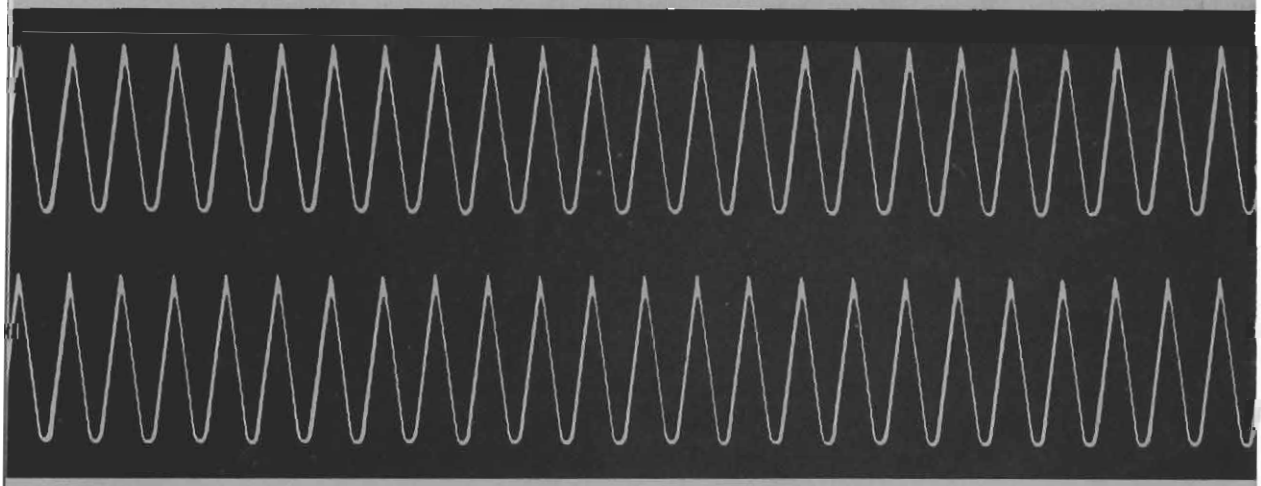
# Patterns of Orofacial Growth and Development

## Proceedings of the Conference



ASHA REPORTS

NUMBER 6 A PUBLICATION OF THE AMERICAN SPEECH AND HEARING ASSOCIATION

PROCEEDINGS OF THE CONFERENCE
PATTERNS OF OROFACIAL GROWTH AND DEVELOPMENT

This Conference was supported in part by
NIH Training Grant DE 00210-03
National Institute of Dental Research
U.S. Department of Health, Education, and Welfare

Proceedings of the Conference

# Patterns of Orofacial Growth and Development

Ann Arbor, Michigan
March 4-6, 1970

*ASHA Reports 6*
AMERICAN SPEECH AND HEARING ASSOCIATION
Washington
March 1971

## A CONFERENCE ON PATTERNS OF OROFACIAL GROWTH AND DEVELOPMENT




ASHA REPORTS NUMBER 6

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# PREFACE

The Conference on Patterns of Orofacial Growth and Development is an outgrowth of deliberations of the Joint Committee on Dentistry and Speech Pathology-Audiology. The Committee was established in 1966 with the support of the National Institute of Dental Research to bring together representatives of the American Speech and Hearing Association and the American Association of Dental Schools to review the common interests and concerns of dentists and communication pathologists.

The basis for joint action between the two groups was found in (1) areas of common interest in orofacial anatomy and physiology; (2) study of growth and development affecting orofacial structure and function; (3) areas of informational overlap and supplementation believed to exist between the educational objectives and curricula of the two academic fields; (4) identification and study of management techniques in the diagnosis and treatment of orofacial pathologies which affect speech and hearing, mastication, and deglutition; and (5) research to identify the areas of ignorance and uncertainty, and to expand the body of knowledge needed by each discipline to assure success in fostering orofacial health and function.

In recognition of these points of common interest, the Joint Committee, acting for its sponsors, established as its major goals the promotion of (1) the continuous exchange of information between the two professions, especially with reference to ongoing research; (2) the education and orientation of researchers in each profession to undertake cooperative research with members of the other profession; (3) the placement of speech pathologists and audiologists and dentists in the educational programs of the other profession; and (4) the development of a widespread awareness of the mutually valuable clinical and research contributions of each profession.

As discussions continued it became evident that to meet these goals a mechanism was needed to encourage joint professional interaction. In February of 1969 NIDR agreed to fund the continuation of the Joint Committee and to support a series of three conferences to discuss patterns of orofacial growth and development, respiratory physiology, and oral sensation and perception.

The charge to participants of this Conference on Patterns of Orofacial Growth and Development is to examine critically the information and the concepts presented, to discuss these matters here, and to leave the conference to apply them toward the further advancement of the joint objectives of Dentistry

and Speech Pathology-Audiology. Out of this conference and the succeeding ones we hope to stimulate new concepts, to generate new information, and to foster the productive efforts of the expanding groups of students of oral biology.

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# KEYNOTE ADDRESS: SOME OBSERVATIONS ON DENTAL RESEARCH AND THE CHANGING HEALTH SCENE

SEYMOUR J. KRESHOVER

*National Institute of Dental Research, Bethesda, Maryland*

In a recent book by Peter Drucker (1969), these times have been labeled the "age of discontinuity." Although Drucker is concerned with world economy and technology, I think it is fair to apply the term to some recent phenomena that will affect biomedical research and practice in the health professions. I should also like to take refuge in the concept of discontinuities, and ask that I be permitted to explore more than one in this paper. Special interests in the area of growth and development have been treated in depth and with great competence elsewhere during this conference. Therefore, my comments will be general ones about some aspects of the changing scene with which we are all very much concerned.

Drucker's premise is that discontinuities will largely shape our times. The revolutionary change in the concept of health as a right rather than a privilege affords a quick parallel to his statement that we are witnessing something new in human history, namely, "The whole world has become one economy in its expectations, in its responses, and in its behavior." Certainly, the thesis that poverty need not be tolerated was inconceivable 30 years ago.

There is also a similar naiveté towards the fulfillment of economic and health expectations. As a poignant example of the hazards inherent in the new economic world view, Drucker cites the growth since the Second World War of so-called "cargo cults" in remote South Sea Islands. These cultists believe that the President of the United States has sent a magic cargo ship "that will one day land and bring to everyone the wealth of the advanced economies—phonographs and smoked meats, transistor radios, motorcycles, and drugs."

The mounting demands for medical services, fueled by Medicare, Medicaid, and insurance plans, are severely taxing already overburdened facilities, and no magic cargo ship can promise instant gratification. Health, Education, and Welfare officials have soberly declared that "this nation is faced with a breakdown in the delivery of health care unless immediate concerted action is taken by Government and the private sector" (*Fortune*, 1970).

Although the demand for dental care has perhaps not kept the top pace, it is expected to leap dramatically. One reason is the growing awareness of

dental disease as a health problem. In a recent survey, more than three out of every four people interviewed agreed that every person has the right to receive needed dental care free of cost if he cannot afford to pay for it. Another reason is the increasing inclusion of dental plans as fringe benefits in union negotiations with employers.

Only 6½ million Americans are estimated to have dental coverage now. Inflation is blamed for this slow growth rate, and this factor may well curtail its expected expansion. Nevertheless, predictions now range from a coverage of 15 million to 30 million persons by 1975.

The fact that needs and opportunities in the health field are out of phase with each other is evident not only in care but also in research. Here, too, the forces of inflation have steadily eroded the nation's biomedical research effort.

The increasing cost of doing research has been as sharp a knife as reduced appropriations. The resultant discontinuity in the orderly growth of biomedical research, viewed against the changing health scene, imposes a stern obligation on all administrators—whether in governmental, educational or other institutions—to strive for a balance of research that will yield the greatest return for the tax dollar.

I believe that dental research leaders in many federal and academic settings have marched ahead of the drummer, in that, for the past several years, they have increasingly considered program relevance as well as scientific merit in setting priorities and administering programs. At the National Institute of Dental Research this focus on relevance has been sharpened as we try to realize the fullest potential of research for advancing levels of oral health. Assessment of our progress has now evolved to the point where we can chart more graphically and precisely a continuum of specific disease-oriented projects. This approach, I believe, will enable us to marshal most effectively the resources at hand; to identify areas where progress is blocked for lack of an adequate underpinning of basic knowledge; and to expedite the progress of research from the fundamental, through the developmental, and thence to the applied stage.

Our so-called National Caries Program is an example of a clearly designed research target. The President's budget requests an additional $5 million in the next fiscal year to enable the National Institute of Dental Research to mount a program of intensified research and development to make caries almost completely preventable by 1980.

Surely, the potential impact of biological research on the delivery of health care needs no fortune-teller to predict. Two examples of current investigation in the caries field illustrate the point. Both in the use of intensive local applications of fluoride and in the use of adhesive sealants, we have potential preventive techniques that can be practiced by auxiliary personnel, thereby freeing the dentist for tasks requiring greater professional judgment and skill.

Needless to say, not all of our oral problems are amenable to simplified approaches. Nor has dentistry always been responsive to the need for interdisciplinary cooperation to meet some of these problems. Improved interaction

between speech science and dentistry is certainly one area of deep concern. Being reminded, as we have, that studies of orofacial and speech problems are in urgent need of increased attention, the Dental Institute has accelerated its efforts for the training of specialists qualified to conduct research in this field. We see, too, an invaluable by-product of attracting young investigators to this field, namely, their recruitment into the dental school through joint appointments. Most gratifying in this latter regard is the number of dental schools that now incorporate speech pathology in their curricula—at last count, at least 10. And so we find with increasing specialization in our dental schools, new strengths in the complementary nature of many sciences—in the unity of knowledge. Let us never be found in the position of the hillbilly husband who one evening, with pencil and paper in hand, suddenly shouted triumphantly to his wife, "By gosh if I ain't learned to write!" "What does it say?" exclaimed the wife. "I don't know," he said, "I ain't learned to read yet!"

Aristotle is supposed to have said that the guests will judge the feast better than the cook. The effective harnessing of all the skills and knowledge that relate to a complex problem is very much in the public interest and will, in the long run, also better serve the respective professions; for our continued functioning, in both research and practice, is dependent on how well we discharge our responsibilities to an ever-demanding clientele.

The January 1970 issue of *Fortune* speaks stridently of our ailing medical system. It predicts radical change in these terms:

> The conversion to modern methods, and the institution of the same degree of efficiency that Americans have reached in other realms, would probably effect enough saving so that good care could be brought to every American with very little increase in costs. Nobody except other physicians should tell physicians how to practice medicine. But the management of medical care has become too important to leave to doctors, who are, after all, not managers to begin with.

Surely, we can predict that the long arm of change must soon reach to the professional school. Indeed, it has already arrived! As observed recently[1] by Robert Q. Marston, the Director of the National Institutes of Health, the relative immunity enjoyed by the health field, particularly education, from the social currents swirling around us is plainly a thing of the past, and "Whether we like it or not, we are involved to the hilt in the social dynamics of our times."

The discontinuity of the times has already had an impact on dental education. Future practitioners are learning how to extend their productivity by working with dental auxiliaries. More recently, the establishment of departments of community dentistry has added a new social dimension to dental education. Similarly, the growing attention to research in new techniques to improve learning is long overdue. An equally compelling need, as yet largely unmet, is to redesign the curriculum to reflect the advances in the dental sciences that will shape the practice of tomorrow. The recent statement of a dental dean, that most dentists are over-educated for what they do and under-

[1] Speech at the Fall Retreat of the McGill University Faculty of Medicine, Montreal, Canada, October 24, 1969.

educated for what they ought to be doing, assumes greater urgency in the light of projected advances against caries and other diseases.

The need to strengthen the science base, of course, implies the availability of research-trained teachers. Here, unfortunately, dental schools are seriously handicapped. The regrettable statistics show a current level of but 2500 full-time equivalent faculty members to staff our 53 operating dental schools (less than 50 per dental school, versus some 175 per medical school). Even more alarming is the urgent need to double this figure by 1975 if we are to adequately staff six new dental schools and those others that are now in process of expansion.

Without question, the late start of dental research has greatly disadvantaged dental schools and the progress of the dental sciences. For example, there is one biomedical research worker for every seven active physicians, versus one dental researcher for every 44 active dentists; and there is one Ph.D. scientist in health research for every 9.5 physicians, versus one Ph.D. researcher for every 53 dentists.

Within our budgetary capabilities, NIDR is attempting to alleviate this disparity through support of training programs, such as those in speech pathology, as well as through research grants. Also, by drawing limited funds from our research grants program, we are initiating a new form of support intended to help newly trained young investigators remain active in research during the developmental stages of their careers. We recognize that these investigators represent a vital resource for maintaining an adequate science base in the field of oral health. It is hoped that this new special dental research awards program will encourage young faculty members in dental schools to engage in research and thereby also increase their students' orientation toward science.

And so we see, by brief example, the variety of forces of discontinuity that are shaping and creating our new roles as members of the health team for tomorrow's society. While the Dental Institute has been privileged to contribute to furthering the frontiers of dental and related sciences, we recognize and appreciate the vital role of such partner organizations as the American Speech and Hearing Association. Let me say, as I conclude, that the kind of leadership such organizations provide in directing that rarest of commodities—skilled and dedicated human resources—to the solution of our mutual problems is the most comforting of signs in meeting the exciting challenges that lie ahead.

## REFERENCES

Drucker, P. F., *The Age of Discontinuity, Guidelines to our Changing Society.* New York: Harper (1969).

It's time to operate. *Fortune,* 81, 79 (January 1970).

# FUNCTIONAL CRANIAL ANALYSIS AND THE FUNCTIONAL MATRIX

MELVIN L. MOSS

*Columbia University School of Dental and Oral Surgery, New York, New York*

A complete understanding of the biological processes of craniofacial growth is the most fundamental requirement for rational, correct, and successful therapeutic intervention in patients presenting developmental or acquired growth disturbances of this region. Beyond the intrinsic correctness of one viewpoint or another, as a topic for scholarly and scientific debate, there is the very practical problem that the approach of any clinician is strongly influenced by what he believes to be the effective cause of the particular growth disharmony he is treating. In large part, he attempts to relate his specific therapy to either eliminating or counterbalancing the presumed etiologically abnormal growth processes in a given patient.

The systematic approach to the treatment of growth disorders of the orofacial region began within the memory of living men. Whether we term this clinical specialty *orthodontics* or *orofacial orthopedics*, it is simply true that the usually accepted corpus of knowledge concerning the processes of normal and abnormal growth of this region is entirely derivative. Prior and contemporary investigations by anatomists and physical anthropologists continue to provide the profession with both a body of presumed facts about growth processes and a conceptual outlook which has had the unfortunate result of confusing the results of growth processes with their effective causation. Specifically, students of craniofacial growth generally have studied the growth of skeletal tissues, per se (cartilage as well as bone), and have mistakenly assumed that these same processes of skeletal tissue growth were their own primary cause. This has led to the more serious clinical error of directing our therapy toward the skeletal tissues themselves, rather than toward those other, nonskeletal, tissues and organs and functioning spaces, which are, in fact, the primary morphogenetic agencies for both normal and abnormal growth. Briefly, the so-called classical theories of craniofacial growth have now been demonstrated, repeatedly, to be unable to account for the totality of the observed phenomena, much to the embarrassment of their proponents.

It is simply a fact to state that most American orthodontic therapy remains "tooth-oriented." No matter to what extent a sense of technical sophistication can correctly be attributed to appliance therapies which are directed to tooth

movement, per se, in their totality they leave a great many problems beyond the scope of their clinical effectiveness. Several schools of European orthodontic thought, on the other hand, have created a number of empiric therapeutic techniques which are more functionally oriented, insofar as they are not primarily tooth-oriented, but rather deal with the several adjacent nondental regions as well. Without in any way attempting here an evaluation of clinical effectiveness of the cis- and transatlantic philosophies, it is a fact that, to the extent that dentofacial or orofacial orthopedics are directed primarily towards tissues other than teeth, their potential effectiveness is greater in those clinical conditions in which factors other than malposition or malrelation of teeth alone are involved. However, here, too, the comment must be made that the theoretical basis for either basic type of therapy has been equally incomplete.

For the past 16 years our laboratory has been engaged in a study of craniofacial growth processes. From this work has emerged the method of Functional Cranial Analysis, and its component hypothesis concerning the morphogenetic primacy of the Functional Matrix in cranial skeletal growth. In recent years, we have been somewhat surprised and gratified at the increasing acceptance our thoughts have had by many clinicians. However, we have been disturbed at the same time by the gratuitous use of our concepts to provide support for one or another specific therapy or appliance. Accordingly, we welcome this opportunity to present this summary of our work so that those interested may read it firsthand and no longer be troubled by derivative and frequently incomplete citations.

## FUNCTIONAL CRANIAL COMPONENTS

The method of Functional Cranial Analysis is based on an operational view of the head and neck. Rather than considering this region either systemically or topographically, as is usual in gross anatomy, we adopt a functional attitude. The head and neck are considered as a region of the body within which a number of functions are carried out. A partial list includes respiration, olfaction, speech, digestion, hearing, balance, vision, and neural integration. Each of these separate functions is carried out completely by what we call a *functional cranial component*—one component for olfaction, another for digestion, yet another for vision, and so on. The size and shape (that is, the form), as well as the position in space, of each functional cranial component is relatively independent of that of all other such components.

In turn, each separate cranial component has two distinct parts: a functional matrix, which actually carries out the particular function; and a skeletal unit, which provides biomechanical protection and support for its uniquely specific functional matrix. Let us consider these terms in detail.

A functional matrix consists of all of the tissues, organs, glands, and functioning spaces necessary to carry out a given single function completely. We do not use the phrase *soft tissues*, since, as we will demonstrate, the teeth individually and collectively are a functional matrix. Muscles, blood vessels, nerves, fat

pads, and salivary glands are all examples of one type of functional matrix. Similarly, we regard the oral, nasal, and pharyngeal functioning spaces as examples of a second type of functional matrix. Accordingly, we believe there are two distinct types of functional matrices: the periosteal and the capsular, respectively. The essential and important differences between these will be discussed after we consider the definition of the skeletal unit.

A skeletal unit is not identical with the word *bone*. It may be composed of osseous, cartilaginous, or tendonous tissues. When it is formed by bone tissue, we specifically acknowledge the important additional role of bone tissue in mineral homeostasis. However, the only other role of bone (to provide biomechanical protection and support) is carried out by bone at the next higher level of organization (as an organ). A skeletal unit is not identical with the named entities of classical osteology. As is true for all "bones," in the classical sense, the splanchno- and neurocranial "bones" are composed, in fact, of a number of contiguous microskeletal units. The mandible, for example, contains alveolar, coronoid, angular, condylar, and basal skeletal units, as well as several others.

It has been proven repeatedly, and without exception, by many workers that the size and shape of each of these mandibular processes (microskeletal units) is always and without exception a secondary response to morphogenetically prior and primary demands of specifically related functional matrices. The presence, number, and position of teeth directly and primarily determine the development and the growth in size and shape, as well as the very maintenance in being of alveolar bone. The clinical experiences following tooth extraction and congenital anodontia are well known. Similarly, the coronoid process is but an expression of the response of this particular skeletal unit to the dynamic demands of the temporalis muscle. Removal or denervation of this muscle results in the loss of the coronoid process, and this result is not caused by loss of vascularization, since blood flow through a denervated muscle increases. Homologous statements can be made for the angular process, where we deal with two muscles, the medial pterygoid and the masseter. Naturally, if absence or nonfunctional atrophy of such functional matrices leads to decreases in the size and shape of their skeletal units, increased demands (as in muscular hypertrophy) produce increases in the size and shape of the skeletal unit, as seen in the eversion of the angular process observed in masseteric hypertrophy. Taken as a whole, the several microskeletal units of the mandible form what we term a *macroskeletal unit*, whose significance will be discussed subsequently.

Next, we will consider periosteal and capsular functional matrices. Every cephalic function is carried out by a functional cranial component, that is, by a specific functional matrix which, in turn, is protected or supported by its own skeletal unit. Further anatomical consideration demonstrates that all skeletal units and one type of functional matrix (the periosteal matrices) actually arise ontogenetically, and exist, operate, and are maintained while completely embedded within a series of cephalic capsules.

The neurocranial capsule extends from the epidermis of the scalp externally

to the fibrous layer of the dura mater internally. Within this capsule lie the several microskeletal units of the calvarial bones, with their individual functional periosteal matrices. Since muscles furnish an excellent example of this type of functional matrix, we collectively term them *periosteal*. So, for example, portions of the outer plates of the temporal, parietal, and frontal bones are found embedded within the capsule, with the temporalis muscle. These together form a single functional cranial component. All of the examples given above regarding the mandible, and the various ramal and alveolar processes, are related to other periosteal matrices: muscles and teeth. The neurocranial capsule, as a whole, with all of its enclosed and embedded functional cranial components (microskeletal units and their periosteal matrices) has yet another function: to protect and to support the enclosed *neural mass*. This latter term includes the brain mass, the volume of the cerebrospinal fluid, and the leptomeninges as a unitary functional matrix; and, since it is protected by a capsule, we term it a *capsular matrix*. The orbital mass (globe, extrinsic and intrinsic orbital muscles, fat, neurovascular structures, and so on) forms a homologous capsular matrix, which is protected and supported by an orbital capsule.

In an entirely homologous manner the splanchnocranial bones arise, grow, and are maintained completely embedded within an orofacial capsule. Specifically, all the microskeletal units of the mandible (here, as a "macro" skeletal unit), the premaxilla, maxilla, palatine "bones," etc., with their specific periosteal matrices, lie within this unitary orofacial capsule, which is limited by epidermis externally and mucosa internally. The capsular matrices of the orofacial region differ from those of the neurocranial and orbital regions. The combined oral, nasal, and pharyngeal "cavities" is the capsular matrix. We prefer the term *functioning space* when discussing these matrices. These spaces are not volumes that are somehow left over after bones, muscles, epithelium, or glands have finished growing. These functioning spaces are brought into being in the embryonic period, by the process of enclosure, as the embryonic facial processes develop. Thereafter, these spaces are functionally maintained. The patency of these spaces is their operational reality, and their occlusion causes fatal results if continued more than temporarily. Admittedly, this viewpoint requires the clinician to adopt a new way of thinking about these functioning spaces, but, as we will show, these concepts are both correct and clinically useful.

Let us summarize now the principal differences between the two types of functional matrices. The proof of these assertions is given subsequently.

1. Periosteal matrices act directly on microskeletal units. They do so primarily by the processes of osseous deposition or resorption, or by effecting peri- or endochondral accretionary growth processes. They produce changes only in the size and shape (form) of their specific microskeletal units. We call such growth processes *transformations*.

2. Capsular matrices act indirectly on macroskeletal units, or more properly, on entire functional cranial components, which happen to be embedded completely within a specific capsule (neurocranial, orbital, orofacial). They do not

act by the processes of osseous deposition or resorption, nor by affecting cartilage directly. They do not alter the shape or size of the skeletal units, but they do change their location in space. Such growth processes are called *translations* (Figure 1) (Moss and Young, 1960; Moss, 1962).

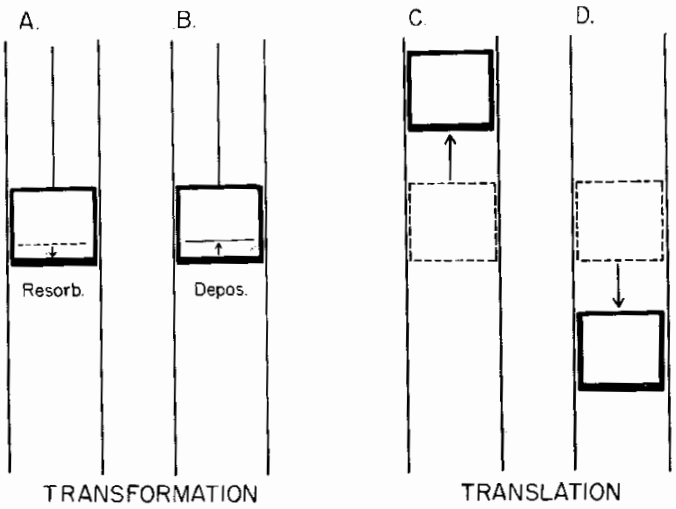



FIGURE 1. Diagrammatic presentation of differences between transformation and translation as processes involved in cranial bone growth. Transformation is brought about by osseous deposition and resorption and is a direct response to the primary morphogenetic demands of specifically related periosteal functional matrices. In this instance, picking up floor boards in an elevator car (representing a cranial capsule) (A) or laying them down (B) would move a passenger (representing a skeletal unit) in the car either down or up relative to the car. Should the car itself move within the shaft (the capsule), the passenger (skeletal unit) would be translated in space, passively and without the necessity of osseous deposition or resorption. Obviously, process A or B can occur in situations C or D. Accordingly, the expansion of cranial capsules in response to the volumetric alterations of enclosed capsular matrices can be distinguished from transformative changes which, in turn, may be either complementary or antagonistic to the direction of such translations. (From Moss and Salentijn, 1969b.)

## CRANIAL GROWTH PROCESSES

The foregoing discussion, essentially theoretical, acquires meaning and clinical significance when we consider problems of cranial growth. It will be demonstrated now that the classical theories of cranial growth cannot possibly provide satisfactory explanations for early observed phenomena, while the application of the method of Functional Cranial Analysis resolves these problems easily.

First, it is of the utmost importance to define the word *growth*. The most

usual definition which has applicability for students of orofacial growth has a dimensional connotation, that is, it is a linear measure. These measurements, again, usually are recorded as indicating directly the changes in size between any two points, and as indirect measurements of changes in shape, when angular changes are included. In other words, the most usual meaning of growth, as indicated by what is measured, is a change in form. It is true, in sensu stricto, that growth can be defined broadly as any change in any parameter that is itself capable of measurement. Nevertheless, in dealing with cephalic growth our usual interests are somewhat more restricted, but yet more encompassing than only changes in form. Specifically, we have an additional need to know about growth changes in the spatial location of the craniofacial "bones."

Rephrasing these statements in our own terminology, it is obvious that most students of cranial growth measure only the results of transformation. In addition, we require information concerning the translatory growth changes.

It is our basic contention that functional matrices are the primary morphogenetic agencies in cranial growth. All skeletal tissue growth is a secondary, compensatory, and mechanically obligatory response to changes in the temporally prior demands of specific functional matrices. Similarly, the continuation in being of skeletal units requires that these matrix demands be maintained. *In a very real sense, functional matrices grow, and skeletal tissues respond.* This statement is strongly supported by considering the fact that it is now quite certain that, with the exception of only a very few and very rare diseases, there is no direct genetic control of skeletal tissue growth, either of cartilage or of bone. Indeed, the best available data strongly indicate that the direct effect of genes is exerted on surrounding tissues (functional matrices) and only through these nonskeletal tissues, and hence, indirectly, to the skeletal tissues themselves (Grüneberg, 1963). The vast body of data on in vivo and in vitro experimentation with skeletal tissues and organs emphasizes this point with considerable force. Skeletal tissues have but one biomechanical role in any vertebrate body: to act biomechanically to protect or to support related tissues, organs, and functioning spaces. The additional role of bone in mineral homeostasis is neither denied nor compromised by this statement, since biomechanical demands are accommodated at the organ level and homeostatic demands at the tissue level of organization.

### *Classical Theories of Cranial Growth Processes*

There are three processes which many earlier workers held to provide a totally self-sufficient explanation of craniofacial growth—although different workers placed varying degrees of emphasis on specific processes. They were (1) interstitial expansive forces generated within soft sutural tissues, (2) interstitial expansive forces generated by cartilaginous tissues; and (3) deposition and resorption of bone.

The first process, intrinsic forces of sutural expansion, was widely proclaimed (see Moss, 1954, for a review). This concept, derived from the Viennese School of Erdheim, were widely disseminated to the English-speaking world by

Weinmann and Sicher (1947). This hypothesis was as simplistically attractive as it was totally incorrect. Once we dismiss appeals to either the Scholastic Method or the authority of the professorial chair and turn to appropriate experiments with growing laboratory animals, we consistently observe undisturbed and normal cephalic growth in the face of sutural extirpation. This is true for the neurocranium, as well as for the splanchnocranium. These findings, published by us in 1954 (Moss, 1954), dealing with the calvarial sutures, have been amply verified by many other workers, dealing with all accessible cephalic sutures (Sarnat, 1963; Selman and Sarnat, 1957; Watanabe, Laskin, and Brodie, 1957). Furthermore, negative data do not exist. Undoubtedly, by many techniques it is possible to demonstrate and measure accurately the deposition of bone at sutural edges during growth. But this only indicates that the sutures are the loci of secondary and compensatory growth, and it does not indicate that these same sutural areas are primary growth centers.

The second process, which implies that the several cephalic cartilaginous areas are capable of exerting expansive forces, suggests that both perichondrial apposition and, more importantly, interstitial expansion of cartilage tissue are causally involved as primary growth centers. Again, as attractive as this concept is, it cannot be sustained in the face of appropriate experimental (and clinical) data.

Three principal cephalic cartilaginous areas must be considered: the mandibular condyle, the nasal septum, and the spheno-occipital synchondrosis. Without question, one of the most deep-seated beliefs in dentistry is that the condylar cartilages are homologous in structure and function with the epiphyseal growth plates of the long bones. However, the facts are otherwise. First, condylar cartilages are similar to articular cartilages in structure (Rönning and Koski, 1967; Durkin, Irving, and Healey, 1969a, b). Second, bilateral removal of condylar cartilages in a young, growing animal does not cause any disturbance of orofacial growth (Gianelly and Moorrees, 1965; Irving and Durkin, 1965; Pimenidis, 1969). Further, when such techniques are applied in appropriate human cases (usually for uni- or bilateral functional ankylosis of the temporomandibular joint), we observe identical results. (We will return to this point later.) Accordingly, all our data convince us that the condylar cartilages are the loci of secondary and compensatory growth and not primary growth sites (Moss, 1960; Moss and Rankow, 1968).

Identical statements can be made with regard to the nasal septal cartilage. While some workers still consider that this area is a primary growth center, whose expansion is responsible for the vertical (and anteroposterior?) growth of the middle face, the data suggest otherwise.

Removal of the entire nasal septal cartilage in a growing animal is not followed by any diminution of midfacial growth (Moss and Bromberg, 1967). The vertical, anteroposterior, and lateral growth of the midface is quite normal. There is a postoperative collapse of the roof (the bridge) of the nose, but this is due to the loss of the strut-like capacity of the septum in the biomechanics of nasal construction (compare Badoux, 1966). Our earlier work on this problem

has recently been confirmed by Stenstrom and Thilander (1970). In reviewing the literature on this problem, it is of crucial importance to inquire into the possibility of postoperative scarring following septal resection. The work of several others, whose data seem to indicate disagreement with our statements concerning the secondary and compensatory role of the septal cartilage, can be brought into harmony when the presence of extensive scar-tissue formation is made clear. As will be shown, such scar tissue will produce an effective "ankylosis," or better, a mechanical blockade which will, quite literally, prevent the passive translation of the midfacial skeleton during growth. When such "ankylosis" is present after septal removal inevitably the result is a malformation of the midface. Indeed, it would be impossible for such deformation not to be produced in this way.

Recently, homologous statements have been proposed for the spheno-occipital synchondrosis (Koski, 1968; Koski and Rönning, 1969). This region is notoriously difficult to approach experimentally in vivo. In the few successful attempts to do so, postoperative scarring has been inevitably produced, and once again the resulting deformations were interpreted incorrectly as demonstrating that this cartilage was a primary growth center rather than a secondary locus of compensatory growth (DuBrul and Laskin, 1961).

A few sentences about the results of the in vitro work with cranial cartilages will be appropriate here, since misunderstanding of these data has led some workers to suggest inappropriate extensions of their conclusions. When any of these three cartilages are explanted and cultured in vitro, they do grow. That is, they undergo a linear expansion! However, several points should be carefully noted. First, such growth occurs primarily in the first week of culture. That is, almost any cartilaginous tissue when grown in vitro will demonstrate linear increase. However, this occurs for a very limited period of time and, despite the fact that external environmental conditions remain satisfactory, further linear growth cannot be observed. More important is the fact that careful observation of the site of this elongation shows that it usually occurs in areas of the transplanted cartilage which are not identical to the areas where such linear growth occurs in vivo. These observations can be repeated in the in vitro study of both mandibular condylar and spheno-occipital cartilages (Mareel, 1969).

All of the voluminous data on the tissue and organ culture experiments of cartilaginous anlagen of endochondral bones demonstrate similar effects. With increased time, the form of the skeletal organ becomes less discrete and, by the time membranous ossification begins, severe deformation is present. Cartilage, as a tissue, but not as an organ, can indeed continue to grow, but in an increasingly formless fashion.

The third of the classical cranial growth processes mentioned above is deposition and resorption of bone tissue (especially of intramembranous bone), and has long been favored by many workers. Dating from the inception of modern investigations of bone growth in the early 18th century by Du Hammel, Belchier, and Hunter, and continuing in our own era with the excellent work of

Brash, this process has received its most recent statement in the detailed work of Enlow (1968). Ignoring many technical problems of interest only to the specialist, this process may be easily described in the following way. Let us consider the results of deposition and resorption of any bone in a given direction—whether it be the outward growth of parietal bone, or the downward growth of the hard palate. At its simplest, this can be imagined to occur as the result of resorption of the endocranial surface of the calvaria or of the nasal surface of the palate, and of deposition on the ectocranial calvarial surface or on the oral palatal surface. This oversimplistic statement, of course, does not consider the more complex biological realities of the processes occurring on the diploic surfaces of both calvarial plates, nor in similar spaces in the palate. However, it does make clear the primary claim of those who view this skeletal growth process as a major contributor to cephalic growth—specifically, that the dimensional enlargements of both neuro- and splanchnocranium are accomplished by the activity of these two reciprocally interacting processes. In other words, such workers specifically consider that cranial growth is an active process, and one which primarily acts through the agency of the skeletal tissues themselves.

As attractive as this concept is, it can be demonstrated easily that such direct skeletal transformative processes cannot possibly account for either the totality or even the majority of the observed cephalic growth and, even more damaging, that there are critical examples of skeletal growth in which the direction of transformative growth due to osseous deposition and resorption is directly opposite to the observed direction of the movement of the bone itself. For example, the work of Cleall, Wilson, and Garnett (1968) has provided us with excellent quantitative data on the cranial growth of the rat, utilizing well-spaced injections of dyes capable of producing well-defined lines of osseous calcification. When we compare the amount of such osseous deposition and resorption with the total dimensional changes observed, we find that the transformative growth processes account for between 25% and 33% only.

Other data of Cleall, Wilson, and Garnett (1968) are equally informative. This same multiple dye injection technique easily showed that the postnatal growth of the rat supraoccipital bone involved deposition on the endocranial surface and resorption on the ectocranial surface. However, this bone, in fact, actually moves posteriorly (Figure 2). Obviously, active transformation occurs in a direction which is directly opposite the direction of passive translation and, furthermore, translative growth is the major factor in rodent cephalic growth.

One final comment. No combination of the three classical processes of cephalic growth processes yields a result which is any more satisfactory than any of them singly. It is somewhat sad to reflect on the years of sterile debate between the conflicting schools of craniological theory that were so noticeable in recent decades, when proponents of the primacy of sutural expansion opposed those who held similar claims for surface deposition and resorption.

If this criticism of the classical growth theories is correct, we must now

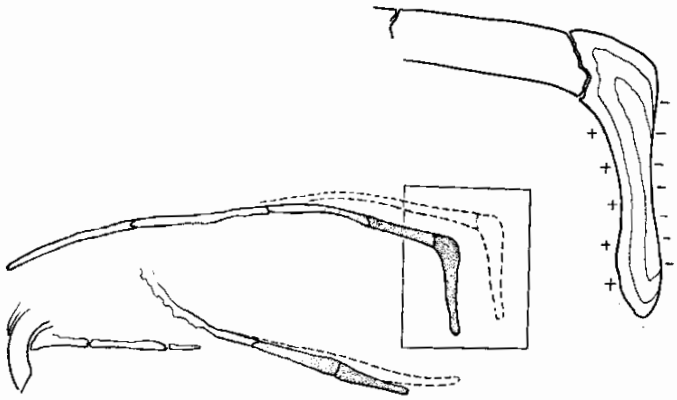


FIGURE 2. These data illustrate that the directions of active, transformative, and passive translative growth can be opposite in sign. In this case the supra-occipital bone of the rat, before 60 days of age, moves posteriorly and upward, in an absolute sense. During this same period deposition occurs endocranially and resorption ectocranially. Hence the algebraic sum of both types of growth is dominated by passive translative growth. (Data derived from Cleall, Wilson, and Garnett [1968] and Vilmann [1968].)

consider what additional cephalic growth processes must be involved. In essence, we have said that all of the classical theories reflect only the active, transformative responses of skeletal units to periosteal matrices. It is to the passive, translative growth processes we must turn now.

## TRANSLATIVE GROWTH

The primary morphogenetic event in this growth process is the volumetric enlargement of the capsular matrices. This, in turn, causes a secondary areal enlargement of the surrounding capsule. All enclosed and embedded skeletal units and periosteal matrices are thus passively translated in space. Without question, transformative growth changes occur at the same time, since the altered conditions of the translating periosteal matrices inevitably bring about such secondary changes in their related skeletal units. But these latter changes must not be confused with the former.

