures and could produce asymmetries in arch form. Although the expected types of displacement of teeth are seen in professional musicians, even in this group the effects are not dramatic, and little or no effect is observed in most children. It seems quite likely that the duration of tongue and lip pressures associated with playing the instrument is too short to make any difference, except in the most devoted musician.

Another possible contributor to the dental equilibrium is the periodontal fiber system, both in the gingival tissues and within the periodontal ligament. We have already noted that if a tooth is lost, the space tends to close, in part because of force created by the transseptal fibers in the gingiva. The importance of this force has been demonstrated experimentally in monkeys by extracting a tooth and then making repeated incisions in the gingiva so that the transseptal fiber network is disrupted and cannot reestablish continuity. Space closure is almost completely abolished under these circumstances.

The same gingival fiber network stretches elastically during orthodontic treatment and tends to pull the teeth back toward their original position. Clinical experience has shown that after orthodontic treatment, it is often wise to eliminate this force by making gingival incisions that sever the stretched transseptal fibers, thereby allowing them to heal with the teeth properly aligned. In the absence of a space created by extraction or orthodontic tooth movement, however, the gingival fiber network apparently has minimal effects on the dental equilibrium.

The periodontal ligament itself can contribute to the forces that make up the dental equilibrium. Exactly how the eruption mechanism works is still not completely understood, but it seems clear now that the eruptive force is generated within the periodontal ligament. This force is large enough and sustained enough to move a tooth. It seems likely that the same metabolic activity can and does produce forces that serve as a part of "active stabilization" for teeth, direcdy contributing to the equilibrium. The extent to which this occurs in teeth that are not erupting is not known. It is known, however, that the eruption mechanism remains at least potentially active throughout life, since a tooth can begin to erupt again many years after eruptive movements have apparently ceased, if its antagonist is extracted. Thus there is at least the potential for metabolic activity in the periodontal ligament to affect equilibrium.

Consideration of eruptive forces leads to a final aspect of the dental equilibrium: the effect of forces against the teeth must be considered, not only in the anteroposterior and transverse planes of space that relate to the position of a tooth within the arch, but also vertically in relation to how much or how little a tooth erupts. T h e vertical position of any tooth, of course, is determined by the equilibrium between the forces that produce eruption and those that oppose it. Forces from mastication are the primary ones opposing eruption, but lighter, more sustained forces from soft tissues such as the tongue interposed between the teeth probably are more important, just as they are for the horizontal equilibrium.

Equilibrium effects on jaw size and shape. The jaws, particularly the mandible, can be thought of as consisting of a core of bone to which functional processes are attached. The functional processes of bones will be altered if the function is lost or changed. For example, the bone of the alveolar process exists only to support the teeth. If a tooth fails to erupt, alveolar bone never forms in the area it would have occupied, and if a tooth is extracted, the alveolus in that region resorbs until finally it completely atrophies. When one of a pair of

opposing teeth is extracted, the other usually begins to erupt again, and even as bone is resorbing in one jaw where the tooth was lost, new alveolar bone forms in the other as the erupting tooth brings bone with it. The position of the tooth, not the functional load on it, determines the shape of the alveolar ridge.

The same is true for the muscular processes: the location of the muscle attachments is more important in determining bone shape than mechanical loading or degree of activity. Growth of the muscle, however, determines the position of the attachment, and so muscle growth can produce a change in shape of the jaw, particularly at the coronoid process and angle of the mandible.

If the condylar processes of the mandible can be considered functional processes serving to articulate the mandible with the rest of the facial skeleton, as apparently they can, the intriguing possibility is raised that altering the position of the mandible might alter mandibular growth. The idea that holding the mandible forward or pressing it backward would change its growth has been accepted, rejected, and partially accepted again during the past century. Obviously, this theory has important implications for the etiology of malocclusion. For example, if a child positions his mandible forward on closure because of incisor interferences or because his tongue is large, will this stimulate the mandible to grow larger and ultimately produce a Class III malocclusion? Would allowing a young child to sleep on his stomach, so that the weight of the head rested on the chin, cause underdevelopment of the mandible and a Class II malocclusion?

The effect of force duration is not as clear for equilibrium effects on the jaws as for the teeth. It appears, however, that the same principle applies: the magnitude of force is less important than its duration. Positioning the jaw forward only when the teeth are brought into occlusion means that most of the time, when the mandible is in its rest position, there is no protrusion. We would expect no effects on a functional process from repeated intermittent force because of the short total duration, and the condylar process seems to respond in accordance with this principle. Neither experimental nor clinical evidence suggests that mandibular growth is any different because of occlusal interferences (though it should be kept in mind that tooth eruption, and thereby the final position of the teeth, can be altered).

If the mandible were protruded at all times, as might well be the case if the tongue were unusually large, the duration threshold could be surpassed, and growth effects might be observed. On clinical examination, individuals who appear to have a large tongue almost always have a well-developed mandible, but it is very difficult to establish tongue size. Only in extreme cases, as with a patient with early-onset thyroid deficiency, is it possible to be reasonably sure that an enlarged tongue contributed to excessive growth of the mandible. This is unlikely to be a major cause of mandibular prognathism.

Although it was widely believed in Edward Angle's era that pressures against the mandible from various habits, particularly sleeping on the stomach, interfered with growth and caused Class II malocclusion, little or no evidence

supports this contention. Growth of the soft tissue matrix that moves the mandible forward and creates a space between the condyle and the temporal fossa is the normal mechanism by which growth occurs. Inhibition of mandibular growth by pressure is not a feature of normal development and is much harder to achieve, if indeed it is possible at all.

From the perspective of equilibrium theory, then, we can conclude that intermittent pressures or forces have little if any effect on either the position of the teeth or the size and shape of the jaws. Density of bone in the alveolar process and throughout the basal areas of the jaws should differ as a function of masticatory forces, but shape should not. Neither masticatory forces nor soft tissue pressures during swallowing and speaking should have any major influence on tooth position.

Major equilibrium influences for the teeth should be the light but long-lasting pressures from tongue, lips, and cheeks at rest. In addition, significant equilibrium effects should be expected from the elasticity of gingival fibers and from metabolic activity within the periodontal ligament. These equilibrium influences would affect the vertical as well as horizontal position of the teeth and could have a profound effect on how much tooth eruption occurred as well as where a tooth was positioned within the dental arch. The major equilibrium influences on the jaws should be positional changes affecting the functional processes, including the condylar process.

In tile remainder of this section, functional patterns and habits that might produce malocclusion are examined as potential etiologic agents from the perspective of equilibrium theory.

4.1. Mastication

The pressures generated by chewing activity potentially could affect dentofacial development in two ways: (1) greater use of the jaws, with higher and/or more prolonged biting force, could increase the dimensions of the jaws and dental arches. Less use of the jaws might then lead to underdeveloped dental arches, and to crowded and irregular teeth; (2) decreased biting force could affect how much the teeth erupt, thereby affecting lower face height and overbite/open bite relationships. Let us now examine both possibilities in more detail.

Function and dental arch size. Equilibrium theory, as reviewed earlier, suggests that the size and shape of the muscular processes of the jaws should reflect muscle size and activity. Enlargement of the mandibular gonial angles can be seen in humans with hypertrophy of the mandibular elevator muscles, and changes in the form of the coronoid processes occur in children when temporalis muscle function is altered after injuries, so there is no doubt that the muscular processes of the jaws are affected by muscle function in humans. Equilibrium theory also suggests that heavy intermittent forces produced during mastication should have little direct effect on tooth positions, and therefore that size of the dental arches would be affected by function only if their bony base were widened. Does the extent of masticatory activity affect the width of the base of the dental arches?

It seems likely that differences between human racial groups, to some extent, reflect dietary differences and the accompanying masticatory effort. The characteristic craniofacial morphology of Eskimos, which includes broad dental arches, is best explained as an adaptation to the extreme stress they place on jaws and teeth and changes in craniofacial dimensions from early to modern human civilizations have been related to the accompanying dietary changes. A number of studies by physical anthropologists indicate that changes in dental occlusion, and an increase in malocclusion, occur along with transitions from a primitive to modern diet and lifestyle, to the point that Corrucini labels malocclusion a "disease of civilization". In the context of adaptations to changes in diet over even a few generations, it appears that dietary changes probably have played a role in the modern increase in malocclusion.

Whether masticatory effort influences the size of the dental arches and the amount of space for the teeth during the development of a single individual is not so clear. Vertical jaw relationships clearly are affected by muscular activity (the effect on tooth eruption is discussed below); the effect on arch width is not so clear.

Animal experiments with soft versus hard diets show that morphologic changes can occur within a single generation when diet consistency is altered. When a pig, for instance, is raised on a soft rather than a normal diet, there are changes in jaw morphology, the orientation of the jaws to the rest of the facial skeleton, and in dental arch dimensions. Whether similar effects exist in humans remains unclear. If dietary consistency affects dental arch size and the amount of space for the teeth as an individual develops, it must do so early in life, because dental arch dimensions are established early. The intercanine distance, the key dimension for the alignment or crowding of incisors that is the major component of non-skeletal malocclusion, increases only modesty after the primary canines erupt at age 2 and tends to decrease after the permanent canines erupt. Is it possible that a child's masticatory effort plays a major role in determining dental arch dimensions? That seems unlikely. Genetic drift toward smaller jaw sizes, accelerated by the dietary changes that have occurred, is a more plausible explanation, but the precise relationship remains unknown.

Biting force and eruption. Patients who have excessive overbite or anterior open bite usually have posterior teeth that are infra- or supra-erupted, respectively. It seems reasonable that how much the teeth erupt should be a function of how much force is placed against them during function. Is it possible that differences in muscle strength, and therefore in biting force, are involved in the etiology of short- and long-face problems?

It was noted some years ago that short-face individuals have higher, and long-face persons lower, maximum biting forces than those with normal vertical dimensions. The difference between long-face and normal-face patients is highly significant statistically for occlusal tooth contacts during swallow, simulated chewing, and maximum biting. Such an association between facial morphology and occlusal force does not prove a cause-and-effect relationship. In the rare muscle

weakness syndromes discussed earlier, there is a downward and backward rotation of the mandible associated with excessive eruption of the posterior teeth, but this is almost a caricature of the more usual long-face condition, not just an extension of it. If there were evidence of decreased occlusal forces in children who were showing the long-face pattern of growth, a possible causative relationship would be strengthened.

It is possible to identify a long-face pattern of growth in pre-pubescent children. Measurement of occlusal forces in this group produces a surprising result: there are no differences between children with long faces and normal faces, nor between either group of children and long-face adults. All three groups have forces far below those of normal adults. Therefore it appears that the differences in occlusal force arise at puberty, when the normal group gains masticatory muscle strength and the longface group does not. Because the long-face growth pattern can be identified before the differences in occlusal force appear, it seems more likely that the different biting force is an effect rather than a cause of the malocclusion.

These findings suggest that the force exerted by the masticatory muscles is not a major environmental factor in controlling tooth eruption and not an etiologic factor for most patients with deep bite or open bite. The effect of muscular dystrophy and related syndromes shows that there can be definite effects on growth if the musculature is abnormal, but in the absence of syndromes of this type, there is no reason to believe that how a patient bites is a major determinant of either dental arch size or vertical dimensions.

4.2. Breathing.

Respiratory needs are the primary determinant of the posture of the jaws and tongue (and of the head itself, to a lesser extent). Therefore it seems entirely reasonable that an altered respiratory pattern, such as breathing through the mouth rather than the nose, could change the posture of the head, jaw, and tongue. This in turn could alter the equilibrium of pressures on the jaws and teeth and affect both jaw growth and tooth position. In order to breathe through the mouth, it is necessary to lower the mandible and tongue, and extend (tip back) the head. If these postural changes were maintained, face height would increase, and posterior teeth would super-erupt; unless there was unusual vertical growth of the ramus, the mandible would rotate down and back, opening the bite anteriorly and increasing overjet; and increased pressure from the stretched cheeks might cause a narrower maxillary dental arch.

Exactly this type of malocclusion often is associated with mouth breathing (note its similarity to the pattern also blamed on sucking habits and tongue thrust swallow). The association has been noted for many years: the descriptive term *adenoid face* has appeared in the English literature for at least a century, probably longer. Unfortunately, the relationship between mouth breathing, altered posture, and the development of malocclusion is not so clear-cut as the theoretical outcome of shifting to oral respiration might appear at first glance. Recent experimental studies have only partially clarified the situation.

In analyzing this, it is important to understand first that although humans are primarily nasal breathers, everyone breathes partially through the mouth under certain physiologic conditions, the most prominent being an increased need for air during exercise. For the average individual, there is a transition to partial oral breathing when ventilator exchange rates above 40 to 45 L/min are reached. At maximum effort, 80 or more L/min of air are needed, about half of which is obtained through the mouth. At rest, minimum airflow is 20 to 25L/min, but heavy mental concentration or even normal conversation lead to increased airflow and a transition to partial mouth breathing.

During resting conditions, greater effort is required to breathe through the nose than through the mouth—the tortuous nasal passages introduce an element of resistance to airflow as they perform their function of warming and humidifying the inspired air. The increased work for nasal respiration is physiologically acceptable up to a point, and indeed respiration is most efficient with modest resistance present in the system. If the nose is partially obstructed, the work associated with nasal breathing increases, and at a certain level of resistance to nasal airflow, the individual switches to partial mouth breathing. This crossover point varies among individuals, but is usually reached at resistance levels of about 3.5 to 4 cm H_2O/L/min. The swelling of the nasal mucosa accompanying a common cold occasionally converts all of us to mouth breathing at rest by this mechanism.

Chronic respiratory obstruction can be produced by prolonged inflammation of the nasal mucosa associated with allergies or chronic infection. It can also be produced by mechanical obstruction anywhere within the nasorespiratory system, from the nares to the posterior nasal choanae. Under normal conditions, the size of the nostril is the limiting factor in nasal airflow. The pharyngeal tonsils or adenoids normally are large in children, and partial obstruction from this source may contribute to mouth breathing in children. Individuals who have had chronic nasal obstruction may continue to breathe partially through the mouth even after the obstruction has been relieved. In this sense, mouth breathing can sometimes be considered a habit.

If respiration had an effect on the jaws and teeth, it should do so by causing a change in posture that secondarily altered long-duration pressures from the soft tissues. Experiments with human subjects have shown that a change in posture does accompany nasal obstruction. For instance, when the nose is completely blocked, usually there is an immediate change of about 5 degrees in the craniovertebral angle. The jaws move apart, as much by elevation of the maxilla because the head tips back, as by depression of the mandible. When the nasal obstruction is removed, the original posture immediately returns. This physiologic response occurs to the same extent, however, in individuals who already have some nasal obstruction, which indicates that it may not totally result from respiratory demands.

Experiments with growing monkeys show that totally obstructing the nostrils for a prolonged period in this species leads to the development of malocclusion but not of the type commonly associated with mouth breathing in humans. Instead, the monkeys tend to develop some degree of mandibular prognathism, although their response shows considerable variety. Placing a block in the roof of a monkey's mouth, which forces a downward position of the tongue and mandible, also produces a variety of malocclusions. It seems clear that altered posture is the mechanism by which growth changes were produced. The variety of responses in the monkeys suggests that the type of malocclusion is determined by the individual animal's pattern of adaptation.

In evaluating these experiments, it must be kept in mind that mouth breathing of any extent is completely unnatural for monkeys, who will the if the nasal passages are obstructed abruptly. To carry out the experiments, it was necessary to gradually obstruct their noses, giving the animals a chance to learn how to survive as mouth breathers. Total nasal obstruction is also extremely rare in humans.

There are only a few well-documented cases of facial growth in children with long-term total nasal obstruction, but it appears that under these circumstances the growth pattern is altered in the way one would predict. Because total nasal obstruction in humans is so rare, the important clinical question is whether partial nasal obstruction, of the type that occurs occasionally for a short time in everyone and chronically in some children, can lead to malocclusion; or more precisely, how close to total obstruction does partial obstruction have to come before it is clinically significant?

The question is difficult to answer, primarily because it is difficult to know what the pattern of respiration really is at any given time in humans. Observers tend to equate lip separation at rest with mouth breathing, but this is simply not correct. It is perfectly possible for an individual to breathe through the nose while the lips are apart. To do this, it is only necessary to seal off the mouth by placing the tongue against the palate. Since some lip separation at rest (lip incompetence) is normal in children, many children who appear to be mouth breathers may not be.

Simple clinical tests for mouth breathing can also be misleading. The highly vascular nasal mucosa undergoes cycles of engorgement with blood and shrinkage. The cycles alternate between the two nostrils: when one is clear, the other is usually somewhat obstructed. For this reason, clinical tests to determine whether the patient can breathe freely through both nostrils nearly always show that one is at least partially blocked. One partially obstructed nostril should not be interpreted as a problem with normal nasal breathing.

The only reliable way to quantify the extent of mouth breathing is to establish how much of the total airflow goes through the nose and how much through the mouth, which requires special instrumentation to simultaneously measure nasal and oral airflow. This allows the percentage of nasal or oral respiration (nasal/oral ratio) to be calculated, for the length of time the subject can tolerate being con-

tinuously monitored. It seems obvious that a certain percentage of oral respiration, maintained for a certain percentage of the time, should be the definition of significant mouth breathing, but despite years of effort such a definition has not been produced.

The best current experimental data for the relationship between malocclusion and mouth breathing are derived from studies of the nasal/oral ratio in normal versus longface children. The relationship is not nearly as clear-cut as theory might predict. A minority of the long-face children had less than 40 % nasal breathing, while none of the normal children had such low nasal percentages. When adult long-face patients are examined, the findings are similar: the number with evidence of nasal obstruction is increased in comparison to a normal population, but the majority are not mouth breathers in the sense of predominantly oral respiration.

It seems reasonable to presume that children who require adenoidectomy and/or tonsillectomy for medical purposes, or those diagnosed as having chronic nasal allergies, would have some degree of nasal obstruction (although it must be kept in mind that this has not been documented). Allergic children tend to have increased anterior face height and the increased overjet/decreased overbite that accompanies it. Studies of Swedish children who underwent adenoidectomy showed that on the average, children in the adenoidectomy group had a significantly longer anterior face height than control children. They also had a tendency toward maxillary constriction and more upright incisors. Furthermore, when children in the adenoidectomy group were followed after their treatment, they tended to return toward the mean of the control group, though the differences persisted. Similar differences from normal control groups were seen in other groups requiring adenoidectomy and/or tonsillectomy.

Although the differences between normal children and those in the allergy or adenoidectomy groups were statistically significant and undoubtedly real, they were not large. Face height on the average was about 3 mm greater in the adenoidectomy group. Earlier English workers indicated that the percentage of children with various malocclusions was about the same in a group being seen in an ear, nose, and throat clinic as in controls without respiratory problems.

It appears therefore that research to this point on respiration has established two opposing principles, leaving a large gray area between them: (1) total nasal obstruction is highly likely to alter the pattern of growth and lead to malocclusion in experimental animals and humans, and individuals with a high percentage of oral respiration are overrepresented in the long-face population; but (2) the majority of individuals with the long-face pattern of deformity have no evidence of nasal obstruction and must therefore have some other etiologic factor as the principal cause. Perhaps the alterations in posture associated with partial nasal obstruction and moderate increases in the percentage of oral respiration are not great enough by themselves to create a severe malocclusion. Mouth breathing, in short, may contribute to the development of orthodontic problems but is difficult to indict as a frequent etiologic agent.

Naso-respiratory function and its relation to craniofacial growth is of great interest today, not only as an example of the basic biologic relationship of form and function, but also is of great concern for orthodontists, pedodontist, pediatricians, otorhinolaryngologists, allergists and speech pathologists for varying reasons.

Infants are obligatory nasal breathers. Everyone breathes partially through the mouth under physiological conditions, the most important being the need for increased air, i.e. physical exertion during strenuous activity and exercise.

During normal mechanism of respiration, the efforts to breathe through the nose is greater. The mouth does not normally participate in respiration. The tortuous nasal passages introduce an element of resistance to airflow as they perform their function of warming and humidifying the inspired air. This modest resistance present in the system makes respiration more efficient.

Definitions:

Chopra R.B. (1951) defined mouth breathing as habitual respiration through the mouth instead of the nose.

Chacker F.M. (1961) defined mouth breathing as a prolonged or continued exposure of the tissues of anterior areas of mouth to the drying effects of inspired air.

Sassouni (1971) defined it as, habitual respiration through the mouth instead of the nose.

Merle (1980) used the term ore-nasal breathing instead of mouth breathing.

Sim and Finn classified mouth breathing as:

1. Obstructive
2. Habitual
3. Anatomic

1. Obstructive .Children with an increased resistance to or a complete obstruction of the normal flow of air through the nasal passages. Seen in ectomorphous individuals with long narrow faces and nasopharyngeal passages.

2. Habitual. Child who continually breathes through the mouth by force of habit, although the obstruction has been removed.

3. Anatomical . Short upper lip does not permit closure without undue effort.

a. Total blockage: Nasal passages are completely blocked. .

b. Partial blockage.

Function of nasal breathing. An important function of the nose is to prepare and modify inspired air to a more physiologic state before it enters the lungs.

The nasal passages are so designed that inspired air is cleaned, warmed and humidified prior to its entry into the lungs, since the quality of air required by the lungs may influence the health and function of the lungs.

Etiology of mouth breathing. Mouth breathing usually results when nasal passage is obstructed or is inadequate for respiratory exchange.

Causes of mouth breathing are:

Nasal Obstruction

1. Enlarged turbinates Infection and increase blood supply produces hypertrophy of the mucosa causing obstruction of the nasal passage unilaterally/bila-

teral1y. This may be due to allergies, chronic infections of mucous membrane, atrophic rhinitis, hot and dry climatic conditions, and polluted air.

2. Hypertrophy of pharyngeal lymphoid tissue (adenoids). Repeated infection resulting in the overgrowth of lymphoid masses blocks the posterior nares, rendering mouth breathing necessary. Enlarged tonsils will cause the soft palate to rest on their upper pole instead of the dorsum of the tongue and further displace the dorsum downward and forward contributing to an open mouth posture, possible nocturnal snoring and sleep apnea.

3. Intranasal defects:

• Deviated nasal septum
• Subluxation of septum
• Thickness of septum
• Bony spurs
• Polyps

4. Allergic rhinitis Continuous infections and toxins of the bacteria may sensitize the tissue to develop allergic reactions.

Facial type. Seen more in ectomorphs, long-faced (*Fig. 4.1*, *4.2*), tall, slender persons with long narrow pharyngeal space.

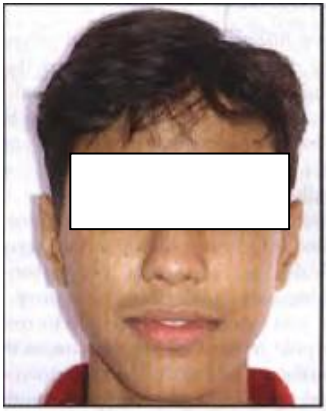

Fig. 4.1. Long face patient

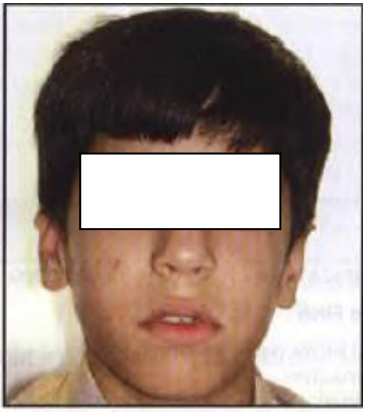

Fig. 4.2. 'Adenoid Face' appearance

Respiratory pattern. Respiratory needs are the primary determinant of the posture of the jaws and tongue. Thus, an altered respiratory pattern, e.g. in mouth breathing could alter the posture of the head, jaw and tongue. This, in turn, could alter the equilibrium of pressure on the jaws and teeth thus, affect, jaw growth and tooth position. To breathe through the mouth, it is necessary to extend lower the mandible and tongue and the head. If these postural changes were maintained:

• Face height would increase.
• Posterior teeth would supra-erupt
• Mandible would rotate down and back

•Open-bite develops anteriorly, increased overjet

• Narrower maxillary arch-increased pressure from stretched cheeks

• ′Adenoid Face' appearance *(Fig. 4.2).*

Effects of mouth breathing:

1. Associated structures and nose When air is inspired through the mouth, it is not cleaned, warmed and moistened, secretion of mucus is stopped gradually. The irritants accumulate resulting in local inflammation discomfort and pain.

2. General health and growth. The child is usually restless and is affected by repeated cold, cough, glandular fever etc., loss of general body resistance to other diseases.

3. Growth and development of the face and jaws

On face:

1. Lips slack and stay open

2. Short upper lip

3. Molding action of upper lip on incisors is lost thereby resulting in proclination and spacing.

4. Lower lip: heavy and everted.

5. Tongue is suspended between upper and lower arches resulting in constriction of buccal segment (V shape arch).

***Effect on occlusion of teeth** (Fig. 4.3).*

• Proclination of anteriors

• Distal relation of mandible to maxilla

• Lower anteriors elongate and touch the palatal tissues.

• Upon gingival tissues constant wetting and drying of the gingiva causes irritation, saliva about the exposed gingiva tends to accumulate debris resulting in an increase in bacterial population.

Hypertrophic mouth breathing gingivitis (Fig. 4.3). Gingival hypertrophy is seen as mouth breathing line/ gum ridge at the junction of edematous and normal tissues. This line marks the limit of the area exposed to air.

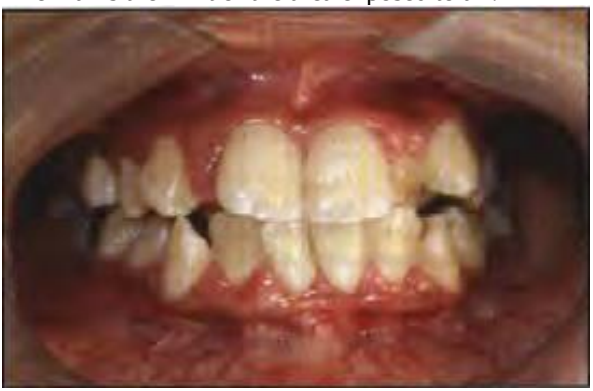

Fig. 4.3. Effect of mouth breathing on gums and occlusion

Non-hypertrophic mouth breathing gingivitis. Non-hypertrophic mouth breathing gingivitis is a marginal gingivitis without edema which develops on the palatal tissues of upper anterior region in mouth breathers even in the presence of good oral hygiene.

