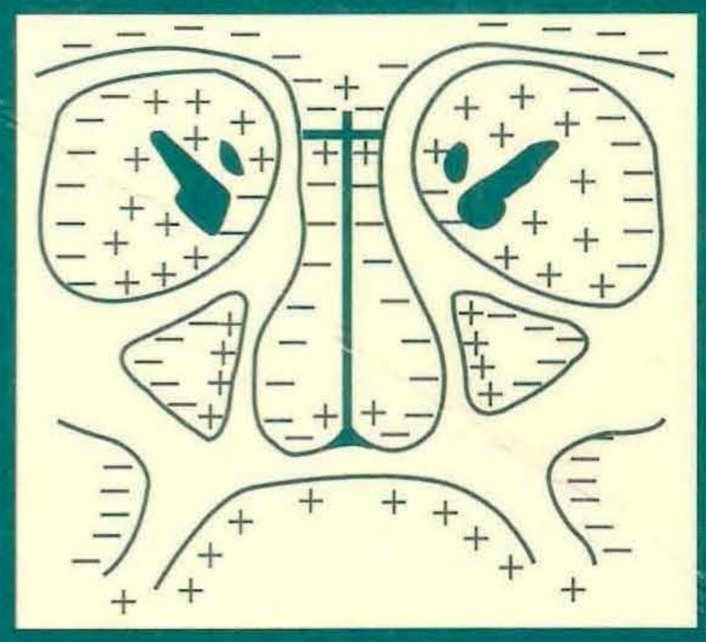
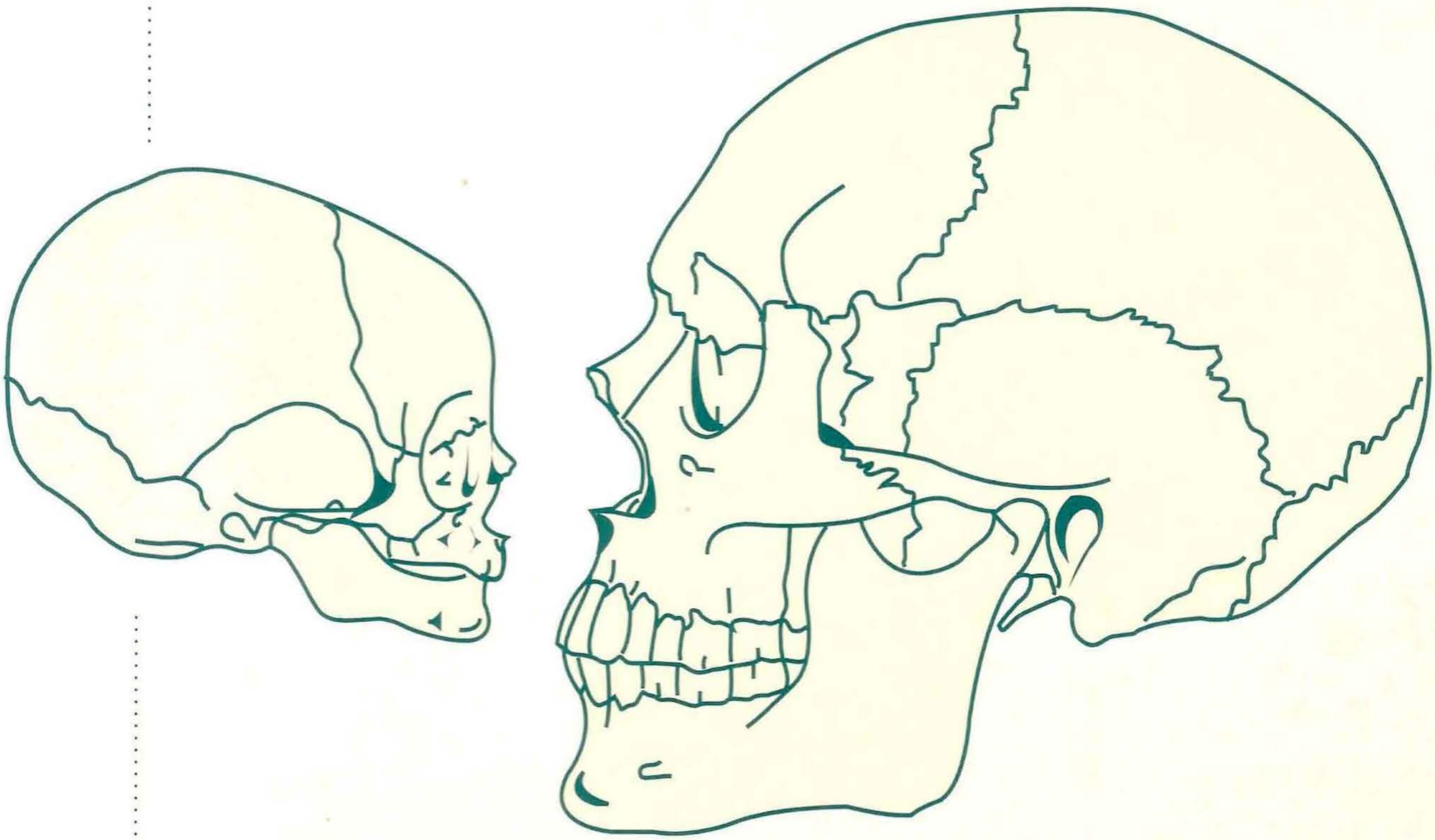
Essentials of
FACIAL
GROWTH





Enlow and Hans

Essentials of FACIAL GROWTH

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To
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PREFACE

Radiographic cephalometry was a pioneering advance introduced decades ago that led to many fundamental insights into the behavior of the face and neurocranium during growth. It remains to this day a basic tool in research and diagnosis. The techniques were necessarily formulated before present-day understanding of the actual biology underlying craniofacial morphogenesis, and thus could not incorporate today's biologic concepts. *This biology is the particular subject of the pages that follow*. The purpose is to outline in *abridged format* the enormous range of morphogenic information dealing with key craniofacial growth concepts. *The biological grounding for basic clinical theory is highlighted*. Just as an understanding of the basics of cephalometry remains essential to academic researchers as well as clinicians, so now certainly is the biological aspect as well. Meaningful insight into diagnosis, treatment planning, treatment selection, treatment effects, and rebound is not possible without the latter. These considerations, without contradiction, are the near and long-term clinical craniofacial future. The predoctoral dental curriculum and, importantly, graduate dental and medical training programs touching this biology only lightly or not at all are seriously incomplete. The present condensed version of previous editions is an attempt to distill details down into the "essentials". An all-new introductory chapter has been added that presents a panorama of the craniofacial growth process as a whole. It emphasizes the developmental interplay among all tissues, soft as well as hard. In effect, this section is a "short course" of facial growth, summarizing highlights and key concepts of the chapters that follow. For teachers and speakers needing to present core material in a limited time frame, these new pages can be useful. The format and selection of content for all the other chapters have been refined, extensively re-organized for teaching and for an interested professional's reading, and the text has been updated and clinically tuned throughout.

The preparation of this extensively revised edition has been strengthened by the talents and gracious tolerance of our office secretary, Mrs. LaVerne Vogel. We are grateful indeed. We all value and appreciate the professional expertise and helpful collaboration provided by the editors and production staff of W.B. Saunders Company.

CONTENTS

PROLOGUE

HISTORICAL PERSPECTIVE

The morphogenic *interrelationships* among the diverse families of soft tissues and the growth and development of the craniofacial skeleton are highlights of the present monograph. However, the early historical story of "bone" is especially interesting because of bone's unusual "oxymoron" character. That is, how in the world can this unique rock-like substance actually grow and develop into constantly changing shapes and sizes *perfectly* matching the developing soft tissues it serves. This question festered in early scholars' minds and has been a particular wonderment since Biblical times. In the book of Ecclesiastes (11:5), for example, it was said that "As thou knowest not the way of the spirit, nor how the bones do grow in the womb of her that is with child, even so thou knowest not the works of God who maketh all." Many Old and New Testament passages make frequent reference to bone in health and disease. That bone is actually a living substance is certainly not a modern notion at all. Greek philosophers and physicians, including Hippocrates (*De carnibus*), Aristotle (*De generatione animalium*), Galen (*Opera omnia*), and Plato (*Timaeus*), all recorded allegories on bone formation, describing how the earthy, less fluid, and thicker seminal parts become solidified by internal body heat, comparable to the manner in which moist clay (i.e., cartilage) is kiln-fired (endochondral ossification) into earthenware. A reasonable analogy at the time, considering that microscopes, histology textbooks, and the Cell Doctrine were in the distant future. Arnobius accounted for the control of childhood bone formation by a goddess "who hardens and solidifies the bones in infants." Then, century by century, many, many of the great names in the anatomy and medicine Hall of Fame assembled our foundations of bone knowledge in a long series of plateaus, each following some technological advance or conceptual breakthrough. These familiar names include Albinus, Vesalius, Bartholin, Harvey, Sue, Havers, Nesbitt, Monro, Leewenhoek (who observed canals in bone years before Havers, but that doesn't detract from the latter's classic monograph "*Osteologia Nova*"), Todd, Bowman, Tomes, Demorgan, Von Ebner, Gagliardi, Malpighi, Bell, Howship, Belchier, Hales, Hunter, Volkmann, Wolff, Hassall, Meckel, Virchow, Purkinje, Sharpey, and Schwann. All, and many more, were directly involved in the quest. (See Enlow, 1963, for a more extensive historical review, including the specific landmark contributions of these early scholars.)

1

Overview of Craniofacial Growth and Development

"Growth" is a general term implying simply that something changes in magnitude. It does not, however, presume to account for **how** it happens. For the professional clinician, such a loose meaning is often used quite properly. However, to try to understand "how" it works, and what actually happens, the more descriptive and explanatory term "development" is added. This connotes a maturational process involving progressive differentiation at the cellular and tissue levels, thereby focusing on the actual biologic mechanism that accounts for growth.