Figure 3 shows the expansion of the neurocranial capsule, as a secondary and compensatory response to the volumetric enlargement of the enclosed neural mass. This dimensional movement will (and does) occur even if little or no transformation occurs, as in extreme hydrocephaly. Normally, such passive translation would tend to cause separation of calvarial bones, which is compensated for by the secondary transformative growth at the sutural margins. Additionally, the calvarial bones may thicken, develop a diploic space, and alter their arcs of curvature—all transformative growth processes.

This is but another way of saying that a microcephalic skull has a small

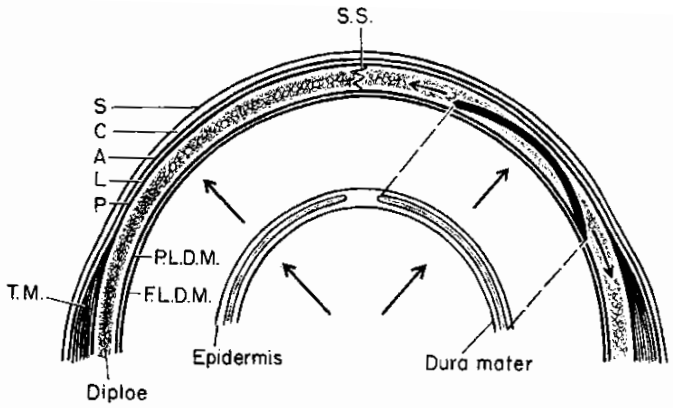



FIGURE 3. The expansion of the neurocranial capsule in response to the morphogenetically primary expansion of the enclosed neural mass. As this capsule expands, the enclosed and embedded calvarial skeletal tissues are passively translated in space in response to the growth of this capsular matrix. At the same time, these same skeletal units respond to the altered demands of their functional periosteal matrices. They do so by expanding in area and in thickness, by altering from a simple system of open, radiating trabeculae to one with inner and outer tables of compact bone with an intervening diploic area. If simple translation occurred alone, the figure on the right would result. It is explicitly stated that the amount and regions of periosteal growth are diagrammatic only. The simultaneous delamination of the neurocranial capsule is also seen. From a simple dura mater/epidermis in the early fetus, we see the full expansion of such changes in the adult. S, skin; C, dense connective tissue layer; A, aponeurotic layer; L, loose connective tissue layer; P, periosteum. (All five of these layers form the scalp.) T.M., temporalis muscle; P.L.D.M., periosteal layer of dura mater; F.L.D.M., fibrous layer and dura mater; S.S., sagittal suture. (From Moss and Salentijn, 1969a.)

neurocranium because the neural mass is abnormally small, while a macrocephaly is the result of an abnormally large neural mass. No amount of periosteal matrix activity (transformation) can account for what is so evidently the result of a primary alteration in the cephalic matrix. What is so abundantly clear in the neural skull is equally true for the facial skull.

The orofacial capsular matrices are the volumes of the several cavities of this region: the oral, nasal, and pharyngeal functioning spaces. The biological bases for considering these functioning spaces as primarily competent morphogenetic agencies has been discussed at length elsewhere, but a brief review of this point is necessary. The oral cavity is not a space somehow haphazardly left over after the growth of the facial bones, muscles, glands, vessels, nerves, mucosa, and skin is completed. In fact, its growth is the primary biological reality, about whose functionally necessary volume (in terms of respiratory needs) all of these other tissues and organs adapt themselves. If any dimension of the body has significance and biological meaning, it is the total volume of the respiratory system. Here, as in the digestive and urogenital systems, it is

the patency or hollowness which is the functionally significant fact. All of these spaces are maintained by function, and should this function cease, obliteration of the lumen will occur.

The oral functioning space comes into being by the growth and fusion of the embryonic facial process, a process we call *enclosure* (Moss and Salentijn, 1969a, b). Once formed, the space is maintained by function. The growth in volume of the oral space most probably is a genetically controlled event, acting primarily on the epithelial tissues, and most likely under afferent neurotrophic control, a point that will be discussed in detail in later publications. The important fact is that the volumetric enlargement of the oral space causes the orofacial capsule to expand secondarily. All enclosed skeletal units and periosteal matrices are passively translated within this same capsule.

Let us consider the mandible. Passive translation of the orofacial capsule tends to carry the mandible (as a macroskeletal unit) down and forward. This same passive movement also tends to distract the head of the condylar process away from the superior surfaces of the temporomandibular joint. The observed increments of growth at the condylar head are not the cause of this mandibular movement but, in reality, secondary and compensatory growth increments which serve to keep the temporomandibular joint in being and in function.

The results of condylectomy, in animals and in man, previously referred to fully substantiate this view. Undoubtedly, active transformative growth occurs. This is certainly the manner in which the ramal processes grow. It is possible to demonstrate the amounts of total mandibular growth due to active transformation and passive translation independently. The former account for between 25% and 33% of the total growth observed.

The methods used by us to demonstrate the morphogenetic role of capsular matrices in orofacial growth have been published elsewhere extensively (Moss, in press; Moss and Salentijn, 1969a, b). Some pictorial data are shown in Figure 4.

What they demonstrate, in essence, is that the majority of the downward and forward growth of the mandible is passive translation. Obviously, the ramal changes are the result of the active transformative growth processes, which, in turn, are secondary and compensatory responses to their periosteal matrices which are themselves being passively translated.

It is clear now that cephalic growth cannot be explained fully by the solitary activity of either periosteal or capsular matrices. Rather it is true that when we combine the action of both matrix types, a comprehensive answer is possible. Finally, it is also clear that any proper consideration of cephalic growth phenomena must include changes in spatial position in addition to changes in size and shape, since each type is produced by a different sort of matrix and by different growth processes.

## SUMMARY

The basic principles of the method of functional cranial analysis are described. Emphasis is placed on the concept that functional matrices are the

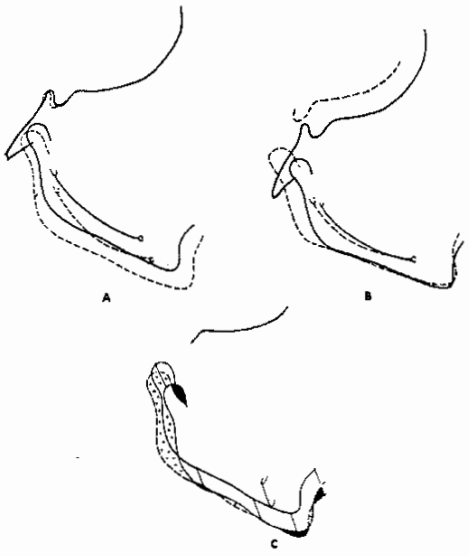



FIGURE 4. Interosseous growth of the mandible is shown by registration of two tracings taken from a longitudinal growth study. The mental and mandibular foramina, as well as the course of the interior alveolar canal, are shown. The totality of the growth changes shown here is the sum of the skeletal unit responses to both periosteal and capsular matrices. B: Intraosseous growth of the same mandible is shown when the two tracings are registered at the mental foramina when the anterior cranial base tracings are kept parallel. C: When the tracings of A and B are superimposed, this figure is produced. The two mandibular outlines with solid lines are the two positions of the earlier tracing; the outline with a broken line is the oldest tracing. It is now seen that most of the growth changes in a downward and forward direction are due to passive translation in response to the expansion of the orofacial functioning spaces. Periosteal appositions (plus signs) and resorption (in black) account for relatively minor changes in the anterior and lower borders, while being totally responsible for the backward and upward motion of the condylar and angular processes. It is seen that B, the intraosseous drawing, in fact showed all of the periosteal growth changes. It is stressed that the upward periosteal growth at the condylar process is a compensation for the downward and forward translation of the mandible, and not its cause. (From Moss and Salentijn, 1969a.)

primary morphogenetic agencies in cephalic growth. Two types of functional matrices are described: the periosteal and the capsular. The first acts directly upon skeletal tissues, by the processes of deposition and resorption of the surfaces of skeletal tissues, by deposition of bone at sutural areas, as well as by endochondral growth. These changes alter the size and shape of the skeletal tissues and are collectively termed *transformations*. The second type of functional matrix (the capsular) acts indirectly on skeletal tissues to alter only their spatial position. It does not utilize the three classical methods of skeletal tissue growth, but acts by virtue of the fact that these skeletal tissues exist within capsules which expand as the volume of the capsular matrices increases. Numerous examples are given, with some emphasis on mandibular growth.

ACKNOWLEDGMENT

This report was supported in part by grant NB 00965 from the National Institutes of Health.

## REFERENCES


BADOUX, D. M., Framed structures in the mammalian skull. *Acta morph. Neerl.-Scand.*, **6**, 239-250 (1966).

CLEALL, J. F., WILSON, G. W., and GARNETT, D. S., Normal craniofacial skeletal growth of the rat. *Amer. J. phys. Anthrop.*, **29**, 225-242 (1968).

DUBRUL, E. L., and LASKIN, D. M., Preadaptive potentialities of the mammalian skull: An experiment in growth and form. *Amer. J. Anat.*, **109**, 117-132 (1961).

DURKIN, J. F., IRVING, J. T., and HEALEY, J. D., A comparison of the circulatory and calcification patterns in the mandibular condyle in the guinea pig with those found in the tibial epiphyseal and articular cartilages. *Arch. oral Biol.*, **14**, 1365-1371 (1969a).

DURKIN, J. F., IRVING, J. T., and HEALEY, J. D., A comparison of circulatory and calcification changes induced in the mandibular condyle, tibial epiphyseal and articular cartilages of the guinea pig by the onset and healing of scurvy. *Arch. oral Biol.*, **14**, 1373-1382 (1969b).

ENLOW, D. H., *The Human Face*. New York: Harper (1968).

GIANELLY, A. A., and MOORREES, C. F. A., Condylectomy in the rat. *Arch. oral Biol.*, **10**, 101-106 (1965).

GRÜNEBERG, H., *The Pathology of Development*. New York: Wiley (1963).

IRVING, J. T., and DURKIN, J. F., A comparison of the changes in the mandibular condyle with those in the upper tibial epiphysis during the onset and healing of scurvy. *Arch. oral Biol.*, **10**, 179-185 (1965).

KOSKI, K., Cranial growth centers: Facts or fallacies. *Amer. J. Orthodont.*, **54**, 566-583 (1968).

KOSKI, K., and RÖNNING, O., Growth potential of subcutaneously transplanted cranial base synchondroses of the rat. *Acta. odont. Scand.*, 27, 343-357 (1969).

MAREEL, M., In vitro studies of the embryonic growth of tibia in chicken. *C. R. Sci. Soc. Biol.*, **163**, 221-224 (1969).

Moss, M. L., Growth of the calvaria in the rat. *Amer. J. Anat.*, **94**, 333-362 (1954).

Moss, M. L., A functional analysis of human mandibular growth. *J. pros. Dent.*, **10**, 1149-1160 (1960).

Moss, M. L., The functional matrix. In B. Kraus and R. Reidel (Eds.), *Vistas in Orthodontics*. Philadelphia: Lea and Febiger, pp. 85-98 (1962).

Moss, M. L., Differential roles of periosteal and capsular matrices in orofacial growth. *Trans. Eur. orthodont. Soc.* (in press).

Moss, M. L., and BROMBERG, B., The passive role of nasal septal cartilage in mid-facial growth. *Plast. reconstr. Surg.*, **41**, 536-542 (1967).

Moss, M. L., and RANKOW, R., The role of the functional matrix in mandibular growth. *Angle Orthodont.*, **38**, 95-103 (1968).

Moss, M. L., and SALENTIJN, L., The primary role of functional matrices in facial growth. *Amer. J. Orthodont.*, **55**, 566-577 (1969a).

Moss, M. L., and SALENTIJN, L., The capsular matrix. *Amer. J. Orthodont.*, **56**, 474-490 (1969b).

Moss, M. L., and YOUNG, R., A functional approach to craniology. *Amer. J. phys. Anthrop.*, **18**, 281-292 (1960).

PIMENIDIS, M. Z., The effects of early post-natal condylectomy on the growth of the mandible in rats. Master's thesis, Boston Univ. (1969).

RÖNNING, O., and KOSKI, K., Observations on the histology and histochemistry of growth cartilages in young rats. *Dent. Practit.*, **17**, 488-450 (1967).

SARNAT, B. G., Post-natal growth of the upper face: Some experimental considerations. *Angle Orthodont.*, **33**, 139-161 (1963).

SELMAN, A. J., and SARNAT, B. G., Growth of the rabbit snout after extirpation of the frontonasal suture. *Amer. J. Anat.*, **101**, 273-293 (1957).

STENSTROM, S. J., and THILANDER, B. L., Effects of nasal septal cartilage resections on young guinea pigs. *Plast. reconstr. Surg.*, **45**, 160-170 (1970).

VILMANN, H., The growth of the parietal bone in the albino rat studied by reontgenocephalometry and vital staining. *Arch. oral. Biol.*, 13, 887-901 (1968).

WATANABE, M., LASKIN, D. M., and BRODIE, A. G., The effect of autotransplantation on growth of the zygomatico-maxillary suture. *Amer. J. Anat.*, **100**, 319-329 (1957).

WEINMANN, J., and SICHER, H., *Bone and Bones*. St. Louis: Mosby (1947).

# HUMAN PRENATAL ACTIVITY SEQUENCES IN THE FACIAL REGION AND THEIR RELATIONSHIP TO POSTNATAL DEVELOPMENT

TRYPHENA HUMPHREY

*University of Alabama in Birmingham, Birmingham, Alabama*

I plan to discuss the development of prenatal activity including the fact that the sequences in which action develops prenatally are repeated perinatally or postnatally, and some of them even earlier. In addition, I want to describe some characteristics of central nervous system development which make such repetitions of activity sequences more or less inevitable.

The material presented here is not based on my own work exclusively, but on the motion picture records of Dr. Davenport Hooker, former Chairman of the Department of Anatomy at the University of Pittsburgh. The program was begun at Pittsburgh in 1932, and continued there from that time until the spring of 1963 when I left that university. I joined the program at Pittsburgh in 1938 and at present I am carrying on the analysis of Hooker's motion picture films which I inherited after his death in 1965. During my 18 years (1938-1956) of association with Hooker, I was usually on hand when the motion pictures were made, helping with the observations and offering suggestions on what we might try. There are many things yet that could be done.

The motion pictures were taken with a 35-mm World War I surplus camera, which Hooker himself bought, and rigged up to run with a foot switch, so that he could use his hands to move or to stimulate the fetus.

At laparotomies done for the therapeutic termination of pregnancy, we were set up next to the operating room with the camera focused on a doll of the expected size. Because histories from pregnant women are often inaccurate concerning the onset of the last menstrual period, the estimated fetal age was not always right. So the expected size of the fetus—even when the age was checked by the obstetrician from his examination—might still be wrong. In that case, although the camera was adjusted for the focus, there was not time for other changes because the small fetuses react to stimuli for only eight to 12 minutes—although larger ones respond for 20 minutes, or even more when they approach the age of viability. If a small fetus was expected and a larger one was delivered, the field covered by the camera might not be large enough for the fetus.

The fetuses were placed in an isotonic bath, at normal body temperature. Most of the motion pictures were made of responses to stimulation, but some activity was recorded for which there was no known stimulation. Fetal behaviorists usually refer to such actions as *spontaneous activity*. As used by the Hamburger school (Hamburger, 1963, p. 350), however, the term refers primarily to the endogenous, automatic build-up of neuronal discharges. The term *spontaneous activity*, as used by others, refers to action due to any unknown stimulation, whatever it might be.

As far as we know, the first action of a human fetus is a reflex, not any type of spontaneous activity. The first reflex is in response to touching the face of the fetus, an exteroceptive stimulus of tactile nature, rather than a stretch stimulus as Windle (1944, 1950) has proposed in his papers.

The stimulus from touching the fetus around the mouth (upper and lower lips or perioral area) is carried by the descending fibers of the fifth cranial (trigeminal) nerve down to the upper cervical segments of the cord, then over an interneuron to the motor neurons and out by the motor fibers to the region of the neck muscles. The reflex consists of bending the head to the side opposite the stimulation (Figure 1). At the time that the reflex first occurs, it involves all of the neuromuscular mechanisms that are sufficiently developed to react (for diagrams, see Figure 4 in Humphrey, 1952; Figure 1 in Humphrey, 1953; or Figure 6 in Hooker, 1952). In the week's time between the first reaction of bending the head to the opposite side, and acquisition of the reflex shown in Figures 1 and 2A, there has been enough development so that the head, the trunk, and the rump all bend to the contralateral (or opposite) side, and the rump rotates to give a little hula wiggle, obviously a very primitive reaction.

In his work on amphibians, Coghill (1929) termed this contralateral flexion reaction a *total pattern reflex* since it involves all of the neuromuscular mechanisms capable of reacting. It is not a reaction to stretch, which is always a localized reflex over a two-neuron arc that functions in response to stretching a muscle, which then contracts, but a response to cutaneous stimulation over a three-neuron reflex arc. Probably the stretch reflex known best to most of us is the one that occurs in the dentist's chair, when he finds it necessary to say, "Open a little wider, please." It is a stretch reflex that closes one's mouth in spite of all efforts to keep it open.

The reaction to touch that turns the face of the fetus (that is, the perioral region) away from the stimulus has been known as an avoiding reaction. Coghill called it that in 1929, in connection with his work on Amblystoma, and a number of other workers on fetal activity have referred to it as an avoiding reaction. It is a negative response, whereas turning toward a stimulus is a positive reaction. The negative reflexes keep free-swimming animals like tadpoles from being eaten up when touched by predators, as would happen if they turned toward, instead of away from, a harmful stimulus; and so they preserve the life of the tadpole. For the human fetus, there is no comparable function of avoiding harm, but perhaps without any movement the fetus might adhere to the amnion. Nevertheless, this type of reaction is retained in human develop-

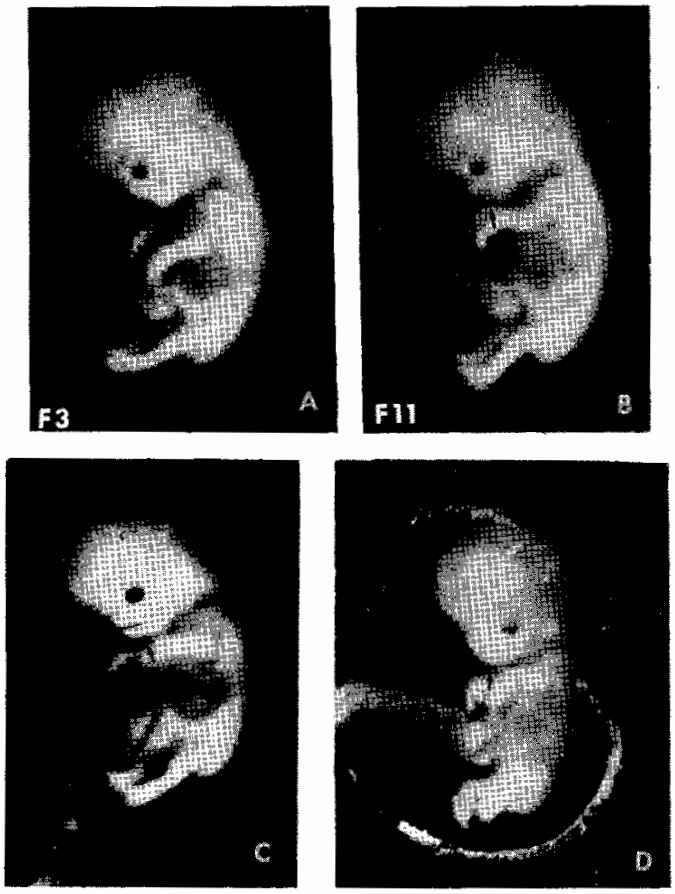



FIGURE 1. Photographs of 3 different fetuses of 8½ weeks of menstrual age, showing part of a contralateral flexion reflex for each one. Note that in each reflex the action includes mouth opening by jaw depression as well as contralateral flexion of the head, trunk, pelvis, and upper and lower extremity movements.

1A and B, third (F3) and eleventh (F11) frames of a reflex of a 27.1-mm fetus; 1C, 26.0-mm fetus 1D, 27.7-mm fetus within the amniotic sac. The photographs vary from approximately 1.5 to 1.7 times the actual size of the fetuses. The arrows point to the open mouth. The dark line over the nose in 1C and at the corner of the mouth in 1A is the stimulator.

ment and insofar as we know now, it is the only type of reflex present for about a week before the opposite response, turning toward the stimulus, makes its appearance.

The motion pictures were usually taken at 16 frames per second, and in illustrating the entire reflex, the frame immediately before the movement has been numbered Frame 1 (F1) and the others in order until the return to the original position is completed. Thus the approximate time required for the reflex can be determined. In Figure 1A and B, therefore, F3 and F11 indicate the second and tenth frames of movement in such a reflex of a 27.1-mm fetus. A comparable stage of a similar reflex of a 27.7-mm fetus in the amniotic sac is shown in Figure 1D.

Menstrual age, which is approximately two weeks more than fertilization age, is used throughout this discussion. When a little fetus 26 mm long (8½ weeks of menstrual age, Figure 1C) is stroked around the mouth with a stimulator, the head and trunk bend toward the side opposite the stimulation (con-

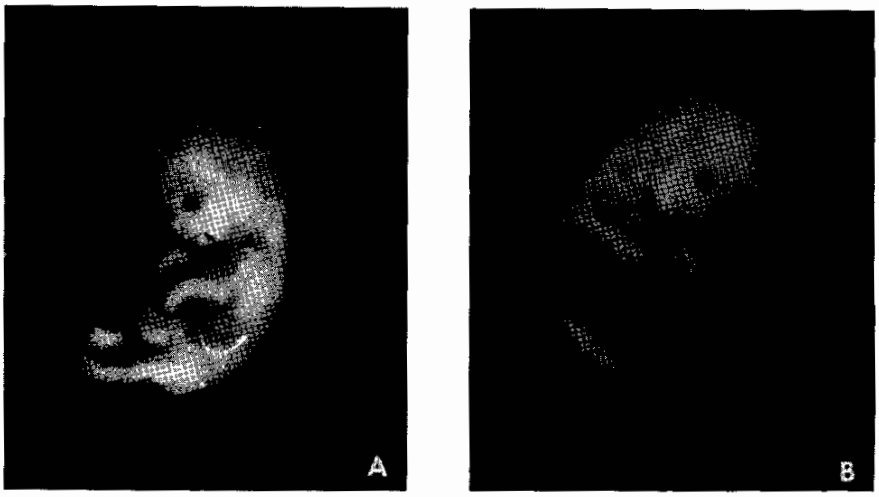



FIGURE 2. Photographs show the peak of action in a contralateral flexion reflex at 8½ weeks (A, 27.1 mm) and 9½ weeks (B, 34.3 mm, crown-rump length). In the fetus of 8½ weeks (A), the lips are just beginning to separate at the corner (at arrow), but in the one a week older (B) the jaw is depressed enough to separate the lips completely at the corners also. The fetus in A is shown at one and one-half times its normal size and that in B is enlarged slightly less.

tralateral head flexion) and then come back to the original position. This 26-mm fetus reacted 16 times with such contralateral reflexes but only two times with an ipsilateral reflex, a response consisting of bending toward the side stimulated. One of the interesting things about the ipsilateral reflex is that, whereas an avoiding type of reflex of this fetus may take as little as half a second (Humphrey, 1968, Figure 2), a positive reflex of turning toward the stimulus required about two and a half seconds.

The difference in time between the two types of reflexes appears to be present all through early stages of fetal activity. The ipsilateral responses, or turning toward the stimulus—that is, the positive reactions—are slower than the contralateral avoiding reactions, or potentially protective reflexes. Phylogenetically the developing animal needs to get out of an enemy's way in a hurry, but can take more time for the positive reactions that later form the basis for eating.

In earlier publications (Humphrey, 1968, Table 1, and 1969a), percentages of the avoiding reaction or turning away were compared to the positive reflexes for the age period 8 to 8½ weeks. The 94 reflexes recorded in the motion pictures include 87 of the avoiding type, or over 92% of them. So the avoiding reflexes not only begin earlier and are more rapid, but are far more numerous during this period and through at least the first 10 weeks.

In the beginning, I said that sometimes, even prenatally, there may be repetition of the motor sequences of events. Here I am referring to the negative and positive reactions, moving away from a stimulus or moving toward it. As

early as 9½ weeks, head extension has been noted after perioral stimulation, and at 11 weeks of menstrual age a perioral stimulus elicits an extension response quite consistently. (In Figure 3A and B, the perioral stimulus was a clamp on the umbilical cord that touched the mouth area. The head almost hyperextended and then returned to position.) This action also brings the area stimulated away from the stimulus.

At 10 weeks (Humphrey, 1964, Table IV) touching the lips rarely results in forward head-bending, or ventral flexion of the head, and this action is uncommon until after 11 weeks. It is illustrated later at 14 weeks as part of a swallowing reflex. The movement brings the lips toward the stimulator and so is a positive response. Therefore, in head extension and in ventral flexion of the head, as in contralateral and ipsilateral bending (or flexion) of the head, the avoiding or negative reflex precedes the positive one in time of appearance. It has also been seen more frequently.

At 11½ weeks of menstrual age, there is another change. Earlier in development the head bends to one side, a difficult thing for an adult to do instead of rotating the head. At this age the face begins to rotate to one side on stimulation of the oral area. Figure 3D shows a combination of rotation of the face to the side opposite the stimulation and extension of the head. This action again brings the area touched away from the stimulus. By 13 weeks of menstrual age, stimulating the same area of the face may cause the face to rotate toward the stimulator (Humphrey, 1968, Figure 12), although there may be a little head extension also.

The extension-flexion and rotation types of activity are repeated in the perinatal and postnatal period. Premature infants more often turn the face away when the mouth is touched, but not always, of course (Figure 6A and B in Humphrey, 1969b). An illustration from Prechtl and Beintema (Figure 6C and D in Humphrey, 1969b) shows one of the four cardinal points (corners of the mouth and upper and lower lips), the stimulation of which in most newborn infants causes them to turn toward the stimulating nipple or finger, which then slides into the mouth and sucking begins. Also from the standpoint of an avoiding (or negative) and a moving toward (or positive) reaction, there are two repetitions in fetal life. Thus, although the actions of extension and ventral flexion of the head and of contralateral and ipsilateral rotation of the face differ from contralateral and ipsilateral head bending, the sequence of first the negative, then the positive reaction is clearly repeated twice.

Now we shall return to the early contralateral flexion reflexes, to take up another part of the activity. In the 26-mm fetus with the stimulator lying across the nose (Figure 1C), there is a small, dark, right-angled triangle below the nose, just between the hands. This is the partially open mouth. For this fetus there were nine motion picture recordings of mouth opening. The mouth of the 27.1-mm fetus in Figure 1A and B also is partly open, but the open mouth of the 27.7-mm fetus within the amniotic sac (Figure 1D) shows less well.

It is hard to demonstrate small movements with photographic prints, but in

watching the motion picture films, in addition to the contralateral bending of the head and trunk, the rump rotation, and the mouth opening, one sees spreading of the fingers and of the toes (Figures 1D and 2). There is also extension of the arms at the shoulders, as seen on comparing Figure 1A with 1B, and sometimes flexion at the elbow and at the wrist as well.

At the peak of another reflex of the 27.1-mm fetus, the lips are beginning to separate all the way to the corner (at arrow, Figure 2A). A week later (at 9½ weeks of menstrual age) as part of the same sort of contralateral flexion reflex to stimulation of the perioral area, the open mouth forms an acute angle (Figure 2B) instead of a right angle. This means that the mouth is open farther toward the corner, probably all the way.

By chance, and because I knew a little embryology, I noted that palatal closure takes place at or just after the time that these mouth-opening reflexes develop. An adult needs only to try to lower his own jaw with his tongue up in the roof of his mouth to feel the pull exerted on it by lowering the jaw. Years ago Wilhelm His (1901), the famous Swiss embryologist, suggested that reflex depression of the jaw and reflexes of the tongue were the factors that removed the tongue from between the palatal shelves so they could change position and close. There was much disagreement at the time, but within 30 years the concept was dropped from the literature, and modern workers on cleft palate have ignored the views of His almost completely. At the 1967 meeting on cleft lip and cleft palate in Denver, however, it was reported by Walker (see Fraser, 1967) that head extension reflexes in the rat might be effective in elevating the palatal shelves. When I noticed the correlation in time of development between mouth-opening reflexes and palatal shelf transposition, it seemed worth pointing out this relationship (Humphrey, 1969c), even though I am not in the field of palatal investigations.

One of our 8½-week fetuses, a fetus 28 mm long, had 16 reflexes that included mouth opening. The mouth opened only part way, and the tongue and palate were well developed, with the palatal shelves in the vertical position along the sides of the tongue (Figures 6 and 7C in Humphrey, 1969c).

In the 27.1-mm fetus shown in Figure 1A and B, the mouth is beginning to open farther (Figure 2A) and the palatal shelves are in the horizontal position above the tongue (Figure 4D in Humphrey, 1969c), but the shelves are poorly developed. They do not touch in the midline and probably they never could. Here an underdevelopment of the palatal shelves could lead to a cleft palate, because the shelves are too short to meet, and to an early change in tongue and palatal shelf position, since there is little resistance to tongue withdrawal.

The 9½-week fetus of 34.3-mm crown-rump length in Figure 2B sometimes opened its mouth farther. The palatal shelves are closed and in contact with the nasal septum. They have indented each other as a ball indents the mitt of the catcher (Figure 14 in Humphrey, 1969c). This probably means that the closure was forceful and therefore could not have been due either to such a slow process as the accumulation of mucopolysaccharides or solely to an

intrinsic shelf force—explanations that are prominently mentioned in cleft-palate literature at the present time, as reported by Fraser (1967).

The tongue and the palatal shelves of two of the 8½-week fetuses (Figure 8 in Humphrey, 1969c) have particularly well-developed vertical palatal shelves along the sides of the tongue. The microscopic sections show the hyoglossus and genioglossus muscles of the tongue and the pull that they would exert on it on opening the mouth, even without muscle contraction. Of course, if these tongue muscles are able to contract at this time, the pull on the tongue is far greater.

As yet there is no published evidence, as far as I know, that the human tongue muscles do contract at this age. But there is evidence that at the time of palatal closure the tongue muscles of the rat, at least, are able to act. Wragg, Smith, and Borden (1968) showed that both the nerves that supply them and the muscles are functional. Walker (1969) has demonstrated that these reflexes—that is, both mouth-opening reflexes and tongue movements—take place in the mouse at the time of palatal closure. (I have included palatal closure here because I thought it might interest the people in speech and orthodontics who are concerned with the problems that arise from cleft palates.)

Let us turn now to the manner in which mouth opening and closure develop, because these actions are necessary for speech. The little 8½-week, 27.1-mm fetus is the smallest one that shows any evidence of the mouth opening to the corners (Figure 3A). At 9½ weeks (Figure 3B) the mouth may open wider, sometimes all the way to the corners, but the jaw depression that separates the lips is still part of a total pattern reflex that bends (or flexes) the head, trunk, and rump contralaterally, rotates the rump, and moves both upper and lower extremities.

If we go now from 8½ and 9½ weeks to 11 weeks (Figure 3A-C), instead of contralateral flexion with mouth opening when the fetus is touched about the mouth (Figures 1 and 2), as a rule there is head and trunk extension with a tightening of the lips (Figure 3A and B). This change in the mouth is hard to see, even with the motion pictures, and even more so in the photographic prints. Also, in the few instances that the mouth has been seen to open at this age, a different type of opening appears, a slit-like separation of the lips (Figure 3C) rather than formation of an acute angle (as in Figures 2B and 3D). The lips open no farther at the midline than at the corners and so give a quite different appearance than earlier. Two such reflexes were recorded in the motion pictures for this 11-week fetus and one for a fetus of 11½ weeks. These slit-like mouth openings are rapid. One of them took place in only six frames (or ⅜ sec), from closed mouth to closure again.

At 11½ weeks of menstrual age, however, there also may be another type of mouth opening (Figure 3D) when the face is rotated contralaterally as a part of the action. If the reflex is primarily a head extension reflex at this age, the mouth opening will be slit-like; if it is a reflex that includes rotation of the face or contralateral flexion of the head, the mouth is open a little more widely in the midline (Figure 3D). Because trunk and extremity action is still present,

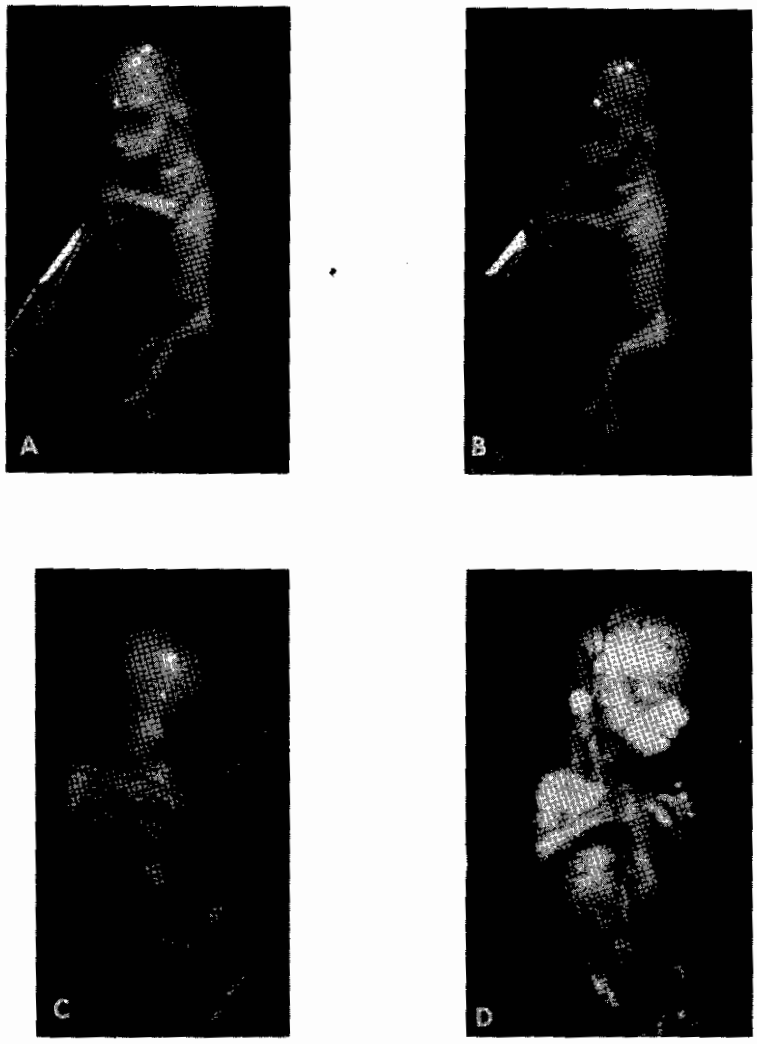



FIGURE 3A and B. Part of an extension reflex following accidental stimulation of the oral area with the clamp on the umbilical cord. Instead of the mouth opening, the lips appear tightly compressed (arrow on B). C shows the slit-like type of mouth opening sometimes seen at 11 and 11½ weeks. The reflex followed pulling of the fetus by the umbilical cord, and included head, trunk, and both upper and lower extremity movements. D illustrates another type of mouth opening at this period, in this case 11½ weeks, where the lips are separated more at the midline than laterally. The face rotated a little contralaterally in this reflex and the head bent contralaterally. There was also a small amount of trunk and extremity movement.

A, B, and C show an 11-week fetus, measuring 48.5 mm crown-rump length at slightly less than its size; D, 56.0-mm fetus, 11½ weeks of menstrual age, photographed at a little over natural size.

these reflexes might be considered of the total pattern type although they are no longer the same as the reactions at 8½-9½ weeks.

Up until about 10 to 10½ weeks of menstrual age, the areas of the face that are supplied by the trigeminal nerve are the only areas of the body surface that have been reported in the literature to be sensitive to stimulation. The eyelids and the more peripheral areas about the mouth and nose have become sensitive by 10½ weeks also. Likewise, by 10 to 10½ weeks, the palm of the hand responds to stimulation, and the sole of the foot soon afterward. After these areas become sensitive, mouth-opening reflexes may be secured by stimulating a variety of other regions in addition to the perioral area.

When one fetus of 12½ weeks (Figure 4A) was moved around by Hooker, touching the forearm with his finger (at the arrow) probably provided the stimulus for the mouth-opening response that followed. As part of the reflex, the head bent a little toward the side touched.

On attempting to elicit the Babinski reflex from the sole of the foot of the same 12½-week fetus, after the third stroke of the hair along the foot an extremely quick opening and closing of the mouth occurred (Figure 4B). The whole reflex from closed mouth to closure again took place in a fourth of a second. One can be sure that this closure is a stretch reflex because it is so rapid. Probably also the rapid closure after slit-like mouth opening at 11 weeks (Figure 3C) is a stretch reflex elicited by pull on the muscles when the jaw is lowered. Prior to 11 weeks, mouth closure has been a passive affair, I believe, not active.