***Diagnosis of mouth breathing*.** Diagnose the habit by looking for the following symptoms.

Subjective Symptoms

1. History. A good history should be recorded from patients and parents also, as children may deny the habit.

2. Clues about nasal stiffness, nasal discharge, sore throat, repeated attacks of cold.

3. Posterior nasal defects.

Objective Symptoms

1. Hoarseness of voice

2. Mouth breathing malocclusion (clinical features)

3. Restlessness at night, feeling thirsty.

4. Mouth breathing gingivitis.

5. Association with other habits.

Cephalometries. Cephalometric examination helps in establishing the amount of nasopharyngeal space, size of adenoids, and also helps in diagnosing the long face associated with mouth breathing.

Rhinomanometry. It is the study of nasal air flow characteristics using devices consisting of flow meters and pressure gauges. These devices help in estimation of air flow through the nasal passage and nasal resistance.

*Methods of examination***:**

1. Study the patient's breathing unobserved: Nasal breather's lips touch lightly during relaxed breathing whereas mouth breathers keep the lips parted.

2. Ask the patient to take a deep breath: Most mouth breathers respond to this request by inspiring through the mouth. The nose, does not change the size or shape of external nares occasionally contracts the nasal orifices while inspiring.

Other tests:

a. Mirror test. A double sided mirror is held between the nose and mouth. Fogging on the nasal side of the mirror indicates nasal breathing while fogging on oral side – mouth breathing.

b. Cotton test/Massler's butterfly test. Butterfly shaped cotton strands are placed over the upper lip below nostrils. If the cotton flutters down it is a sign of nasal breathing. This test can be used to determine unilateral nasal blockage.

c. Water test. The patient is asked to fill the mouth with water and retain it for a period of time. Mouth breathers find this task difficult.

Management.

1. ENT referral for management of nasopharyngeal obstruction.

2. Prevention and interception. It usually ceases at puberty or after it due to increase in size of passage during period of rapid growth. Mouth breathing can be intercepted by use of an oral screen.

3. Myofunctional therapy

• During day time - hold pencil between the lips.

• During night time – tape the lips together with surgical tape in habitual mouth breathing.

• Hold a sheet of paper between the lips.

• Piece of card 1 × 1 1/2 held between the lips.

• Patients with short hypotonic upper lip stretch the upper lip to maintain lip seal or stretch in downward direction towards the chin.

• Button pull exercise. A button of 1½ diameter is taken and a thread is passed through the button hold. The patient is asked to place the button behind the lip and pull the thread, while restricting it from being pulled out by using lip pressure.

• Tug of war exercise. This involves 2 buttons, with one placed behind the lips while the other button is held by another person to pull the thread.

• Blow under the upper lip and hold under tension to a slow count of 4 repeat 25 times a day.

• Draw upper lip over the upper incisors and hold under tension for a count of 10.

Oral screen vestibular. Screen/oral shield (*Fig. 4.4*).

Fig. 4.4. Vestibular screen. Small air holes may be drilled to aid breathing

The oral screen is a device fitting in the vestibule which shuts off the ingress of air through the mouth and directs contraction of lips against any anterior teeth in labioversion. It is used to retrain the lips.

1. Corrects simple labioversion of the maxillary anterior teeth.

2. Habit correcting appliance - as it helps retrain and strengthen lip action.

It should not be used if the child has nasorespiratory distress or nasal obstruction. It is of no use for correction of Class II malocclusion.

Breathing holes can be bored initially. This allows passage of some amount of air into the mouth. As the child learns to breathe through nose, fill some holes with acrylic so that less and less air enters through the mouth and finally close all the holes.

Rapid maxillary expansion. Patients with narrow, constricted maxillary arches benefit from RME procedures aimed at widening of the arch. It is found to increase nasal air flow and decrease nasal air resistance. Following RME an increase in intranasal space occurs due to outer walls of nasal cavity moving apart. This increase in nasal cavity width is maximum in the anterior and inferior region and gradually decreases towards superior and posterior aspect respiratively.

4.3. Swallowing.

Humans show 2 types of swallow patterns:

1. Infantile and neonates swallow *(Fig. 4.5. b).*
2. Mature/adult swallow *(Fig. 4.5. c).*

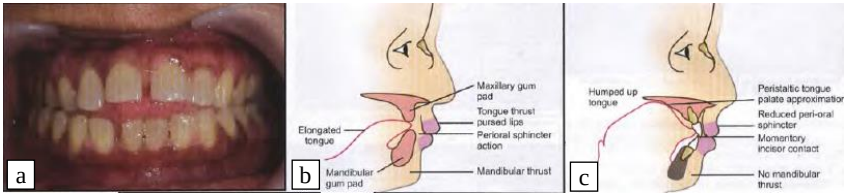

Fig. 4.5. a. – Abnormal placement of the tongue/tongue thrust swallow; bю – Infantile swallow; c. – Mature (somatic) swallow

Infantile swallow is characterized by:

• Active contractions of the lip muscles.

• Tongue is placed between the gum pads and tongue tip is brought forward into contact with the lower lip *(Fig. 4.5. a).*

• Little posterior tongue activity / pharyngeal muscle activity.

• Tongue-to-lower lip posture adopted by infants at rest.

• Contraction of lips and facial muscles helps to stabilize the mandible.

• Vigorous mandibular thrust.

Physiologic transition of swallow begins daring the 1st year of life and continues for several years. Mature swallow is seen usually by 2.5–3 years. Maturation of swallow pattern occurs with the addition of semisolid and solid food to the diet. Increasing activation of the elevator muscles of mandible is seen. When sucking activity stops, a continued transition of swallow leads to acquisition of adult pattern of swallow.

This swallow is characterized by:

• Cessation of lip activity, i.e, lips relaxed.

• Placement of tongue tip against the palate and behind upper incisor

• Posterior teeth into occlusion during swallow.

• Downward and forward mandibular growth increases intraoral volume and vertical growth of the alveolar process changes tongue posture.

• Mandible stabilized by contraction of muscles of mastication.

Retained infantile swallow. Retained infantile swallow is defined as predominant persistence of the infantile swallowing reflex after the eruption of permanent teeth.

Clinical features:

• Strong contractions of lips and facial musculature especially buccinator.

• Massive grimace.

• Anterior and lateral thrusting *(Fig. 4.6).*

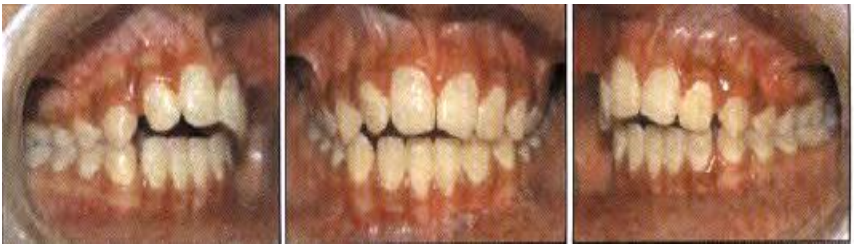

Fig. 4.6. Simple tongue thrust

Inexpressive face due to use of facial muscles for swallowing.

• Difficulty in mastication since they normally occlude on only one molar in each quadrant.

• Low gag threshold

• Poor prognosis

Usually associated with skeletal craniofacial developmental syndromes and neural deficits.

4.4. Specking.

The soft tissues show remarkable adaptation to the changes that occur during the transition between the primary and mixed dentitions, and when the incisors have been lost owing to trauma or disease. In the main, speech is little affected by malocclusion, and correction of an occlusal anomaly has little effect upon abnormal speech. However, if a patient cannot attain contact between the incisors intraorally, this may contribute to the production of a lisp (interdental sigmatism).

The ability to communicate effectively is vital to a person's functioning in society. Speech and language acquisition is a developmental process occurring most dramatically in the first years of life but one that proceeds throughout a person's life-time. Difficulties may be encountered at any point during the language acquisition process. Children may experience problems acquiring the sounds of the language, learning how to combine words meaningfully or comprehending others' questions and instructions. Communication problems in adults may arise from loss of language functioning associated with stroke or head injury. In all these cases, a speech pathologist is the primary health-care professional responsible for the identification and treatment of individuals with communication problems. Pediatric dentists should be aware of the symptoms and problems associated with communication difficulties and should know how to refer children and their families to speech pathologists.

Communication disorders. There are five main areas to be considered when assessing a child's communication:

- Oral motor and feeding problems.
- Articulation.
- Language.
- Voice.
- Fluency.

Oral motor and feeding problems. Problems in this area constitute the earliest at which children are referred to a speech pathologist. Significant problems can result when an infant does not develop control of the oral mechanism sufficient for successful feeding. Early reflex development typically facilitates feeding behavior, but neuromotor factors, prematurity, cleft lip and palate, long-term non-oral feeding and other reasons may interfere with a child's development of the movement patterns essential for sucking, swallowing and feeding. Because these patterns form the scaffolding of movement for early speech sound development, it is not uncommon for children with a history of feeding difficulties to have subsequent difficulties in producing sounds for speech.

Referral:

- Sucking, swallowing or chewing difficulties.
- Coughing or choking with feeds.
- Moist vocal quality.
- Poor cough or gag reflex.
- Persistent drooling (not coincident with teething).
- Recurrent chest infections.
- Presence of a craniofacial malformation.
- Parental report of feeding difficulty or refusal.

Articulation. Articulation refers to the production of speech sounds by modification of the breath stream by using the various valves along the vocal tract: lips, tongue, teeth and palate. Problems in these areas can vary from a fairly mild distortion of sounds, such as a lisp, where the child's speech is still easy to understand, through to a more severe speech production problem in which all speech attempts are unintelligible or the child makes very few speech attempts. Errors are classified in the following ways:

- *Speech sound omissions*

'cu' for 'cup', 'te-y' for 'teddy'.

- *Substitutions of sounds*

'wed' for 'red', 'tun' for 'sun'.

- *Distortions*

lateral lisp–'s' that sounds slushy.

Children learn to produce sounds in a developmental sequence, with adult-like sound systems expected by 8 years of age. For example, it is acceptable for a 2-year-old to mispronounce an 's' sound but it would be considered a problem if a similar error were made by a 5-year-old child.

Language. In contrast to the fairly straightforward examples listed above to illustrate speech sound learning, language development is much more complex. Skills emerge on two parallel levels.

Receptive language This is the ability to understand language.

Expressive language This refers to the ability to initiate communication.

A child with a language disorder may present with difficulties in both comprehension and expression of language, or in only one area of language learning.

Language learning proceeds in a predictable order but there is more variability in the emergence of these skills than the acquisition of speech sounds. Vocabulary grows, as does a child's ability to progressively understand more complex language. Words are combined into phrases and eventually sentences, and comprehension becomes more adult-like over time. Eventually, language that is heard and said becomes the language of literacy, reading and writing. School success is highly correlated with language learning, especially in the early years.

Children may experience language learning problems at any stage of the acquisition process. There may be:

- Difficulties interpreting the meaning of words and gestures.
- Excessive delay in the production of first words and phrases.
- Lack of understanding of questions and instructions.
- Inability to produce sentences that are grammatically correct.
- Inability to participate in conversations.

A delay at any single stage may not necessarily constitute a long-standing problem, although it should be investigated further. Problems with language acquisition are the most subtle indicators of difficulties with childhood development, and therefore should never be ignored.

Voice. The voice is produced when the vocal cords in the larynx are vibrated. Changes in air flow and the shape of the vocal folds can affect the loudness, pitch and quality of the voice. Once the voice is produced, its tone (resonance) and quality are modified by the throat, oral and nasal cavities. A child with a voice problem may present with:

Abnormal voice quality Harsh, breathy or hoarse in the absence of upper respiratory tract infection.

Abnormal resonance or tone Hypernasal (excessive nasal resonance). Hyponasal (lack of nasal resonance, due to some type of nasopharyngeal obstruction).

Inappropriate loudness levels Voice too soft to be heard or so loud that it distracts from the message of the speaker.

Problems with pitch Pitch too high or low for age or sex. Causes of voice problems include:

- Vocal abuse and misuse (in some cases producing vocal nodules).
- Neurological problems.
- Vocal polyps.
- Muscular pathology.
- Vocal cord paralysis.

- Irritants caused by exposure to smoking or aerosol sprays.
- Physical conditions including cleft palate, laryngectomy and hearing loss.

Fluency. Fluency refers to the smooth flow of speech. Where there are interruptions in the flow of speech, stuttering occurs. Many children experience brief periods of stuttering as they learn to speak in longer sentences. The phenomenon is not a disordered pattern that will persist, and is best resolved by reacting to the message the child is attempting to convey. When stuttering outlasts this normal period and when it is becoming stressful for the child, referral is indicated.

A child or an adult with a stutter experiences involuntary repetition of words, prolongations of sounds in words and blocks where no sound is produced at all. Some speakers with a stutter will use words like 'urn' to help them initiate speaking. Often secondary features such as a facial grimace will occur with the stutter.

Recent research suggests that the problem is not psychological in origin. The majority of people who stutter have been born with an inherited predisposition to disfluency (e.g. it is usually a familial trait). Stutterers do not have more emotional or psychological problems when compared with the general population, nor is there evidence of decreased mental aptitude. Approximately 3 % of the population stutter, with a predominance of males (3:1). The disorder usually has its onset in the early years of life with an improved prognosis when treatment is undertaken early. Hence the importance of prompt referral.

Structural anomalies and their relationship to speech. The speech pathologist is actively involved in the assessment of the structural and functional status of the orofacial, pharyngeal and laryngeal areas. These structures, as well as the nasal and respiratory areas, are often objectively assessed by video-fluoroscopic X-ray studies or by naso-endoscopy during both speech production and swallowing. These important diagnostic procedures are often critical in the diagnosis of structural and functional problems affecting feeding skills and speech production.

Dental anomalies

Severe class-III malocclusion Tongue tip elevation to the alveolar ridge may be restricted, resulting in speech sound distortions and an interdental tongue position for sounds such as 's', 'z', 't', 'd,' T and 'n'.

Severe class II malocclusion Lip closure may be poor during eating and drinking. Distortion or substitution of the lip sounds 'p', 'b' and 'm' may occur with the child compensating by placing the lower lip and upper teeth together.

Maxillary collapse This condition may occur after cleft palate surgery. Distortion of sounds requiring tongue and palatal contact–'s', 'z', 'sh', 'ch' and 'dge'– may result.

Absence of teeth Chewing difficulties may result. Lateral or forward displacement of the tongue during speech may occur, resulting in distortion of sounds.

It is important to note that speech and feeding difficulties are not always associated with these dental conditions. Each patient must be considered individually in the light of his or her ability to compensate for these conditions. In cases where problems are identified, treatment is coordinated with dental and

orthodontic management, as some individuals may not be able to improve their speech or feeding until the dental anomaly is remedied.

Palatal anomalies. The soft palate and pharyngeal walls work simultaneously to close off the nasopharynx during speech production and while swallowing. This action prevents excessive airflow into the nasal cavity during speech and prevents nasal regurgitation during the swallow. If there is a palatal abnormality, this action cannot take place efficiently and there is often escape of food and liquid into the nose. Speech is nasal and breathy, sounds are unclear and volume may be reduced.

Palatal anomalies:

- Cleft palate (with or without cleft lip).
- Submucous cleft palate, characterized by a bifid uvula, separation of the palatal muscles, which results in a midline furrow on the soft palate, and a notching of the posterior margin of the hard palate.
- Congenital palatal abnormalities: short palate, deep nasopharynx, uncoordinated or inefficient velopharyngeal movement.
- Acquired palatal abnormalities resulting from neurological damage, from surgery or from neoplasms.

Children with palatal anomalies are best referred to a specialist cleft palate clinic where they can receive multidisciplinary assessment and management.

Lingual anomalies. Abnormalities of the tongue may affect the precision, range and speed of tongue movement, resulting in speech or feeding difficulties. The most common expression of a lingual anomaly is the tongue tie.

Ankyloglossia — 'tongue tie'. This may occur with varying degrees of severity and does not always result in any functional problems. Some of the possible presenting speech problems of concern to the speech pathologist are:

- Substitutions and distortion of tongue tip sounds caused by restricted elevation of the tongue tip (t, f, 'd', 'n', 's', 'z').
- Slower than normal speech rate Reduced speech precision during shouting (shouting requires the mouth to open more widely and thereby may result in the tongue tie having a more negative effect on speech precision).
- Feeding difficulties, such as difficulty sucking in infancy and persistent 'messy' eating.

If a child's speech or feeding appears to be affected by the presence of a tongue tie, comprehensive assessment by both a speech pathologist and a dentist is indicated to determine whether surgical release is required.

Maxillofacial surgery and its relation to speech production. When orthognathic surgery is being considered, consultation with a speech pathologist should be made to determine the possible consequences of the procedure for speech production. It is important to assess the position of the frenum in the body of the tongue as well as any attachment into the gingival margin that may cause periodontal complications.

Maxillary advancement procedures. When the maxilla is advanced anteriorly, the hard and soft palatal structures are also displaced forwards, increasing the distance that the soft palate must move to achieve velopharyngeal closure. Most patients seem able to compensate for this alteration in the nasopharyngeal area, and their speech and swallowing are not affected. Some patients (e.g. those with cleft palate) are at risk of developing velopharyngeal incompetence and the consequent hypernasal and distorted speech production. Forward placement of the maxilla also means that tongue contact on the palate may be altered for some sounds. In patients with severe class-III malocclusions, tongue placement and sound production may be improved with this procedure.

Referral to a speech pathologist. When the presence of a communication or feeding problem is suspected, referral should be made as soon as possible.

Dentists should refer any child who experiences the difficulties outlined below.

Feeding and swallowing

- Has difficulty sucking, swallowing and chewing.
- Is coughing and choking during feeds.
- Is drooling excessively.

Articulation

- Is not babbling a wide variety of sounds by 8–10 month
- Is not easily understood by caregivers by 2 years.
- Is not easily understood by familiar adults by 3 years.
- Is having difficulty in producing sounds accurately by 5 years.

Language

- Is not understanding a variety of questions/instructions by 18 months.
- Is not using single words by 18 months.
- Is not combining two words by 2 years (e.g. want drink).
- Has difficulty following instructions or answering questions.
- Gives inappropriate answers or frequently ignores language spoken to them.
- Is constructing sentences that are incorrect or immature by 3–4 years (e.g. 'me go to him house').
- Cannot maintain a topic of conversation by 4 years.

Voice

- Has a hoarse voice or often loses their voice.
- Has a nasal voice.
- Often sounds as though they have a cold.
- Has a voice that seems too high or low for their gender or age.
- Continually speaks abnormally loudly or softly.
- Has a sudden onset of any of these problems.

Fluency

- Shows evidence of stuttering at any age.

Referral procedures.

It will be necessary to locate the most appropriate service to meet your patients' needs. Most often, access to a speech pathologist through a local

community health centre or hospital will be all that is required. Some patients may require a more specialized service such as a developmental disability service or cleft palate clinic. Some patients may prefer the services of a private practitioner.

Referral is most efficient when the dentist provides a written referral outlining the areas of concern. Following an assessment, a treatment plan will be devised according to the individual needs of the patient. Treatment can be provided in individual or group sessions, and it may extend over a period of time depending on the nature and severity of the condition. Most speech, language and feeding problems are best treated with parental participation and a programme to facilitate carryover at home. Occasionally, it is useful to include a school component to fully integrate speech and language services into the child's daily life experiences.

Chapter 5. Different types of malloclusion. Orthodontic dispensary system

5.1. Different types of malloclusion

There exist a lot of anomalies and deformations with similar clinical presentation. In this connection there have been offered numerous different classifications which allow systematizing various types of dentognathic anomalies. Anomalies systematization enables to choose a correct approach to their understanding, study etiopathogenetic factors of their origination, put a diagnosis and plan treatment. All the classifications of dentognathic anomalies are mainly built on the registration of morphologic deviations, functional disorders, etiologic factors or their combination.

The most wide-spread are the classifications built on the basis of morphologic changes. They are grounded on the immovable junction of the facial skeleton, excluding the lower jaw, with other cranial bones. Thus, according to scientists, the facial skeleton does not undergo the harmful influence of internal and external factors and is not exposed to such changes as the dentognathic apparatus is exposed to.

The first morphologic classification, based on the principle of dental arches correlation on the whole, was offered by E. Angle in 1889. The classification is grounded on the mesiodistal correlation of the 1^{st} permanent molars of both jaws, which are defined by the author with the term "occlusion key". Angle considered that the localization of the, upper 6^{th} tooth always corresponds to the localization of the crista zygomatica thanks to its eruption in this place only. The author named the 6^{th} tooth "punctum fixum" (the fixed point). The permanent localization of the 6^{th} tooth, according to the scientist, is determined, first of all, by the immovable junction of the upper jaw with the cranial base, and secondly, by the fact that it always comes out behind the 2^{nd} temporary molar. Therefore all atypical correlations of permanent molars arise only at the expense of the irregular position of the lower jaw.

Angle divided occlusion anomalies into three classes.

- The 1^{st} class *(Fig. 5.1. a)* – "occlusion key" is not violated: the mesial buccal tubercle of the 1^{st} upper permanent molar is located in the intertubercular sulcus of the lower 1^{st} permanent molar. Thus, the pathology is only localized in

front of the Ist molars and concerns either teeth arrangement or alveolar process and jaws bodies development.

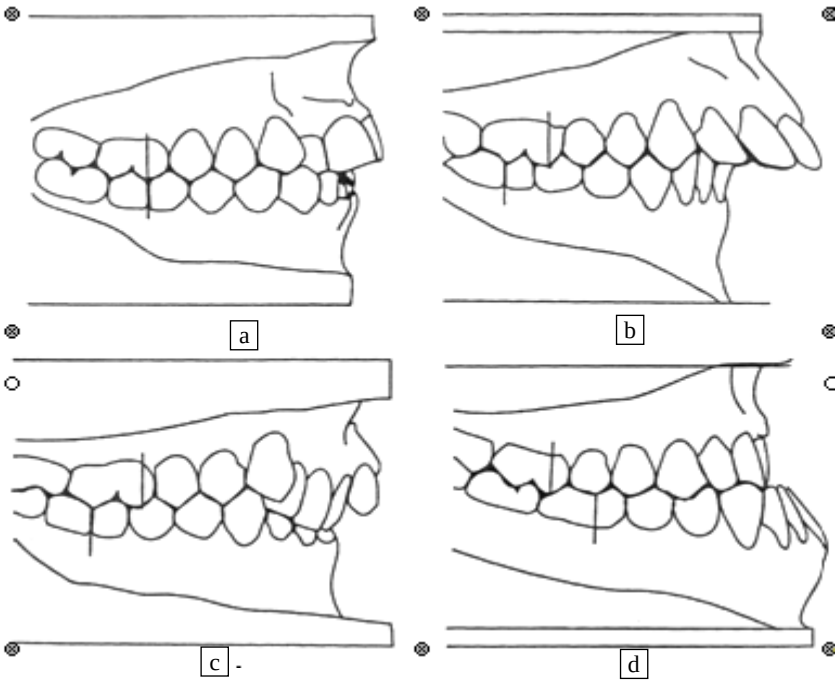

Fig. 5.1. Angle's classification: a – The Ist class;
b – Class II division I; c – Class II division II; d – The 3rd class

- The ***2nd*** class is characterised by the distal localization of the lower Ist permanent molar. At such correlation the mesial buccal tubercle of the upper Ist permanent molar is in front of the intertubercular sulcus of the lower Ist permanent molar. Either the contact is defined by similar tubercles or the mesial-buccal tubercle of the upper 1st permanent molar is located between the tubercle of the 2nd premolar and the mesial buccal tubercle of the lower 1st permanent molar, which depends on the complexity of the deformity. The changes of teeth correlation concern the whole dental arch. The 2nd class may have two subclasses of the anomaly:
- Class II division I (*Fig. 5.1. b*) — the distal location of the lower jaw, at which the upper frontal teeth are inclined forward and are fanlike allocated, with diastems and diaereses.
- Class II division II (*Fig. 5.1. c*) — the upper frontal teeth are inclined in the oral direction, deeply covering the lower ones. In both subclasses the distal correlation in lateral areas may be uni- and bilateral.

- The 3rd class (*Fig. 5.1. d*) is characterized by the mesial position of the lower 1st permanent molar relative to the similar upper tooth. At such correlation the mesial buccal tubercle of the upper 1st permanent molar is behind the intertubercular sulcus of the lower 1st permanent molar. The lower frontal teeth cover the upper ones.

Angle's classification was very popular at the end of the 19lh century as it somehow ordered the diagnostics of dentognathic anomalies and deformations. This was the first accessible by its simplicity, the only generally accepted occlusion anomalies classification in the world, which is eagerly used by specialists also nowadays.

Occlusions characterised by the anomalous position of individual teeth, deformed dental arches and their anomalous interrelation (a shift in the sagittal, vertical or transversal direction) are named anomalous or pathologic. There are distinguished such types of main anomalous occlusions:

in the sagittal direction

- *prognathic (distal occlusion); the 2nd class*
- *progenic (mesial occlusion); the 3d class*

in the vertical direction

- *open bite;*
- *deep bite*;

in the transversal direction

- *cross bite;*

Each type can have a number of variants, resulting from a characteristic position of the Ist molars (neutral, distal or mesial), a peculiarity of alveolar crests development and jaws position (dentoalveolar and gnathic varieties), basic anomaly signs combined with the signs of other occlusion types, presence of dental arches deformations (narrowing, compression or dilation, lengthening or shortening), incomplete or excessive teeth quantity.

Pathological types of occlusion in the sagittal direction prognathic (distal occlusion); Class II Malocclusion.

Extraoral Class II Division I features (Fig. 5.2. a. b):

- In frontal view, face is usually oval (mesocephalic to dolichocephalic).
- In profile view, exhibits a convex profile.
- Posterlorly divergent face.
- Incompetent and stretched upper lip due to procllned incisors.
- Lower lip is invariably everted and placed behind the upper incisors exhibiting a deep mento-labial sulcus.
- There is lack of lip seals.

lntraoral Class II Division I characteristics (Fig. 5.3. c. d. e; Fig. 5.4).

- Class II molar relationship indicating distal relationship of mandible to maxilla (*Fig. 5.4*).
- Upper incisors are proclined increasing the overjet *(Fig. 5.4).*
- There is a deep bite which may be traumatic in nature *(Fig. 5.3. c).*

- An exaggerated curve of Spee.
- Upper arch is usually narrow, 'V' shaped *(Fig. 5.3. d)*.
- The palatal vault is usually deep but may be average.

