Growth and Maxillofacial Surgery - A Plea for a more Physiological Approach to Surgery

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Abstract

The present article is the unfinished final scientific work of the late Professor Jean Delaire and offers insights into his innovative ideas during the concluding phase of his career. Right at the beginning, Delaire challenges traditional teachings by asserting that all growth sites in the skeleton share the same histological origin, namely the cephalic neural ectomesenchyme. He advocates for a comprehensive understanding of biokinetic effects and sutural physiology beyond mere union elements. In addition, embryological, morphological, and biomechanical aspects are combined to present a comprehensive understanding of the development and evolution of maxillary and premaxillary structures in humans. Delaire argues that the development of the cranio-facial skeletal construct strictly adheres to universal laws governing harmonious and balanced states and highlights the adherence to universal mathematics in life while emphasizing that life does not accept fixity. Case presentations are used to demonstrate how orthopedic and surgical interventions are aimed at preventing, interrupting, and healing issues related to various growth sites in the facial skeleton, such as the premaxilla, tuberosity regions, mid-palatal suture, condylar region, and bony chin. These treatments also address soft tissue involvement, including masticatory muscles, nasolabial muscles, muscles affecting the chin, and soft palate muscles. Pioneering work by renowned anatomists accompanies and substantiates Delaire's explanations by tracing the historical development of these ideas, with the realization that the actual physiology of the sutures only became widely known in the orthodontic and maxillofacial world at a very late stage.

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Preface

The present article is the unfinished final scientific work of the late Professor Jean Delaire, a distinguished personality in maxillofacial surgery. Professor Delaire, recognized for his significant contributions to craniofacial reconstruction, left an incomplete manuscript that we have diligently reviewed and compiled for publication.

The manuscript offers insights into Professor Delaire's innovative ideas during the concluding phase of his career. Despite its incomplete nature, the material presents potential advancements that could impact the field significantly.

Our editorial team has organized Professor Delaire's notes, sketches, and unfinished sections to reveal the essence of his final scholarly endeavor. The publication is accompanied by a homage to Jean Delaire, written by Professor Ulrich Joos, which provides a rounded perspective on the significance of his contributions.

We invite the scientific community to engage with this publication, aiming to acknowledge and analyze Professor Delaire's work objectively. By doing so, we hope to foster a continued dialogue around his ideas and encourage future advancements in orthodontics and maxillofacial surgery.

Homage to Professor Jean Delaire

In 2022, Professor Jean Delaire died at the age of 99, and was fortunate enough to remain mentally fit until the end. He was the founder of physiological therapy in the treatment of malformations using both orthodontic and surgical methods. He wanted his students to continue along this path for the benefit of patients and to develop further their knowledge. In addition to the enormous services he rendered to science, I always saw him as a patron of the arts, open-minded and able to talk about anything without intellectual boundaries.

With his philanthropic and accessible manner, he was not only a professional but also a human role model for us all.

In 1972, as a German student, I received a scholarship from the University of Nantes and was able to study with Professor Delaire for four months. During this time, I got to know Professor Delaire as a tolerant and cosmopolitan university teacher, who was happy to explain his treatment methods and his vast knowledge to a young German student with only an moderate knowledge of French. Professor Delaire also allowed me to take part in several demonstrations of craniofacial surgery that Dr. Paul Tessier was performing for a small circle of American colleagues.

I was so inspired by my time studying in Nantes that I decided to make it my profession. Professor Delaire and I had an intensive teacher-student relationship, which developed into a deep friendship. Professor Delaire allowed me to come to his clinic in Nantes at any time, which I was happy to do 4 or 5 times a year. In 1979, I was able to obtain an official post as a doctor at the University of Nantes. During the many courses on craniofacial growth, the Delaire mask and the orthodontic and surgical treatment of facial malformations that Professor Delaire gave in Germany, I was always his companion and his translator in the courses and also in his German-language publications. The teacher-pupil relationship developed into what he saw as a brotherly relationship and a deep friendship that lasted until his death (**Figure 1**).

Professor Jean Delaire born in 1923 was Director and Chairman of the Stomatology and Maxillofacial Clinic at the University Hospital in Nantes from 1959-1990. Prior to studying medicine, he had trained as an orthodontist. As an additional responsibility, as Dean of

Medical Faculty and Director of Dentistry he was responsible for the building of a new dental school and hospital. During his scientific life he published over 300 articles and 8 manuals.

Figure 1. Memories of Prof. Jean Delaire. **Top, from left to right:** President of the European Association for Cranio-Maxillo-Facial Surgery 1984. Course about the Delaire mask in Karlsruhe, Germany 1982. Prof. Schilli, Prof. Joos and Prof. Delaire at the European Congress of Cranio-Maxillo-Facial Surgery in Münster 2002. **Bottom, from left to right:** In the audience during the inauguration lecture of Ulrich Joos at the University of Münster, Germany 1992. Interview about physiological treatment concepts in Nantes 2015. Last visit in Nantes 2016.

Through his teacher Lucien Lebourg, he was inspired at an early age to become intensively involved with the craniofacial growth of the skull and face. This passion accompanied him throughout his life and he was still able to inspire young people for this field well into his 90s. At an early age, he was confronted with the treatment of deformities, especially cleft lip and palate. He began to develop the Delaire mask for the treatment of Class III patients and, in parallel, to place the surgical methods of treatment on a physiological basis and was thus able to revolutionise the results. His physiological methods were recognised worldwide and he had a large number of students from all over the world. Prof. Jean Delaire's motto, which can be seen as his legacy, is as follows.

"Ce sont tousjours les petites gestes qui font la difference."

(It's always the little things that make the difference.)

– Jean Delaire, Nantes, France

1 Introduction

Despite substantial progress in understanding the growth of the facial skeleton, many surgeons continue to operate:

- in accordance with the long-standing surgical principles according to which correction of dysgnathia must be performed once growth is complete, at the end of adolescence and perhaps in the early years of adulthood,
- based more on criteria of morphological and aesthetic normalisation than on a physiological basis.

In fact, systematically postponing the date of orthognathic surgery (until the end of sutural growth) remains a good precaution in correcting skeletal Class III malocclusions, as persistent excess mandibular growth following surgery can compromise surgical outcomes. This is not the case for skeletal Class II malocclusions, where Trauner (1967) advised systematic intervention before the end of the pubertal period (or even at the start of it), since continued mandibular growth could, in his view, only improve the quality of surgical results in the long term.

It is important that surgeons have a good understanding of the fundamentals of normal development of the craniofacial skeleton. This enables them (possibly assisted by an orthodontist) to see patients early and so reduce the severity of the developing deformity, or even to normalize the abnormalities of the craniofacial skeleton, leading to less surgical intervention or even none at all.

Several examples, although not exhaustive, will be given below, illustrating the value of this surgical (and also orthopaedic) approach, often referred to as *interceptive*, which deserves to be called *physiological* because it does not only correct skeletal shapes and structures, but also the growth potential of the regions concerned and the functions involved.

2 Fundamental principles of craniofacial skeletal growth

It is imperative to recognize, right from the outset, that, in contrast to the classical doctrine, all growth sites within this skeletal structure share the same histological origin: the **cephalic neural ectomesenchyme**. This specific tissue gives rise to both the bones of the face and the skull. This includes, but is not limited to: i) the membranous sutures connecting the bones of the skull and face, ii) the periosteum enveloping them, iii) the chondrocranium and its derivatives (such as the basi-cranial synchondrosis, mesethmoid, and nasal cartilage septum), and iv) the condylar cartilage.

Their histological differences (bone tissue and cartilage) are basically the result of the *biokinetic* effects to which the embryonic neural ectomesenchyme has been subjected. These *biokinetic* effects, responsible for the different tissue differentiations of the cephalic ectomesenchyme, have been well described and illustrated by Blechschmidt (1951), according to whom (**Figure 1**):

- The *cranial bone tissue* appears in the membranous blastema (condensation of the cells of the cranial embryonic mesenchymal cap) of the skull vault, in territories subject to pressure-sliding forces (*detraction*), due mainly to the expansion of the cranial contents.

- The *cartilaginous tissue* appears, at about the same time, in those areas subjected to moderate pressures (*densification*). It then turns into cartilaginous *plates* when these forces are exaggerated (*contusion* or *distusion*).

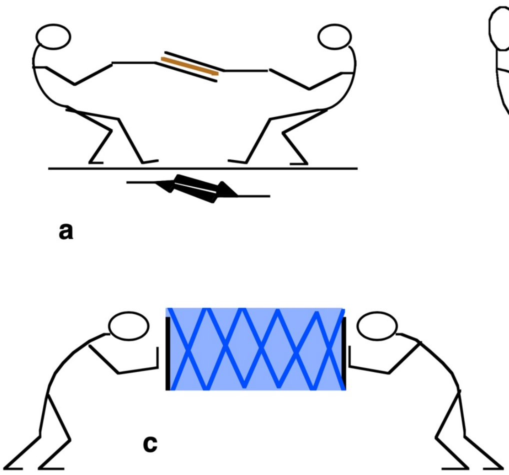

Figure 1. *Biokinetic* phenomena underlying the differentiation of ectomesenchyme into bone and cartilage according to Bleschschmidt (1951). *Detraction* (**a**), *Condensation* (**b**), *Contusion* (**c**) and *Distusion* (**d**).

A very good demonstration of these biokinetic effects is observed in the anterior and inferior frontal region and in the anterior and medial part of the upper face where bone and cartilage are parallelized (**Figure 2**).

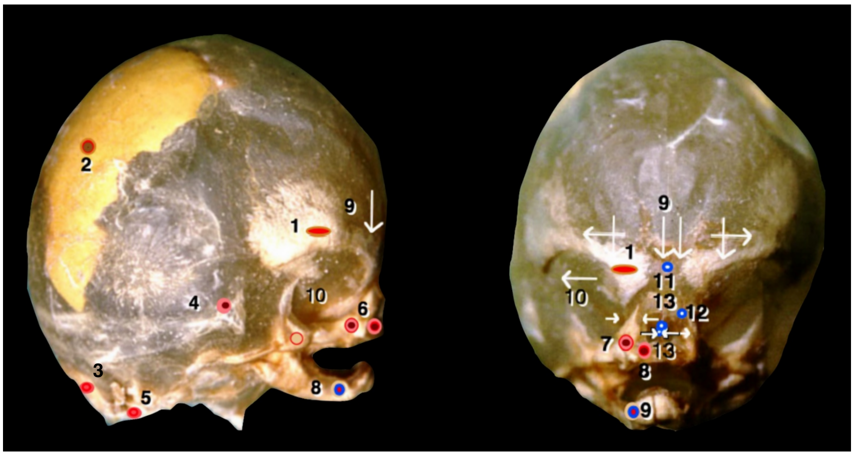

Figure 2. Profile (**left**) and enface (**right**) view of the locations and dates of the appearance of the first ossification points of the cranial skeleton (Mall, 1906). 1) frontal (56 days), 2) parietal (56 days), 3) supra-occipital (56 days), 4) exo-occipital (56 days), 5) squamosal (56 days), 6) maxilla (39-40 days), 7) premaxilla (42 days), 8) mandible (39 days), 9) encephalon, 10) eyeball, 11) anterior chondrocranium, 12) lateral part of the nasal capsule, 13) median nasal septum.

2.1 Histophysiology of ossification and bone growth

In the cranial vault (as an example of the description of membranous development), the process of ossification is preceded by the appearance, in the membranous blastema, of fibroblasts which will provide collagen fibre and will turn into osteoblasts. Ossification itself begins between the 45th and 55th day (Augier, 1912) in the form of tiny islets of *pre-osseous* substance (*calcafine*) around which the osteoblasts are arranged (**Figure 3, left**). The shape of these first *islands*, which resembles that of a dragon, indicates the direction of their displacement.

These early ossification nuclei fuse rapidly, giving rise to an *open reticular bone plate* whose osteoblasts become osteocytes, at the same time that the *osteoid* substance they have secreted (and which surrounds them) is transformed into bone and organized into trabeculae.

New osteoblasts form on the surface and periphery of this plate, which will follow the same pattern. In its peripheral extension, the *reticular* plate slips between two mesenchymal plates that it has helped to individualize (**Figure 3, right**).

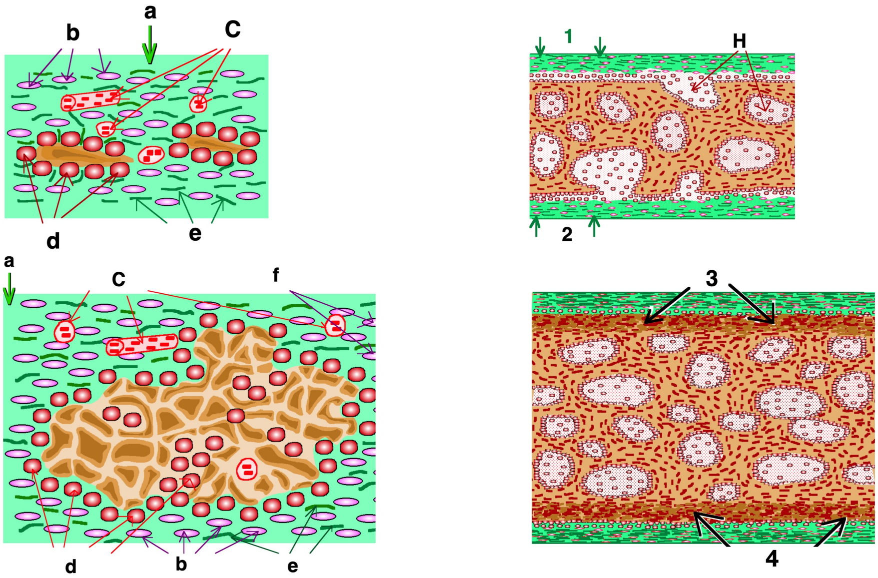

Figure 3. Left: Schematic representation of the first islands of ossification (**top**) and the open reticular plate, consecutive to their union (**bottom**). a) membranous blastema, b) fibroblasts (precursors of osteoblasts), c) blood capillaries, d) osteoblasts, e) collagen fibers, f) fibroblasts (precursors of osteoblasts). **Right:** The production (by the superficial and deep periosteum) of cortical bone transforms the *open plate* into a *closed plate.* **Top:** Open reticular plate open in a flat bone of the vault of the skull of a fetus (Kolliker, 1860). 1) exo-cranial periosteum, 2) endo-cranial periosteum (dura mater), 3) external cortex, 4) internal cortex, H) primitive channels of Havers whose walls are lined with osteoblasts (some open in the periosteum). The vessels have not been represented. **Bottom:** Early closure of the reticular plate. The exo- and endocranial surfaces are closed by recent cortices (vessels not shown).

The superficial layer becomes the external periosteum, while the deep layer becomes the *dura mater*, the equivalent of an internal periosteum. The production of a layer of cortical bone by each of these *periosteal* blades transforms the *open plates* into *closed plates* which extend at their periphery into the neighboring mesenchyme.

According to Athenstaedt (1969), the directions of these extensions are the result of piezoelectric fluxes produced by forces from both the encephalic expansions and the pressures and traction of the neighboring muscles. These actions begin as early as the end of the second embryonic month (**Figure 4, left**).

In the foetus of 3-4 months (**Figure 4, right**), these already well developed plates leave large bands of mesenchymal tissue between them, known as fontanellar bands, which will gradually reduce to form the membranous sutures, which at birth, still leave non-ossified spaces at their intersections, known as *fontanelles.* These will close normally in the months following birth (**Figure 5**).

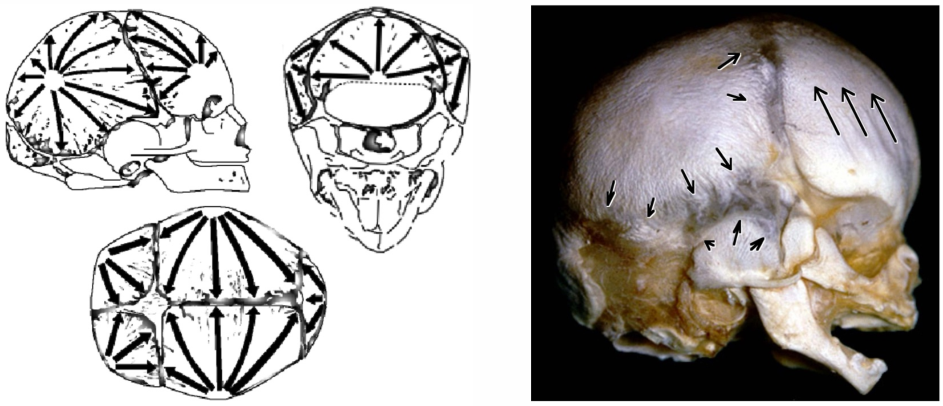

Figure 4. Left: The location of the ossification points of the skull vault and the direction of the the electrical polarization of its mesenchymal cap (Athenstaedt, 1969). **Right:** State of cephalic ossification in the foetus of 3-4 months. At this age, between already well developed bony plates, wide bands of mesenchymal tissue are present, and these are called *fontanellar bands*, which will gradually shrink to form the membranous sutures which, at birth, still persist where they meet and known as *fontanelles.*

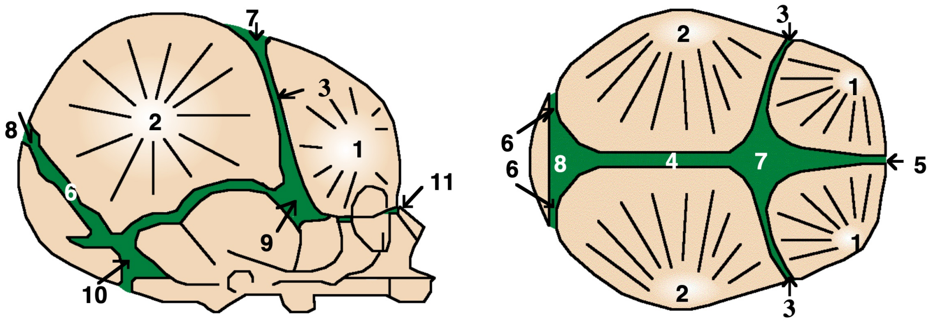

Figure 5. State of cranial sutures at birth. The margins of the bone plates have come closer. The fontanellar bands, gradually becoming narrower, become the membranous sutures. Expanded spaces existing at their confluence points are called *fontanelles* 1) frontal tuberosities, 2) parietal tuberosities, 3) coronal sutures, 4) interparietal (mid-sagittal) suture, 5) metopic suture, 6) parieto-occipital suture, 7) bregma, 8) pterion, 9) asterion, 10) fronto-naso-maxillary fontanelle.

It is important to note, that *closure* does not mean *loss of activity* as was once thought, at a time when sutures were considered as simple elements of union between the skeletal parts they separate.

In fact, the true nature of membranous sutures and their role in craniofacial growth have already been fully understood and clarified in 1931 and 1933 by L. Lebourg (Parisian stomatologist) in his medical thesis on *La dysarthrose craniofacial* and his article on *Nature, évolution et rôle des articulations de la face; leur importance physiologique* (Lebourg and Seydel, 1932).

He was the first to understand their periosteal nature and exact physiology, the anatomical and functional similarity of the sutures of the skull, face and alveolar-dental ligaments, and their responsibility in determining dentofacial dentofacial deformities.

The following sentences, taken from his work, testify this and confirm not only how right he was but also how advanced his concepts were (generally attributed to J.H. Scott and J.J. Pritchard).

> *"The osteogenesis of the face, like that of the cranial vault, takes place in a fibrous matrix which remains for a variable time between the bony parts. Classically, this fibrous interposition behaves as a conventional simple interosseous ligament. In reality, it's a genuine growth periosteum, bipolarized, in continuity with the covering periosteum, allowing the bones to expand on the surface."*
>
> *"The histological appearance of the sutures sometimes gives the impression of intense activity. Capillaries, often voluminous, pour into this veritable construction site the materials required for osteogenesis. Far from playing a modest ligamentary role, this tissue exhibits remarkable osteogenic activity."*
>
> *"As development progresses, this plate narrows, and the contiguous bone surfaces emit serrations that mesh with each other; the evolution culminates, more or less late, in complete fusion."*
>
> *"From an anatomical point of view, we can assign the three stages of synfibrosis, synarthrosis and synostosis to this evolution, depending on whether the bones are still mobile one on top of the other (loose fibrous suture), whether they are already meshed, or finally whether they are fused together."*
>
> *"For us, the periodontum is nothing more than a specialized conjugation periosteum."*

It was only in 1953 and 1956 that the same conceptions were published by Scott *Growth at facial sutures* (Scott, 1956) then Pritchard, Scott and Girgis in their article *The structure and development of cranial and facial sutures* (Pritchard et al., 1956). It was not until 1967, when Scott's book *Dento-Facial Development and Growth* was published, that the anatomy and actual physiology of sutures was known to the orthodontic world (35 years after Lebourg's thesis).