Swallowing was first noted at 12½ weeks (Hooker, 1952, 1958) and at 13 weeks it was recorded on motion pictures (Figure 13 in Humphrey, 1968). On stimulation, the head extended a trifle, the mouth opened a little, and the larynx was pulled upward so the contour under the chin changed. The sternomastoid muscle was no longer visible because of the fullness in the neck area. As the reflex was completed, some head flexion reappeared, but the lips remained somewhat parted. So swallowing begins with a slight head extension, but in completing the swallowing reflex there is ventral head flexion. If one extends his own head back as far as he can, then tries to swallow, he will see how difficult it is to do so without head flexion.

In a swallowing reflex a week later (14 weeks, Figure 5) the stimulator touched the lips and was moved to one corner. Here also there was an initial slight head extension, followed by ventral flexion as swallowing was completed. Unfortunately, in Figure 5 a shadow over the mouth area makes it hard to see that the mouth closed and then reopened as swallowing was completed.

At 14 weeks there are other combinations of mouth opening with still different activities. In one reflex (Figure 6A and B), the stimulator was drawn along the back from below, upwards. The reaction included shrugging the shoulder, mouth opening and a sharp intake of breath or inspiratory gasp, then return to the original position. The action reminds one of a postnatal reaction to tickling, particularly as seen in the motion pictures. Even in the prints, without evidence of movement, the similarity to a reaction to tickling is evident.

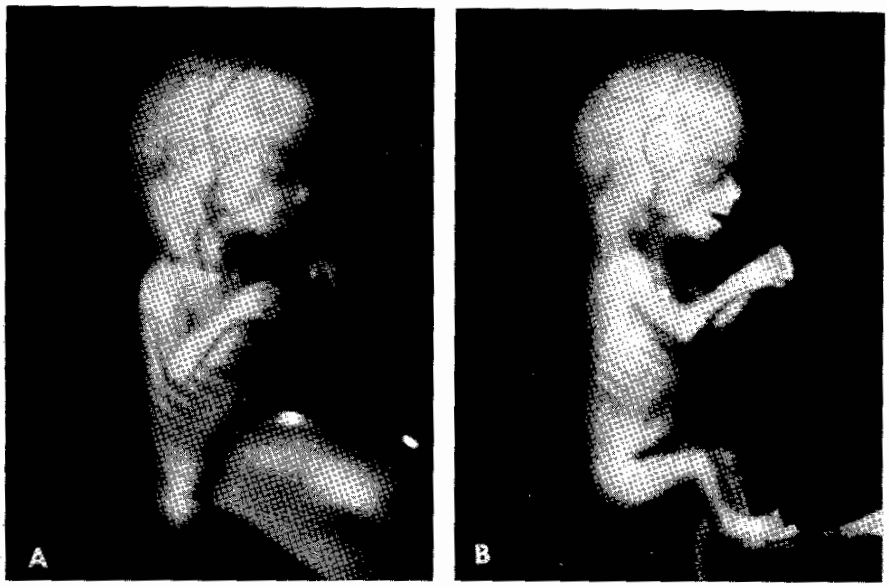




FIGURE 4. Three different types of mouth-opening reflexes resulting from jaw depression. A and B illustrate two mouth-opening reactions of a fetus of 12½ weeks, menstrual age (70.0 mm, crown-rump length) that occurred after stimulation of the forearm with a finger when the fetus was moved (A) and stroking of the sole of the foot with a hair esthesiometer (B). In A the tongue obscures the corner of the mouth; the reflex lasted about 1½ seconds. In B the action was rapid with the mouth open for only two motion picture frames (⅛ second). The photographs are slightly less than the size of the fetus. In C the mouth opening and the tongue elevation, forward tongue thrust, and tongue withdrawal followed stimulation of the palm of the hand. At the stage of the reflex reproduced here, the round contour of the tongue is easily seen at the corner of the mouth. This 14-week-old fetus (85.5 mm, crown-rump length) is illustrated at about 0.6 of its true size.

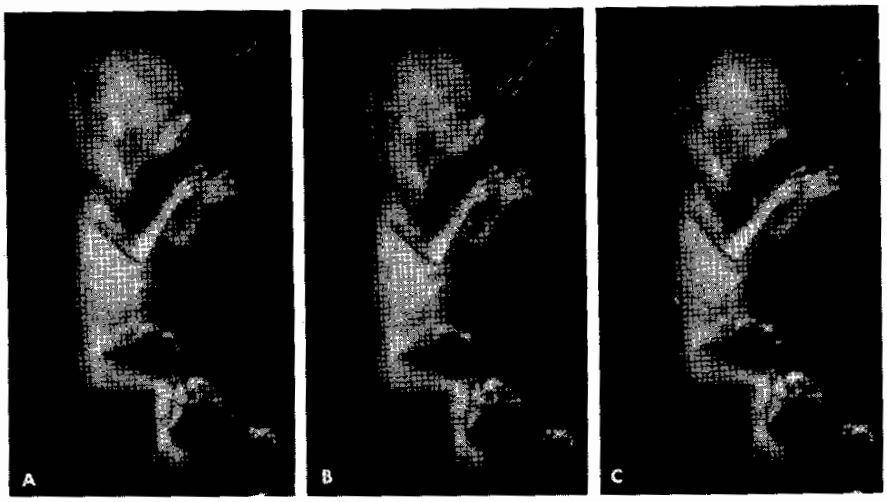



FIGURE 5. Three stages in a swallowing reflex of a 14-week fetus (88.5 mm, crown-rump length) elicited by moving the stimulator (A) from the midline to the corner of the lips. Ventral head flexion followed the initial head extension and closure of the mouth (B) as swallowing was completed (C). The mouth reopened at the end of this reflex. The photographs here are a little over half the size of the fetus.

In another reflex of the same 14-week fetus, the stimulator came up from the cheek over the fused eyelids (Figure 6C and D). In response to this stimulus the fusion line between the eyelids almost disappeared and the eyeball flattened (Figure 6D). This means that the eyelid muscle (orbicularis oculi) contracted. The result is a squint-like reflex. In addition, in the oral region there was a retraction of the corner of the mouth and the upper lip was raised near the corner (Figure 6D). The result is a sneer-like action. Both eyelid and lip reflexes are ipsilateral, or on the side stimulated.

Also at 14 weeks, two types of oral reflexes have been seen after stimulation of the palm of the hand. One of them (Figure 4C) consists of mouth opening by depressing the jaw and is accompanied by elevating the tongue, thrusting it forward, and then withdrawing it. In the other reflex (Figure 16 in Humphrey, 1969b) the lips separate due to action of the muscles of facial expression supplied by the facial or seventh cranial nerve. This reflex has been seen also at 18½ weeks, and reflexes like both of these reactions are found in premature and newborn infants. Thus again there is repetition of activity sequences.

At 15½ weeks (Figure 7), still other combinations of reflexes have developed. After stimulation over the eyelid, in addition to disappearance of the fusion line and flattening of the eyeball the mouth may open (Figure 18 in Humphrey, 1968). The reflex appears to be purely from the eyelid stimulation, but the stimulus to the eyelids involves the maxillary division of the trigeminal nerve to the lower lid as well as the ophthalmic branch to the upper one. In a similar reflex at 16 weeks (Figure 8A), after the stimulator was drawn down over the eyelids, the highlight over the ipsilateral eyebrow became smaller as

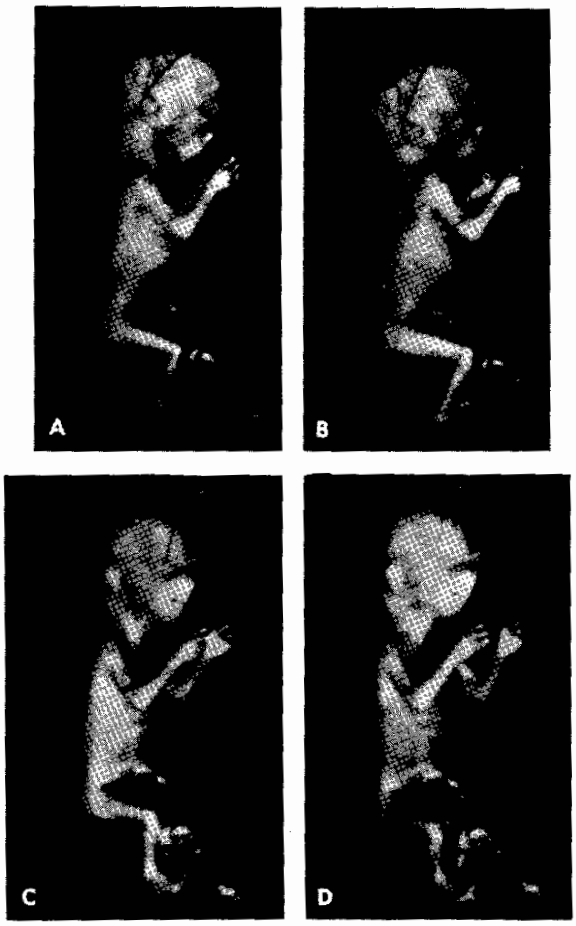



FIGURE 6. Part of the action from two reflexes elicited from the same 14-week-old fetus shown in Figure 5 and reproduced at approximately half the size of the fetus. In A and B, drawing the stimulator up the back resulted in an inspiratory gasp with mouth opening, head and trunk extension, expansion of the chest, and shoulder elevation with twisting of the trunk resembling a response to tickling. In C and D, a stimulus upward over the eyelids was followed by eyelid muscle action (orbicularis oculi contraction) and lip movement that retracted the corner of the mouth and lifted the upper lip near the corner—reactions producing squint-like and sneer-like movements, respectively.

the corrugator muscles contracted to give a scowl-like reflex. The fusion line of the eyelids also disappeared and the eyeball flattened. Comparison of Figure 8A with 8C shows these differences. In this reflex, there were both squint and scowl-like types of action. The lips also closed a little, then parted again (Figure 21 in Humphrey, 1968).

As early as 10½ weeks, when the first squint-like reflex was seen, and 11 weeks, when scowl-like actions have appeared, some muscles of facial expression

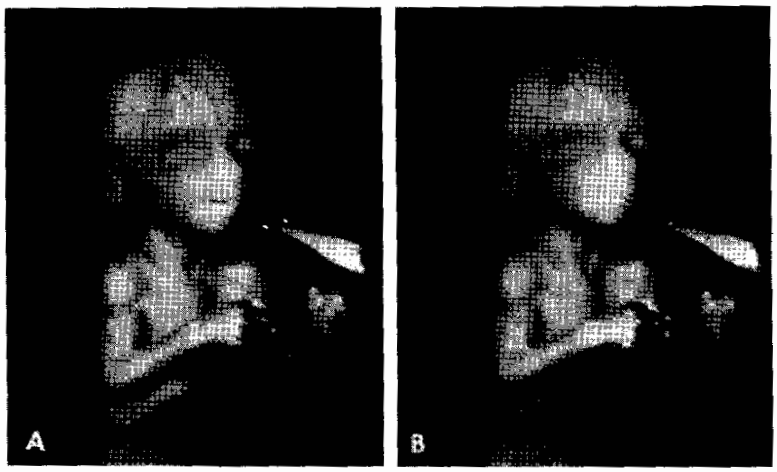



FIGURE 7. Tongue and lip movements of a fetus of 15½ weeks of menstrual age (107.5 mm, crown-rump length) reproduced at about 0.7 normal size. The tongue and lips had been stimulated when separation of the lips in A revealed a dark area at the midline interpreted as a groove lengthwise of the tongue. Later the lips closed tightly on the stimulator as shown in B.

are functioning. As soon as these muscles can react, the reflex mechanism for the expression of emotion is functioning. It is only the reflex mechanism for the expression of emotions, however. No emotion (as we usually think of it) is present, only the reflex action. Postnatally, the squint, the scowl, and the retraction of the angle of the mouth into a sneer-like appearance all become expressions of true emotion.

A glass rod—instead of the hairs usually employed—was used as a stimulator of one reflex from a fetus 18½ weeks of menstrual age. After the rod was inserted into the mouth for a considerable way, a gag reflex resulted (Figure 23 in Humphrey, 1968). This reflex is included to show that the facial expression that accompanies a gag reflex at this age is much like those seen on the faces of children and adults under similar circumstances. The appearance produced in this reflex by the muscles of facial expression is another example of the functioning of these mechanisms for the expression of emotion before emotion itself is possible.

The beginning of tongue movements in human fetuses has never been observed. There is no proof, but the first movement of the human tongue is probably a downward pull when it is withdrawn from between the palatal shelves. Both the hyoglossus muscles, from the hyoid bone into the tongue, and the genioglossus muscles, from the mandible into the tongue, would exert a pull downward on the tongue when they contract, possibly as early as 8½ weeks, when the tongue and palatal shelves change position and mouth opening first begins. In the rat, at least, tongue movements begin early (Angulo, 1932) at the time shown by others for palatal closure.

FIGURE 8. Photographs of a fetus of 16 weeks, menstrual age (114.0 mm, crown-rump length) illustrating a combined squint-like and scowl-like reflex accompanied by slight separation of the lips (A). The eyebrow change in A is evident if the reduced size of the highlight over the eyebrow is compared with this area in C. The faint fusion line of the eyelids (arrow on A) and the flattened contour of the eyeball is evident also on comparison with C. B and C illustrate the slight protrusion of the lips that is beginning at this age. The photographs are about 0.4 the size of the fetus.

Stimulation of the tongue was first noted to produce a reflex at 12½ weeks of menstrual age (Hooker, 1952, 1958; Humphrey, 1964). In Figure 4A the corner of the open mouth is obscured so that the acute angle is not clear all of the way to the corner, evidently because the tongue is elevated enough to obscure the corner of the mouth (Figure 4A). Later in this reflex the corner of the mouth is clear, apparently because the tongue is withdrawn.

At 14 weeks of menstrual age, the tongue was clearly thrust forward and withdrawn when the jaw was lowered in a mouth-opening reflex that followed stimulation of the hand by pulling out a bit of amnion caught by the fingers (Figure 4C). The corner of the mouth is completely invisible due to the tongue movement forward in the part of the reflex shown. In one frame of this reflex the rapid withdrawal of the tongue leaves a streak reminiscent of a snake's tongue.

At 15½ weeks tongue movements are more varied. In one reflex (Figure 19 in Humphrey, 1968) the lips closed on the stimulator after it was drawn back and forth across the lips and the tongue. Then the mouth opened to show the tongue (Figure 7A) with a little dark spot where the stimulator touched it. This dark spot, I believe, is a longitudinal groove, or lengthwise furrow along the tongue. At this stage, evidently the sides of the tongue can turn upward to produce such a longitudinal furrow. Later the lips of the fetus closed tightly on the stimulator (Figure 7B). In another area of the motion picture record of this fetus, the tongue was held at the inner border of the lips with no indication that a groove was formed along it. At 20 weeks (Figure 9C) when the lips neared each other and the upper one puckered and the lower one protruded, the tip of the tongue appeared along their inner border.

Protrusion of the upper lip was reported by Hooker (1952, 1958) at 17 weeks and the lower lip at 20 weeks (Humphrey, 1964). Beginning protrusion of the lips on mouth opening appears to be present as early as 16 weeks, but is very difficult to see (Figure 8A-B). The upper lip protrudes before the lower lip, we believe, then both lips protrude.

At 20 weeks of menstrual age, the upper lip sometimes puckers and the lower lip protrudes (Figure 9). In the position of the mouth at rest in this particular fetus, the upper lip is elevated near the corner for part of the time (Figure 9A). Following their stimulation as shown in Figure 9, the lips came closer together, the elevation near the corner disappeared (Figure 9B), and the lower lip protruded more definitely (Figure 9C). The outline of the mentalis muscle is apparent in 9A and 9B, but is more prominent in 9C. This protrusion in the postnatal child becomes a pout. The small up-and-down (or vertical) lines on the upper lip are the pucker lines. They became accentuated when the lips closed (Figure 9C). The protrusion and puckering of the lips are preliminary developments for the complex sucking reflex. The lips must be both puckered and closed before sucking can occur.[1]

[1]At the conference, the author showed a motion picture of several fetal activity sequences.—Ed.

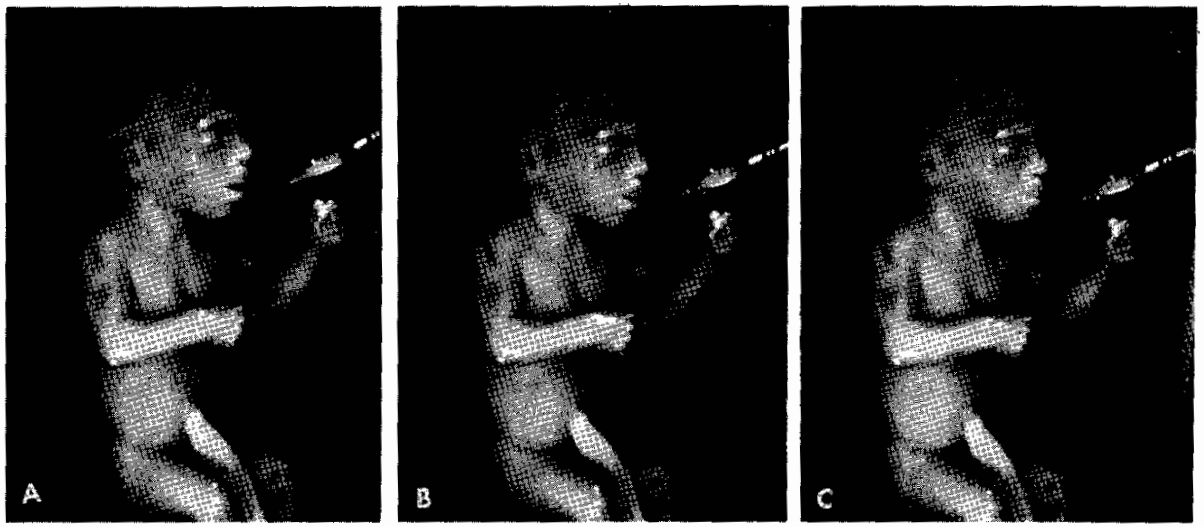



FIGURE 9. Puckering of the upper lip and protrusion of the lower lip of a 20-week fetus (166.0 mm, crown-rump length). The outline of the mentalis muscle, which protrudes the lower lip, is present in A and B, but clearest in C where the lip action is greatest. The vertical pucker lines on the upper lip in A and B are accentuated in C as the upper lip is pursed and the slight elevation near the corner in A disappears. When the pucker and protrusion occurred in C, the tongue moved up and forward behind the lips. These illustrations are only about one-third the size of the fetus.

I want now to point out the neuroanatomical basis for the repetition of motor sequences postnatally and to emphasize the fact that this is not a repetition of identical movements, but of the sequence or order in which movements develop, which is another matter entirely.

First, embryologically, the neural tube closes in the upper cervical region, above the cervical enlargement that supplies motor nerve fibers for the arms. Closure then proceeds cephalad to the uppermost cord levels and brain stem and caudad to the cervical enlargement that innervates the arms, to the thoracic region, and to lumbosacral cord levels for the legs. Thus closure proceeds in two directions. Differentiation will also begin in the upper cervical levels and proceed cephalad and caudad in the same manner. Consequently, function follows in the same order, beginning in the region that closes first, and proceeding both caudad and cephalad (Humphrey, 1969b).

I have pointed out that the first movement is bending the head to the side contralateral to the stimulus. This action takes place over the motor neurons and nerves in the region where the neural tube first closed. As differentiation progresses caudalward from the region of neural tube closure, the trunk bends to the opposite side and the rump rotates. Differentiation and function have then reached caudal spinal cord levels.

Differentiation also proceeds cephalad from the upper cervical region, so that active partial mouth opening is added to the reflex. This mouth opening is the result of the lowering of the mandible (or jaw depression) and may be due partly to the action of the muscles that are supplied by the first and second cervical nerves. As differentiation proceeds and the levels of the seventh (or facial) motor nucleus and the fifth (or trigeminal) motor nucleus are reached, the action of the mylohyoid and the digastric muscles aid in lowering the mandible. The neurons that are thought to supply these muscles differentiate earlier than the other nerve cells in the motor trigeminal and facial nuclei (Jacobs, 1970).

The neurons which supply the tongue muscles are located throughout most of the medulla, in the hypoglossal nucleus, beginning slightly above the spinal cord. As differentiation proceeds upward, it is reasonable to assume that these motor neurons will both develop and become active early also and that active tongue reflexes will then begin.

When the descending pathways from upper levels of the nervous system grow down to synapse with the lower motor neurons of cranial and spinal nerves, the fibers will reach the cervical spinal cord before thoracic levels, then arrive at lumbar levels, and last at sacral regions. Therefore, the neurons innervating the upper extremities, the trunk, and the lower extremities will develop synapses in the same general sequence in which the development of the lower motor neurons took place and the reflex arcs began functioning. This order of development and function is primarily due to the fact that the fibers arise at higher levels of the nervous system and so reach the spinal cord segments in a descending order. Neurophysiologically, the neurons that have functioned the longest require the least stimulation to fire them, and less oxygen to react, so they are

discharged more readily and therefore will function earlier in development. These factors will contribute also to a repetition of the same order (or sequence) of motor events as the higher nervous system levels become active. However, there will be a variation in the movements themselves, even though the sequence is retained (Humphrey, 1969b).

The reflex arcs are regulated—activated or inhibited—from upper brain-stem levels before higher levels like the basal ganglia (or striatal complex) influence them. The extrapyramidal systems from the cortex which also produce gross motions, such as one sees in spastic children, will activate the motor neurons of cranial and spinal nerves in a similar sequence developmentally. Of course, some actions are never performed until postnatal life, when the motor cortex (area 4) becomes functional. In the development of finger action, for instance, the fine action of opposing the thumb and fingers develops last and is considered to be purely postnatal.

I would like to mention one other point. In response to external tactile stimulation about the mouth when reflex activity first begins, the reaction includes the organism as a whole, and so is a total pattern type of reflex as the term was used by Coghill (1929). All of the neuromuscular mechanisms that are capable of functioning do react. As the contralateral flexion reflexes increase in extent, they include mouth opening along with head, trunk, and extremity activity. Then suppression or inhibition of part of the action begins and the trunk and extremity activity is mostly eliminated (on perioral stimulation) and localized actions appear. For the infant or the adult, gross movements must be suppressed if fine ones are to be made. At the higher levels of the nervous system, including the cortex, the gross movements must be suppressed or controlled, in order to perform fine movements.

## ACKNOWLEDGMENT

This investigation was supported by a Public Health Service career program award NB-K6-16716 from the National Institute of Neurological Diseases and Stroke, and was aided by research grant HD-00230 from the National Institute of Child Health and Human Development, National Institutes of Health.

## REFERENCES

Angulo y Gonzáles, A. W., The prenatal development of behavior in the albino rat. *J. comp. Neurol.*, **55**, 395-442 (1932).

Coghill, G. E., *Anatomy and the Problem of Behaviour.* London: Cambridge Univ. Press (1929). New York: Hafner (1964).

Fraser, F. C., Meetings. Cleft lip and cleft palate. *Science*, **158**, 1603-1606 (1967).

Hamburger, V., Some aspects of the embryology of behavior. *Quart. Rev. Biol.*, **38**, 342-365 (1963).

His, W., Beobachtungen zur Geschichte der Nasen- und Gaumen-bildung beim menschlichen Embryo. *Abh. sächs. Akad. Wiss. Leipzig, Math. Phys. Kl.*, **27**, 349-389 (1901).

Hooker, D., *The Prenatal Origin of Behavior.* Lawrence, Kans.: Univ. of Kansas Press (1952). New York: Hafner (1969).

HOOKER, D., Evidence of the prenatal function of the nervous system in man. James Arthur Lecture on the Evolution of the Human Brain. New York: American Museum of Natural History (1958).

HUMPHREY, TRYPHENA, The spinal tract of the trigeminal nerve in human embryos between 7½ and 8½ weeks of menstrual age and its relation to fetal behavior. *J. comp. Neurol.*, **97**, 143-209 (1952).

HUMPHREY, TRYPHENA, The relation of oxygen deprivation to fetal reflex arcs and the development of fetal behavior. *J. Psychol.*, **35**, 3-43 (1953).

HUMPHREY, TRYPHENA, Some correlations between the appearance of human fetal reflexes and the development of the nervous system. *Prog. brain Res.*, **4**, 93-135 (1964).

HUMPHREY, TRYPHENA, The development of mouth opening and related reflexes involving the oral area of human fetuses. *Ala. J. med. Sci.*, **5**, 126-157 (1968).

HUMPHREY, TRYPHENA, Reflex activity in the oral and facial area of human fetuses. Chapter 12 in J. F. Bosma (Ed.), *Second Symposium on Oral Sensation and Perception.* Springfield, Ill.: Charles C Thomas, pp. 195-233 (1969a).

HUMPHREY, TRYPHENA, Postnatal repetition of human prenatal activity sequences with some suggestions on their neuroanatomical basis. In R. J. Robinson (Ed.), *Brain and Early Behaviour, Development of the Foetus and Infant.* New York: Academic, pp. 43-84 (1969b).

HUMPHREY, TRYPHENA, The relation between human fetal mouth opening reflexes and closure of the palate. *Amer. J. Anat.*, **125**, 317-344 (1969c).

JACOBS, M. J., The development of the human motor trigeminal complex and accessory facial nucleus and their topographic relations with the facial and abducens nuclei. *J. comp. Neurol.*, **138**, 161-194 (1970).

PRECHTL, H. F. R., and BEINTEMA, D., *The Neurological Examination of the Full-Term Newborn Infant.* London: William Heinemann Medical Books (1964).

WALKER, B. E., Correlation of embryonic movement with palate closure in mice. *Teratology*, **2**, 191-197 (1969).

WINDLE, W. F., Genesis of somatic motor function in mammalian embryos: A synthesizing article. *Physiol. Zool.*, **17**, 247-260 (1944).

WINDLE, W. F., Reflexes of mammalian embryos and fetuses. In P. Weiss (Ed.), *Genetic Neurology.* Chicago: Univ. Chicago Press, pp. 214-222 (1950).

WRAGG, L. E., SMITH, J., and BORDEN, C., Tongue muscle morphology and function at the time of secondary palate closure in the rate. (Abstract) *Anat. Rec.*, **163**, 188 (1968).

# POSTNATAL DEVELOPMENT OF THE OROFACIAL MUSCULATURE

ROBERT E. MOYERS

*University of Michigan, Ann Arbor, Michigan*

In this discussion of the maturation of the orofacial neuromusculature, emphasis will be placed on its significance to workers in facial growth and speech. A number of areas will be identified where our knowledge is incomplete, and some logical sequences for research strategy and clinical therapy will be suggested.

Few workers who spend their time solely on the early postnatal maturation of the orofacial neuromusculature have a solid background in developmental neurobiology. The rest of us—speech pathologists, pathologists, pediatricians, and orthodontists—have a secondarily dependent interest in developmental neurobiology, so we approach research in this area with a bias. Walt Whitman once said that our attitudes are the victims of our experience. When we look, for example, at a neonate with some neural deficits, if we're pediatricians, we have one idea in mind; if we're speech pathologists we have another; if we're orthodontists, we have another. All of us probably ought to have a developmental neuroscientist working with us who, so far as possible, avoids the clinical bias. In turn, we might help him keep his own work pointed to that which is of significance to society.

We often tend to choose the work that pleases us most, whether our own ideas are tenable with related research findings or not. Therefore, it is important in a paper of this type to label areas of incomplete knowledge, or findings which seem to conflict, as well as to document facts. In our Center for Human Growth and Development, we have a variety of people from several fields who bring their different viewpoints together with the hope of giving each individual more balance in his approaches to his own work. Intellectual hybrid vigor is badly needed, hence this conference.

## CLASSES OF NEUROMUSCULAR ACTIVITIES

Sir William Osler once said, in talking about diagnosis, that clinicians are inclined to believe that the problem begins when the patient first is examined. The conflagrations we call disease and disorders are the result of a constant battle that may have been going on for a number of years between forces of

pathology and the forces of repair, until a moment arrives when one side dominates and the patient seeks help. Our ability to diagnose the problem and to treat it successfully is largely dependent upon our ability to reconstruct what went on before the patient came to us.

When we see a patient in orthodontic or speech pathology practice, and observe a functioning orofacial neuromuscular system, we look at a number of combined reflexes which may best be fractionated and seen on a developmental scale. Some reflexes may go as far back as prenatal days, and our ability to condition them, even by the most elaborate methods, is more difficult than changing reflexes developed postnatally.

*"Unconditioned" Reflexes or Responses*

By unconditioned reflexes, we mean those which are present at birth, having appeared as a normal part of the prenatal maturation of the neuromuscular system, a process which does not involve conditioning or learning in the ordinary sense. If such maturation has not occurred by birth, the infant may not survive. Such reflexes range from the simplest monosynaptic responses, such as tendon jerk, to the very complex reflexes involved in infant nursing. Most importantly, nearly all of these responses are related to food-getting, to protection of the airway, and to body regulatory mechanisms.

Among the unconditioned congenital reflexes operable in the oropharyngeal region of the neonate are those of respiration, mandibular posture, tongue posture, infantile swallowing, suckling, gagging, vomiting, coughing, and sneezing. The infant does not learn to cough, for example, for he might die of asphyxiation by the time he'd had sufficient "practice" in breathing. Congenital unconditioned reflexes such as these require minimal reinforcement and are very difficult to alter by usual conditioning procedures.

*Conditioned Reflexes*

The second category of neuromuscular activity is conditioned reflexes, of which there are two types. The first of these are *reflexes which appear with normal growth and development.* Of course, no conditioned reflex is capable of being learned until all of the necessary parts in the central nervous system and musculature have matured sufficiently to make that learning possible. This is really Piaget's concept of readiness applied to the central nervous system. One can't have an occlusal reflex, for example, unless he has some occlusion. But there are many arguments here. For example, crawling, walking, grasping: does a baby learn to grasp and crawl, or was he taught? Would he have acquired these motor skills at a certain age with no teacher to encourage him?

In the orofacial region, the mature swallow and mastication are examples of reflexes which normally appear with growth and development. The child's swallow changes after the teeth appear. The mature swallow, requiring a sensory input from the teeth and an exchange of sensation between the upper and lower teeth, occurs as a conditioned reflex, learned in adjustment to the developing occlusion and maturing musculature.

The second type of conditioned reflex we are concerned with is that of the so-called "bad habits," for example, thumb sucking and some tongue-thrust swallows.

*Voluntary Activities*

Finally, willful acts under cortical control must be considered. The infantile swallow of the neonate is an example of an unconditioned reflex, since infants are swallowing reflexly by the time they are born. The mature teeth-together swallow, which appears during the first year of life, is an example of a reflex which appears with normal growth and development. The learned tooth-apart swallow, caused by a painful tooth, an occlusal interference, inflamed tonsils, or pharyngitis is an example of the conditioned reflex swallow. And of course one can swallow voluntarily as well.

If we separate these reflexes that can be conditioned from those which resist conditioning and which are basic to the very maintenance of life, we automatically reshape our views.

What is the significance of this? One can read articles in our orthodontic and speech pathology literature telling how to correct abnormal swallow, without even separating the kinds of swallows being discussed. Some are not even aware that a tooth-apart swallow and an infantile swallow are physiologically and developmentally two quite separate and different things. Furthermore, a test of whether or not the swallow has been corrected is rarely given.

The main problem is not only to succeed in conditioning the swallow while the patient is in your office; he must adopt the new reflex and automatically reinforce it for us to claim to have "corrected" the problem.

## DEVELOPMENT OF OROPHARYNGEAL FUNCTIONS

*Prenatal Maturation*

During prenatal life the neuromuscular system does not mature evenly (Hooker, 1944). It is not accidental that the orofacial region matures (in the neurophysiological sense) ahead of limb regions, since the mouth so concerns a number of vital functions which must be operable by birth—for example, respiration, nursing, and protection of the oropharyngeal airway (Hooker, 1939). In the human fetus, by about the eighth week generalized uniform reflex movements of the entire body can be elicited by tactile stimulation. The few spontaneous movements, in response to as yet unidentified stimuli, have been observed as early as nine-and-a-half weeks. Localized specific and more peripheral responses can be produced before 11 weeks. At this time, stimulation of the nose-mouth region causes lateral body flexion (Hooker, 1939). By 14 weeks, the movements have become much more individualized, so very delicate activities can be executed. When the mouth area is stimulated, general bodily movements no longer are seen, but instead, facial and oricular muscle responses are produced (Humphrey, 1968). Stimulating the lower lip causes the tongue to

move, for example. Stimulation of the upper lip causes the mouth to close, and, often, deglutition to occur (Humphrey, 1970).

Respiratory movements of the chest and abdomen are first seen at about 16 weeks. The gag reflex has been demonstrated in the human fetus at about 18½ weeks of menstrual age (Humphrey, 1970). By 25 weeks, respiration is shallow, but may support life for a few hours if it has once been established.

Stimulation of the mouth at 29 weeks menstrual age has elicited suckling, although complete suckling and swallowing are not thought to be developed until at least 32 weeks (Hooker, 1939, 1944; Humphrey, 1964, 1968, 1970).

Davenport Hooker and Tryphena Humphrey have shown us that there is an orderly, sequential staging of events in prenatal orofacial maturation—a staging seen throughout the body, but much more advanced in the oropharyngeal region. All this has to be established by the time of birth in order for the child to survive.

*Neonatal Oral Functions*

At birth, the tactile acuity is much more highly developed in the lips and mouth than it is in the fingers. The infant carries objects to his mouth to aid in the perception of size and texture long before he inserts them into his mouth as a part of teething. The neonate slobbers, drools, chews his toe, sucks his thumb, and discovers that gurgling sounds can be made with his mouth (Bosma, 1967).

Freudians consider all of this oral eroticism, as they do adult smoking; but in the infant it surely is also exploratory and exercising the most sensitive perceptual system in the body at that time. Oral functions in the neonate are guided primarily by local tactile stimuli, particularly those in the lips and the front part of the tongue.

The tongue at this age does not guide itself; rather, it follows superficial sensation. The posture of the neonate's tongue is between the gum pads, and often it is far enough forward to rest between the lips, where it can perform its role of sensory guidance more easily. The young infant, to a great extent, interprets the world with his mouth, and the integration of oral activities is therefore by sensory mechanisms.

If you touch a young child's lips or tongue and have him follow your finger, his whole head and body turns. A little bit later he turns his head separately from his body, and still later he will move his mandible without moving his head. It is only last of all that he can follow the tongue without moving his mandible. These stages appear in a natural sequence, just as teeth erupt on a kind of schedule.

The infant uses his mouth for many purposes. The perceptual functions of the mouth and face are combined with the sensory functions of taste, smell, and jaw position. The neonate's primary relationship to his environment is by means of his mouth, pharynx, and larynx. Here a high concentration of readily available receptors become stimulated and modulate the already matured brain-stem

coordinations which regulate respiration and nursing and determine head and neck positions during breathing and feeding.

The sensitivity of the tongue and lips is perhaps greater than that of any other body area. The sensory guidance for oral functioning, including jaw movements, is from a remarkably large area (Bosma, 1967). These sensory inputs are compounded by many dual contacting surfaces, such as tongue and lips, soft palate and posterial pharyngeal wall, and the compartments of the temporomandibular articulation (Bosma, 1967). A great array of sensory signals is required for integration, coordination, and interpretation of this complex system.

### *Infantile Suckling and Swallowing*

The effectiveness of these activities is a good indication of the neurological maturation of premature infants. It has been found that a child will follow the same patterns in certain oral reflex movements years after initial learning. For example, a study was made of children whose records had been kept from infancy. As long as five years after weaning, if given a bottle from which to suckle, they produced the same suckling, swallowing, and respiratory rhythms as they had when infants (Moyers, 1964, and author's unpublished data). If they swallowed in a suckle-suckle-swallow type of pattern, which we call a two-for-one, this same rhythm appeared years later. It may be a three-for-one or even a four-for-one, but the pattern is maintained. Such primitive reflexes are difficult for us to change. How foolish it is for us, with our present ignorance about conditioning mechanisms, to try to alter some of these reflexes. We must spend more time with those problems that we have at least a theoretical chance to condition.

Rhythmic elevation and lowering of the jaw provide sequential changes in positions of the tongue in coordination with its suckling contractions. The activities of suckling are closely related temporally to the motor functions of positional maintenance of the airway.

Electromyographic studies in our own laboratory have confirmed visual observation reported in England by a number of people revealing that, while the mandibular movements are carried out by the muscles of mastication, the mandible is primarily stabilized during the actual act of infantile swallowing by concomitant contractions of the tongue and facial muscles (Moyers, 1964). At the actual time of the infantile swallow, the tongue lies between the gum pads and in close approximation with the lingual surface of the lips. So the infantile swallow is neuromuscularly a different mechanism than the mature swallow.

Characteristic features of the infantile swallow are that (1) the jaws are apart, with the tongue between the gum pads; (2) the mandible is stabilized primarily by contractions of the muscles of the VIIth cranial nerve and the interposed tongue; and (3) the swallow is guided, and to a great extent controlled, by sensory interchange between the lips and tongue.