"Growth and development" is an essential topic in many clinical disciplines and specialties, and the reason is important. Morphogenesis is a biologic process having an underlying **control** system at the cellular and tissue levels. The clinician intervenes in the course of this control process at some appropriate stage and substitutes (augments, overpowers, or replaces) some activities of the control mechanism with calculated clinical regulation. It is important to understand that the actual biologic process of development itself is the same. That is, the histogenic functioning of the cells and tissues still carry out their individual roles, but the **control signals** that selectively activate the composite of them are now clinically manipulated. It is the rate, timing, direction, and magnitude of cellular divisions and tissue differentiation that become altered when the clinician's signals modify or complement the body's own intrinsic growth signals. The subsequent course of development thus proceeds according to a programmed treatment plan by "working with growth" (an old clinical tenet). Of course, if one does not understand the workings of the underlying biology, any real grasp of the actual basis for treatment design and results, and why, is an illusion. Importantly, craniofacial biology is independent of treatment intervention strategy. Therefore, some clinicians may argue about the relative merits of different intervention strategies (e.g., headgear versus Frankel appliance therapies). The biologic rules of the game are the same.

Morphogenesis works constantly toward a state of composite, architectonic **balance** among all of the separate growing parts. This means that the various parts developmentally merge into a functional whole, with each part complementing the others as they all grow and function together.

During development, balance is continuously transient and can never actually be achieved because growth itself constantly creates ongoing, normal regional imbalances. This requires other parts to constantly adapt (develop) as they all work toward composite equilibrium. It is such an imbalance itself that fires the signals which activate the interplay of histogenic responses. Balance, when achieved for a time, turns off the signals, and regional growth activity ceases. The process recycles throughout childhood, into and through adulthood (with changing magnitude), and finally on to old age sustaining a changing morphologic equilibrium in response to ever-changing intrinsic and external conditions.

For example, as a muscle continues to develop in mass and function, it would outpace the bone to which it inserts, both in size and in mechanical capacity. However, this imbalance signals the osteogenic, chondrogenic, neurogenic, and fibrogenic tissues to immediately respond, and the whole bone with its connective tissues, vascular supply, and innervation develops (remodels) to work continuously toward homeostasis.

By an understanding of how this process of progressive morphogenic and histogenic differentiation operates, the clinical specialist thus selectively augments the body's own intrinsic activating signals using controlled procedures to jump-start the remodeling process in a way that achieves an intended treatment result. For example, rapid palatal expansion separates the right and left halves of the maxilla (displacement) and initiates a period of increased remodeling in the midpalatal suture.

The genetic and functional **determinants** of a bone's development (i.e., the origin of the growth-regulating signals) reside in the composite of soft tissues that turn on or turn off, or speed up or slow down, the histogenic actions of the osteogenic connective tissues (periosteum, endosteum, sutures, periodontal membrane). Growth is not "programmed" within the bone itself or its enclosing membranes. The "blueprint" for the design, construction, and growth of a bone thus lies in the muscles, tongue, lips, cheeks, integument, mucosae, connective tissues, nerves, blood vessels, airway, pharynx, the brain as an organ mass, tonsils, adenoids, and so forth, all of which provide information signals that pace the histogenic tissues producing a bone's development.

A major problem in orthodontics and orthognathic surgery can be *relapse* (rebound subsequent to treatment). The potential for relapse exists when the functional, developmental, or biomechanical aspects of growth among key parts are clinically altered to a physiologically imbalanced state. The possibility of instability exists because clinicians strive to bring about a state of aesthetic balance that at times produces physiologic imbalance. The underlying conditions that led to the pretreatment dysplasia can still exist and thus trigger the growth process to rebound in response to those conditions. The "genic" tissues (see below) are brought into play in a way attempting to restore balance,* thereby returning in a developmental direction toward the pretreatment state or some combination between. It is, in effect, a built-in protective mechanism but, again, is the same growth process working toward physiologic, biomechanical, and developmental equilibrium. (See Fig. 1–1.)

The evolutionary design of the human head is such that certain regional clinical situations naturally exist. For example, variations in headform design establish natural tendencies toward different kinds of malocclusions. The growth process, in response, develops some regional imbalances, the aggregate of which serves to make corrective adjustments. A

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*A malocclusion or other dysplasia (including congenital malformations), although clinically abnormal, is nonetheless in a "balanced" state.

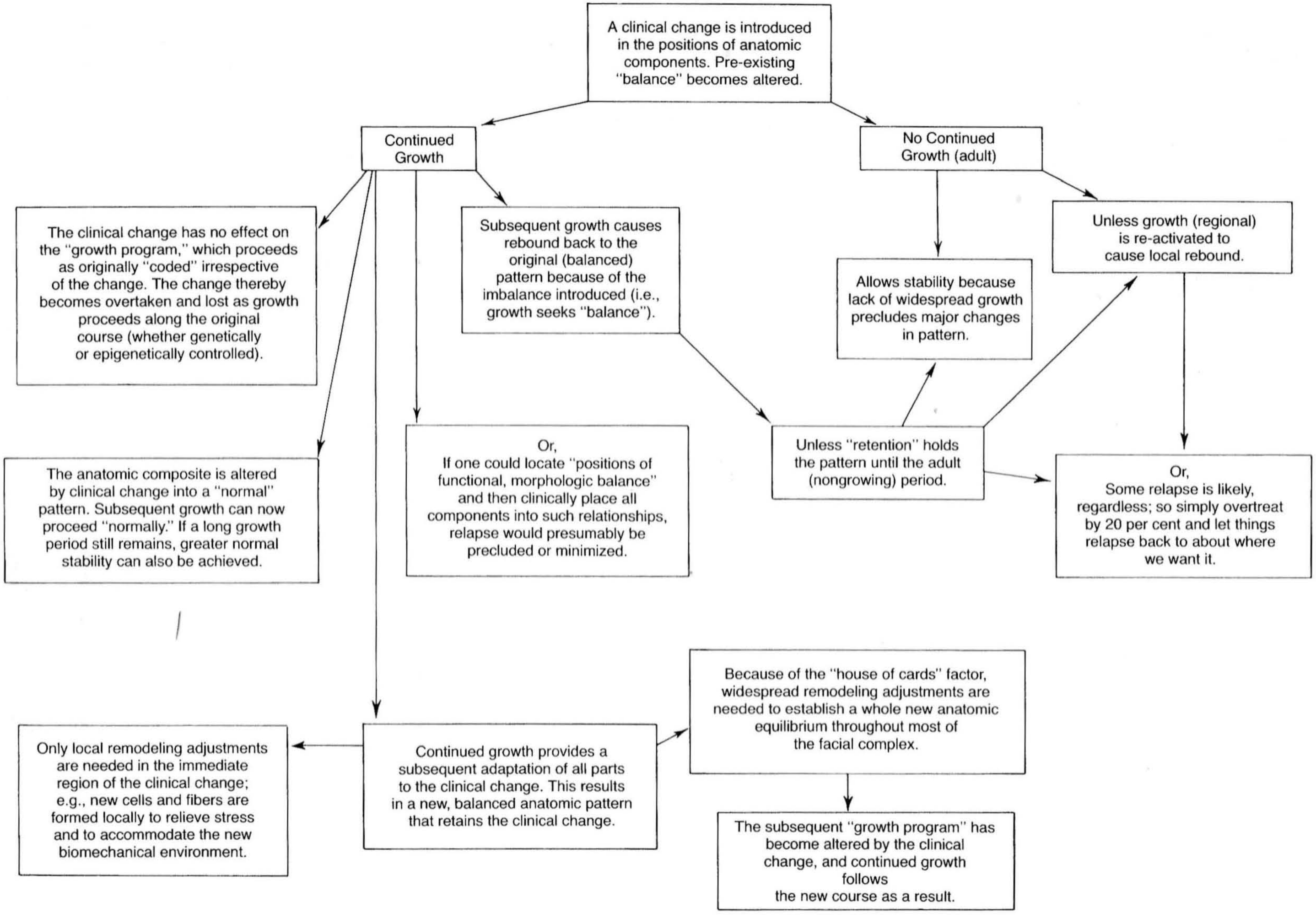

A clinical change is introduced in the positions of anatomic components. Pre-existing "balance" becomes altered.
Continued Growth
No Continued Growth (adult)
The clinical change has no effect on the "growth program," which proceeds as originally "coded" irrespective of the change. The change thereby becomes overtaken and lost as growth proceeds along the original course (whether genetically or epigenetically controlled).
Subsequent growth causes rebound back to the original (balanced) pattern because of the imbalance introduced (i.e., growth seeks "balance").
Allows stability because lack of widespread growth precludes major changes in pattern.
Unless growth (regional) is re-activated to cause local rebound.
The anatomic composite is altered by clinical change into a "normal" pattern. Subsequent growth can now proceed "normally." If a long growth period still remains, greater normal stability can also be achieved.
Or,
If one could locate "positions of functional, morphologic balance" and then clinically place all components into such relationships, relapse would presumably be precluded or minimized.
Unless "retention" holds the pattern until the adult (nongrowing) period.
Or,
Some relapse is likely, regardless; so simply overtreat by 20 per cent and let things relapse back to about where we want it.
Only local remodeling adjustments are needed in the immediate region of the clinical change; e.g., new cells and fibers are formed locally to relieve stress and to accommodate the new biomechanical environment.
Continued growth provides a subsequent adaptation of all parts to the clinical change. This results in a new, balanced anatomic pattern that retains the clinical change.
Because of the "house of cards" factor, widespread remodeling adjustments are needed to establish a whole new anatomic equilibrium throughout most of the facial complex.
The subsequent "growth program" has become altered by the clinical change, and continued growth follows the new course as a result.