Scott confirmed the secondary character of the sutural proliferation and the primordial role of the *separating* factors, in particular the cranial contents and the cartilages of the base of the skull and the face, on the growth of the craniofacial skeleton.

Lebourg's original cross-section (**Figure 6, left**) and Scott's schematic representation of sutural anatomy (**Figure 6, right**) support this physiological concept, which is now universally recognized. In fact, sutural physiology is more extensive and needs to be well understood.

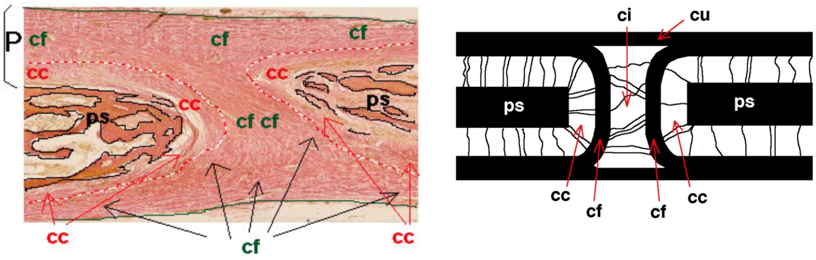

Figure 6. Left: Histological appearance of cranio-facial synfibrosis at birth according to Lebourg (1931); Plate VII: *"This section was cut perpendicular to the fronto-malar joint. At low magnification, we can see the connective tissue between the two bony plates, in continuity with that which covers them. The concentric orientation of the fibers is clearly visible. Clearly, there is no fibrous ligament between the two bone blanks, but rather an active, osteogenic tissue in histological continuity with the periosteum - in short, a genuine periosteum of growth."* **Right:** Schematic representation of sutural anatomy after Scott (1967).

2.2 The sutural functions

Schematically, three sutural functions can be identified (**Figure 7, upper left**).

- A *ligamentary* function, limiting and dampening excessive deviation and displacement of the sutural edges.
- An *osteogenic* function of adaptive marginal growth of the osseous margins, stimulated by distension and, conversely, slowed by sutural compression.
- An *articular* function allowing the skeletal parts united by the suture to orientate themselves differently with respect to each other.

In other words, the sutures are at the same time *(i) joints of rupture*, *(ii) expansion joints with marginal ossification* and *(iii) movement joints.*

In young people, the three functions of the suture are linked as follows. The damping function regulates the fit and alignment of the suture, the osteogenic function adjusts the size of the skeletal parts to the volume of the growing tissues and viscera, the joint function ensures their best orientation and the best overall architectural balance.

Taking into account the diversity of forces acting on the craniofacial skeleton and the movements of the skeletal parts that they unite, the state of the sutures (direction, forms, growth activity) can therefore be very different (**Figures 7 and 8**), so that conclusions about their functional present (and past) can be drawn from their morphological appearance alone.

Another, very important point of sutural growth must be emphasized: the (relative) independence of the growth of external and internal cortices, both in the cranium and in the face. Topinard (1876) was probably the first to have insisted on this concept when he wrote:

> *"The skull is formed of two independent lamellae, not obeying the same physiological influences, here making contact, there spreading, either leaving between them a more or less abundant spongy tissue called diploe, or supplements of compact tissue at the level of external projections and ridges, sometimes finally*

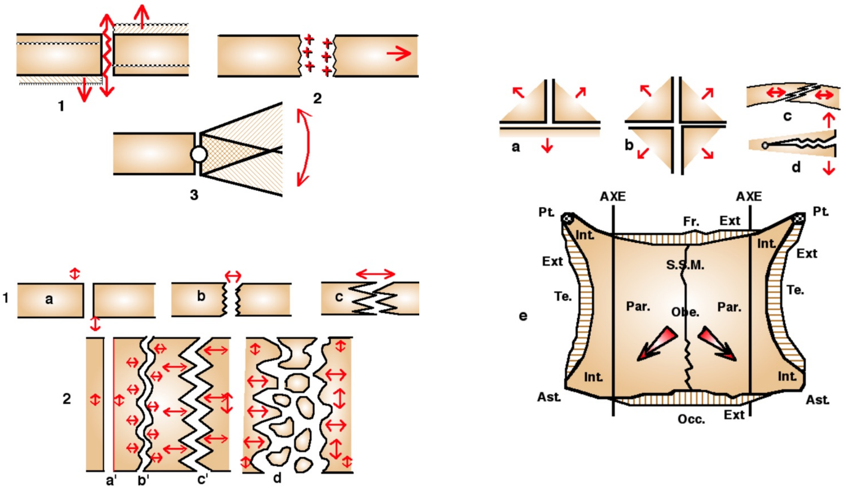

Figure 7. Upper left: The three sutural functions: 1) *ligamentary* = *cushioning / rupture joint*, 2) *osteogenic* = *expansion joint* and *articular* = *movement joint*. **Lower left:** Morphological consequences of sutural physiology. 1) view in section: (a) *linear* suture = vertical offsets, (b) *slightly serrated* suture = small transverse movements, (c) *very serrated* suture = large transverse movements. 2) surface view: (a') linear suture = displacements along the sutural axis, (b') undulating suture = weak transverse movements, (c') serrated suture = significant transverse movements, (d) multifragmental sutural band = large spreading movements with vertical offsets. **Right:** Morphological consequences of sutural physiology (continued). (a) three-branched fontanelle, (b) four-branched fontanelle, (c) beveled suture, (d) v-shaped suture: limited to one end and (e) sutural bevels. Asterion (Ast), pivot axis (AXE), external table (Ext), internal table (Int), frontal (Fr), obelion (Obe), Occipital (Occ), parietal (Par), pterion (Pt), medial sagittal suture (SSM), temporal (Te).

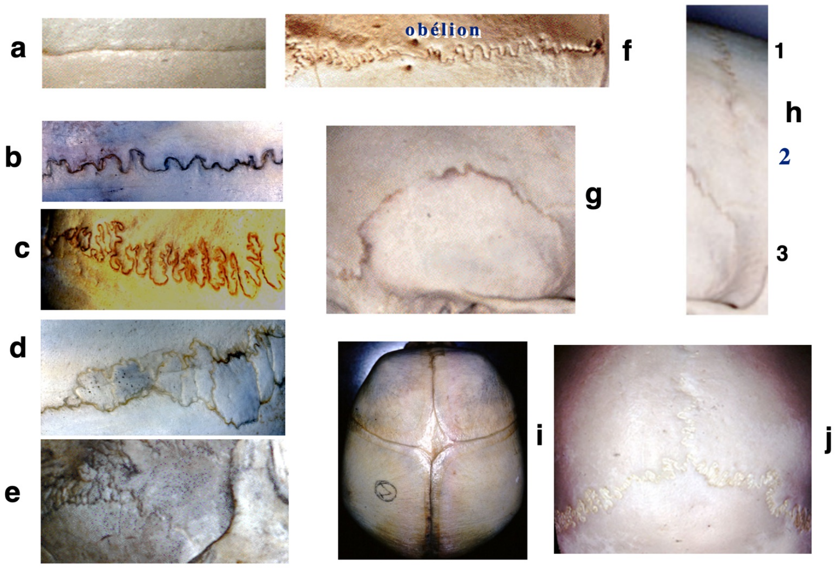

Figure 8. Different clinical aspects of cranial membranous sutures. a) linear suture. b) serrated suture, c) large sutural interdigitations, d) wormian bones, e) multiple micro wormian bones (hydrocephalus), f) median sagittal suture. Many small interdigitations on both sides of the obelion, g) parieto-temporal suture (arciform, bevelled), h) coronal suture (1) serrated part, (2) straight part, (3) pterion), i) newborn lambda (vertical view), j) adult bregma (posterior view).

> *leaving between them more or less large voids called sinuses as at the base of the forehead, where these sinuses sometimes extend almost to the frontal bosses."*
>
> *"Ligaments and muscles are attached to the outer blade, thickening it and developing it to a greater or lesser extent, depending on how robust the subject is. While the internal lamina is governed by the brain, the external lamina is in touch with external life."*

Similarly, in 1952, Lucien De Coster placed much emphasis on these particularities of development of the frontal bone (De Coster, 1952):

> *"The frontal bone is the type of bone where the inner blade and the outer table best show their differences. In the beginning, the outer and inner blades are absolutely parallel. But the inner blade stops developing very early on, while the outer blade lengthens. At first, spongy tissue forms between the two blades, then a true sinus is created, lined with mucous membrane, forming a cavity" ... "This is a dynamic formation related to the direction of masticatory forces in relation to the frontal."*

Occlusal forces that advance the frontal bone cortices should, however, add thrust to the the anterior part of the cartilaginous mesethmoid and nasal catilaginous septum (**Figure 9**).

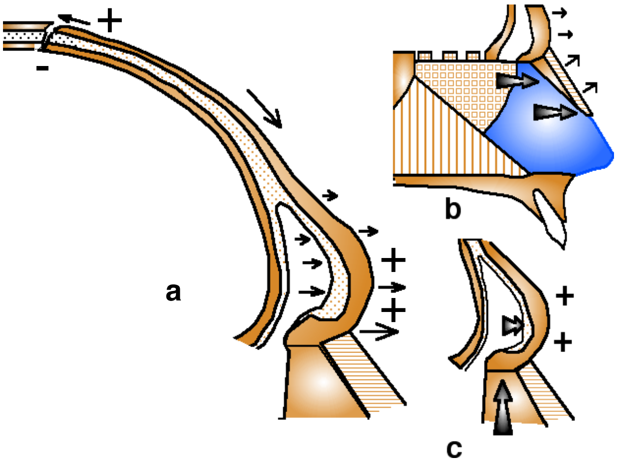

Figure 9. Schematic representation of the frontal bone. Section showing the differental growth of the external and internal cortices: (**a**) The selective advance of the frontal cortex of the frontal bone involves differentially more activity of the external cortical suture than the internal cortical suture. (**b**) The forward thrust of the septal cartilage in the median plan plays an important role in the specific advance and the development of frontal sinuses. (**c**) The occlusal forces, in particular those coming from the incisivo-canine regions and premolars which also play a significant role in the development of these sinuses. They also cause superficial periosteal appositions, especially at the level of the glabella and supra-orbital ridges.

Cornelis Van der Klaauw and Melvin Moss also emphasised the individuality of the two cranial bone cortices, which they consider as true *skeletal units*. In fact, this bi-cortical discrepancy of the membranous bones is part of their normal evolution. It also exists in the maxilla (Van der Klaauw, 1946; Moss, 1958).

Another peculiarity of the sutural physiology that is characteristic of them is the significant chronological differences between their times of closure and their fusion, with more or less early (or more or less late) disappearance of certain sutures. This is essentially related to the local conditions to which they are subjected.

The earliest fusions occur as early as the embryonic period. This is true for the external parts of the premaxillo-maxillary sutures which, in man (Cadenat, 1924), normally fuse at the end of the 7th week, thus leading to the assimilation of the premaxillary skeletal unit to the maxilla. In other words, the loss of its individuality and its transformation into *skeletal unity.*

Also, the medio-frontal (metopic) suture closes very early and begins as soon as the infant begins to raise his head (in his cot and then in the arms of his mother) and is usually almost complete at age three, when upright posture and upright walking are well acquired.

Conversely, other sutures fuse very late, if ever. This was popular belief amoung earlier anatomists who insisted on the very late closure of the parieto-occipital sutures in particular, in contrast to the almost total absence of fusion of the circum-temporal sutures due to the persistent mobility of the temporal bones. An exception is the totally edentulous, according to Firu (1959).

This great chronological variability in closure of membranous sutures, very different from that which occurs in the cartilages of all the long bones, proves that they are basically *secondary* (adaptive) growth sites. We shall soon see that this variability can also be observed in the synchondroses at the base of the skull, leading us to discuss their true nature. All in all, to summarize the physiology of cranial membranous sutures, we can say the following.

1. Thanks to their adaptive growth, the internal cortical sutures contribute to the increase in the volume of the cranial cavity, normally perfectly adapted to its contents. Its internal shape is different as a result of tensions from the intracranial fascia (falx cerebri, cerebellum, and tentorium cerebelli).

2. The external shapes of the cranial sutures depend, of course, on those of the endocranium, but also on external muscular forces applied directly to the external cranial cortices or which are transmitted to them.

3. The appearance and general distribution of cranial sutures reinforces the concept that the state of sutures and the general morphogenesis of the craniofacial skeleton result from the combined effects of two fundamental mechanics (Leroi-Gourhahn, 1954; **Figure 10**).

2.3 Periostal physiology

Directly derived from the *skeletogenic membrane* in particular the membranous layers located on the surface and the deeper layers of reticular bones (**Figure 3, right**), the periosteum retains its fundamental properties, that is to say, it is able to produce fibroblasts, which will become bone-forming osteoblasts.

In the young, the activity of the periosteum is very intense and effectively contributes to bone growth and bone morphogenesis. In adults, and even into quite advanced age, it remains active and continues to influence the normal shape and external form of the skeleton.

Histologically, the periosteum covering the outer and inner surfaces of flat bones, is in section, bilaminar, with an outer *fibrous* layer and an inner *cellular* layer (**Figure 11, left**).

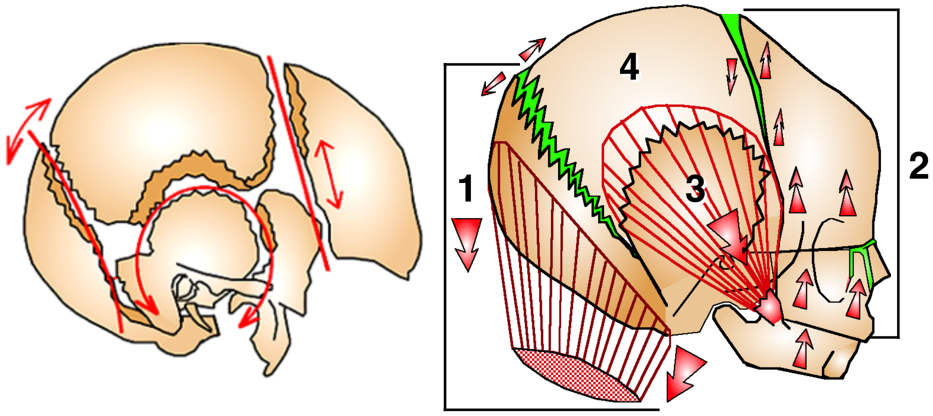

Figure 10. Cranial sutures and morphogenesis of the craniofacial skeleton. **Left:** Movements responsible for the state of cranial sutures. **Right:** The *mechanics* at the origin of morphogenetic forces (Leroi-Gourhahn, 1954). (**1**) Occipito-cervical postural mechanics condition the state of the occipital and occipito-parietal and occipito-temporal sutures. (**2**) The masticatory mechanics, originating from the occlusal forces transmitted to the anterior base of the skull, conditions the location and state of the coronal suture. (**3**) Tractions on the temporal muscles also condition the state of this suture. The arciform appearance of the parieto-temporal sutures also reflects the particular mobility of the temporal bone. (**4**) The state of the parietal territory reflects its adaptation to the forces arriving from the two mechanics located, on the one hand in front of and below it, and on the other hand behind it.

This commonly used terminology is, unfortunately, totally inappropriate as it may lead to the mistaken belief that the outer fibrous layer is inactive and that only the inner layer produces bone, and this is incorrect.

In fact, the outer layer is rich in embryonic mesenchymal cells, in osteoblast precursor fibroblasts (DOPC) as well as collagen fibres (which give the outer layer of the periosteum its characteristic appearance). They represent the *reservoir* from which the osteoblasts of the underlying layer will develop the type I collagen necessary for ossification. This generative layer is highly vascularized by numerous arterioles derived from the superficial muscle attachments to the periosteum and which extend into the inner layer.

Conversely, the inner, ossifying layer is made up mainly of cells: fibroblasts, pre-osteoblasts and osteoblasts. Between them there are fine-calibre collagen fibres running perpendicular to the bone, which are buried with osteocytes (derived from osteoblasts) in the bone tissue, where they form Sharpey fibres. These fibers, produced by osteoblasts from the collagen reserve of the outer layer, are loosely bound to it. All in all, the outer layer is the true generative layer of the periosteum, while the inner layer is merely ossificatory.

During growth, the periosteum is largely *appositional*. However, it can, conversely, be *resorbant*, by inversion of the activity of its cellular layer. This is due to the replacement of osteoblasts by osteoclasts. This complete reversal in activity of the periosteum (and its cellular organization) is due to environmental conditions, not from the *type* of periosteum (**Figure 11, right**). In other words: there is no *appositional* periosteum or *resorbent* periosteum; the periosteum always and everywhere has these two potentialities.

According to Enlow (1962), these different actions of the periosteum result directly from the increase or decrease of its vascularity. External pressure, reducing it, would thus result in external cortical resorption. Conversely depression, could cause superficial apposition

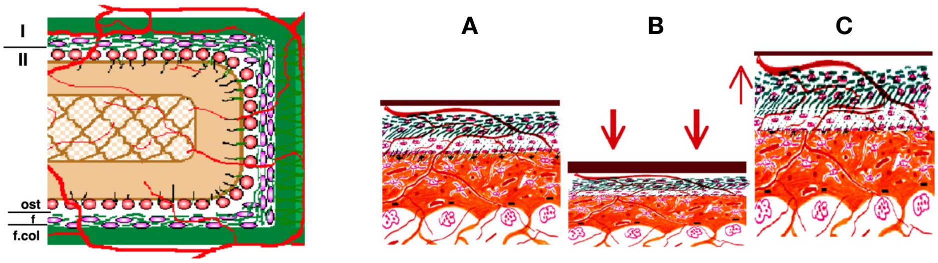

Figure 11. **Left:** Very schematic representation of periosteal histology. The outer layer (**I**), rich in embryonic mesenchymal cells, fibroblasts (f) precursors of osteoblasts (ost.) and, as well as, collagen fibers (f.col.) secreted by these cells. This generative layer is richly vascularized by numerous arterioles arising from the surface. The inner ossifying layer (**II**), is made up mainly of fibroblasts, pre-osteoblasts and osteoblasts, which are arranged in a veritable carpet on the surface of the bone. Between them are thin caliber collagen fibers running perpendicular to the bone, which, together with osteocytes (derived from osteoblasts), are embedded in the bone tissue to form Sharpey fibres. At the level of a suture these periosteum are invaginated and continue with those coming from the other side of the bone. **Right:** Schematic representation of the effects of pressures / depressions on the vascularization of the periosteum and cortical ossification (Enlow, 1962). (**A**) normal periosteum. (**B**) excessive pressure = decreased vascularity and activity of fibroblasts and osteoblasts = thinned cortices. (**C**) depression = increased vascularization and cellular activity = thickened cortices.

(**Figure 11, right**). Periosteal growth, like that of sutures, is therefore fundamentally secondary to environmental influences. Periosteal growth (like that of sutures) is therefore fundamentally secondary to environmental influences.

It should be noted that the superficial action of the periosteum is counterbalanced by that (deep) of the endosteum. The latter, made up exclusively of a cellular layer (osteoblasts or osteoclasts), helps to regulate the thickness of the external periosteal bone layer (or cortical bone) by compensating for periosteal actions. Thus, when the periosteum is appositive and produces *periosteal* cortical bone, the cortical bone is resorbed on its deep surface by endosteal osteoclasts. Conversely, when the periosteum is resorbent, the endosteum produces endosteal bone and the thickness of the external cortex is maintained.

Thanks to this balanced interplay of the *functional matrix* and the endosteal-periosteal complex, flat bones can either be thickened, as occurs normally during growth, or thinned, shaped (in particular, a change in their curvature) or undergo a kind of *drift*, corresponding to a *displacement without real mobility* (Enlow, 1965). Indeed, when the phenomena of periosteal apposition and endosteal resorption take place in the same direction on the two cortices of the bone, the latter retains the same thickness while moving in the direction of the apposition (= *relocation*).

These bone *drifts* may affect the entire skeletal part, or only part of it. They cannot, however, explain all bone displacements, which, during growth, are mainly due to the actual separation of skeletal parts from each other. This mobility, it should be remembered, is essentially the result of the particular physiology of membranous sutures (expansion joints and movement joints).

2.4 Cartilage differentiation and growth of the skull base

In dentofacial orthopedics, it is traditional to teach that the growth of the cranial base (from nasion to basion) is controlled by the chondrocranium and its remnants: the synchondroses, similiar to the diaphyso-epiphyseal cartilages of the long bones. Of *primary cartilaginous* nature, the growth of the cranial base would therefore be totally predetermined by genetics and, therefore, not the result of epigenetic influences.