### *Maintenance of the Airway*

The oral jaw musculature is responsible for the vital positional relationships which maintain the oral pharyngeal airway. While the infant is resting, a rather uniform diameter for the airway is provided by (1) maintaining the mandible anteroposteriorly, and (2) stabilizing the tongue and posterior pharyngeal wall relationships.

The axial musculature around the vertebrae is also concerned. These primitive neonatal protective mechanisms provide the motor background upon which, with growth, all of the postural mechanisms of the head and neck region are developed. Physiologic maintenance of the airway is of vital, continuing importance from the first day throughout life.

This little neonate that can't focus its eyes, that can't make a purposeful movement with any of its limbs, that can't hold its head upright, that has absolutely no control of the lower end of its GI tract, has absolutely exquisite control of some functions in the orofacial regions. Why? He must in order to survive!

The gag reflex is also present at birth to protect the airway, but it changes as the child grows older, to accommodate visual, acoustic, olfactory, and psychic stimuli which are remembered and thus condition the gag reflex.

### *Infant Cry*

The involvement of the orofacial and jaw musculature in infant crying has been reported in classic studies by Bosma, Truby, and Lind (1965). When the aroused baby is crying, the oral region is unresponsive to local stimulation. The mouth is held wide open, while the tongue is separated from the lower lip and from the palate. The steady stabilization of the size of the pharyngeal airway is given up during crying; and there are irregular, varying constrictions during expiration of the cry, and large, reciprocal expansions during the alternating inspirations.

## EARLY POSTNATAL DEVELOPMENT OF ORAL FUNCTIONS

### *Mastication*

The interaction between the rapidly and differentially growing craniofacial skeleton and the maturing neuromuscular system brings about sequentially progressive modifications of the elementary oral functions seen in the neonate. Mandibular growth, downward and forward, is greater during this time than is midface growth, giving rise to a greater separation of the thyroid bone and thyroid cartilage from the cranial base and mandible.

Maturation of the musculature and delineation of the temporomandibular joint help provide a more stable mandible. Although the mandibular growth carries the tongue away from the palate and helps provide differential enlargement of the pharynx, patency of the airway is maintained—a most important point.

The soft palate and the tongue are commonly held in apposition, but as the tongue is no longer lowered by mandibular growth, its functional relationship with the lips is altered, an alteration aided by the vertical development of the alveolar process. So the morphologic relationship of the tongue and lips is strained. At rest now, the tongue is no longer in generalized apposition with the lips, buccal wall, and soft palate. The lips elongate and become more selectively mobile; the tongue develops discrete movements which are separate from lip and mandibular movements. The labial valve mechanism is constantly maintained during rest and feeding so that food is not lost.

The development of speech and mastication as well as facial expression requires a furthering of the independent mobility of the separate parts. In the neonate, however, the lips tightly surround a plunger-like tongue, moving in synchrony with gross mandibular movements. Speech, facial expression, and mastication require the development of new motor patterns as well as greater autonomy of the motor elements. Not all of the developmental aspects of these functions are known. But mastication certainly does not gradually develop from infantile nursing. Rather, it seems, the maturation of the central nervous system permits completely new functions to develop. These functions are triggered to an important extent by the eruption of the teeth.

One of the most important factors in the maturation of mastication is the sensory aspect of newly arriving teeth. The muscles controlling mandibular position are cued by the first occlusal contacts of the antagonistic incisors. Serial electromyographic studies at frequent intervals during the arrival of the incisors have demonstrated conclusively that the very instant the maxillary and mandibular incisors accidently touch one another, the jaw musculature begins to learn to function in accommodation to the arrival of the teeth (Moyers, 1964, and author's unpublished data).

Thus, since the incisors arrive first, the closure pattern becomes more precise anteroposteriorly before it does mediolaterally. All occlusal functions are learned in stages. The central nervous system and the orofacial and jaw musculature mature concomitantly, and usually synchronously, with the development of the jaws and dentition (Moyers, 1971).

The earliest chewing movements are irregular and poorly coordinated, like those during the early stages of learning of any motor skills. As the primary dentition is completed, the chewing cycle becomes more stabilized, using more efficiently the individual's pattern of occlusal intercuspation (Moyers, 1971). In the very young child, sensory guidance for masticatory movement is provided by the receptors in the temporomandibular articulation, the periodontal ligament, the tongue, and the oral mucosa and muscles; and, of these, it seems by far that the most important are those of the temporomandibular articulations, and next those of the periodontal ligament. Cuspal height, cuspal angle, and incisal guidance (which is usually minimal in the primary dentition) play a role in establishment of chewing patterns in the infant. However, condylar guidance is not important, since the eminentia articularis is ill-defined and the temporal fossae are shallow (Moyers, 1971).

Rather, it may be supposed that the bone of the eminentia articularis forms where temporomandibular function permits it to develop (Moyers, 1971). In a similar fashion, the plane of occlusion is established by the growth of the alveolar process, during eruption of the teeth, to heights permitted by the configuration and functioning of the neuromusculature.

The individual's movements during the chewing cycle are a developed, integrated pattern of many functional elements. In the young child, at the time of completion of the primary dentition, masticatory relationships are nearly ideal, since all three systems (bone, teeth, and muscle) still show the lability of development and are highly adaptive. Cusp height and overbite in the primary dentition are more shallow, bone growth more rapid and adaptive, and neuromuscular learning more easily obtained, since pathways and patterns of activity are not yet well established. Adaptations to masticatory change are much more difficult in later years, as every dentist knows.

*Facial Expression*

In a not dissimilar way, most subtle facial expressions are learned, largely by imitation, so we think, and begin about the time the primitive uses of the VIIth nerve musculature for infantile swallowing are abandoned. Those of us who are parents imagine all sorts of facial expressions in the young neonate. Actually, observing the infant objectively, we must admit that the expression is often rather blank. The reason is that the facial muscles are busy being used for the massive efforts of mandibular stabilization necessary during infantile swallowing. Eventually the VIIth nerve muscles and the mandible become controlled and stabilized more by the muscles of mastication, particularly during unconscious reflex swallowing, and the delicate muscles of facial expression become free to be truly "muscles of facial expression."

Although many facial expressions are learned through imitation, some facial responses are unlearned and can be traced back to reflexes of earlier primates. Similar facial displays have evolved in the four lines of modern primates in which monkey-like forms have developed. Comparative studies have been made revealing similar reflex expressions of basic protective anger, for example, in various primates—the same primitive instinctive expressions you have seen on your best friend.

*Speech*

Purposeful speech is different from reflex infant cry. Infant crying is associated with irregular tongue and mandibular positions related to sporadic inspirations and expirations during crying (Bosma, Truby, and Lind, 1965). Speech, on the other hand, is performed on a background of stabilized and learned positions of the mandible, pharynx, and tongue. Infant cry is usually a simple displacement of parts, accompanied by a single explosive emission, whereas speech can only be carried out by polyphasic and sequential motor activities synchronized closely with breathing (Bosma, Truby, and Lind, 1965).

Speech is regular; infant cry is sporadic. Speech requires complicated, sophisticated, varying sensory conditioning elements during learning; infant cry is primitive and unlearned. There is an analogy here between the mature swallow and the infantile swallow, between mastication and infantile suckling, and between learned speech and infant crying, all of which are best seen on a developmental scale.

### *Mature Swallow*

During the latter half of the first year of life, several maturational events occur which markedly alter the orofacial musculature's functioning. The arrival of the incisors cues the more precise opening and closing movements of the mandible, compels a more retracted tongue posture, and initiates the learning of mastication.

As soon as bilateral posterior occlusion is established, usually with the eruption of the first primary molars, true chewing motions start, and the learning of the mature or teeth-together swallow begins (Moyers, 1971). Gradually the Vth cranial nerve muscles assume the role of mandibular stabilization during the swallow, and the muscles of facial expression abandon the crude, infantile function of suckling and swallowing and begin to learn the more delicate and complicated functions of speech and facial expression (Moyers, 1964).

The transition from infantile to mature swallow takes place over several months, aided by maturation of neuromuscular elements, the appearance of upright head posture, and hence a change in the direction of gravitational forces on the mandible, the instinctive desire to chew, the necessity to handle textured food, dentitional development, and so on (Moyers, 1964). Most children achieve most features of the mature swallow by 12 to 15 months of age.

Characteristic features of the mature swallow are that (1) the teeth are together; (2) the mandible is stabilized by contractions of the mandibular elevators, which are primarily Vth cranial nerve muscles; (3) the tongue tip is held against the palate above and behind the incisors; and (4) there are minimal contractions of the lips during the mature swallow.

### *Development of Neurophysiologic Regulation of Jaw Positions and Functions*

Jaw position, like a number of other automatic-somatic activities, normally is largely reflexly controlled, even though it can be altered voluntarily. A surprising number of jaw functions are carried on at the subconscious level, even though conscious control is possible and sometimes necessary. Recent research has shown that the receptors in the temporomandibular capsule area are far more important than previously thought. Many of the enigmas of prosthetic dentistry have fallen into place logically as a result of the research efforts of Thilander (1964) in Sweden and Greenfield and Wyke (1966) in England.

Since most research on the neurophysiological regulation of jaw position and function has been done on the adult, there has been a tendency to transfer prosthodontically oriented concepts, based on sound adult clinical practice, to

children. During development, before all of the system's parts have appeared, and while growth is dominant, it is hazardous to maintain the same clinical assumptions which are so useful in understanding the adult.

Our knowledge about the developmental aspects of orofacial and jaw neurophysiology is most incomplete at this time, though much research is under way. We must remember that many of our attitudes are victims of our experience with degenerating occlusions in adults, and the critical clinical factors which obtain under those circumstances may not be present in the child, or may have different relative significance during development.

## CONCLUDING REMARKS

Aristotle, though not writing about child growth and development, said that every man ought to be judged by a ruler within him. The problem is: what is the ruler? One of the crude rulers useful for this conference, and for our collaborative research in the future, is a ruler which is a developmental scale. Not a time scale, necessarily, and not a quantified scale, but a crude, ordinal concept that some of these reflexes we treat have their origins in prenatal days. Our ability to condition them, even by the most elaborate conditioning methods we have, is quite different from our ability to condition other reflexes learned during postnatal growth or in response to environmental stimuli. Each child must be judged by his progress along such a developmental neurological ruler and our treatments cannot naively and neatly presume to avoid the rules of neurobiology. We need much more collaborative research among dentists, speech scientists, and developmental neurobiologists to maintain steady progress toward practical goals. A thorough understanding of the development of orofacial neurobiology is the only basis for such cooperation in research, the only foundation for clinical advancement.

## REFERENCES


Bosma, J. F. (Ed.), *Symposium on Oral Sensation and Perception.* Springfield, Ill.: Charles C Thomas (1967).

Bosma, J. F., Truby, H. M., and Lind, J., Cry motions of the newborn infant. *Acta paediat. Scand.*, Suppl. 163, 61-92 (1965).

Greenfield, B. E., and Wyke, B., Reflex innervation of the temporomandibular joint. *Nature*, 211, 940-941 (1966).

Hooker, D., Fetal behavior. *Association for Research in Nervous and Mental Disease.* XIX. *Inter-relationship of Mind and Body.* Baltimore: Williams and Wilkins, pp. 237-243 (1939).

Hooker, D., *The Origin of Overt Behavior.* Ann Arbor: Univ. of Mich. (1944).

Humphrey, T. J., Some correlations between the appearance of human fetal reflexes and the development of the nervous system, *Progr. brain Res.*, 4, 93-133 (1964).

Humphrey, T. J., Development of mouth opening and related reflexes involving oral area of human fetuses. *Ala. J. med Sci.*, 5, 126-157 (1968).

Humphrey, T. J., Reflex activity in the oral and facial area of the human fetus. Chap. 12 in J. F. Bosma (Ed.) *Second Symposium on Oral Sensation and Perception.* Springfield, Ill.: Charles C Thomas (1970).

Moyers, R. E., The infantile swallow. *Trans. Eur. orthodont. Soc.*, 40, 180-187 (1964).

Moyers, R. E., *A Handbook of Orthodontics.* (3rd ed.) Chicago: Yearbook Medical Publishers (1971) (Chap. V).

Thilander, B., Innervation of the temporomandibular disc in man. *Acta odont. scand.*, 22, 151-156 (1964).

# NEUROMUSCULAR DYSFUNCTION AND THE REHABILITATION OF CHILDREN WITH COINCIDENT SPEECH AND OROFACIAL PROBLEMS

RICHARD M. FLOWER

*University of California Medical Center, San Francisco, California*

LUCIE I. LAWSON

*University of California School of Dentistry, San Francisco, California*

Whether necessary or not, we feel the need to qualify our presence among a group of authors who have made notable contributions to the research literature in the fields under discussion in this *Report*. For the past 20 years or so we have confronted clinical problems that relate to these fields and have tried to establish some perspectives about these problems with our students, both in dentistry and in speech pathology. We shall direct our remarks from the basis of these qualifications.

This is the first paper in this *Report* to be presented by speech pathologists. It seems advisable, therefore, to fit it into some broader context. The following areas of mutual clinical concern can be defined with special reference to our work with problems in orofacial growth and development among children.

1. Interrelated dental problems and problems in speech acquisition may occur among children with congenital malformations of the orofacial complex. These problems may be directly related to the malformations or result from attendant maldevelopment or from certain types of surgical intervention.
2. The absence of tissue that may result from these congenital malformations may require prosthetic management which will also influence speech acquisition.
3. The speech of a previously normal speaker may be affected by the removal of teeth or by the traumatic loss or surgical excision of orofacial tissues. These patients may require both prosthetic management and speech reeducation.
4. Malocclusions occurring in children with otherwise normal potential for speech production may, on occasion, create specific interference to the precise articulation of individual speech sounds. Thus, the orthodontist and the speech clinician may be mutually concerned.

5. Dysfunction of the orofacial neuromuscular system may interfere with speech acquisition and speech production. Many dentists believe that it may also interfere with the development of orofacial morphology. Therefore, our professions may be mutually concerned about children with oral neuromuscular problems.

Our paper discusses only this last area of mutual concern—children with neuromuscular dysfunction. We do not minimize the importance of the other areas, but they will be discussed elsewhere in this *Report.* Furthermore, we must discuss this area from our point of view as speech pathologists and leave more detailed discussion of relationships between neuromuscular dysfunction and the development of oral morphology to our colleagues in dentistry.

Arthur Miller, in his play *All My Sons,* has one of his characters ask another why he reads book reviews when he never reads a book. "Because," he answers, "I like to keep abreast of my ignorance." At this moment in history, the most that anyone can do in discussing our topic is to bring us abreast of our ignorance. Or perhaps, stated more positively, to define some of our quandaries.

We will discuss the topic by considering three general questions: (1) How do we define neuromuscular dysfunction as it relates to speech and orofacial problems? (2) What are feasible terminal goals for the rehabilitation of these children? (3) What are the implications of neuromuscular dysfunction on new response learning?

## DEFINING NEUROMUSCULAR DYSFUNCTION

Neuromuscular dysfunction has been implicated as a cause of speech disorders since the earliest works in our clinical literature. Yet, except at the extreme of the continuum where such diagnoses as cerebral palsy are applied, neuromuscular dysfunction remains more of a loosely defined clinical construct than an objectively describable pathologic entity. Less pervasive or less debilitating neuromuscular dysfunction substantially remains in the eye of the beholder. It is probably best considered a manifestation of what MacKeith (1968) calls "neurodevelopmental disorders." In proposing this nomenclature, he wrote, "The term is used not as 'minimal brain damage' and 'minimal brain dysfunction' have too often been used, to put the problem to rest in a pigeonhole, but into a mailing box so that it goes on to a sorting office for intensive study and analysis."

Three general approaches to the clinical diagnosis of neuromuscular dysfunction have been used, either single or in combination. The first approach involves guilt by association. Diagnoses may be based on poor performances on such tests as the Osertsky Tests of Motor Proficiency or the Heath Rail-Walking Test. Or neurologic tests—usually unstandardized—may be used to elicit "soft signs" of central nervous system deficits. Some clinicians even base diagnoses upon tests of visual perception or visual-motor integration.

None of these tests involves the motor or sensory processes directly involved

in speech production. How, then, can they help us understand problems in speech production, let alone problems in tongue control related to problems in dental development?

A second diagnostic approach emphasizes the historical description of the development of such functions as chewing, sucking, and swallowing, and observation of these processes as they are currently accomplished. In this instance the observation centers on muscle groups that participate in speech production. But speech production and these functions, even though they involve the same muscle groups, represent very different processes. Presumed deviations in the development or execution of mastication and deglutition cannot, therefore, be cited to account for deviations in speech production. Or conversely, deviations in speech production cannot be cited to attribute dental maldevelopment to neuromotor problems.

A third diagnostic approach centers on measurement of components of speech-related neuromotor functions. As a measure of motor functioning, diadochokinesis has probably been explored most thoroughly, and tentative age norms have been established (Johnson, Darley, Spriestersbach, 1963, Chap. 5). Currently, however, some workers are questioning the assumption that the component movements in the rapid or alternating repetition of syllables are similar to actual speech production. McDonald (1964) questioned the relevance of measures of diadochokinetic rates on a theoretical basis. Hixon and Hardy (1964) have offered experimental evidence to support the irrelevance of measurements of syllable repetition, at least among children with sufficient neuromotor dysfunction to warrant a diagnosis of cerebral palsy.

Another approach to measuring speech-related neuromotor functions is the assessment of tactile-kinesthetic sensitivity of oral structures by form discrimination and two-point discrimination. Again some standardization of technique has been accomplished and age-expectancies have been described (see Bosma, 1967). There is some disparity in research findings, but these techniques probably distinguish between certain speech defective groups and normal-speaking groups. They may also correlate with measures of severity of articulatory disorders (Ringel, 1970) and predict children's success in articulation learning (Locke, 1968a). In a recent review, however, Hardy (1970) cites evidence from neurophysiologic research suggesting that "a relationship between deficits of somesthetic discrimination and abnormal muscular patterning may not be a direct one." He concludes that "the recent work in mapping the two-point discrimination and stereognostic capabilities of the oral cavity . . . may not have as much implication for detecting and understanding deficits of afferent systems related to sensorimotor function for speech production as might be assumed." It seems, therefore, that the final definition of the validity of tests of oral sensation and perception as measures of speech-related functions is yet to be accomplished.

The obvious answer to the problem of describing neuromotor problems as a basis for deviant speech production seems to lie in the observation of neuromotor processes during actual speech production. But the rapidity, complexity,

and invisibility of speech movements create a host of frustrations to attempts at direct observation and measurement. It is also important to recognize that speech is a complex of learned behaviors. Winitz (1969) reminds us that when particular phonemes and phoneme clusters are present in an individual's spoken language he is more likely to be proficient in the motor behaviors involved in the production of these phonemes and clusters than when they are not present in his phonology.

For the moment we can only say that the quest for approaches to better observation of the neuromotor aspects of speech continues. Two recently suggested approaches deserve special mention. One is the observation, by electromyographic recording, of the lack of stereotypy in the articulation of adults with acquired dysarthria, preliminarily reported by Shankweiler, Harris, and Taylor (1968). The other is the approach to the description of the coordination of successive pressure events in speech production, described by Harvold elsewhere in this *Report*.

In the final analysis, then, our diagnostic criteria for speech-related neuromuscular dysfunction are primarily unstandardized clinical impressions. With full recognition of their woeful inadequacies, we can further timidly advance five clinical formulations to serve as a basis for discussing approaches to rehabilitation:

First, children with neuromuscular dysfunction may be less able to effect the relatively precise relationships among the articulators required for normal speech. Their difficulties in effecting these relationships may be increasingly apparent, or may only become apparent, as the motor demands of speech become increasingly complex. Therefore, wide inconsistencies in articulation may appear, depending upon the over-all rate of speech, upon the phonemic context, or upon other determinants of task complexity.

Second, neuromuscular dysfunction may not only influence the component functions of the speech process, such as controlled expiration of the breath stream, phonation, velopharyngeal valving, and movements of the tongue, lips, and jaw. It may also disturb the temporal coordination of the series of motor events involved in speech production.

Third, neuromuscular dysfunction should not be considered only in terms of output. Impairment of precision in tactile-kinesthetic monitoring may be equally important. Auditory monitoring is a prime factor in the regulation of speech production, particularly during the years of speech learning. But we must also recognize the probable importance of tactile-kinesthetic monitoring.

Fourth, the relevance of neuromuscular dysfunction to speech production cannot be assessed only in terms of its immediate influence. Even more important is the influence of neuromuscular dysfunction at the time the child is learning his repertoire of articulatory skills.

Fifth, neuromuscular dysfunction must be described with reference to the specific process under scrutiny. It is, for example, precarious to relate neuromuscular dysfunction in mastication and deglutition to neuromuscular dysfunction in speech, even though both may be found in the same patient.

## TERMINAL GOALS FOR REHABILITATING CHILDREN WITH NEUROMUSCULAR DYSFUNCTION

If we are to plan a rehabilitation program for any patient, we must first formulate a reasonably precise picture of the terminal goals toward which we are programming. When we read reports of the prevalence of malocclusions ranging from 20 to 70% of the population, we suspect that dentists may be in less than monolithic agreement on what constitutes acceptable dentition. We speech pathologists have done little better, if indeed as well, in arriving at definitions of what constitutes acceptable communication. We have, for example, approached the description of articulatory defectiveness from the premise that absolute criteria of correct vs incorrect can be established. We have then compounded the folly by behaving as though—as Locke (1968b) stated it—"The best measure of severity of misarticulation is the number of defective sounds."

In Winitz's (1969) excellent discussion of phonology, he reminds us that, at least as the term is used by linguists, the phoneme is defined as a unit of language signaling semantic distinctiveness. One criterion for judging the misarticulation of phonemes must then relate to the influence of those misarticulations on the semantic distinctiveness of the words in the patient's spoken language. The only possible additional criteria would emerge from judgments of acceptability of articulation—virtually in aesthetic terms.

As we observed earlier, lacking more precise diagnostic criteria, we are left with the assumption that a neuromuscular deficit is immediately significant to speech rehabilitation only when it reduces the potential for normal speech production. In defining rehabilitation goals, therefore, we usually must assign priorities to effecting changes that will have the greatest influence on the over-all acceptability of the patient's speech. The following suggestions are incomplete but may provide some general approaches to goal formulation.

Very generally, the intelligibility of a particular phoneme is reduced in proportion to the number of distinctive features altered by the misarticulation. To illustrate: If we analyze consonant articulation according to the relatively simple distinctive feature classification suggested by Miller and Nicely (1961), we see that each of the five features is probably ascribable to different motor patterns. The potential for mastery of these features may, therefore, be roughly predictable. Neuromuscular impairment might, for example, preclude consistent, accurate targeting of the place of articulation but might not preclude reasonably accurate use of the affrication feature. In this instance, if the child learns to use the affrication feature correctly, his speech is more likely to be intelligible, even though place features remain deviant.

Misarticulations may be more deleterious to communication when they involve phonemes that determine common grammatical forms. Therefore, a high priority should be assigned to achieving consistent production of approximations of such morphologically crucial phonemes as /s/ and /z/.

Even if many of the phones that characterize a patient's speech do not lie within acceptable phoneme boundaries, a listener is more likely to understand him if he uses these phones consistently. Noll (1970) has expressed this

concept as follows: ". . . if a speaker is fairly consistent in his sound errors, the listener may be able to decode the message easily, once he has learned the speaker's code. If he is randomly inconsistent with his misarticulations, the code is always changing, and thus intelligibility is affected." A tenable goal for rehabilitation may, therefore, be developing consistent use of a child's most nearly acceptable misarticulations.

Speech is primarily a series of acoustic events. Nevertheless, judgments of acceptability may depend heavily upon coincident visual phenomena. For example, a child who accomplishes his version of /s/ by protruding his tongue between his teeth may appear to have much more defective articulation than a child who produces this sound by contact between the lower lip and the upper central incisors, even though the acoustic result no more nearly approximates an acceptable /s/. An important goal for rehabilitation, therefore, may be the achievement of greater visual acceptability during speech production.

This aspect of rehabilitation may involve both of our professions. Research findings on the direct relationships between dentition and the acoustic aspects of speech articulation are equivocal, but the visual aspects of speech production may concern both dentists and speech pathologists.

Obviously, many other factors must be considered, but the four we have mentioned illustrate some possible bases for assigning priorities to goals for the rehabilitation of children with significant neuromuscular dysfunction.

## NEUROMUSCULAR DYSFUNCTION AND NEW RESPONSE LEARNING

If we were to capsulize what clinical speech pathology is all about, we would say that it deals with new response learning. As we overhear the shop talk of our colleagues in dentistry, we suspect that new response learning is an important area of their concern as well. Regardless of a dentist's skill in modifying or restoring structure, his efforts may yield little long-term benefit unless the patient concomitantly learns some new responses. These responses may comprise the components of better dental hygiene, or different patterns of lingual movement, or adaptations to prostheses. Here again is an area where the two professions share some mutual concerns.

Despite the primary involvement of speech pathology in new response learning, our research has traditionally been much more concerned with the measurement of various behaviors than with orderly explorations of approaches to modifying these behaviors. In a recent paper, Shelton (1970) observed:

> Better speech therapy may result from improved understanding of speech production. For example, knowledge about how the articulation of one phone influences the movements used in the production of a neighboring phone . . . may lead to the identification of speech contexts that will facilitate the acquisition of a specific articulatory skill. . . . However, treatment applications will occur only if someone uses the speech production research in the formulation and testing of new treatment. This requires research as careful and complex as that involved in basic research. Basic knowledge by itself will not solve our applied problems. Good clinical work cannot be founded on pronouncements whether delivered by scientists or senior clinicians.

With some notable exceptions, most orderly investigation of speech therapeutic approaches has involved either children with normal speech or children for whom normal speech was a feasible terminal behavior. To discuss the rehabilitation of children with neuromuscular dysfunction, we must again rely substantially on hypothetical formulations based on fragmentary evidence. Yet one fundamental tenet seems important: To our knowledge, no one has yet demonstrated that any widely accepted theory of new response acquisition is invalidated by the existence of central nervous system impairment. In fact, some of the best research on new response acquisition has been conducted with children whose central nervous system deficits are reflected in mental retardation.

We often regard disease-model diagnostic categories as meaningful descriptions of behavior. For example, we discuss "cleft-palate speech," as though the dimensions of the speech behavior are specified by a single underlying disease entity. Yet the patterns of misarticulation found among children with cleft palate vary widely and are often not attributable to disordered orofacial morphology. Similarly, all undesirable speech behaviors found among children with neuromuscular dysfunction may not necessarily be attributable to neuromuscular dysfunction.

Also, recall our earlier assertion that the influence of neuromuscular dysfunction at the time particular speech behaviors are being learned does not necessarily mean that neuromuscular dysfunction will interfere with a child's learning more acceptable speech behaviors at the present time. Sometimes, therefore, neuromuscular problems will be irrelevant in the learning of particular new speech behaviors.

A child's success in acquiring new responses is one of the strongest motivations for his acquisition of other related responses. Too frequently, in teaching speech skills we offer no alternatives between completely successful execution of a normal speech response and a totally unacceptable response. Instead, we should try to analyze acceptable speech responses into component tasks so that a child can direct his attention to these components and be rewarded for mastery of something less than the total task. This is particularly important when eventual mastery of normal speech seems unlikely from the outset.

Much of our therapy for children with speech problems, particularly articulatory problems, seems to be based on a notion Curtis (1968) characterized as regarding speech as a sequence of discrete elements, one following another like beads on a string. We direct therapeutic programs to these discrete elements, assuming that if these elements are learned, this learning will be reflected in total speech production. When a child's speech problem is characterized primarily by "phonetic errors that are the result of incorrectly learned phonemic systems" (Winitz, 1969, p. 125), this approach to therapy may have some validity. In that instance the speech behavior is, as Harris (1970) expresses it, stereotyped, albeit inaccurately stereotyped. On the other hand, if neuromuscular dysfunction is characterized by lack of stereotypy in the coordination of movements, we may not effect significant changes in speech by concentrating

attention at the phoneme level. To our knowledge, McDonald (1964) is the only writer in the field of speech pathology who has devoted extensive consideration to this area.

We have already observed that one of the possible components of neuromuscular dysfunction is impairment in tactile-kinesthetic monitoring of speech. Although it has been demonstrated that tactile-kinesthetic perception improves with age, it has not, to our knowledge, been demonstrated whether improved tactile-kinesthetic discrimination can be learned in the same way that improved visual and auditory discrimination can be learned. It may, in this instance, be important to differentiate, in Galambos' (1967) terms, between neural maturation and neural learning. Winitz (1969), among other authors, has suggested some guidelines for programming activities for the auditory monitoring of speech production. These guidelines are based both on his own work and on research in the field of visual discrimination learning. It is interesting to speculate whether similarly structured programs would lead to comparable refinement of oral tactile-kinesthetic perception. Currently, we do not know whether we should program for improved speech facility despite impaired monitoring, or whether we should attempt to ameliorate the monitoring deficits themselves.

We noted earlier that it is precarious to generalize from neuromuscular dysfunction, as manifested in mastication and deglutition, to speech production or vice versa. It is similarly precarious to assume that teaching new responses for one of these processes will result in new responses in another process. Hixon and Hardy (1964) have offered experimental evidence to support their conclusion that "the neurophysiological mechanisms which evoke speech movements of the articulators may be dissimilar from those which evoke nonspeech movements of the same structures."

In 1965, Subtelny wrote, ". . . to date it has not been conclusively shown by controlled research studies that re-education of swallowing activity will, by itself, effect an appreciable improvement in malocclusion and/or defective speech production." We know of no subsequent research that would seriously challenge this conclusion.

We will leave to others the question of whether or not programs for learning new responses in mastication and deglutition should be conducted. If such programs are pursued, however, we must conclude at the moment that they are essentially unrelated to programs designed for learning new speech responses.

## SUMMARY

In summary, we have attempted to offer glimpses of our ignorance in three different areas related to neuromuscular dysfunction and speech problems in children: diagnosis, identifying goals for rehabilitation, and the implications of neuromuscular dysfunction for new response learning. For the purposes of this *Report*, it may seem that we have placed too little emphasis on the relationships between dental and speech problems. This is only partially attributable

to our even greater ignorance of the relationship between neuromuscular and dental deficits. We also maintain basic questions regarding some assumptions about the interrelatedness of our concerns for the children who present these problems.

In some respects the relationship between dentistry and speech pathology has been like a summer romance, effected on the basis of superficial glimpses of compatability. If it is to ripen into anything more lasting, we must find some more binding ties.

From a clinical standpoint, we are convinced that no further reason is needed for cooperation than that speech pathologists and dentists often work for the rehabilitation of the same human beings. To justify our relationship we do not need to demonstrate that the speech clinician's efforts will contribute to the ultimate achievement of better orofacial morphology. Nor need we demonstrate that better speech will be effected by improved dental occlusion. When the final evidence is in, we may find that either or both of these hypotheses are true. Even if they are not, it is quite enough to believe that the highest level of cooperation among all concerned professions is always in the best interest of patients who require our assistance. This obtains regardless of the immediate effects of the efforts of one profession on the efforts of another.

## REFERENCES


Bosma, J. (Ed.), *Symposium on Oral Sensation and Perception.* Springfield, Ill.: Charles C Thomas (1967).

Curtis, J., Conceptual models of speech production and the description of articulation. Paper presented at Annual Convention of the American Speech and Hearing Association, Denver (1968).

Galambos, R., Brain correlates of learning. In G. Quarton et al. (Eds.), *The Neurosciences.* New York: Rockefeller Univ. Press (1967).

Hardy, J. C., Development of neuromuscular systems underlying speech production. *Speech and the Dentofacial Complex: The State of the Art. ASHA Reports Number 5,* 49-68 (1970).

Harris, K., Physiological measures of speech movements: EMG and fiberoptic studies. *Speech and the Dentofacial Complex: The State of the Art. ASHA Reports Number 5,* 271-282 (1970).

Hixon, T., and Hardy, J., Restricted motility of the speech articulators in cerebral palsy. *J. Speech Hearing Dis.,* **29,** 293-306 (1964).

Johnson, W., Darley, F., and Spriestersbach, D., *Diagnostic Methods in Speech Pathology.* New York: Harper and Row (1963).

Locke, J., Oral perception and articulation learning. *Percept. mot. Skills,* **26,** 1259-1264 (1968a).

Locke, J., Questionable assumptions underlying articulation research. *J. Speech Hearing Dis.,* **33,** 112-116 (1968b).

MacKeith, R., Maximal clarity on neurodevelopmental disorders. *Develpm. Med. child Neurol.,* **12,** 143-144 (1968).

McDonald, E., *Articulation Testing and Treatment: A Sensory-Motor Approach.* Pittsburgh: Stanwix House (1964).

Miller, G., and Nicely, P., An analysis of perceptual confusions among some English consonants. In S. Saporta (Ed.), *Psycholinguistics: A Book of Readings.* New York: Holt, Rinehart, and Winston (1961).

Noll, J. D., Articulatory assessment. *Speech and the Dentofacial Complex: The State of the Art. ASHA Reports Number 5,* 283-298 (1970).

RINGEL, R. L., Oral sensation and perception: A selective review. *Speech and the Dentofacial Complex: The State of the Art. ASHA Reports Number 5,* 188-206 (1970).
SHANKWEILER, D., HARRIS, K., and TAYLOR, M., Electromyographic studies of articulation and aphasia. *Arch. phys. Med. Rehab.,* 49, 1-8 (1968).
SHELTON, R. L., Modification of the oral-facial function during speech. *Speech and the Dentofacial Complex: The State of the Art. ASHA Reports Number 5,* 146-164 (1970).
SUBTELNY, JOANNE D., Examination of current philosophies associated with swallowing behavior. *Amer. J. Orthodont.,* 51, 161-182 (1965).
WINITZ, H., *Articulation and Behavior.* New York: Appleton-Century-Crofts (1969).

# SKELETAL AND DENTAL IRREGULARITIES IN RELATION TO NEUROMUSCULAR DYSFUNCTIONS

EGIL P. HARVOLD

*University of California School of Dentistry, San Francisco, California*

Conflicts of interest may readily develop among professional groups dedicated to specific subjects when their magic circles overlap. It is surprising, therefore, that the two professions, speech science and dentistry, both searching in the close quarters of the oral cavity, have barely noticed the presence of each other. Apparently they have been focusing in opposite directions. Looking around certainly will widen the horizon for both groups, while looking together may be stimulating. Both identify spheres of interest within the same anatomical and physiological boundaries. Both serve the same section of the population. The problems puzzling them deserve attention for many practical reasons, in addition to the inherent intellectual challenge.

It is my task to provide a brief overview of certain problems confronting dentistry, problems which we may solve more expeditiously if we can work in conjunction with the speech scientists. Dentistry is laboring on two fronts. The first front is the combatting of diseases manifested in oral symptoms, of which caries and periodontal problems are the most frequent. Also involved are soft tissue lesions and bone diseases which, though occurring less frequently, are not less complicated. The second front is aimed at maldevelopments and dysfunctions ranging from dental malocclusions to congenital malformations. It is on this second sphere of action, maldevelopment and malfunction, that speech science and odontology may join forces.

Dental malocclusion is a prevalent malady in the total spectrum of all dental irregularities, and in our culture it is viewed as a significant impairment. The paucity of knowledge regarding the causes of malpositioned teeth presents a significant problem to dentists. The etiologies of only a few categories of irregular tooth positions are clearly delineated; for example, finger sucking and lip biting. Otherwise, causation is hazy in the majority of dental malocclusions.

The prevailing concept in the dental profession holds that the facial skeleton and its neuromuscular structures are strongly controlled by genetic factors and not readily influenced by any therapeutic measures. According to this school of thought, only tooth position and alveolar bone respond readily to environmental stimuli. This concept is not a flimsy one. It is based on a long

series of research projects. Among the more important are the longitudinal cephalometric studies by Broadbent (1937), Brodie (1941), Meredith (1954), and Bjork (1947). These investigations unanimously indicate that individual growth patterns of the facial skeleton follow rather narrow channels and make few unexpected deviations. Clinical observations by orthodontists seem to support this concept of developmental stability, since conventional correction of dental malocclusions apparently has little effect on the anatomical structures outside the dental arches.

The tenuousness of this concept is obvious, as it is based on data obtained from cephalometric radiographs and plaster models, neither of which yields information on function. The possible interrelationship between function or behavior and structural development is not considered in the classical cephalometric studies, and it is therefore not recognized. Parenthetically, this results not from ignorance but from lack of proper research methods. While some orthodontists may assume that only the dental alveolar structures can be altered by conventional orthodontic treatment, this notion is open for discussion. In his early electromyographic studies, Moyers (1949) was the first to provide scientific evidence of more fundamental effects.

However, still undeveloped are the instrumentation and methods which will permit a more meaningful examination of the relationship between muscle function, skeleton, and dentition. We can study the skeleton and the dentition with x rays and plaster models. We can derive some observations on neuromuscular structures and activities from electromyography. However, we cannot synthesize the information. My assumption is that, with appropriate tools and methods which allow the recording and analysis of the interplay between neuromuscular activity and skeletal morphogenesis, eventually an understanding of the etiology of malocclusion will emerge. Then we can prevent some malocclusions and correct others more efficiently.