FIGURE 1–1

Class I molar relationship with an aesthetically pleasing face is the common result in which the underlying factors that would otherwise have led to a more severe Class II or III malocclusion still exist but have been "compensated for" by the growth process itself. The net effect is an overall, composite balance.

As pointed out above, clinical treatment can disturb a state of structural and functional equilibrium, and a natural rebound can follow. For example, a premature fusion of some cranial sutures can result in growth-retarded development of the nasomaxillary complex because the anterior endocranial fossae (a template for midfacial development) are foreshortened, as in the Crouzon or Apert syndromes. The altered nasomaxillary complex itself nonetheless has grown in a balanced state proportionate to its basicranial template, even though abnormal in comparison with a population norm for esthetics and function. Craniofacial surgery has disturbed the former balance, and some degree of natural rebound can be expected. The growth process attempts to restore the original state of equilibrium, since some extent of the original underlying conditions (e.g., the basicranium) can still exist that were not, or could not be, altered clinically. These are examples in which the biology of the growth process is essentially normal, either with treatment or without, but is producing abnormal results because of altered input control signals.

The Big Picture

The following paragraph outlines a growth concept basic to the overall developmental process. It deals with the separate but **interrelated** and **interdependent** nature of the assembly of all the regional parts comprising the neurocranium (for the brain and associated sensory organs) and viscerocranium (face). It underscores the variety of developmental conditions in any given local region, but at the same time points to the necessary morphogenic and functional interplay among them.

No craniofacial component is developmentally self-contained and self-regulated. Growth of a component is not an isolated event unrelated to other parts. Growth is the composite change of all components. While this seems self-evident, it might be perceived, for example, that the developing palate is essentially responsible for its own intrinsic growth and anatomic positioning, and that an infant's palate is the same palate in the adult simply grown larger. The palate in later childhood, however, is not composed of the same tissue (but with more simply added), and it does not occupy the same actual position. Many factors influence (impact) that growing palate from without, such as developmental rotations, displacements in conjunction with growth at sutures far removed, and multiple remodeling movements that relocate it to progressively new positions and adjust its size, shape, and alignment continuously throughout the growth period. Similarly, for the mandible, the multiple factors of middle cranial fossa expansion; anterior cranial fossa rotations; tooth eruption; pharyngeal growth; bilateral asymmetries; enlarging tongue, lips, and cheeks; changing muscle actions; headform variations; an enlarging nasal airway; changing infant and childhood swallowing patterns; adenoids; head position associated with sleeping habits; body stance; and an infinite spread of morphologic and functional variations, all have input in creating constantly changing states of structural balance. As emphasized above, **development** is an architectonic process leading to an aggregate state of structural and functional equilibrium, with or without an imposed malocclusion or other morphologic dysplasia. Very little, if anything, can be exempted from the "big picture" of factors affecting the operation of the

growth control process, and no region can be isolated. Meaningful insight into all of this underlies the basis for clinical diagnosis and treatment planning. The direct target for clinical intervention must be the control process regulating the biology of growth and development.

A Cornerstone of the Growth Process

A grasp of how facial growth operates begins with distinction between the two basic kinds of **growth movement**. These are (1) remodeling and (2) displacement (Fig. 1–2). Each category of movement involves virtually all developing hard and soft tissues.

For the bony craniofacial complex, the process of growth **remodeling** is paced by the composite of soft tissues relating to each of the bones. The functions of remodeling are to (1) progressively create the changing **size** of each whole bone; (2) sequentially **relocate** each of the component regions of the whole bone to allow for overall enlargement; (3) progressively **shape** the bone to accommodate its various functions; (4) provide progressive fine-tune **fitting** of all the separate bones to each other and to their contiguous, growing, functioning soft tissues; and (5) carry out continuous structural adjustments to **adapt** to the intrinsic and extrinsic changes in conditions. Although these remodeling functions relate to childhood growth, most also continue on into adulthood and old age in reduced degree to provide the same ongoing functions. This is what in freshman histology is meant when it is stated that bones "remodel throughout life," but without an explanation of the reasons. Added to this, now, is that all soft tissues *also* undergo equivalent remodeling and for all of the same reasons.

In Figures 1–3 and 1–4, note that many external (periosteal) surfaces are actually resorptive. Opposite surfaces are depository. This is required in order to sculpture the complex configurations involved.

As a bone enlarges, it is simultaneously carried away from other bones in direct articulation with it. This creates the "space" within which bony enlargement takes place at the interface between bone-to-bone joint contacts. The process is termed **displacement** (also called "translation"). It is a physical movement of a whole bone and occurs while the bone simultaneously remodels by resorption and deposition (to an equivalent extent).