In fact, the chondrocranium, like the craniofacial skeletal parts, results from the effects of the environment on the primordial cephalic mesenchyme, in this case from the pressure and compression that the base undergoes as a result of the expansion of the cranial cavity and facial and cervical resistance.

In the six-week-old embryo it appears in the form of multiple cartilaginous islands, fused as early as the 12th week, the cartilaginous plate then occupying the entire base of the skull (**Figure 12, left**). At birth, the chondrocranium is limited to the central axis extending from the anterior border of the cartilaginous mesethmoid to the basion (**Figure 12, right**).

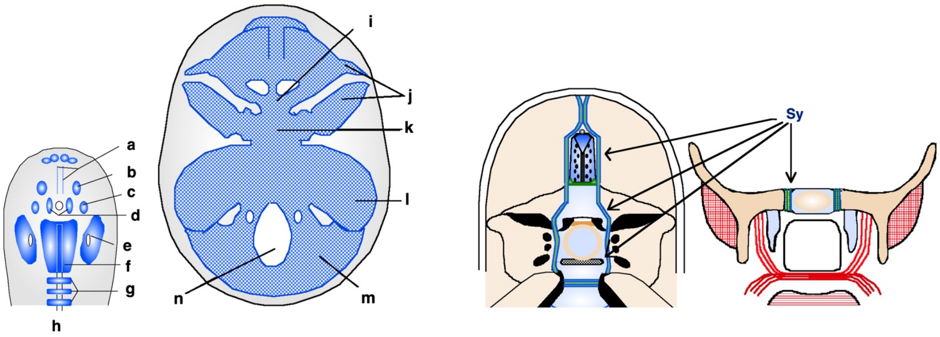

Figure 12. Left: Schematic representation of the formation of the chondrocranium. Superior views according to Moore (1974). Six weeks: The various cartilages that will merge to constitute the chondrocranium: (a) cranial struts, (b) orbital wing, (c) temporal wing, (d) pituitary cartilage, (e) auditory capsule, (f) para-chordal cartilage (basal lamina), (g) occipital sclerotomes, (h) chorda, (i) ethmoid, (j) small and large sphenoid wings, (k) sphenoid body, (l) bulge of the temporal bone, (m) occipital bone, (n) foramen magnum. Twelve weeks (right): the cartilaginous base of the constituted skull, or *chondrocranium*. **Right:** Very schematic representations, according to Scott (1967), of paramedian *mixed* synchondroses (or *chondro-sutures*), extended from frontal to the foramen magnum at birth. (Middle left) vertical view. (Far right) frontal section through the body of the sphenoid. (Sy) lateral synchondrosis.

Anteriorally and laterally, the horizontal parts of the frontal (still apart) and the lesser and greater wings of the sphenoid (all the anterior and middle cerebral fossae) are formed of membranous bones. It is the same posteriorally, where the majority of the occipital plate is also ossified. Moreover, the synchondrosis between the body of the sphenoid and its lateral parts disappear in the first year of life. Whilst Enlow (1965) cosiders their role incidental, Scott (1967) believes the contrary.

To summarize, the parts located within the paramedian synchondroses could (at best) be considered *cartilaginous* in origin. On the other hand, all the bony elements located out with (external) them should be considered as being of *membranous* origin. The transverse development of the *anterior* (ethmoid-frontal) and *middle* (sphenoidal) stages is thus mainly caused by the lateral expansion of the cranial contents. For Enlow, transverse and thickness growth are essentially secondary to periosteal apposition-resorptions with the sutures of the base acting mainly in the sagittal direction (Enlow, 1965). However, we must also take into account the activity of the sutures located in the cranial vault and base: the pterion, parieto-temporal suture, the asterion and the parieto-occipital suture. The outward displacement of these sutures contributes to the more oblique orientation of the lateral walls of the base bones (particularly the large wings of the sphenoid), and thus to the widening of the skull.

The sagittal development of the base of the skull, is generally described, before and after birth from the very classic schemes (**Figure 13**) of Augier (1912), Baume (1961) and Scott (1967).

At birth, three cartilaginous zones can clearly be seen and individualized - from front to back: the cartilaginous mesethmoid,the inter-sphenoid synchondrosis, and the occipital synchondrosis. But by the 6th month (**Figure 13, right B**), the inter-sphenoid synchondrosis is completely ossified, the basi-sphenoid and the basi-post sphenoid then forming a single skeletal unit. However, about the same time, another synchondrosis appears between the first form of the ethmoid and the anterior part of the body of the sphenoid - the sphenoethmoidal synchondrosis. By the end of the 3rd year, this is replaced by a true membranous suture (without chondrification), with occlusal force-breaking functions (without any capacity for growth). After six years (**Figure 13, right C**), only elements of chondro-cranial origin persist - in front of the medial cartilaginous septum (and the paramedian parts of the nasal capsules), behind the spheno-occipital synchondrosis and between the body of the

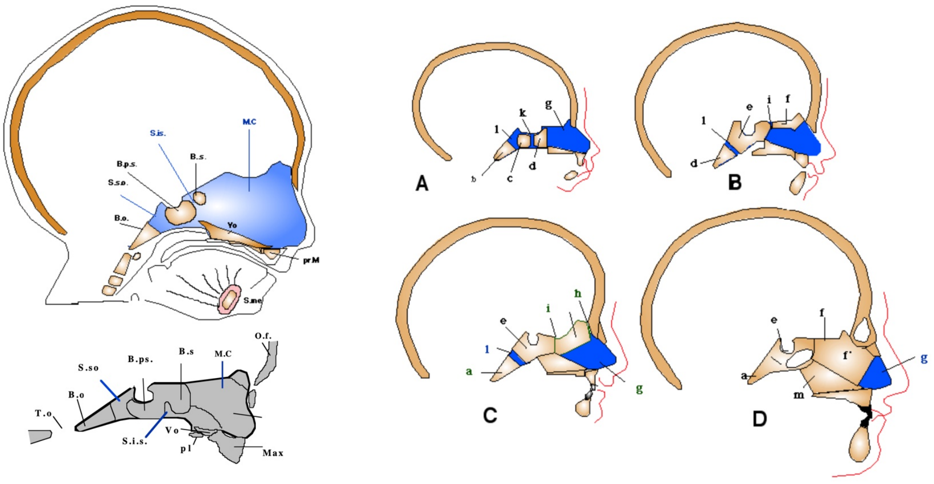

Figure 13. Left: Schematic representation, in profile view, of the first elements of the bones of the skull base. (Top) Cartilages of the base of the skull in fetal life (Scott, 1967). (Bottom) Sagittal medial skull of a newborn (Augier, 1912). Basion (B.o), basi-postsphenoid (B.ps), basi-sphenoid (B.s), cartilaginous mesethmoid (M.C), frontal bone (O.f), palatal (pl), maxillary (Max), intersphenoidal synchondrosis (S.i.s), nasal septum (S.nl), spheno-occipital synchondrosis (S.so), occipital foramen (T.o), vomer (Vo). **Right:** Post-natal development of basi-cranial synchondrosis (Baume, 1961). (**A**) term, (**B**) 6 months, (**C**) 6 years, (**D**) 40 years. (a) basion, (b) basi-occiput, (c) basi-post-sphenoid, (d) basi-sphenoid, (e) sphenoid body, (f) ethmoid bone: cribriform plate and perpendicular plate (quadrilatere lamina), (f') perpendicular plate (quadrilatere lamina), (g) nasal septum, (h) frontoethmoidal suture, (i) spheno-ethmoidal suture, (j) inter-sphenoidal synchondrosis, (k) spheno-ethmoidal synchondrosis, (l) spheno-occipital synchondrosis, (m) vomer.

sphenoid and the basi-occiput, this latter normally until adulthood. At 40 years (**Figure 13, right D**), it persists only in trace amounts.

Classically, as long as they are active, these various synchondroses grow like the diaphyso-epiphyseal cartilages of the long bones, so according to a primary mode, reacting essentially to general factors and not to local factors. But these conceptions have been much disputed by Enlow and Hans (1996) in these terms:

> *"Historically, the spheno-occipital synchondrosis has been regarded as the growth center and pacemaker that programs the development of the basicranium. This overly simplistic notion, however, as with the mandibular condyle, is a conceptual anachronism. The development of the basicranium is quite multifactorial and not merely the product of localised, midline cartilages that do not relate to the many regional growth circumstances throughout all parts of the basicranium as a whole. Only a very small percentage of the actual bone of the cranial floor is formed endochondrally in conjunction with the synchondroses, a parallel truism previously noted for the mandibular condyle."*
>
> *"Two key questions exist with regard to the lenthening of the basicranium at a synchondrosis and the process of displacement that accompanies the elongation of each whole bone. First, do the synchondroses cause displacement by the process of growth expansion, or is their endochondral growth a response to displacement caused by other forces (such as brain expansion)? Second, does the cartilage have an intrinsic genetic program that actually regulates the rate, amount, and direction of growth by the cranial base? Or is the cartilage dependent on some other pacemaking factors for growth control and secondarily responsive to them?"*
>
> *"Traditionaly, the cranial cartilages (and the whole basicranium in general) have been regarded as essentially autonomous growth units that develop in conjunction with the brain, but somehow independent of it. This is difficult to understand."*

At the same time, Melvin Moss argues the following (Moss, 1997a; 1997b; 1997c; 1997d).

> *"It is proposed that all of cephalic cartilages act similarly to the cranial sutures, i.e., they are loci of secondary compensatory growth. In conformity with the hypotheses of the method of functional cranial analysis, the cranial base is viewed as consisting of skeletal units responsive superiorly to the functional demand of the functional components of the neural mass (brain and cerebro-spinal fluid), transmitting their functional demands by means of the dura mater of the neurocranial capsule, and responsive inferiorly to the functional demand of related pharyngeal musculature (periosteal matrix) and pharyngeal functioning spaces (capsular matrix)".*
>
> *"The principal regulatory process, by means of which functional matrices influence basal synchondrosal growth, is by alteration in the relative degree of biomechanical compressive or tensile forces acting on the cranial basal skeletal elements."*
>
> *"The neural capsular matrices are viewed as affecting synchondrosal growth by means of the relative degree of axial compressive and tensile loading exerted extrinsically upon the synchondroses. This conclusion explicitly denies validity to the older hypotheses that presumed intrisic, genomic regulation of synchondrosal and, hence, of basicranial growth".*

The author is in complete agreement with these last opinions, the great differences in the chronology of the different sutures of the cranial base, according to the development of the neighboring nervous elements, being a further argument in favour of the concept according to which the cranial synchondroses are in reality *chondrofied sutures.* Nevertheless, as long as they contain cartilage this *tissue* is sensitive to general influences (in particular to somatotropic hormones), which is not the case with sutures.

In summary, from the age of six to adolescence, the base of the skull,from the foramen caecum (anterior) and the basion (posterior) has two parts behaving differently during growth: the anterior segment, extended foramen cecum (in front of the crista galli) to the anterior edge of the sella turcica, remains unchanged. The cranial upper slope corresponds to the line of De Coster (1952), demonstrating its stability. On the other hand, the segment extending from the sella turcica to the top of the basion may increase, partly due to residual (primary-type) activity of the spheno-occipital synchondrosis, and partly due to variations in angulation between the dorsal surface of the sphenoid body and that of the basi-occipital. These changes in the spheno-occipital angle can be observed in particular during changes in cephalo-rachidian posture, for example when oral ventilation is suppressed.

As well growth of these two segments, the base of the skull can still lengthen, anteriorally from the foramen cecum to the nasion and posteriorally from the opisthion (middle of the posterior border of the foramen magnum) to the maximal posterior bulge of the occipital bone. Anteriorally, the frontal bone develops in response to enlargement of the frontal sinus and, from below, anterior migration of the summit of the anterior maxillary pillars in relation to its base. It results mainly from the thrust of the nasal cartilaginous septum (on the medial plane) and the occlusal forces of the incisivocanine and premolar teeth (on either side). Development of the occipital region usually accompanies the temporo-occipital anti-clockwise rotation without modifiying the volume of the cerebellar compartment whose development in any case does not continue beyond the age of six.

2.5 Growth of the maxilla

Enlow's ideas on this topic have become classic and commonly accepted as the main reference. The reason is their simplicity, the excellence of the diagrams that illustrate them, and also the multiplicity of books and articles produced by the author.

According to Enlow (1996), the entire development of the maxillary mass would result, on the one hand, from the growth activities of the multiple sutures which separate the skeletal pieces from the upper surface of each other and the bones of the base of the skull (**Figure 14, upper row**). On the other hand the very important phenomena of apposition-periosteal resorption (**Figure 14, lower row**) capable not only of reducing or increasing their surfaces considerably, but also of ensuring their real displacements, according to a process he calls *relocation.*

In the sagittal direction, maxillary growth is mainly due to considerable tuberosity periosteal apposition. The very significant anterior resorptions would also be responsible for a genuine posterior facial rotation, which Enlow (1996) was the only person to have described and illustrated (**Figure 15, left**).

In fact, this interpretation of the mechanisms of maxillary sagittal growth has been contradicted by many other authors, among which we must remember in particular, Hellmann (1929), De Coster (1952) and Björk (1968). The reason for this divergence of opinion is due to, obviously, variations of superimpositions on the contour of orbits (chosen by Enlow) whose orientation varies a lot from birth to adulthood.

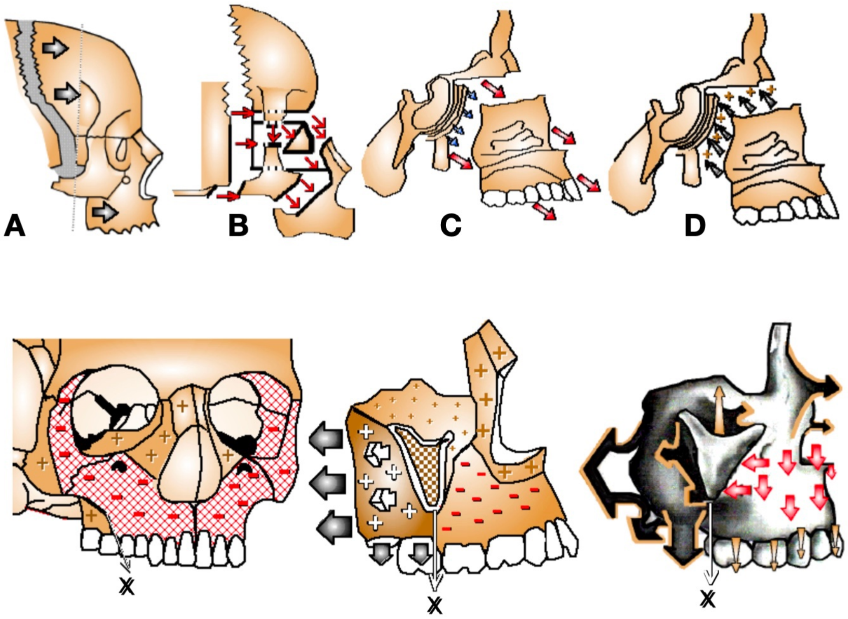

Figure 14. Top: The sutural gaps at the origin of membranous ossification *compensators* (Enlow, 1965). (**A**) Between the parietal bones and the greater wings of the sphenoid posteriorly and the fronto-facial complex anteriorly, (**B**) between each of the skeletal parts of the fronto-facial complex, and (**C**, **D**) between the large facial bones and bones of the anterior skull base (*circum-maxillary sutures*). **Bottom:** Schematic representations of the areas of apposition (+) and resorption (-) periosteal maxilla, in the sagittal direction (Enlow, 1965). Inversion line (**X**) between the anterior resorptions and posterior appositions.

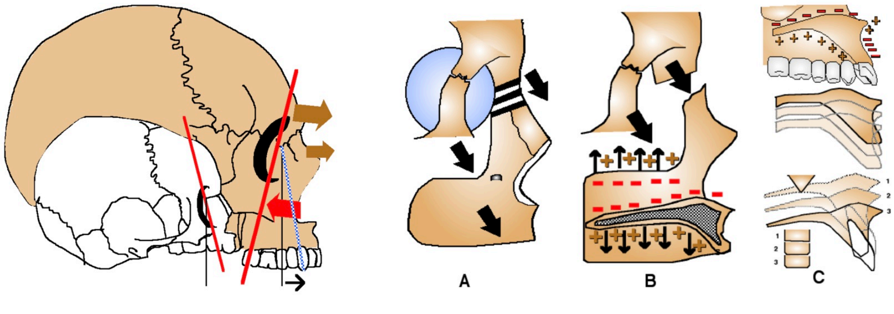

Figure 15. Schematic representation of maxillary posterior rotation (**left**) and vertical maxillary development, according to (Enlow, 1965). (**A**) Lowering of the maxilla under the influence of the forces from the expanding orbital contents, septal cartilage and muscle traction acting on the lower parts of the maxilla. (**B**) Effects of apposition- resorption phenomena of the periosteum on the maintenance, at the correct height and the descent of the palatal plane. (**C**) Schematic representation of the *relocation* of the bony palate which (according to Enlow) is lowered mainly by resorption of its upper surface and compensatory apposition of its inferior surface.

Those carried out on the Basion-Nasion and Sella-Nasion lines at the base of De Coster's or Björk's skulls clearly show the opposite. As for the dynamic role of tuberosity appositions invoked by Enlow, this is contradicted by what we know periosteal physiology, which is always secondary to skeletal displacement and never primitive.

In the vertical direction, Enlow exclusively supports the two phenomena of perimaxillary sutural growth and periosteal apposition/resorption (**Figure 15, right**). This phenomenon of *relocation* of the bony palate is highly disputed, in particular by Björk who showed that his lateral maxillary metal implant remained stable (was not eliminated) from the age of 4, which shows that his area implantation (at the level of the external maxillary process) does not undergo any bone resorption. It is therefore much more likely that the lowering of the palate occurs simultaneously with the lower part of the maxilla and according to the same processes (essentially sutural).

In the transverse direction, the mode of development described by Enlow (**Figure 16**) (which also favors external periosteal appositions/resorptions) is even more disputed, notably by De Coster (1952), Delattre and Fenart (1960), and Björk (1968).

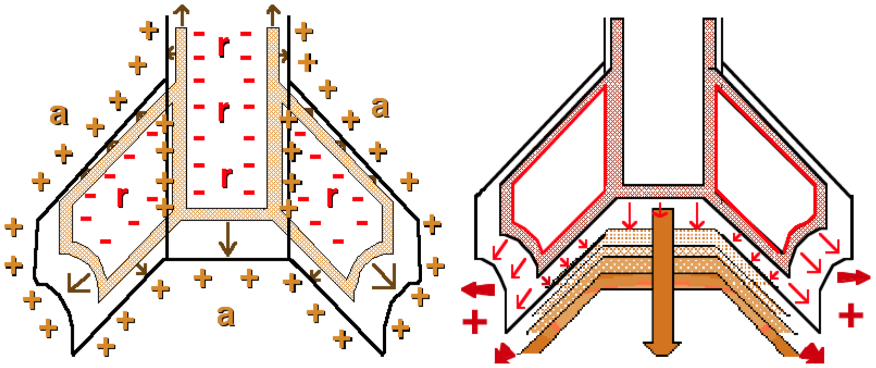

Figure 16. Left: Schematic representation of the transversal maxillary growth of sutural origin and especially periostal origin (diagram of Enlow and Moyers, 1971). It occurs mainly in its lateral parts (the maximum width of the nasal cavity is reached at 6 years of age) and would basically result from external appositions and internal resorptions (endonasal, endosinus and palate). **Right:** Mode of widening and deepening of the palatal vault (according to these concepts).

According to De Coster, around the *central nucleus* of the maxilla, corresponding to the nasal fossae (whose maximal dimensions are obtained by the end of the 6th year), there is in fact an *exo-peri-maxilla*, formed of cortical bone, able to move and develop under the influence of occlusal forces (as is observed in the frontal sinus).

In the same spirit, Delattre and Fenart (1960) have also individualized an endo-face (deep) and an exo-face (peripheral). As for Björk, he asserts that his lateral maxillary metal implant would be rapidly eliminated if there really existed peripheral appositions-resorptions as important as those described by Enlow.

Enlow's strongly-held belief that no sutural action would participate in the transverse expansion of the palatal vault, are not without practical consequences. Indeed, he formally condemns maxillary transverse expansion after the age of 6, an orthopedic therapy that he claims is *non-physiological* and even iatrogenic, a source of recurrence and even vestibular alveolysis.

Many clinical observations prove that this is not always the case. The explanation lies in the presence (too often overlooked) of a lateral maxillo-palatal sutural complex (**Figure 17**), extending the transverse palatal suture, located on either side between the outer surface of the vertical lamina of the palatal bone and the inner surface of the maxillary tuberosity (**Figure 18**).