My optimism is inherited, I suppose; but it is also based on certain observations of coordinated animal experiments and clinical studies, which will be mentioned briefly. Numerous publications over the last century have indicated that mouth breathing, with the mandible maintained in a low postural position, influences the shape of the jaws and frequently leads to malocclusion (Linder-Aronson, 1970). The underlying hypothesis holds that the neuromuscular system subserving respiration has some control over the position of the mandible relative to the maxilla and hence influences both skeletal morphogenesis and tooth position (Harvold, 1960, 1968).

The hypothesis has been tested in animal studies with the rhesus monkey as an experimental model. It was demonstrated that if a normal animal lowers the position of the mandible relative to the maxilla for a period of six to ten months, then the shape of the mandible undergoes predictable changes. A more open gonial angle and a steeper inclination of the lower border are produced (Figure 1). Furthermore, when the distance between the maxilla and the mandible is increased, the muscular environment surrounding the dentition changes and malocclusion can develop. The type of dental malocclusion depends on a series

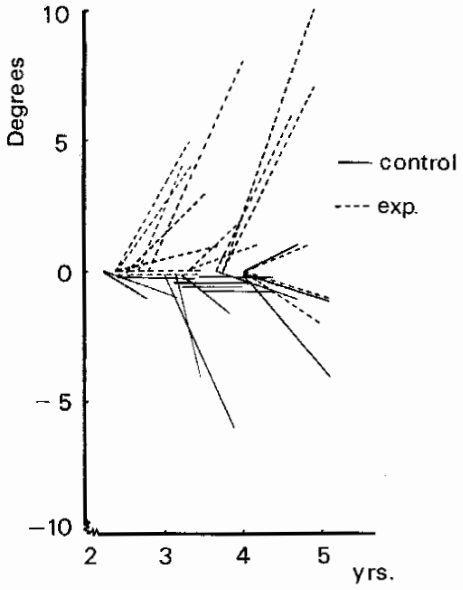



FIGURE 1. The lines on the diagram show the ages of the animals at the beginning of the experiment and at the end of the observation periods. The lines also indicate the changes in gonial angle during the experiment. An alteration in mandibular form is associated with a long-term change in the mandibular position relative to the mandible. The ages of the animals were established on the basis of dental development.

of factors, such as the new posture and activity of the tongue, cheeks, and lips, as well as the movements of the mandible. In these pilot studies with the rhesus monkey (Dawson, 1968; Harvold, 1968), various malocclusions have been produced which are similar to those found in the human who has that particular type of mandible (Figure 2). However, we do not yet have sufficient animals in the various categories to justify further statements.

In a parallel investigation, we are testing the hypothesis in reverse. Children with malocclusions involving an open gonial angle and a steep lower mandibular border are treated with appliances designed to strengthen the muscles elevating the chin, particularly the facial muscle complex with the orbicularis oris and the buccinators. Half of the children in this study serve as controls for one year before entering the treatment group. The exercise appliances are used mainly at night, and they are not designed to move any particular teeth. Yet the response to these exercises is as striking as is the response in the experimental animals (Figures 3, 4).

At this point one realizes that missing in this work are the tools and methods which permit recording and study of neuromuscular activity in relation to skeletal morphogenesis and morphology as well as to speech and behavior. Available in the literature and in the laboratories are vast quantities of information regarding the central nervous system, bone, muscles, correlative neurophysiology, and functions such as respiration, mastication, and speech. The presentations in this *Report* have sifted out some central problems. The situa-

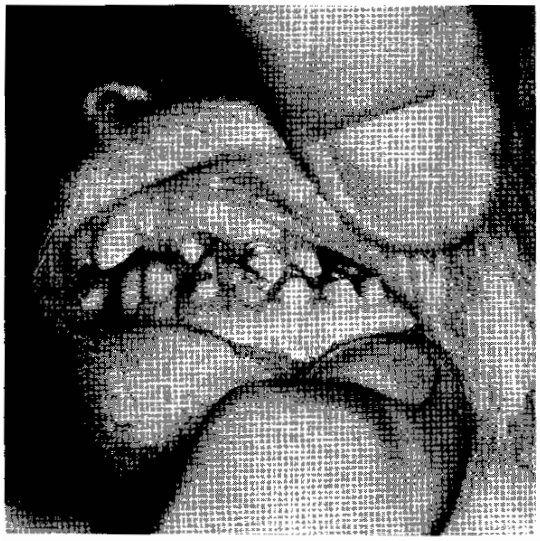


FIGURE 2. Rhesus monkeys with normal dental occlusion develop malocclusions when jaw relations are altered and the teeth subjected to deviant muscle function. This animal developed a Class II malocclusion in less than a year.

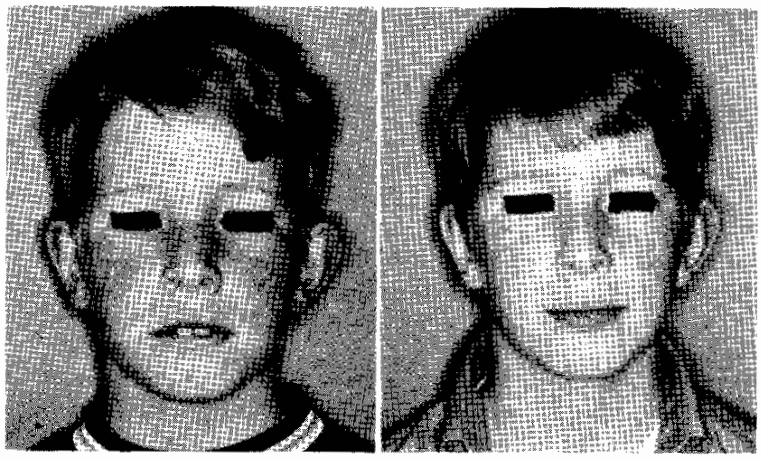


FIGURE 3. The change in facial appearance after nine months of treatment with an activator appliance. The use of orthodontic devices which affect the facial musculature may also alter the dentition.

tion is reminiscent of the story of the young, healthy, and happy California rancher who was sitting in the sunshine gazing at his green acres and his sturdy cattle, when a county adviser on agricultural problems arrived. The "expert" recognized room for improvement on the ranch and asked the young proprietor, "Would you like to take some courses on new farming methods?" The young rancher looked at him and replied, "No. I know so much already I don't use half of it."

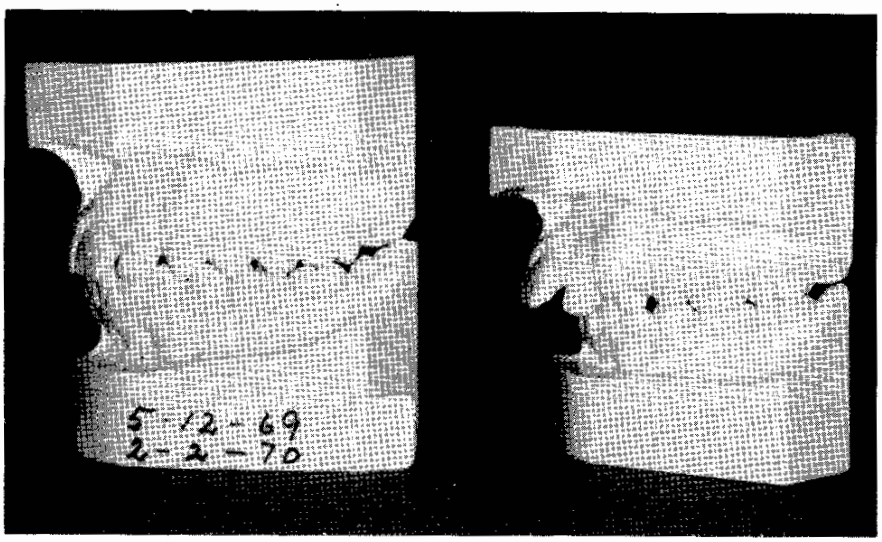



FIGURE 4. Tooth position changed during the ninth month of treatment with an activator appliance designed to strengthen the facial muscles. Similar changes were not observed in the control group in this clinical study.

I do not know what fraction of our knowledge we can apply in speech and dentistry, but we certainly need more differentiating information. We may compare the neural network serving the orofacial region to the telephone system servicing the states. In the clinical sciences we want to know how, when, and where those calls are made which influence the life and welfare of the various communities. The details are, of course, important: the number of stations, the shape and color of the telephones, the thickness and length of the wires, quality of insulation, reliability of connections, signal coding, and transmission. This information is basic and is accumulating more rapidly than we can read it.

In our fields we need a "bugging" system to watch the action and separate the meaningful communication from the meaningless, relative to morphogenesis, speech, and language development. In searching for such knowledge at the clinical level, speech scientists and dentists find themselves putting needles, wires, transducers, and microphones into the same muscles and cavities of the head. Sometimes they are surprised to find the other party engaged in staking the same ground.

The orofacial, pharyngeal, and laryngeal structures subserve respiration, mastication, speech, and facial expression. The motor control systems underlying these functions are highly integrated, but with specialized areas and centers subject to complex priority regulations. These priorities must be unraveled before many significant problems can be solved. The neuromuscular activity underlying these functions is not readily available for observation and analysis because of the irregularity of onset and the voluntary influence on performance.

It is not comparable to the activity of the heart, with a built-in monitor and reflexes producing a stable rhythm which exposes the responses to the stimuli. The only oral function with some periodicity, which is the prerequisite for pattern recognition and analysis, is swallowing. It is not surprising, therefore, that pediatrician Bosma (1937), dentist Kaires (1957), otolaryngologist Kawasaki and his colleagues (Kawasaki, Ozura, and Takenouchi, 1964), neurologist Kawamura (1968), speech pathologist Fletcher (elsewhere in this *Report*), and many others have entered the arena through the subject of deglutition.

The soft palate, with its participation in velopharyngeal constriction and closure, is another neuromuscular mechanism which has been subjected to concerted research. The palate apparently participates in deglutition, respiration, vocalization, and speech articulation. With relatively little difficulty one may elicit a predictable velopharyngeal constriction as part of the speech function and study the muscles involved. Therefore, the explorers from medicine, speech science, and dentistry congregate at the soft palate and scrutinize the speech mechanism, disregarding momentarily the possible involvement of other mechanisms. I must include myself in this category.

There is no need to proceed further on this subject, since the various orofacial functions—respiration, mastication, deglutition, speech—are discussed in detail elsewhere in this *Report*. I may turn, then, to a specific objective which, in my opinion, is crucial for dentistry and which may be achieved more readily when pursued in cooperation with speech scientists and other professional groups, namely, the development of adequate methods for assessment of orofacial functions. This is concerned directly with the neuromuscular activity and not with subjective criteria, such as appearance of the face and dentition and the phonetics of speech.

Growth and development are slow processes at the macroscopic level which we are examining. Data on neuromuscular activity are relevant to the problem of growth if they demonstrate a comparable stability and rate of change. Only "patterns" of function and behavior may have such stability. It seems imperative, therefore, to develop techniques by which functional patterns can be recognized, recorded, and related to each other as well as to the morphologic changes in the subserving structures.

The identification of functional models may enable us to study independence and interaction among the various neuromuscular functions, such as speech and mastication, or speech and facial expression. The study of functional patterns may also provide information on the relation between speech and dental malocclusion. These morphologic deviations not only coexist with, but may also sustain or even cause, speech disorders (Bloomer, 1957). Dental abnormalities probably place excessive demands on the neuromuscular system ordinarily serving speech articulation.

Several exploratory studies have been undertaken to test the feasibility of such an approach. Certain of the observations may be mentioned. The parameters for these investigations are time and signal amplitude. The time measurements provide information regarding onset and duration of muscle activity,

which in turn exposes the sequence of actions in the various muscles. If the action can be repeated, then the pattern can be recognized and its stability determined. This is an aspect of motor skill which can be related to growth and development. The neuromuscular system serving respiration has a rhythmic function, and can be monitored to perform with a relatively stable pattern during the experiments. The modifying influence of respiration on other functional patterns is therefore accessible for study. In the speech system the conditions are indeed complex and require clarification in many dimensions. The first question becomes: Is it possible to monitor a rhythmic function in the speech system without engaging the masticatory mechanism and the system underlying facial expression?

Our experiments have been based on the assumption that a relatively simple function of the speech mechanism would be a relaxed repetition of short, familiar syllables without any attempt to communicate or be understood. A selection of syllables serves to provide information on proficiency of sequential action in the various muscle groups. We have found that recordings of the functional pattern underlying a syllable are reproducible, with a statisfactory level of reliability, at intervals of several weeks. We have also established that the rhythmic inspiration has a distinct influence on syllable patterns. Furthermore, several functional skills appear to develop with age, while others suffer when there are congenital malformations, neurologic disorders, and anatomical deviations such as dental malocclusions.

These experiments indicate that it is possible to provide new information on some of dentistry's problems regarding growth and development. However, the investigations also demonstrate that the problems of neuromuscular activity in the orofacial region are influenced by many factors which are not readily accessible to recording of valid data; for example, intensity of expression, attempts to articulate with precision, and the subject's awareness of the testing procedure. The parameters for assessment of these factors are expressed in signal amplitude.

In short, these observations focus our attention on the significance of threshold levels, interaction, and feedback systems. It is at this junction that the need for cooperation with other professionals, contributing their special knowledge, becomes an urgency.

## REFERENCES


BJORK, A., The face in profile. *Svensk Tandlak Tidskr.*, 40 sup. (1947).

BLOOMER, H. H., Speech defects associated with dental abnormalities and malocclusion. In L. E. Travis (Ed.), *Handbook of Speech Pathology*. New York: Appleton-Century-Crofts, pp. 608-652 (1957).

BOSMA, J. F., Deglutition: Pharyngeal stage. *Physiol. Rev.*, 37, 275-300 (1937).

BROADBENT, H. B., The face of the normal child. *Angle Orthodont.*, 7, 183-208 (1937).

BRODIE, H. G., On the growth pattern of the human head from the third month to the eighth year of life. *Amer. J. Anat.*, 68, 209-262 (1941).

DAWSON, W. J., A study of the effects of tongue depression and resultant mandibular postural change on the growth and development of the orofacial complex of rhesus monkeys. Thesis submitted as partial fulfillment for certification by the American Board of Orthodontics. University of California San Francisco Library (1968).

HARVOLD, E. P., The role of function in the etiology and treatment of malocclusion. *Amer. J. Orthodont.*, **54**, 883-898 (1968).
HARVOLD, E. P., Mandibular morphogenesis. (Abstract) *Amer. J. Orthodont.*, **46**, 144 (1960).
KAIRES, A. K., Palatal pressures of the tongue in phonetics and deglutition. *J. pros. Dent.*, **7**, 305-317 (1957).
KAWAMURA, Y., Recent concepts of the physiology of mastication. *Advances in Oral Physiology*. Osaka, Japan: Osaka University, pp. 77-109 (1968).
KAWASAKI, M., OZURA, J. H., and TAKENOUCHI, S., Neurophysiologic observations of normal deglutition. I. Its relationship to the respiratory cycle. *Laryngoscope*, **74**, 1747-1765 (1964).
LINDER-ARONSON, S., Adenoids. *Acta oto-laryng.* Suppl., 265 (1970).
MEREDITH, H. V., Growth in bizygomatic face breadth during childhood. *Growth*, **18**, 111-134 (1954).
MOYERS, R. E., Temporomandibular muscle contraction patterns in Angle Class II, Division I malocclusions: An electromyographic analysis. *Amer. J. Orthodont.*, **35**, 853-857 (1949).

# DEGLUTITION

SAMUEL G. FLETCHER

*University of Alabama in Birmingham, Birmingham, Alabama*

An attempt will be made here to draw together the diverse material that has been published relating to deglutition and to present it in a theoretical framework so that it can be examined and evaluated more readily.

First, four theories of normal deglutition which seem to emerge from the multitudinous subjective and objective reports will be considered. Information and conjectures concerning a particular pattern of oral physiology most typically called "tongue thrust" will then be reviewed. Finally, an attempt will be made to relate findings about tongue thrust to a theory of maturation.

Interest in deglutition has been extensive, especially during the last century. A rather large proportion of the discussions of this topic by various writers consists of unverified personal observations and speculations. Nevertheless, a thread of continuity for four major lines of thought may be recognized. These lines of thought were presented in the form of the following contrasting theories by Wildman, Fletcher, and Cox (1964): (1) the theory of constant propulsion, (2) the theory of oral expulsion, (3) the theory of negative pressure, and (4) the theory of integral function.

The theory of constant propulsion emerged from Magendie's (1816) descriptions of the swallowing act. His beliefs were drawn from anatomical dissections supplemented by animal experiments and have survived as a basic, classical conceptualization of these phenomena. The theory of constant propulsion described the passage of the food bolus through the upper digestive tract in three stages: the oral stage, the pharyngeal stage, and the esophageal stage. During the oral stage, the food bolus is formed and transported under voluntary control to the pharynx. Upon receipt of the bolus, the pharynx was postulated to be activated and, thereafter, to propel the food to the esophagus. The esophagus then transported the food to the stomach. Magendie viewed each stage in this process as essentially independent in action, although he recognized rather close synchrony as essential in order to maintain propulsion of the bolus through the different parts of the tract.

The next major expansion in theories relevant to swallowing patterning came with the work of Kronecker and Melzer in the late 1800s. They conducted a series of very ingenious experiments on themselves and on dogs in an attempt

to establish some time relationships during the swallowing act. Their basic approach was to use balloons attached to a string at one end. The string was then used to activate a kymograph and to record the time intervals. Using themselves as subjects, they observed the time from the onset of the swallow to the balloon's arrival at the stomach. On the dogs, they opened the channel at different points along the tract and attempted to document time factors for transport within different segments of the tract. They found the time lapses to be so brief (approximately 0.1 sec for the total tract) that they concluded that the expelled bolus was literally thrown from the mouth to the stomach and that the intervening channels served only to provide a passageway and perhaps a final squeezing action to disgorge the remnants which fell short of the mark. This theory is labeled the *theory of oral expulsion*, since primary attention is given to the powerful expulsive force generated by the tongue and mylohyoid muscles.

A third major theory was advanced in the 1930s by a British radiologist, A. E. Barclay. During fluoroscopic examination, Barclay observed what appeared to be a "period of radiolucency" in the pharynx just before the actual movement of the bolus was initiated and at a time when all of the orofices of possible exit appeared to be closed. He also noted that, simultaneously, the laryngeal pharynx and the esophagus appeared to be elevated and dilated momentarily, then dropped. His interpretation of these phenomena was that the tongue was brought forward to create a negative pressure and that this pressure was accentuated by the drop of the larynx, so that the food was literally sucked from the mouth to the esophagus.

The theory of negative pressure was discussed in detail and essentially refuted by Saunders, Davis, and Miller in 1951 on the basis of cinefluorographic observations. In 1957, Atkinson et al. conducted a crucial experiment which provided rather conclusive evidence that Barclay's interpretation of his observations was in error. They placed small pressure transducers in the pharynx and found double-peaked positive pressure fluctuations. At no time did they report pressure differentials below the resting levels.

The theory of integral function is the final of the four theories of deglutition and will be discussed in detail here. The orientation of this theory is drawn from recent cinefluorographic, electromyographic, and myometric observations, and places special emphasis on the highly integrated, synergistic coordination of swallowing as a total, dynamic process. Although it is segmented for discussion purposes, the theory stresses a highly refined, integrated, total physiologic act.

For convenience, the theory of integral function in deglutition will be discussed in four phases, although it should be kept clearly in mind that there are no clear demarcations between these phases. That is, the total activity is set into motion, and no boundaries separate the phases other than the anatomical designations of the various channels through which the bolus passes. Thus, all phases are activated with the onset of the act. The act is discussed according to each successive area where the most prominent changes are occurring.

*Preparatory Swallow*

The first phase of the integrated swallow is signalled by the collection of the food and the formation of a more or less compact bolus on the dorsal surface of the tongue. This is initiation of the preparatory swallow and begins immediately after liquids are taken into the mouth, or as soon as solid foods are suitably masticated. The bolus thus formed is moved into a characteristic position on the posterior dorsum of the tongue, and a complete peripheral seal is established around it by the lips and teeth anteriorly, the tongue and tensor-depressed soft palate posteriorly, the hard palate superiorly, and the buccal teeth and adjacent mucosae laterally. Bosma and Fletcher (1962) have described a second site of bolus accumulation in the infant, between the base of the tongue and the epiglottis.

Positioning of a liquid on the dorsum of the tongue before transporting it to the final swallow-preparatory position may be facilitated through suction created by moving the tongue posteriorly after a peripheral seal has been established within the cavity.

The proportion of a mouthful of food to be swallowed at one time is apparently somewhat constant. Jones and Work (1961) measured the volume of water consumed per swallow by adults and by young children. They reported an average of 21.3 cc per swallow by men, 13.6 per swallow by women, and 4.6 cc per swallow by children in the 1¼ to 3½ years age range. They also found a relatively constant ratio of 0.27 cc per kilogram of body weight.

A final characteristic of the preparatory swallow is the stabilization of the oral cavity. Proffit et al. (1964) called attention to pressure of "considerable magnitude" exerted between the teeth in the molar region as the tongue tip was elevated to position the bolus in adult subjects. Whether or not a similar condition exists in young persons has not been established. It seems that in predentition or mixed dentition other mechanisms would need to be utilized for this purpose.

*Oral Phase of Deglutition*

The oral phase of swallowing may be detected slightly before positioning of the bolus is completed in the preparatory swallow. This phase is introduced by withdrawal of the palate from its position against the root of the tongue, where it has been held by the tensor veli palatini muscles (Fletcher, 1958). A simultaneous drop and posterior movement of the tongue and vertical elevation of the hyoid bone and larynx further open the channel for passage of the bolus (Shelton, Bosma, and Sheets, 1960). The elevation of the hyoid may actually be initiated as the bolus is positioned in preparatory swallow, according to Ardran and Kemp (1954). A short respiratory pause also occurs as the food is released from the mouth. This pause extends to the esophageal phase, and evidences fine neurologic coordination between respiration and deglutition (Kawasaki and Ogura, 1968).

As soon as the posterior seal around the food bolus is broken by separation

of the tongue and palate, an anterior-to-posterior rippling motion is activated in the tongue at its contact point with the hard palate. The result of this action is to discharge the food from the mouth, much as toothpaste is squeezed from a tube (Ardran and Kemp, 1954). The toothpaste analogy is appropriate only in descriptions of swallowing a solid bolus. The force of gravity and the release of linguapalatal pressure cause the bulk of a liquid bolus to flow ahead of the tongue and later pharyngeal constrictions. A momentary pooling of the bolus is evident as the bolus arrives at the upper and lower esophageal sphincters, regardless of the consistency of the material swallowed. Ramsey et al. (1955) termed the progressive tongue-palate contact and the later tongue-pharynx contact a *stripping wave*, thereby emphasizing its function as a final clearance activity rather than the primary driving force.

When a large bolus is to be swallowed, most or all of it is moved into the swallow-preparatory position, then neatly sectioned by the tongue in consecutive swallows until the oral cavity is emptied of its contents (Ardran and Kemp, 1955).

This interpretation contrasts rather sharply with that postulated earlier by Kronecker and Meltzer, who did not have the advantages of cinefluorographic observation.

Kydd and Toda (1962) and Proffit et al. (1964) found that during the oral phase of swallowing, lingual pressures were almost identical around the anterior and lateral edges of the perimeter of the oral cavity. Kydd and Toda noted that pressure in the center of the palatal vault reached only about 60% of that at the peripheries. In later observations, Proffit and Norton (1970) reported that pressures exerted at the lateral margins of tongue-palate contact are somewhat higher than those at the anterior margin. Nevertheless, the observations from each of these studies of oral pressure are consistent with the concept of the establishment and maintenance of an anterior and lateral seal during swallowing.

External postural stability during the oral phase of the mature swallow is provided by active contraction of the muscles of mastication (Kawamura, 1961; Sheppard et al., 1959).

*Pharyngeal Phase of Swallowing*

The pharyngeal phase of deglutition is begun as the bolus is released from the tongue and thereafter passes through the fauces. Active participation of the pharynx is elicited by palate and bolus contact with the pharyngeal wall (Fletcher, 1958; Sumi, 1964). This action consists of an elevation of the entire pharyngeal tube and a sphincteric reduction in the lumen between the upper pharyngeal wall and the soft palate (Bosma, 1957). A ridge of Passavant may or may not be present to augment this valvular closure (Fletcher, 1957).

Closure of the palatopharyngeal portal and entrance of the bolus into the pharynx is followed shortly by a synergistic, peristaltic-like tongue-pharynx stripping wave that progressively propels the remnants of the bolus from the pharynx. There is some controversy about whether the tongue or the pharyngeal

wall is the major contributor to the propelling force developed. Ramsey et al. (1955) pointed out that attempts to resolve this question are rather irrelevant, since the actions of both structures are plainly complementary.

Passage of the bolus through the pharynx during mature swallowing is enhanced by an anterior movement of the hyoid bone and the root of the tongue (Shelton, Bosma, and Sheets, 1960). Shelton, Bosma, and Sheets described an alternative pattern in the swallow pattern of some people, an oblique upward and forward movement of the hyoid. This variation apparently reflects different ways in which the bolus is being handled. Ramsey et al. (1955) reported that when one subject swallowed a large bolus, the hyoid bone moved in the oblique upward and forward pattern; whereas when he swallowed a small bolus, the hyoid motion pattern was first upward, then forward. Cinefluorographic observation of swallowing by infants displays yet a third pattern. In infantile swallowing, the first vector is a strong forward thrust. The second vector is a vertical rise toward the end of the act as the bolus passes through the pharynx.

Bosma (1957) described an abrupt final elevation of the larynx as the bolus reaches the hypopharynx. This rise is followed by elevation of the floor of the hypopharynx and opening of the esophageal sphincter (Negus, 1948).

### *Esophageal Phase of Swallowing*

The esophageal phase of swallowing is introduced by passage of the bolus through the cricopharyngeal sphincter. True peristaltic movements then move the bolus through the upper part of the passageway. The peristaltic wave may be replaced as the propulsive force in the distal parts of the esophagus by simultaneous contraction along lower segments (Vantrappan and Hellemans, 1967). During the esophageal phase of deglutition the tongue, palate, and hyoid bone return to their preswallow positions.

The total processes of deglutition encompass a highly coordinated and complex system in which all phases are integrated with few points of clear demarcation.

## THE TONGUE-THRUST PATTERN OF ORAL ACTIVITY

### *General Description*

In recent years considerable attention has been focused on a pattern of oral activity which includes the following attributes: minimal contraction of the muscles of mastication during swallowing (Rix, 1946 and 1948; Tulley, 1953); strong contraction of the perioral musculature (Rix, 1946 and 1948; Tulley, 1953); a thrusting movement of the tongue against or between the incisal dentition (Neff and Kydd, 1966); movement of the hyoid bone in the oblique or in the infantile pattern (Shelton, Haskins, and Bosma, 1959); and distortion of the linguapalatal speech sounds, especially the sibilants (Fletcher, Casteel,

and Bradley, 1961). The criterion characteristic of this pattern is the anterior directional orientation of lingual movements, hence its label of tongue thrust. Other terms such as *perverted swallow, orofacial muscle imbalance, infantile swallow,* and *reversed swallow* have been suggested from time to time but have not gained general usage, because they reflect specific and restrictive interpretations concerning the presence and etiology of the pattern as a pathological condition. The term *tongue thrust* is a descriptive rather than an etiologic or prescriptive term and seems to have survived by default—no more acceptable scientific term has been proposed which maintains the neutral connotation.

Perhaps the most significant statement which could be made about the so-called tongue-thrust pattern of oral activity at this time is that no single characteristic of the pattern is constant. Neff and Kydd (1966) have shown that persons who are legitimately classified as tongue-thrusters may bring the teeth into occlusion during swallowing. Leech (1958) and Subtelny (1965) question the validity of unusual contraction of the perioral musculature as a sign of tongue-thrust swallowing. Fletcher, Casteel, and Bradley (1961) and Subtelny, Mestre, and Subtelny (1964) have shown that many children who may demonstrate a tongue-thrust pattern of swallowing do not have associated speech errors. Writers such as Rix (1946) and Straub (1951) stress the deleterious effects of this "abnormal" swallowing pattern on dental occlusion; however Cleall (1965) has observed that a thrust of the tongue tip between the incisors, and lack of contact between the molars, can be shown in subjects with normal dental occlusion. This variability led Scott (1961) to conclude that, insofar as dental occlusion was concerned, the pattern of oral activity is somewhat irrelevant since a basic cause-and-effect relationship between intraoral and perioral muscular forces and arch form has not been established. This last position was recently strongly attacked by Jacobs (1969), who observed that, when any sum of dynamic movement forces, static isometric forces, and passive tonic forces are not balanced by equal opposing forces, "even minor muscular forces of such low values as 1.68 g . . . [are] . . . capable of moving teeth."

Cleall (1965) suggested that describing various patterns of oral activity as "normal" or "abnormal" has hindered development of an accurate concept of the relationships between soft tissue behavior, skeletodental configuration, and the adaptive capabilities of the stomognathic system. Attempting to be accurate in describing swallowing phenomena along the total continuum seems to be more critical at this time than attempting to dichotomize the behavior into the two exclusive categories of normal or abnormal.

In spite of the inconsistencies and unanswered questions noted, most clinical and experimental evidence continues to suggest that the tongue-thrust pattern of oral activity is likely a meaningful cluster of behavioral signs. As such, it is worthy of continued scrutiny to determine more precisely its specific characteristics and possible place in the hierarchy of oral physiology.

### *Etiology of Tongue Thrusting*

Another point of considerable controversy with respect to the tongue-

thrust pattern of oral activity has to do with etiology. Speculation about etiology may be summarized under the six alphabetically arranged headings. The references indicated in this section are not meant to indicate that the particular person cited believes only in that particular explanation, nor that the ones cited are the only proponents of that particular viewpoint. Rather, they are included to document the fact that one writer has indicated in a formal publication the etiological explanation described. A further observation is that the establishment of all of these possible etiologic factors has been after the fact. So far as is known, no one has attempted to manipulate them in a person who does not have a tongue-thrusting pattern of movement, then study the outcome. That is, no one has demonstrated the capability of both generating and modifying the pattern by manipulating these factors which have been labeled as predisposing for tongue thrust.

Genetic

1. Inherited variations in orofacial morphology which precipitate a tongue-thrust motion pattern (Ballard and Bond, 1960; Gwynne-Evans, 1956).
2. Inherited orbicularis oris hypertony resulting from specific anatomical configuration and neuromuscular interplay and generating a tongue-thrust pattern of motion (Cauhépé, 1955).
3. Genetically predetermined pattern of mouth behavior (Ballard, 1959).

Learned Behavior (Habit)

1. Improper bottle feeding which results in "abnormal" functional patterns of lingual movement in the form of tongue thrust (Straub, 1951).
2. Protracted period of tenderness or soreness of gum tissue and teeth that keeps the teeth apart during swallowing and thereby changes the swallowing pattern (Truesdell and Truesdell, 1937-1938).
3. Prolonged thumb sucking with the habitual movements generalized to tongue activity (Teuscher, 1940).
4. Tongue held in open spaces during mixed dentition and extension and habituation of such postures into other mobile activities of the tongue (Rogers, 1927).
5. Prolonged tonsillar and other upper respiratory infections which cause adaptive patterns in tongue movements, which are retained after the infection subsides (Moyers, 1958, pp. 118-119).

Maturation

1. Tongue thrust present as part of normal childhood oral behavioral pattern which is gradually modified as the lingual space and suspensory system change (Fletcher, Casteel, and Bradley, 1961; Wildman, Fletcher, and Cox, 1964).
2. Tongue-thrust pattern as evidence of late maturation from infantile suckle-swallow (Rix, 1953).

3. Late maturation from or retention of immature patterns of general oral behavior of which tongue thrust is a symptom (Tulley, 1956).

Mechanical Restriction

1. Constricted dental arches which cause the tongue to function in a higher-than-usual position (Gwynne-Evans, 1956).
2. Macroglossia which limits space in the oral cavity and forces a forward thrust to manipulate the bolus (Breitner, 1942).
3. Enlargement of the tonsils and adenoids which reduces the space available for lingual movements (Moyers, 1958, p. 227; Strang and Thompson, 1958, p. 206).

Neurological Disturbances

1. Hyposensitive palate which precipitates crude patterns of food manipulation and swallowing (Ray and Santos, 1954).
2. Disruption in the tactile sensory control and coordination of swallowing because of inadequate underlying skeletodental configuration (Cleall, 1965).
3. Gross neuromuscular deficiency which includes a tongue-thrusting movement as part of a general extensor thrust pattern (Ingram, 1957; Palmer, 1948).
4. Moderate motor disability and loss of precision in oral function (Bloomer, 1963; Shelton, Haskins, and Bosma, 1959; Strang and Thompson, 1958, pp. 74-76).

Psychogenic

1. Substitution of tongue thrust for forcibly discontinued finger sucking (Teuscher, 1940).
2. Exaggerated motor image of tongue (Froeschels and Jellinek, 1941).

## A BIOGENETIC MODEL OF ORAL ACTIVITY

An attempt will now be made to organize the data and observations concerning oral structure and function into a general model. Although the discussion to this point has made relatively little mention of sensory feedback in the process of physiologic development, Bobath (1967), Smith (1961), and Rood (1954) have all stressed its importance in the integration of any emerging motor system. The theory of neuromotor integration proposed by Smith is especially cogent to the present considerations. This extension of cybernetic theory states that sensory feedback organized in space and time dynamically links and controls the receptor and motor systems of an organism. This control is accomplished at three levels: control of postural movements in relation to gravity, control of transport movements in relation to bilateral differences in stimulation, and control of manipulative movements in space.

According to this formulation, any disturbance in the neurogeometric organization of motion may be expected to result in new patterns of perceptual-motor function. Thus, if the feedback loop operating to maintain a geometric system is altered, some reorganization of the motor output may be expected.

The oral cavity of the human infant is vastly different from that of an adult. The most striking characteristic of this difference lies in the comparative massiveness of the tongue and in the orientation of its suspensory system. Although the general dimensions of the human body change by a ratio of five to one during maturation, Hopkin (1967) showed that the mean dimensions of the adult tongue are only double those of the neonate. Thus, the human infant has a relatively massive tongue housed in a comparatively small oral cavity.

The suspensory system of the infant tongue also differs markedly from that of a mature person. The orientation of the corpus of the tongue to its suspensory system gradually expands during the prenatal period of development from an anteroposterior source of motive power to a multidirectional force capability. This trend toward expansion of the peripheral attachment continues well into postnatal development (Fletcher, in press).

The lingual system of the newborn infant is basically a dual reciprocating structure with the mandible as the anterior hub and the hyoid-styloid complex as the posterior hub. Thus the system is highly stabilized in space and structurally capable of functioning in the gross activities of the infant suckle-swallow.

When the nipple is placed in the infant's mouth, he immediately generates a complete peripheral seal around it by apposing the tongue and lower lip against its inferior and lateral surface and by drawing the soft palate firmly against the tongue root. The lateral seal is supported by the fat pad embedded in the cheeks of the infant, and the posterior seal is facilitated by the leverage obtained as the tensor veli palatini muscles pass around the hamular processes, which are oriented much lower with respect to the tongue of the infant than of the adult. This seal allows the infant to generate the suction through which he can stabilize the nipple within the oral cavity. The tongue, lip, and mandible are then raised in concert and simultaneously the tongue tip is thrust forward against the base of the nipple. The compression which results from these movements is further supported by a rippling motion along the tongue dorsum, beginning slightly behind the tip. This total action expresses the milk from the nipple and carries it to a swallow-preparatory position between the palate and the tongue (Ardran, Kemp, and Lind, 1958; Bosma, 1963; Gwynne-Evans, 1951).

The primary neurogeometric mechanisms to handle the suckle-swallow are differentiated during fetal development (Pritchard, 1965), although they are not fully developed until after birth (Crump, Gore, and Horton, 1958). Gryboski (1969) observed that swallows of premature infants are preceded by three or four suckling bursts and occur during pauses between the suckling bursts; whereas swallows of the more mature infant are frequently present

during suckling itself, which bursts contain as many as 30 consecutive suckle movements without pause.

Postnatal ingestion of solid foods involves differentiation of postural and transport motions which will allow more bulky foods to be handled. Consistent with neurogeometric theory, the initial attempts by the infant to ingest solid foods are in the form of suckle movements. These movements are characterized by elevation of the mandible and forward thrust of the tongue in contact with the lower lip.

Nonliquid foods, however, provide a new set of sensory inputs which, according to the theory, would be expected to set into play a motor reorganization and a new set of outputs. The reactive feedback from the new bodily movements would in turn tend to bring the new response system into stabilized congruence. This new cybernetically generated and regulated reorganization would be posited as the activation of a genetically preprogrammed sequence which lies dormant until the appropriate set of sensory experiences precipitates its arousal.[1]

Cinefluorographic observation of infant ingestion of solid foods indicates that postural stability is achieved by elevating the mandible and thrusting the tongue firmly forward to elevate the bolus crudely to the dorsum of the tongue and transport it into the swallow-preparatory position. In this action, mandibular posturing seems to continue to serve primarily as a supporting buttress to aid in gross positioning of the tongue. Perioral contraction provides additional stability in the anterior oral region. Bosma (1963) called attention to the importance of the labial enclosure of the oral cavity as a developmental landmark in oral function.