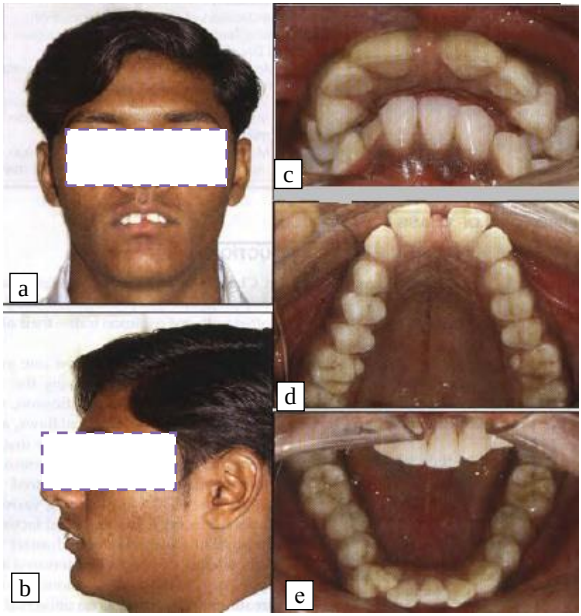

Fig. 5.3. Angle's Class II Division I
a–b – profile and close up of the lowerhalf of the face, highlighting the averted lower lip, convex profile and the retropositioned mandible,
c – intraoral view highlighting the increased overjet and trauma caused due to the deep bite,
d–e – occlusal views, note the 'V' shaped maxillary arch.

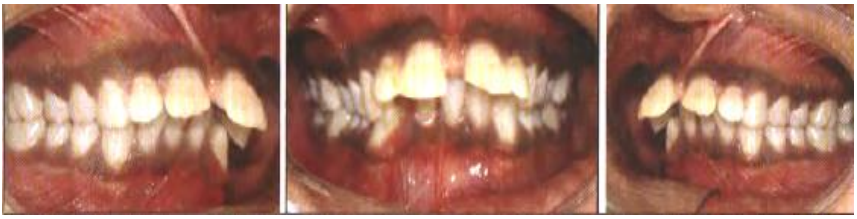

Fig. 5.4. Angle's Class II Division I

Extraoral Class II Division II features (Fig. 5.6. a. b):

- Squarish face (brachycephalic).
- Usually straight to mildly convex profile because of less skeletal discrepancy and the retroclined incisors.
- Usually straight face.

- Upper lip is invariably short and positioned high with respect to upper anteriors.
- Lower lip is thick flabby covering the upper incisors and exhibiting a shallow sulcus.
- Adequate lip seal is present. lntraoral Class II Division II characteristics *(Fig. 5.5; Fig. 5.6. c. d)*:

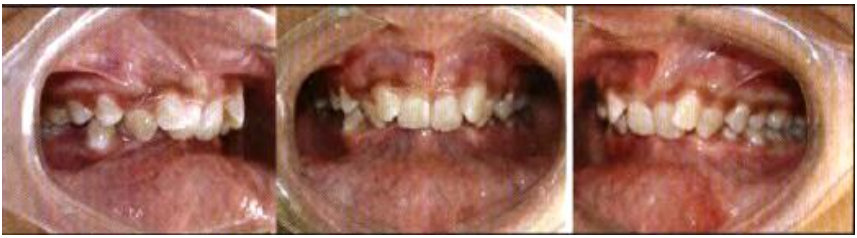

Fig. 5.5. Angle's Class II Division II

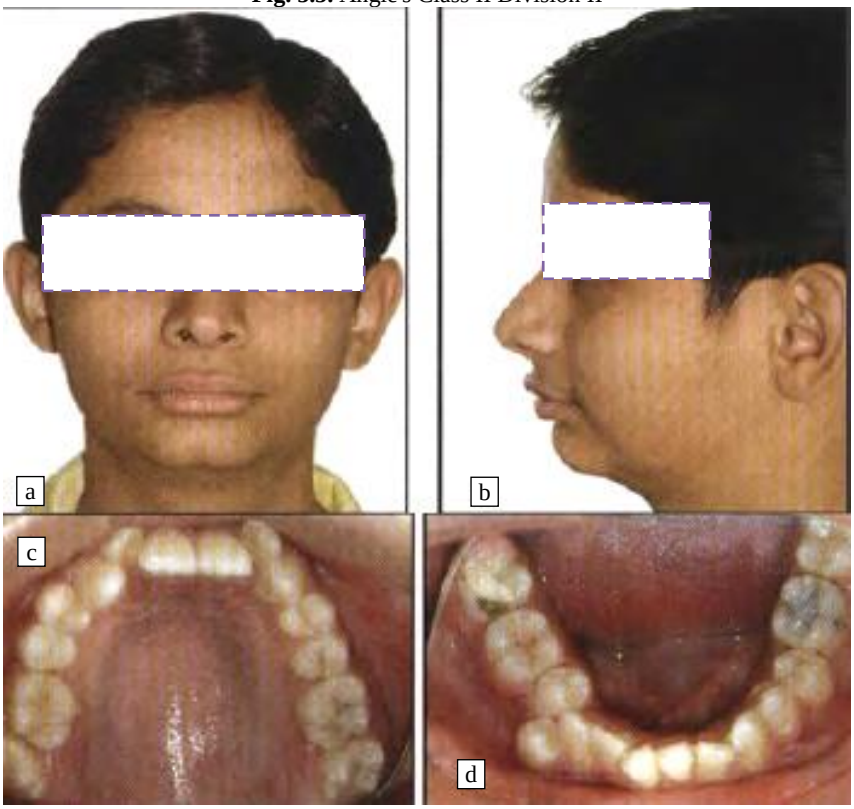

Fig. 5.6. Angle's Class II Division II

- Class II molar relationship indicating distal relationship of mandible to maxilla.
- Upper incisors retroclined exhibiting a decreased overjet and an increased overbite.
- Deep bite usually traumatic.
- An exaggerated curve of Spee.
- Other characteristics are rarely seen but may be present occasionally.
- The upper arch is usually broad, 'U' shaped.
- The palatal vault is usually deep.

Pathological types of occlusion in the sagittal direction progenic (mesial occlusion);Class III Malocclusion.

Class III malocclusion on clinical examination may have the following features:

Extraoral Features (Fig. 5.7. a):

- A straight to concave profile.
- Anteriorly divergent profile.
- Long face (increased lower face height), which may be pointed at the chin.
- Mandible appears to be well developed (with an obtuse gonial angle).

Intraoral Features (Fig. 5.7. b):

- A Class III molar relationship.
- A reverse overjet with possibly labially inclined lower incisors and lingually inclined upper incisions.
- A posterior cross-bite unilateral or bilateral (or functional) due to a constricted maxillary arch or a more forward positioned lower arch.

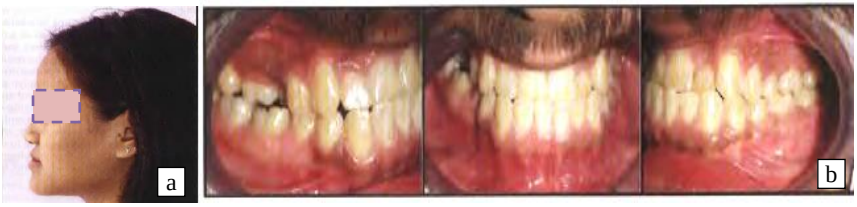

Fig. 5.7. a – Class III malocclusion, extraoral features.
b – Class III malocclusion, intraoral features

Pathological types of occlusion in the vertical direction – open bite *(Fig. 5.8).* An open bite is said to exist when there is a lack of vertical overlap between the maxillary and mandibular teeth. In normal circumstances the mandibular dental arch is contained within the maxillary arch. In other words the maxillary teeth overlap the mandibular teeth labially and buccally. Depending upon the lack of this overlap an open bite is said to exist.

Open bites can exist in the anterior as well as the posterior region. Extent can vary from being simply dental in nature to involving the underlying skeletal structures. The classification and treatment will depend mainly on the location, etiology and the extent of the open bite.

An open bite present in the anterior segment is the most unaesthetic, as the patient has to bring his tongue anteriorly between the teeth and the lips during speech and while swallowing. Posterior open bites may hamper mastication and are more difficult to treat.

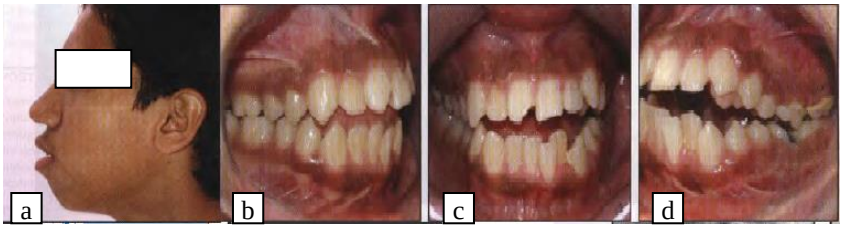

Fig. 5.8. Pathological types of occlusion in the vertical direction – open bite: a – extraoral features, b, c, d – intraoral features

Extraoral features (Fig. 5.8. a):

- Long face due to increased lower anterior face height
- Incompetent lips
- An increased mandibular plane angle
- An increased gonial angle
- Marked antegonial notch
- A short mandible is a possibility
- Maxillary base may be more inferiorly placed (vertical maxillary excess)
- The angle formed by the mandibular and maxillary planes is also increased

Intraoral features (Fig. 5.8. b. c. d):

- Mild crowding with upright incisors
- Gingival hypertrophy
- Maxillary, occlusal and palatal planes tilt upwards
- Mandibular, occlusal plane canted downwards
- Open bite limited to the anterior segment, often asymmetrical.
- Proclincd maxillary and/or mandibular incisors.
- Spacing between maxiliary and/or mandibular anteriors.
- Narrow maxillary arch is a possibility.
- "Fish mouth" appearance.

Posterior open bite *(Fig. 5.9).* Posterior open bites are characterized by a lack of contact between the posterior teeth when the teeth are brought in occlusion. Posterior open bites are relatively rare and are caused mainly because of a lateral tongue thrust habit or submerged/ ankylosed posterior teeth.

Pathological types of occlusion in the transversal direction – cross bite *(Fig. 5.10).* Cross bites are a deviation of the normal bucco-Iingual relationship of the teeth of one arch with those of the opposing arch. Graber defined cross bites as a condition where one or more teeth may be malposed abnormally, buccally or lingually or labially with reference t0 the opposing tooth or teeth.

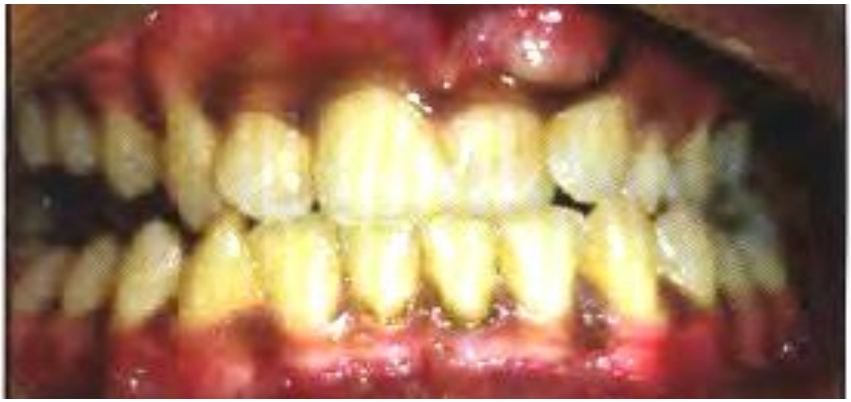

Fig. 5.9. Posterior open bite

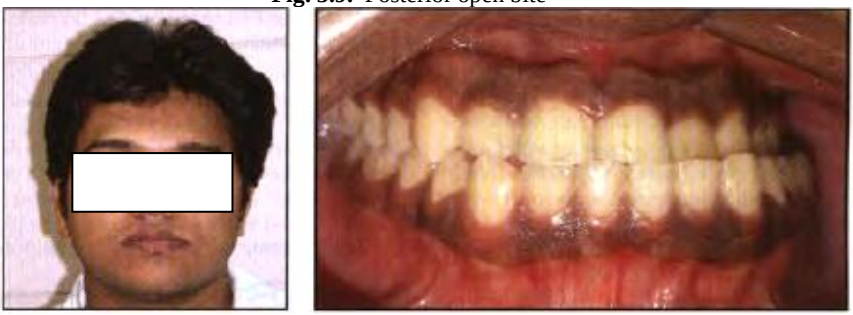

Fig. 5.10. Pathological types of occlusion in the transversal direction – cross bite

Under normal circumstances the maxillary arch overlaps the mandibular arch both labially and buccally. But when the mandibular teeth, single tooth or a segment of teeth, overlap the opposing maxillary teeth labially or buccally, depending upon their location in the arch, a cross bite is said to exist.

- Cross bite causing facial asymmetry
- Here the buccal cusps of left posterior teeth occlude lingual to the buccal cusps of the mandibular teeth.

Prophylactic measures organization. Every orthodontist should organize his work in such a way that at least one day a week is dedicated to prophylaxis, i.e. reception of children of preschool age. At that the doctor should not wait till children come to his consulting room, but is to examine them routinely in infant schools and detect initial stages of occlusion anomalies.

The children, in which endocrine system disorders, upper airways pathology, dentognathic apparatus pathology, rachitic, and other diseases are found, are referred to children's polyclinics, to the doctors of corresponding profession. Simultaneously these children are subject to prophylactic measures and early treatment by an orthodontist. Orthodontists are to talk regularly with mothers about

the importance of correct air-and-sunshine regimen, pay attention to the necessity of rational nutrition and intensive chewing of food. The doctor also gives to children a demonstrative myogymnastics lesson, and also instructs mothers in the technique of doing the gymnastics of masticatory and expression muscles at home.

In the fight against occlusion anomalies not only orthodontists but also children's dentists should participate. The oral cavity can not be considered sanified if dental system deformations are not eliminated, especially since for the true and full-value caries prophylaxis not only the fight against initial caries of hard tissues matters, but also the elimination of factors which may cause the initial stages of teeth affection by carious disease. And the presence of an anomaly of teeth or dental arches position is just a reason for caries, just as caries is often the reason for dental arches deformation. Thus, occlusion anomalies prophylaxis is an integral part of oral cavity sanation.

Therefore, the ways of fighting for the healthy mouth in a child almost completely coincide in the work of the orthodontist and the children's dentist.

Pediatricians, otolaringologists, endocrinologists, hygienists, pedagogues, and all other workers of children's institutions are to be involved in the sanitary and educational work directed at fighting occlusion anomalies; then orthodontists will manage to find children with occlusion anomalies at the earliest development stage and the fight of dentists for the healthy children's organism and full-value mastication apparatus will be more successful and fruitful.

5.2. Dispensary System in Orthodontics

Dispensary system is the system of Ukrainian treatment-and-prophylactic institutions work, which provides diseases prevention, their early detection and treatment at systematic observation of patients. Health examinations are conducted by local children's stomatological polyclinics and orthodontists in particular, who have a prophylactic day a week. It is conducted in children's institutions and consists of several stages.

- The first stage – registration of all children. Age, sex, and general state of health are taken into consideration.
- The second stage – specialized examination of every child.
- The third stage – their distribution into groups.
- The fourth stage – observation of patients, oral cavity sanation, conducting lessons of hygiene and other mass prophylactic measures.
- The fifth stage – studying the effectiveness of orthodontic health examination.

A complex of treatment-and-prophylactic measures, outlined.at the examination of a child, is registered in the card of health examination, after what children are distributed into dispensary groups. Osadchyi has singled out 4 dispensary groups:

4 dispensary groups:

To the 1st group are included children with correct lips closure, normal functioning of the dentognathic apparatus and regular occlusion. These are practically healthy children examined once a year.

The 2nd group includes children with risk factors, i.e. with functional disorders of breathing, swallowing, speech, mastication, mimicry, pernicious habits, with shortened frenula of lips, shallow vestibule of oral cavity. In such children the reasons for disorders should be eliminated and favorable conditions fro the normal growth of jaws and occlusion formation should be created. Oral cavity sanation is conducted, methods of pernicious habits control and curative myogymnastics are recommended, specialists' (otorhinolaryngologist, orthopedist, pediatrician, and others) consultations are recommended; such children should be watched by parents, teachers, medical staff of the children's institution. Orthodontist's examination is carried twice a year.

The 3d group includes children with not full-blown morphologic changes and anomalies of teeth or their groups position, changes of dental arches shape, occlusion deviations, caused by functional changes. To help such patients measures are taken aimed at the elimination of reasons for disorders development, including orthodontic appliances usage. After treatment examination is carried out once a year.

To the 4th dispensary group are included children with evident changes in the dentognathic apparatus: derangements of the functions of breathing, swallowing, speech, biting and chewing food. Such children require specialized help and complex treatment measures which fix the function of the dentognathic apparatus and the organism on the whole.

Chapter 6. Oral habits in child age. Classification. Mechanism of development of dentognathic system anomalies of children with oral habits

Oral habits in children are a prime concern for the dentist, be it an orthodontist, pedodontist or a general practitioner (*Fig. 6.1*). The neonate uses its mouth as a primary device for exploring the environment and his survival depends on instinctive sucking when his lips and tongue are stimulated. By random movements, infants discover their hands and toes, and use these to continue stimulation of the mouth and related structures. Normal habits grow out of these early developmental stages smoothly. Occasionally, a retained infantile pattern can cause an evident oral habit.

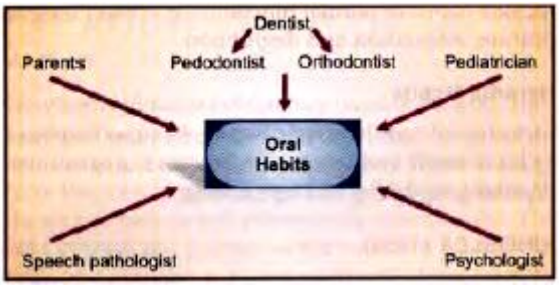

Fig. 6.1. Confluence of specialists

Finn says that habits cause concern because they **cause:**

I. *Oral structural changes* Harmful, unbalanced pressures bear upon the immature, highly malleable alveolar ridges and bring about potential changes in position of teeth and occlusion.

II. *Behavioral problems.*

III. *Socially unacceptable act.*

Definitions of habits:

1. Dorland (1.963): Fixed or constant practice established by frequent repetition.

2. William James: A new pathway of discharge formed in the brain by which certain incoming currents lead to escape.

3. Maslow (1949): A habit is a formed reaction that is resistant to change, whether useful or harmful, depending on the degree to which it interferes with the child's physical, emotional and social functions.

4. Moyers: Habits are learned patterns of muscle contraction, which are complex in nature.

5. Johnson (1938): A habit is an inclination or aptitude for some action acquired by frequent repetition and showing itself in increased facility to performance and reduced power of resistance.

6. Stedman: Habit is an act, behavioral response, practice or custom established in one's repertoire by frequent repetitions of the same act.

7. Habit is an autonomic response to a situation acquired normally as the result of repetition and learning, strictly applicable only to motor responses. At each repetition the act becomes less conscious and can lead 017 to a unconscious habit.

Classification of habits:

Over time various authors have classified habits in differing ways:

William James (1923) classified habits into:

- *Useful Habits***.** Include habits of normal function, e.g. correct tongue posture, respiration and deglutition.
- *Harmful Habits***.** Includes all habits which exert pressures/stresses against teeth and dental arches and also mouth breathing, lip biting and lip sucking.

Based on the nature of the habits, **Kingsley (1956)** classified habits as:

a. *Functional oral habit,* e.g. mouth breathing.

b. *Muscular habits* Tongue thrusting, cheek/lip biting.

c. *Combined muscular habits* Thumb and finger sucking.

d. *Postural habits.*

- Chin-propping
- Face leaning on hand
- Abnormal pillowing.

Earnest Klein (1971):

- *Intentional/Meaningful Habits*. Intentional meaningful habits are caused by a definite underlying psychological disturbance.
- *Unintentional/Empty Habits.* A meaningless habit has no need for support. They can be easily treated by reminder appliances.

Graber (1976) included all habits under extrinsic factors of general causes of malocclusion.

1. Thumb/digit sucking
2. Tongue thrusting
3. Lip/nail biting, bobby pin opening
4. Mouth breathing
5. Abnormal swallow
6. Speech defects
7. Postural defects
8. Psychogenic habits-bruxism
9. Defective occlusal habits.

Finn and Sim (1975):

• Compulsive oral habits
• Non-compulsive oral habits.

Compulsive Habits. An oral habit is compulsive when it has acquired affixation in the child to the extent that he retreats to the practice of this habit whenever his security is threatened by events which occur in his world. They express deep-seated emotional need and attempts to correct them may cause increased anxiety. The act serves as a bulwark against society or a safety valve when emotional pressures are too much to bear. Various etiologies often implicated are:

• Rapid feeding patterns
• Too little feeding at a time
• Too much tension during feeding
• Bottle-feeding
• Insecurity brought by a lack of love and tenderness by the mother.

Non-compulsive Habits. Non-compulsive habits are the ones that are easily added or dropped from the child's behavior pattern as he matures. Continual behavior modification causes release of undesirable habits and addition of new socially acceptable ones. No abnormal response results from attempts to retrain the child to form a pattern of behavior consistent with his increased level of maturity. Some of the other common classifications of habits are as follows:

Habits can be classified according to the cause of the habit:

• *Physiologic Habits*. Those required for normal physiologic functioning, e.g. nasal breathing, sucking during infancy.

• *Pathologic Habits*. Those that are pursued due to pathologic reasons e.g. mouth breathing due to deviated nasal septum (ONS)/enlarge adenoids.

Classification based on the origin of the habit:

• *Retained Habits.* Those that are carried over from childhood into adulthood.

• *Cultivated Habits*. Those that are cultivated during socioactive life of an individual.

Classification based on the patient awareness to the habit:

• *Unconscious Habits*. Unconscious habits are sustained by unconscious behavior. Simple attenuation of sensory feedback mechanism aid in cessation.

• *Conscious Habits.* Involve choice or need, making treatment more difficult and complex.

Okushko (1975) classified habits into:

Functions are divided into incorrect type of the function and unnecessary correct type of the function.

I. Oral habits of sucking (fixed reaction for movement):

1. Finger sucking.
2. Lips/cheeks/object (pen, dummy) sucking and biting.
3. Tongue sucking and biting.

II. Pathologic function (fixed function which is incorrect):

1. Incorrect mastication (unilateral mastication, lazy mastication).
2. Incorrect swallowing and tongue thrusting.
3. Mouth breathing.
4. Incorrect articulate (speech).

III. Fixed postural and tonic reflexes, which determine incorrect position of parts of the body in the rest:

1. Incorrect position of the body and incorrect posture.
2. Incorrect position of the mandibulare and tongue in the rest.

Maturation of oral function. Principle physiologic functions of oral cavity are respiration, swallowing, mastication and speech. Respiration needs are the primary determinants of the posture of the mandible and tongue.

Fetal life. Respirative movements are seen in utero but lungs do not inflate. Swallowing occurs during the last months of fetal life.

At Birth. Newborn infants are obligatory nasal breathers. Thus, to open the airway, mandible is positioned downward and tongue is moved downward and forward away from the posterior pharyngeal wall. Breathing through the mouth becomes physiologically possible later. The next physiologic priority of the newborn is to obtain milk and transfer it to the gastrointestinal tract by sucking and swallowing.

Suckling. Milk ducts of lactating mammals are surrounded by smooth muscles and the infant stimulates their contraction by suckling, i.e, small nibbling movements of lips. This causes squirting of milk into the mouth. Infant grooves the tongue to allow milk to flow posteriorly. This sequence of events is called an *infantile swallow,* which later on matures to an adult swallow.

At Birth. Maturation of oral function is characterized as a gradient from anterior to posterior. Hence, at birth, we see that lips are more mature as compared to the tongue and greater activity by posterior parts of the tongue and complex motion of pharyngeal structures is seen as time passes and maturation proceeds.

Acquisition of speech takes place with development of pharyngeal structures.

After sucking habits are extinguished, a complete transition into adult swallow requires some months. Delay can occur in the presence of an anterior open bite (due to a habit) due to the physiologic need to seal the anterior space.

Development of habit.

A newborn instinctively develops certain habits essential for his survival in the state of neuromuscular immaturity.

There are 5 sources of unconscious mental patterns in childhood which may lead to the development of a habit:

1. Instinct
2. Insufficient outlet for energy
3. Pain/discomfort or insecurity
4. Abnormal physical size of parts
5. Limitation/imposition by parents or others.

Etiological agents in the development of oral habits.

Anatomical. For example, posture of the tongue. Infantile swallow occurs due to a large tongue in a small oral cavity coupled with anterior open bite of gum pads *(Fig. 6.2. a).*

Mechanical interferences. Mechanical interferences lead to undesirable oral habits, e.g. in a child with normal breathing and swallowing, if permanent incisors erupt ectopically (*Fig. 6.2. b*), then to achieve a proper anterior seal/vacuum when swallowing, the child must thrust the tongue and resultant mouth breathing occurs due to loss of 1iP seal. Again if the succedaneous teeth are missing *(Fig. 6.2. c)*, an abnormal habit can develop.

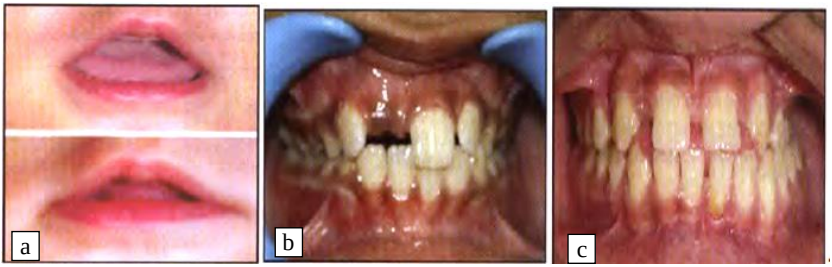

Fig. 6.2. a – Infantile swallow; note the placement of the tongue at rest and its position just before the act of swallowing. The tongue comes in between the gum pads to obtain the vacuum required to suck.
b – Ectopically erupting central incisor resulting in the tongue be incisors
c – Anterior tongue thrust habit due to the congenitally missing permanent maxillary lateral incisors

Pathological. Certain conditions of oral and perioral structures can cause an undesirable oral habit, e.g. tonsillitis, DNS, hypertrophy of inferior nasal turbinates (can cause mouth breathing).

Emotional. Upset children regress towards infancy, assume infantile postures, e.g. digit sucking which gives the child a feeling of security.