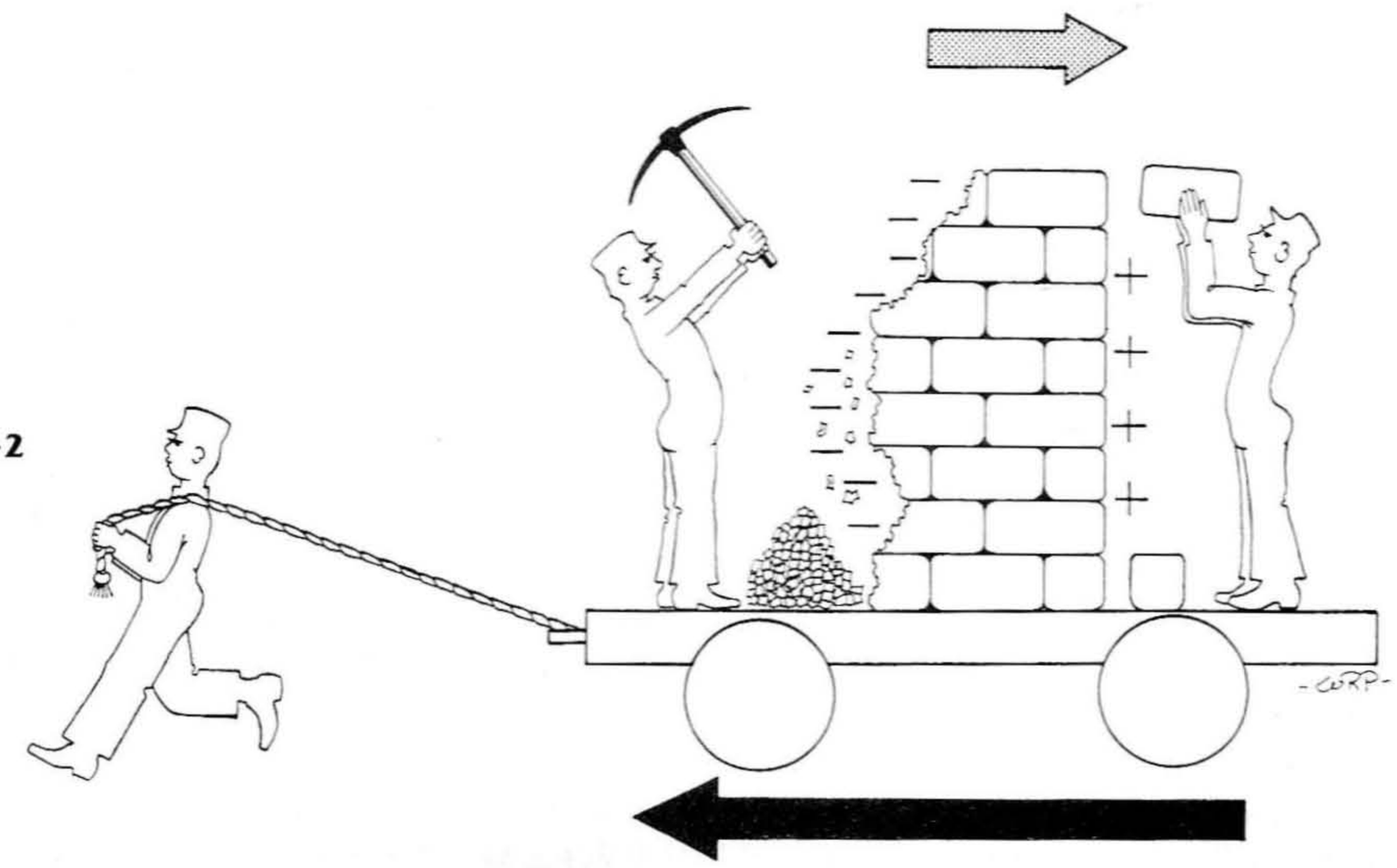


FIGURE 1–2

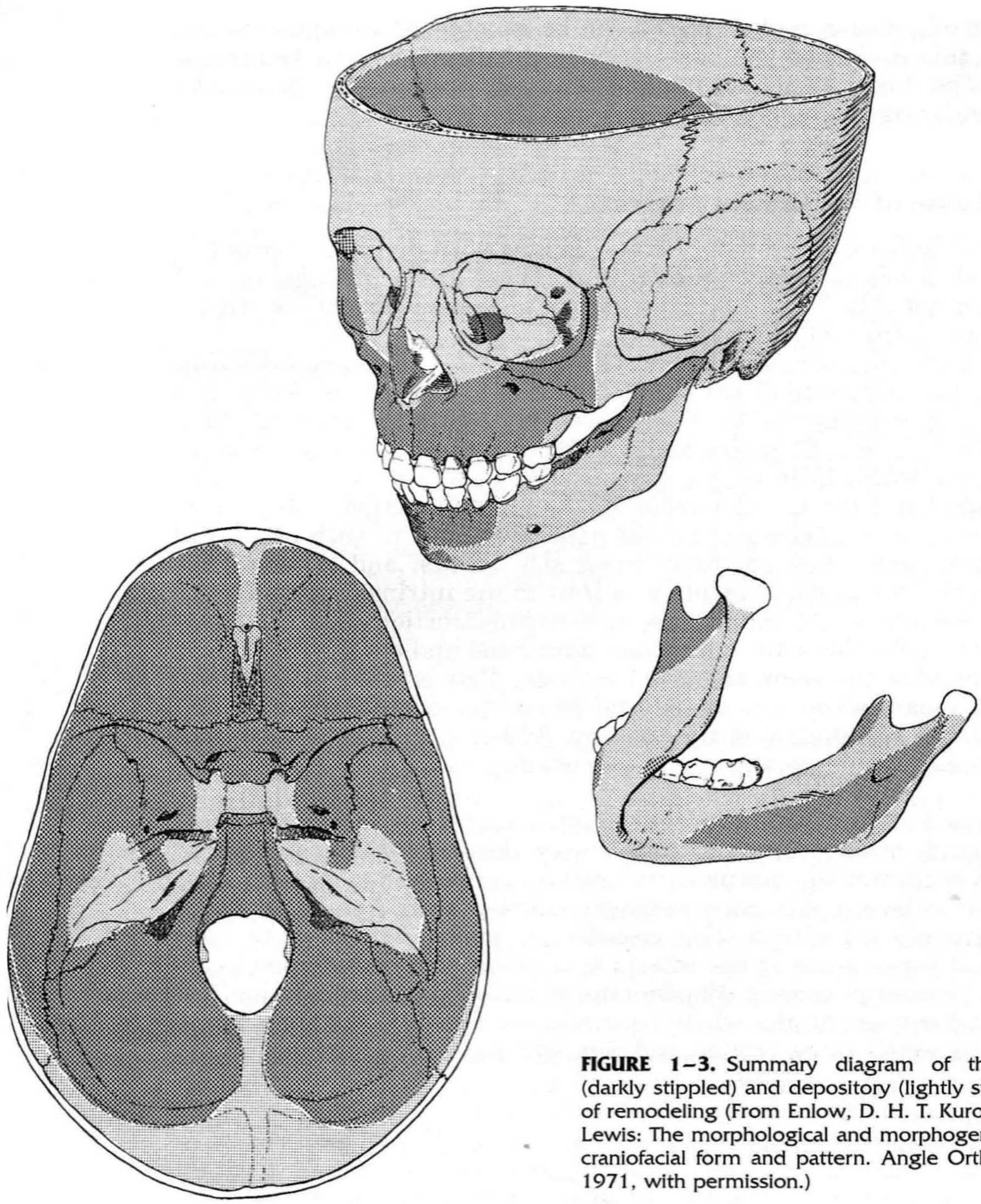


FIGURE 1–3. Summary diagram of the resorptive (darkly stippled) and depository (lightly stippled) fields of remodeling (From Enlow, D. H. T. Kuroda, and A. B. Lewis: The morphological and morphogenetic basis for craniofacial form and pattern. Angle Orthod., 41:161, 1971, with permission.)

As the bone enlarges in a given direction within the joint, it is simultaneously displaced in the **opposite** direction (Fig. 1–5). The relationships underscore why facial articulations (sutures and condyles) are important factors; they are direct clinical targets.

The process of new bone deposition does not cause displacement by **pushing** against the articular contact surface of another bone. Rather, the bone is **carried** away by the expansive force of all the growing soft tissues surrounding and attached to it by anchoring fibers. As this takes place, new bone is added immediately (remodeling), the whole bone enlarges, and the two separate bones thereby remain in constant articular junction. The nasomaxillary complex, for example, is in sutural contact with the floor of the cranium. The whole maxillary region, **in toto**, is **displaced** downward and forward away from the cranium by the expansive growth of the soft

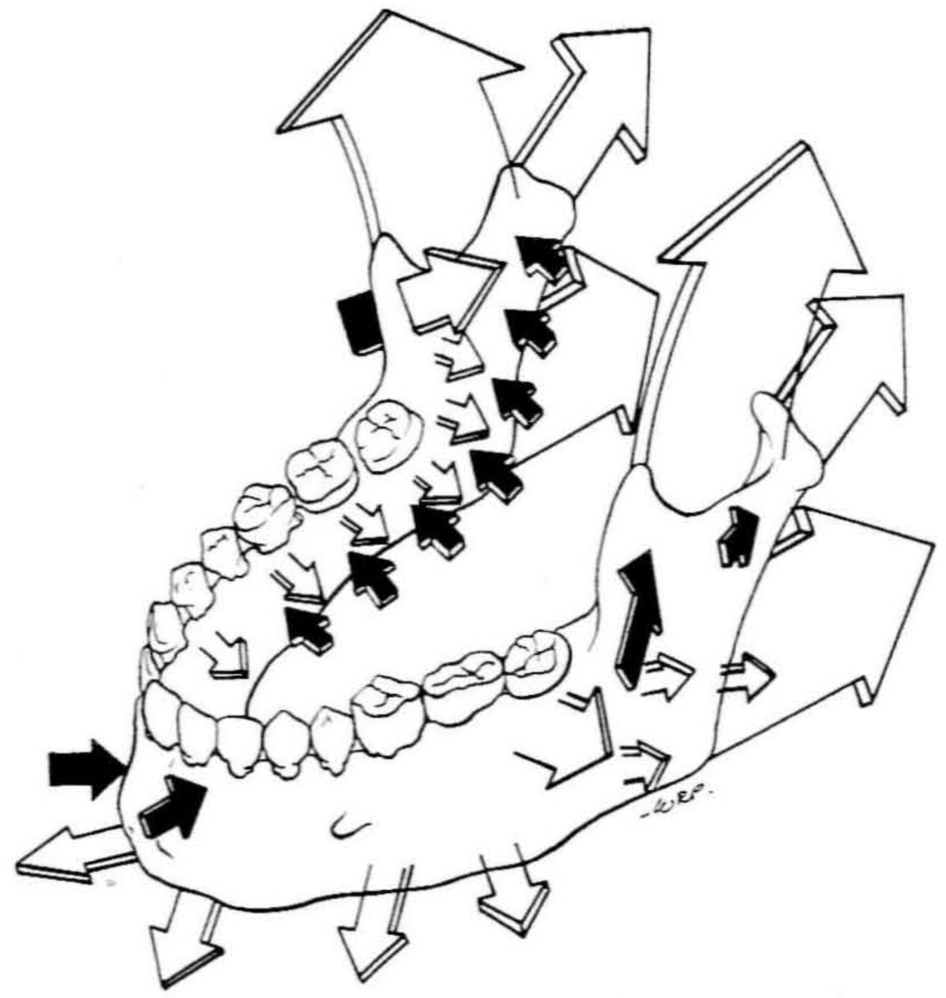


FIGURE 1–4. Black arrows are surface resorptive, and white arrows are depository.

tissues in the midfacial region (Fig. 1–6*A*). This then triggers new bone growth at the various sutural contact surfaces between the nasomaxillary composite and the cranial floor (Fig. 1–6*B*). Displacement thus proceeds downward and forward an equivalent amount as maxillary remodeling simultaneously takes place in an opposite upward and backward direction (i.e., **toward** its contact with the cranial floor).

Similarly, the whole mandible (Fig. 1–5) is **displaced** "away" from its articulation in each glenoid fossa by the growth enlargement of the composite of soft tissues in the developing face. As this occurs, the condyle and ramus grow upward and backward (relocate) into the "space" created by the displacement process. Note that the ramus also remodels in shape and size as it relocates posterosuperiorly. It becomes longer and wider to accommodate (1) the increasing mass of masticatory muscles inserted onto it, (2) the enlarged breadth of the pharyngeal space, and (3) the vertical lengthening of the nasomaxillary part of the growing face.

A beginning student is always confused because it is repeatedly heard and read that the face "grows forward and downward." It would seem reasonable, then, that the growth activity of the mandible or the maxilla would be in their anterior, forward-facing parts. However, it is mostly the displacement movement that is forward and downward, thereby complementing the predominantly posterosuperior vectors of remodeling. This is one fundamental reason, as mentioned above, that all joint contacts and bone ends are of basic significance in the growth picture. They are the points away from which displacement proceeds and, at the same time, the sites where remodeling lengthens a given bone. Thus, they are key locations where certain clinical procedures affect the growth process.

Note this significant point. If a non-biologic material, such as a metal or plastic plate or other prosthetic appliance, is implanted within the developing craniofacial complex, it lacks both of these two systems of growth movement! It cannot (1) move by patterns of REMODELING since resorptive and depository fields do not exist. It (2) cannot become moved by DISPLACEMENT through traction growth forces because the enlarging soft tissues are not anchored into its substance by Sharpey's fibers. The