As the oral cavity expands through growth, the intrinsic lingual musculature matures and becomes capable of more refined movements, and the teeth erupt to serve as a new potential source of sensory input, new sets of reactive responses may be anticipated. In 1886, Wassilieff described two patterns of lingual reflex which suggest that such reorganization does indeed occur. He found that mucosal stimulation by touching or rubbing the surface of the infant tongue elicited suckling movements in the infant. Repeatedly touching or rubbing the adult tongue caused it to curve into the shape of a spoon. This latter posture is that seen as the bolus is collected in the mature preparatory swallow.

The addition of dental occlusion provides an important new source of stabilization for swallowing. Sensory feedback from the teeth in occlusion during swallowing could signal the presence of a new avenue to achieve a stable postural balance of the oral cavity and thereby release the tongue for more precise control of the bolus during deglutition. From myometric data, Proffit,

[1] An additional important point is that the development of a delicately coordinated pattern of action presupposes the maturation of all tissues and processes involved, to the level at which the action demands will be imposed upon them. As Meader and Muyskens (1962, p. 53) point out, the tissues and processes do not mature simultaneously but, rather, they mature in accordance with a certain "time-place framework." Thus, the tongue may have achieved certain gross developmental characteristics but still be incapable of many finer movements because it lacks the internal structural refinement to perform them.

Chastain, and Norton (1969) suggest just such a developmental pattern. They noted two patterns of swallow which they felt were intermediate between the traditionally described infantile and adult patterns. These patterns consisted of a tongue thrust with the teeth apart and a tongue thrust with the teeth together. They felt the adult pattern would emerge from the latter movements.

Within the foregoing framework, the tongue-thrust pattern of oral activity could represent either a form of developmental arrest or a regression to a less mature phase of physiologic function. Thus, if a particular child (such as one with cerebral palsy) did not achieve the neurological competence demanded to segment the lip-tongue-mandible complex in response to the changing cues from an expanding oral cavity, he could remain in an extensor thrust-like, immature swallow pattern.

Alternatively, if neurologic maturation progressed adequately, but the geometric configuration of the mouth or the tongue did not change sufficiently to provide appropriately different sets of new sensory cues, the swallow pattern would remain at some less-than-mature level. A similar arrest in development could be conceived as the result of more transitory phenomena, such as prolonged infection and missing dentition, which would disrupt sensory cues during critical developmental periods. Timing and intensity of disruptive forces would be critical considerations in this event.

Finally, certain types of trauma to the neurological system may be expected to precipitate regression to previous patterns of oral activity, including the tongue-thrust swallow, as the earlier sensory cues reestablish their prominence.

## ACKNOWLEDGMENT

The author was affiliated with the University of New Mexico, Albuquerque, New Mexico, at the time of this research.

## REFERENCES

ARDRAN, G. M., and KEMP, F. H., Radiographic study of movments of the tongue in swallowing. *Brit Soc. Study Orthodont.*, **30**, 119-128 (1954).

ARDRAN, G. M., and KEMP, F. H., A radiographic study of movements of the tongue in swallowing. *Dent. Practit.*, **5**, 252-263 (1955).

ARDRAN, G. M., KEMP, F. H., and LIND, J., A cineradiographic study of breast feeding. *Brit. J. Radiol.*, 31, 156-162 (1958).

ATKINSON, M., KRAMER, F., WYMAN, S. M., and INGELFINGER, F. J., The dynamics of swallowing. I. Normal pharyngeal mechanisms. *J. clin. Invest.*, **36**, 581-588 (1957).

BALLARD, C. F., Ugly teeth. *Med. Press*, **141**, 78-83 (1959).

BALLARD, C. F., and BOND, E. K., Clinical observations on the correlation between variations of jaw form and variations of oro-facial behavior, including those for articulation. *Speech Path. Ther.*, 3, 55-63 (1960).

BARCLAY, A. E., The normal mechanism of swallowing. *Proc. Mayo Clin.*, **5**, 251-257 (1930).

BLOOMER, H. H., Speech defects in relation to orthodontics. *Amer. J. Orthodont.*, **49**, 920-929 (1963).

BOBATH, B., The very early treatment of cerebral palsy. *Dev. Med. child Neurol.*, **9**, 373-390 (1967).

BOSMA, J. F., Deglutition: Pharyngeal stage. *Physiol. Rev.*, 37, 275-300 (1957).

BOSMA, J. F., Maturation of function of the oral and pharyngeal region. *Amer. J. Orthodont.*, **49**, 94-104 (1963).

Bosma, J. F., and Fletcher, S. G., The upper pharynx: A review. Part II. Physiology. *Ann. Otol. Rhinol. Laryng.*, **71**, 134-157 (1962).
Breitner, C., Tooth supporting apparatus under occlusal changes. *J. Periodontol.*, **13**, 72 (1942).
Cauhépé, J., Antlitz und Vererbung. *Dtsch. Zahn. Z.*, **10**, 17 (1955).
Cleall, J. F., A study of form and function. *Amer. J. Orthodont.* **51**, 566-594 (1965).
Crump, E. P., Gore, P. M., and Horton, C. P., The sucking behavior of premature infants. *Hum. Biol.*, **30**, 128-141 (1958).
Fletcher, S. G., A cinefluorographic study of the movements of the posterior wall of the pharynx during speech and deglutition. Master's thesis, University of Utah (1957).
Fletcher, S. G., Hypernasal voice: Its relation to growth disturbance and physiological activity. Doctoral dissertation, University of Utah (1958).
Fletcher, S. G., Maturation of the speech mechanism. *Folia phoniat.* (in press).
Fletcher, S. G., Casteel, R. L., and Bradley, D. P., Tongue thrust swallow, speech articulation, and age. *J. Speech Hearing Dis.*, **26**, 219-225 (1961).
Froeschels, E., and Jellinek, A., *Speech Therapy.* Boston: Expression (1941).
Gryboski, J. D., Suck and swallow in the premature infant. *Pediatrics*, **43**, 96-102 (1969).
Gwynne-Evans, E., The organization of the oro-facial muscles in relation to breathing and feeding. *Brit. dent. J.*, **41**, 135-142 (1951).
Gwynne-Evans, E., Clinical types. Part I. *Dent. Practit.*, **6**, 222-235 (1956).
Hopkin, G. B., Neonatal and adult tongue dimensions. *Angle Orthodont.*, **37**, 132-133 (1967).
Ingram, T. T. S., Personal communication cited in G. B. Hopkin and J. D. McEwen, Speech and the orthodontist. *Dent. Practit.*, **7**, 324 (1957).
Jacobs, R. M., Muscle equilibrium: Fact or fancy? *Angle Orthodont.*, **39**, 11-21 (1969).
Jones, P. V., and Work, C. E., Volume of a swallow. *Amer. J. Dis. Child.*, **102**, 427 (1961).
Kawamura, Y., Neuromuscular mechanisms of jaw and tongue movements. *J. Amer. dent. Ass.*, **62**, 545-551 (1961).
Kawasaki, M., and Ogura, J. H., Interdependence of deglutition with respiration. *Ann. Otol. Rhinol. Laryn.*, **77**, 906-913 (1968).
Kronecker, H., and Melzer, S., Der Schluckmechanismus, seine Erregung und seine Hemmung. *Arch. Anat. Physiol.*, Supp. (1883).
Kydd, W. L., and Toda, J. M., Tongue pressures exerted on the hard palate during swallowing. *J. Amer. dent. Ass.*, **65**, 321-330 (1962).
Leech, H. L., Clinical analysis of oro-facial morphology and behavior of 500 patients attending an upper respiratory research clinic. *Dent. Practit.*, **9**, 57 (1958).
Magendie, F., *Précis Élémentaire de Physiologie.* Vol. 2. Paris: Mequignon-Marvis (1816).
Meader, C. L., and Muyskens, J. H., *Handbook of Biolinguistics, Part One.* (Rev. ed.) Toledo: Herbert C. Weller (1962).
Moyers, R. E., *Handbook of Orthodontics.* Chicago: Year Book Pub. (1958).
Neff, C. W., and Kydd, W. L., The open bite: Physiology and occlusion. *Angle Orthodont.*, **36**, 351-357 (1966).
Negus, V. E., The second stage of swallowing. *Acta Otolaryng.*, Supp., **78**, 79-82 (1948).
Palmer, M. F., Orthodontics and the disorders of speech. *Amer. J. Orthodont.*, **34**, 579-588 (1948).
Pritchard, J. A., Deglutition by normal and anencephalic fetuses. *Obstet. Gynec.*, **25**, 289-297 (1965).
Proffit, W. R., Chastain, B. B., and Norton, L. A., Linguopalatal pressure in children. *Amer. J. Orthodont.*, **55**, 154-166 (1969).
Proffit, W. R., Kydd, W. L., Wilskie, G. H., and Taylor, D. T., Intra-oral pressures in a young adult group. *J. dent. Res.*, **43**, 555-562 (1964).
Proffit, W. R., and Norton, L. A., The tongue and oral morphology: Influences of tongue activity during speech and swallowing. *Speech and the Dentofacial Complex: The State of the Art. ASHA Reports Number 5*, 106-115 (1970).
Ramsey, G. H., Watson, J. S., Gramiak, R., and Weinberg, S. A., Cinefluorographic analysis of the mechanism of swallowing. *Radiol.*, **64**, 498-518 (1955).
Ray, H. G., and Santos, H. A., Consideration of tongue-thrusting as a factor in periodontal disease. *J. Periodontol.*, **25**, 250-256 (1954).
Rix, R. E., Deglutition and teeth. *Dent. Rec.*, **66**, 103-108 (1946).
Rix, R. E., Deglutition. *Eur. Orthodont. Soc. Trans.*, 191-202 (1947-1948).

Rix, E., Some observations upon the environment of the incisors. *Dent. Rec.*, 73, 427-441 (1953).

Rogers, A. P., Open-bite cases involving tongue habits. *Int. J. Orthodont.*, 13, 837-844 (1927).

Rood, M. S., Neurophysiological reactions as a basis for physical therapy. *Phys. Ther. Rev.*, 24, 440-449 (1954).

Saunders, M. B., Davis, C., and Miller, E. R., The mechanism of deglutition (second stage) as recorded by cinefluorography. *Ann. Otol. Rhinol. Laryng.*, 60, 897-916 (1951).

Scott, J. H., The role of soft tissues in determining normal and abnormal dental occlusion. *Dent. Practit.* 11, 302-308 (1961).

Shelton, R. L., Jr., Bosma, J. F., and Sheets, B. V., Tongue, hyoid and larynx displacement in swallow and phonation. *J. appl. Physiol.*, 15, 283-288 (1960).

Shelton, R. J., Jr., Haskins, R. C., and Bosma, J. F., Tongue thrusting in one of monozygotic twins. *J. Speech Hearing Dis.*, 24, 105-117 (1959).

Sheppard, I. M., Jacobson, H. C., Zaino, D., and Poppel, M. H., Dynamics of occlusion. *J. Amer. dent. Ass.*, 58, 77-84 (1959).

Smith, K. U., The geometry of human motion and its neural foundations. II. Neurogeometric theory and its experimental basis. *Amer. J. phys. Med.*, 40, 109-129 (1961).

Strang, R., and Thompson, W. M., *A Textbook of Orthodontia.* Philadelphia: Lea and Febiger (1958).

Straub, W. J., The etiology of the perverted swallowing habit. *Amer. J. Orthodont.*, 37, 603-610 (1951).

Subtelny, J. D., Examination of current philosophies associated with swallowing behavior., *Amer. J. Orthodont.*, 51, 161-182 (1965).

Subtelny, Joanne D., Mestre, J. C., and Subtelny, J. D., Comparative study of normal and defective articulation of /s/ as related to malocclusion and deglutition. *J. Speech Hearing Dis.*, 29, 269-285 (1964).

Sumi, T., Neuronal mechanisms in swallowing. *Pflügers Arch.*, 278, 467-477 (1964).

Teuscher, F. W., Suggestions for treatment of abnormal mouth habits. *J. Amer. dent. Ass.*, 27, 1703-1714 (1940).

Truesdell, B., and Truesdell, F. B., Deglutition: With special reference to normal function and the diagnosis, analysis and correction of abnormalities. *Angle Orthodont.*, 7-8, 90-99 (1937-1938).

Tulley, W. J., Methods of recording patterns of behavior of the oro-facial muscles using the electromyograph. *Brit. Soc. Stud. Orthodont. Trans.*, 88-95 (1953).

Tulley, W. J., Adverse muscle forces—their diagnostic significance. *Amer. J. Orthodont.*, 42, 801-814 (1956).

Vantrappan, G., and Hellemans, J., Studies on the normal deglutition complex. *Amer. J. digest. Dis.*, 12, 255-266 (1967).

Wassilieff, N. W., Ueber eine localisirte reflectorische Bewegung der Zunge. *Zbl. med. Wiss.*, 24, 209-210 (1886).

Wildman, A. J., Fletcher, S. G., and Cox, B., Patterns of deglutition. *Angle Orthodont.*, 34, 271-291 (1964).

# SPEECH

RICHARD M. COLE

*Lancaster Cleft Palate Clinic, Lancaster, Pennsylvania*

Egil P. Harvold opened his oral presentation in this symposium by saying that an audience always should ignore titles. I should open with the same statement, and for several reasons. My topic is the growth and development of a particular physiologic system called speech. As we all know quite well, the anatomy and physiology of such a system are quite similar, if not identical, to that of the system concerned with the functions of mastication and deglutition. As I listened to the oral presentation of the excellent papers published in this *Report,* it struck me that they covered mastication, deglutition, and speech rather well from a number of standpoints ranging from neurophysiology to remediation. I was somewhat troubled, also, by the fact that I found it difficult to imagine how a didactic presentation concerned with the physiology of speech could help to fulfill the charge of our Joint Committee, to promote a closer working relationship between the professions of speech pathology/audiology and dentistry in training, service, and research. This concern was reinforced when I looked at the audience, 50% of whom were leaders in the field of speech pathology, and 50% leaders in the field of dentistry. They came from institutions with highly recognized programs in speech pathology and audiology, thus posed the likelihood that they had a more than casual acquaintance with the physiology of speech.

All of this must seem to be a series of rationalizations which is leading to a complete change of topic. This is not quite the case. Rather, I am going to discuss briefly the anatomy, physiology, and some of the research concerned with a series of anatomic or functional units often considered as making up the speech mechanism. I will then move quickly into an area that is of great interest to me and one that I think comes much closer to the concern of our Joint Committee: that dealing with research issues of particular concern to both our professions.

## EARLY DEVELOPMENT

Later in this *Report,* Arend Bouhuys will discuss the "power supply" for the speech mechanism. After touching briefly on the larynx, I will move upward, discussing the pharynx, the palate, pharyngopalatal synchrony, the hyoid

system, the tongue, the bony oral structures, and conclude with the facial and labial muscles.

Before beginning this mechanistic march upward through the speech system, I will discuss briefly the growth and development aspects of this system. Man is said to represent the summit of the phylogenetic scale, although current events give us reason to doubt the accuracy of the statement. Nonetheless, as the scale progresses from invertebrate to man we see a series of changes—the gradual recession of the snout and mandible; the shortening and broadening of the facial muscles, tongue, and muscles of the floor of the mouth; the descent of the larynx; and the assumption of the upright posture, which has freed the hands for feeding and thus freed the oral cavity, the mouth, the lips, and the face for speech. Yet, when we look at the human fetus, there appears to be a kind of exaggeration of this trend, in that the snout and mandible have assumed a position of extreme retrusion. We are all aware of the downward and forward growth of the lower two-thirds of the face during infancy; however, some excellent work at the University of Michigan (Burdi, 1969) now tells us that this very orderly tendency may be seen in the second and third trimesters of embryonic life, so that by adulthood we are left with a relatively straight facial profile.

Moving from phylogeny to embryology, it appears that the newborn infant may already have had a relatively lengthy period of rehearsal of a number of movements that we say are requisite to speech. Studies of prenatal reflexes (Humphrey, 1969) tell us that the fetus of 8½ weeks menstrual age exhibits lowering of the mandible upon stimulation, and, at 12½ weeks, exhibits elevation and possible protrusion of the tongue as well as swallowing behavior upon stimulation. In the past we have been inclined to say that movements of the fetus and the newborn infant represent undifferentiated neuromotor acts. It seems that a more appropriate description would state that these acts are poorly differentiated. One cannot help but wonder whether these prenatal activities have left any imprint upon the central nervous system, as crude as it is at this stage of development. Is it possible that a kind of primitive learning is taking place during this period? It may be, also, that we have underestimated the sensorimotor abilities of this little creature. Some current research in England[1] tells us that newborns taken from mothers with absolutely negative pre- and paranatal histories may demonstrate visual and auditory tracking through 180°. This, of course, defies every norm that we have ever had regarding infant behavior, and raises the possibility that we should take a much closer look not only at the first months, but at the first days of life.

As the infant matures, we see a dropping out of a number of primitive reflexes that actually interfere with the early acquisition of speech. Some of these reflexes may represent a kind of short-circuiting in the central nervous system due to incomplete myelinization. It appears that we are witnessing, gradually, a kind of cumulative quantitative dominance over the final common

[1]Richards, Martin, personal communication (1970).

pathway, and that not until such dominance has taken place can we expect reasonably well-differentiated speech sounds.

A number of events seem to correlate with the appearance of these speech sounds, such as the assumption of the sitting posture and its accompanying changes in respiratory physiology and activity, the emergence of teeth, and the transition from soft foods to hard foods, which demands a greater specificity of tongue function. Elsewhere in this *Report*, Moyers discusses the importance of the emergence of teeth in effecting changes in orofacial motor patterns and the precision of speech sounds.

Certainly, the gradual maturation of neuromotor activity is quite obvious, as is the gradual change in the process of "stimulus fusion." Less clear, however, is the developmental schedule of perceptual skills. Considerable controversy continues as to whether the infant's gradual mastery over articulatory movements results in sharper limits in his self-monitoring, or whether the reverse is the case (Menyuk and Anderson, 1969). I am tempted to say that this represents a rather artificial approach to the issue. I am reminded that one of the foremost speech scientists in our field once made the statement that tactile and kinesthetic cues are relatively unimportant, giving as evidence the fact that congenitally deaf children generally are unable to master articulate speech. This, it seems to me, is a dangerous line of reasoning. I am reminded of the strict localizationists who insist that every definable aspect of sensory or motor behavior has as its basis a relatively specific locus within the central nervous system. Over a century ago John Hughlings Jackson (Taylor, 1958) spoke of the "positive and negative elements" in the symptomatology of disturbed behavior, whether speech or otherwise. In these terms, Jackson was speaking essentially of the dynamic and plastic nature of the nervous system—of the richness of interaction among its elements. The failure of many initiates to the field of speech pathology to look beyond the possibility of a motor etiology for defects in articulation may stem primarily from the failure to recognize the dynamic and interactive nature of the central nervous system.

## ANATOMY AND PHYSIOLOGY

I will move now to some consideration of the anatomy and physiology of speech production. My intent here is not so much to cover a particular body of information as to present some of the issues that have remained controversial and some of the issues that have remained relatively undefined and are in need of careful and controlled research. Most of the material to be presented is concerned with the adult, since we still are loathe to subject the infant to needle electrodes, radiation, and most of the transducers currently available for the study of speech physiology. In some cases, however, we do feel reasonably secure in extrapolating backward in time to infancy our research results derived from adults. As I mentioned earlier, I will begin with the discussion of the mechanism of phonation rather than respiration.

Air ascending through the trachea, under varying degrees of pressure, im-

pinges upon the subglottic laryngeal structures, primarily the conus elasticus and the inferior surfaces of the vocal folds themselves. If the folds are completely adducted, the subglottic pressure will increase, and, depending upon the amount of this subglottic pressure and the degree of tension imposed by the laryngeal adductors, produce an increase in the intensity of the voice or a rise in pitch. We continue to list as adductors of the vocal folds, the muscles of the folds themselves, the vocalis and the thyroarytenoid, the lateral cricoarytenoid, the cricothyroid and, very likely, the transverse and oblique arytenoids. From the work of Isshiki (1969) and Hirano, Ohala, and Vennard (1969) it appears reasonable to conclude that adductor tension increases as fundamental frequency increases. With increases in intensity level, however, cricothyroid muscle activity appears to decrease, particularly in the high frequencies.

Since Goerttler (1950) published his findings regarding oblique fibers in the vocalis muscle and Husson (1953) advanced his "neurochronaxic" theory, controversy has continued to rage between the proponents of the neurochronaxic school and the advocates of what generally may be called a myoelastic-aerodynamic basis for vocal-fold vibration. Although research evidence for some years has weighed heavily against the neurochronaxic theory, its advocates continue to appear in the literature from time to time. As an aside, perhaps the issue will be clarified somewhat by research concerned with Teflon injections of partially or totally paralyzed or atrophied vocal folds. In many such instances a breathy, hoarse pre-injection voice quality is rendered entirely normal by the injection of Teflon into an essentially passive vocal fold. Perhaps those concerned with mechanisms underlying voice production could profit from studying the results of those concerned with voice rehabilitation. One interesting facet of working with these Teflon-rehabilitated patients is that the sound spectrographs taken from their postinjection voices are indistinguishable from those taken from individuals with entirely normal laryngeal mechanisms. Yet, the injected vocal fold is entirely passive.

As the pulsating airstream emerges from the vibrating vocal folds, it enters what amounts to a muscular tube—the pharynx. This tube generally is considered to have three closely associated component muscles: the inferior, medial, and superior pharyngeal constrictors, all of which have a common origin (the pharyngeal raphe). The anterior insertions are different for each muscle. This muscular tube represents an ideal resonator, since not only can each muscle contract individually, but it appears that portions of an individual muscle can contract selectively. An increasing body of evidence from a number of sources (such as electromyography, cinefluorography, pulsed ultrasound, and capacitance) indicates that there is differential and selective contraction of the various pharyngeal constrictors and that asymmetry of pharyngeal constriction is the rule rather than the exception.

The neurophysiologic basis for pharyngeal function, as was the case with laryngeal function, is the pharyngeal plexus, though it is likely that the accessory nerve has more to do with laryngeal function. The pharyngeal plexus also accounts primarily for velopharyngeal closure. The exception to this is the

tensor palati muscle, which is innervated by the motor branch of the trigeminal nerve.

The vibrated airstream, or raw sound, now has passed through a variable resonator and, depending upon the nature of the sounds to be articulated, will emerge from the oral or nasal cavities, which themselves are additional resonators. The palatopharyngeal valving mechanism will determine the direction of this pulsated, resonated airstream, and the size, shape, and tonicity of the walls of these two cavities will determine the total resonance characteristics of the voice. It is the activity of the velopharyngeal mechanism which determines whether the nasal cavity is to be coupled to the oral cavity during speech production.

The mechanism of velopharyngeal closure is relatively straightforward (Figure 1). We may consider two components, each contributed to by two muscles.

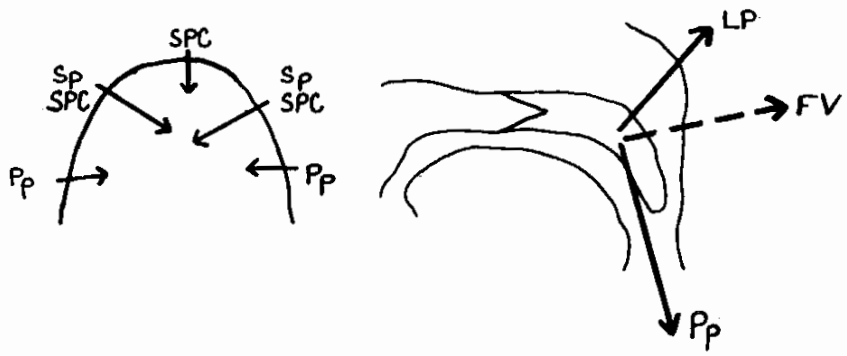



FIGURE 1. Schematic representation of the muscles controlling velopharyngeal closure. Left, superior view of pharyngeal walls at the level of the palatal plane: Pp, palatopharyngeus; SPC, superior pharyngeal constrictor; Sp, salpingopharyngeus. Right, lateral view of velopharyngeal area: Pp, palatopharyngeus; LP, levator palati; FV, force vector resulting from synchronous activity of LP and Pp.

The first component is that concerned with the upward and backward movement of the velum. The upward and backward movement is primarily the function of levator palati, and the palatopharyngeus contributes a downward and backward movement. The resultant vector of forces produces a slightly upward, but essentially backward, movement of the velum to make contact with the posterior pharyngeal wall. The second component of velopharyngeal closure is composed of the superior pharyngeal constrictor and the salpingopharyngeus muscles. Both muscles, though acting nearly at right angles to each other, tend to narrow the pharynx, usually making contact with the lateral borders of the velum. Anterior movement of the posterior pharyngeal wall during function is rather slight and often inconsistent. Usually the anterior excursion is 2 to 3 mm at best.

At least two other muscles have not been mentioned with regard to palatal

activity: the palatoglossus and the tensor palati. Neither clinical examination, dissection, nor electromyographic research has made entirely clear the functions of these two muscles. Certainly one role of the tensor palati is to accomplish exactly what its name implies—to tense the palate; however, since the tensor is located at the junction of the hard and soft palates, and since the tissue is primarily aponeurotic, it is unlikely that it has any direct function in velopharyngeal closure. Because of the origins and insertions of the palatoglossus, we know that it could either depress the palate or elevate the tongue. Unfortunately, none of the techniques mentioned has served to clarify the issue entirely. The electromyographic work of Fritzell (1969) and the electromyographic-cinefluorographic work of Lubker (1968) have helped to clarify the role of individual muscles in velopharyngeal closure. Considerable clinical experience might lead one to believe that the Moll-Shriner hypothesis (1967) concerning discrete levels of velar activity could be more appropriate to pharyngeal function than velar function.

A number of phenomena associated with velopharyngeal closure are puzzling to those concerned with this mechanism, for whatever reason. For example, the linearity of the relationship betwen velopharyngeal orifice size and (1) nasal/oral sound-pressure levels, (2) nasal airflow, and (3) articulation skills is considerably less than perfect (Shelton et al., 1969; Warren, 1967; Brandt and Morris, 1965). We know that a number of variables may influence perceived hypernasality. From an organic standpoint, a number of structures may exert varying degrees of influence upon perceived nasal resonance, such as the size and shape of the nasal resonating chamber, the size of the inferior turbinates, the thickness and contour of the nasal septum, the condition of the nasal mucous membranes, and even the size and shape of the nares, to mention but a few. All of these may act quite independently of velopharyngeal orifice size in determining hypernasality.

Another structure offering a totally unknown contribution to oral/nasal resonance balance is the maxillary sinus, or antrum. I have examined several clinical patients who have had surgical removal of tissue because of carcinoma, leaving the antrum widely exposed to the oral and nasal cavities. These patients were excessively hypernasal; however, the degree of hypernasality was strikingly reduced when the relative isolation of the antrum was restored prosthetically.

Before leaving this anatomic area, I will comment only briefly on the issue of velopharyngeal inadequacy. The usual results of the condition are, as one would expect, hypernasality, nasal air escape during speech, and some loss of precision during the articulation of certain phonemes, particularly fricatives and sibilants. Remediation usually takes one of three courses: (1) any of a variety of surgical procedures, including pharyngeal implants, (2) prosthetics, and (3) direct muscle stimulation. It is the last point which prompted me to raise the issue of velopharyngeal inadequacy. Clinical experience has told us that direct muscle stimulation of palatal and pharyngeal musculature will elicit some degree of increase in the activity of these muscles. Unfortunately, our

documentation of this increase in activity is inadequate. One reason for this is that we have had only crude means of assessing the mesial movement of the lateral pharyngeal walls (Cole, 1971; Campos-Giral and Cole, 1963). Recently, however, two vastly improved radiographic approaches to this problem have given promise of a much greater degree of precision in evaluating this mesial movement, which is so critical in surgical or prosthetic rehabilitation of velopharyngeal inadequacy (Kelsey, Ewanowski, and Crummy, 1970; Skolnick, 1970).

I will move on now to the hyoid system. I use the word *system* since there is good reason to believe that this structure and its attendant muscles may assist or even govern a number of functions. From Figure 2 one can see that two

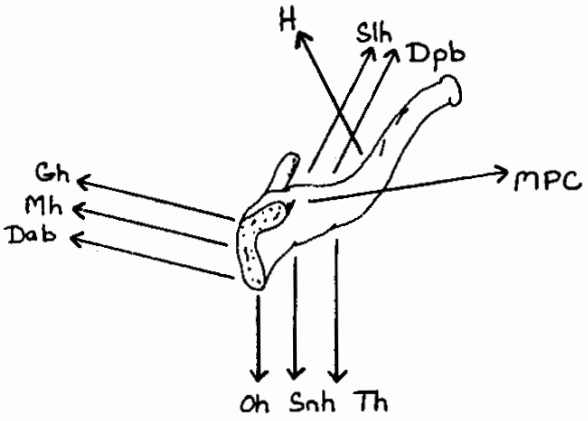



FIGURE 2. Schematic representation of the muscles controlling hyoid movement; medial aspect of hyoid bone, right half: Gh, geniohyoid; Mh, mylohyoid; Dab, anterior belly of digastricus; Oh, omohyoid; Snh, sternohyoid; Th, thyrohyoid; MPC, medial pharyngeal constrictor; Dpb, posterior belly of digastricus; Slh, stylohyoid; H, hyoglossus.

muscles may act to pull the hyoid upward and backward; four may act to pull it upward and forward, or if the hyoid is stablized to pull the mandible downward; and at least three muscles can lower the hyoid, and even move it somewhat anteriorly. The hyoid system is believed to assist materially in depressing the mandible. Certainly, the anatomy of the anterior suprahyoids would lead one to theorize this possibility. Unfortunately, our evidence is imperfect, since it relies primarily on electromyographic evidence. EMG results derived from surface electrodes are difficult to interpret because of the influence of adjacent muscles; results derived from needle electrodes are difficult to interpret, since one never knows exactly what muscle is providing the signal. The role of the hyoid system in assisting or regulating tongue movements is virtually unknown. I will speak to this issue in the section concerned with research questions.

As complex as are the movements of the tongue, its anatomy is relatively straightforward. One may diagram a series of force vectors to represent the myriad possibilities of movement of the tongue by its four extrinsic and its four intrinsic muscles (Figure 3). From their anatomic course alone, it seems rea-

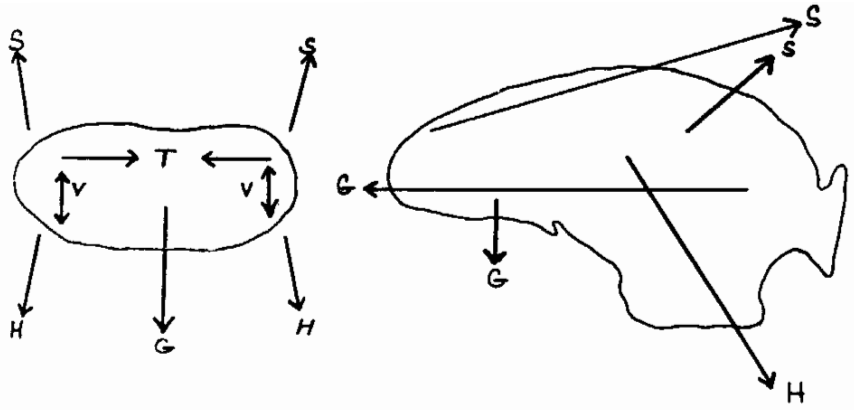



FIGURE 3. Schematic representation of the muscles controlling tongue movement. Left, frontal section of anterior third of tongue: S, styloglossus; H, hyoglossus; G, genioglossus; V, vertical fibers; T, transverse fibers. Right, lateral view of tongue: G, genioglossus; H, hyoglossus; S, styloglossus.

sonable to assume that the styloglossus is the muscle primarily responsible for elevating the tongue. Likewise, the genioglossus seems primarily responsible for lowering the tongue in the mouth and for protruding the tongue. The hyoglossus brings the tongue sharply downward and backward during deglutition, and anchors the base of the tongue during the production of a number of tongue-tip sounds. Quite frankly, I am not sure what the palatoglossus does. Theoretically, it can elevate the tongue; if it does, I am inclined to believe that it does not elevate it very much. The intrinsic muscles may serve to narrow, thin, curl the tip of, shorten, or lengthen the tongue.

As complex as the motions of the tongue may be during mastication and deglutition, this complexity does not begin to approach that seen during rapid, continuous speech. One can only marvel at the fantastic synchrony exhibited by this structure during speech. For example, fibers of the genioglossus inserting in the posterior portion of the tongue may bring the tongue forward in the mouth so that the styloglossus can elevate the tip to make contact with the alveolar margins. At the same time, genioglossal fibers inserting into the midline of the tongue may serve to depress the midline in conjunction with the elevation of the lateral margins of the tongue by the styloglossus, thus assisting in the production of sibilant sounds. The hyoglossus also is active during this period, anchoring the posterior portion of the tongue so that the tongue tip can be raised selectively.

The neural innervation of the tongue is as simple as the innervation of the hyoid musculature is complex. With the exception of the palatoglossus, which is supplied by the pharyngeal plexus, all muscles of the tongue are supplied by the twelfth cranial nerve, the hypoglossal nerve. The controversy as to whether there are sensory components to this nerve continues, and I will not take time to discuss the issue. Likewise, controversy continues to rage over

whether there are proprioceptive/kinesthetic endings in the tongue. Certainly, there does appear to be evidence for some kind of mechanorceptors in the tongue from which we derive position sense at times when the tongue is not in contact with other oral structures. Certainly the ratio of nerve fiber to muscle fiber lends reinforcement to the fact that the tongue is capable of an amazing variety of movements and is capable of these movements early in life. The data of Humphrey (1969) indicate that the tongue is capable of contracting during the 11th week of fetal life, and that it demonstrates overt activity at 12½ weeks of fetal life.

We know, too, that the tongue is rich in sensory innervation. We have a number of articles, texts, and colleagues who can attest to the tongue's tactile awareness, two-point discrimination, and stereognostic skills (Bosma, 1967). These functions appear to be governed entirely by the sensory portion of the trigeminal nerve. Taste, of course, is governed by the seventh cranial nerve, anteriorly, and the ninth cranial nerve, posteriorly.

The sensory and motor capabilities of the tongue imply that it may serve as a sensitive indicator of central or peripheral nervous system dysfunction. What continues to amaze and distress me is that, for the most part, our friends in neurology have failed to take advantage of this fact, looking only for signs of atrophy or fasciculation. A diagnostic task no more complex than the demand for rapid diadochokinetic activities of the tongue may yield obvious information about central nervous system dysfunction, in the type and degree of synkinetic activity, or "overflow," observed. It is certainly not chance that one of the greatest aggregates of reticular neurons in the central nervous system is in the region of the hypoglossal nucleus.

The next functional unit to be discussed is the mandible. By now I am sure that my breezy descriptions of rather complex structures and functions have appalled my colleagues in speech pathology; now I will run the risk of appalling my friends in dentistry. Mandibular function is far more than a series of up-and-down movements. For example, the translatory movements of the condyle in the articular fossa during mastication are incredibly complex. Fortunately for me, however, my topic is speech, and I can legitimately avoid this complex subject.

The force vectors of the mandible may be sketched rather simply (Figure 4). You recall that the elevation of the mandible is achieved, in part, through the synchronous activity of two muscles, the masseter and the medial pterygoid. The two muscles form a "sling" at the angle of the ramus, to bring the mandible upward and slightly forward. The third muscle of elevation is the temporalis, running from the temporal fossa to the coronoid process. Depending upon which fibers of this rather broad muscle are firing, the mandible may be moved directly upward, upward and slightly forward, or upward and slightly backward. Mandibular depression has been discussed in relation to the hyoid musculature. It is believed by some that a portion of the lateral pterygoid also can assist in mandibular depression.

It is difficult to assess the importance of mandibular movement to speech.

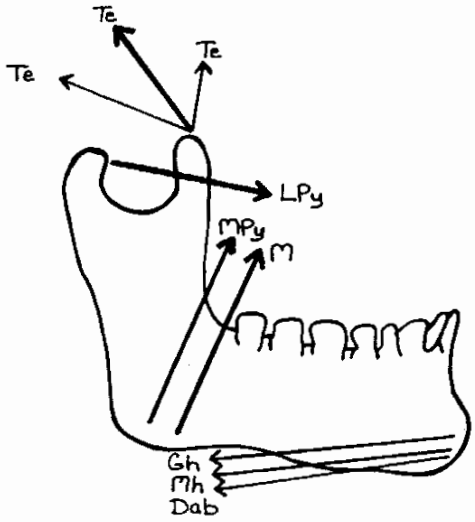



FIGURE 4. Schematic representation of the muscles controlling mandibular movement: Gh, geniohyoid; Mh, mylohyoid; Dab, anterior belly of digastricus; M, masseter; MPy, medial pterygoid; LPy, lateral pterygoid; Te, temporalis.