Imitation. Young children are extremely observant and sensitive to environment and highly affected by parents and siblings. The child may imitate jaw positions/speech disorders of parents.

Random behavior. Behavior appears purposeless if not completely accidental.

Equilibrium theory. Weinstein *et al* (1963) observed: "An object subjected to an unequal force will get accelerated and thereby will move to a different position in space. Hence, any object subjected to a set of forces remains in place if forces are balanced". In dentition, small imbalance of forces maintained for a long time (6 yrs) can upset the equilibrium. This depends upon the duration of the habit.

Tretment philosophy and consideration.

Three main variables need to be considered.

1. Emotional significance of a habit for the child in relation to family and peer group.

2. Age.

3. Existing or potential malocclusions associated with a force exerting habit.

It should be kept in mind that:

a. Active intervention before 3 years, other than ignoring the habit is unadvisable. Contingency behavior modification should be done.

b. A 3-year-old child with a skeletal Class II may not be correctable by any measure.

c. A5-year-old with Class I and anterior open bite: correction is expected only if the habit is stopped before eruption of the permanent incision.

d. Malocclusion development and correction are most dramatic during active eruption of permanent dentition.

Restracting habit. Process of progressive elimination of an undesirable oral habit from a child's behavior pattern involves use of various approches:

Psychologic Methods/Approach. Examine duration, frequency, osteogenic development, genetic endowment, state of health of the child. It is a clinical rule of thumb – A habit can be overcome only by the conscious efforts of the child himself guided by the dentist and parent. This is possible only if a child is ready psychologically and wants to break the habit.

Parents should cooperate by:

a. Setting a short term goal for dropping the habit

b. Do not criticize the child if the habit continues

c. Offer a small reward if the habit is extinguished.

*Extra-oral Methods***.**

• Painting the child's finger/thumb with an unpleasant tasting substance *(Fig. 6.3. a).*

• Arranging for offending digit to be taped or glove taped at the wrist to hold it in place *(Fig. 6.3. b).*

• Rewarding a child for growing out of the habit during a procedure, makes a deep impression and orients him towards the goal.

• Parents must not demand perfection, with which a child cannot comply.

Intra-oral Methods. Appliances fabricated by the dentist and placed in the child's mouth with or without his overt permission.

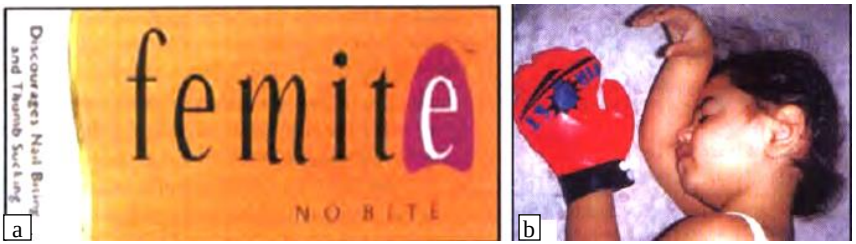

Fig. 6.3. *a – Commercially available bitter material for application on the digit that is put in the mouth. b – Patient wearing a boxing glove to prevent digit sucking*

Digit-sucking habit (thumb/finger-aucking.

Defenitions:

Gellin (1978): Defines digit-sucking as placement of thumb or one or more fingers in varying depths into the mouth.

Moyers: Repeated and forceful sucking of thumb with associated strong buccal and lip contractions.

Practically all children take up this habit, but eventually discontinue it spontaneously with age and maturation, as growth unfolds.

Sucking reflex.

Anatomy and Physiology of Sucking. Engel on direct observation of infants during the first year of *life* revealed their organization to be an oral and clinging one. At birth, the child has a reflex pattern of neuromuscular functions such as sucking.

The habit of sucking is a reflex occurring in the oral stage of development and disappears during normal growth between 1–3½ years. Even before birth, oral contractions and other reflexes have been observed. This early neural organization allows the infant to nurse and cling to the mother as evidenced by the sucking and the grasping reflexes. With hearing and vision development, the baby tries to reach and transport to its mouth, what it has seen and heard at a distance. The baby tends to persist until all possible objects are carried into his mouth.

There are two forms of sucking:

The Nutritive Form. Breast and bottle-feeding, which provides essential nutrients.

Breast-feeding. Flow of milk is regulated by biting and releasing the lacteal glands. Rapid mandibular protrusive movement and buccinator mechanism alternately contracts and relaxes during breastfeeding. Infantile swallowing exerts a progressive pressure towards the rear to drain milk. Gum pads are apart; tongue and lower lip are in constant contact.

Bottle-feeding. Warmth of association with mother's body is lacking. Two types of nipples are available:

• Non-physiologic nipple

• Physiologic nipple.

Non-physiologic/conventional nipple:

• End of the nipple is almost against the posterior pharyngeal wall.

• Milk is directly released into the digestive tract red ucing the period of predigestion.

• Flow of milk may be too rapid.

• Mouth is held wide open.

• Greater demand on buccinator mechanism. Suckling is now converted to sucking.

Physiologic nipple:

• Forward movement of tongue under the flat surface of nipple.

• Nipple is drawn upward and backward towards the palate.

• Child has to work and exercise the lower jaw.

• Posterior part of the tongue awaits milk and pushes it into esophageal area.

• Milk flows down by the peristaltic action of the tongue and cheeks.

• Flat shape of the nipple improves lip seal, e,g, functionally designed latex nuk sauger nipple,

Hence physiologic nipple serves better adapted to anatomy and physiology of sucking.

The Non-nutritive Form. Larsson (1993) described non-nutritive sucking (NNS) to be the earliest sucking habit adopted by infants in response to frustration and to satisfy their urge and need for contact. Children who neither receive unrestricted breast feeding nor have access to a pacifier may satisfy their need with habits like thumb sucking which ensures a feeling of warmth and sense of security but may be detrimental to their dentofacial development.

Psychology of non-nutritive digital-sucking (NNS). Developmental psychologists have produced numerous theories regarding digit sucking.

1. *Psychoanalytical theory of psychosexual development (Sigmund Freud)* NNS arises from an inherent psychosexual drive. It is a pleasurable, erotic structuralization of lips and mouth. Orality in an infant is related to pregenital organizations thus, the object of thumb sucking is nursing, Abrupt interference with such a basic mechanism is likely to lead to stuttering and other antisocial tendencies. Thumb sucking may be the only manifestation of insecurity maladjustment/deep-seated internal conflicts.

2. *Learning theory-Palermo* (1956) NNS stems from an adaptive response. Infants associate sucking with such pleasurable feelings as hunger, satiety and being held.

3. *Eric Johnson and Brent Larsson* (1993). Origin of NNS is a combination of psychoanalytical and learning theories. At will feeding causes less digit sucking when compared to widely separate feeding.

4. *Oral drive theory-Sears and Wise (1960).* Strength of oral drive is, in part, a function of how long a child continues to feed by sucking. The oral drive is strengthened by prolongation of nursing, this is the cause and not frustration of weaning.

5. *Benjamin* (1962). Thumb sucking is an expression of a need to suck that arises simply from rooting and placing reflexes. These reflexes are maximal during the 1st three months of life.

6. Oral gratification theory-Sheldon (1932). If a child is not satisfied with sucking during the feeding period, it will persist as a symptom of an emotional disturbance by digit sucking.

7. Maintenance of habit as explained jointly by Freudian and the learning theory. Prolonged NNS is a learned habit in most children. In fewer children, it may represent some underlying psychological disturbance caused by an acute increase in the level of anxiety.

Classification of thumb sucking:

I. *Cook (1958***)** 3 patterns of thumb sucking:

1. ά Group: Pushed palate in a vertical direction and displayed only little buccal wall contractions *(Fig. 6.4. a–b).*

2. β Croup: Registered strong buccal wall contractions and a negative pressure in the oral cavity show posterior cross bite *(Fig. 6.4. c–d).*

3. γ Group: Alternate positive and negative pressure; least effect on anterior occlusion.

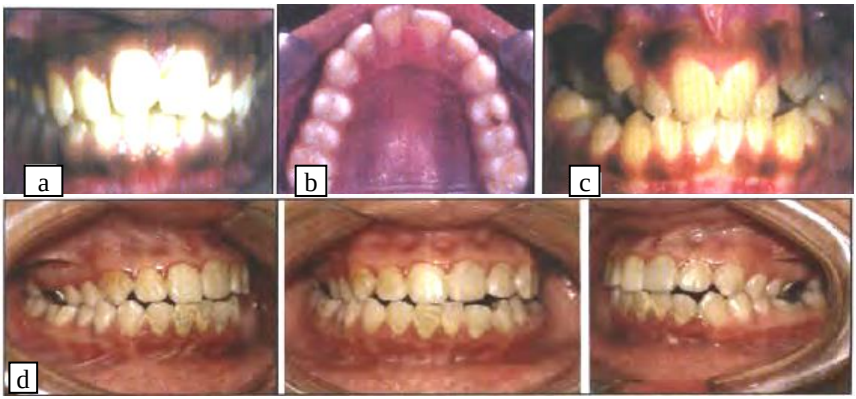

Fig. 6.4. a–b – Vertical placement of the digit in the palate only causes anterior tooth dearrangements. c – Horizontal placement of the digit in the palate and sucking on it can cause bilateral posterior cross-bites. d – Anterior open-bite caused due to a digit-sucking habit

II. *Subtleny et al (1973)*

Four types of thumb-sucking:

Group 1. Thumb was inserted into the mouth considerably beyond the first joint. The thumb occupies a large area of hard palate vault pressing against the palatal mucosa and alveolar tissue. Lower incisors press out the thumb and contact it beyond the first joint. This type was seen in 50 percent of children.

Group II. The thumb extended into mouth around the first joint or just anterior to it. No palatal contact, contacts only maxillary and mandibular anteriors (24 %).

Group III. Thumb placed fully into mouth in contact with the palate as in group I; without any contact with the mandibular incisors (18 %).

Group IV. Thumb did not progress appreciably into the mouth. The lower incisors made contact approximately at the level of thumb nail (8 %).

Index finger may be curled over the bridge of the nose or rolled into a fist with other fingers.

Clinical aspects of digit-sucking. *Moyers* divided the thumb sucking habit into 3 distinct stages:

Phase I. Normal and subclinical1y significant sucking:

• From birth to 3 yrs of age depending on the child's social development.

• Most infants exhibit digit sucking especially during weaning. Usually sucking is resolved towards the end of phase I.

• If vigorous sucking persists at the end of phase I, then a definite prophylactic approach is to be taken due to possible occlusal harm, e.g. pacifier (oval, flat shape) is less harmful.

Phase II. Clinically significant sucking:

Features:

• From 3–6/7 years

• More serious attention required for purposefull digit sucking because:

a. Possibility of clinically significant anxiety.

b. Best time to solve dental problems related to digit sucking.

• Firm and definite corrective program is indicated.

Phase III.

- *Intractable-sucking.*

Persistence of thumb-sucking till phase III is a symptom of significant problem that can be associated with a malocclusion.

- *Finger Sucking from Birth* to 4 *Years.*

The newborn child exhibits a well developed circumoral and intraoral muscular activity. It is the most important means of his exchange with the outside world. During the first few days of life, apart from sucking at meal times, child attempts to suck his fingers or a dummy.

Weaning to the cup is postponed to at least the first birthday. For the first 3 years, damage due to thumb sucking is largely confined to the anterior segment. Usually it is temporary, provided the child starts with a normal occlusion.

- *Finger-sucking After Age 4.*

If habit continues beyond the time it would have been automatically dropped, the child normally comes from homes where ineffectual badgering attempts by the family to break the habit is carried out.

Incidence of finger-sucking habit. Literature surveys of last 40 years show that incidence of digit sucking is highly age dependent.

• Fifty two to sixty seven percent-Incidence in newborn.

• Thirty percent-l year old rate of occurrence.

• Twelve percent-by 9 years number of new cases/year.

• Two percent-12 years or beyond.

Efect of digit-sucking. Dentofacial changes associated with NNS can affect:

I. Maxilla
II. Mandible
III. Inter-arch relationship
IV. Lip placement and function
V. Other effects.

Effects on Maxilla (Fig. 6.5. a–b)

1. Proc1ination of maxillary incisors: When a child places a thumb/finger between the teeth, it is usually positioned at an angle so that it presses against the lingual palatal surface of the upper incisors and the lingual surface of the lower incisors. This direct pressure causes displacement of incisors.

2. Increased arch length.

3. Increased anterior placement of apical base of maxilla: maxillary teeth experience a labial and apical force resulting in flared and labially inclined anteriors with or without a diastema.

4. Increase in SNA angle.

5. Increased clinical crown length of maxillary incisors.

6. Increased counter clockwise rotation of occlusal plane.

7. Decreased width of palate. Left/right side of anterior maxillary arch is usually deformed with deformation related to whether the right or left thumb is sucked.

8. Atypical root resorption of primary central incisors.

9. Trauma to maxillary central incisors (Primarily due to their prominance).

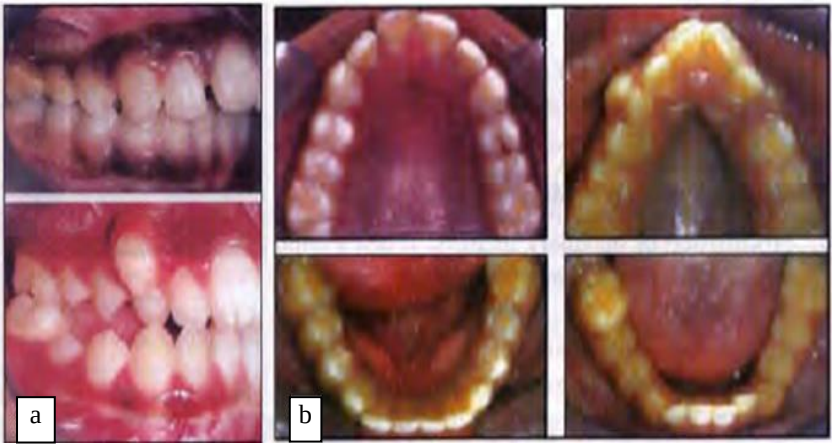

Fig. 6.5. a – Inter-arch relationship in vertical and horizontally placed digit suckers. b – The maxillary and mandibular arches in the vertical and horizontally placed digit suckers

Effects on Mandible (Fig. 6.5. b)

1. Proclination of mandibular incisors.

2. Increased mandibular inter-molar width.

3. More distal position of point B: Mandible is more distally placed relative to the maxilla.

4. Mandibular incisors experience a lingual and apical force.

Inter-arch Relationship (Fig. 6.5. a)

1. Decreased inter-incisal angle.

2. Increased overjet.

3. Decreased overbite.

4. Posterior cross-bite (*Fig. 6.5. a*). If the thumb is placed between the upper and lower teeth, tongue is lowered, which decreases the pressure exerted by the tongue against the lingual aspect of upper posterior teeth, at the same time, cheek pressure against these teeth is increased as buccinators contracts during sucking. Cheek pressures are greatest at the corner of the mouth, therefore, maxillary arch tends to become V-shaped with more constriction across the canines than molars. Hence, the maxillary arm becomes narrower than the mandibular arch.

5. Anterior open-bite (*Fig. 6.4. d*) Arises by a combination of interference to normal eruption of incisors and excessive eruption of posterior teeth. When a thumb or a finger is placed between the jaws the mandible must be positioned downward to accommodate it. The interposed thumb directly impedes incisor eruption. With the separation of jaws, there is an alteration in the vertical equilibrium, which causes more eruption of posterior teeth; about 1 mm supraeruption posterioriy, opens the bite about 2 mm anteriorly resulting in an open bite.

6. Narrow nasal floor and high palatal vault results from loss of equilibrium in the force system in and around the maxillary complex, it is possible for the nasal floor to drop down vertically from its expected position during growth. These are commonly seen and is dependent on the position and pressure exerted by the digit during sucking.

*Efect on lip placement and function***.**

1. Lip incompetence

2. Hypotonic upper lip

3. Hyperactive lower lip: Since it must be elevated by contractions of orbicularis oris and mentalis muscle to a position between malposed incisors during swallowing.

Effect on tongue placement and function.

1. Tongue thrust.

2. Lip to-tongue rest position.

3. Lower tongue position: Tongue is displaced inferiorly towards the floor of mouth and laterally between posterior teeth.

Other Effects

1. Affects psychological health.

2. Risk of malpositioning of the teeth and jaws.

3. Deformation of digits.

4. Speech defects (lisping).

Diagnosis. According to Nanda and Sorokohit (1989) the type of malocclusion that may develop in a thumb sucker is dependent on a number of variables. These include:

1. Position of digit.
2. Associated orofacial muscle contractions.
3. Position of the mandible during sucking.
4. The facial skeletal morphology.
5. Duration of sucking.

The diagnosis of thumb sucking consists of the following diagnostic procedures:

1. *History of Digit Sucking***.** Information on whether the child has had a history of digit sucking is obtained from the parents. When there is a positive answer, one should inquire about:

I. Frequency: Number of times/day habit is practiced.

II. Duration: Amount of time spent on habit.

III. Intensity: Amount of force applied to the teeth during sucking.

More damage occurs to the child with a constant sucking habit, also if sucking can be heard loudly and visible perioral muscle functions and facial contortions are seen, it is more harmful.

2. *Extra-oral Examination***.** Casual examination of the upper extremities can reveal considerable information about the digit used for NNS habit:

a. Cleaner digit

b. Redness, wrinkling or chapped and blistered due to regular sucking.

c. Dishpan thumb-dean thumb with short nails.

d. Fibrous/roughened wart like callus on superior aspect of the digit, ulceration, corn formation.

e. Rarely finger deformity seen.

f. Short upper lip

g. Higher incidence of middle ear infections, blocked eustachian tubes, enlarged tonsils and mouth breathing.

3. *Intra oral Examination.* As discussed in the effects of digit sucking.

Treatment of digit sucking.

Pretreatment Screening.

a. Social background: Inquire into the family status of the child, whether the parents are married/single, lack of jobs, family stress, evidence of child abuse, etc. All factors causing turbulent home environments are ruled out before eliminating the habit.

b. School and peer relationship: Pour school performance and self image diminishes chances for success.

*Dental Consultation***.** Confirm habit, its etiology, duration, frequency, intensity and direction of force as well as the presence of other habits.

*Clinical treatment***.** The child, parent and dentist will be amalgamated together to form a team to assist the child in stopping the habit. According to **Pinkham** there are three categories of treatment:

1. Reminder therapy

2. Reward system

3. Appliance therapy.

Younger than 3 *years:*

I. No active intervention regardless of type and severity of malocclusion because of general emotional immaturity.

II. Most children out grow the habit by 5 years of age.

III. Malocclusion is self-correcting if ceased by the time of eruption of permanent teeth.

IV. Parents are advised to ignore habit.

V. Give more attention to the child when not sucking.

VI. If occlusion Class II, advise need for future orthodontic treatment.

3–7 year old. More concern about finger sucking than thumb sucking due to anterior orthopedic force vectors associated with finger sucking leverage. *Watching and counseling:* working with parent on contingent behavior modification.

7 years and older Anterior open bite will not close by itself due to established functional patterns. Therefore, orthodontic intervention is needed.

Psychologic approach. Duniop's theory "Beta hypothesis" states that the best way to break a habit is by conscious, purposeful repetitions, i.e. the subject should sit in front of a large mirror and suck observing as he does so. The timing of sucking should conflict with some pleasurable activity the child enjoys. By practicing the bad habit with the intent to stop it, one learns not to perform that undesirable act. This is especially practiced in older children (8 yrs and over).

*Reminder therapy: appliance***.** An appliance may be used to control a habit only in the capacity of a psychologic reminder. Appliances must be used after trying psychologic non-appliance approach. Appliances act as reminders for control of habit to break the chain of association with tactile gratification.

A number of factors should be considered before giving the appliance to the child. The patient should be at least 7 years old to reason and understand the need for an appliance. The child should understand the problem and have a desire to correct it. Support and encouragement is necessary from the parents to help the child through the treatment period.

Graber explained the working of these appliances. The appliances:

1. Render finger habit meaningless by breaking suction.

2. Prevents finger pressure from displacing maxillary central incisors thus, avoids/labially from creating worse a malocclusion.

3. Forces tongue backwards changing its postural rest position, thus exerting more lateral pressures.

Reminder therapy: non-appliance. Best suited for those patients who desire to stop the habit but need assistance to do so.

Includes adhesive tapes, bandages to offending digits, mittens, socks, or distasteful liquid/ointments (*Fig. 6.3. a–b*). These serve as reminders for child to remove the finger from the mouth.

Norton and Gellin (1968): Proposed a 3-alarm system often effective in children between 3–7 yrs (Mature children).

1. Offending digit is taped and when the child feels the tape in the mouth it serves as the first alarm.

2. Bandage tied on the elbow of the arm with the offending digit, a safety pin is placed lengthwise. When child flexes the elbow, the closed pin mildly jabs indicating a second alarm.

3. Bandage tightens if the child persists serving as a third alarm.

Chemical approach to habit control. Recommends the use of hot flavored, bitter tasting or foul smelling preparations, placed on the thumb or fingers that are sucked. The chemical therapy uses cayenne (red) pepper dissolved in a volatile liquid medium. Quinine and Asafoetida, which have a bitter taste and an offensive odour respectively, also may be used. This should be done only when the patient has a positive attitude and wants treatment to break the habit.

Corrective therap. Appliances are indicated only when the child wants to discontinue habit and needs only a reminder.

Classification of appliances for thumb-sucking:

1. Removable appliances These are passive appliances which are retained in the oral cavity by means of clasps and usually have one of the following additional components:

a. Tongue spikes *(Fig. 6.6. a.)*

b. Tongue guard *(Fig. 6.6. b).*

c. Spurs/rake (*Fig. 6.7. a*).

2. Fixed appliances

a. Quad helix (*Fig. 6.7. b*)

b. Hay rakes (*Fig. 6.7. c*)

c. Maxillary lingual arch with palatal crib (*Fig. 6.7. d*).

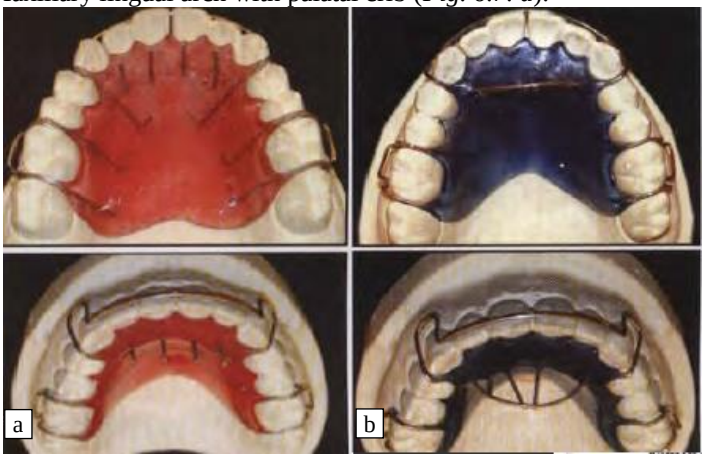

Fig. 6.6. a – Tongue spike. b – Tongue guard

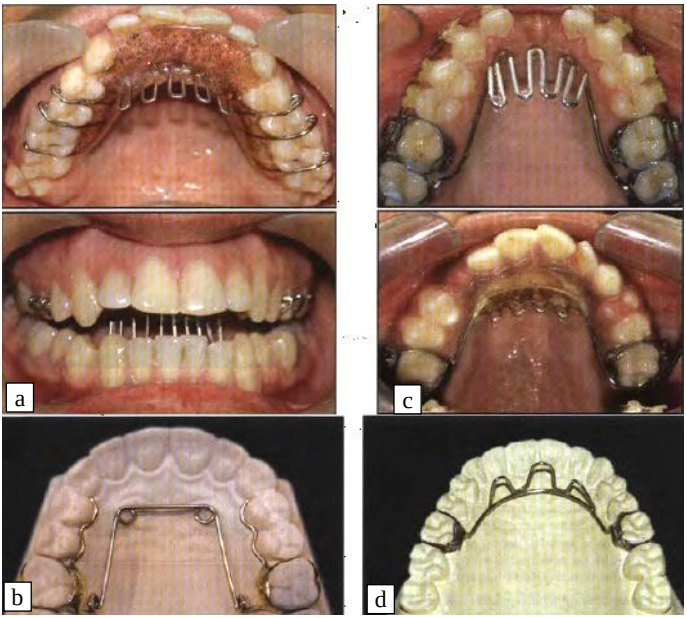

Fig. 6.7. a – Removable tongue crib. b – Quad helix appliance.
c – Fixed tongue rake with and without Nance palatal button.
d –Maxillary lingual arch with palatal crib

One of the best appliances is a lingual arch wire with a short spur soldered at strategic locations, i.e. maxillary lingual arch with anterior crib device to remind the thumb to keep out. It should be well adapted, out of the way of normal oral functioning and contain sufficient sharp, short spurs to provide mild afferent signals of discomfort each time the thumb is inserted.

A clear signal of discomfort or mild pain reminds the neuromuscular system, even when the child is asleep, that the thumb best not be inserted.

This appliance consists of molar bands/crowns on 1st permanent molars with a palatal assembly and soldered spurs made of 0.040" nickel-chrome/S.S. wire. This appliance serves as a reminder and not punishment if its understood by the child as a "helping hand", treatment will be successful and psychologic problems will not result. When sucking ceases, appliance should be retained for approximately 3 months to ensure that the habit has truly stopped.

*Time of therapy***.** Four to six months. A period of 3 months of total absence of finger sucking is convincing evidence of absence of relapse. The ideal appliance for correction of posterior cross-bite due to a digit sucking habit is the Quad Helix *(Fig. 6.7. b)*. It is an adjustable lingual arch that requires little patient cooperation as it is fixed and is reliable and easy to use. Constructed with 38 mil steel wire shaped in the form of a 'W', consisting of 2 anterior and 2 posterior helices.

These helices increase the range and springiness of the appliance. The helicesin the anterior palate are bulky, which can effectively serve as reminders to aid in stopping the habit. Quad helix is activated by opening the helices. The activation of anterior helices produces posterior expansion and activation of posterior helices produce causes anterior expansion. Three months of retention are recommended with this appliance.

Fig. 6.8 demonstrates the sequence to be followed in treating NNS in patients who are able to understand their condition.