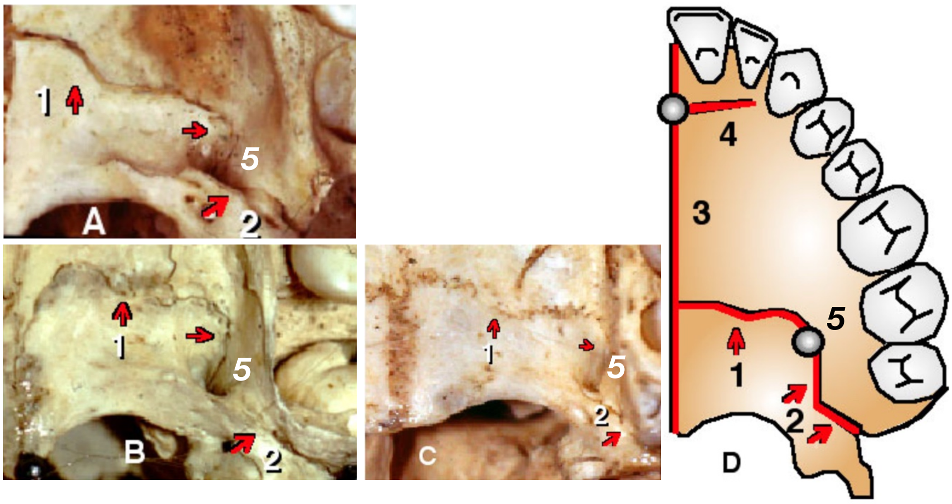

Figure 17. Lateral maxillofacial sutural complex: Aspects of the transverse palatine (1) and lateral (sagittal) maxillo-palatal (2) sutures at 4.5 years (**A**), 8 years (**B**) and 12 years (**C**). Schematic representation (**D**) of the position of these sutures in relation to the mid-palate (3) and incisivo-canine sutures (4). Greater palatine foramen (5).

Normally ensuring the separation of the tuberosity cortices and the advancement of the upper second molars (and Björk's lateral meticulous implants), the intermediate sutural blade at these bone surfaces seems to remain active during the normal period of eruption of the higher wisdom teeth until the end of pubertal growth.

Another reason to disagree with Enlow is because he totally ignored the premaxilla and its role in the development of the anterior parts of the maxilla, in contrast, in particular, to Broadbent (1937), De Coster (1952), Scott (1956) and Moss (1958). As early as 1937, Broadbent emphasized its early development in contrast to the relative stability of the posterior parts of the hard palate.

De Coster (1952), thanks to superimpositions carried out on his line of the anterior base of the skull, confirmed that after the age of six there is no further development of the parts of the maxilla located below this line. He opposes it at the still important growth of the naso-premaxillary complex (secondary, according to him, away from the anterior cortex of the frontal sinus and maxilla).

Moss (1958) confirmed these facts, created the term *intramaxillary growth* and confirmed the individuality of the premaxillary skeletal unit. Scott, in 1956, perfectly described the anatomical and developmental particularities. The author also carried out a number of studies on this subject, particularly in relation to its development factors.

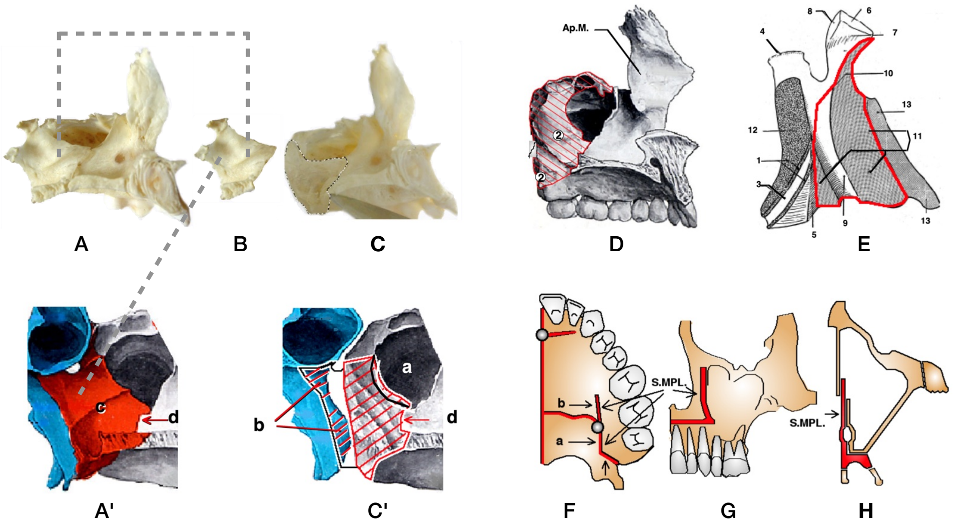

Figure 18. Upper left: Specimen of a 2-year-old child. Skeletal aspect of the lateral wall of the left nasal cavity. (**A**) Vertical palatal bone in place, (**B**) palatal bone removed, (**C**) palatal bone absent. **Lower left:** Schematic representations (same view as above) of the maxillary and palatal bone surfaces separated by the lateral sagittal maxillo-palatal sutural lamina (Rouviere, 1920). Os sphenoidale and Proc. pterygoideus in blue. (**A'**) Palatal bone in place. (**C'**) Palatal bone absent, (a) extent of the maxillo-palatine and (b) pterygo-palatine sutural areas to the (c) vertical part of the palatal bone. Note the presence of a *palatal fissure* (d) which is actually *maxillary* into which the *sinus segment* of the palatal bone penetrates. **Upper right:** (**D**) Inner side of the left maxilla, (2) the sutural area. (Ap.M) apophysis maxillaire (Proc. frontalis). (**E**) External face of the right palatine. The vertical lamina of the palatine bone occupies the entire territory behind the vast entrance hole (hiatus) of the maxillary sinus. Its anterior part (a) extends, from front to back, from the sinus hiatus to the gutter of the posterior palatal canal (slightly oblique downwards and forwards). Its middle part (b) corresponds to the posterior palatal canal. Its posterior part (c) connects the maxillary tuberosity to the anterior surface of the pyramidal process of the palatal bone (embedded between the ends of the medial and lateral plates of the pterygoid process). (1) Pyramidal process, edges of posterior surface (2) Proc. maxillaris, (3) pyramidal process (pterygoid fossa), (4) sphenoidal process, (5) pyramidal process (anterior face), (6) ethmoidal face, (7) orbital face, (8) sphenoidal face, (9) posterior palatal gutter, (10) inter-pterygo-maxillary segment, (11) maxillary segment, (12) pterygoid segment, (13) segment sinusian. **Lower right:** Schematic representation of the position (in projection) of the lateral maxillo-palatine suture (S.MPL). (**F**) palatal view, (**G**) frontal view, (**H**) horizontal section (on the dry bone, only the posterior palatal part of the suture is visible).

2.6 Growth of the premaxilla

2.6.1 Observed phenomena

According to Augier (1912), the human maxilla and premaxilla are derived from two small bony islands which appear at the beginning of the 7th embryonic week, in the future canine region (**Figure 19, A**). By the 8th week (**Figure 19, B**), however, their external cortices are fused, giving rise to an incomplete suture, extended on each side from the midline nasopalatine canal to the alveolus (Cadenat, 1924). Another suture, median interincisive, is clearly seen, extending from the nasopalatine canal to what will become the apex of the anterior nasal spine. The two hemi-premaxillas are well delimited between each other and with respect to the rest of the maxilla (located behind incisivo-canine sutures), the development of which will fundamentally arise from local factors that will stimulate each of them (**Figure 19, C-G**).

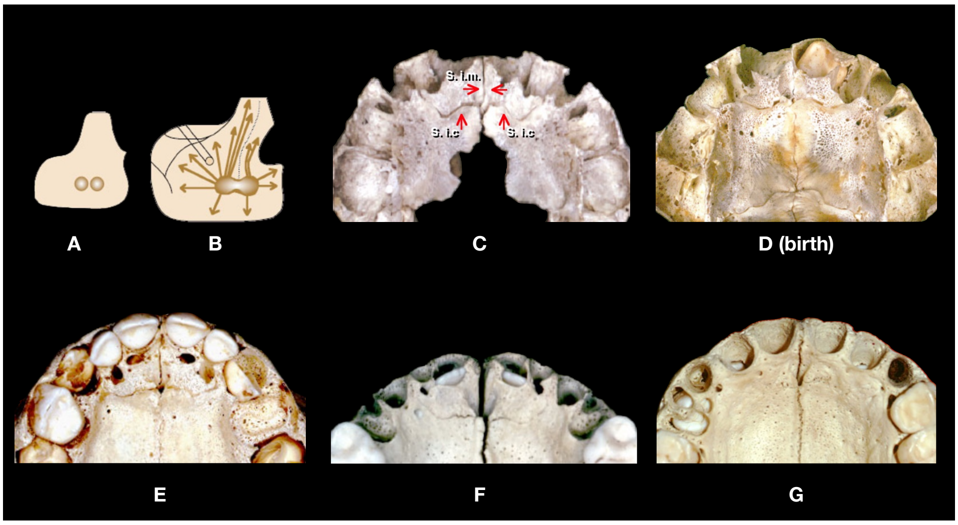

Figure 19. Early stages of development of the premaxilla. (**A**) 7th embryonic week, the first two points of ossification of the premaxilla and maxilla. (**B**) 8th embryonic week, fusion of *primary* points and peripheral extension of maxillary ossification. (**C**) Premaxilla (fetus about 7 months). Medial inter-incisor suture (S.i.m). Incisivo-canine suture (S.i.c). At birth the incisivo-canine suture extends from the inside between the upper deciduous canine tooth buds (**D**). At 18 months, it stops a little before reaching these tooth buds (**E**). At 6 years old its external parts are in the process of synostosis (**F**). At 8 years, the remainders of this suture are usually limited to the immediate vicinity of the nasopalatine canal (**G**).

At birth (**Figure 19, D**) the incisivo-canine suture extends from one side of the alveolus of an upper deciduous canine to the other. At 18 months (**Figure 19, E**), it ceases to extend a little before reaching these alveoli.

At 6 years old (**Figure 19, F**) its external parts are in the process of synostosis. At 8 years (**Figure 19, G**), the remains of this suture are sometimes already limited to the immediate vicinity of the naso-palatine canal. Most often, however, this very important reduction in visibility of the incisivo-canine suture occurs in adolescence. The inner part of the suture may even remain open in some adults.

These medial and incisivo-canine inter-incisal sutures are not only visible on the oral side of the palatal vault. They are also found at the nasal surface of the latter where they are even, often, more marked and more long-lasting. On the other hand, the incisivo-canine sutures (right and left) also include a vertical extension, rising higher or lower (depending on the age) on the internal surface of the ascending process (**Figure 20**).

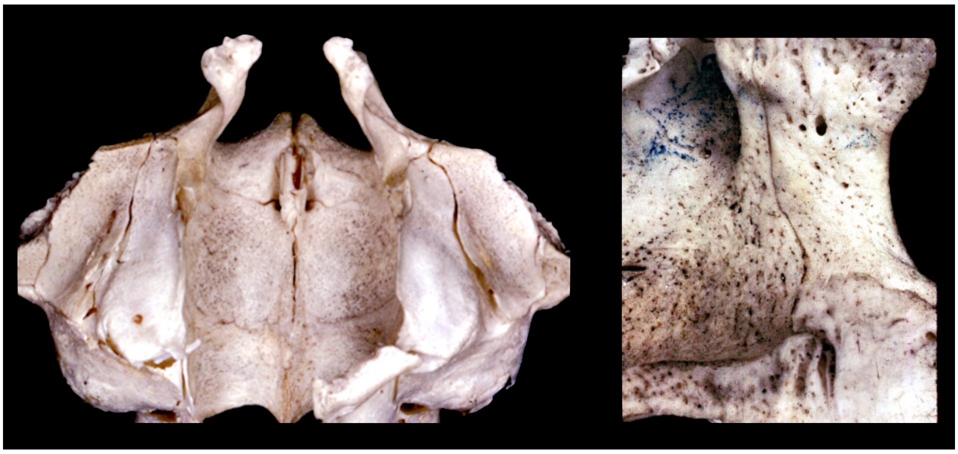

Figure 20. View of the floor of the nose showing the incisivo-canine (*premaxillo-maxillary*) and median inter-incisal sutures (details in the text). **Left:** Nasal floor with various palatal sutures in a 20-month-old child. **Right:** Detail of the vertical extension of the incisivo-canine suture to the internal surface of the maxillary ascending process in a 30-month-old child.

Given the direct relationship between the appearance of the sutures, their arrangement, their evolution, and the forces that apply to the skeletal elements they separate, it can be deduced that the two hemi-premaxillas perform two types of movements.

Firstly movements like a *double opening bridge*, advancing mainly their medial parts, spreading the sutural sides between each other, as well as the anterior sides of the incisivo-canine sutures (**Figure 21, top**). The reality of these movements, at a young age, is demonstrated by the presence of newly formed osteoid bone on both sides of the sutures and is of comparable size. It is likely that from 2-3 years, these *en bloc* movements of the two hemi-premaxillas are replaced by comparable movements of the anterolateral cortices, which explains why, after this age, incisivo-canine sutures no longer show signs of osteogenesis, their function is subsequently reduced to damping the incisive occlusal forces as well as the role of a *hinge*.

Secondly a hinge function (**Figure 21, bottom**), allowing the raising or the lowering of the anterior parts (nasal spine) of each hemimaxilla, attested by the existence of a kind of pseudo-condylar joint between the posterior part of the incisal crest and the corresponding part of the secondary palate.

2.6.2 Agents (factors) of these phenomena

They are twofold. First and foremost, dental through expansion (**Figure 22**) of incisive dental buds (deciduous then permanent) and which Broadbent (1932) and De Coster (1932) both have placed particular emphasis on the precocity of premaxillary development and its hypodevelopement in cases of incisor agenesis. Secondly, there are muscular factors, with lingual thrusts at the back and, anteriorly, upper lip traction transmitted to the anterior surfaces of each hemi-premaxilla by the *incisor* muscles and to the medial inter-incisor suture by the medial septum of the lip.

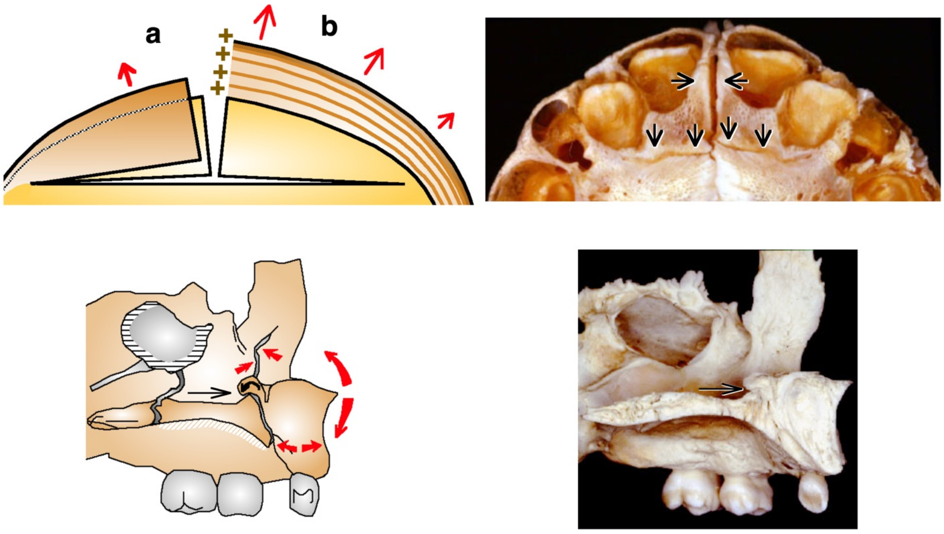

Figure 21. Upper left: Diagram showing the *double bridge opening* movements of the hemi-premaxillas (a, b) and their anterolateral cortices, generating the incisivo-canine sutures (*premaxillo-maxillary*). **Upper right:** Aspect of incisivo-canine and medial intercisal sutures, in a newborn. Note the presence of osteoid tissue at the margins of these sutures (arrows), testifying to their osteogenic activity. **Lower left:** Diagram showing the vertical movements of the premaxilla in relation to the anterior part of the bony palate (red arrows) and the situation of the pseudo-condylar joint that is formed here (black arrow). **Lower right:** Internal view of the left maxilla of a 4.5 year old child, showing this condylar pseudo-articulation (black arrow).

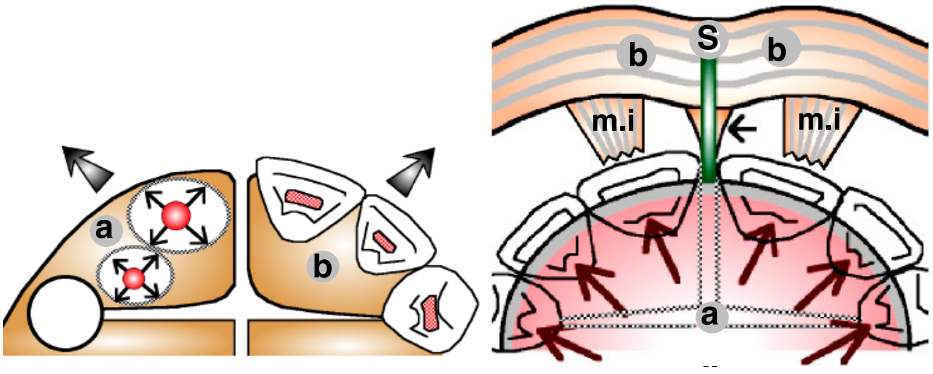

Figure 22. Agents of premaxillary development. **Left:** Dental factors. (a) expansion of deciduous and then permanent incisor tooth buds, (b) occlusal forces against the lingual surface of the upper incisors and canines. **Right:** Other factors. (a) tongue thrusts (b) forward traction of the upper lip, transmitted to the premaxilla by the *incisor* muscles (m.i) and to the median inter-incisor suture by the medial septum of the upper lip (S).

2.7 Growth of the mandible

According to modern classical authors, the entire development of the mandible is due essentially to *secondary* adaptive growth of the condylar cartilages and periosteum of the posterior parts of the ascending mandibular rami, under the influence of mandibular advancements (**Figure 23**).

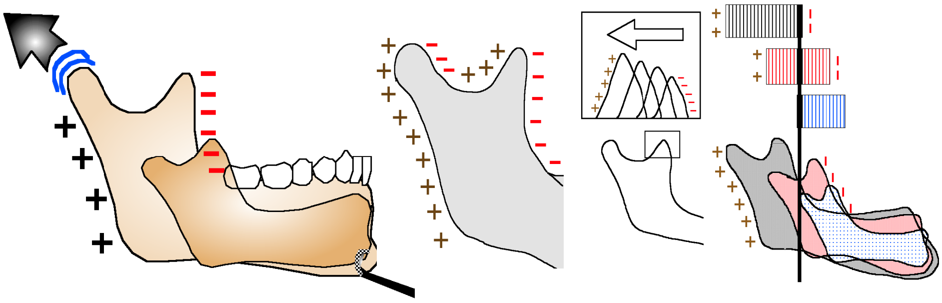

Figure 23. Left: Schematic representation of mandibular growth, by bone apposition below the condylar cartilage (*secondary* type) and periosteum of the posterior parts of the ascending rami at the expense of the distal mandibular body (Enlow, 1965). **Right:** The process of *relocation* invoked to explain growth posterior of the mandibular body.

This concept results from a misinterpretation of the results obtained by Humphry (1878) who inserted metallic threads into the lower jaw of still growing young pigs to study their growth. In fact it is contradicted by the two other diagrams of the same author present in his article and also by the condylectomies performed by Sarnat in young monkeys, which showed only localized changes to the condylar processes and their underlying ascending rami (Sarnat and Engel, 1951). The mandibular body was practically unchanged.

In humans, there are also cases of condylar agenesis or even absence of the entire ramus of the mandible in which there is no significant reduction of the dimensions of the mandibular body. The very important downward growth of the ascending rami that this interpretation would require would not exist if, instead of a symphyseal or condylar mandibular registration point, the line of De Coster (1932) is used, where the *post-maxillary plane* which, according to Enlow, is *"the only interface that is consistent during growth and development of the skull and the face"* (Rozencweig, 1994).