Certainly these movements are minimal during speech. The neurology of this area would lead us to believe that a far more important function is that of mastication. For example, it has been discovered that periodontal receptors may travel to all three of the brain-stem trigeminal nuclei, and that these impulses may travel directly to the parietal lobe. Yet, in spite of the richness of periodontal innervation, the teeth seem unable to discriminate accurately the direction of force applied to their surfaces. If one taps on the teeth during closure, electromyographic activity suddenly ceases. This does not occur when the teeth are clenched. EMG opening and closing patterns resulting from voluntary tapping of the teeth and from occlusal contacts during mastication are quite different (Butler and Stallard, 1969). Does this mean that different pathways are responsible for reflex and for voluntary activities? The complexity of the trigeminal system becomes apparent when one realizes that the spinal root of the trigeminal nerve also receives information from the seventh, ninth, and tenth cranial nerves, and that any point on the face is represented along the entire length of the spinal root as well as in the main sensory nucleus of the trigeminal. There is evidence, too, that the trigeminal nerve is the first cranial nerve to become active during fetal life (Humphrey, 1969).

The last functional unit concerned with speech physiology to be discussed is that involving the facial, buccal, and labial musculature. Although there is a variety of muscles, and an even greater variety of movements made possible by these muscles, their functions largely can be represented by a simple schematic diagram (Figure 5). Such a diagram says much of what can be said about these muscles. The great variety of facial and labial movements possible is a result of varying combinations of these muscles and varying degrees of tension they exert. Perhaps two or three muscles do warrant special mention.

It is likely that the buccinator muscle plays a modest role in mastication and in speech. As the "cheek" muscle, it acts to oppose the tongue in keeping food on the occlusal table, and varying degrees of tonicity during speech may have some slight influence upon resonance. To experience the latter, one need only pull the cheeks outward while speaking. The orbicularis oris, perhaps the best known muscle of the anterior face, actually is a composite of many muscles. I know of few anatomists who would be willing to isolate a particular fiber and say that it belonged to this muscle. Its primary purpose is to purse, or pucker, the lips, rather than to close them, per se. Thus, the semisphincteric posture the lips assume during the production of the *oo* sound primarily is a function of orbicularis oris; whereas the tense and somewhat spread posture of the lips during the *ee* sound primarily is a function of buccinator and its smaller overlying muscle, the risorius. An excellent test of the seventh cranial nerve, which innervates all of these muscles, is simply to ask the patient to alternate between these two vowel sounds. There is some experimental evidence indicating that the posture and tonicity of the labial musculature also can influence oral resonance. During the production of vowels such as *oo*, one may consider lip rounding as serving to lengthen the vocal tract, since it is known that the larynx descends during the production of this vowel. Conversely, during the production of vowels such as *eh*, and *ah*, the larynx is in a relatively higher position and the lips are flattened.

To conclude this section concerned with the anatomy and physiology of speech production, I should mention the phenomenon referred to as *coarticulation.* Simply stated, the concept concerns the types of anticipatory activity seen in one or several structures during function. For example, in the word *construe,* we see that the lips are being rounded for the *oo* vowel as the *s* consonant is being formed. An excellent medium for demonstrating coarticulation is a cinefluorographic film of the speech mechanism during the utterance

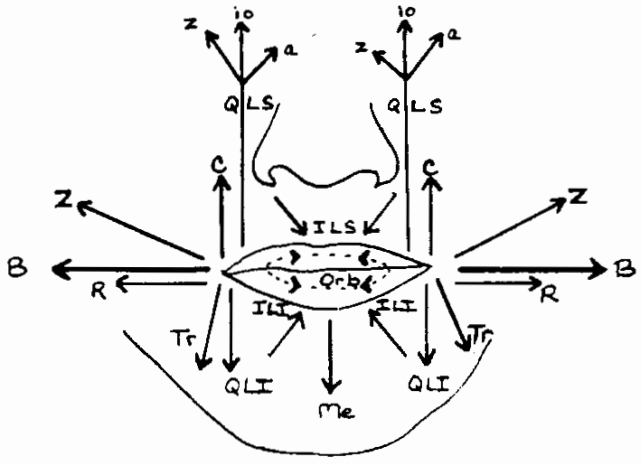



FIGURE 5. Schematic representation of muscles controlling labial and buccal movement: B, buccinator; R, risorius; Tr, triangularis; QLI, quadratus labii inferioris; ILI, incisivus labii inferioris; Me, mentalis; Z, zygomaticus; C, caninus; ILS, incisivus labii superioris; QLS, quadratus labii superioris; z, zygomatic head; io, infraorbital head; a, angular head; Orb, orbicularis oris.

of a complex sentence. These anticipatory activities may be seen in all of the structures that I have discussed—the larynx, the pharynx, the soft palate, the tongue, the hyoid bone, the mandible, and the facial and labial musculature.

## RESEARCH ISSUES OF MUTUAL CONCERN TO DENTISTRY AND SPEECH PATHOLOGY/AUDIOLOGY

Before beginning this section, I want to comment on a tendency that I sense from time to time within my own profession, that of extrapolating from models, from animal work, and from pathology. By way of illustration, I should like to comment on a newly published work by Perkell (1969), entitled *Physiology of Speech Production.* At one point, Perkell postulates that the speech-production mechanism can be thought of as being composed of two neuromuscular systems, and that the articulation of vowel sounds is accomplished primarily by the extrinsic tongue muscles and the production of consonant sounds is a function of the intrinsic musculature of the tongue. The book is quite interesting and it is well worth reading. It offers an elaborate means of analyzing, if you will, the ballistics of speech activity. My concern is that there is little experimental evidence to support some of the hypotheses stated. A greater concern, however, is the tendency of some to treat hypotheses and theories as fact. There is the tendency, also, to extrapolate from animal experimentation. We are often so eager to have explanations for various behavioral phenomena that, unknowingly, we perform the extrapolation and take that huge leap from animal to man, and are left with the feeling that issues have been settled. Another danger is that of extrapolating normal anatomy and function from pathologic anatomy and function. This is done in my own profession, particularly regarding aphasia and brain function.

Enough of digressions. If there is any one theme to these remarks concerned with research issues, it is that we must constantly strive for better definition and quantification of the various oral-facial motor and sensory skills that we feel are requisite to speech production, mastication, and deglutition. Perhaps another theme would be homogeneity versus heterogeneity. Let me give you two examples, both of particular concern to the dental profession. Tongue-thrusters continue to be treated as though they were a homogeneous population. Only recently has there been any concerted attempt to factor-analyze the variables often thought to be associated with this phenomenon (Hanson, Barnard, and Case, 1969, 1970). Another example is that of articles appearing in the orthodontic literature espousing one treatment technique over another. The treatment sample may have been a group of very severe Angle Class III malocclusions, yet nowhere in the article can one find any definitive statements regarding the population studied, how they came to be severe Class III malocclusions, or the existence of other behavioral or neurophysiologic phenomena that should have been studied to better define the sample and to allow a more accurate and realistic interpretation of the results.

Let me present briefly some examples of issues that appear to be in need of

quantification, and issues that appear to remain vague with regard to the matter of homogeneity versus heterogeneity. Those of us involved in the treatment of patients with cleft palate often come across patients said to have Pierre Robin Syndrome. One aspect of this syndrome is said to be micrognathia, yet to my knowledge nobody has ever defined micrognathia in objective terms. The diagnosis of micrognathia continues to result entirely from subjective impressions. I am not a dentist, but I am more than a little impressed by the radiologic techniques that dentists have developed and use. Is there really any good reason why we cannot establish an arbitrary boundary, and say that dimensions beyond that point represent normalcy and dimensions below that point represent micrognathia? It seems to me that normative data already are at hand in this regard.

Can we do no better than to classify malocclusion or crossbite as Class I, II, or III, or as mild, moderate, or severe? These are functional, pragmatic, and descriptive terms, but surely there must be more objective ways of defining the two conditions. For not until we have such objective means at hand will we be able to make meaningful statements about the effectiveness of our treatment techniques or to make realistic statements about the merits of one treatment as contrasted with another.

When I speak of the failure to define adequately the population under study, or the general failure to quantify precisely the variables being studied, or the results obtained, I am not speaking only of the dental profession; my own profession is as guilty as any other. In fact, two of the issues to which I have alluded (those concerned with a precise quantification of malocclusion and crossbite) are considerably closer to being resolved as the result of recent research by an orthodontist. Stockli's work at Northwestern University (1969, 1970) gives great promise in this regard. Speech pathologists have speculated for years about possible relationships between atypical arch dimensions and articulation problems. Research now is underway to quantify precisely a number of maxillary arch dimensions, through a variety of means, in normal individuals and in individuals with cleft lip and palate, and to relate these dimensions to patterns of articulation.[2] By way of an aside, I suspect that most clinicians who have had extensive experience in dealing with orofacial abnormalities would guess that most children could compensate or adapt to such conditions, and I am inclined to agree. Ironically, adaptive mechanisms have been considerably better defined on a cellular level than they have been on a neurophysiological or behavioral level.

Another example of the need for quantification is the assessment of soft tissue configuration, symmetry, and growth of the anterior face. Such data, at the very least, would provide plastic surgeons, orthodontists, and prosthodontists considerably more objective guidelines in their approach to treatment, regardless of the nature of the problem. Why not also apply some of the relatively new techniques (Haring and McCormack, 1964; Damon, 1967) to quan-

[2]Work of this author with M. Mazaheri, in preparation.

tifying the extent of the cleft lip? It troubles me to realize that we can provide our computers with precise anthropometric-cephalometric data regarding the craniofacial growth of our patients, yet when we come to correlate these data with cleft type, we can do no better than talk about a lip cleft that is narrow, moderate, or wide, and one-, two-, or three-thirds in extent. Our ratio-scale data is diluted to an ordinal level. We must do better than this, since, if we are going to understand the nature and degree of heterogeneity of the cleft-palate population, we must relate these kinds of data.

Let me mention now a number of issues that need not only quantification, but definition as well. For example, experimental phoneticians continue to tell us that the perceptual and spectral characteristics of vowel sounds largely are dependent upon such factors as the anteroposterior and the vertical positioning of the tongue in the oral cavity, as well as the degree of tonicity of the tongue. We are inclined to accept such theories until we come face to face with individuals who have experienced total glossectomies and whose vowel sounds are entirely normal by listener judgment and sound spectrographic analyses. The two individuals with whom I have had personal experience had not one shred of tongue tissue; in one, the geniohyoid, the mylohyoid, and the anterior belly of the digastricus had been removed. Even more troublesome, from the standpoint of our present inability to understand such phenomena, was the fact that the speech of both individuals was entirely intelligible. Quite obviously there were articulatory defects, yet the nature and extent of these defects was not at all what our present knowledge in experimental phonetics would lead us to expect. Closely associated with the need to acquire a better understanding of the contribution of the tongue to spectral characteristics of the voice, is the need to understand the exact role of the hyoid system, particularly as it may serve as a kind of mobile platform for the tongue during speech production.

Two issues of joint concern to dental specialists and speech pathologists are the role of the palatine tonsils and the nature and disturbances of swallowing. Plastic surgeons, prosthodontists, and speech pathologists continue to be troubled by the exact nature of the role of the palatine tonsils with respect to their possible contributions to palatal activity, oral-nasal resonance balance, and tongue function. This is particularly true in the case of individuals with cleft lip and palate. There appears to be considerably more understanding of the role of the pharyngeal tonsils (the adenoids) with respect to these functions. We know, for example, that the hypertrophied adenoid can assist the palate in achieving velopharyngeal closure, in some cases, by the mere fact of its bulk. We know, too, that the hypertrophied adenoid may produce a hyponasal voice quality. We are somewhat less certain about the possible relationship between hypertrophied adenoid tissue and chronic otitis media. Yet, about the palatine tonsils we display none of this certainty. I am inclined to agree with those who postulate a role for hypertrophied palatine tonsils in causing or maintaining tongue thrust, but there is virtually no evidence to indicate whether we should expect hypernasality or hyponasality in cases with massive hyper-

trophy of the tonsils. Likewise, in patients demonstrating limited palatal activity and hypertrophy of the tonsils, can we expect an increased excursion of the palate after removal of tonsils? Many plastic surgeons avoid this procedure, since they feel that scarring of the palatal pharyngeal pillars actually may impede palatal activity.

I cannot resist one additional example of the need to define, or redefine, as well as to quantify. Approximately 11 years ago, our attention was directed forcibly to a clinical entity that has earned a number of descriptions, including tongue thrust, reversed swallowing, visceral swallowing, and others. Quite properly, reversed swallowing was defined as one or more deviations from several factors cited as characteristic of the normal swallow, normal swallow being defined by (1) molar contact, (2) the absence of facial activity, (3) lip contact, (4) the tongue not in contact with the teeth during rest, and (5) a frequency of swallow ranging from 2000 to 3000 times per day. Within the last five years a number of studies have disproven every point of this so-called definition of the "normal" swallow. For example, Cleall (1965) and others conducted cinefluorographic studies of deglutition in individuals with and without malocclusions and with and without tongue thrust, and found that upwards of 40% of subjects without malocclusion and without tongue thrust do not have molar contact during swallowing, and that a sizable percentage of these adults did not demonstrate lip contact during swallowing. A number of other studies have indicated that the tongue virtually always is in contact with the anterior dentition during rest and during swallowing (Peat, 1968; Fishman, 1969; Hedges, McLean, and Thompson, 1965). Still another study (Lear and Moorrees, 1966) has indicated that the average daily frequency of swallowing is closer to 600 than to 2000 or 3000. It should be clear that the "normal swallow," let alone what any of us might agree to call "tongue thrust," remains to be defined.

I could go on, since the list of issues that are of joint concern to speech pathology/audiology and dentistry is quite long. Some of these issues are not entirely obvious, such as the influence of extraction and the creation of full dentures upon auditory acuity (King, Reid, and Belting, 1970). Others, such as the assessment of lingual forces within the oral cavity and their possible influence upon arch form, have been studied from several standpoints (Proffit, Chastain, and Norton, 1969; Lear and Moorrees, 1969; Lowe, Kydd, and Smith, 1970). There remain, however, a number of issues of very direct relevance to both professions that have received little or no research attention despite the persistence of clinical problems in these areas. Certainly it would be appropriate to ask about the forces or tongue habits that may be incompatible with the health of the periodontal tissues, or about the forces or conditions which lead to stability or instability of full dentures. Perhaps the many questions that we might ask center around the single broad question, "What are the anatomic, physiologic, neurologic, and behavioral substrates requisite to dental health and, if you will, to speech and language health?" Conversely, we might ask what are the true hazards to an effective speech and language mechanism and

an effective dental mechanism. Of equal importance, perhaps, is the determination of those conditions which are hazards to the acquisition of certain skills and those which are hazards to the retention of those skills.


## ACKNOWLEDGMENT

This work was supported by Research Grant DE 02172 from the National Institute of Dental Research.


## REFERENCES


Bosma, J. F., *Symposium on Oral Sensation and Perception.* Springfield, Ill.: Charles C Thomas (1967).

Brandt, Sally D., and Morris, H. L., The linearity of the relationship between articulation errors and velopharyngeal competence. *Cleft Pal. J.*, **2**, 176-183 (1965).

Burdi, A. R., Cephalometric growth analysis of the human upper face during the last two trimesters of gestation. *Amer. J. Anat.*, **125**, 113-122 (1969).

Butler, J. H., and Stallard, R. E., Effect of occlusal relationships on neurophysiological pathways. *J. peridont. Res.*, **4**, 141-151 (1969).

Campos-Giral, R., and Cole, R. M., A method for evaluating the degree of mesial movements of the lateral pharyngeal walls. *Cleft Pal. Bull.*, **13**, 64-66 (1963).

Cole, R. M., Electrical capacitance studies of oral-pharyngeal functions. Chap. 53 in W. Grabb, K. Bzoch, and S. Rosenstein (Eds.), *Cleft Lip and Palate.* Boston: Little, Brown (1971).

Cleall, J. F., Deglutition: A Study of form and function. *Amer. J. Orthodont.*, **51**, 566-594 (1965).

Damon, F., An anteroposterior photographic and cephalometric study of soft tissue and skeletal asymmetry in normal and unilateral cleft lip and palate patients. Master's thesis, Northwestern Univ. (1967).

Fishman, L. S., Postural and dimensional changes in the tongue from rest position to occlusion. *Angle Orthodont.*, **39**, 109-113 (1969).

Fritzell, B., The velopharyngeal muscles in speech. *Acta Otolaryng.*, Suppl., **250**, 1-81 (1969).

Goerttler, K., Die Anordnung, histologie und histogenese der quergestreiften muskulatur im menschlichen stimmband. *Z. Anat. Entw.*, **115**, 352-401 (1950).

Hanson, M. L., Barnard, L. W., and Case, J. L., Tongue-thrust in preschool children. *Amer. J. Orthodont.*, **56**, 60-69 (1969).

Hanson, M. L., Barnard, L. W., and Case, J. L., Tongue-thrust in preschool children. Part II: Dental occlusal patterns. *Amer. J. Orthodont.*, **57**, 15-22 (1970).

Haring, F. N., and McCormack, R. M., A physiometric analysis of lip function in cleft and noncleft subjects. *Cleft Pal. J.*, **1**, 320-328 (1964).

Hedges, R. B., McLean, C. D., and Thompson, F. A., A cinefluorographic study of tongue patterns in function. *Angle Orthodont.*, **35**, 253-268 (1965).

Hirano, M., Ohala, J., and Vennard, W., The function of laryngeal muscles in regulating fundamental frequency and intensity of phonation. *J. Speech Hearing Res.*, **12**, 616-628 (1969).

Humphrey, Tryphena, The prenatal development of mouth-opening and mouth-closure reflexes. *Pediat. Dig.*, **11**, 28-40 (1969).

Husson, R., Théorie de la vibration des cordes vocales. *C. R. Acad. Sci.*, **236**, 1697-1699 (1953).

Isshiki, N., Remarks on mechanism for vocal intensity variation. *J. Speech Hearing Res.*, **12**, 669-672 (1969).

Kelsey, C. A., Ewanowski, S. J., and Crummy, A. B., Techniques for the evaluation of lateral pharyngeal wall motion in cleft palate patients. Paper presented at the meeting of the American Cleft Palate Association, Portland, Oregon (1970).

King, W. H., Reid, P., and Belting, C. M., The influence of extraction and replacement of teeth on hearing. *Pros. Dent.*, **23**, 148-153 (1970).

LEAR, C. S., and MOORREES, C. F., Swallowing frequency: A detection system employing FM telemetry. *J. dent. Res.*, **45**, 1222-1227 (1966).
LEAR, C. S., and MOORREES, C. F., Buccolingual force and dental arch form. *Amer. J. Orthodont.*, **56**, 379-393 (1969).
LOWE, R. D., KYDD, W. L., and SMITH, D. E., Swallowing and resting forces related to lingual flange thickness in removable partial dentures. *Pros. Dent.*, **23**, 279-288 (1970).
LUBKER, J. F., An Electromygraphic-cinefluorographic investigation of velar function during normal speech production. *Cleft Pal. J.*, **5**, 1-18 (1968).
MENYUK, PAUL, and ANDERSON, S., Children's identification and reproduction of /w/, /r/, and /l/. *J. Speech Hearing Res.*, **12**, 39-52 (1969).
MOLL, K. L., and SHRINER, T. H., Preliminary investigation of a new concept of velar activity during speech. *Cleft Pal. J.*, **4**, 58-69 (1967).
PEAT, J. H., A cephalometric study of tongue position. *Amer. J. Orthodont.*, **54**, 339-354 (1968).
PERKELL, J. S., *Physiology of Speech Production.* Cambridge, Mass.: MIT Press (1969).
PROFFIT, W. R., CHASTAIN, B. G., and NORTON, L. A., Linguopalatal pressure in children. *Amer. J. Orthodont.*, **55**, 154-166 (1969).
SHELTON, R. L., JR., ARNDT, W. B., JR., KNOX, A. W., ELBERT, MARY, CHISUM, L., and YOUNGSTROM, K. A., The relationship between nasal sound pressure level and palatopharyngeal closure. *J. Speech Hearing Res.* **12**, 193-198 (1969).
SKOLNICK, M. L., Videofluoroscopic examination of the velopharyngeal portal in lateral and base projections—a new technique for studying velopharyngeal closure during phonation. Paper presented at the meeting of the American Cleft Palate Association, Portland, Oregon (1970).
STOCKLI, P. W., A method for evaluation of maxillary arch form in complete unilateral cleft lip and palate cases. Master's thesis, Northwestern Univ. (1969).
STOCKLI, P. W., Initiale kieferbogenform und deren prognostische bedeutung bei eiseitig totalen lippen-kiefer-gaumen-spalten. *Schweiz. Mschr. Zahnheilk.*, **80**, 605-621 (1970).
TAYLOR, J., *Selected Writings of John Hughlings Jackson.* New York: Basic Books (1958).
WARREN, D. W., Nasal emission of air and velopharyngeal function. *Cleft Pal. J.*, **4**, 148-150 (1967).

# RESPIRATION: GROWTH AND DEVELOPMENT

AREND BOUHUYS

*Yale University School of Medicine, New Haven, Connecticut*

This presentation covers selected aspects of developmental anatomy of the lungs, as well as some problems related to the development of breathing and some specialized respiratory acts. I will not discuss respiratory mechanics in detail, and I will completely avoid gas exchange. These two areas are important for clinical purposes, but I do not see that they are of special interest to a person who is chiefly interested in child development.

## DEVELOPMENT OF THE LUNGS

The lungs begin to form when the human fetus is 3 mm long. A groove forms in the foregut; it ends in a small pouch. This is the primary lung bud, which consists of this pouch and a mass of mesenchymal tissue around it. The foregut grows rapidly, and forms the esophagus and trachea. The primary lung bud divides into a left and a right one, and these in turn divide further to begin the formation of the bronchial tree. During the first four months, the whole tissue mass grows very rapidly, and the cells derived from the entoderm are tall. The tubes are never closed, and thus the tissue is never solid. This is the *glandular period*. Next, after the fourth month, the bronchi divide further and the amount of connective tissue becomes less; in addition, more blood vessels enter the lung. This is called the *canalicular period*. The epithelium becomes somewhat flatter, but there is not yet good contact between the lumen of the tubes and the blood. From about six months onward, the *alveolar period* begins. The epithelium flattens out, and blood vessels come closer to the epithelium. There is still a fair amount of connective tissue between the lumina of the cavities, at least in the beginning of the alveolar period.

There is a dispute among anatomists about the formation of lung alveoli. Some say that all alveoli are formed after birth. Others say that their formation depends mainly on age and maturity of the fetus, and consider birth almost incidental. The main source of the quarrel seems to be a matter of semantics: at what stage does one call superficial, thin-walled formations by the name *alveoli*? An important point, however, is that the lungs are not collapsed before

birth. It used to be thought that the first breath pulls the thin walls of the alveoli apart, as one inflates a thoroughly crumpled toy balloon. However, it has been shown recently that the lungs before birth have the same volume as the lungs directly after the first few breaths. Actually, the lungs are filled with fluid during gestation, and at birth this fluid is rapidly absorbed and replaced by air.

In spite of the quarrel about the beginning of alveolar formation, there is agreement that most of the alveoli are formed after birth. How does this come about? The development of the network of elastic tissue seems to be very important for the growth and development of the lungs, and therefore we must start there. Almost all cells in the body can form *collagen,* fibers made of long molecules, joined end-to-end. About 50% of the solid stuff in lungs is collagen, and the amount does not change a great deal with age. What does change is the quality of the collagen. As we grow older, the collagen molecules develop cross-links, and this makes their network less and less mobile. This decreased collagen mobility may be the main feature of senility. In the lungs, the main function of the collagen network is probably to limit the extent to which the lungs can be inflated. Part of the collagen fibers are converted to *elastic fibers,* and this occurs mostly after birth. There are only a few, around the terminal airways, at birth, but during postnatal growth this network becomes more extensive and dense. They help the lung to recoil during expiration, and Emery believes that the development of the elastic fiber network gives important clues on the development of alveoli after birth. In his recent book, Emery (1969) describes three different ways in which changes in the elastic structure of the lungs may contribute to the development of new alveoli. The most interesting one of the three is perhaps what Emery calls "fragmentation of terminal respiratory passages." After the first postnatal year, the number of airways covered with epithelium decreases, and it is likely that the more peripheral of them are converted into alveoli. The elastic fiber layer in their walls breaks up, and the epithelium protrudes through the newly developed holes in the elastic network. These protrusions become new alveoli; eventually, nothing may remain of the original airway wall but the tips of the alveolar septa.

This view of alveolar development, if correct, has important practical implications. There is evidence that the number of alveoli continues to increase at least until early adulthood (Emery, 1969). If a part of these new alveoli is formed from peripheral airways, early damage to some peripheral airways may cause arrest of alveolar development in that part of the lungs. Such damage might follow not only from, for instance, bronchiolitis in infancy, but also perhaps even from cigarette smoking in adolescence. There is evidence that teen-agers who smoke have relatively poor performance in certain lung function tests, compared with nonsmokers of similar age, sex, and stature. This difference may result from a direct toxic effect of some component of the smoke on airways, but it may also depend on a more indirect effect such as developmental arrest.

The preceding discussion brings us from anatomical data to lung function studies during life. Figure 1 shows some results obtained in our laboratory

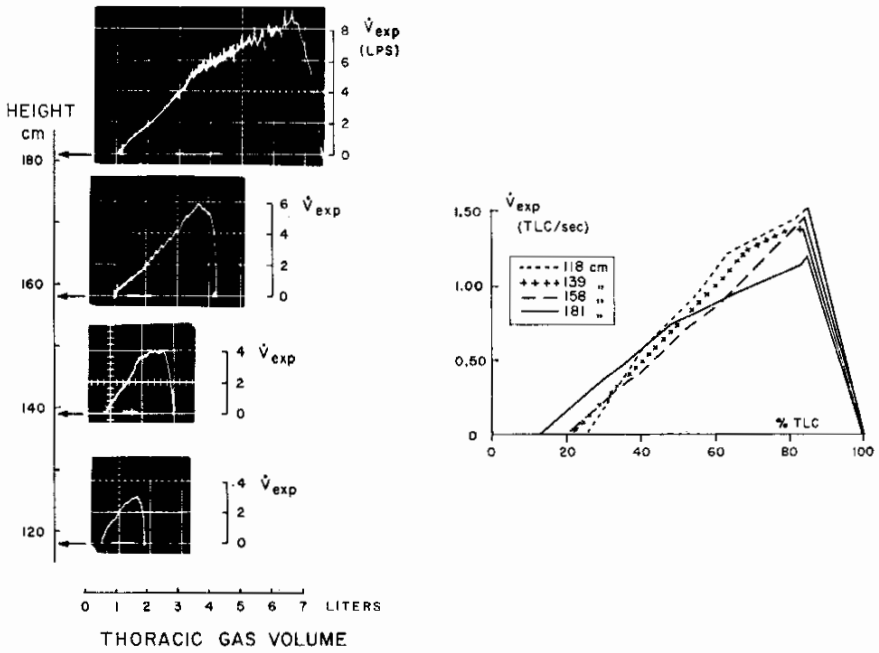



FIGURE 1. Left, MEFV curves of four boys; standing height indicated on scale at left. All curves have been lined up to fit the TGV scale at the bottom. TLC at right-hand side, RV at left-hand side on each curve. Right, MEFV curves of A replotted with lung volume in % TLC on abscissa and maximum flow rates in TLC/sec on ordinate. (From Zapletal et al., 1969.)

(Zapletal et al., 1969). The test consists of a maximally rapid expiration starting with the lungs fully inflated. The subject takes a maximum inspiration, and this brings him to the point at the right-hand side of the graph. Next, he expires as fast and as completely as possible. The abscissa of each graph shows how much he can blow out (volume), and the ordinate shows how fast he can blow the air out (flow rate, volume per unit time). There is an absolute volume scale underneath, and this fits all curves. The small boy has a maximum lung inflation volume of about two liters, and the tallest young man one of about seven liters. This value is called the total lung capacity (TLC). These curves show very well how the lungs grow. Is there any regularity in this process? There is indeed, and this can be seen when one normalizes these curves for lung size. We do this by expressing the volume axis in percentage of TLC. We must also normalize the flow rates, since obviously more air per

unit time can flow out of big lungs than out of small ones. Therefore, we use a new unit for flow rate, that is, TLC/sec (peak flow is about 1.5 TLC/sec for all subjects discussed here). With both axes normalized, the four curves become very closely similar. This fixed relationship of maximum flow rates to TLC is of additional interest if we consider sex differences in growth. During adolescence, both TLC and vital capacity (VC) of the lungs increase more with height in boys than in girls (Zapletal et al., 1969). This may be related to the boys' muscular development, which helps them to increase the volume of their lungs at maximum inspiration. Nearly everything we measure about lungs is some function of lung volume, and since TLC and VC differ, most functional data may differ between boys and girls of similar height. This is also true for the flow rates on maximum expiratory flow volume (MEFV) curves such as those of Figure 1. However, by using units of TLC/sec we can take the difference in lung volumes into account; the sex difference in flow rates then becomes nonsignificant.

Thus, our lung volume data suggest that human lungs grow as if they behave isotropically—that is, as if all linear dimensions increased in equal proportions throughout the period of growth from about age five to adulthood. Although the real process of growth may be much more complex than this statement suggests, it is interesting that such a simple rule can describe at least some aspects of lung growth.

## CONTROL OF BREATHING

To breathe, one needs lungs, but breathing itself is a complicated motor act in which the lungs function only as passive bellows. The frequency and range of motion of the cyclical movements of the chest wall and the diaphragm determine how much fresh air will reach the lung alveoli per unit of time. This, in turn, determines to a large extent the amount of oxygen and carbon dioxide that is exchanged between air and blood. Our breathing movements must be adjusted to suit the requirements of our body for oxygen, and its need to eliminate the waste product, carbon dioxide ($CO_2$). If we do not refresh the lung air at a sufficient rate, we accumulate $CO_2$ in the blood and we develop an oxygen deficit. The former we can stand to some extent, the latter is quickly lethal if severe enough. Strangely enough, we seem to do much of our adjustment of ventilation to altered conditions of gas exchange by sampling arterial blood for $CO_2$, rather than for $O_2$, and we adjust ventilation according to the result of that sample. If the sampler, which is located centrally, close to coordinating centers in the midbrain and medulla, detects too much $CO_2$, it signals to the centers to increase the number of motor signals to respiratory muscles. Increased ventilation then results. If the sampler detects too little $CO_2$, it may tell the centers to stop motor output altogether for a short while. In addition, we have oxygen samplers, but they fulfill an emergency function and come into play mostly when oxygen lack is pronounced, although there are some indications that they are always somewhat active as long as we

breathe room air. But we have to decrease oxygen content of inspired air considerably before we begin to breathe deeper and faster.

The blood gases are not the only controlling quantities. The brain centers also get signals from the lungs themselves, and these tell them about the inflation status of the lungs. Many other factors also modify breathing: just thinking about it may change your breathing pattern. So will pinching your toe or thinking about emotional subjects.

The brain centers send appropriate messages, via motoneurons, to respiratory muscles. We do not use this motor system for breathing only, but also for speaking, laughing, crying, singing, and shouting. We are able to integrate the control of breathing per se with the control of several other motor acts, all of them complex ones in their own right. It would be interesting to speculate on the development of this precisely regulated and integrated motor control, but to do this we must first discuss more elementary aspects of the development of nervous functions.

## FUNCTIONAL MATURATION OF NERVE FIBERS

In addition to somatic motor fibers to respiratory muscles, the breathing apparatus contains autonomous nerve fibers that supply stimuli to the smooth muscle in the walls of airways and blood vessels.

The tenth cranial nerve, the vagus, sends autonomic nerve fibers to many different organs, including the airways. Vagus fibers have been described in the lungs of 10- and 16-cm human fetuses (Emery, 1969), but their terminals on smooth muscle develop later. At birth there appears to be some terminal network, but only in an 8-month-old child was there a complex network of vagus fibers which ramified in the muscle coat of the airways. These data, from a study by Spencer and Loef in 1964 (reported in Emery, 1969), strongly suggest that at birth the development of the peripheral nerve fiber network in the airways is very incomplete. Anatomical studies have shown that, in other organs as well, terminal nerve fiber networks, the sites of neuromuscular impulse transmission, are not fully developed in man at birth.

The details of functional maturation must be studied in experimental animals. In man, behavioral studies can teach us how the system as a whole develops, but we would also like to know which parts of the total system limit development.

In our laboratory, we studied the response of airway smooth muscle and of the heart to vagal and sympathetic stimulation, in immature as well as adult rabbits (Schwieler, Douglas, and Bouhuys, 1970). Figure 2 shows some of the results. In the adult animal (four weeks old) one can stimulate the vagal nerve 150 times per second, and the smooth muscle contracts as long as one maintains stimulation. In contrast, in the seven-day-old rabbit the response begins to decrease at much lower rates of stimulation (30/sec). In this immature animal, some element in the system, that is, nerve fiber-neuromuscular junction-muscle fiber, is already getting "tired" at low rates of stimulation.

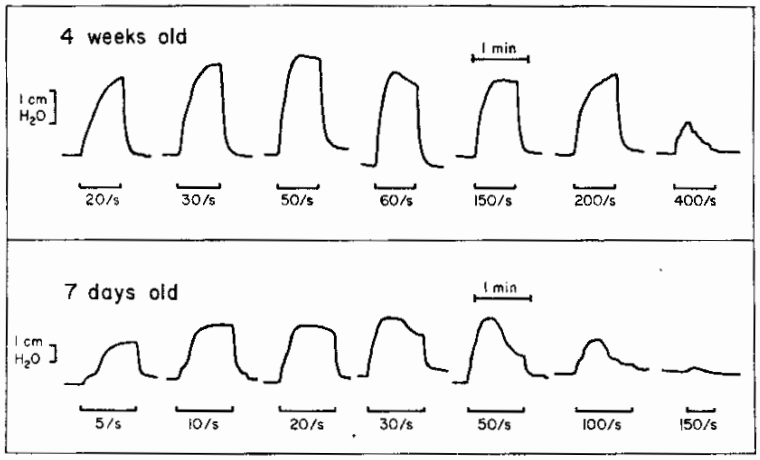



FIGURE 2. Intratracheal pressure changes in response to supramaximal vagal stimulation in a 4-week-old and a 7-day-old rabbit. The contractions are well maintained at stimulation frequencies up to 200 imp/sec in the older animal, but decrease in the younger animal before the end of the stimulation period at 30 and 50 imp/sec, and is reduced in amplitude at 100 imp/sec. Upward deflection = pressure increase. (From Schwieler et al., 1970.)

Other experiments suggest that the limiting factor here is the nerve fiber. The immature nerve fiber is apparently unable to conduct impulses at high rates for prolonged periods of time. There are several reasons for this. One reason is that immature fibers are small, which means that they have a large surface area per unit of volume. Once the membrane has been depolarized, the cellular ion pump has to perform a lot of metabolic work to restore intracellular ion composition to its prestimulus condition. In addition, the pump of immature nerve fibers is not yet in good shape. There are few mitochondria in maturing nerve fibers, and therefore they have an insufficient aerobic machinery. Fiber size and metabolic incompetence are probably both important factors in the functional immaturity of peripheral autonomic nerve fibers. The autonomic fibers to the heart behave similarly, and it seems likely that a similar functional immaturity exists in all peripheral autonomic nerve fibers.

Other studies suggest that somatic nerve fibers and central neurons are similarly immature. For instance, Huttenlocher (1969) found inability to fire at rapid rates in central neurons of immature cats. Thus, this may well be a general property of immature nerve fibers. If this is so, it may be very difficult to find out what element in the nervous system limits the development of motor control. Depending on slight differences in the rate of development, central neurons might be limiting in some cases and peripheral ones in others. The firing rates associated with the input and output signals may be important. Reflex events requiring slow rates of signaling could conceivably develop at an earlier stage than those requiring high rates of firing.

Apart from the properties of nerve fibers, immaturity of the peripheral organ itself may influence the requirements for the control system. The dependence of immature animals upon vagal efferents for their control of ventilation may be an example.

Schwieler (1967) has shown that newborn, decerebrated rabbits with the vagi cut do not increase inspiratory muscle force when the trachea is clamped. Adult animals respond to this procedure with vigorous attempts to inspire, even though their vagi are cut. Apparently, the newborn rabbit gets much of its sensory information on breathing via vagal afferents from the lungs. These afferent impulses come probably mostly from inflation and deflation receptors in lung tissue. These tell the coordinating centers in the midbrain and medulla what the state of lung inflation is. This kind of input appears to be important for an immature animal. Its lungs are still in a relatively unstable state; they are prone to collapse, for instance when surfactant production is insufficient (Avery, 1964). The control of breathing appears to serve, first of all, the goal of keeping the lungs adequately inflated. Appropriate messages reach the centers whenever the lungs are either insufficiently inflated or overinflated, and breathing movements are adjusted accordingly. This type of control, which seems eminently suitable for the immature animal, in a teleological sense, is dependent on intact vagus nerves.