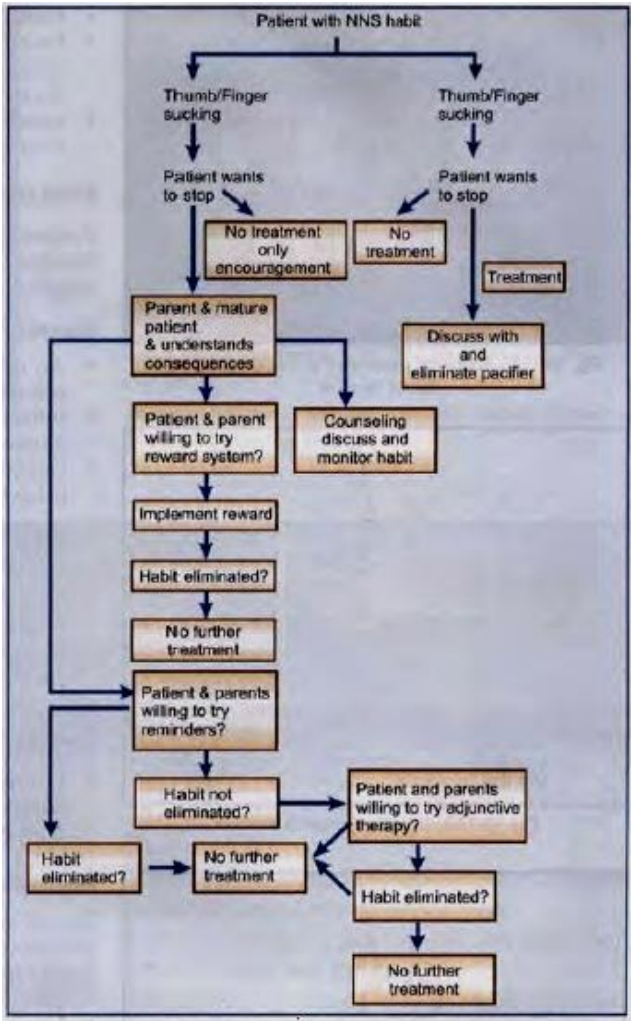

Fig. 6.8. Sequence followed in NNS patients who can understand their condition

Tongue thrusting habit.

Tongue thrusting is the most controversial of all oral habits. Considerable attention has been paid at various times to the tongue and tongue habits as possible factors in malocclusion.

Proffit defined **tongue thrust swallowing** as placement of the tongue tip forward between incisors during swallowing *(Fig. 4.5. a).* This anterior tongue position may be termed as tongue thrust, deviate swallow, visual swallow or infantile swallow. Tongue thrust is actually a 'misnomer' as it means that tongue is forcefully thrusted forward whereas actually. The tongue is only placed forward.

Some of the other definitions are Norton and Gellin (1978): Condition in which the tongue protrudes between anterior and posterior teeth during swallowing with or without affecting tooth position.

*Mechanical restriction***.** Constricted arches which cause tongue to function in a lower than usual position.

• *Macroglossia:* Limits space in the oral cavity and forces a forward thrust *(Fig. 6.9. a–b).*

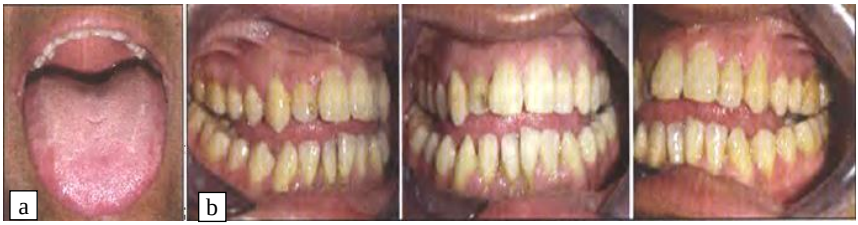

Fig. 6.9. a–b – Abnormally large tongue causing a tongue thrust

Etiology of tongue thrusting. Various factors have been proposed for tongue thrust. Fletcher (1975) reviewed the etiological factors in tongue thrust as follows:

Genetic factors:

• An inherited variation in oro-facial form that precipitates a tongue thrust pattern.

• Inherited anatomic configuration and neuromuscular interplay generating a tongue thrust.

• Genetically predetermined pattern of mouth behavior.

*Learned behavior***.** Improper bottle feeding which results in abnormal functional pattern.

• Protracted period of soreness/tenderness of gum tissue or teeth thereby keeping teeth apart during swallowing.

• Prolonged thumb sucking.

• Tongue held in open spaces during natural exfoliation/ extractions.

• Prolonged tonsillar/upper respiratory tract infection which cause adaptive patterns that are retained even after the infection subsides.

Maturational factors. Tongue thrust presents as a part of normal childhood oral behavior pattern that is gradually 'modified as the lingual space and suspensory system change.

• Late maturation from infantile swallow patterns

• Late maturation from immature patterns of general oral behavior.

• *Enlarged tonsils and adenoids:* Reduce space available for lingual movement.

1. Younger children with reasonably normal occlusion: It is a transitional stage in normal physiologic maturation.

2. Individuals of any age with displaced teeth (incisors): It is an adaptation to the space between teeth (overjet and anterior open bite) since correcting tooth position should cause change in swallowing pattern.

Classification of tongue thrust.

Backlund 1963

Anterior tongue thrust (Fig. 6.10) Forceful anterior thrust.

Posterior tongue thrust (Fig. 6.11) Lateral thrusting in case of missing teeth.

*Neurological disturbances***.** Hypersensitive palate which precipitates crude patterns of food manipulation and swallowing.

• Disruption in tactile sensory control and coordination of swallowing.

• Moderate motor disability and loss of precision in oral function.

*Psychogenic Factors***:**

• Substitution of tongue thrust for forcibly discontinued thumb sucking.

• Exaggerated motor image of tongue.

Modern View. Tongue thrust is seen in 2 circumstances:

*Pickett's 1966***.** *Adaptive tongue* thrust-Tongue adapts to an open bite caused by missing teeth/thumb sucking.

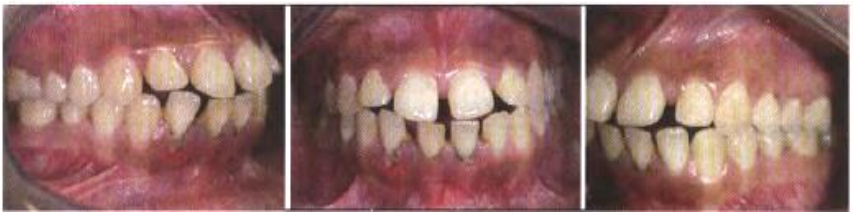

Fig. 6.10. Anterior tongue thrust

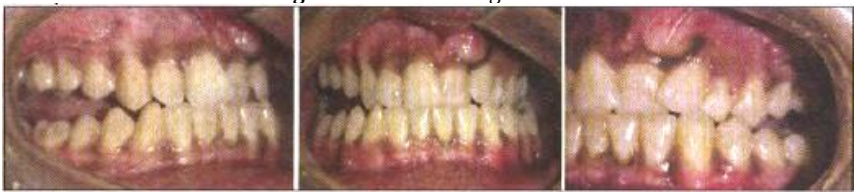

Fig. 6.11. Lateral tongue thrust

Transitory. Tongue is put forward only for a short period. Forceful and rapid.

Habitual. Due to postural problem, a habit or presence of open bite.

Moyers, 1970. *Simple tongue thrust (Fig. 6.12)* Teeth are together.

Complex tongue thrust (Fig. 6.13). Teeth are apart and buccal occlusion is deranged.

Retained Infantile swallow Persistence of infantile swallow even after permanent teeth appear.

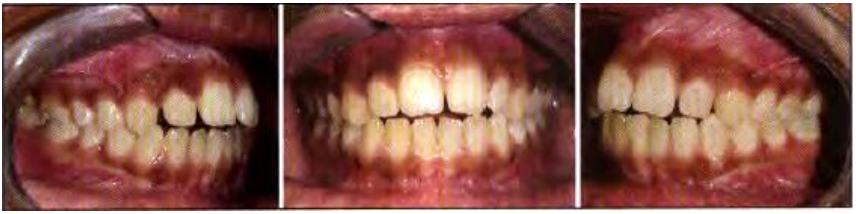

Fig. 6.12. Simple tongue thrust

Fig. 6.13. Complex tongue thrust

James Braner and Holt

Type I: Non-deforming tongue thrust.

Type II: Deforming anterior tongue thrust

• Sub-group 1: Anterior open-bite

• Sub-group 2: Anterior proclination

• Sub-group 3: Posterior cross-bite.

Type III: Deforming lateral tongue thrust:

• Sub-group 1: Posterior open-bite

• Sub-group 2: Posterior cross-bite

• Sub-group 3: Deep over-bite.

Type IV: Deforming anterior and lateral tongue thrust.

Sub-group 1: Anterior and posterior open-bite

Sub-group 2: Proclination of anterior teeth

Sub-group 3: Posterior cross-bite.

Clinical features effects of tongue thrust on dento-facial structures *(Figs 6.14).*

1. Open-bite-anterior and posterior (lateral tongue thrust).

2. Proclination of upper anterior teeth.

3. Protrusion of anterior segments of both arches with spaces between incisors and canines.

4. Narrow and constricted maxillary arch-posterior cross-bite.

Diagnosis.

*Extra-oral examination***.** The facial profile is examined to confirm favorable unfavorable growth pattern, steepness of mandibular plane angle, anterior facial height.

Examination of tongue

• Tongue posture.

• Tongue function.

Tongue Posture. Tongue posture is examined when the mandible is in a postural rest position either using a cephalogram or with the patient seated upright. During rest, dorsum of tongue touches the palate, while the tip rests against the cingula or fossa of mandibular incisors.

*Differential diagnosis***.** Abnormal tongue posture is related to malocclusion and skeletal morphology. In Class III-tongue lies below occlusal plane and in Class II-tongue is positioned forward.

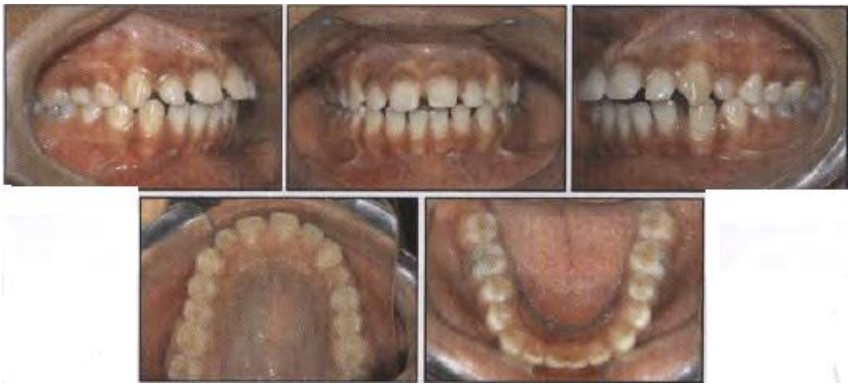

Fig. 6.14. Features of anterior tongue thrust

Two significant variations may be seen in tongue posture.

Retracted. Tip is withdrawn behind all the incisors, seen in 10 percent children.

• Associated with posterior open bite because of edentulous/multiple loss of posteriors.

• Loss of positional sense due to removal of periodontal ligament, tongue retracts to establish contact with the alveolus and used to obtain a seal when swallowing.

Protracted.

• Resting tongue is between the upper incisors.

• Serious, since it results in an anterior open bite.

Two forms:

1. *Endogenous protracted:* Retention of infantile swallow pattern.

• Mild open bite.

• No certain treatment-surgery caused relapse due to failure of tongue to adapt to the new posture.

2. Acquired adaptive: Transitory adaptation to enlarged tonsils and pharyngitis.

Retained infantile swallow. Retained infantile swallow is defined as predominant persistence of the infantile swallowing reflex after the eruption of permanent teeth *(Fig. 6.15).*

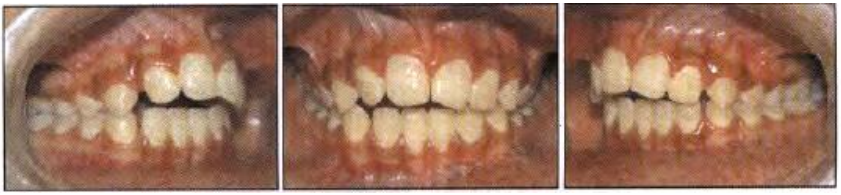

Fig. 6.15. Infantile swallow

Clinical features:

• Strong contractions of lips and facial musculature especially buccinator.

• Massive grimace.

• Anterior and lateral thrusting• Inexpressive face due to use of facial muscles for swallowing.

• Difficulty in mastication since they normally occlude on only one molar in each quadrant.

• Low gag threshold.

• Poor prognosis.

Usually associated with skeletal craniofacial developmental syndromes and neural deficits.

Management of tongue thrusing. The management of tongue thrust involves interception of the habit followed by treatment of the malocclusion.

Since tongue thrust decreases with age, treatment must be based on age.

1. 3–11 *years.* Normal occurrence, not to be concerned, reassure parents. If child is under 7 yrs, there is no need to be concerned since speech sound that elicits a lisp are not matured until 7–8 years of age.

Conservative approach: Demonstrate correct swallow and observe the child.

2. 11 *yrs or* older. Tongue thrust is not a normal pattern.

Management of simple tongue thrust.

Three phases (Moyers):

1. Conscious learning of new reflex-cognitive approach
2. Transferring to subconscious level-reflexive approach
3. Reinforcement of new reflex.

*Cognitive Approach***:** Functional therapy.

*Myofunctional Therapy***.** It is based on the fact that form of the occlusion adapts to function. The sum total forces exerted by the muscles through well motivated, controlled tongue function, and lip exercises, child can be trained to develop a new swallowing pattern and through this altered tongue and lip function correct a malocclusion or for orthodontically treated patients, prevent relapse. Functional therapy is attempted before appliance treatment,

Advantages of postponing tongue therapy until treatment of malocclusion is begun include:

1. In absence of obvious predisposing factors, correction of malocclusion results in disappearance of habit.

2. Gives maximum opportunity for transition to mature adult swallow.

3. Therapy is most effective when carried out with orthodontic treatment.

Muscle exercises:

1. Barnet's tongue positioning exercises.

a. Identify the incisal papilla as the spot behind front teeth.

b. Practice touching spot with the tongue tip.

c. Swallow with lips and teeth closed and tongue tip touching the incisal papilla.

d. Have patient practice this with lips apart.

2. Andrews recommends practice of swallow correctly 20 times before meals with water in the mouth and mirror in hand. Each practice is followed by relaxation of muscles until the swallowing progress smoothly.

3. Use of sugarless mint: Held against roof of the mouth stimulates saliva and makes it necessary to swallow.

4. Single elastic swallow of gardiner.

Using orthodontic elastic band of 1/4" or 5/16" placed on the tip of the tongue plus speech exercises- ”D”, “t”.

5. Double elastic swallow:

• Place 1 elastic each at tip and middle of tongue contact with tip and mid part of palate.

• Lips open with buccal teeth together.

• Speech exercises 'C', 'h', 'g'.

6. Peanuts and elastic band: Patient chews peanuts but not to swallow it. The chewed peanuts are placed in the middle of the tongue.

• Place elastic at tip of tongue.

• Instruct the practice of swallow.

• Speech exercises – 'C', 'g', 'k'.

7. Lip exercises

a. Lip pull exercises - to strengthen lips.

b. Lip over lip exercises - to strengthen lips.

*Reflective approach***.** When new swallowing pattern has been learned at a conscious level, it is necessary to transfer it to the subconscious level. At the second appointment, the patient should be able to swallow correctly at will.

Neuromuscular facilitation: Subcortical method of affecting swallowing act. Sensory input is correlated with motor activities on a subconscious level.

Reinforcement of new reflex. This is achieved by means of mechanical restraints which may be removable or fixed. Cribs or rakes are valuable in breaking the habit *(Fig. 6.6 and 6.7).* Oral screen also may be used *(Fig. 4.4).*

Treatment of complex tongue thrust:

1. Treat the occlusion first.

2. When orthodontic treatment is in its retentive stages, careful occlusal equilibration is completed.

3. The muscle training is begun similar to simple tongue-thrust with minor modifications.

Mouth breathing habit (see Chapter 4).

Lip habits. Lips play an important role in deglutition, speech and maintenance of normal occlusion**.** Three lip habits:

1. Lip-sucking *(Fig. 6.16. a–b.)*
2. Lip-wetting.
3. Lip-biting *(Fig. 6.16. c).*

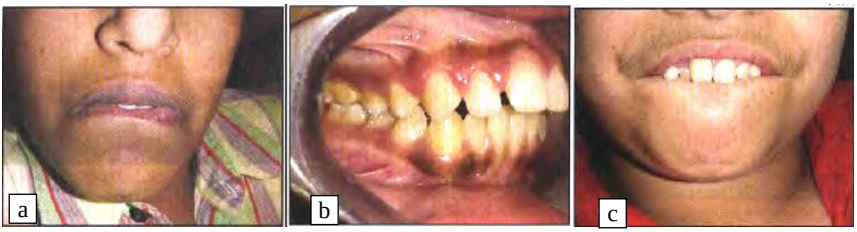

Fig. 6.16. a–c – Lip-sucking habit.
b – Increased overjet and upright mandibular incisors

Lip-sucking. In many instances, lip-sucking (*Fig. 6.16. a–c*) is a compensatory activity that results from an excessive overjet and the relative difficulty of closing the lips properly during deglutition. It is easy for the child to cushion the lip to the lingual side of maxillary incisors. To achieve this position, mentalis muscle extends the lower lip upwards.

The patient may exhibit the following features:

I. When the habit has become pernicious, a marked flattening and crowding of lower anterior segment occurs.

II. Retraction of teeth occurs which depends upon whether the upper or lower lip is sucked.

III. Maxillary incisors may be forced upward and forward into a protrusive relationship (*Fig. 6.16. b*) in lower lip-sucking.

In severe cases, the lip itself shows the effects of abnormal habit.

- The vermilion border becomes hypertrophic and redundant during rest.
- Reddening below vermilion border is seen.
- Flaccid lip due to lengthening caused by frequent practice.
- Mento-labial sulcus becomes accentuated.
- Chronic herpes with areas of irritation and cracking of lip appears sometimes.

Mentalis muscle habit. It is a variation of lip-sucking. Lip-sucking will involve the whole lip, whereas in mentalis habit, the lower lip is everted and only involves the vermilion border. An area of contraction between lips and chin will result in protrusion of maxillary incisors and collapse of mandibular incisors.

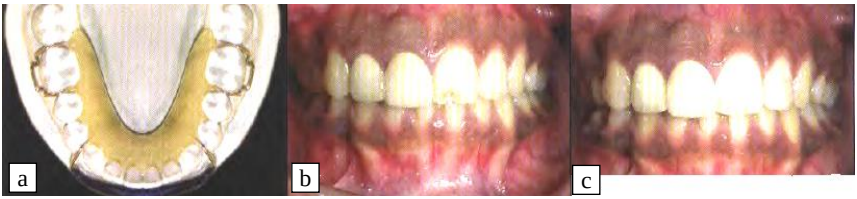

Fig. 6.17. a – The lip bumper appliance used to prevent lip sucking/biting.
b – Defect caused by a bobby pin opening habit.
c – Defect restored with light cure composites

Management.

• Lip over lip exercises
• Flaying bass instruments.
• Lips bumper/shield (*Fig. 6.17. a*)
• Oral screen (*Fig. 4.4*).

Lip-wetting. Tongue constantly wets the lips due to dryness/irritation which later becomes a habit.

Lip-biting. May involve either of the lips (*Fig. 6.16. c*), features seen are cuts and abrasions, marks of incisors on lips along with reddening of lips.

Cheek biting. Biting the cheeks, if unchecked may contribute to ulceration, pain, discomfort or malignancy.

Etiology. Buccoversion of erupting third molar
• Flabby cheeks
• Lack of proper coverage of lower teeth by upper teeth buccally.
• Atrophy of muscles seen in paralysis.

Treatment:
• Identify the cause
• Analgesics
• Appliance therapy - oral screen
• Oral screen (*Fig. 4.4*).

Frenum-thrusting. Rarely seen:
• Patient has spaced upper permanent incisors.
• Child holds the labial frenum between teeth for several hours.
• Starts as idle play and develops into a tooth displacing habit by keeping centrals apart.

Treatment. Orthodontic correction for spacing.

Bobby Pin Opening

Habitual opening of pins with incisors *(Fig. 6.17. b).*

Features:

• Notched incisors

• Teeth derided of labial enamel.

• Treatment involves counseling and restoration of the defect with light cure composites (*Fig. 6.17. c*).

Masochistic habit. It is a prevention in which the sufferer derives pleasure from his own pain. It is the enjoyment of what appears to be painful to others. Ayer and Levin 1974: Based on the etiology divided this self destructive habit into:

Organic. Includes various syndromes it is seen in 19-20percent of mentally retarded population.

Functional.

a. Injuries superimposed on preexisting lesion.

b. Injuries secondary to another established habit.

c. Injuries of unknown and complex etiology with more of a psychological component.

Postural habit.

Chin-propping habit. It is an extrinsic pressure, unintentional habit which causes a deep anterior closed bite. It may cause retraction of mandible.

Face leaning. Lateral pressure from face leaning which is an unintentional, extrinsic pressure habit, may cause lingual movement of maxillary teeth on that side. The mandible is less affected as it does not have a rigid attachment and can slide away from the pressure.

Abnormal pillowing/habitual sleeping on right or left side of face. Normally children do not lie in one position during sleep. The movements are largely involuntary and are produced by nervous reflexes in order to prevent pressure interferences with circulation. Pillowing habits may cause flattening of the skull, facial asymmetry in infants.

In conclusion, the interceptive and preventive orthodontic procedures enable the clinician to treat and/or make a developing malocclusion less severe so as to allow the corrective orthodontist to deliver a stable and more conservative (non-extraction) treatment plan benefiting the patient. Certain patients may need to undergo a 2 phase treatment plan but, the end results are more rewarding. Therefore, the interceptive orthodontic procedures should not be made controversial as being done at present by a section of the dental specialists and instead there should be a team approach towards early detection of malocclusion, patient and parent counseling towards the long-term benefits of the same, cross referral as well as treatment of the malocclusion.

Chapter7. Muscle exercises and functional appliances in preventive and interceptive orthodontics

A malocclusion, if detected as soon as possible, can be eliminated or made less severe, by initiation of interceptive orthodontic procedures. Preventive orthodontic procedures are also interceptive in nature if undertaken soon after the development of the malocclusion.

An interceptive procedure undertaken at the right time can, therefore, either eliminate a developing mal occlusion or make it less severe, so as to allow corrective orthodontics to deliver a stable and conservative result, in the shortest treatment time possible with least discomfort to the patient.

An interceptive orthodontic procedure will ensure that an oral habit does not become fixed and its deleterious effects do not affect the normal growth and development of the patient. It will also ensure that there is no loss of arch length due to the premature loss of deciduous tooth/teeth or due to rotated teeth or on crowding of teeth and guide the growth of mandible by using myofunctional appliances so as to deliver greater benefits to the patient.

The basic interceptive procedures that are undertaken by the interceptive pedodontist are:

1. Space regaining.
2. Correction of anterior and posterior cross bites.
3. Elimination of oral habits.
4. Muscle exercises.
5. Removal of soft or hard tissue impediments in the pathway of emption.
6. Resolution of crowding.
7. Interception of developing skeletal malocclusions.

Muscle exercises. The normal development of the occlusion depends on the nature of the muscles of the face. If the oramaxillofacial musculature were in a state of balance, a good occlusion would develop and if any of the muscle groups were aberrant it would result in a malocclusion in some form or the other. Muscle exercises allow a clinician to bring such aberrant muscular functions into normal functioning, to create normal health and function, as they are important elements in aiding growth and development of normal occlusion.

Uses

1. To guide the development of occlusion.
2. To allow optimal growth patterns.
3. To provide retention and stability in post-corrective (mechanical) orthodontic cases.

Exercises

Exercises of orbicularis and circumoral group of muscles:

A. Upper lip is stretched in the posteroinferior direction by overlapping the lower lip. Such muscular exercises allow the hypotonic lips to form an oral seal labially.

B. Hypotonic lips can also be exercised by holding a piece of paper between the lips.

C. Parents can stretch the lips of the child in the posteroinferior direction at regular intervals.

D. Swishing of water between the lips until they get tired.

E. Massaging of the lips.

F. Playing a reed musical instrument-produces fine lip tonicity.

G. Placement of scotch tape over the lips helps to train them to remain sealed.

H. Use of an oral screen with a holder-to exercise the lips.

I. Button pull exercise-a 1½ inch diameter button is taken through which a thread is passed. The patient is asked to place the button behind the lips and pull the thread while the lips try to resist the same.

K. Tug of war exercise-is similar to the button pull exercise, where the difference is that 2 buttons are used and another individual pulls the thread gently while the same movement is resisted, by the patient.

Exercises of the tongue. Exercises of the tongue are done to correct any aberrant tongue swallow patterns:

a. *One elastic swallow.* An orthodontic elastic, usually 5/16th of an inch, is placed on the tip of the tongue and the patient is asked to raise the same to rugae area and swallow**.**

b. *Two elastic swallow* 5*/16th* inch elastics are used and one is placed on the lip of the tongue whereas the other is placed on the dorsum of the tongue in the midline and asked to swallow.

c. *Tongue hold exercise* A 5/16th inch elastic is used and the patient is asked to place the same on a designated spot over a definite period of time with the lips closed. The patient is asked to swallow with the elastic in the designated position and lips apart.

d. *Hold pull exercise.* The tip of the tongue is made to contact the palate in the midline and the mandible is gradually opened. This allows the stretching of the frenum to relieve a mild tongue-tie.

***Exercises of masseter muscles*.** At times it is advised to strengthen the masseter muscles. The patient is asked to clench his teeth, count up to 10 in his mind and then relax them. This has to be repeated over a period of time, until the masseter muscles feel tired.

Exercises of pterygoid muscles. In case of disto-occlusion cases the patient is asked to protrude the mandible as much as possible and then retracted. Repeat the exercises until the muscles feel tired. The ability to keep the mandible in correct position gradually improves.

Limitations of muscle exercises:

1. Exercises are not known to drastically alter any bone growth pattern.
2. They are not a substitute for corrective orthodontic treatment.
3. Patient compliance is extremely important.
4. If not done correctly, can be counter productive.