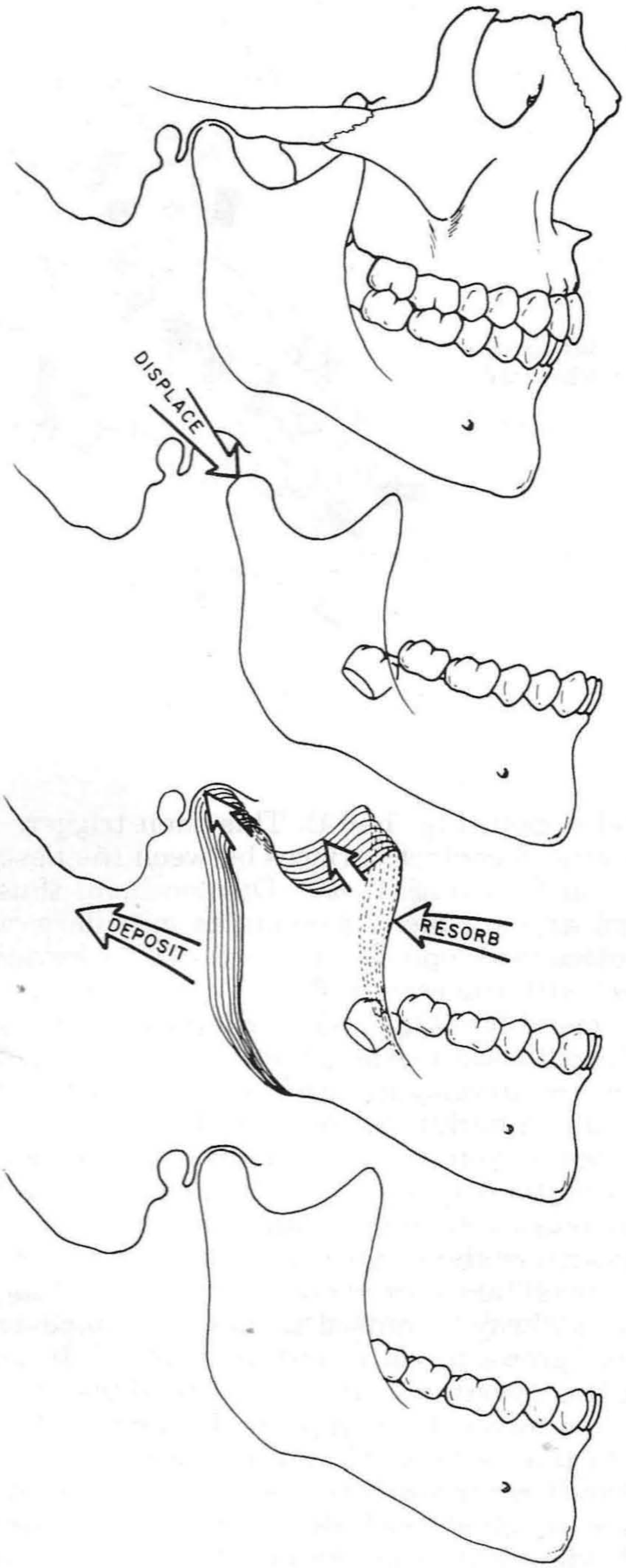


FIGURE 1–5

growing bone contiguous with any non-biologic material, thus, simply grows away from it as they become progressively disjoined. The skeletal as well as soft tissue parts previously around it when originally implanted thereby continue to (1) remodel and (2) displace, while the non-biologic material itself remains behind without dual biologic growth movement capacity. It now becomes a developmental block against any advancing tissues behind.

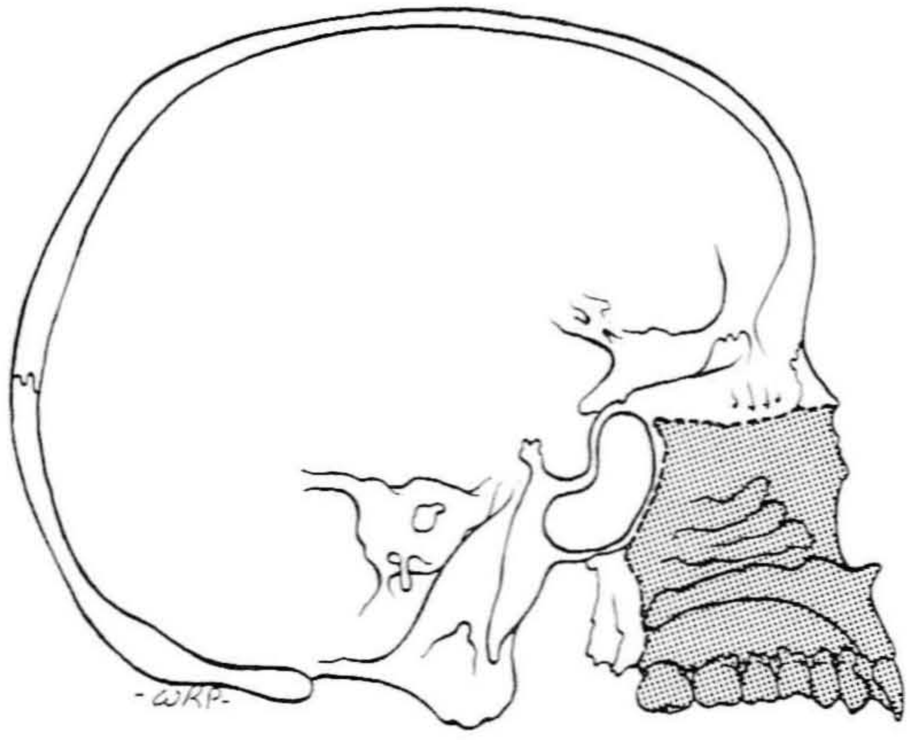
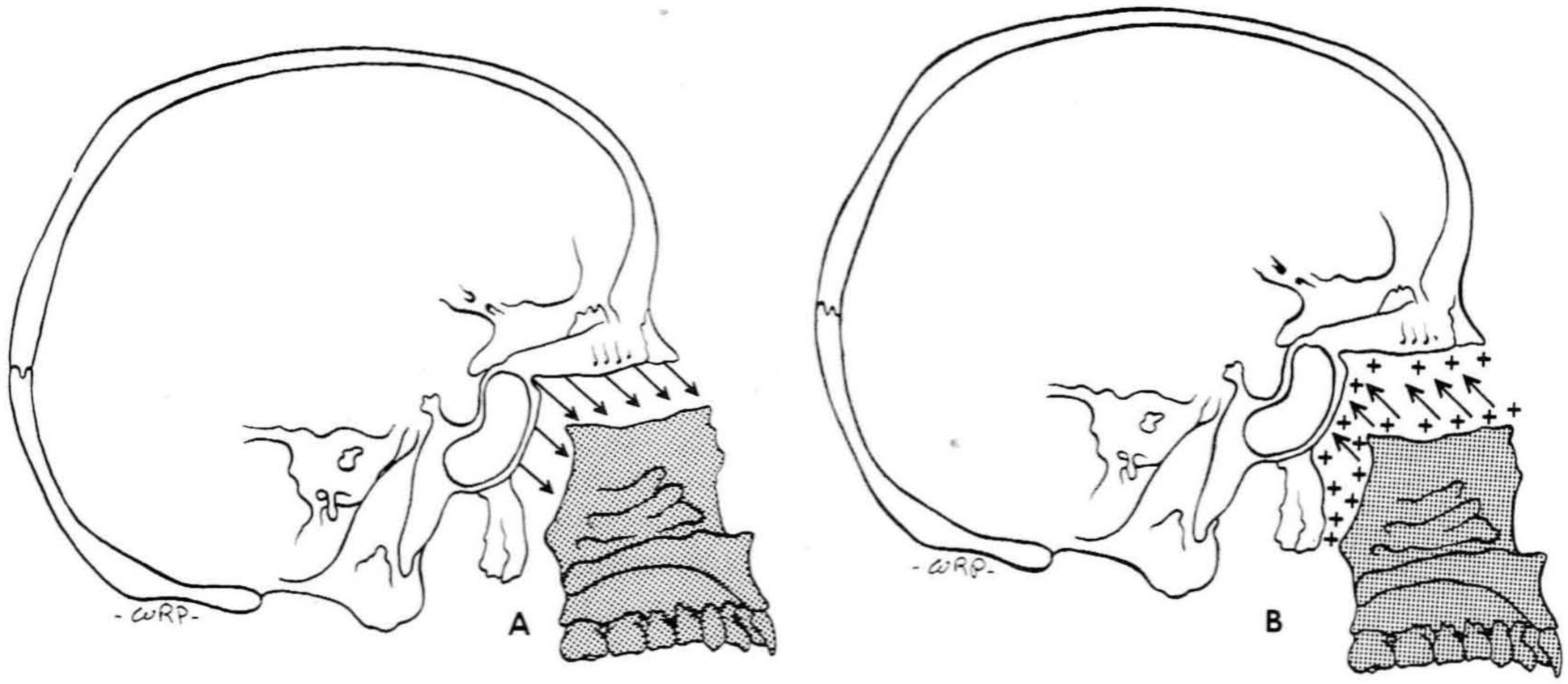


FIGURE 1–6



The "Genic" Tissues

The histogenic "blast" cells and tissues are activated by a shower of intercellular signals targeted toward the signal-sensitive cell membrane receptors of each cell type, including chondroblasts, osteoblasts, myoblasts, fibroblasts, neuroblastic (satellite) cells, and any other progenitor or undifferentiated cellular types. Signals include mechanical forces, bioelectric potentials, hormones, enzymes, oxygen tension, and other similar agents. Within each cell, a chain of reactions then passes through the cytoplasm to and from the nucleus, endoplasmic reticulum, or organelles such as lysosomes and mitochondria, ending either in (1) output of secretions such as alkaline and acid phosphatase, ground substance (protein mucopolysaccharides), and collagen; or (2) differentiation by cell divisions and maturation into specific tissue types comprising cartilage, bone, periosteum, muscle, epithelia, blood vessels, and lengthening nerves. Ongoing activating signals are "intrinsic" during development, but, as emphasized herein, are subject to clinical modification that then alters regulation of the same underlying biology. This affects the **timing** and **duration** of the cellular activity, and the growth **vectors** (magnitude and direction). It is the selective nature of the signals that governs the pattern of developmental activity that leads to variations in morphology, not any real change in the growth biology itself.

Regional Control of Development

Replacing the archaic notion of "master growth control centers" of yesteryear, is the understanding that tissues within each **local** area contain an array of cell types carrying out the specific developmental requirements of that area. Sensitive to the play of "primary messengers" (activating signals) relating to particular localized **functions** and structural relationships, each and every location has a developing size and shape that is custom-made by its own "genic" cells receiving the local information that determines it. Because the local signals continuously change, regional size and shape correspondingly and progressively adapt. Complex architectonic combinations of regional parts, such as those comprising the mandible and maxilla as a whole and all of the soft tissues associated with them, achieve their differentiating morphology by continuous adjustments among the developing local parts (condyles, coronoid processes, tuberosities, alveolar sockets, tubercles, etc.). This provides a precise and ongoing "fit" among all of them. Everything continues to function all the while.