Finally, in his remarkable works, comprising successive superimpositions with registration on the (intra-sphenoidal) R-point, Broadbent (1931; 1932) insisted on the existence of very important growth of the region of the mandibular angles, associated with that of the ascending rami in these terms:

> *"The corresponding increase in size in the mandible has taken place in the length of the body where it joins the ramus along the line between the internal angle and the mandibular notch. The area of the mandible along the line between the body and the ramus appears to be comparable to the epiphyseal line of the long bones."*

In the last re-editions of his book *Essentials of Facial Growth*, Enlow (1996) took account of this work, firstly by reducing, in his drawings, the importance of the resorption of the ascending rami, on the other hand by introducing a mandibular tuberosity, physiologically comparable to the maxillary tuberosity and normally providing the same amount of posterior growth of the mandible as the latter (**Figure 24, upper row**).

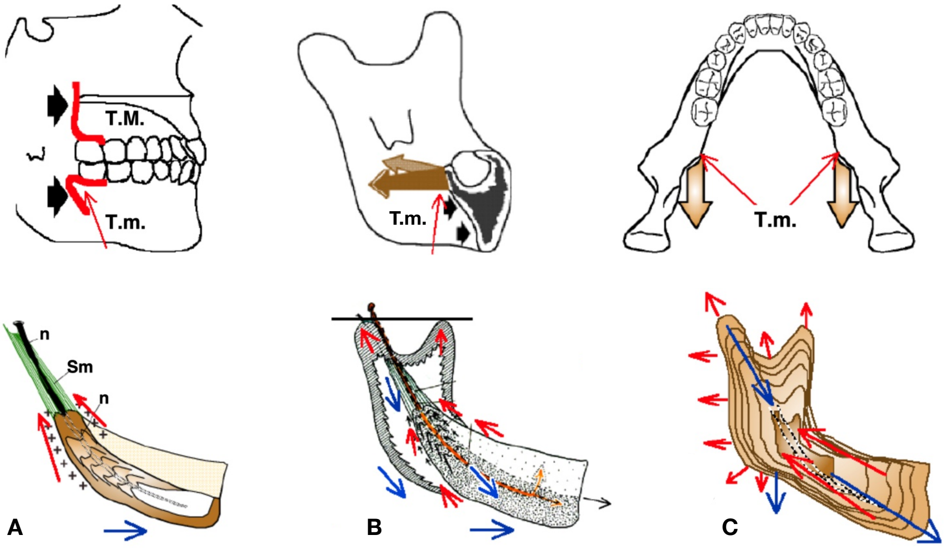

Figure 24. Upper row: Schematic representation of maxillary and mandibular tuberosities and their growth directions (Enlow and Hans, 1996). (T.M.) maxillary tuberosity, (T.m.) mandibular tuberosity. **Lower row:** Schematic representation of the sphenomandibular ligament (Sm) and periosteal appositions, in response to mandibular advancement (**A**). Periosteal appositions following advancement of the mandibular body (**B**). Schematic representation of the development of the ascending rami and body of the mandible. Blue arrows: Direction of mandibular movements. Red arrows: Direction of skeletal growth (**C**).

The hypothesis of mandibular tuberosity can not be supported, firstly because, as in the maxilla, the alveolar tuberosity cannot cause displacements of the bone elements. Indeed, on the contrary, these determine the superficial and alveolar periosteal compensations, and secondly because the growth of the mandibular body occurs almost normally in the total edentulous.

Much more likely, the site of posterior growth of the mandibular body is located in the spine region where the sphenomandibular ligament (anchored at the other end to the sphenoid spine) is inserted (**Figure 24, lower row**). This ligament, in fact, is virtually inextensible, so that during advancement of the mandibular body, its energization causes traction on the mandibular periosteum into which the ligament is inserted and compensatory ossification. A comparable system exists at the level of the anterior nasal spine where the septo-premaxillary ligament and the nasolabial muscles are inserted.

To fully understand the development of the mandible, one must also know the exact nature of the condylar cartilage and its true physiology of growth. Sicher (1947) addressed this question:

> *"The condyle of the mandible contains a cartilage up to the late twenties, but this cartilage has been very much misrepresented in the literature, some maintain that it is an articular cartilage; others identify it as an epiphyseal cartilage, analogous to that existing in long bones. It is neither. In a young individual the bony part of the mandibular condyle is overlaid by a cap of cartilage, but this in turn is covered by a thick layer of connective tissue which continues in the periosteum of the mandibular neck."*
>
> *"Cartilage just as it stands physically between soft connective tissue and hard*

> *bone, can grow, and grows by both modes of growth, interstitially, that is, by expansion, and appositionally by addition."*
>
> *"The growth of the cartilage in the mandible, therefore, may be inhibited or stimulated by factors, which do not interfere with the interstitial growth of cartilage in other bones. For instance, chondrodystrophic dwarfs, their mandible is not smaller; on the contrary, very often it protrudes ..."*

Petrovic (1972) confirmed the particular nature of the condylar cartilage, which he describes as *secondary*, and emphasized its adaptive properties. In fact, this cartilage is periosteum which is not yet chondrified at birth (Testut, 1895), but which rapidly becomes so under the effects of the movements and forces acting on it during suckling and mastication. This periosteal nature was well demonstrated experimentally by McNamara and Graber (1975) using bi-maxillary blocks practiced in Macaca mulatta, responsible in only two months for the disappearance of the cartilaginous tissue and the return to the initial membranous cuff in direct continuity with the periosteum of the ramus.

Therefore, it is logical to describe the temporomandibular joint as an *open suture* (Couly, 1975) or even as a *dislocated suture*, which makes it possible to better understand the favorable role of condylar advancement on the growth of the condyle and its neck. The presence of cartilage, even if it is not a true growth cartilage, also explains the excessive mandibular increase caused by large condyles (with more cartilage) hypercondylies and conversely (in hypocondylies).

From these data concerning the different growth rates of the mandibular body and the ascending rami, it should be noted that, in dentofacial orthopedics, an insufficiency of height of the ascending rami will require activation of the condylar growth, if the insufficiency is not too significant, or a surgical elongation, if it is too severe. In contrast, a reduction of the length of the body requires, as the main activator of growth, good posture and function of the lingual mass.

2.8 Balance of the cranio-facial skeleton

Already Broadbent (1931), in his lead article on radiologic cephalometry wrote:

> *"Growth of the face is not the complex erratic process that it seems to be by similar superimposition of tracing from craniostatic drawing of skulls of dead children."*

The cranio-facial skeletal *construct* develops, in fact, strictly respecting the universal laws that regulate the harmonious and balanced states of all the other constructs present in the cosmos and the phenomena that occur there. These laws are basically those of *Equivalence of Matter and Energy, Global Harmony of the Universe, The Mathematical Universality of the Cosmos* to which, in living beings, must be added *Vital Laws* that characterize them.

1. Law of Equivalence of Matter and Energy. It was well summed up by D'Arcy Wentworth Thompson (1917), according to which:

   > *"The form of any portion of matter whether it be living or dead and the change of forms which are apparent in its movements and in its growth, may in all case alike be discribed as due to the action of force. In short, the form of an object is a diagram of forces."*

2. Law of the harmony of all the constituents of the universe. According to Cuvier (1825), it postulates that:

   > *"Every organized being forms a set, a unique and closed system whose parts mutually correspond and contribute to the same final action by a reciprocal action. None of these parts can change without the others changing too. Consequently, each of them, taken separately, indicates and gives rise to all the others."*

3. Law of mathematical universality of the cosmos. Known since the highest antiquity (Pythagoras), this law was enacted in these terms by Plato:

   > *"Everything is arranged according to the number."*

   For Galileo:

   > *"The book of nature is written in mathematical language, the characters are triangles, spheres and other figures, without the help of which it is impossible to understand a single word."*

   According to D'Arcy Wentworth Thompson:

   > *"The different parts of a whole, even if they are not directly shaped by the action of physical forces, all adopt an optimal geometrical form, which materializes the solution of a morphological problem."*

4. Vital laws = specific properties of living matter (living beings). *Life* obeys very scrupulously the fundamental *universal* physical laws, nevertheless it differs profoundly from inorganic phenomena, by (i) its autonomous development (by cellular multiplications and intra and extra cellular accumulations), (ii) its extraordinary creative inventiveness (even its fantasy), (iii) its marvelous adaptability in response to the variability of external and internal influences that all *organisms* face.

Moreover, if life respects the imperatives of universal mathematics, it does not accept fixity. This explains the multiplication and evolution of different species, their final diversity, the originality of each of them, their great differences, despite their fundamental similarities. In all, diversity is the essence of life. There are not two absolutely similar beings. This condemns conventional cephalometric analyzes whose *norms* are, in fact, only statistical averages.

These considerations have been the basis for the development of *architectural* cephalometry, the aim of which is, firstly, to trace the optimal individual pattern of the craniofacial skeleton of the patient, and secondly to compare the relationship between the components of the craniofacial skeleton.

This optimal scheme is itself inferred from the work of Leroi-Gourhahn (1954) which showed that the cephalic skeleton of all vertebrates basically comprises two mechanisms (postural and masticatory) and an intermediate *damping* system of forces generated by them.

The concept I personally developed (**Figure 25**) is that the *postural* region has two triangles, one *equilateral*, representing the vault of the skull, the other representing the base of the skull. The masticatory mechanics area below is represented by a triangle with its base lying anterior. The intermediate damping system interposed between the preceding lines consists of a parallelogram whose principal lines coincide with the hypotenuse of the right triangle representing the skull and the upper line of the masticatory triangle.

The cranio-facial architectural analysis, which has been developed from it, represents the optimal harmonious skeletal balance of the subject, with respect to which all its anomalies will be highlighted. In the normal state, therefore, the *orthognathic* lines objectifying the peculiarities of the subject coincide exactly with the architectural reference lines.

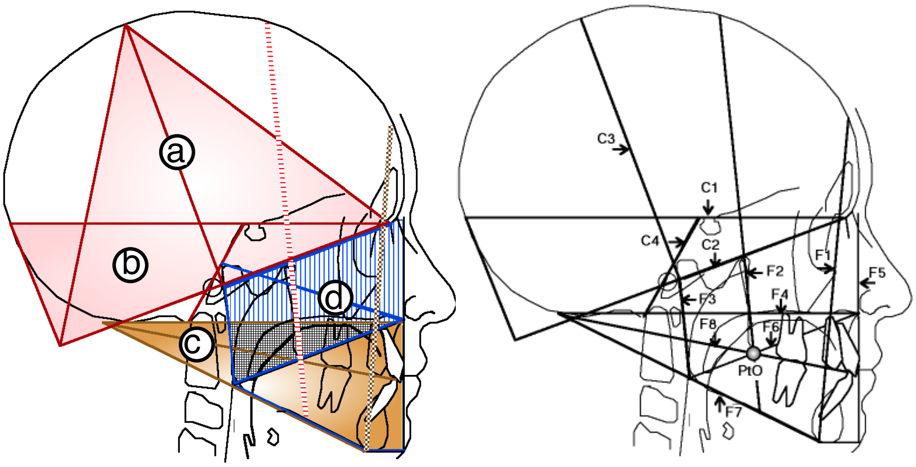

Figure 25. **Left:** Schematic geometric representation of the optimal balance of the Caucasian craniofacial skeleton: (a) the *postural* equilateral triangle of the vault of the skull, (b) the *postural* right-angled triangle of the base, (c) the *masticatory* triangle, and (d) the facial parallelogram. **Right:** *Architectural* cephalometric analysis to highlight the subject's anomalies.

Any shift in these lines easily highlights the nature and importance of dysmorphosis, thus making it possible to determine and quantify the best treatment (**Figure 26**). Architectural cephalometry also includes traces of the soft tissues (cutaneous profile, velo-lingual-pharyngeal spaces), normally in harmony with each other and with the skeletal elements. Interesting data can thus be obtained concerning, for example, the major potential role of maxillary osteotomies in the correct repositioning of the lingual mass (**Figure 27**).

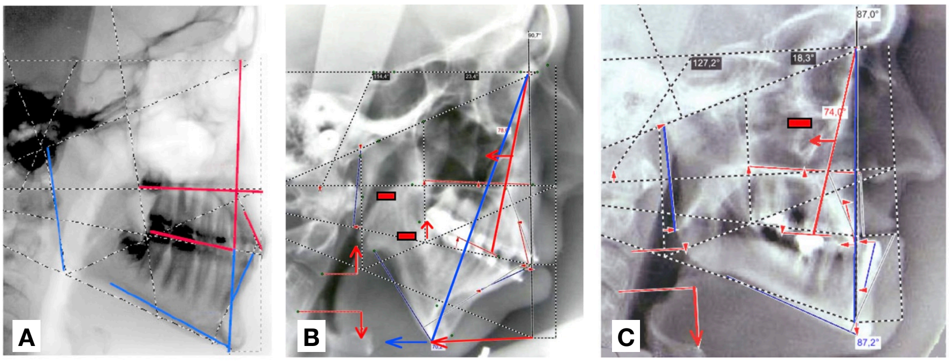

Figure 26. Architectural analysis. (**A**) Harmonious balance of a normal orthofrontal Caucasian. The (colored) features of the orthognathic analysis coincide exactly with those of the architectural analysis of reference. (**B**) Orthognathic analysis of a mandibular micrognathia and (**C**) a Class III due to a retromaxilla.

The benefit of this cephalometric approach, the conceptual basis of which is derived directly from the laws of cephalic morphogenesis, clearly demonstrates the abnormalities of skeletal development responsible for dentofacial dysmorphoses, and those of the soft tissues that accompany them. The Orthodontist and the Surgeon can easily deduce the treatment which, by acting on growth, will be all the more effective the earlier it takes place.

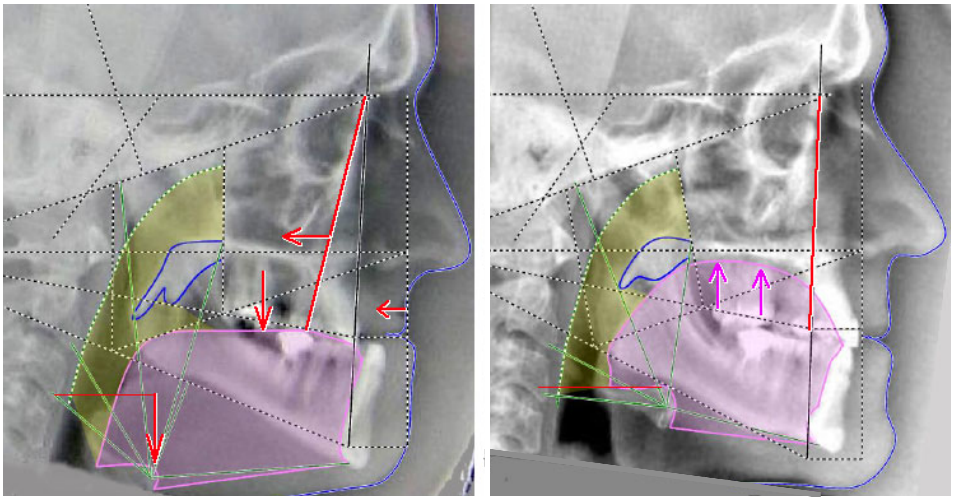

Figure 27. Example of the effects of maxillary advancement osteotomy on the position of the lingual base and hyoid bone. (**Left**) Before the osteotomy and (**right**) two years later (courtesy of P. Delcampe).

2.9 Examples of physiological treatments in maxillofacial surgery

These treatments can be preventive, interceptive and curative with respect to (i) the different growth sites of the facial skeleton, the premaxilla, the tuberosity regions, the mid-palatal suture, the condylar region and the bony chin, as well as (ii) the soft tissues acting on them, including the masticatory muscles, the nasolabial muscles, the muscles acting on the chin and the muscles of the soft palate.

The functional treatment of fractures of the mandibular condyle is one of the most interesting treatments whose application has revolutionized the post-therapeutic prognosis. Fifty years ago, in fact, the treatment of these fractures consisted of a systematic intermaxillary wire fixation obtaining good occlusal relations, considered to be the position of optimal function. After 3-4 weeks, the fixation was released and the jaw actively mobilised, with forced mouth opening using a gag, so as to improve ultimate function. Unfortunately, the most frequent outcome was less than satisfactory mouth opening of no more than two centimetres inter-incisally on average.

A much better treatment is based on the fact that the temporomandibular joint is actually a moving joint. Rather than immobilisation, the mandible should be subjected as soon as possible to active forward and lateral movements under the influence of Class II intermaxillary elastics which return the mandible to the correct occlusion. The results obtained by this therapy are usually excellent, not only in cases of non-dislocated fracture of the condylar head (**Figure 28**) but also in fractures with internal dislocation of the condylar fragment (**Figure 29**).

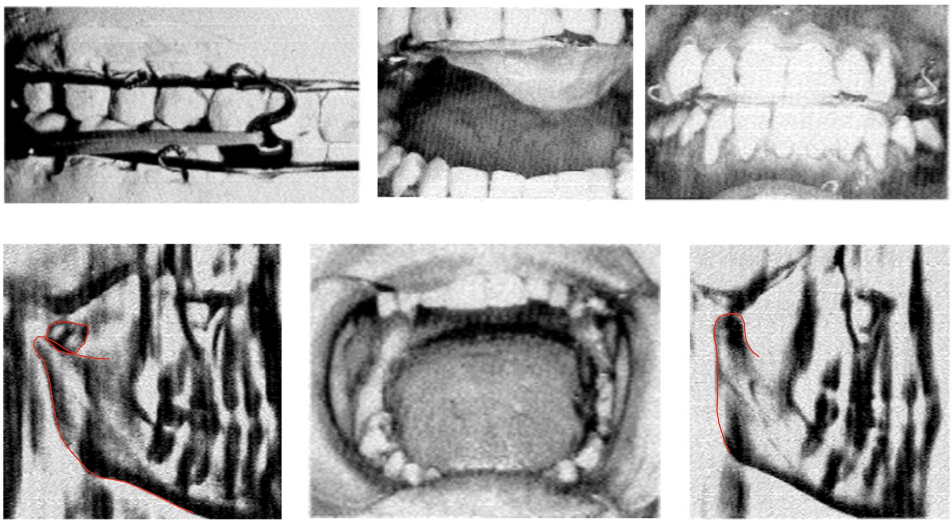

Figure 28. Upper row: Example of devices used for fractures of the right mandibular condyle or its neck. Oblique unilateral traction for right fracture (**left**), anterolateral inclined plane (**middle**) forcing the mandible to move forward and left (**right**), during mouth closing. **Lower row:** Example of a result obtained after functional treatment of a fracture of the condyle. The right condylar fracture (**left**). The mouth opening (**middle**) six months after the accident; note the extent of the opening without any lateral deviation. The state of the condyle five years later; note its good mobility (**right**).

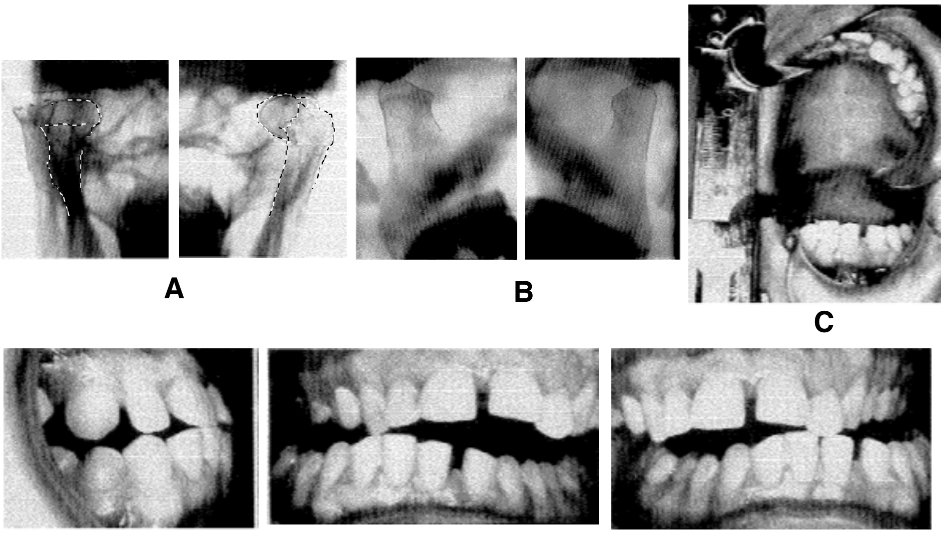

Figure 29. Subcondylar fracture with medial dislocation of the condylar heads (**A**), position of the condyles six months later (**B**), mouth opening at the same time (**C**). (**Lower row:**) Forward and lateral mandibular movements.

The early physiological treatment of Class III by postero-anterior orthopaedic traction on the maxilla is also one of the preventive treatments of orthognathic surgery. Not so long ago, Class III was considered by orthodontists as impossible to correct and therefore systematically assigned to the surgeon who would wait till the end of growth before intervening. Based on the physiology of facial sutures and the ability to stimulate transverse maxillo-palatal growth with devices that spread their bony margins, I have since 1969 used extra-oral elastic forces anchored on a metal arch ligated to the whole lower and upper dental arches as well as hooked on to a fronto-maxillary support called orthopedic mask (**Figure 30**).