A developing skeletal rnalocclusion if detected at an earlier stage can be intercepted so as to decrease its severity and at times even resulting in a normal

occlusion. These changes are brought about by myofunctional therapy, which more appropriately is known as Functional Jaw Orthopedics today. Mills (1991) has defined *a functional appliance as a removable or fixed appliance, which changes the position of the mandible* so *as to transmit forces generated by the stretching of the muscles, fascia and/or periosteum, through the acrylic and wirework to the dentition and the underlying skeletal structures.*

CAD/CAM myofunctional appliances. The concept of myofunctional appliances, which developed in Scandinavian countries, shifted to Germany before the World War II, got stagnated there and finally spread to Europe, in the post-wartime. It became accepted over a period of time. It however, did not become popular as most of the myofunctional appliances were difficult to fabricate, had frequent breakages and were hard.

Farrell (Myofunctional Research Company) in Australia using CAD/CAM techniques and the flexibility as well as inherent memory effects of silicone/non-thermoplastic polyurethane produced myofunctional appliances. The appliance was developed to bring about tooth guidance effects as well as have a functional effect too.

Design. The appliance has been designed using CAD/CAM techniques. The appliances are soft and are shaped in the form of the normal parabolic shape of the dental arches. It has channels for the maxillary and mandibular teeth. The labial/buccal screen has premolded condensations of the material, which act as labial bow, thereby having an effect similar to the arch wire in corrective (fixed) treatment. This allows the irregular teeth to get aligned and the tooth channels further guide the teeth into the normal arch form.

The oral screen like structure enveloping the teeth buccally/labially help in treating the mouth breathing or thumb sucking habits. This allows for the child to shift from oral to nasal breathing, which in turn allows the nasal passages to develop and the palate to descend. The maxillary arch therefore tends to develop into a shallow arch and a U shaped arch develops due to the parabolic natural like shape of the appliance. Promotion of development of a U shaped arch allows an increase in the inter-canine dimensions of the maxilla, which in turn allows an increase in the intercanine dimensions of the mandible, thereby allowing resolution of mandibular anterior crowding.

Small projections on the labial aspect of the oral screen like structure in the region relating to the mandibular anteriors, behaves as a lip bumper or mentalis stretcher, which in turn deactivates an overactive mentalis muscle, thereby allowing a mandibular anterior flat arch to develop into a rounded one and thereby increasing arch perimeter. It also allows the perioral group of muscles to become normotonic thereby ensuring a lip seal. A tongue tag has also been incorporated in the maxillary palatal aspect, which is used to train aberrant tongue habits such as retained infantile or tongue thrust cases. Thus, the imbalance of forces acting on the developing arches if any from the lingual aspect are also taken care of.

The tooth channels are designed in such a way so as behave like an activator guiding the teeth into the occlusion.

These are used in 2 phases-the softer blue pre-orthodontic trainer first, which allows for correction of aberrant muscle movements and mild tooth movements. This is generally worn for about 6 months or until one finds that the aberrant movement of the groups of muscles have ceased considerably followed by the firmer pink pre-orthodontic trainer, which exerts slightly greater forces for the alignment of teeth. The CAD/CAM process has allowed the appliance to be developed in such a way that a single size is applicable to all the patients. The only adjustments required are in case of the distal aspects, which can be easily trimmed. In case of open bite cases the appliances may need to be trimmed distally so that the maxillary anterior teeth get to lie below the maxillary labial bow like premolded area so that the appliance exerts the corrective forces.

Indications:

1. Mandibular anterior crowding.
2. Class II Division I and II.
3. Anterior open bite.
4. Deep bite.
5. Mild Class III/ Pseudo Class III.
6. Tongue thrusters, thumb sucking and oral breathing habits.

Contraindications:

1. Posterior cross bite-which is uncorrected.
2. Severe Class III.
3. Complete nasal obstruction.
4. Non-cooperative child / parent.

The appliance should be inserted for a minimum of one hour daily during the day and also be worn while sleeping. Initially, the appliance may fall out while sleeping at night, this would decrease over a couple of weeks and finally the appliance would not fall out in sleep, as the aberrant muscular forces become normal. The daily one hour wearing is important so as to unlearn the old habits and learn the correct habits at the conscious levels, e.g. in case of aberrant tongue swallow patterns and the night time wear during sleep is equally important so as to convert the same i.nto a subconscious habit. The blue trainer is made to be worn for 6–8 months followed by the pink trainer for 12 months or so until the treatment objectives are achieved. Alignment of teeth will begin to occur in 3–6 months time. A clinical review once every month is important to review as well as motivate the child to wear the appliance regularly and for the parents to see the changes and regulate the child use of the appliance at home. The appliance is kept clean by brushing the same with lukewarm soft soapy water every day.

Adjustment Required. Generally the appliance does not require to be adjusted, except for a few cases such as:

1. Narrow mouths may require 2–3 mm of the distal ends of the appliance to be trimmed, if the patient finds the appliance to be long or the lips do not close over the appliance.

2. If the tongue tag area hurts the V cuts on either side of the appliance needs to deepen.

3. If there is an exacerbated mouth opening or in extreme Class II cases, where the maxillary anterior teeth do not come into the tooth guidance system, 2–3 mm of the distal ends are trimmed to correct the same. If the maxillary anterior teeth still do not fit into the appliance well, then the trimming of the upper labial bow of the appliance is indicated.

4. In children less than 6 years of age, where the permanent first molars have not erupted, 4–6 mm of the distal ends are trimmed off.

5. Upper labial bow is removed in case of Class III cases so as to allow the maxillary arch to advance anteriorly over the mandibular arch.

6. The lower labial bow is removed to have an enhanced lip bumper effect so as to increased arch length. This is more effective in the pink pre-orthodontic trainer.

7. A more compliant patient with the use of the appliance may result in an edge-to-edge bite; this can be corrected by removing the upper labial bow to allow an overjet and overbite to develop.

This way the treatment is not only more economical, but also more stable, with an improvement in facial profile and features, decrease in the need for extractions and decreased corrective orthodontic treatment time, if required. The pre-orthodontic trainer can also be used with the Farrel Bent Wire system, so as to bring about arch development by lateral expansion forces once the appliance is activated. These can be placed on both the maxillary and mandibular arches in a phased manner, starting from the maxilla. It along with the pre-orthodontic trainer corrects the tongue positioning, which in turn brings about a slight anterior tipping of the anteriors thus increasing the arch perimeter. Thus, arch length deficiency including lost canine space can be regained without the opening of the bite. This generally should take about 6 to 9 months if the patient wears the appliance properly.

Certain other appliances have also been introduced which allows the aberrant muscular forces to be corrected along with the use of corrective fixed appliances (*Fig, 7.1. a–b, 7.2*).

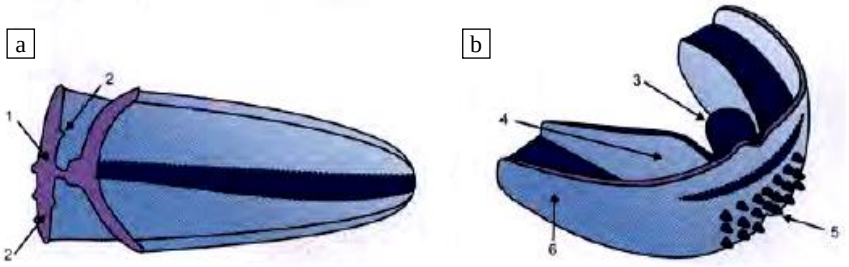

Fig. 7.1. a – Cut section of the pre-orthodontic trainer: 1-Tooth channels, 2-Labial bows.
b – Parts of the pre-orthodontic trainer 3-Tongue tag, 4-Tongue guard,
5-Lip bumpers and 6-Enables jaw positioning into edge-to-edge Class I occlusion

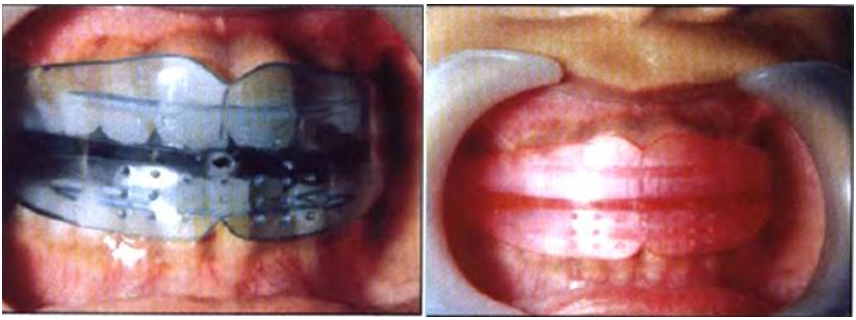

Fig. 7.2. Blue and pink pre-orthodontic appliance in place

Functional Appliances.

Conventional orthodontic appliances use mechanical force to alter the position of tooth/ teeth into a more favorable position. However, the scope of these fixed appliances is greatly limited by certain morphological conditions which are caused due to aberrations in the developmental process or the neuromuscular capsule surrounding the orofacial skeleton. To over come this limitation, functional appliances came into being.

These appliances are considered to be primarily orthopedic tools to influence the facial skeleton of the growing child. The uniqueness of these appliances lies in the fact that instead of applying active forces, they transmit, eliminate and guide the natural forces (e.g. muscle activity, growth, tooth eruption) to eliminate the morphological aberrations and try to create conditions for the harmonious development of the stomatognathic system.

Most of the functional appliances are intraoral devices, and nearly all of them are tooth borne or supported by teeth. With a few exceptions, these appliances are removable, consisting primarily of acrylic with wire components for retention and support.

Most of the functional appliances are used to correct early Class II malocclusions and some cases of Class III malocclusion. About 40 percent of all malocclusions treated belong to the Class II category. This preponderance of the Class II malocclusions seen in orthodontic practice is partly a result of public awareness of aberrant characteristics associated with the malocclusion and education of the public by the concerned dental practitioners.

Too often, Class II malocclusions have been treated with extra-oral forces directed against the maxilla. However, abundant research has shown that much of the problem lies in the mandible, which is retrognathic (small) or retropositioned (backwardly placed) or a combination of both. Nevertheless the past 30 years have seen an increasing awareness regarding the potential of functional appliances as a valuable tool in the armamentarium of an orthodontist.

Basis for functional appliances. Although functional appliances have been in use for over two centuries, their scope and potential has been realized only recently with the recognition of interrelationship between form and function and the realization that neuro-muscular involvement is vital in treatment.

Graber described the 'Three Ms: Muscles, Malformation and Malocclusion in 1963. Melvin Moss, again validated the concept of form and function through his theory 'The Functional Matrix Hypothesis'.

Another factor that gave impetus to the use of functional appliances (especially mandibular hyperpropulsers) was the identification of certain cartilages in the body as "'Secondary cartilages".

Secondary cartilage is that' cartilage which is not of developmental origin but rather differentiates from the bony periosteum in response to the needs of the body, e.g. condylar cartilage.

Among the other features of the secondary cartilages (*Table 7.1*) the characteristics which make it useful for functional appliance therapy is its adaptive growth response to the local intrinsic and extrinsic stimuli, unlike the primary cartilages which are minimally responsive to local factors/stimuli.

Table 7.1

Differences between primary and secondary cartilalage

Biological criteria	Epiphyseal growth plate or primary cartilage	Condyles or secondary cartilage
Origin	Derivative of primordial cartilage (chondroblasts)	Secondary cartilages form on original membrane bone
Growth	Interstitial, three dimensional, in hyaline cartilage	Peripheral in Fibro-cartilage covering: proliferating cells are not cartilage cells but rather resemble undifferentiated mesenchymal cells
Maturation	Secondary ossification center, final fusion, disappearance all cartilage. Only the degenerative zone is mineralizing: Primary spongiosa	Conversion from hypertrophic to non-hypertrophic state, but no complete conversion into bone. Whole hypertrophic area in a state of mineralization: No primary spongiosa
Histology	Well arranged histologic features	Haphazard arrangement
Hormonal control	Marked response to thyroxine deficiency. After final fusion; no further response to growth hormones	Minimal response to tryroxine deficiency. Mature condyle can be awakened by growth hormone
Vitamin response	Ascorbic acid deficiency leads to Gerustmark zone; Vitamin D deficiency results in classic picture of Rickets	Vitamin C deficiency elicits minimal response; Vitamin D deficiency causes reversion to more immature state
Mechanical stimuli	Unresponsive	Responsive
Antigenic difference	Possesses antigenic determinants-common to condylar cartilage and nasal septum	Possesses one or more unique antigenic determinants distinct from the epiphyseal cartilages and the nasal septum

Other factors/theories have also been proposed in favor of the use of Functional appliances which include – hyperactivity of the lateral pterygoid and the Cybernetic

growth theory (proposed by Petrovic and associates) and more recently the 'Growth Relativity Theory' (Vodouris & associates) which was proposed after extensive research and shows that significant remodeling in the glenoid fossa and the mandibular condyle takes place in response to the mandibular hyperpropulsers.

However, one question that still looms large is whether the functional appliance therapy actually causes growth modification (beyond genetic potential) or just growth re-direction.

Although answers to these questions are still at large, one thing is clear that growth modification and growth re-direction both take place to varying degrees depending on various factors like the age of the patient, diagnosis and skill of the clinician, etc.

Classification of functional appliances

Myofunctional appliances are classified as:

I. Classification put forth by Tom Graber when functional appliances were removable:

1. Group A-Teeth supported appliances, e.g. catlans appliance, inclined planes, etc.

2. Group B-Teeth/tissue supported, e.g. activator, bionator, etc.

3. Group C-Vestibular positioned appliances with isolated support from tooth/tissue, e.g. Frankel appliance, lip bumpers.

II. With advent of fixed functional appliances, a new classification evolved:

1. Removable functionals, e.g. activator, bionator, frankel, etc.

2. Semi-fixed functional appliances, e.g. Den Holtz, Bass appliances, etc.

3. Fixed functional appliances, e.g. Herbst, Jasper jumper, Churro jumper, Salf springs, adjustable corrector, Eureka spring, mandibular anterior repositioning appliance, (MARA), Klapper super spring, Sabbagh universal spring (SUS).

III. With concept of hybridization by Peter Vig, functionals were classified as:

1. Classical functional appliances, e.g. activator, Frankels appliance, etc.

2. Hybrid appliances, e.g. propulsor, double oral screen, hybrid bionators, Bass appliance.

IV. Classification put forth by Profitt:

1. Teeth borne passive appliances-myotonic appliances, e.g. Andresen/Haupl activator, Herren activator, Woodside, activator, Balter's bionator etc.

2. Tooth borne active applianccs-c-myodynamic appliances, e.g. elastic open activator (EOA), Bimler's appliance, modified bionator, stockfish appliance, etc.

3. Tissue borne passive appliance, e.g. oral screen, lip bumpers, etc.

4. Tissue borne active appliances, e.g. Frankel appliances.

5. Functional orthopedic magnetic appliances.

Cephalometric diagnosis for functional appliance therapy. Cephalometric analysis attempts to define normal/abnormal craniofacial pattern by examining the angular and linear relationships of clearly defined skeletal landmarks.

However, as far as patients for functional appliance therapy are concerned, this seemingly simple approach is complicated by the unpredictability in the nature, dimension, rate and direction of growth. Therefore, cephalometric diagnostic assessment for functional appliance patient include 4 areas of emphasis:

1. Increment of growth direction vector.
2. Assessment of magnitude of growth change.
3. Assessment of constantly changing inclination of upper and lower incisors.
4. Radiographic cephalometries.

The various cephalometric analysis for functional appliance therapy for patients can be divided into 3 groups:

1. Facial skeleton.
2. Jaw bases.
3. Dentoalveolar relationships.

Activator *(Fig. 7.3).* Activator is a loose fitting appliance which was designed by Andreasen and Haupl to correct retrognathic mandible. The present form of the appliance came through various stages of development starting with the concept of 'bite jumping' introduced by Norman Kingsley (1879). He used a vulcanite palatal plate consisting of an anterior inclined plane, which guided the mandible into a forward position when the patient closed on it.

This was followed by Hotz's Vorbissplatte which was a modification of Kingsley's plate and was used to correct retrognathic mandible with deep bite.

Monobloc which was made up of a single block of vulcanite, was used by Pierre Robin to correct the airway obstruction in patients with micrognathia.

Later in *1913* Viggo Andreasen, modified the Hawley's type of retainer, on the maxillary arch, to which he added a lower lingual horse shoe shaped flange which helped to position the mandible forward. He called it the biomechanical working retainer. Later he teamed with Karl Haupl, and developed an appliance which they called as Norwegian appliance and later came to be known as the activator.

Fig. 5.3. The activator (lateral view)

Indications. Actively growing individual with favorable growth pattern are good candidates for the activator therapy. Various types of activators have been devised for the treatment of various conditions like:

- Class II division I malocclusion.
- Class II division II malocclusion.
- Class III malocclusion.
- Class I open bite malocclusion.
- Class I deep bite malocclusion.
- For post-treatment retension.
- Children with decreased facial height.

Contraindications:

- Cannot be used in correction of Class I problems of crowded teeth where there is disharmony between tooth size and jaw size.
- Cannot be used in children with excess lower facial height.
- Cannot be given in cases with lower proclination.
- In case of nasal stenosis.
- In non-growing individuals.

Advantages:

- Uses existing growth.
- Minimal oral hygiene problems.
- Appointments usually short.

Disadvantages:

- Requires good patient co-operation.
- Cannot produce precise detailing and finishing of occlusion.

Philosophy of activator. Various views have been put forward to explain the mode of action of the activator. Some implicate the reflex myotactic activity and isometric contractions while others attribute the results to the viscoelastic properties and stretching of the muscles and soft tissues.

However the basic fact remains that most of the changes are induced by holding the mandible forward and the ensuing reaction of the stretched muscles and soft tissues, transmitted to the periosteum, bones and the teeth.

A restraining effect on the growth of the maxilla and the maxillary dentoalveolar complex is also seen along with the stimulation of mandibular growth and mandibular alveolar adaptation.

Research has also shown favorable changes in the TMJ region.

Components of the activator. It consists of the following elements *(Fig. 7.3. a–c).*

1. Labial bow The wire used is spring hardened 0.9 mm stainless steel. The primary wire element of the activator consists of an upper and/or lower labial bow. It consist of horizontal middle sections, two vertical loops and wire extensions through the canine-deciduous first molar embrasure into the acrylic body.

2. Jack screw Optional (fitted to maxillary arch).

3. Acrylic portion This can be fabricated in cold cure acrylic directly on the models or a wax matrix can be made first and then invested in the flask.

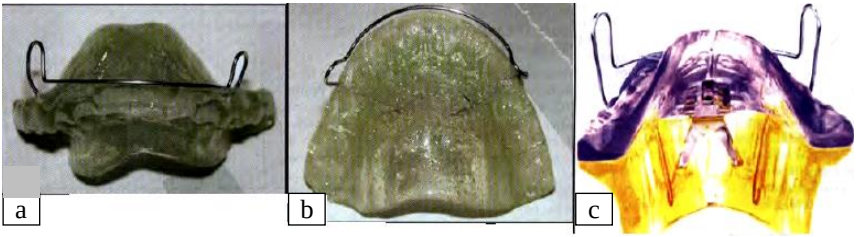

Fig. 5.3. a – The activator (frontal view). b –The activator (superior view). c – The activator (lingual view) showing he jack screw

Construction bite. It is an intermaxillary wax record used to relate the mandible to the maxilla. This is done to improve the skeletal inter-jaw relationship. In most cases bite opening is by 2–3 mm and advancement is by 4–5 mm.

General considerations for construction bite:

1. In case the *overjet* is too large, forward positioning is done in 2–3 stages.

2. In case of forward positioning of the mandible by 7–8 mm, the vertical opening should be slight to moderate i.e. 2–4 mm.

3. If the forward positioning is not more than 3–5 mm then the vertical opening can be 4–6 mm.

Lower construction bite with marked mandibular forward positioning. This kind of construction bite is characterized by marked forward positioning of the mandible with minimum vertical opening. As a rule of the thumb the anterior advancement should not exceed more than 70% of the most *protrusive* position, and vertically it should be within the limits of inter occlusal clearance. This kind of an activator with marked sagittal advancement with minimal bite opening is called H-activator and is indicated in persons with Class II Division I malocclusion with horiozontal growth pattern.

High construction bite with slight anterior mandibular positioning. Here mandible is positioned anterioriy by 3–5 mm only and the bite is opened vertically by 4–6 mm. This kind of activator constructed with minimal sagittal advancement but marked vertical opening is called a 'V' activator and is indicated in Class II Division I malocclusion with vertical growth pattern.

Construction bite without forward positioning of mandible. This done in cases with deep bite, and open bite.

Construction bite with opening and posterior positioning of the mandible. In Class III cases bite is taken after retruding the mandible to a more posterior position. In this a vertical opening of 5 mm and a posterior positioning of 2 mm is required.

Fabrication.

Impressions. Impressions of upper and lower arches are made to construct 2 pairs of models – study models, working models.

Bite Registration:

• Amount of sagittal and vertical advancement is planned.

• Horse-shoe shaped wax block is prepared. It should be 2–3 mm more than the vertical opening planned.

• Patient is asked to practice placement of mandible at the desired position.

• Horse-shoe shaped wax block is placed on the occlusal surface of one of the cast, maxillary or mandibular (maxillary preferred) and is pressed gently to form indentation of the teeth on the wax.

• It is then removed and placed in the patient's mouth and the patient is asked to bite in the proposed sagittal position.

• If found all right it is chilled and once again tried on the cast and then checked again in the patients mouth.

Articulation of the Model. Upper and lower casts are articulated with the construction bite in place. The upper and lower casts are articulated in a reverse direction facing the hinge. This is to get easy access to the palatal surface of the upper and lingual surface of the lower casts.

Preparation of Wire Elements. A labial bow is prepared with 0,8 or 0,9 mm wire. The ends of the wire enter the acrylic body. The labial bow can be active or passive.

Fabrication of Acrylic Portions. Appliance consists of three parts *(Fig. 7.3. a–b):*

• Maxillary part.

• Mandibular part.

• lnterocclusal part.

The maxillary and mandibular parts are same as the acrylic portions of upper and lower Hawley's plate, but these are joined by an interocclusal part which makes this appliance into a single block. The inter occlusal portion has the indentations of upper and lower teeth and caps the lower anteriors, which controls their supra-eruption and proclination.

Trimming of the Activator. Activator therapy aims at providing a good skeletal as well as dentoalveolar relationship of upper and lower arches. However, this is not possible by simply holding/posturing the mandible forward, in a predetermined position, without appropriate guidance for the erupting teeth. Therefore, to achieve a proper three dimensional relationship of teeth, selective trimming of the activator is carried out. Trimming can be done at the time of appliance insertion or as some clinicians suggest, it can be done after about a week's time.

Trimming for Sagittal Control.

a. Class II correction: Trimming is done so as to encourage the mesial movement of the lower molar and distal movement of the upper molar. Therefore, the distopalatal surface in the maxillary and mesio lingual surface in the mandibular posterior segments are trimmed. This pattern of trimming is particularly useful in horizontally growing patients as it tends to open the bite due to molar eruption *(Fig. 7.4. a).*

b. Protrusion of incisors • In this case lingual surfaces of teeth are loaded with acrylic and a passive labial bow is given *(Fig. 7.4. b).*

c. Retrusion of incisors:

• Here the lingual surface is made totally free of acrylic and an active labial bow is given *(Fig. 7.4. c).*

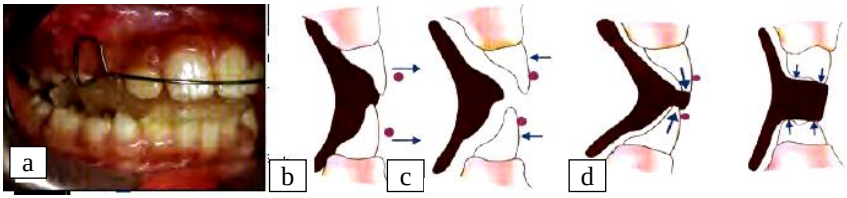

Fig. 7.4. a – Trimming of the activator for Class II correction. Note the lower posterior segment is free to erupt vertically and mesially. thus helping in the correction of deep overbite and Class II relation. b – Activator design for protrusion of incisors. Note the loading of the entire lingual surface and labial bow away from the incisors to encourage labial movement of the incisors. c – Activator design for retrusion of incisors. The labial bow here is active and the lingual surfaces of the incisors are relieved for lingual movement of the teeth. d – Activator design for intrusion of teeth (for anterior intrusion the labial bow is placed below the greatest convexity in the upper and above the greatest convexity in the lower)

Trimming for Vertical Control:

a. lntrusion of teeth *(Fig. 7.4. d)*:

• For this the incisal area is loaded with acrylic.

• Labial bow is placed below the greatest convexity at the incisal area for intrusion.

• In case of intrusion of posteriors load the surfaces of the teeth with acrylic

b. Extrusion of teeth *(Fig. 7.5. a)*

• Here the lingual surface is loaded above the area of greatest convexity in the maxilla and below the area of greatest convexity in the mandible

• Also the labial bow can be placed at the gingival 1/3 i.e. below the greatest convexity

• In case of posterior extrusion the lingual surfaces below the greatest convexity are loaded.

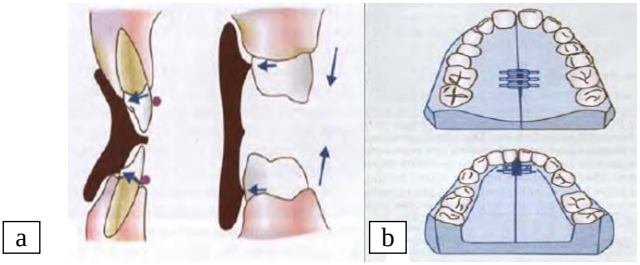

Fig. 7.5. *a – Activator design for extrusion of teeth. (I) Anteriors. (II) Posteriors. b – Activator with jack screw, for transverse control*

For Transverse Control *(Fig. 7.5. b).* Jack screw is incorporated into the activator for expansion (trasnverse control) as and when required.

Management of the appliance. The patient is demonstrated to place and remove the appliance in mouth. The appliance is to be worn 2 to 3 hours during

the day for the first week. During the second week the patient sleeps with the appliance in mouth and wears it for 1–3 hours each day.

The appliance is checked during the third week to evaluate the trimming.

If the patient is wearing the appliance without any difficulty and following the instructions, checkup appointments are scheduled every 6 weeks.

Bionator (*Fig. 7.6. a–f*). The bulkiness of the activator and its limitation to night-time wear was a major deterrent in its greater use by clinicians to obtain maximum potential of functional growth guidance. The appliance was too bulky for day-time wear. Moreover, during sleep, the function is minimized or virtually nonexistent.