With regard to the "goodness of fit" of separate bones, muscles, teeth, blood vessels, and all other such anatomic parts to each other, consider several examples illustrating the remarkable developmental interplay characterizing "growth." This interplay is a key factor that makes the whole thing work. When dealing with the growth process, we sometimes forget to appreciate this, or, actually, don't even think of it at all.

For example, a tooth **precisely** matches its alveolar socket in shape, size, and the timing of developmental changes and movements during growth. The osteogenic and fibrogenic periodontal connective tissue (1) shapes and progressively reshapes the bony socket, (2) moves the tooth (drift and eruption) by mechanical traction forces mediated by the collagenous fibers of the periodontal connective tissue, (3) moves the socket by remodeling, and (4) remodels its own periodontal connective tissue (fibrogenic) to sustain continuous attachment and to move itself in precise lock step with the moving tooth and bone.[†]

Another example of "goodness of fit" is a cranial nerve with its sheath of vascular connective tissue passing through a basicranial foramen. The configurational and dimensional fit and the positioning of the foramen must be absolutely perfect. As the nerve constantly moves with the growing brain, the remodeling of the bony passage precisely conforms. If such were not the case, development itself would reach a dead end. (See page 103 Chapter 6 for further phylogenic insight.) Another example is the precise match of a bony tuberosity to which a muscle inserts. There can be no misfit whatever between the two. The match is perfect because of their constant histogenic interplay. Also, any given bone fits precisely within its articular joint. Actually, tissues everywhere throughout the whole body involve virtually limitless adaptive interactions as a part of the growth process, and function continues all the while it happens.

Figure 1–7 schematizes this process. Although the growth activities involved are separated into little boxes, in real life such isolation of relationships, of course, is not possible because of the interdependence among them. This is one basic reason why so many laboratory experiments addressing the "determinants of growth" have historically yielded equivocal results: either (1) **all** of the categories were not taken into account (almost always), or (2) the experimental design often calls for separation of the

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[†]It is this same histogenic ("growth") process that is utilized in orthodontic tooth movement. Only the signals are changed in order to alter the directions and amounts of tooth movement.

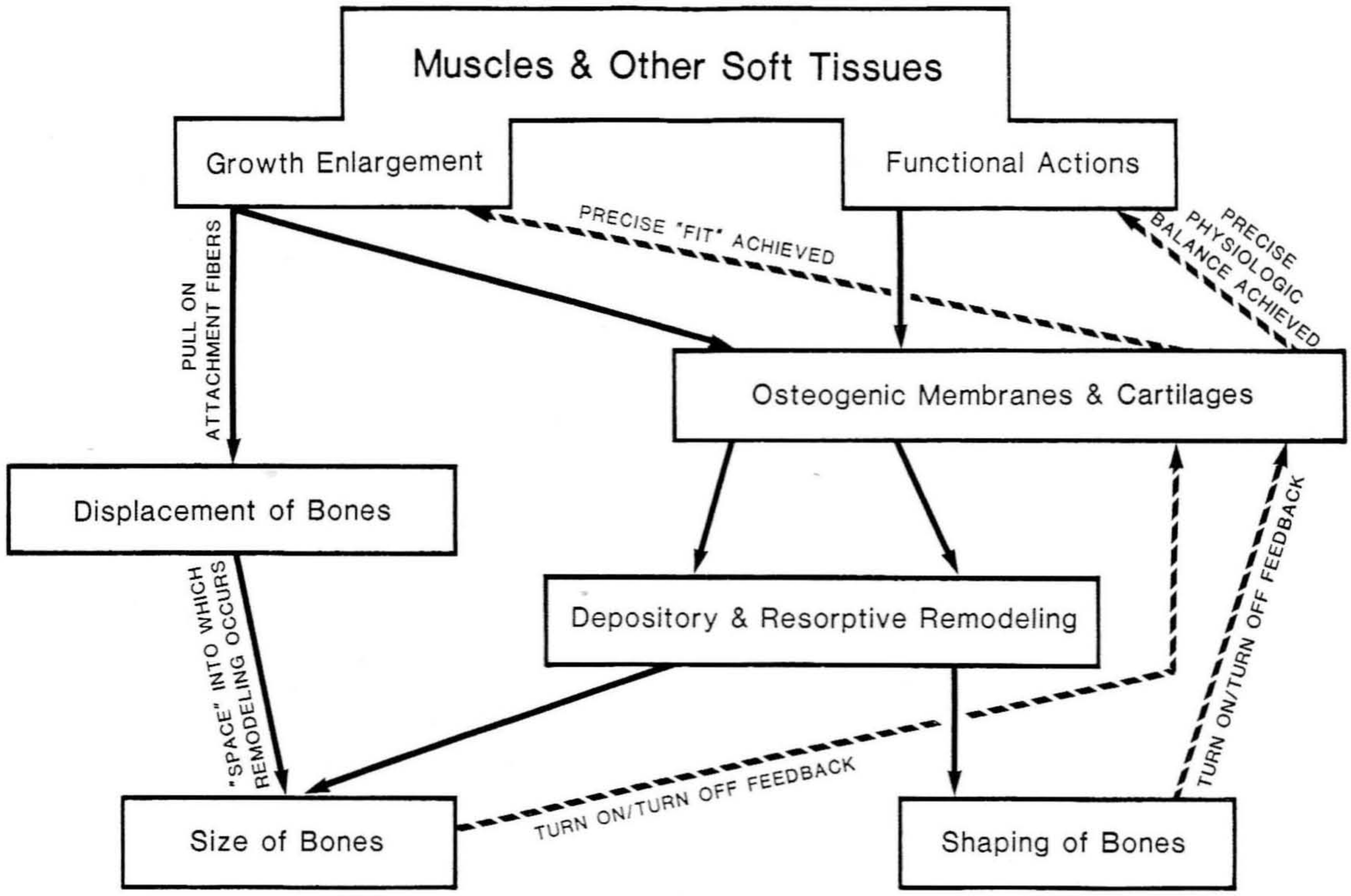

FIGURE 1–7. See text for discussion. (From Enlow, D. H.: Structural and functional "balance" during craniofacial growth. In: *Orthodontics: State of the Art, Essence of the Science*. Ed. by L. W. Graber, St. Louis, C. V. Mosby, 1986, with permission.)

categories in order to attempt to control variables, but which simply cannot be done. (See also later chapters.)

THE THREE PRINCIPAL REGIONS OF FACIAL AND NEUROCRANIAL DEVELOPMENT

The major but mutually interrelated form/function components involved in development are the brain with its associated sensory organs and basicranium, the facial and pharyngeal airway, and the oral complex. Although discussed below separately, they are, of course, developmentally inseparable. This interrelation factor is important in the clinical application of growth concepts since the developmental factors underlying most craniofacial dysplasias involve all three.

The Brain and Basicranium

The configuration of the neurocranium (and brain) determines a person's headform type which, in turn, sets up many of the proportionate and topographic features characterizing facial type. A long and narrow basicranium (dolichocephalic) with its more elongate and open-angle configuration, for example, programs the developmental process so that it characteristically leads to an anteroposteriorly and vertically elongate facial pattern and a more frequent built-in tendency for mandibular retrusion (Fig. 1–8, top).