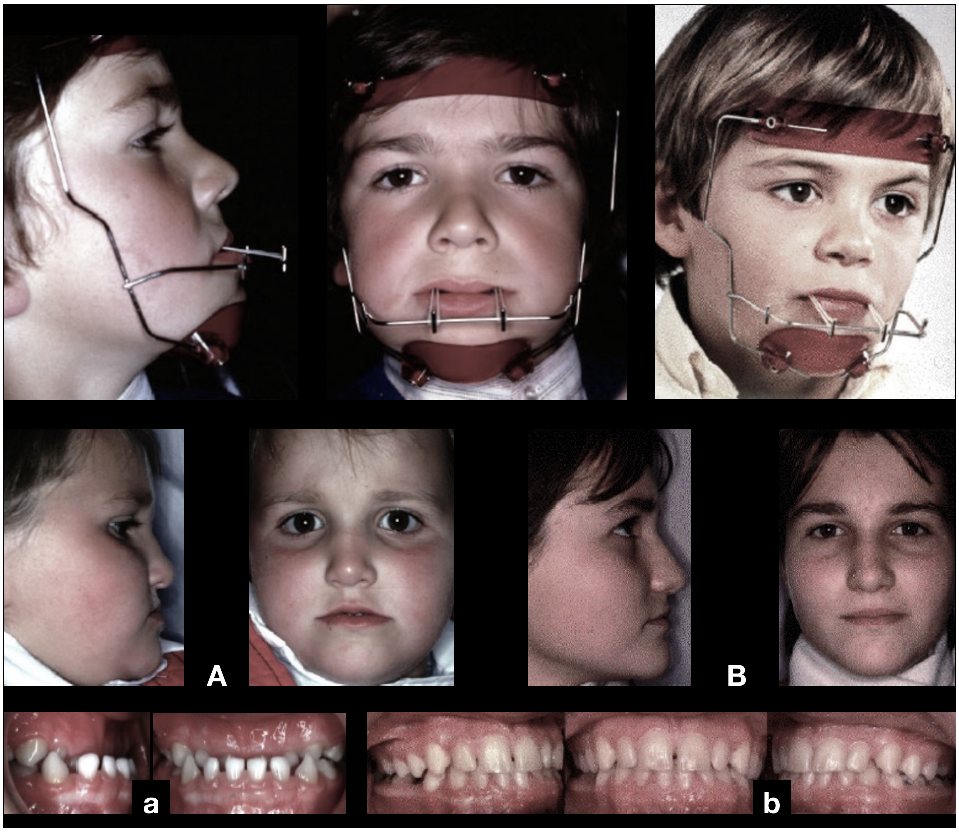

Figure 30. Upper row: An example of orthopedic mask with lateral bars, for nocturnal postero-anterior traction on the upper dental arch. **Lower rows:** Example of results obtained by extraoral posterior-anterior traction in an early treated Class III. Patient H., five years old, before extra-oral tractions (**A, a**). Same patient, 13 years old, after one year of extra-oral posterior-anterior traction plus one year wearing a Balters III appliance (**B, b**).

These relatively heavy forces (1200 to 1600 grams) should be applied only at night. Previous maxillary expansion is required when the maxilla is transversely narrowed. During the day and after stopping the traction, a Balters III tongue-guide appliance (Balters, 1955) is worn to help normalize swallowing. The results of this treatment are even better when the subject is younger (**Figure 30, 31**).

The age of six to nine years has clearly emerged as the best period for its implementation and is therefore systematically recommended. In the best cases, normalization of occlusal relations does not take more than 9-12 months. In boys, a slight tendency to relapse can

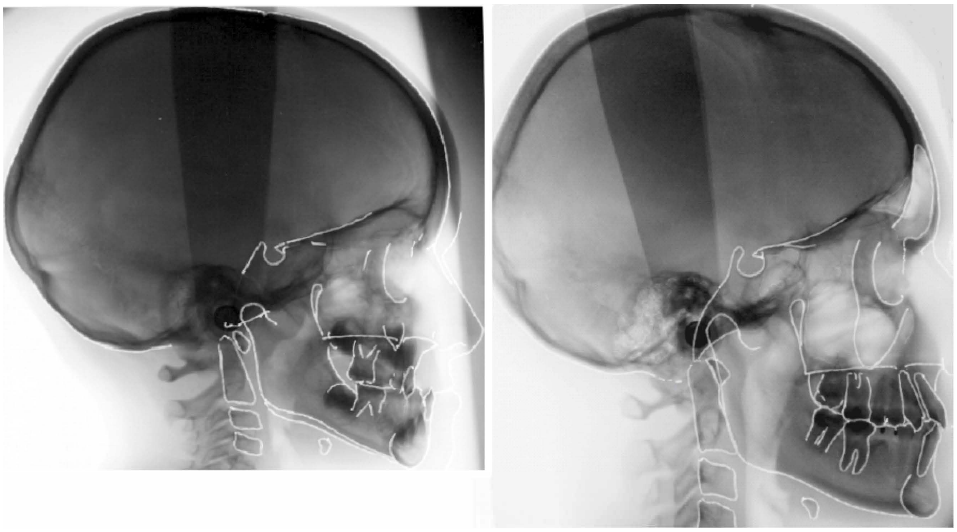

Figure 31. Lateral skull radiographs and cephalometric tracings of the patient in Figure 30, before (**left**) and after treatment (**right**).

be observed during puberty, which is usually quickly brought under control by resuming treatment and strengthening the regulation of swallowing, chewing and nasal ventilation functions. At present, this treatment is very widely used with, on the whole, very good results, except in subjects treated after 11 years (all the failures of which I knew personally had been treated after this age). The mask has prevented many operations for Class III correction at the end of growth. Orthodontists and surgeons must be familiar with the possibilities and limitations of this technique to effectively counsel their patients.

Interceptive physiological condylar surgery is an important part of this subject. Based on the double *adaptive* (membranous) and *constitutional* (cartilaginous) potentiality of the condylar cartilage. Either by very early orthopaedic treatment, if possible as early as six years old, or by slightly more delayed surgery at 11-12 years old, it is possible to prevent most of the associated alveolar and maxillary anomalies, which (in the absence of such treatment) develop in adolescence and sometimes make surgery very difficult in adulthood.

A good example is the development of the so-called appearance of *camel's humps* in condylo-mandibulo-dysplasia, usually considered to be a clinical variety of hemifacial microsomia. In fact, it is quite different since there is no involvement of the ears, conjunctiva and labial commissures (**Figure 32**). The very peculiar aspect of the upper part of the affected mandibular ramus (the condition is always unilateral) is pathognomonic with a condyle reduced to a small, rounded apophysis, contrasting with a tapering corona.

In subjects seen later the results of orthopaedic treatment are more uncertain. In these cases, chondro-costal grafting may be preferred to orthopedic treatment, as it is less restrictive overall (**Figure 33**). In both cases (orthopedic and surgical), the maxilla adapts to mandibular improvements in all three directions (sagittal, vertical and transverse), thus avoiding the need for bimaxillary surgery in adulthood (classic in the past).

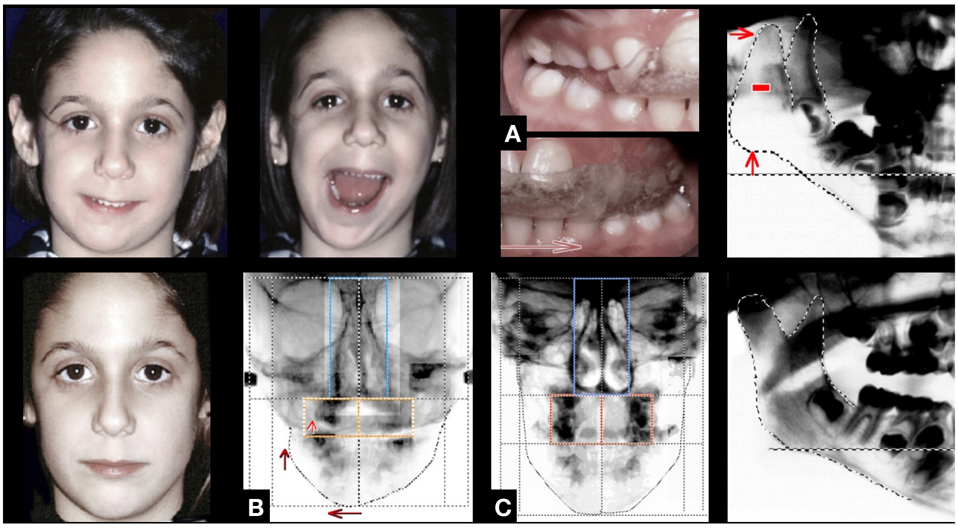

Figure 32. Upper row: Right condylo-mandibulo-dysplasia of a 5.5 years old patient at first appointment. (**A**) Orthopedic treatment with occlusal plates deviating the mandible to the left. The panoramic x-ray represents a *camel's hump* like shape of the right ramus and condyle region. **Lower row:** Enface photograph at the end of treatment at 8 years. Frontal cephalometric analysis before (**B**) and after treatment (**C**). Note the large volume of the neoformed right condyle in the panoramic x-ray.

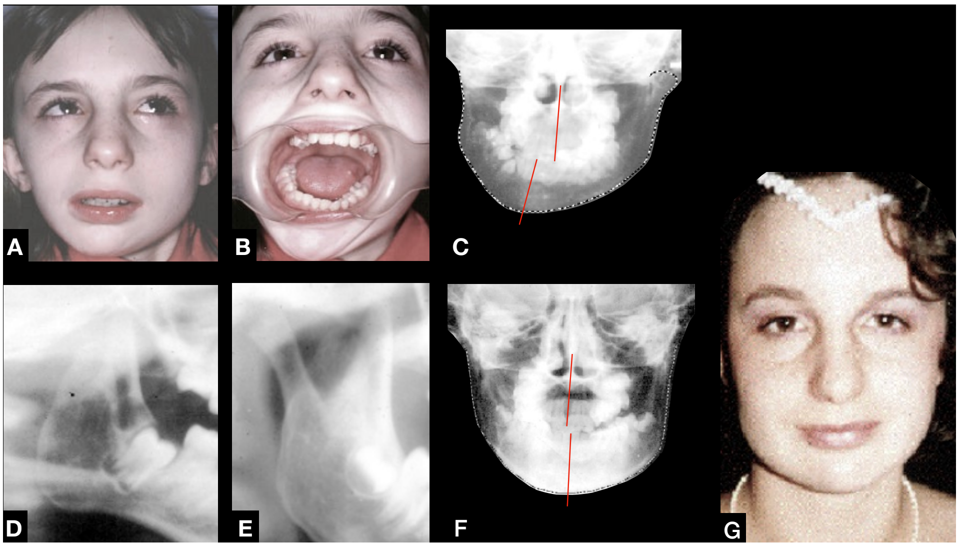

Figure 33. Patient with condylo-mandibulo-dysplasia, 12 years old at first appointment (**A-C**). Deviation to the affected side (**C**) with *camel's hump* like shape of the right ramus ascendens in the panoramic x-ray (**D**). Results of the chondro-costal graft placement before the operation at 12 years old (**D**), at 14.5 years old (**E, F**) and at 24 years old (**G**).

All forms of hemifacial microsomia, including bilateral, are amenable to the same early surgical treatment, although the results are generally less good because of the muscular agenesis which always accompany the bone abnormalities and hamper mandibular movements, essential for good condylar cartilage activity. In this situations the maxillary improvements that accompany the forward and downwards growth of the mandibular body make late surgery, when growth has ceased, both less extensive and more effective. The pathologies to which these therapeutic principles apply also include temporomandibular ankylosis in young patients.

Hypercondylar problems are subject to the same concepts. Unlike the preceding cases they are characterized by hyperactivity of growth of one of the condylar cartilages (the anomaly is usually unilateral) presenting as a large condyle, elongation of the condylar neck, increased height of the ascending ramus, ipsilateral lowering of the occlusal plane and, more often than not, deviation of the chin towards the opposite side (**Figure 34**).

The increased activity of the condylar cartilage cells, as demonstrated by scintigraphy, are responsible for the condylar enlargement. It is interesting to note, however, that subjects with this syndrome usually have a more or less apparent hemi-hypertrophy of the whole body, including the upper and lower limbs (and breasts in the females). It is therefore not unreasonable to think that condylar hyperactivity is essentially the result of an increase in the size of the cartilage covering the large condyle, which is an additional argument for early treatment of the condyle, by resection in proportion to the excess height observed. Results are rapid and highly satisfactory (**Figure 34**).

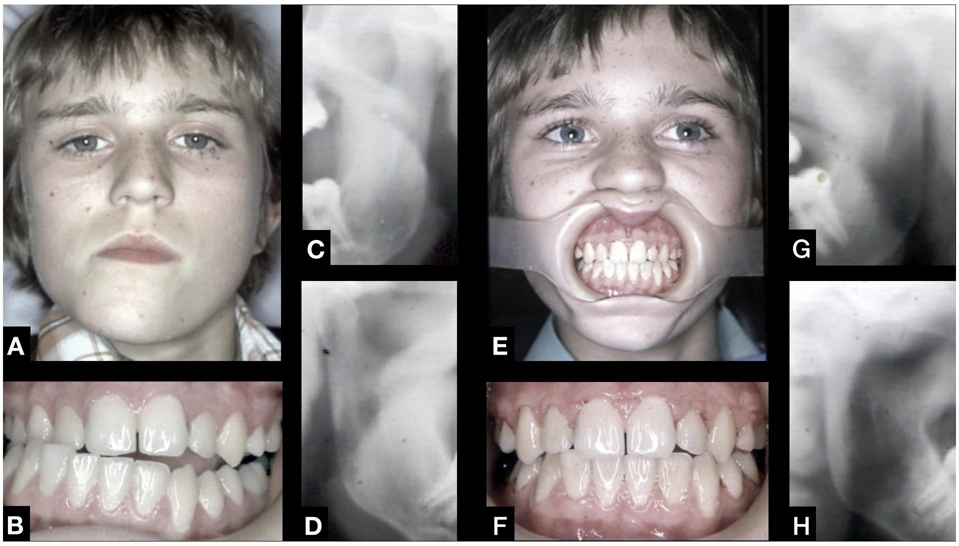

Figure 34. Patient with left hypercondylia, 12 years old at first appointment (**A**), presenting a *mixed* form, both lateral and vertical, with deviation of the lower interincisal midpoint to the contralateral side with a lateral open bite ipsilateral (**B**). Note the significant volumetric increase of the left mandibular condyle and the length and width of its neck (**C**), compared to the right side (**D**). **E-H:** Same patient 4 months after the left partial condylectomy at the day of removal of nocturnal intermaxillary elastic traction. Transverse and vertical asymmetries have gradually been corrected. Dental occlusion will further improve with the return of normal chewing and swallowing.

In the absence of this early operation, the mandibular and maxillary abnormalities that result can, on the other hand, become very complex and require both the reduction of the excess height of the affected ramus and transverse and vertical changes of the maxilla and the upper dentoalveolar arch, changes which accompany the mandibular anomalies. In some adolescents and even adults, however, condylar resection may be all that is necessary due to the periosteal morphogenetic phenomena that occur after the operation, although the postoperative improvement is slower (**Figure 35**).

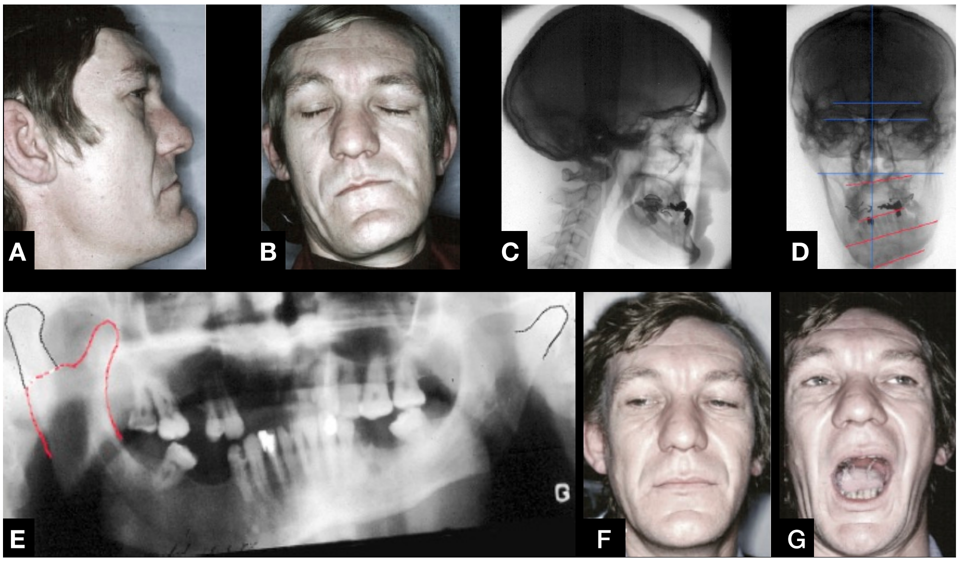

Figure 35. Profile (**A**) and enface (**B**) photographs as well as lateral (**C**) and frontal (**D**) radiographs of an adult patient with right hypercondylia, 36 years old at first consultation. Projected resection of the hypertrophied condylar process on the panoramic radiograph (**E**). Patient status one year and 10 months after condylectomy (**F, G**).

One of its advantages of this approach is that by definitively reducing the length (height) of the condylar process, the adverse effects on mandibular functioning are avoided, an otherwise frequent cause of a progressive relapse of the immediate post-operative results.

As illustrated in **Figure 36** the best movements of the mandibular condyles and, more generally, of the mandibular body are obtained when the condylar and coronoidal processes are of equal lengths with the centre of mandibular rotation in the vicinity of the spine of Spix (Eramo et al., 2014) which is a bony protrusion resembling a tongue, situated on the inner aspect of the mandibular ramus, covering the mandibular foramen and connected to the sphenomandibular ligament (Lingula mandibularis).

2.10 The physiological surgery of the premaxilla

Another region where the physiological surgery gives a very interesting result, in some cases even very spectacular is that of the premaxilla. Classically, the premaxilla does not exist in humans. However, as early as 1937, Broadbent (the father of modern radiological cephalometry) in his remarkable article, *The Face of the Normal Child*, wrote:

> *"It is interesting to note the forward developmental growth of the premaxilla in contrast to the relative stability of the posterior termination of the hard palate."*

As early as 1933, De Coster had already insisted on these developmental features of the premaxillary region. He wrote:

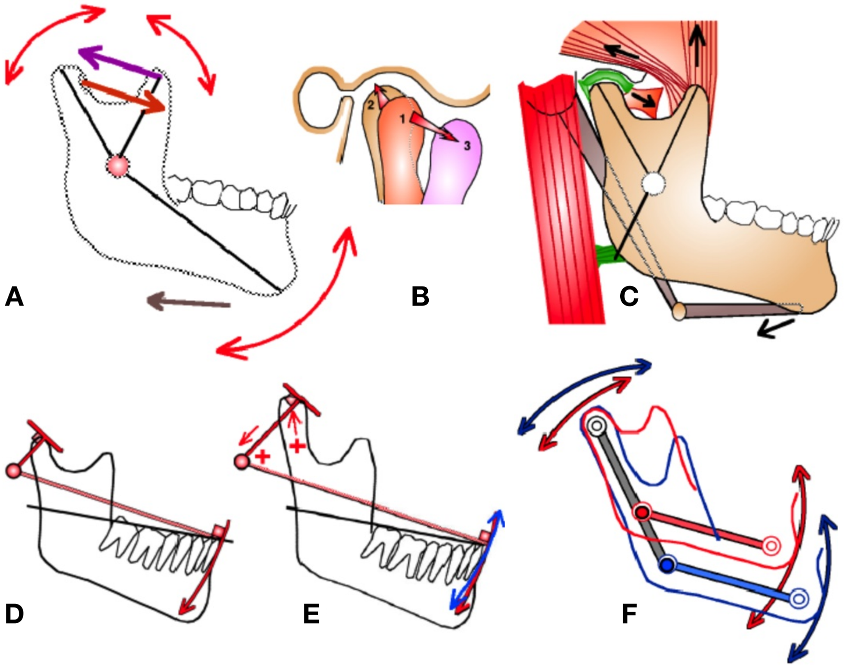

Figure 36. **Upper row:** Schematic representation of the main movements of the mandible, in general (**A**), movements of the resulting condyles (**B**) representing rest position (**1**), tightened teeth contracted jaws (**2**), wide mouth opening (**3**), and agents of these movements (**C**). The best movements of the mandibular condyles and, more generally, of the mandibular body are obtained when the condylar and coronoid process have equal lengths with the mandibular center of rotation in the vicinity of the spine of Spix. **Lower row:** Constructions according to Darcissac (1921) highlighting the differences in mandibular movements as a function of the length of the condylar processes compared to the coronoid processes. Normal condyle (**D**), very elongated condyle (**E**) and excessive lengthening of the condylar process (superposition on the head of the condyle of a normal mandible). As the condylar process lengthens, the mandibular center of rotation lowers and vice versa (**F**). As a result, the curves traced by the chin symphysis differ in direction.