This led to the development of the **bionator**, a less bulky appliance. Its lower portion is narrow, and its upper component has only lateral extensions, with a crosspalatal stabilizing bar. The palate is free for proprioceptive contact with the tongue and the buccinator wire loops hold away the potentially deforming muscles. The appliance developed by **Balters** in 1960, can be worn all the time, except during meals.

Philosophy of bionator. According to Balters, "the equilibrium between the tongue and the circumoral muscles is responsible for the shape of the dental arches and that the functional space for the tongue is essential for the normal development of the orofacial system" e.g. posterior displacement of the tongue could cause Class II malocclusion. Taking into consideration the dominant role of the tongue, Balters designed an appliance, which could take advantage of tongue posture. Thus he constructed an appliance whereby the mandible was positioned anteriorly, with the incisors in an edge to edge position. This forward positioning brought the dorsum of the tongue in contact with the soft palate and helped accomplish lip closure.

Thus the principle of bionator is not to activate the muscles but to modulate muscle activity, thereby enhancing the normal development of the inherent growth pattern and eliminate abnormal and potentially deforming environmental factors.

Bionator types. Three basic constructions are common in bionator:

- Standard appliance.
- Open-bite appliance.
- Class III or reverse bionator.

Standard Appliance *(Fig. 7.6. a–c).* It consists of a lower horse-shoe shaped acrylic lingual plate extending from the distal of the last erupted molar to the corresponding point on the other side. For the upper arch the appliance has only posterior lingual extensions that cover the molar and premolar regions. The anterior portion is open from canine to canine (*Fig. 7.6. a).* The upper and lower parts, which are joined interocclusally, extend 2 mm above the upper gingival margin and 2 mm below the lower gingival margin.

The palatal bar is formed of 1.2 mm hard stainless steel wire extending from the top edges of the lingual acrylic flanges in the middle area of the deciduous

first molars *(Fig. 7.6. b).* The palatal bar forms an oval, posteriorly directed loop that orients the tongue and mandible anteriorly to achieve a Class I relationship.

The labial bow is made from 0.9 mm hard stainless steel. It starts above the contact point between the canine and deciduous upper first molar/premolar. It then extends vertically, making a rounded 90° bend to the distal along the middle of the crowns of the posterior teeth and extends as far as the embrasure between deciduous 2nd molar and permanent 1st molar. It then makes a gentle downward and forward curve running anteriorly till the lower canine. From there, it forms a sharp curve extending obliquely till the upper canine, bends to a level at approximately the incisal third of the incisors and extends to the canine on the opposite side *(Fig. 7.6. c).*

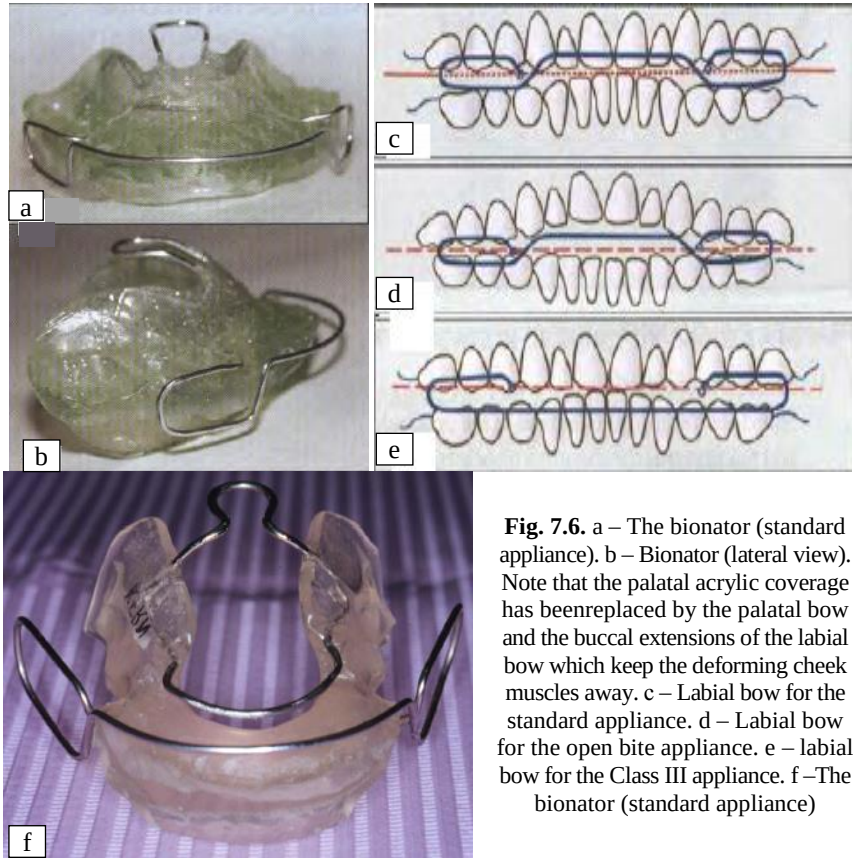

Fig. 7.6. a – The bionator (standard appliance). b – Bionator (lateral view). Note that the palatal acrylic coverage has beenreplaced by the palatal bow and the buccal extensions of the labial bow which keep the deforming cheek muscles away. c – Labial bow for the standard appliance. d – Labial bow for the open bite appliance. e – labial bow for the Class III appliance. f –The bionator (standard appliance)

Open Bite Appliance. This is used to inhibit abnormal posture and function of the tongue. The construction bite is kept as low as possible with acrylic bite blocks between the posterior teeth to prevent their extrusion. The acrylic portion

of the lower lingual part extends onto/up to the upper incisor region as lingual shield, to prevent tongue movements. The palatal bar has the same configuration. The labial bow is quite similar with the exception that the wire runs approximately between the incisal edges *(Fig. 7.6. d).*

Class III or Reverse Bionator. This type of appliance is used to encourage the development of maxilla. The bite is taken in most possible retruded position, to allow labial movement of the maxillary incisors and reciprocally a slight restrictive effect on the lower arch. The bite is opened about 2 mm only in the interincisal region.

The palatal bar configuration runs forward instead of posteriorly, with the loop extending as far as the deciduous 1st molar or premolar. The labial bow runs in front of the lower incisors rather than in front of the upper incisors *(Fig. 7.6. e).*

Indications for bionator therapy. Bionator is indicated for the treatment of Class II Division I malocclusion in the mixed dentition using the standard bionator under the following conditions:

• Well aligned dental arches.
• Functional retrusion.
• Mild to moderate skeletal discrepancy.
• No evidence of labial tipping seen.

Contraindications:

• Class II relationship caused by maxillary prognathism.
• Vertical growth pattern.
• Labially tipped lower incisors.

Advantages of bionator:

1. Appliance is less bulky.
2. Can be worn full time, except during meals.
3. Appliance exerts a constant influence on the tongue and perioral muscles.

Disadvantage of appliance. The main disadvantage lies in the difficulty of correctly managing it.

The Frankel function regulator (FR) *(Fig. 7.7).* Function regulator appliances were developed by Rolf Frankel (Germany). Frankel believed that the active muscle and tissue mass i.e., the buccinator mechanism and the orbicularis oris complex have a major role in the development of skeletal and dentofacial deformities.

Hence he developed function regulators as orthopedic exercise devices, to aid in the maturation, training and reprogramming of the orofacial neuromuscular system.

Frankel philosophy:

1. Vestibular area of operation:

– Shields of the appliance extend to the vestibule and this prevents the abnormal muscle function.

2. Sagittal correction via tooth borne maxillary anchorage:

– Appliance is fixed on the upper arch by grooves mesial to the 1st permanent molar and distal to the canine in the mixed dentition period.

– Presence of the lingual pad acts as proprioceptive stimulus and helps in the forward posturing of the mandible.

3. Differential eruption guidance:

– Frankel is placed on the upper teeth.

– Mandibular posterior teeth are free to erupt and their unrestricted upward and forward movement contributes to both vertical as well as horizontal correction of the malocclusion.

4. Periosteal pull by buccal shields and lip pad:

– Presence of buccal shields and lip pads exert the periosteal pull which helps in bone formation and lateral expansion of the maxillary apical base.

5. Minimal maxillary basal effect:

– Downward and forward growth of maxilla seems to be restricted, even though lateral maxillary expansion in seen.

Mode of action of FR:

1. Increase in transverse sagittal direction

– by use of buccal shields and lip pads

2. Increase in *vertical direction*

– by allowing the lower molar to erupt freely because appliance is fixed to the upper arch

3. Muscle adaptation

– The form and extension of the buccal shields and lip pads along with the prescribed excercises corrects the abnormal perioral muscle activity.

Oral exercises with Frankel:

– Frankel-full time wear appliance.

– Lips to be closed at all times or keep a paper between the lips.

– Swallowing, speaking, etc. with the appliance in mouth, itself serves as an exercise.

Types of function regulator:

1. FR l-used for Class I and Class II, Division I.

FR l a -used for Class I, moderate crowding and deep bite.

PR l b-used for Class II Division I overjet less than 7 mm.

FR l c-used for Class II Division I overjet more than 7 mm.

2. FR II-used for Class II Division II and Division I *(Fig. 7.7. a-b).*

3. FR Ill-used for Class III.

4. FR IV-used for cases with open bite and bimaxillary protrusion.

5. FR V-FR with headgear.

Fubrication of the function regulator

Parts of the appliance *(Fig. 7.7.a–b, 7.8. a–d):*

- Acrylic part: buccal shields, lip pads, lower lingual pads.

Wire parts: palatal bow, labial bow, canine extensions, upper lingual wire (only in FR II), lingual cross over wire, lip pads, lower lingual springs.

Impressions. The impressions should reproduce the whole alveolar process to the depths of the sulci, including the maxillary tuberosities. The soft tissues and the muscle attachments should not be distorted. A custom tray can also be fabricated based on the study models, if desired. Since the appliance is anchored in

the maxillary arch between the deciduous second molar and the permanent first molar, separators should be placed between these teeth prior to impressions; otherwise, disking of the distal surface of the primary second molar, can also be done after fabrication of appliance.

Fig. 7.7. Frankel II (lateral, frontal and superior view showing parts of the appliance)

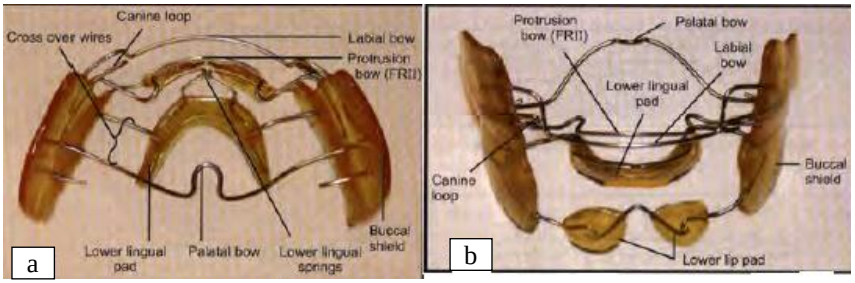

Fig. 7.7. a – Frankel II (superior view showing parts of the appliance.
b –Frankel II (frontal view showing parts of the appliance)

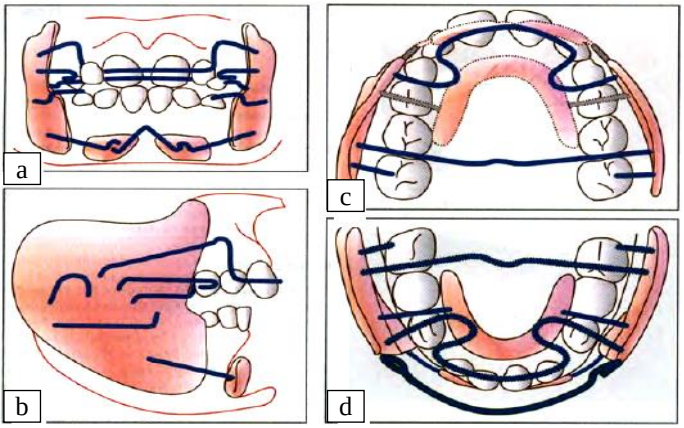

Fig. 7.8. a–d – Basic components as described for a FA II appliance.
a – Buccal shields, lip pads, labial wire. b – Buccal shields, lip pads, canine clasp, labial arch and labial arch loop. c – Appliance on the maxillary cast.
d – Lingual acrylic pad, lingual wires and lingual springs

Working model pouring and trimming:

– Model base must extend away from alveolar process by at least 5 mm.

– Correct model trimming is necessary before appliance fabrication.

– The desired amount of stone to be cut is outlined with a pencil before trimming.

– Then it should be cut with a round bur.

– Final detailing is done with plaster knife.

– No trimming required for buccal shields on mandible.

– But trimming is required in the maxillary buccal shield area and lower lip pad area.

– Care must be taken not to disturb the muscle attachments.

Trimming for lip pads:

– 5 mm from greatest curvature of *alveolar* base to ensure optimum extension.

– Lower relief should be 12 mm below gingival margin.

Trimming for buccal shield:

– Su1cular depth must be 10–12 mm above the *gingival* margin of posterior teeth.

– Region next to the muscle attachment over the deciduous 1st molar and the superior limit of the lateral incisor depression must be well defined.

– This allows optimal extension of buccal shields for deposition of bone.

Construction bite *(Fig. 7.9. a).* For minor sagittal problems, the construction bite is taken at and end-to-end incisor relationship, with the mandible position forward not more than 2.5 to 3 mm. A clearance of at least 2.5 to 3.5 mm in the buccal segments is necessary to allow the crossover wires to pass through in the Frankel appliance.

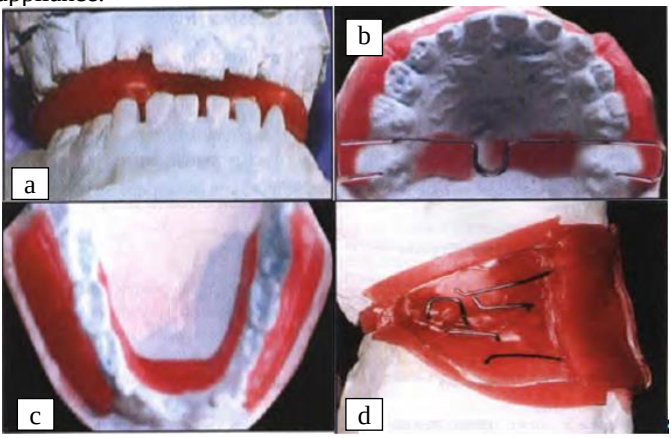

Fig. 7.9. a – Construction bite for FR II. Note that the dental midline deviation should not be corrected in the construction bite unless the skeletal midlines are deviating as well. b – Wax relief in the maxillary arch. Note the configuration of the palatal bow as well. c – Wax relief on the mandibular cast. d – Complete wax up of the Frankel. Ready for the fabrication of the buccal shields in cold cure acrylic

Dental midline discrepancies should not be corrected in the bite by manipulation during forward posturing. The construction bite should be checked on the casts and the cases should be mounted with the bite.

Wax relief *(Fig. 7.9. b–d).* Relief is placed such that the buccal shields and lip pads stay away from teeth and tissues to achieve the desired expansion. Thickness of wax depends on the expansion required but should not exceed 4–5 mm in the tooth area and 2.5–3 mm in the alveolar area in the maxilla *(Fig. 7.9. b).* In the mandible, only 0.5 mm of relief is given *(Fig, 7.9. c).* Thickness of relief wax is greater in maxilla because of arch narrowing in case of Class II Division I malocclusions.

Wire components (FR I b) *(Fig. 7.7. a–b, 7.8. a–d).* Consists of:

• Stabilizing wires.

• Tooth moving wires.

Lower Lingual Support Wire:

– Made of 1.25 mm wire

– Can be one unit or 3 separate parts

– Horizontal reinforcing wire element contours to the lingual apical base 1–2 mm away from the mucosa and 3–4 mm below the gingival margin so as to permit adding acrylic to the pad.

– Cross over wire pass between deciduous first and 2nd molar

– Ends are then bent at 90° to insert into the buccal shields.

The ends must be parallel to each other and the occlusal plane to allow for advancement of the anterior section later if needed.

Lower Lingual Springs:

–Made of 0.8 mm wire.

– Right above the cingula the wire is curved about 3 mm below the incisal margin.

– Function-is to prevent extrusion of lower incision.

– Should not be active only passive.

– If tooth movement is required the spring is made active.

Lower Labial Wires:

– Made of 9 mm wire

– Acts as a skeleton for lower lip pads

– It can be one piece or three piece

– It should be 1 mm away from the tissue.

– Wire frame-work should be 7 mm below the gingival margin

– Middle part should be inverted 'V' shaped for labial frenum relief.

Palatal Bow *(Fig. 5.9. b–d):*

– Made of 1 mm wire.

– Should have a curve in the center for lateral expansion.

– Wire should pass into the groove between deciduous 2^{nd} and 1^{st} permanent molar.

– Wire emerges out of wax relief makes a loop in the buccal shield and lies between maxillary1^{st} molar buccal cusp ending in the fossa as an occlusal rest.

– This provides a positive seat to the FR as well as prevents eruption of the upper 1st permanent molar.

Labial Bow:

– Made of 0,9 mm wire

– Originates in buccal shield curves upwards and lies in the depression between canine and lateral. It is in the middle of labial surface of incisors and leaves the acrylic with slight bend towards the sulcus.

– Should be 2 mm away from mucosa

– Permits canine eruption and expansion without contacting the labial wire

– Loops should be wide enough to allow activation later to close anterior space if required.

Canine Loops (For FR la):

– Made of 0,9 mm.

– Embedded in buccal shield at occlusal plane level

– Turned sharply towards gingival margin of upper deciduous 1st molar and fit in embrasure between deciduous first molar and canines.

– Wire wraps around the lingual surface of the canine emerges labially at the canine-lateral embrasure curves distally over the canine cusps. Free ends can be bent occlusally if required.

Canine Loops (For FR 11):

– Made of 0,8 mm wire.

– Originate in buccal shield

– Contact canines on buccal surface as recurved loop.

– Serves as extension of buccal shield in the canine area whim is normally narrowed by the peri-oral muscles.

– Should be 2–3 mm away from canines to prevent restrictive muscle function.

Upper Lingual Wire (Protrusion Bow):

– Made of 0.8 mm wire

– Mostly seen in FR II and FR III.

– Lingual bow behind the maxillary incisors serves to maintain pro-functional appliance alignment achieved and also stabilizes the FR by locking it on to the maxillary arch.

– 0.8 wire is used if the centrals are retroclined, but if the anterior alignment is already achieved a stiffer wire can be used i.e. 0.9 mm wire.

– It originates in the vestibular shield and passes to the lingual through the canine-deciduous 1st molar embrasure.

– Wire forms loops at the palatal mucosa and curve vertically to contact the incisor at the embrasure between canine and lateral incisor.

Acrylic parts:

– After the wires are fabricated and adapted they are secured in place with sticky wax.

– The lip pad, buccal shields, lower lingual pads are fabricated in cold cure acrylic.

– Total thickness of the acrylic should not exceed 2.5 mm.

– Lip pads look like parallelogram (tear drop shape in longitudinal cross-section).

– Lip pads should be 5 mm from the gingival margin.

– Presence of lip pads eliminates mentalis hyperactivity and abnormal functional lip trap, thereby helps correcting the overjet.

– Buccal shields should extend past the canine deciduous first molar embrasure to the middle of the canine.

Components of various FR appliances:

FR la is not popular. Replaced by **FR Ib:**

– Used for Class I moderate crowding and deep bite.

– Lingual wire loops are given instead of acrylic lingual pads to posture the mandible forwards.

– The cross over wire (passing between upper and lower occlusal surface) is an extension of the lingual loops.

FR lc:

– used in Class II Division I with over jet greater than 7 mm.

– It has been observed that posturing the mandible forward into a Class I relationship and eliminating excessive overjet in one step for a Frankel appliance is neither feasible nor necessary. Because tissue response is less favorable and there is increased patient discomfort or compliance.

– So mandibular protraction is done in 2 or 3 steps.

– Horizontal and vertical cuts are made on the buccal shield and then made to slide along the hanger *(Fig. 7.10)*

– The cuts are then filled with cold cure acrylic.

– FR I c is seldom used because FR I b and FR II can be modified in the same way.

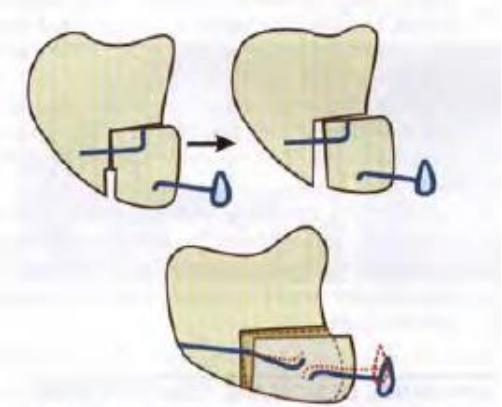

Fig. 7.10. Mandibular advancement done in stages. Horizontal and vertical cuts are made in the acrylic and the lower lingual pad and lip pads are advanced. The gap is later filled with acrylic

FR II:

– Used for Class II Division II and Division I.

– Protrusion bow is made unlike in FR I.

– Canine loops.

• It is only a recurved loop.

• It originates in the buccal shield and contacts the buccal surface of the canine as a recurved loop.

• It shields the canine against the buccinator action.

• It is placed 2–3 mm away from the deciduous canines.

FR III:

– Used for Class III correction.

– Lip pads are in the maxillary arch.

– Labial bow resting against mandibular teeth. Protrusion bow is on the upper teeth and is made of 0.8 mm wire for forward movement of maxillary incisiors if desired.

– The occlusal rest is on the mandibular molar unlike in FRII where it is on the maxillary molar.

FR IV:

– Used for open bite and bimaxillary protrusion.

– Has no canine loops.

– Has no protrusion bow.

– Four occlusal rests present i.e, on deciduous 1st molar and permanent 1st molar on each side to prevent eruption of posterior teeth.

– Palatal bar resembles FR III i.e. it does not contact the teeth.

– The buccal shield in FR IV should be wafer thin to enable lip closure and exercise without which the appliance will be a failure.

FR V:

– Frankel appliance used along with head gear.

Treatment timing. The best therapeutic effect of the Frankel appliance is achieved during the late mixed and transitional dentition period, when both the soft and hard tissues are undergoing their greatest transitional changes.

Treatment for Class III and open bite cases should usually start sooner than for Class II problems.

Twin-block *(Fig. 7.11).*

Evolution of twin-block. The twin block appliance was developed by Clark in 1977, and it consists of an upper and lower device with simple bite blocks that engage on occlusal inclined planes.

The appliance became popular due to a number of advantages *over* other functional appliances namely:

1. The functional mechanism is very similar to that of the natural dentition.

2. The occlusal inclined planes give greater freedom of movement in lateral and anterior excursion and cause less interference with normal function.

3. Appearance is noticeably improved.

4. Less bulk, therefore, better patient compliance.

5. Can be used in later stages of growth (late mixed dentition/early permanent dentition).

6. The appliance can be cemented in mouth, without disrupting the normal oral functions, to improve patient compliance.

7. Absence of lip pads and buccal shields, allow patient a much better comfort, however, modifications containing lip pads can be incorporated as and when required.

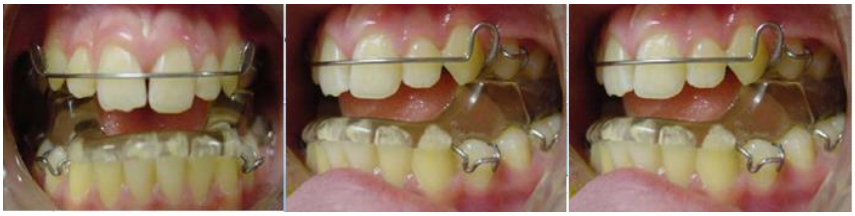

Fig. 7.11. Patient with a twinblock

Development of twin-block. The twin block appliance evolved in response to a clinical problem that presented when a young patient, the son of a dental colleague, fell and completely luxated an upper central incisor. The incisor was reimplanted and a temporary splint was constructed to hold the tooth in position.

After 6 months with a stabilizing splint, the tooth had partially reattached, but there was evidence of severe root resorption and the long-term prognosis for the reimplanted incisor was poor.

The occlusal relationship was Class II Division I with an overjet of 9 mm and lower lip was trapped lingual to the upper incisors. Adverse lip action on the reimplanted incisor was causing mobility, and root resorption. To prevent the lip from trapping in the overjet it was necessary to design an appliance that could be worn full time to posture the mandible forward. At that time no such appliance was available and simple bite blocks were therefore, designed to achieve this objective. The appliance mechanism was designed to harness, the forces of occlusion to correct the distal occlusion and also reduce the overjet without applying direct pressure to the upper incisors. The first twin block appliances were fitted on 7th September 1977. The upper and lower bite blocks engaged mesial to the 1st permanent molar at 90° to the occlusal plane, when the mandible postured forward. *This* positioned the incisors edge-to-edge with 2 mm *vertical* separation to hold the incisors out of occlusion. The patient had to make a positive effort to posture the mandible forward to occlude the bite blocks in protrusive bite.

Case selection for twin-block appliance. Case selection for clinical use of twin-block should, display the following criteria:

1. Angle's Class II Division I malocclusion with good arch form.
2. A lower arch that is uncrowded or decrowded and aligned.
3. An upper arch that is aligned or can be easily aligned.
4. An overjet of 10–12mm and a deep overbite.

S. A full unit distal occlusion in the buccal segments.

6. On examination of models in occlusion with the lower model advanced to correct the increased overjet, the distal occlusion is also corrected and it can be seen that a potentially good occlusion of the buccal teeth will result.

7. On clinical examination the profile should be noticeably improved when the patient advances the mandible voluntarily to correct the overjet.

8. To achieve a favorable skeletal change, during treatment, patient should be actively growing. A more rapid growth response may be observed when the treatment coincides with the potential growth spurt.

Appliance design and construction

Evolution of the appliance design *(Fig. 7.11, 7.11. a–b).* The present form of the appliance evolved over a period following clinical experience with the appliance. The earliest design of the twin-block consisted of:

1. A midline screw to expand the upper arch.
2. Occlusal bite block (at 90° to occlusal plane).
3. Clasps on upper molars and premolars (Adams' clasp),
4. Clasps on lower premolars and incisors.
5. Springs to move the individual teeth.
6. Provision for extraoral traction in some cases. (esp. maxillary protrusion cases).