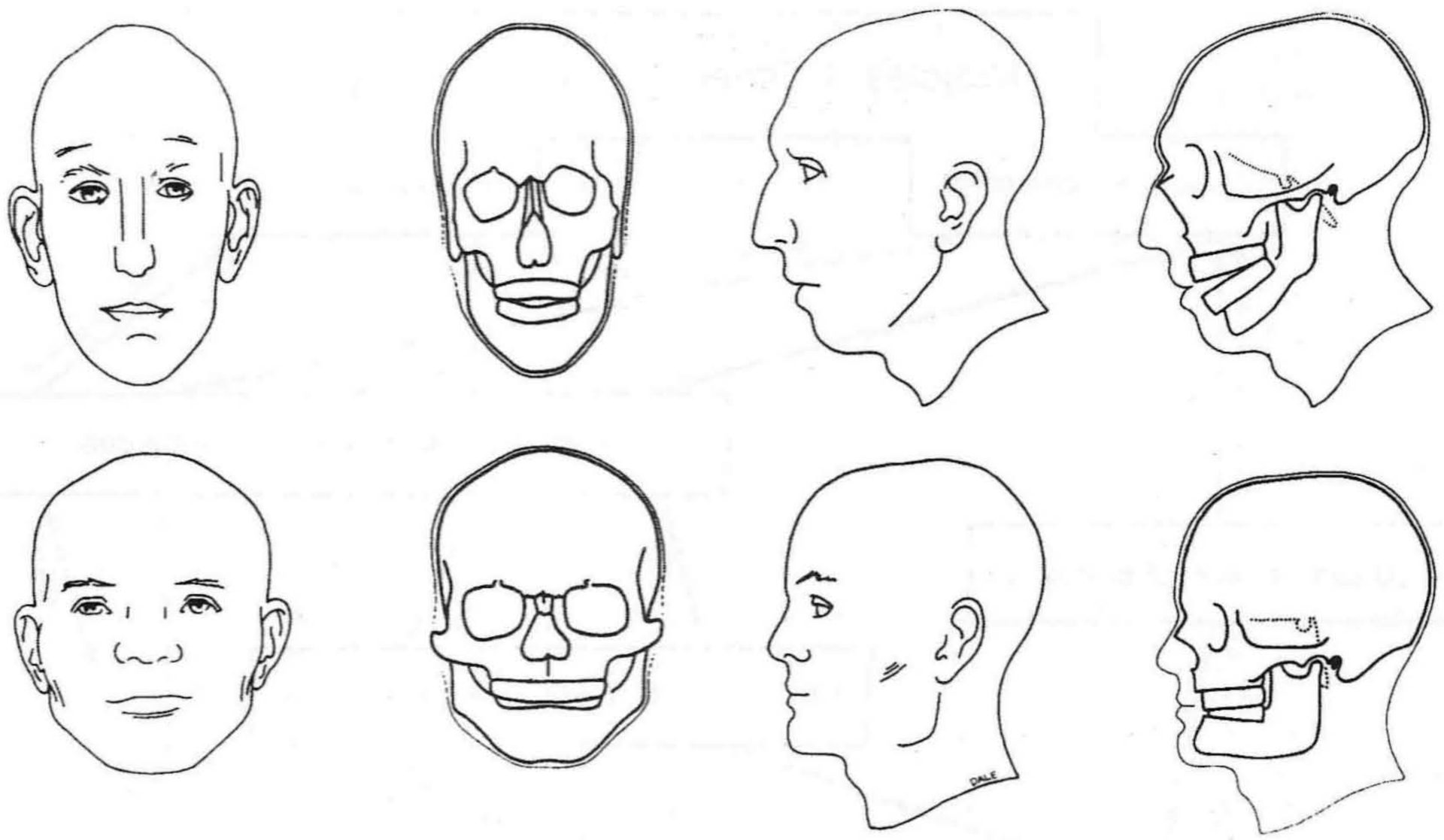

FIGURE 1–8. (From Enlow, D. H., and J. Dale: In: *Oral Histology*, 4th Ed. Ed. by R. Ten Cate, St. Louis, C. V. Mosby, 1994, with permission.)

A rounder basicranium (brachycephalic) is characterized by a proportionately wider but anteroposteriorly shorter configuration, a more obtuse basicranial flexure, and a vertically and protrusively shorter but wider midface (nasomaxillary complex). These features generally underlie a more orthognathic (or less retrognathic) profile or, in the extreme, a tendency for mandibular or bimaxillary protrusion (Fig. 1–8, bottom).

These characteristic features exist because the basicranium is the template that establishes the shape and perimeter of the facial growth field. The mandible attaches by its condyles onto the ectocranial side of the middle endocranial fossae, and the bicondylar dimension is thus determined by this part of the cranial floor. The nasomaxillary complex is suspended from the anterior endocranial fossae, and the width of the facial airway, the configuration of the palate and maxillary arch, and the placement of all these parts are thus established by it.

The Airway

The facial and pharyngeal airway is a space determined by the multitude of separate parts comprising its enclosing walls. The configuration and dimensions of the airway are thus a product of the composite growth and development of many hard and soft tissues along its pathway from nares to glottis.

Although determined by surrounding parts, those parts in turn are dependent upon the airway for maintenance of their own functional and anatomic positions. If there develops any regional childhood variation along the course of the airway that significantly alters its configuration or size, growth then proceeds along a different course, leading to a variation in overall facial assembly that may exceed the bounds of normal pattern. The airway functions, in a real sense, as a keystone for the face. A keystone, as you know, is that part of an arch which, if of proper shape and size, stabilizes the positions of the remaining parts of the arch. In Figure

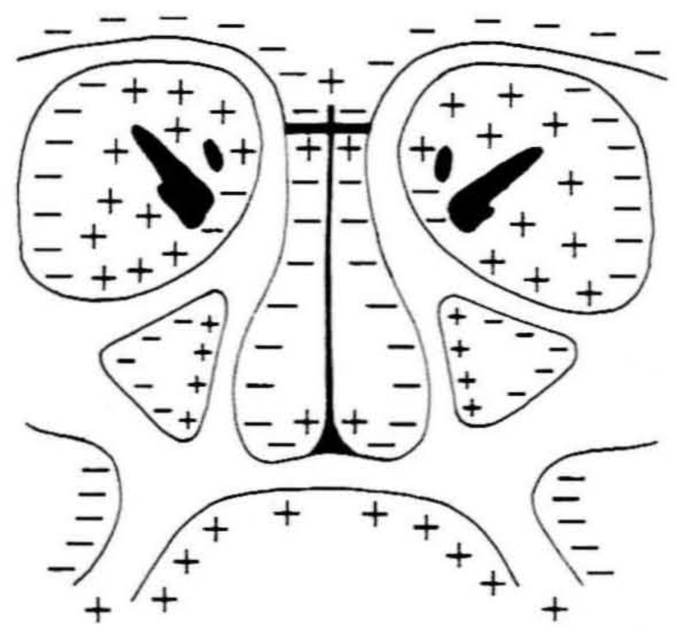

FIGURE 1–9

1–9 a few of the many "arches" in a face can be recognized, and the bony remodeling (+ and −) producing them. Horizontally and vertically, the archform of the orbits, the nasal and oral sides of the palate, the maxillary arch, the sinuses, the zygomatic arches, and so forth are all subject to airway configuration, size, and integrity. Note that the airway is strategically pivotal to all of them.

Two easy personal tests can be performed illustrating the airway as a significant factor in programming the developmental course of the facial "genic" tissues. This is useful in explanations of malocclusion etiology for patients or their parents.

First, starting with an open mouth, close the lips and jaws, noticing that you likely raise the tongue against the palate and, momentarily, swallow. This evacuates the oral air into the pharynx, creating an oral vacuum. The effect is to stabilize the mandible and hold it in a closed position with minimal muscle effort. Now, open the jaws and lips, feeling a rush of air into the mouth. To hold the lower jaw in this "mouth breathing" posture requires a different pattern of muscle activity, and the osteogenic, chondrogenic, periodontal, fibrogenic, and other histogenic tissues thereby receive a correspondingly different pattern of signals. This causes different developmental responses to a different functional morphology adapted to the conditions. As emphasized before, the operation of the growth process itself functions normally. It is the nature of the **activating signals** that produces emerging deviations in the course of development that results in any morphologic variation and perhaps malocclusion.

Another test is similar. With closed jaws and lips compared to open, try swallowing. Open-jawed swallows are possible, but can be difficult when one is accustomed to a closed mouth. Note the very different pattern of masticatory and hyoid muscle actions required. As with the mouth-breathing test outlined above, altered signals are generated, and the genic tissues work toward a different balance combination, producing a variation in facial morphology. A factor often overlooked by clinicians is that these altered signals may result in different treatment responses to the same intervention. For example, a patient's response to a Frankel appliance may vary dramatically based upon the patient's mode of breathing.

The Oral Region

In addition to the basicranial and airway factors described above affecting mandibular and maxillary shape, size, and positioning, other basic considerations are involved. If a brain and basicranial asymmetry exists, this condition can either be (1) passed on to cause a corresponding facial asymmetry, or (2) compensated by the facial developmental process to

either offset or reduce its magnitude. For the latter, remodeling adjustments produce an actual opposite asymmetry in the nasomaxillary complex and/or mandible that counteracts the basicranial condition.

For the maxilla, if not developmentally compensated or only partially so, the maxillary arch can become deviated laterally, matching the lateral asymmetry of the anterior endocranial fossae. (See Thimaporn et al., 1990.) Or, vertically, one side can become lowered or elevated relative to the other, including the orbits, palate, and maxillary arch. For the mandible, the middle endocranial fossae determine the placement of the temporomandibular joints and, if asymmetric, one or the other will be lower or higher, forward or back. Whole-mandible alignment necessarily follows if not fully or partially adjusted by remodeling during development.

Many other such compensatory adjustments by the remodeling process occur throughout growth and development in many ways, as discussed in subsequent chapters. It involves the development of certain regional imbalances to offset others, resulting in a composite overall structural and functional equilibrium.

Craniofacial Levels

There exists a descending, cause-and-effect stratographic arrangement of structural **levels** in the design of the face. Beginning with the frontal lobes of the cerebrum, the floor of the anterior endocranial fossae become adapted in size and shape during their interrelated development. The ectocranial side of this floor is the roof of the nasal chambers, thus programming the perimeter of that key facial part of the airway. This configuration, in turn, is projected inferiorly to the next level, establishing the proportions and configuration of the nasal side of the palate. Then, the perimeter of the apical base of the maxillary dental arch is set by the oral side of the hard palate, all representing configurational projections from the anterior endocranial fossae. The next level following is the maxillary cuspal arch, and then the mandibular bony and dental arches, all preprogrammed in configuration and proportions in descending order from the basicranium.

The mandible has a component not represented in the maxilla, and that is its **ramus**. The anteroposterior size of the ramus develops by an amount approximating the horizontal span of the pharynx, which has a programmed anteroposterior dimension established by **its** ceiling, which is the ectocranial side of the middle endocranial fossae underlying the temporal lobes of the cerebrum. The ramus, thus, places the mandibular arch in occlusion with the maxillary arch following a pattern set up by the basicranium. Vertically, the developing ramus lowers the corpus by progressive amounts, adapting to the vertical growth of the middle cranial fossae (clivus) as well as the vertical expansion of the nasal airway and developing dentition.

The face, thus, is a stratified series of vertical levels all sharing a common developmental template. This makes possible a workable morphogenic system having a structural design allowing large numbers of separate parts to develop together in harmony and to carry out respective functions while it happens.

The Two Basic Clinical Targets

There is one developmental concept that needs to be addressed with particular emphasis because of its great significance to the old clinical axiom "working with growth." While a factor such as the basicranium can

prescribe and determine a "growth field" in the contiguous facial complex, as described above, it is within the boundaries of that field that **remodeling** then engineers the **shape and size and functional fit** of all parts and develops them through time. However, it can be misunderstood if one presumes that all "local growth" is regulated solely by a single local, intrinsic growth system. Remember, there are **two** kinds of growth activity: (1) localized, regional **remodeling** ("genic" tissues), and (2) the **displacement** movements of all the separate parts as they remodel. Thus, there are two corresponding histogenic recipients of clinical intervention.