> *"Although it does not strictly speaking constitute an anatomical unit, nevertheless, from the indiviudal point of view, it exists very much as a centre of activity in the life of the individual, an area sometimes referred to as a part of the maxilla called the intermaxillary bone."*

More recently, in particular Moss (1958) has contributed greatly to the disemination and development of the ideas of Van der Klaauw (1952), father of the subdivision of the cranio-facial skeleton into *skeletal pieces*, *skeletal units* and *skeletal ensembles* that one must always have in mind in order to understand the *state* and *becoming of the cephalic skeletal puzzle*. Among the skeletal units of the maxilla that he has identified is the premaxilla, to which he attributes particular importance for the specific development of the *maxillary ensemble*.

I have already summarised (in section 2.6.1 of my work) the main phenomena and factors responsible for good premaxillary development, essential for the optimum state of the upper incisivo-canine region and, in general, of the entire *fronto-naso-premaxillary* region. In particular, I emphasised the fundamental role of the forces exerted on the anterior premaxilla (vestibular cortices and median inter-incisive suture) and from the septal cartilage and muscles attached to it.

From the surgical point of view, physiological surgery reconstructing the anatomy and physiology of growth of these muscular and skeletal elements (including the periosteum) gives very positive outcomes, especially in the correction of anomalies associated with Binder syndrome and in congenital labio-maxillary clefts.

The Binder syndrome is characterized by the absence of insertion of the nasolabial muscles and the oblique upper layer of the orbicularis of the upper lip onto the lower margin of the nasal septal cartilage (**Figure 37**). Consequently, where these muscles would have pulled in a forward direction during the primary growth spurt of this cartilage, they collapse against the anterior part of the maxilla. This collapse reduces its development, especially that of the lateral parts of the piriform apertures, and, above all, is responsible for the total agenesis of the anterior nasal spine.

The anterior part of the palatal vault, situated in front of the palatal papilla, is thus reduced. As a result there is inadequate space for the tip of the tongue, which is depressed, so causing mandibular advancement together with retroposition of the upper incisors and canines and with vestibulo-positioning of the lower incisors and canines. The law of universal harmony applying not only to skeletal elements but also to functions explains why there are also postural disorders of the cervical spine with, in about 50% of the cases, vertebral anomalies which, without treatment, exaggerate with age, leading to extremely severe cephalic malformations.

Physiological treatment, started very early, from six years old or even earlier at the end of the temporary dentition, can facilitate best treatment outcomes. It consists primarily of postero-anterior traction using an orthopedic mask (**Figure 37 F, G**), and as soon as the maxillary positon has been corrected (**Figure 37 H**), followed by a careful reconstitution of the soft tissue abnormalities (**Figure 37 I, J**). The normalization of the nasolabial and lingual muscular functions thus obtained will gradually lead to bone corrections and a good final result (**Figure 37 L, M and Figure 38**).

In labio-maxillary clefts on the cleft side, there is always (as in Binder's syndrome) an absence of insertion of the nasolabial muscles and the upper layer of the oblique fibres of the orbicularis of the upper lip onto the lower edge of the septal cartilage. This results in collapse and retroposition of the nostril and surrounding soft tissues. It is the same with the hypodevelopment of the hemi-premaxilla on this side, secondary to the absence of insertions of this muscle into the medial inter-incisive suture. Contrary to common opinion, this is not a primary agenesis but, as Veau (1928) already pointed out, a secondary hypodevelopment.

In both unilateral and bilateral labio-maxillary clefts, careful reconstruction of the muscular complex is essential. It ensures correct growth and yields results much superior to those of surgical techniques that do not pay meticulous attention to muscle reconstruction. Although these techniques may bring about initial but unsustained improvement in the whole region, they result in poor later development.

In practice, the essential stage of physiological labio-maxillary surgery is not the labial skin tracing, which simply follows the cutaneous and mucosal limits (**Figure 39**), but the individualization of the various naso-labial and labio-mentalis muscles and their correct, well-symmetrical insertions on the lower edge of the nasal cartilaginous septum and the muscles on the opposite side. To achieve this result, the following steps are essential (**Figure 40**).

1. Releasing the attachments of the alar cartilages from the nasal septum, the cartilage of the opposite side and the superficial muscular envelope, allowing them to be sym-

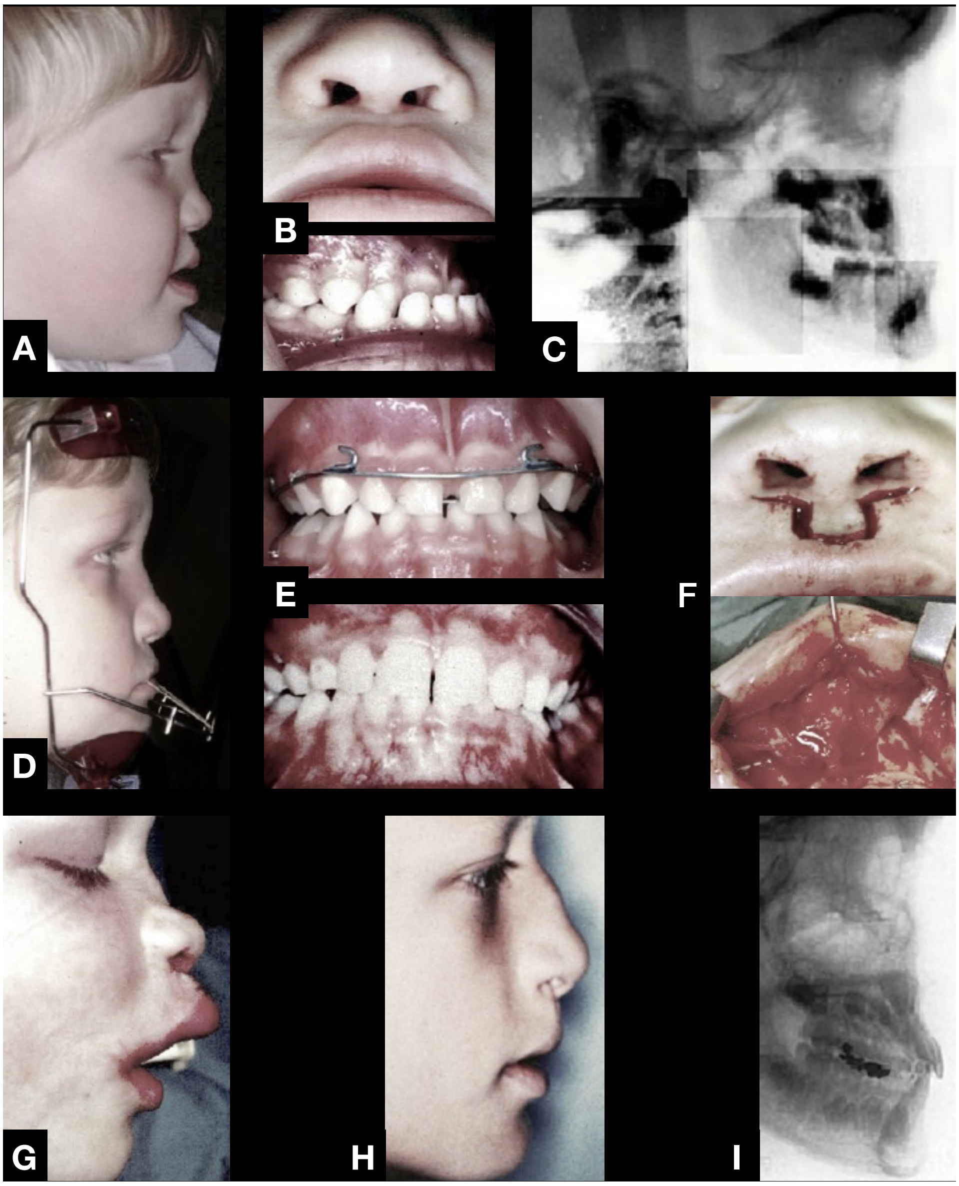

Figure 37. Three year old child with Binder syndrome (**A**). Typical appearance of nasal apertures with notch at the base of the columella and anterior crossbite (**B**). Note the total absence of nasal spine (**C**). At age four the hypopremaxilla and retromaxilla were treated with extra-oral postero-anterior traction on an orthopedic mask (**D**). The good occlusion of the anterior teeth was obtained in six months (**E**). Restoration of the normal anatomy of the nasolabial muscles, by a sub-columellar external route and a vestibular internal pathway (**F**). This muscle reconstruction produces a significant labial projection (**G**) that will disappear when the muscles have functioned normally. Reorganization of the muscles below the nose that attach to the nasal septum (**H, I**).

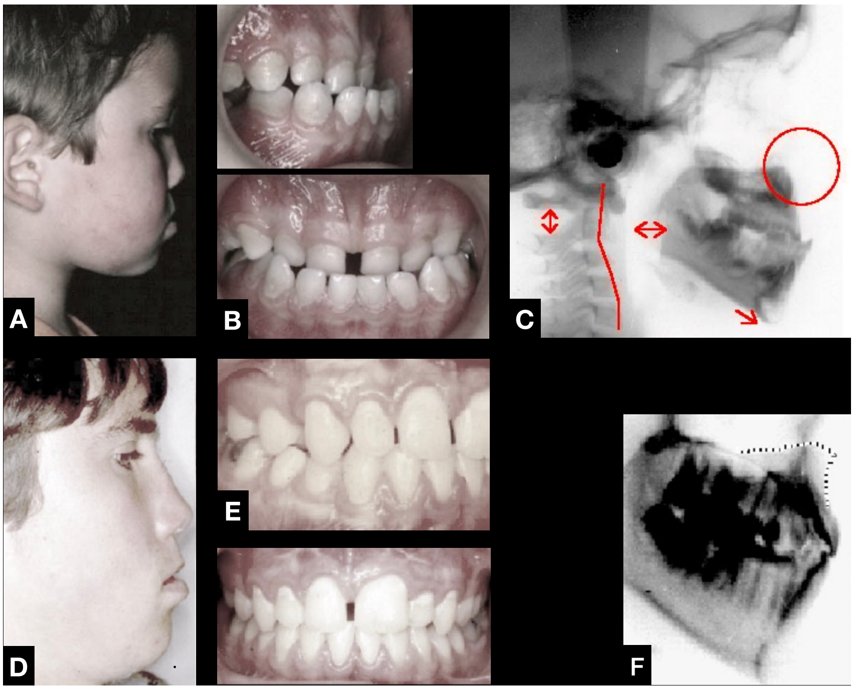

Figure 38. Another example of a Binder syndrome, five years old, having benefited from the same treatment as in Figure 37. Typical aspects of the nose, lips (**A**), incisivo-canine occlusion of the maxilla (**B**) with agenesis of the anterior nasal spine, excess height of the anterior face and vertebral curvature anomalies (**C**). Same patient 12 years old (**D**). Note the volume of the anterior nasal spine (**F**) and the existence of a gigantism of the upper central incisor on the left (**E**).

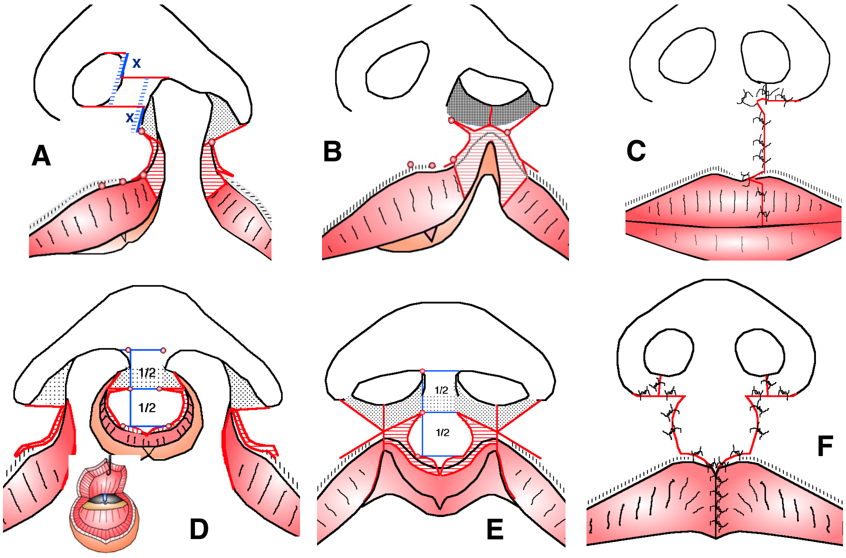

Figure 39. The *physiological* tracing of the skin incisions. Strictly based on the anatomical limits of the skin of the columella, the floor and threshold of the nostrils, the vermillion and the muco-cutaneous margin of the lip (without secondary tracing). They must remain superficial in relation to the underlying muscles and strictly respect the philtrum. Complete unilateral cleft (**A**), incomplete unilateral cleft (**B**), at the end of the operation (**C**), complete bilateral cleft (**D**), incomplete bilateral cleft (**E**) and result at the end of the operation (**F**).

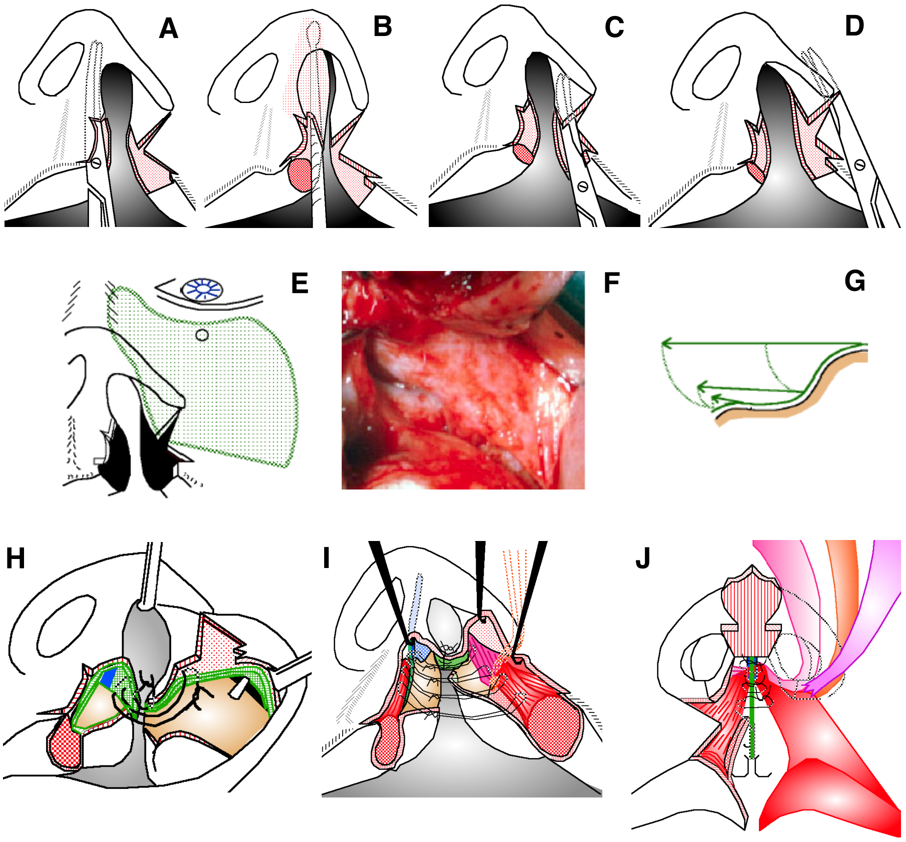

Figure 40. A-D: Release of the left alar cartilage from its attachments with the the nasal septum, the cartilage on the opposite side and the superficial muscular envelope. **E, F:** Large periosteal undermining and detachment from the entire anterior surface of the maxilla, extended from the infra-orbital rim to the nasal bridge and to the top of the malar bone, facilitating easy advancement of the musculo-periosteal flap (**G**). **Lower row:** Different stages of physiological lip reconstruction. (**H**) Suture of the nasal muco-periosteum. (**I**) Suture of the muscular elements of the nasal floor, the nostril threshold and the oblique heads of the orbicularis of the upper lip. (**J**) Insertions of the oblique heads of the orbicularis in the region of the anterior nasal spine (bilateral total cleft).

metrically attached to the septal cartilage, the median frenulum, and the muscles on the non-cleft side.

2. Performing a very wide undermining, of the periosteum of the entire anterior surface of the maxilla, possibly extended along the orbital rim and from the nasal ridge on one side to the superior margin of the zygomatic bone allowing the easy advancement of the labial flap.
3. Systematically and separately reconstruct the continuity of the muco-periosteum of the nasal floor, and the cartilaginous and muscular insertions of the constrictor naris and the oblique and transverse heads of the orbicularis of the lips.

Suturing of the vestibular mucosa should be extended as inferiorly as possible, ideally to the gingival fibro-mucosa. This can only be obtained in incomplete or very narrow clefts. In these cases, and provided that the procedure was carried out at the no later than 6 months (before the eruption of the temporary lateral incisors), it is not uncommon to obtain immediate bony fusion of the cleft margins (**Figure 41**).

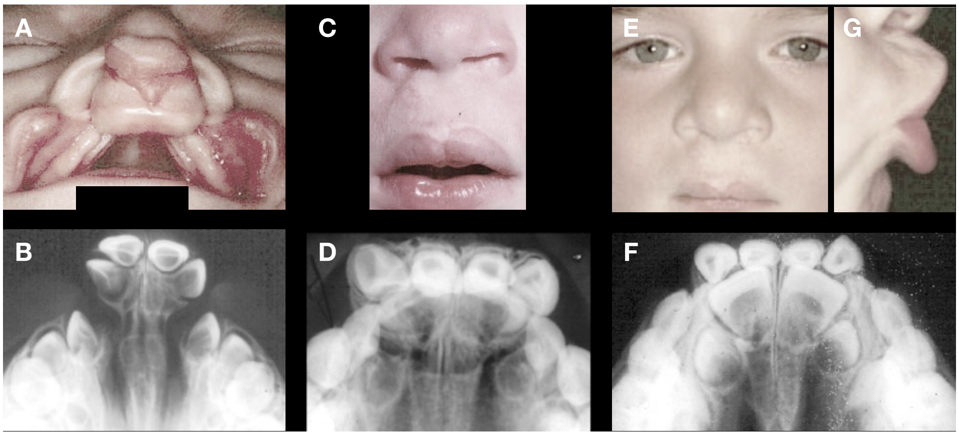

Figure 41. Example of primary gingivo-periostoplasty in a case of bilateral cleft lip and maxilla without palatal separation. The gingivoplasty carried out at the same operating time as the closure of the labio-maxillary clefts. Situation at the day of the operation (**A, B**) and 17 months later (**C, D**). Four years later a complete bone unification is present (**F**) due to the good *physiological* reconstruction of the nasolabial muscles attested by good projection movements of the lips (**G**).

In all other cases, the closure of the residual cleft will be carried out, ideally, at around 18 months, about the time when the deciduous canine teeth are erupting and at the same time as the second stage of closure of the cleft palate, the first stage having been closure of the soft palate only. A large mucoperiosteal flap will be raised, widely undermined from the anterior maxillary, advanced across the alveolar cleft and sutured to the muco-periosteum and the gingiva on the medial side of the cleft (**Figure 42**).

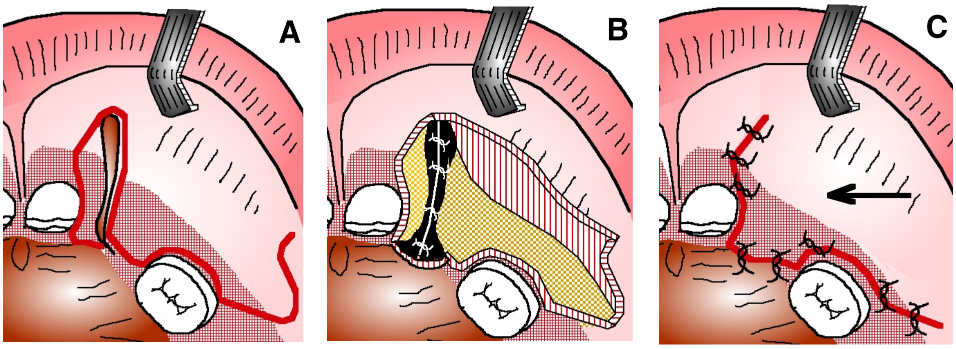

Figure 42. Example of gingivo-periostoplasty for residual alveolar cleft in a case of complete left unilateral cleft. It unites the hemi-premaxilla on the side of the cleft with the adjacent maxilla. The forces from the latter can then activate the growth potential of the median inter-incisal suture, and therefore the premaxilla. Bone exposure can be extended on the anterior surface of the maxilla, up to the pyriform aperture, but never on the palatal side where the buds of the permanent incisors are already forming.