Delta clasps. After initial use of the Adams' clasp, Clark introduced the delta clasp in 1985.The basic premise was to reduce the incidence of breakages (as seen with the Adams' clasp) due to repeated adjustments and consequent metal fatigue.

Labial bow. The use of labial bow in the upper plate was discontinued after it was noticed that it causes unwanted retroclination of upper incisors with consequent deepening of the bite. This is turn, limited the scope of mandibular correction possible. However, in certain cases with severe upper proclination, labial bow in the upper arch may be placed.

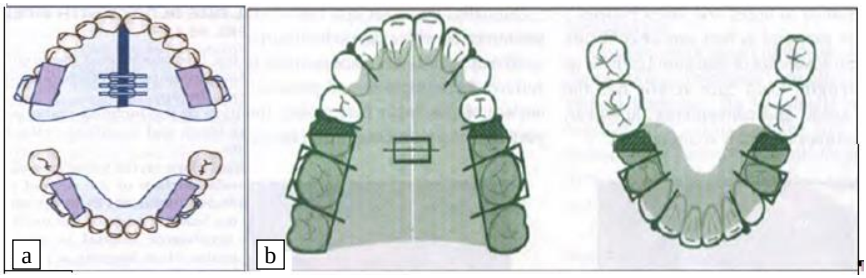

Fig. 7.11. a – The twinblock appliance. b – Clark's standard twinblock appliance

Base plate. The design is similar to upper and lower Hawley's plate and can be prepared in heat cure or cold cure acrylic. The main advantage of heat cure acrylic is its additional strength. Cold cure acrylic has the advantage of speed and convenience, however, strength and accuracy is slightly compromised.

Occlusal bite blocks with inclined planes. Position of the bite blocks and the angulation of its inclined planes are important factors in the success of the twin-block therapy.

The position of the inclined plane is determined by the lower block and is critical in the treatment of deep overbite.

The inclined plane on the lower bite-block is angled from the mesial surface of the second pre-molar or second deciduous molar at 70° to the occlusal plane. This places the leading edge of the inclined plane on the upper appliance mesial to the lower first permanent molar, thus keeping a provision for the unhindered eruption of the lower 1st permanent molar. Mesially, the lower bite block extends up to the canine region with a flat occlusal surface.

The upper inclined plane is angled from the mesial surface of the upper second premolar to the mesial surface of the upper first molar. The flat occlusal portion then passes distally over the remaining upper posterior teeth in a wedge shape, reducing m thicken as it extends distal1y.

The height of the bite blocks is determined by the vertical opening planned and recorded in the construction bite. For a twin block therapy, it is recommended that the vertical opening in the construction bite be beyond the free way space. This implies that the height of the bite block should be 4–6 mm so that the mandible does not go back even in physiologic rest position.

Angulation of the inclined planes. Initially, inclined planes were at 90° to occlusal plane. However, adjustment to this sort of inclined plane was difficult for a lot of patients.

Therefore, for patient convenience inclined planes were reduced to 45° but since, this angulation caused equal vertical and horizontal movement, the angulation was further changed to 70°, so that more horizontal vector of force would be produced.

Nevertheless, the inclined plane angulation can vary between 45° and 70° depending upon the patient comfort levels.

Construction bite. Bite registration for construction of twin-blocks for class 11 division I malocclusion. Construction bite for twin-block can be taken is the conventional manner, by means of an inter-occlusal wax bite, as described earlier, for the activator, or by the use of an 'Exactobite'.

Exactobite or project-bite gauge is a horse-shoe shaped device with an anterior handle with various grooves, designed for accurate control m registering a protrusive bite for construction of twin block. The blue bite gauge registers 2 mm vertical clearance between the incisal edges of the upper and lower incisors, which are in appropriate interincisal clearance for bite registration in most Class II Division I malocclusions with increased overbite.

In a Class 11 Division I malocclusion a protrusive bite is registered to reduce the overjet and distal occlusion on average 5–10 mm on initial activation, depending on the freedom of movement in protrusive functions. The length of the patient's protrusive path is determined by recording the overjet in centric occlusion and fully protrusive occlusion. The activation should not exceed 70 % of the maximum protrusive path.

In growing child, with an overjet of up to 10 mm, provided the patient can posture comfortably forwards, bite may be activated up to edge-to-edge on the incisors with a 2 mm interincisal clearance. Larger overjet requires partial correction.

It is best, first to rehearse the procedure of bite registration with the patient using a mirror. The patient is instructed to close correctly into the bite gauge before applying the wax. Once the patient understands, what is required, softened wax is applied to the bite gauge from a hot water bath.

The clinician can then place the bite gauge in the patient's mouth to register the bite. Midlines should be coincident, however, if dental mid lines are deviating, skeletal midlines should be taken into consideration.

One important aspect of the construction bite for the twin-block appliance is to establish the correct vertical dimension, The bite should be open slightly beyond the clearance of the free way space to encourage the patient to close into the appliance rather than allow the mandible to drop out of contact into rest position.

Hence, an interincisal clearance of about 2–3 mm is established, which is equivalent to an approximately 5–6 mm clearance m the 1st premolar region and about 3 mm clearance distally m the molar region.

This amount of vertical clearance ensures that the mandible does not drop back at rest and that enough space is available for the vertical development of the posterior teeth to red uce the over bite.

Establishing the correct vertical dimensions-the intergingival height. A simple guide is used to establish the correct vertical dimension during the twin block phase of treatment. The intergingival height is measured from the gingival margin of the upper incisor to the ginglval margin of the lower incisor when the teeth are mocclusion.

The 'comfort zone' for intergingival height for adult patients is about 17–19 mm. This is equivalent to combined heights of the upper and lower incisors minus an overbite within the range of normal. Patients whose intergingival height varies significantly from comfort zone are at a greater risk of developing TMD. This applies both to the patients with a deep overbite whose intergingival height is reduced and to patients with an anterior open bite who have an increased intergingival height.

The intergingival height is a useful guideline to check progress and to establish the correct vertical dimensions during treatment. Measurement of intergingival height is done by using a mm ruler and divider or with a Vernier scale to measure the distance between the upper and lower gingival margins.

In twin block treatment the correct intergingival height is achieved with great consistency. Deep overbite may be corrected to an intergingival height of 20 mm to allow for a slight settling in with a resultant overbite increase after treatment.

In the younger patient's a range of 15–17 mm is normal and allowance should be made for the diminutive height of the clinical crowns.

Fitting twin·blocks:

Instructions to patient. Patient motivation is an important factor in all removable appliance therapy. The process of patient education and motivation continues when the patient attends to have twin block fitted. It is often helpful to the patient if the clinician demonstrates twin blocks on models to confirm that it is a simple appliance system and is easy to wear with no visible anterior wires.

Simply biting the blocks together guides the lower jaw forward to correct the bite. The appliance system is easily understood even by young patients, who see that biting the blocks together corrects the jaw position. It is important to emphasize positive factors and to motivate the patient before treatment.

The patient is shown how to insert the twin blocks with the help of a mirror, pointing out the immediate improvement in facial appearance when the twin blocks is inserted and explaining that the appliance will produce this change, in a few months, provided they are worn full time. A removable appliance only corrects the teeth when it is in mouth, and not in the pocket. Both appliances must be worn full time, especially during eating with sole exception being removed for cleaning and during swimming and contact sports.

At first the appliance may feel large in the mouth, but within a few days, it will be very comfortable and easy to wear. Twin blocks cause much less interference with speech than other, one piece functional appliances. For a first few days, speech will be affected, but will improve and should return to normal within a week.

As with any new appliances it is normal to expect a little initial discomfort. But it is important to encourage the patient to preserve and keep the appliance in mouth at all times except for hygiene process.

The patient should be advised to remove the appliance during eating for first few days. Then it is important to learn to eat with the appliance. The force of biting on the appliance corrects the jaw position, and learning to eat with the appliance is important to accelerate the treatment. In a few days patient should be eating with the twin block and within a week should be more comfortable with the appliance in the mouth than they are without it.

It is necessary to check the initial activation and confirm that the patient closes consistently on the inclined plane with the mandible protruded in new position. The overjet is marked with a mandible fully retruded and this measurement should be recorded and checked at every visit to monitor progress.

Stages of Treatment. Twin-block treatment is described in two stages. Twin blocks are used in the active phase to correct the anteroposterior relationship and establish the correct vertical dimension. Once this phase is completed, the twin-blocks are replaced with an upper Hawley's type of appliance with an anterior inclined plane which is then used to support the corrected position as the posterior teeth settled fully into the occlusion.

Fixed functional appliances.

Herbst Appliance. This device *(Fig. 7.12)*, developed in the early 1900s and reintroduced in the 1970s by Pancherz, can be either a fixed or removable appliance. The maxillary and mandibular arches are splinted with frameworks that usually are cemented or bonded but can be removable, and connected with a pin-and-tube device that holds the mandible forward. Occasionally a modification of this appliance is superimposed on traditional fixed appliances. Jaw position is controlled by a pin and tube apparatus that runs between the arches. Pressure against the teeth can produce significant tooth movement in addition to any skeletal effects and, even if the appliance is fixed in place, the amount of skeletal vs. dental change is affected by patient compliance.

Disadvantages:

• Appliance is prone to breakage.
• Lateral movement is restricted

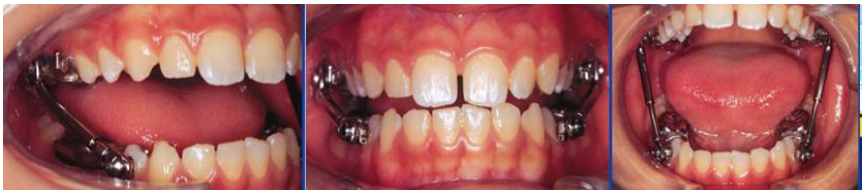

Fig. 7.12. Banded Herbst appliance

Jasper jumper *(Fig. 7.13).* The Jasper Jumper (American Orthodontics) consists of a heavy coil spring encased in vinyl coating. The flexible springs are attached to the maxillary 1st molar posteriorly and distal to the mandibular canine, either directly onto the lower arch wire or by means of an out-rigger.

Fig. 7.13. Jasper Jumper in mouth

Indications:

• Dental Class II malocclusion
• Deep bite with retroclined mandibular incisors.

Contraindications:

• Dental and skeletal open bites.
• Minimum buccal vestibular space.
• Vertical growth pattern with increased lower facial height.
• Cases prone to root resorption

Advantages:
– Ease of insertion and activation
– Generation of intrusive forces on molars and incisors.
Disadvantages*:*
– Frequent breakages
– Compromised oral hygiene

The mandibular anterior repositioning appliance. (MARA) *(Fig. 7.14).* The MARA consists of cams made from 0.060 square wire attached to tubes (0.062 square) on upper first - Externally perceivable bulge in the cheeks molar bands or stainless steel crown. A lower first molar crown has a 0.059 arm projecting perpendicular to its buccal surface, which engages the cam of the upper molar.

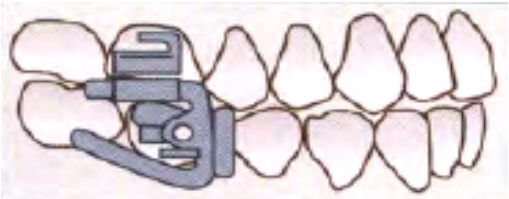

Fig. 7.14. The mandibular anterior positioning appliance

The appliance is adjusted so that when patient closes the mouth, the cam on upper molar guides and repositions the mandible into a Class I relationship.

Its main disadvantage is that temporary stainless steel crowns are needed on all first molars.

Indication: Skeletal Class II with mandibular deficiency.

Contraindications:
• Cases prone to root resorption
• Dental and skeletal open bite
• Vertical growth pattem.

Biopedic appliance *(Fig. 7.15).* Developed by Jay Collins in 1997 consists of buccal attachment on upper and lower molar crowns which includes the standard edge wise tubes and a large 0.70 inch molar tube. Large rods pass through these tubes. The mandibular rod inserts from mesial of the molar tube and is fixed at the distal by its screw clamp. Activation is done by moving the rod mesially.

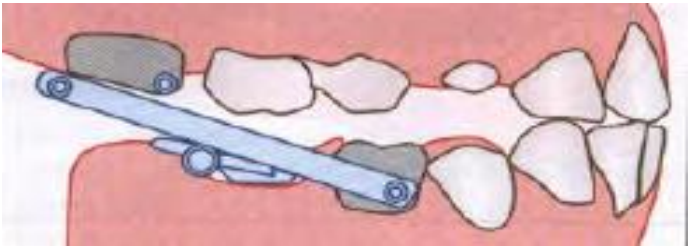

Fig. 7.15. The biopedic appliance

Maxillary rod inserts from distal of the tube and is fixed at the mesial by screw clamp. Two rods are connected by a rigid shaft and have pivotal regions at their ends.

Eureka spring *(Fig. 7.16).* Developed by De-vincenzo in 1996. One of the first inter arch appliances to utilize the compressive forces.

Advantages:

– Good patient acceptance

– Can be used for Class II and Class III correction as well as in conjunction with extraoral force.

– Possibility of alteration in the amount and direction of force during treatment.

– Components are available separately

– Significantly less expensive than other appliances.

Disadvantages:

– Technique sensitive insertion procedure

– Frequent breakages of interval spring

– Less force levels than forsus and twin force corrector.

– Tissue irritation.

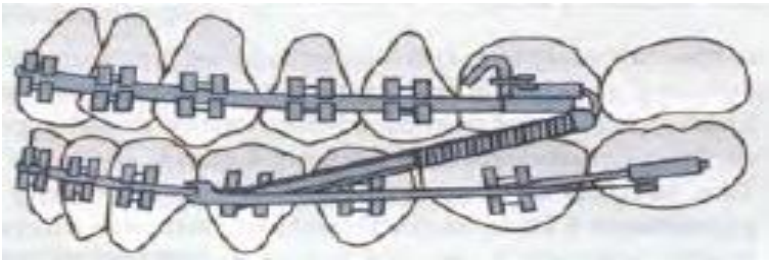

Fig. 7.16. Eureka spring

Saif spring *(Fig. 7.17).* Introduced by Arrnstrong, consists of two Ni-Ti coil springs, one inside the other with soldered loops on both ends. Used for Class II and Class III correction and available in 2 lengths: 7 and 10 mm. Delivers the force of 200–400 gm.

Disadvantage:

– Bulky, therefore oral hygiene maintenance is problem.

– Large inventory

– Oral hygiene is compromised

– Breakages are often seen.

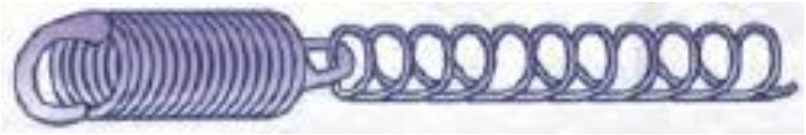

Fig. 7.17. Saif spring

The Klapper super spring *(Fig. 7.18).* Introduced by Lewis Klapper in 1997. Resembles jasper jumper except that instead of coil spring, cable is used. In 1998, the cable was wrapped with a coil and Klappcr super spring II came into being.

Fig. 7.18. Klapper super spring

Advantages: More vertical force vector, therefore useful for intrusion.

Disadvantages: Unlike, jasper Jumper it enters the molar tube from mesial and requires special molar tube for engagement.

Forsus fatigue resistant device *(Fig. 7.19. a–b).* The appliance consists of:

• Spring module.

• L bail pin.

• Push rod installation. The push rods are available in following sizes 25, 29, 32 and 35 mm which are available for right and left side.

• The L pin with the spring module is attached to upper first molar after selecting the appropriate push rod.

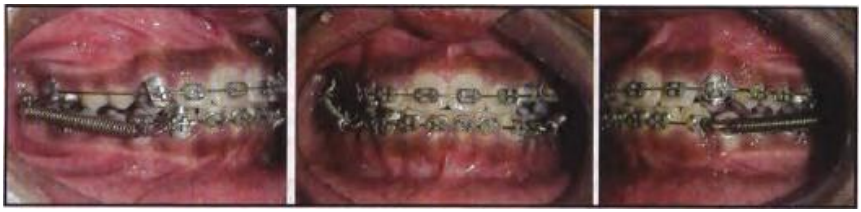

Fig. 7.19. a – Forsus fatigue resistance device

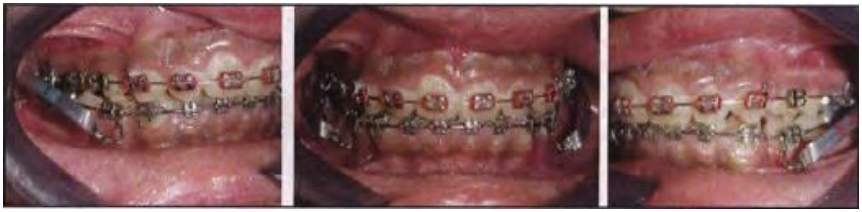

Fig. 7.19. b – Forsus flat spring

Its loop is attached to arch wire between the cuspid and first bicuspid and the other end is inserted into the compressed spring module.

Advantages

• Unequal push rods can be used for midline correction

• Spring can be reactivated by placing crimp split ring bushings on push rod

• Relative ease of installation and removal.

Sabbagh universal spring (SUS) (*Fig. 7.20***).** It is the latest interarch compressive spring to be introduced and has a number of unique features as:

– Slotted screw for partial adjustment of distal aspect of the plunger assembly (up to 4 mm).

– The second coil spring inserted at the time of placement which in combination with the internal spring permits a greater active extension of force than any other appliance.

– Available in one standard link.

– No difference in appliance for the right and left sides.

– Lateral mandibular movement possible.

– More resistant to fatigue fracture.

Fig. 7.20. Sabbagh universal spring

Disadvantages:

– Unsuitability for Class III treatment.

– Limitations in patients with maximum opening of less than 48 mm.

– Increased force levels.

– Considerably greater cost.

Conclusion. The purpose of this chapter was to discuss the biological basis and clinical management of the various functional appliances.

Today, with this important tool in the hands of the orthodontist, the speciality has truly evolved from just the ability to move teeth to the ability of influencing and transforming the dentofacial structures, thus, permitting the attainment of the achievable optimum.

However, as with any other speciality of medicine, the importance of proper diagnosis and treatment planning in the success of the functional therapy cannot be over emphasized. One must not forget the importance of correct timing for achieving the best results with functional appliances.

Literature

1. Aggarwal P., Kharbanda OP., Mathur R., Duggal R., Parkash H. Muscle response to the twin-block appliance: an electromyographic study of the masseterand anterior temporal muscles // Am. J. Orthod. 1999. – Vol. 116, N 4. – P. 405–414.
2. Akkaya S., Haydar S., Bilir E. Effects of spring-loaded posterior bite-block appliance on masticatory muscles // Am. J. Orthod. 2000. – Vol. 118, N 2. – P. 179–183.
3. Artun J., Hollender LG., Truelove EL. Relationship between orthodontic treatment, condylar position, and internal derangement in the temporomandibular joint // Am. J. Orthod. 1992. – Vol. 101, N 1. – P. 48–53.
4. Baccetti T., Antonini A., Franchi L., Tonti M., Tollaro I. Glenoid fossa position in different facial types: a cephalometric study // Br. J. Orthod. 1997. – Vol. 24, N1. – P. 55–59.
5. Bench R.W. Gugino C.F. Bioprogressive prefabricated arches // Saunders company. – 1980. – 34 p.
6. Bishara SE, Ziaja RR. Functional appliances: A review, Am J Orthod Dentofac Orthop 1989;95:250–6.
7. Bishara S.E. Textbook of Orthodontics // W.B. Saunders company 2001. – 592 p.
8. Bishara L.E., Justas R. Proceeding of the Workshop Discussion on Early treatment // Am. J. Orthod. Dentotaeial Orthop. – 1998. – Vol. 113. – P. 5–6.
9. Brower H. Child dental care and serial extraction: a long-term survey // Brit. J. Orthod. 1986. – Vol. 13, N 3. – P. 135–145.
10. Cameron A, Widmer R. Handbook of pediatric dentistry. – 3rd ed. Philadelphia, Pa.: Elsevier; 2008:503.
11. Clark WJ. The twin block technique. A functional orthopedic appliance system, Am J Orthod Dentofaclal Orthop 1988;93:1-18.
12. Eirew 1-1L.The Bionator, Brit J Ortho 1981;8:33–36.
13. Frazier-Bowers SA, Long S, Tucker M. Primary failure of eruption and other eruption disorders–Considerations for management by the orthodontist and oral surgeon. Semin Orthod 2016;22(1):34–44.
14. Frankel R. A functional approach to orofacial orthopedics // Brit J Ortho 1980;7:41–51.
15. Gottfried PF, Schmuth GP. Milestones in the development and practical applications of functional appliances // Am J Orthod 1983;84:48–53.
16. Grippaudoa C, Cafierob C, D'Apolitoc I, Riccic B, Frazier-Bowers SA. Primary failure of eruption: Clinical and genetic findings in the mixed dentition // Angle Orthod 2018;88(3):275–82.
17. Hanisch M, Hanisch L, Kleinheinz J, Jung S. Primary failure of eruption (PFE): A systematic review // Head Face Med 2018;14(1):5.
18. Hartsfield JK, Jacob GJ, Morford LA. Heredity, genetics and orthodontics: How much has this research really helped? // Semin Orthod 2017;23(4):336–47.
19. Jeong WS, Choi JW, Kim DY, et al. Can a surgery-first orthognathic approach reduce the total treatment time? // Int J Oral Maxillofac Surg. 2017;46:473–482.
20. Keeling SD, Wheeler TT, King Gj. Anteroposterior skeletal and dental changes after early Class II treatment with bionators and headgear // Am J Orthod Dentofac Orthop 1998;113:40–50.
21. Kondo E., Aoba Tj. Case report of malocclusion with abnormal head posture and TMJ symptoms // Am. J. Orthod. Denthofacial. Orthop. 1999. – Vol. 116, N 5. – P. 481–493.

22. Lione R, Buongiomo M, Lagana G, Cozza P, Franchi L. Early treatment of Class III malocclusion with RME and facial mask: Evaluation of dentoalveolar effects on digital dental casts // Eur J Pediatr Dent 2015;16(3):217–20.

23. McNamara A, Howe Rp. Clinical management of the acrylic splint Herbst appliance // Am J Orthod Denrofac Orthop 1988; 94:142–9.

24. Millet D, Welbury R. Orthodontics and pediatric dentistry / D. Millet, R. Welbury. – London : Harcourt Publishers Limited, 2000. – 167.

25. Mitchell L. An introduction to orthodontics / L. Mitchell. – Oxford : Oxford University Press, 1996.

26. Nakhjavani Y, Nakhjavani F, Jaferi A. Mesial stripping of mandibular deciduous canines for correction of permanent lateral incisors // Int J Clin Pediatr Dent 2017;10(3): 229–33.

27. Oevincenzo 1. The Eureka Spring: A new in ternrch delivery system // J Clin Ortho 1997;32:454–67.

28. Orthodontics / Edited by Professor P.S.Flis – Kyiv : Medicine, 2008. – 334 p.

29. Orton H.S. Functional appliances in orthodontic treatment: an atlas of clinical prescription and laboratory construction, 1990, Quintessence Publishing Company.

30. Pediatric dental prosthetics/ Edited by Professor P.S.Flis – Kyiv: AUS Medicine, 2012. – P. 9–21, 38–01

31. Proffit WR. Contemporary orthodontics. – 6th ed. – Philadelphia, Pa. : Elsevier; 2019:746.

32. Raberin M. Muscular equilibrium and orthognathic surgery. A preliminary electromyographic study // Orthod. Fr. 2000. – Vol. 71, N 1. – P. 37–48.

33. Roth R.H. Functional occlusion for the orthodontist, part I // J. Clin. Orthod. – 1981. – 15. – P. 32–51.

34. Ricketts R.M., Bench R., Gugino C. Bioprogressive therapy. – Denver : Rocky Mountain. – 1989. – 123 p.

35. Singh G. Textbook of orthodontics. – Jaypee Brothers Medical Publishers (P) Ltd, 2007. – 704 p.

36. Slavichek R. The masticatoty organ: functions and disfanctions. – Klosterneuburg : Gamma med. – wiss. Fortbidung; 2002:554.

37. Stormer K., Pancherz H. Electromyography of the perioral and masticatory muscles in orthodontic patients with atypical swallowing // J. Orofac. Orthop. 1999. – Vol. 60, N 1. – P. 13–23.

38. Stucki N, Ingervall B. The use of the jasper jumper for correction of Class II malocclusion in the young permanent dentition // Eur I Orthod 1998;20:271–81.

39. Schwartz SB, Christensen JR. Examination, diagnosis, and treatment planning. In: Nowak AJ, Christensen JR, Mabry TR, Townsend JA, Wells MH, eds. Pediatric Dentistry: Infancy through Adolescence. – 6th Ed. – Philadelphia, Pa. : Elsevier; 2019: 434–5.

40. Taner Sarisoy L., Darendeliler N. The influence of extraction orthodontic treatment on craniofacial structures: evaluation according to two different factors // Am. J. Orthod. 1999. – Vol. 115, N 5. – P. 508–514.

41. Woodside DG, Mctaxas A, AJtuna G. The influence of functional appliance therapy on glenoid fossa rernodeling // Am J Orthod Dentofacial Orthop 1987;92:181–98.

42. Yamin Lacouture C., Woodside D.R., Sectakof P., Sessle B. The action of three types of functional appliances on the activity of the masticatory muscles // Am. J. Orthod, 1997. – Vol. 112, N 5. – P. 560–572.

43. Yashiro K., Takada K. Tongue muscle activity after orthodontic treatment of anterior open bite: a case report // Am. J. Orthod. 1999. – Vol 115, N 6. – P. 660–666.

CONTENTS

Навчальне видання

Назарян Розана Степанівна
Хмиз Тетяна Григорівна
Кузіна Вікторія Вадимівна

ПРОФІЛАКТИЧНА ТА ПРЕВЕНТИВНА ОРТОДОНТІЯ

Навчальний посібник для англомовних студентів

Відповідальний за випуск Т.Г. Хмиз

Комп'ютерна верстка О.Ю. Лавриненко

Формат А5. Ум. друк. арк. 9,5. Зам. № 22-34257.

Редакційно-видавничий відділ
ХНМУ, пр. Науки, 4, м. Харків, 61022
izdatknmurio@gmail.com, vid.redact@knmu.edu.ua

Свідоцтво про внесення суб'єкта видавничої справи до Державного реєстру видавництв, виготівників і розповсюджувачів видавничої продукції серії ДК № 3242 від 18.07.2008 р.