To illustrate this fundamental concept, the incisor and premaxillary alveolar region of the maxilla develops into its adult shape and dimensions by the local remodeling process. But the principal source of the considerable extent of its downward-and-forward growth movement is by displacement, and **that** comes from biomechanical forces of growth enlargement occurring **outside** the premaxillary region itself. Thus, most of the growth movements responsible for the anatomic **placement** of this region, along with, passively, its teeth, are not controlled within its own tissues or any genetic blueprint therein, even though this might be a natural presumption. **Two** clinical targets thereby exist for orthodontists: local remodeling and, separately, the displacement of some whole part produced by the sum of developmental expansions occurring everywhere. There are certain clinical procedures that relate specifically to one or the other target, and some that involve both. For example, rapid palatal expansion mimics displacement; incisor retraction primarily involves remodeling of the anterior portion of the alveolar arch, and Bionator treatment involves both remodeling of the alveolar process and displacement via changes in the ramus.

These two basic growth movements are often not separated in the clinical literature. Unfortunately, this historical oversight severely limits the clinician's ability to provide a biologic explanation of how any given treatment procedure actually operates.

Child-to-Adult Changing Proportions

The three principal craniofacial growing parts (brain and basicranium, airway, oral region) each has its own separate timetable of development even though all are inseparately bound as an interrelated whole. Some body systems, such as the nervous and cardiovascular systems, develop earlier and faster compared to others, including the airway and oral regions. The reason is that airway growth is proportionate to growing body and lung size, and the oral region is linked to developmental stages involving the fifth and seventh cranial nerves and associated musculature, the suckling process, dental eruption stages, and masticatory development.

The infant and young child are characterized by a wide-appearing face because of the precociously broad basicranial template, but the face otherwise is vertically short (Fig. 1–10). This is because the nasal and oral regions are yet diminutive, matching the smallish body and pulmonary parts and with masticatory development in a transitory state. The mandibular ramus is vertically yet short because it is linked in developmental feedback with the shorter, later-maturing nasal and dental regions. Masticatory musculature is proportionately sized and shaped to progressively match increasing function and to interplay developmentally with the ramus.

During later childhood and into adolescence, vertical nasal enlargement keeps pace with growing body and lung size, and dental and other oral components have approached adult sizes and configuration. The mandibular arch is lowered by increasing vertical ramus length. Overall, the early

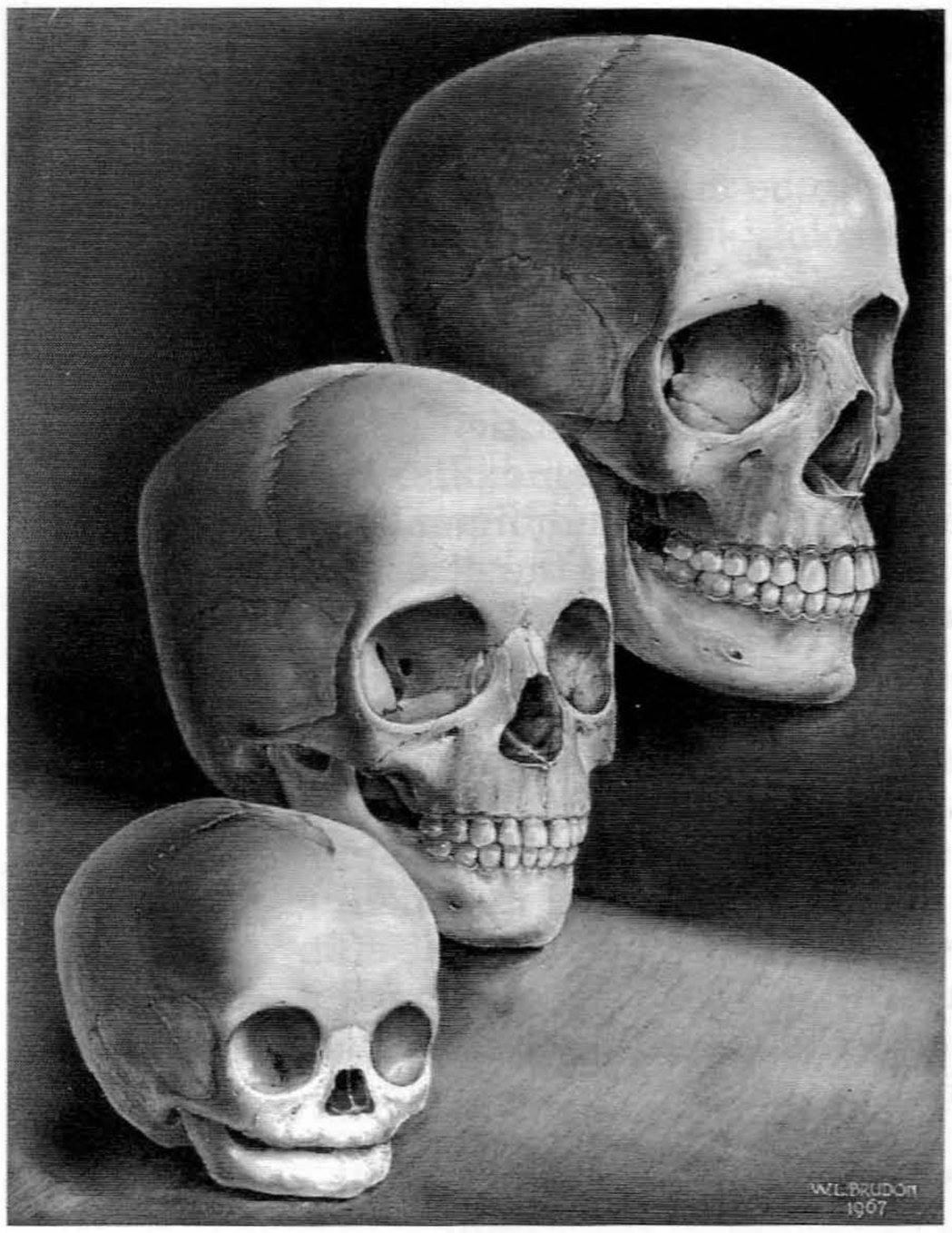

FIGURE 1–10. (Courtesy of William L. Brudon. From Enlow, D. H.: *The Human Face.* New York, Harper & Row, 1968, with permission.)

wide face has become altered in proportion by the later vertical changes. The end effect is particularly marked in the dolichocephalic long-headed and long-face pattern, and less so in the brachycephalic headform type.

Tooth Movement

As with all other sections of this introductory chapter, the subject of how a tooth undergoes intrinsic growth or clinical movements is elaborated in subsequent pages.

To begin, a tooth is moved by either or both of two developmental means: (1) by becoming actively moved in combination with its own remodeling periodontal connective tissue and alveolar socket; and (2) by being carried along passively as each entire maxilla or mandible becomes displaced anteroinferiorly during facial morphogenesis. A second basic and clinically significant concept is that **bone and connective tissues** (such as the periodontal connective tissue, periosteum, endosteum, and submucosa, all of which participate directly and actively in a tooth's movement) have an intrinsic remodeling process that, when activated, move themselves as a growth function. When a tooth is moved, these other contiguous parts move with it by their own "genic" remodeling process to sustain relationships. A tooth, however, **cannot move itself** in a comparable manner by its own remodeling. It is mobile, but not motile. A tooth is **moved** by a biomechanical force external to the tooth itself, and there is an elaborate "biology" in the composite process that produces a tooth's growth movements. A tooth must move (drift, erupt, etc.) during maxillary and mandibular growth in order to become properly placed in progressively changing anatomic positions (see Chapters 3, 4, and 5). Whether the force producing the tooth's change in position is intrinsic *or clinically induced*,

the actual biology is the same. As mentioned again because the point is important, it is the nature of the activating **signals** that is different, and this causes (1) the multiple array of genic tissues to alter the course of remodeling or (2) the displacement process of a whole bone to become altered in direction or magnitude.

Drift

A worthy advance was made when it was realized that teeth undergo a process of ***drift***. For many years this fundamental concept was limited to horizontal (mesial and distal) movements, and the essential function was held to be a stabilization of the dental palisade to compensate for interproximal attrition. Added to this, now, is that drift has a basic **growth** function. It serves to anatomically place the teeth as the maxilla and mandible enlarge. Such movements are significant considering that a jawbone lengthens considerably from prenatal to adult sizes. Also, the original drift concept was for horizontal movement. Important to the clinician, now, is awareness that teeth, especially maxillary, have a marked **vertical** extent of drift. This is in addition to "eruption" and should not be so termed. **Vertical drift** is a basic growth movement the clinician "works with" because it can be modified by clinical intervention (i.e., orthodontic treatment).

Just as teeth undergo a drifting movement, the bone housing them also moves. Unlike a tooth, however, bone moves by the remodeling action of its enclosing osteogenic membranes, and this is also a direct target for clinical intervention. The intrinsic coordination of these bone-tooth movements is remarkable.

A Fundamental Principle of Growth

It has been emphasized in the preceding pages that facial growth is a process requiring intimate morphogenic **interrelationships** among all of its component growing, changing, and functioning soft and hard tissue parts. No part is developmentally independent and self-contained. This is a fundamental and very important principle of growth. As underscored earlier, the growth process works toward an ongoing state of composite functional and structural equilibrium. In clinical treatment, no key anatomic part can be fully segregated and altered without affecting "balance" with other parts and their state of physiologic equilibrium as well.

In essence, orthodontic treatment seeks to maximize the effectiveness of anatomic compensations to achieve an aesthetically harmonious masticatory system.