One of the most significant advantages of this physiological approach to the treatment of the labio-maxillary clefts is the resumption (almost normal) of the premaxillary development of the cleft side, so long as the anatomy and muscular physiology has been normalised (or as near normalised as possible) sufficiently early (between 3 to 6 months). The bony cleft may even disappear completely, provided that the gingivoplasty was sufficiently early, as soon as the alveolar margins are fairly close usually at 18 months, the normal period of eruption of deciduous canines (**Figure 43**).

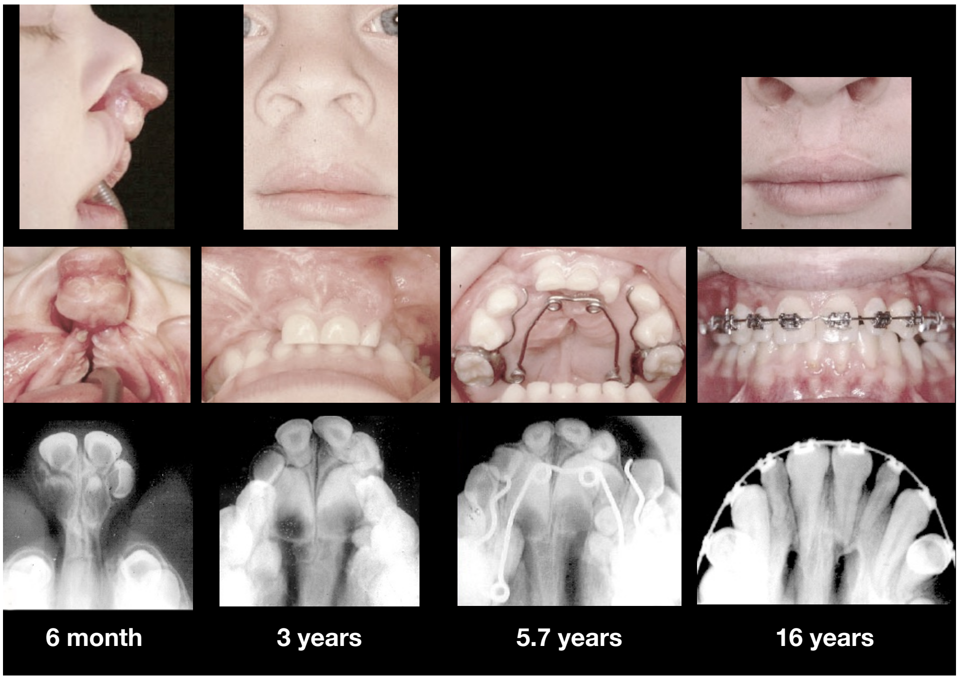

Figure 43. Bilateral total cleft lip and palate with velo-palatal cleft at six months. Note the significant anterior projection of the premaxillary median tooth buds. Then at three years, 1.5 years after bilateral gingivo-periostoplasty. Insertion of a quadhelix at 5.7 years to increase the width of the incisal region. Completing treatment at 16 years. Note the continuity of the alveolar bone in the incisor, canine and premolar regions.

In the complete labio-maxillofacial cleft (uni- or bilateral), the labial reconstruction and closure of the soft palate will be performed simulaneously, between three months (in bilateral forms) and six months (in unilateral forms) so that the approximation of the two sides of the divided bony palate occurs both anteriorly as well as posteriorly.

At the same time as they are getting closer, the palatine shelves widen, so that at 18 months the mid-palatal gap is usually narrow enough to be closed with the smooth mucosa of the mid-palatal vault, without using either the adjacent striated mucosa, nor a vomerine mucosal flap (**Figure 44**).

The great disadvantage of these (yet classic) uses is that, on the one hand, they narrow and reduce the dimensions of the palatal concavity, and on the other hand, they limit the descent of the palate, two factors responsible for the Class III malocclusions so often observed in subjects operated on in this way. During this operation to close the residual palatal gap, the surgeon must also check that the posterior pillars of the soft palate, i.e. the *pharyngo-staphylins*, meet well behind the base of the uvula, an essential condition for correct lengthening behind the uvula and, at the same time, for correct retraction of the lingual mass, without which too many mandibular prognathia tend to develop.

In the Robin sequence, as described by Pierre Robin in 1934, characterized by mandibular retrognathia, cleft palate, glossoptosis, and more or less serious apneic phenomena which can lead to death. The technique for closing the cleft palate should be the same as for wide clefts. (refer to **Figure 45**).

However, there is one imperative that influences the timing of closure. By the end of the

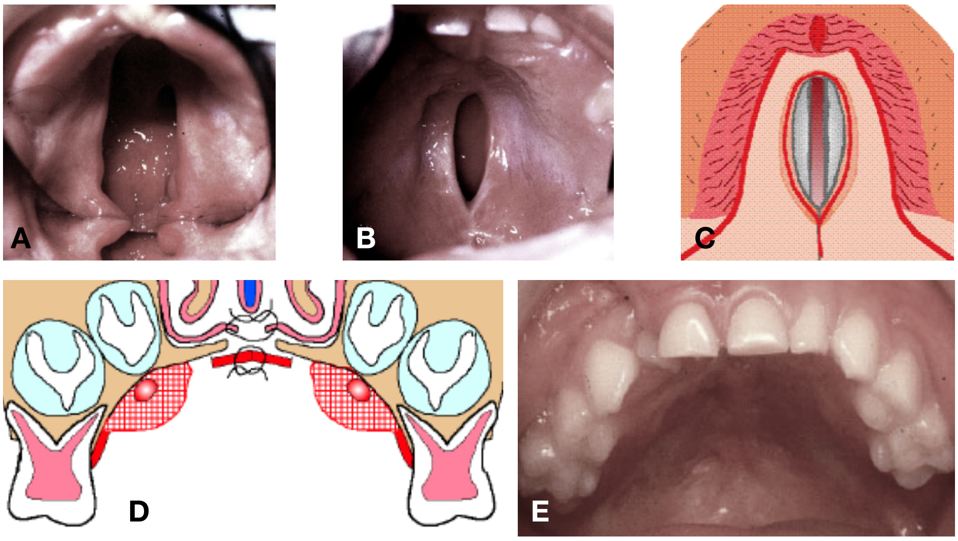

Figure 44. Example of a 2-stage closure of a wide cleft of the palate. (**A**) Palatal cleft at 6 months, at the time of closure of the soft palate. (**B**) At 18 months, the palatal shelves have developed horizontally so narrowing the width of the cleft. (**C**) The line of the palatal incisions passes through the union of the smooth mucosa and the striated palatal fibro-mucosa. (**D**) The margin of the nasal floor mucosa is everted to form a deep layer above the smooth palatal mucosa (the vomerine mucosa is not used). (**E**) At four years of age, the palatal vault, as a whole, is appropriately wide and deep.

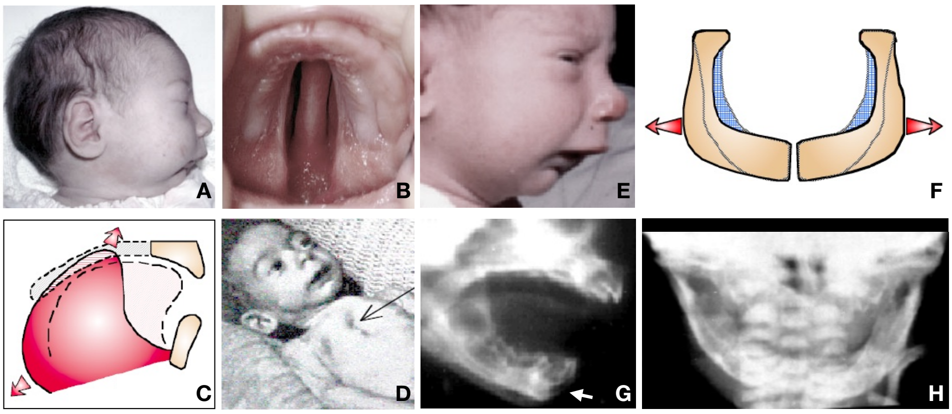

Figure 45. Cardinal signs of Pierre Robin syndrome (sequence). Chin retraction (**A, E**), velo-palatal cleft (**B**, glossoptosis (after Psaume et al., 1986) and therefore airway obstruction (**C**); note the sternal depression (**D**). The setback of the chin, with an apparent brachy-mandible is essentially due to the widening of the mandibular angles (**F**), itself caused by lingual ptosis (**C**). Note the normal angular width in a newborn (**H**). The widening of the mandibular angles is responsible for the pathognomonic reduction of mandibular body length and retraction of the mandibular symphysis (**G**).

first year of life, the mandibular symphysis, which separates the anterior parts of the two hemi-mandibles until this age, has synostosed. Before this date, its existence had allowed the ptosis of the lingual mass between the mandibular angles to exaggerate their distance. This is the main cause of the receding chin and lingual mass (**Figure 45**).

Early reconstitution of the correct anatomy and normal physiology of the soft palate (before symphyseal synostosis) by eliminating lingual ptosis inversely causes the symphysis to advance, and generally also improves ventilatory disorders. After one year, on the other hand, symphyseal synostosis no longer allows the hemi-mandibles to move closer together. In summary, closure of the soft palate in Robin sequence must be performed before the age of one year.

Early physiological surgery of the cranio-facio-cervical complex applies not only to the indivual to parts of the craniofacial skeleton and/or the soft tissues contained therein, as well the entire complex. Early, meticulous surgeries addressing the factor responsible for the global anomaly can therefore radically transform the evolution of the latter. Many other examples could still be given concerning the interest of early interceptive and/or repair surgery, avoiding aggravation of skeletal abnormalities (dental included) and/or correcting those already present during the management of the subject. This applies to the early performance of glossectomy in cases of genuine congenital macroglossia (such as in Wiedemann-Beckwith syndrome), functional reduction genioplasty, and anatomical and physiological surgeries for temporomandibular ankylosis. Additionally, it involves the restoration of the nasal septum's anatomy and the selection of the appropriate site for bone distractors.

Conclusions

Many modern maxillofacial surgeons have more or less abandoned early surgery in favor of majority surgery. Admittedly, advances in the latter, mainly linked to a more precise preoperative lesion assessment, improved surgical techniques and much more rigorous means of restraint, have encouraged them to do so. However, late surgery is not without its difficulties and drawbacks, even in the most classic orthognathic surgery. It is inconceivable for most congenital malformations (particularly facial clefts).

In all cases, early orthognathic surgery requires a good knowledge of the growth physiology of the various components of the cephalic extremity. However, there is no doubt that, in view of the benefits that can be derived, surgeons (like dento-maxillo-facial orthopaedists) will, in the future, accept to acquire at least the essentials.

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Ethical approval

No ethical approval is required for this article as no sensitive data was processed. The non-anonymized image material was created under the ethical standards at the time the patients were treated. It can be assumed that appropriate permission has been granted and, due to the time that has elapsed, the image material does not relate to any currently identifiable persons.

Consent for publication

Not applicable.

Authors' contributions

The author(s) declare that all the criteria for authorship designated by the International Committee of Medical Journal Editors have been met. More specifically, these are: (a) Substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work; AND (b) Drafting the work or revising it critically for important intellectual content; AND (c) Final approval of the version to be published; AND (d) Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Competing interests

The author(s) declare that there are no competing interests related to this work.

Author notes

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References

Athenstaedt, H. (1969). Permanent electric polarization of the meninges of man. Z. Zellforsch. 98, 300–322.

Augier, M. (1912). Squelette céphalique: morphogenèse, morphologie, craniométrie. Masson, Paris.

Balters, W. (1955). Reflexmechanismus und Funktionsablauf. Fortschritte der Kieferorthopädie, 16(4), 325–327.

Baume, L. J. (1961). Principles of cephalofacial development revealed by experimental biology. American journal of orthodontics, 47(12), 881-901.

Björk, A. (1968). The use of metallic implants in the study of facial growth in children: method and application. American Journal of Physical Anthropology, 29(2), 243-254.

Blechschmidt, E. (1951). Die frühembryonale Strukturentwicklung der Gliedmaßen: Entwicklung der Extremitäten beim Menschen. Teil III. Zeitschrift für Anatomie und Entwicklungsgeschichte, 115(6), 617–657.

Broadbent, B. H. (1931). A new x-ray technique and its application to orthodontia. The angle orthodontist, 1(2), 45-66.

Broadbent, B. H. (1932). The Use of X-ray Silhouettes in Tracing Facial Growth (Abstract) Journal of Dental Research vol. XIII, no. 2, Apr. 1933. Proc. Edward H. Angle Society of Orthodontia, April, 151-153.

Broadbent, B. H. (1937). The face of the normal child. The Angle Orthodontist, 7(4), 183-208.

Cadenat, E. (1924). Recherche sur l'embryogenèse et la pathogénie du Bec de Lièvre. Thèse, Toulouse, 1924, Douladoure, Imprimeur.

Couly, G. (1975). Bone statics of the face: the frontal-sphenoid-pterygoid bone pillars as biomechanic equivalents of the mandible. Revue de Stomatologie et de Chirurgie Maxillo-faciale, 76(8), 607-619.

Cuvier, G. (1825). LXXIII. On the osteology of reptiles, and on the geological position of their fossil remains. The Philosophical Magazine, 65(326), 447-457.

Darcissac, M. (1921). De la mobilisation physiologique et permanente du maxillaire inférieur en chirurgie maxillo-faciale: travail du service de M. le professeur Sebileau (Doctoral dissertation).

De Coster, L. (1932). A new method of analysis of dentofacial malformations. International Journal of Orthodontia, Oral Surgery and Radiography, 18(9), 942–956.

De Coster, L. (1952). A new orientation line for the study of dento-facial deformities. Anales espanoles de odontoestomatologia, 11(7), 564-577.

Delattre, A., Fenart, R. (1960). L'hominisation du crâne: étudiée par la méthode vestibulaire. Éditions du Centre national de la recherche scientifique.

Enlow, D. H. (1962). Functions of the haversian system. American Journal of Anatomy, 110(3), 269–305.

Enlow, D. H. (1965). Mesial drift as a function of growth. West Indian med. J., 124-124.

Enlow, D. H., Moyers, R. E. (1971). 1 Growth and Architecture of the Face. The Journal of the American Dental Association, 82(4), 763-774.

Enlow, D. H., Hans, M. G. (1996). Essentials of facial growth. American Journal of Orthodontics and Dentofacial Orthopedics, 110(5), 568.

Enlow, D. H., Hans, M. G. (1996). Essentials of facial growth. Saunders.

Eramo, S., De Carolis, C., Pagano, S. (2014). Johann Baptist Spix and the "lingula mandibularis". Journal of the history of dentistry, 62(3), 116–121.

Firu, P., Neagu, N., & Georgescu, V. (1959). Correlations between edentation and cranial sutures. Rumanian Medical Review, 3, 69-72.

Hellman, M. (1929). The face and teeth of man: a study of growth and position. Journal of Dental Research, 9(2), 179-201.

Humphry, G. M. (1878). On the Growth of the Jaws. Journal of anatomy and physiology, 12(Pt 2), 288–293.

Kolliker, A. V. (1860). V. On the structure of the chorda dorsalis of the plagiostomes and some other fishes, and on the relation of its proper sheath to the development of the vertebræ. Proceedings of the Royal Society of London, (10), 214-222.

Lebourg L. & Université de Paris Faculté de médecine. (1931). La dysarthrose cranio-faciale : mobilité anormale entre la face et le crane : etudes clinique anatomique embryologique et étiologique : rôle dans la pathogénie des dysmorphoses de la face : thèse pour le doctorat en médecine (dissertation). Amédée Legrand.

Lebourg, L., Seydel, S. (1932). Nature, évolution et rôle des articulations de la face; leur importance physio-pathologique. Rev Stomatol, 34, 193-210.

Leroi-Gourhahn, A. (1954). Influence of the denture on the facial bones and on the base of the cranium. Revue francaise d'odonto-stomatologie, 1(1), 47-61.

Mall, F. P. (1906). On ossification centers in human embryos less than one hundred days old. American Journal of Anatomy, 5(4), 433-458.

McNamara Jr, J. A., Graber, L. W. (1975). Mandibular growth in the rhesus monkey (Macaca mulatta). American Journal of Physical Anthropology, 42(1), 15-24.

Moore, Keith L. Before We Are Born—Basic Embryology and Birth Defects. JOGN nursing 3, no. 5 (1974): 56-57.

Moss, M. L. (1958). The pathogenesis of artificial cranial deformation. American Journal of Physical Anthropology, 16(3), 269–286.

Moss, M. L. (1997a). The functional matrix hypothesis revisited. 1. The role of mechanotransduction. American journal of orthodontics and dentofacial orthopedics, 112(1), 8-11.

Moss, M. L. (1997b). The functional matrix hypothesis revisited. 2. The role of an osseous connected cellular network. American Journal of Orthodontics and dentofacial orthopedics, 112(2), 221-226.

Moss, M. L. (1997c). The functional matrix hypothesis revisited. 3. The genomic thesis. American journal of orthodontics and dentofacial orthopedics, 112(3), 338-342.

Moss, M. L. (1997d). The functional matrix hypothesis revisited. 4. The epigenetic antithesis and the resolving synthesis. American journal of orthodontics and dentofacial orthopedics, 112(4), 410-417.

Petrovic, A. G. (1972). Mechanisms and regulation of mandibular condylar growth. Acta Morphologica Neerlando-Scandinavica, 10(1), 25-34.

Petrovic, A. G. (1972). Low-muscle pterygoidien externe et la crossance du condyle madiblare. Recherches experimentales chez le jeune rat. L'Orthodont Franc, 43, 271-285.

Pritchard, J. J., Scott, J. H., Girgis, F. G. (1956). The structure and development of cranial and facial sutures. Journal of anatomy, 90(Pt 1), 73.

Psaume, J., Malek, R., Mousset, M. R., Trichet, C., Martinez, H. (1986). Technique et résultats du traitement total précoce des fentes labio-palatines. Folia Phoniatrica et Logopaedica, 38(2-4), 176-220.

Robin, P. (1934). Glossoptosis due to atresia and hypotrophy of the mandible. American journal of diseases of children, 48(3), 541-547.

Rouvière, H. (1920). Précis d'anatomie et de dissection (Vol. 1). Masson.

Rozencweig, S. (1994). Entretien avec le Dr D.H. Enlow. Rev Orthop Dento Faciale 28 (4) 423-430.

Sarnat, B. G., Engel, M. B. (1951). A serial study of mandibular growth after removal of the condyle in the Macaca rhesus monkey. Plastic and Reconstructive Surgery, 7(5), 364-380.

Scott, J. H. (1956). Growth at facial sutures. American journal of orthodontics, 42(5), 381-387.

Scott, James Henderson. Dento-facial Development and Growth. United Kingdom, Elsevier Science & Technology, 1967.

Sicher, H. (1947). The growth of the mandible. American journal of orthodontics and Oral surgery, 33(1), 30-35.

Testut, L. (1895). Traité d'anatomie humaine: anatomie descriptive, histologie, développement: Appareil de la digestion (Histologie, par G. Ferré) Appareil de la respiration. Appareil uro-génital. Embryologie (par L. Vialleton) (Vol. 3). Doin.

Thompson D. W. (1917). On Growth and Form. Cambridge University Press.

Topinard, P. (1876). L'anthropologie. C. REINWALD ET CIE, LIBRAIRES-ÉDITEURS 15, RUE DES SAINTS-PÈRES, Paris.

Trauner R. (1967). Le traitement chirurgical de la prognathie, de la rétrognathie mandibulaire et de prognathie supérieure. Annales d'oto-laryngologie et de chirurgie cervico faciale : bulletin de la Societe d'oto-laryngologie des hopitaux de Paris, 84(12), 855–866.

Van der Klaauw, C. J. (1946). Cerebral skull and facial skull. Archives Neerlandaises de Zoologie, 7(1), 16-37.

Van der Klaauw, C. J. (1952). Size and position of the functional components of the skull. A contribution to the knowledge of the architecture of the skull, based on data in the literature. Archives Neerlandaises de Zoologie, 9(1), 1-556.

Veau, V. (1928). Discussion on the Treatment of Hare-Lip. Proceedings of the Royal Society of Medicine, 21(12), 1868–1888.