In decerebrated adult animals with the vagus nerves cut, appropriate respiratory responses occur when the trachea is clamped. Apparently, the relevant messages reach the centers via afferent fibers other than those that run in the vagus nerves. The source of these afferents is clear if one considers another of Schwieler's experiments (1967). He recorded the electromyogram (EMG) of inspiratory intercostal muscles in newborn kittens and young cats, before and after sectioning seven successive dorsal roots in the thoracic region of the spinal cord. In the newborn animal, sectioning these roots did not alter the EMG. In the more mature animal, the inspiratory intercostal EMG activity decreased considerably. One concludes from this experiment that the older animals may require afferent signals via fibers in the dorsal roots to keep their motor output to respiratory muscles going. These dorsal root afferents come from specialized muscle fibers, the muscle spindles. Their sensory elements feed information on the contraction status of the muscle to the coordinating centers in the spinal cord, and this information is used to modulate the motor output. For instance, if one clamps the trachea in an animal, the spindle senses a passively increased muscle tension due to the added load. As a response, motor output is increased so that active muscle tension may cope with the added load. The spindle system may thus act as a rapid feedback system, involving only one synapse in its simplest form.

Again, it would seem to make perfect sense that the adult animal depends to a large extent on afferent input from his respiratory muscles to keep his ventilation going. In the adult animal, the lungs collapse only under unusual conditions; this may decrease the importance of vagal afferent signals from

the lungs under normal conditions. On the other hand, the older animal has a much greater range of motor acts (walking, running, posture maintenance) than the immature animal. Many of these other movements involve the respiratory muscles. It is therefore important that the control of the respiratory muscles can be integrated with the control of other motor acts. For instance, if some respiratory muscles change their tension when the animal adopts a different posture, this altered tension can be signaled to the spinal cord and higher coordinating centers by the spindles. These centers can then modify the motor output to the same or to other respiratory muscles, so that the aims of ventilatory control remain fulfilled. The only respiratory muscle which subserves few other needs than those of respiration, the diaphragm, also happens to be the one with the fewest muscle spindles.

## SINGING AND SPEECH

Very precise control of respiratory movements is required for these specialized, skilled acts. Figure 3 illustrates this with a simple example: singing a constant tone, through the full vital capacity range.

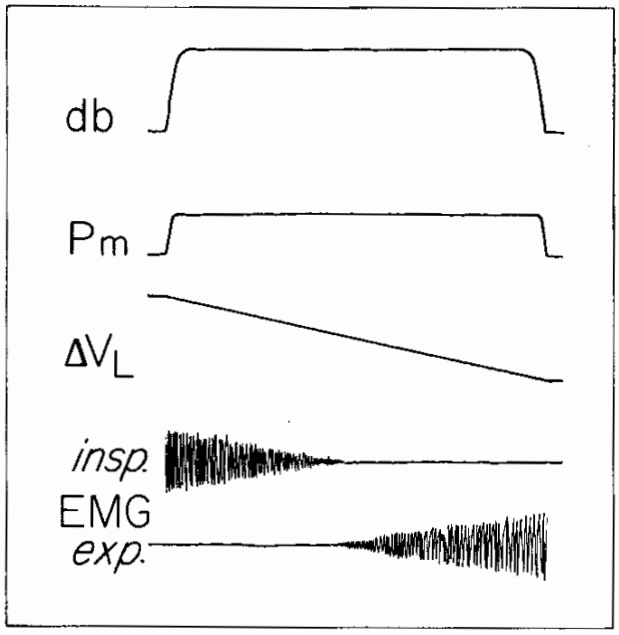



FIGURE 3. Sound level (dB), mouth pressure (Pm), lung volume ($\Delta V_L$) change and electromyogram (EMG) from inspiratory and expiratory intercostal muscles, during production of a constant tone on a wind instrument. EMG pattern based on data of Sears and Newsom-Davis (1968). (From Bouhuys, 1969.)

Initially, the singer must "brake" the expiration in order to keep expiratory air flow at the very low rate required for this tone, and to prevent the subglottic pressure from increasing above the required level. This "braking" is a

function of inspiratory muscles. To let out air at a constant rate and under a low head of pressure, the brake must be released very gradually. Figure 3 shows that the EMG activity of inspiratory muscles decreases as the tone is produced. Approximately at midvolume (in this particular case) a point is reached where the combined elastic recoil of lungs and chest just balances the pressure head required for the tone. At this volume, the system is in equilibrium, with all respiratory muscles relaxed. To continue to sing the tone, one must provide pressure with expiratory muscles, and increasingly so as lung volume decreases further. This is again reflected in the EMG record of Figure 3: expiratory rib cage muscles become increasingly active as the subject approaches the maximum lung volume level. There are simple mechanical explanations for these muscular events; it simply has to be done this way! Further discussions on the mechanics of this process may be found in previous papers (Bouhuys, Proctor, and Mead, 1966; Bouhuys, 1968).

A more complex situation is illustrated in Figure 4, based on work done by Klatt et al. (in Bouhuys, 1968). When we say, "Ha-ha-ha," most air is expired on the consonant, while the sound energy is concentrated in the vowel. Air flow rate varies according to the condition of the glottis: wide open during the consonant, and phonating during the vowel. Subglottic pressures (not

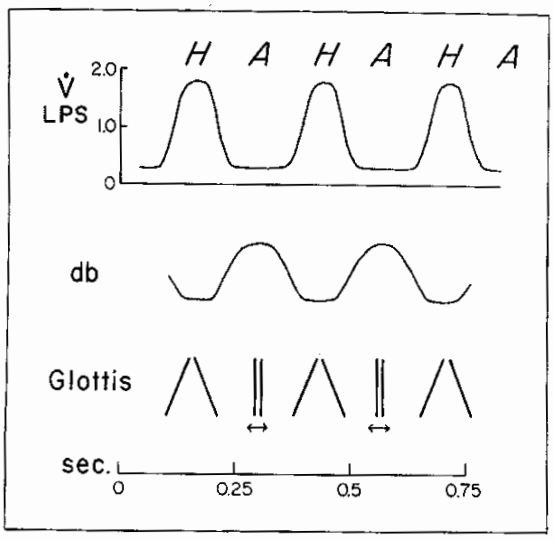



FIGURE 4. $\dot{V}$ = flow rate. For further explanation see text. (From Bouhuys, 1968; copyright, The New York Academy of Sciences; 1968; reprinted by permission.)

recorded in Figure 4) must also vary with glottal configuration. Thus, motor output to respiratory muscles has to be modulated according to the requirements of the glottis, and at the same time the laryngeal muscles must be controlled very accurately. After each *h*, when the vocal folds are wide open, the glottis closes rapidly so that they vibrate at exactly the same frequency

as they did during the previous *a*. Actually, they already vibrate at that rate before they are fully closed (Stevens, in Bouhuys, 1968).

These events occur in very brief spans of time. Some of them are so rapid that time is insufficient for control via reflex pathways. Several neurophysiologists now believe that acts like these are programmed rather than that they evolve through a set of consecutive reflexes. In this concept, sensory inputs from the muscles and other peripheral sensors may serve functions such as error detection, and they may not control the output as long as events proceed according to the learned program. The process of developing the programs for these skilled motor acts takes place when one learns to speak or to sing. Elsewhere I have discussed these aspects of respiratory control in some detail (Bouhuys, 1969).

The mature animal can control his breathing apparatus adequately even under adverse conditions such as heavy exercise or altitude. Man has abilities far superior to those of animals. The integration of his control systems has developed to such a state of sophistication that he is able to keep his arterial blood gas tensions constant within reasonable limits, while using the same motor system and bellows to discuss the events of the day with his friends, and at the same time using a major portion of his grey cells to think about tomorrow!

## REFERENCES


Avery, M. E., *The Lung and Its Disorders in the Newborn Infant.* Philadelphia: W. B. Saunders (1964).

Bouhuys, A., Physiology and musical instruments. *Nature,* **221**, 1199-1204 (1969).

Bouhuys, A. (Ed.), Sound production in man. *Ann.. N.Y. Acad. Sci.,* **155**, Article 1, 1-381 (1968).

Bouhuys, A., Proctor, D. F., and Mead, J., Kinetic aspects of singing. *J. appl. Physiol.,* **21**, 483-496 (1966).

Emery, J. (Ed.), *The Anatomy of the Developing Lung.* Suffolk, England: Lavenham Press (1969).

Huttenlocher, P. R., Functional properties of pyramidal neurons during development of cerebral cortex in the kitten. *Fed. Proc.,* **28**, 825 (1969).

Schwieler, G. H., Some aspects of respiratory regulation during the postnatal development in cats and rabbits. *Life Sci.,* **6**, 1803-1810 (1967).

Schwieler, G. H., Douglas, J. S., and Bouhuys, A., Postnatal development of autonomic efferent innervation in the rabbit. *Amer. J. Physiol.,* **219**, 391-397 (1970).

Sears, T. A., and Newsom-Davis, J., The control of respiratory muscles during voluntary breathing. *Ann. N.Y. Acad. Sci.,* **155**, 183-190 (1968).

Zapletal, A., Motoyama, E. K., van de Woestijne, K. P., Hunt, V. R., and Bouhuys, A., Maximum expiratory flow-volume curves and airway conductance in children and adolescents. *J. appl. Physiol.,* **26**, 308-316 (1969).

# DEVELOPMENT ASPECTS OF SPEECH AND LANGUAGE

HARRIS WINITZ

*University of Missouri, Kansas City, Missouri*

The year 1957 signaled the beginning of a new age in linguistic theory. In that year, Noam Chomsky's *Syntactic Structures*, a thin paperback of less than 120 pages, which in the next decade was to have seismic impact on the many human disciplines, was published. Things just will never be the same again.

Chomsky's intellectual influence has been so great that, without it, the discipline of language development would have continued as an uninteresting chronicle of developmental events whose only common feature was that the responses, events, or measures were described as vocal or verbal in kind. Heralded by many (*Time*, 1968) as a Galileo or Freud in his impact on linguistics, psychology, philosophy, and speech and hearing science, Chomsky's contribution has essentially been twofold: he derived a conceptual system, a generative or transformational grammar, to describe linguistic structures the many products of which have been to question current behavioral approaches to higher-order mental process; and he aroused and stimulated those whose interests lie in resolving the fundamental question of the theory and philosophy of human language.

The priority of the sentence was generally ignored prior to the Chomsky revolution. However, because of his work, speech production models can no longer ignore the syntactic component. Phonological rules clothe the syntactic component into phonetic structure which is issued forth by complex motor command units.

A good way to begin a description of the complexity of the syntactic component is by looking at a simple rule which shares some features with rules of English sentences. Assume you are placed before a computer and a typewriter, and the computer instructs you to type any number of *A*s and any number of *B*s in any order you wish, but that when you type it in a certain order, or a certain way, the computer will respond, *Right*.

So the subject plays the game and decides to type *A*,*A*,*A*,*A*, or *B*,*B*,*B*,*B*, and the computer indicates that in both instances the subject is wrong. The subject tries again. He types three *A*s and three *B*s (*AAA*, *BBB*), and the computer types *Right*. The subject tries again with three *B*s and two *A*s (*BBB*, *AA*), and

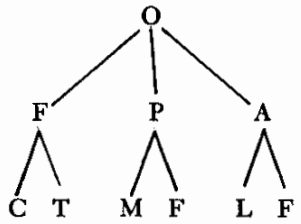
the computer types *Wrong*. The subject types two *A*s and two *B*s (*AA*, *BB*), and the computer types *Right*. The subject tries it in the reverse order, two *B*s and two *A*s (*BB*, *AA*), and the computer signals *Wrong*. Finally, the subject tries four *A*s and four *B*s (*AAAA*, *BBBB*), and the computer types *Right*. Knowing he has the rule, the subject types *Stop* and the computer becomes inactive.

We can symbolize the rule for these correct typings as S → A(S)B, meaning S is rewritten as A(S)B, the parentheses indicating optionality. Note that (S) is imbedded within the boundaries of A . . . B. Imbedding, as in the example above, is a common sentential rule. For example, in "The car . . ." (relative S) ". . . is old," we can form the new sentence "The car which I bought from Tom is old."

Using the preceding example, we can see that imbedding a relative clause more than once results in a nongrammatical sequence: "The car which Tom, whom Mary loved, sold, is parked on the corner." This sentence is not grammatical; that is, we would not recognize this as an English sentence.

Once again let's look at the way in which the arrow, the rewrite rule (→), is used. Assume that you wanted to characterize all the objects in this room. Let's symbolize objects by *O*. Given the *O*, you want to enumerate the furniture, the people, the appliances, and so forth.

You might enumerate as follows: O → FPA where *F* means furniture, *P* means people, and *A* means appliances. *P* can be rewritten as male or female, depending on the categories desired; *F* as chairs and tables; and appliances as lights, fans, and so forth. The tree diagram for these categories would look as follows:

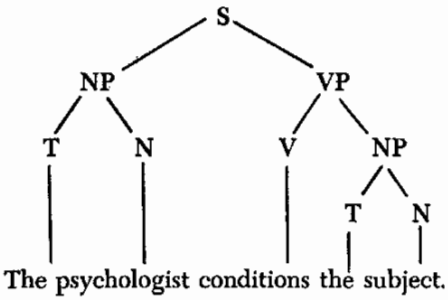



For sentence generation, assume we start with the abstract sentence, denoted S, and that the basic constituents of the sentence are noun phrase and verb phrase, or NP + VP.

The noun and verb phrases are like the categories of furniture, people, and appliances. Using a set of rewrite rules, we can generate the sentence "The psychologist conditions the subject" as follows:

S → NP + VP
NP → T + N
VP → V + NP

The resultant tree diagram is:




Linguistic rules, of which the foregoing is an example, give us the opportunity to describe, in a very explicit way, all of the sentences which we use. We will never reach this goal, but we may come very close to it.

Using a set of complex rules, we may be able to match the speaker-hearer's knowledge of his language. For example, the phrase "the boy the hits monkey" is regarded as nongrammatical. An explicit grammar will tell us that this sentence cannot be used in English.

To illustrate this point further, consider a pair of sentences which has gained notoriety of late:

John is easy to please.
John is eager to please.

On the surface, these sentences might be regarded as similar if we used our high school techniques of parsing. But note you can have:

John is easy to please

and

John is eager to please.

You can have:

It is easy to please John,

but you cannot have:

It is eager to please John.

Why do we regard this last sentence as nongrammatical? Well, the underlying structures of these two sentences are supposedly different, and they're marked in different ways. A complete grammatical analysis of these two sentences would indicate that having said:

John is easy to please,

we can also say:

It is easy to please John,

or

To please John is easy,

or

What pleases John is easy.

Having uttered:

John is eager to please,

we would not construct the following sentences:

It is eager to please John.
To please John is eager.
What pleases John is eager.

On the surface, these utterance pairs are identical in form, but at a very deep and abstract level, they are different, for when "John is easy to please," "John" is the direct object of "please," which is not shown in a superficial or surface analysis. In the case of "John is eager to please," "John" is the subject of "please."

The deep level will show this history of derivation; it will explain that someone is pleasing John when "easy" is the adjective, and John is pleasing someone when "eager" is the adjective. The underlying linguistic structure for

John is easy to please

will be represented at some point by

John is easy for someone to please,

while

John is eager to please

will have an underlying form that might look like

John is eager for John to please.

So we find that these two utterance pairs are complex, no doubt a reason why children take time to acquire them.

Take a look at the next utterance pair:

Bill called up Mary.
Bill called upstairs.

On the surface they look very similar, but "Bill called up Mary" and "Bill called upstairs" would be regarded by all of us as nongrammatical; we would reject it.

Look at the next sequence:

pleasing table
rotting table
dining table

You can have a "very pleasing table," but you cannot have a "very rotting table" or "very dining table." Although you might think of "pleasing, rotting, and dining" as enjoying the same adjectival frame, the use of "very" tells us that these words are not marked in the same way.

On the surface they seem to be the same thing; they're sharing the same slots, they introduce "table," and so on. But when we introduce "very" before these words we find that this is not the case.

Note that we can utter:

a table which is pleasing
a table which is rotting

but not:

a table which is dining.

Somewhere along the way we've learned the rules for grammatical constructions and we use them with great ease.

Take a look at the next pair:

Dale likes Kansas City as much as Betty

implies Dale likes Kansas City as much as Betty likes Kansas City, but not Dale likes Kansas City as much as Betty likes Ann Arbor.

Perhaps it might mean that Dale likes Kansas City as much as he likes Betty. In any event, a deletion has taken place. Originally, children learn that the above sentence means:

Dale likes Kansas City as much as Betty likes Kansas City,

and they also learn a deletion principle allowing for occasional instances of ambiguity.

Examine voice (passive or active) in

Jennifer likes Flora Sue.
Flora Sue is liked by Jennifer.

Note that if we use the word "resemble" the passive construction is rejected:

Jennifer resembles Flora Sue.
Flora Sue is resembled by Jennifer.

How complex it is just to make a passive sentence!

Linguistic devices which reveal the abstract and complex nature of sentences are called *transformations*. A transformation is a rather complex linguistic device, a set of rules, some of which have been introduced in the preceding material. With regard to the development of transformations, "An analysis of the discourse patterns between child and parent may be the richest data for the discovery of grammar," Roger Brown (1968) believes. The abstract manipulations of deep structures may develop as a result of certain instructional patterns imposed on the child.

Consider the transformational character of the *Wh* questions. A superficial description of the underlying strings would involve the assignment of *Wh* to the noun phrase to be questioned, and permutation of the *Wh* to the front of the sentence, as I shall describe. A child will often utter sentences which seem to match this underlying process. His speech will be regarded as defective,

substandard, or immature. Presumably, these variations reflect the child's outward production of a rule which he is internalizing, or which he is learning at this time. Possibly, hints as to how mastery of internal structures is obtained can be gained by examining in detail the verbal exchanges between child and parent, and relating these to linguistic abstracts.

Brown (1968) suggests that the occasion form of *Wh* questions may weigh heavily in the development of the abstract structures of the normal form of the *Wh* questions.

In English we usually use the normal question, such as:

What will John read?

and

Where will John read?

But occasionally we will say:

John will read what?

and

John will read where?

I would guess that we would use the second type of question if we didn't understand a reply because of cocktail noise or other interfering associations.

Linguistic theory suggests that normal questions are generated from occasional questions, really the underlying forms common to both questions.

Note the next example for a sketch of this preposing process:

John will read what?
What John will read?
What will John read?

Roger Brown has observed that the occasional question is relatively frequent in mother-child conversations, far more frequent than occurs in English in general. It apparently clarifies children's solicitations most efficiently.

Children's requests, possibly expressed as "I want milk," or "I want it," usually stimulate parental replies like "You want what?" This verbal interchange seems to tell a child that "what" can replace specific instances as well as general instances of nouns; "what" for "milk" and "what" for "it."

Brown's subjects said sentences that were similar in form to:

What I want milk.

and

What I want it.

Presumably, these response types indicate that children learn the preposing processes of the *Wh*-questions.

This study by Roger Brown illustrates a valuable experimental routine, since "discourse patterns which are rich in structural information may constitute the basis of a learning process" (Brown, 1968).

This valuable finding has been quickly incorporated into the routines of

speech clinicians. A language development clinician realizes that at certain points in the language training sessions, sentences like "You want what?" and "You want where?" are preferred to sentences like "What do you want?" and "Where do you want it?" More adequate understanding of the development of transformations will allow precise presentations of stimuli in clinic lessons.

What did language development look like before the Chomsky revolution? It was the organization of the development of language around certain structures. For example, we had norms which expressed sentence length, giving results like: at 1½ years the child is saying a sentence which is approximately 1.2 words or longer; at two years, he says sentences of 1.8 words; and by eight years, 7.6 words in length. We ignored, however, the primacy of syntactic (also semantic) rules, as we chronicled the development of language.

I have emphasized here the cognitive side of speech and language development and have left unmentioned the physiological determiners of speech development, perhaps an unfair gesture in a conference on oral-facial development. It was my way of saying that speech utterances reflect an abstract psycholinguistic apparatus of which the peripheral print-out (articulation) is only one part.


## ACKNOWLEDGMENT

Supported by Grants 5-K04 HD 38907 and R01 HD 3571 from the National Institute of Child Health and Human Development.


## REFERENCES


BROWN, R., The development of the *Wh* question in child speech. *J. verb. Learn. verb. Behav.*, **7**, 279-290 (1968).

CHOMSKY, N., *Syntactic Structures*. The Hague: Mouton (1957).

Academic discipline. *Time*, **91**, 69-70 (February 16, 1968).

# PSYCHOLOGICAL DEVELOPMENT

DONALD G. KRUPER

*School of Dental Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania*

Psychological development is generally discussed from viewpoints of ages and stages, but a different emphasis will be explored in this discussion. The genetic contribution to a person's development will be accepted as a fact, and primary importance will be placed upon a person's interaction with his world. Therefore, behavioral interactions of many kinds will be the subject of this paper.

Since the reasons for the conference upon which this *Report* is based include a special concern with the orofacial complex, I shall attempt to dwell on environmental influences upon behavior which have special relevance to those interests. Special concern with orofacial factors can have full meaning only when cast in a framework that shows the person as just that—a whole, individual person. As professionals with special interests in segments of people's behavior or segments of their bodies, we can easily allow this specialization to focus too finely on those areas, at the expense of the larger unity—the person. This paper will, no doubt, show some of the same tendency. I hope that it will show an effort to surmount that tendency.

As suggested, the paper will deal with factors that influence psychological development. It is important at the outset to make clear that the factors which influence development or change in a person's commerce with his world may not always have positive consequences for that development.

To avoid giving more meaning to the term *factor* than is intended, it may be useful to think of a concept that can be expressed by the phrase *consequence of behavior*. Parents, teachers, speech pathologists, dentists, or indeed all who interact with another person help to determine what consequences will follow particular behaviors of that person. In a sense then, some of the factors which influence development or change are those things which we do as a result of the behaviors of others. There is, in fact, a close interaction between our own development and the development of others with whom we interact.

My purpose in this paper is to share with others who have some communality of professional interests, some data, some ideas, and some feelings about change in behavior and the consequences of behavior. These will include tests of hypotheses, applications of behavior principles, research ideas, and the like.

Over the past half century the influence of Sigmund Freud has been important and pervasive. Some may thank him for demanding that we accept a

simple dictum that behavior is caused. Others have condemned him for generating hypotheses that do not seem to be testable by experiment. History has confirmed his stand on behavioral causation, and progress is being made in experimental confirmation or rejection of some of his other hypotheses.

A big step, taken in 1950 by Dollard and Miller, was to translate some Freudian concepts into the terms of learning theory. As one example, the concept of transference has a considerable degree of credibility and is an important phenomenon to anyone offering therapies to patients or clients. Transferring feelings, that were once appropriately associated with a parent, to some new authority figure seems to become even more credible when analyzed as an example of a response to stimuli that are generalized from the original conditioned stimuli. In any given situation there are a multitude of potential stimulus elements. When a situation appears later, in which stimuli of a highly similar nature occur, one would predict a similarity in the responses that will also occur. Stated in this way, one has an option for experimental analysis of a phenomenon that may fit into a larger behavioral system. Experimental analysis offers us a better chance to gain understanding and control than the acceptance of a phenomenon as an entity and explanation in itself.

Perhaps one of the most recent, interesting tests of a Freudian assumption comes from the field of dentistry. Psychoanalytic theory argues that persistent thumb sucking is a sign of deeper emotional troubles, that the sucking reduces anxiety associated with those deep troubles, and that it will be replaced by some other symptom if it is altered without removing the deeper problem also (Davidson et al., 1967). However, consistent experimental support for that position has not been presented. On the other hand, behavior theorists see persistent thumb or finger sucking as a habit that has not yet been extinguished. Reinforcement maintains the habit. One way to change the behavior is to allow the response to occur but to remove the reinforcement. Although application of the behavioral principle of extinction should decrease the sucking, what about symptom substitution? Based on behavior theory there should be none, since, in a sense, the symptom is the disease.

Considering these positions, the experimenters ask two questions. First, would application of extinction lead to dropping the habit? Secondly, would more symptoms be found in patients who had treatment, as compared to patients without treatment or children who were not thumb or finger suckers? The experimental approach was to use a palatal crib which greatly changed the usual stimulation, and thus presumably removed reinforcement. They established, with the use of various relatively standard psychological tests for children, that thumb suckers did not show anxiety levels higher than those expected from children who were not thumb suckers. Such a finding immediately questions the presence of deep problems in thumb suckers.

Then they studied 65 thumb-sucking children from 4 to 12 years in age. In 22, the palatal crib was used and in 43 no cribs were used. Twelve months later, 5 of the 22 with the crib had mannerisms (symptoms) other than sucking, whereas 12 of the 43 without the crib showed similar mannerisms. Thus

they reported no significant differences between the groups in symptom substitution. Apparently, new symptoms do not necessarily follow treatment. Incidentally, behavioral extinction from the use of the palatal crib had removed the thumb-sucking habit from all 22 treated subjects. When checked as long as three years later only two of these had relapsed.

Haryett, Hansen, and Davidson (1970) recently reported another study. They found that 6 to 10 months of treatment with a palatal crib, either with or without spurs, was effective for arresting sucking in a majority of cases. Once more, there was no sign of symptom substitution. In this study they pointed out some disadvantages of the method: a temporary upset, with sleeplessness, some difficulty with speech, and some difficulty with eating. None of these problems was long lasting.

Habits involving the tongue, lips, and cheeks are other behaviors that, with the proper ingenuity, should be amenable to alteration by the application of behavioral principles. Fischer (1969) pointed out that anything which will help bring the habit to a conscious level may be effective in changing the habit. However, he failed to deal with the question of continued influences, of how to get a transfer of the new learning, and how to maintain the new level. Hanson (1967) emphasized the latter factor when he stressed the value of charts, signs, and signals for carry-over.

If one concentrates on an effective use of a chart, for instance, several principles that underlie the effects of reinforcers on behavior can be applied to help transfer and maintain behavior changes. Charts are used in our orthodontic department, as is probably common elsewhere, to record the hours patients wear orthodontic appliances. The patient is instructed to wear the appliance for a certain amount of time each day. To make this instruction easier to remember and to carry out, the patient is provided with a chart on which he is to plot the actual times of wearing. When the patient returns for his dental visits he is rewarded by attention and commendation for the wearing and for the charting.

Behaviorally, there are several distinct advantages that should accrue from this procedure. Each point that the patient plots should reinforce his wearing behavior by some small amount. The charted increments can provide immediate positive reinforcement for compliance. When the dentist shows interest, satisfaction, or pleasure, verbally and nonverbally, while viewing the progress chart which the patient brings with him, he is providing additional positive reinforcement for the procedures of chart keeping and appliance wearing. Another source of reinforcement is the long-term improvement of the original dental problems, as viewed by both the patient and the dentist. The probable final reinforcements are derived from more positive social interactions, favorable comments, feelings of relief, a shorter length of treatment time, and so on, as the problem is decreased.

There are potential weaknesses if full cooperation is not achieved and if the dentist isn't alert to additional feedback about compliance. Parents may be of help here. It should be clear, however, that in each case there are certain

events that follow certain behavior. To be able to use these events or consequences, we need to be aware of them and of their potentially reinforcing effects.

The basic procedure just described is currently being applied in therapy for correction of tongue thrusting in one young patient that I know very well. At the speech clinic he learned new swallowing patterns, but apparently was not provided with a plan for the necessary continuation of those behavior patterns. The charting procedure with appropriate attention, praise, and so on, was later applied. The prescribed treatment is now guaranteed a better chance of fulfilling its promise.

Although other examples of similar applications of behavioral principles will be given later in the paper, there are two more that seem appropriate to mention now. Both are based upon a fundamental fact that consequences which directly follow behavior will effect similar behavior.

In a summer camp, children were allowed to go swimming only when they had brushed their teeth after lunch, but that requirement was never stated verbally (Lattal, 1969). As long as the consequences (swimming) depended on tooth brushing, brushing occurred. Before establishing that contingency they had virtually never brushed after lunch. When the contingency was removed they returned to the no-brushing level. The actual relationship of brushing to swimming was never described to the children, but the effect was clear.

That example sounds like common sense and is certainly not very dramatic. Application of a similar principle of reinforcement in another situation, however, is dramatic and sounds much less like good, common sense. Wright, Nunnery, Eichel, and Scott (1968) tackled a relatively uncommon, but serious, problem called tracheostomy addiction.

Their report dealt with two, eight-month-old infants, one with laryngeal malformation (cannulated at the age of one week) and the other with respiratory arrest associated with encephalitis (cannulated at age seven weeks). The problem was that after initial treatment when the patients should have been physiologically ready to go back to regular breathing, they did not do so.

The authors believed that learning was the chief factor in the infants' continued dependence upon the tracheostomy. If this was so, they reasoned, reinforcement principles should be a means to return the infants to normal breathing behaviors. First, reinforcers were identified for each child: for one, his walker, rough-housing with the father, "tender, loving care," and so on; and for the other, "tender, loving care" and contact with toys.

The basic procedure was to provide a minimum of the reinforcers during the time that the cannula was open, but to maximize the reinforcers for the initially very brief time during which the cannula was occluded. Next, the time for occlusion and stimulation was slowly lengthened. At first the occlusion times were for three minutes or less, then were slowly stretched to 30 minutes, one hour, five hours, and so on, until the cannula was removed. In brief, it worked, and was considered a great improvement over the long-term use of cannulas reported by others. One case cited was for 12 years.

Before going on to some research and application of these principles to speech and audiology, I would like to identify two especially interesting and effective aids to teaching the fundamentals of a reinforcement theory. Fred S. Keller has written a delightful and informative paperback (Keller, 1969) and Ellen P. Reese (1966)[1] has produced a fine set of four movies on the same subject. These sources can be of considerable help to us in our functions as clinicians, teachers, or advisers. Let us not forget that psychological development never ends, and whether we are involved with treatment or teaching we will be interacting with others at important times.

One of the most interesting aspects of preparing this paper has been a realization of the degree to which behavioral principles have already been adopted as useful to some of the problems in the disciplines of speech and audiology. A large part of the work appears to concentrate on aspects of stuttering and ranges from an experimental analysis of that behavior to the production of a portable tactile-stimulation apparatus, to be used for pacing vocal production of words (Azrin, Jones, and Flye, 1968; Flanagan, Goldiamond, and Azrin, 1958, 1959; Goldiamond, 1962, 1965; Martin and Siegel, 1966a, 1966b; Quist and Martin, 1967; Shames and Sherrick, 1963).

At least two recent papers have directly focused on the use of an experimental analysis of behavior and the use of behavioral principles in the area of clinical speech problems (Brookshire, 1967; Holland, 1967). These two papers are among those that should be required reading for anyone motivated to apply behavioral principles to speech or audiology problems.

Of course, these principles may be applied to research problems as well as to clinical problems. Stebbins (1966) and Stebbins and Miller (1964), for example, have used behavioral reinforcement techniques to study auditory functions in monkeys. The particular challenge in work of this kind with animals is to communicate to them what they must do. For instance, it takes some special skills to tell a monkey, "Hold down that lever until you are signaled to release it, and then release it as fast as possible." This may not be a speech problem, but it is certainly a language problem!

While there is no particular reason to go into the details of the research here, the Stebbins and Miller project shows another example of the range of problems that can be examined if one chooses to develop skills in this area of behavioral research. With such skills, or in association with someone in that research area, it may be possible to find solutions to one's own research problems.

As another example, one may be able to capitalize on the use of informational feedback, as Yules and Chase (1969) did when they used visual feedback about throat-muscle movements. The feedback was generated indirectly by acoustical energy detected at the nose and lips. The set-up was used as a training device to reduce nasal air leakage and to eliminate hypernasality. Other possibilities exist for similar application.

[1] Reese, Ellen P., "Behavior Theory in Practice," films. New York: Appleton-Century-Crofts (1966).

Couldn't one build devices that respond when the lips take a particular configuration, when the pressures of the tongue move to a particular position, when a desired increase in vocal intensity is achieved, or when an increased pressure of tooth contact is sensed? Couldn't these sensors feed back information to let the subject know when the performance is appropriate, and couldn't specific reinforcement contingencies be tied to the desired kind of behavioral output? The answer to all of these questions is almost certainly yes. Only the right kind of motivation and the right kind of ingenuity are required.

With the kind of mechanical intervention that is being suggested here, how can the researcher possibly be concerned with the patient as anything but an extension of all of these proposed gadgets? This would be a reasonable question if it were true that our concern is limited to testing the devices.

However, because of the discrete control of the timing of reinforcements, the accurate record of ongoing performance, and the quick feedback derived from the use of devices, one expects that skilled performance will be more effectively learned with, than without, such devices. More effective solutions to the presenting problems almost certainly will mean more effective psychological growth for the individual involved. It is not necessarily true than an automated approach will make person-to-person interaction less important. In fact, there are reasons to believe that it may improve the clinician-to-patient relationship.

The question of concern with the complete individual patient happens to be one of the high-priority items in dental education today. Around the country we find an increasing number of departments of community or social dentistry or departments of behavioral sciences in dental schools. Ten years ago, Blackerby (1960) asked, "Why not a Department of Social Dentistry?" While I cannot say that he was the first to ask that question, nor can I say that no one had already moved in that direction, I can, with safety, say that his question was a rallying point for discussion and action.

Over the past seven to 10 years there has been a steady growth of interrelationships between people in dentistry and those in psychology, sociology, education, public health, and quite obviously also with people in speech and audiology. Apparently, only a few dental schools have active teaching programs in the behavioral sciences, but more are developing programs each year. Much of this development seems to be in answer to a call to dentistry for even greater personal and social concern. Leaders in dental education and government and even the public seem to be making this call.

There are many reasons for this concern. I will not attempt to document this subject but will cite only one example as a source of this sentiment.

A psychologist, D. M. Morc (1962), extensively studied a large number of dental students in 1958 and 1962. He pointed out that students rarely saw handling of patients to be a serious problem for them. Furthermore, his observations led him to state, "They exhibit an air of omniscience in handling difficult emotional reactions that is positively amazing to anyone with training in the field of psychology and psychiatry" (p. 132). No doubt some of the interest

in bringing the behavioral sciences to dental students represents a reaction to this kind of evaluation.

Wechstein (1968) has clearly pointed out an attempt to lead dental students toward a person orientation rather than toward a task orientation. He suggests that the dentist ask himself a series of questions about each patient. These would be of value to anyone in a helping profession that offers individual treatment:

1. Is the patient frightened?
2. Will there be pain or some unacceptable change?
3. Has the patient had enough time to adjust to the idea of change?
4. Is the patient angry; at adults, parents, authority?
5. Are his expectations of therapy realistic?
6. Does he see me as some kind of god?

While this series of questions is related to knowledge of the patient, we cannot really stop there. Self knowledge may be as important as, and perhaps even more important than, knowledge about others. What makes me angry? What kind of statements or behaviors make me anxious? Are there things that I really fear? What can I do about some of these feelings? This list could be expanded, but the important factor is to be aware that these kinds of questions need to be asked.

Fright, fear, and anxiety are words that have a tradition in dentistry. Despite effective anesthetic procedures, new equipment, and sincere educational efforts by many dentists, these feelings and expectations seem to linger. Whether it is a so-called cultural lag or whether there are better explanations, fear of dental treatment persists at a seemingly high level. It will not be possible here to specify in any comprehensive way the large number of variables that may affect any individual as he develops the fears and anxieties that tend to beset him. However, I would like to weave in the results of a few research studies that, although focusing on the whole person, have special relevance to the orofacial region.

The now classic study of Janis and Feshbach (1953) seemed to show that a high level of fear, generated by a vivid depiction of decayed teeth and diseased gums, did not result in a pronounced change in attitudes toward better dental care. A minimum fear appeal seemed to produce the most change on attitude scale measurements. I do not cite this particular study to push for or against fear appeal to change attitudes, but to contrast its method of procedure with a different emphasis upon observable behavior. If we assume that an attitude is a propensity to action of a certain kind, why not look more directly at the actions?

A recent study in persuasive communications moves one step closer to measuring the actual change desired (Evans et al., 1968). In this study, the experimenters probably were not concerned with measurable changes in attitude. They wanted to see whether subjects' teeth were cleaner after the subjects heard statements about the positive consequences of brushing or the

negative consequences of not brushing. They found that either appeal was better than a control discussion of science and dentistry in general. The most important aspect of this procedure was the use of an objective measurement of differences in the amount of stained plaque as seen on photographs taken before and after the communications. The inference, of course, is that the actual target behavior, brushing (or better, cleaning the teeth) is what was being influenced and that that behavior was of more importance than a stated attitude about cleaning teeth.

In two more studies, we can see some potential weaknesses of attending to attitude rather than behavior itself. Evans (1967) cites the Friedson and Feldman report that, although 88% of a large national sample of people believed it was prudent to see a dentist at least once a year, 51% said that they had not seen a dentist within the past year. Evans also cites a report by Kegeles which concluded that the best single predictor of a patient's attention to dental care was his past behavior rather than his attitude toward care.

Lest it appear that this paper is moving completely away from a concern with psychological development, let me hasten to emphasize that observable behavior is an important aspect of development, although certainly not the totality of it. If we are to evaluate baseline levels of development before certain therapies, changes in development as the result of therapies, and levels of maintained change, observable behavior ranks high on any scale of importance. True, much goes on inside a person which is never observable, so we must not make a fetish out of observable behavior. On the other hand, one does want to stay with methods that succeed, and this kind of approach is enjoying considerable success in psychology today.

Earlier, this discussion dealt with fear in dentistry. I am simply not knowledgeable enough about speech and audiology to appreciate how important fear may be as a problem in those areas. Based on a minimal interaction with some of the people in our Cleft Palate Research Center at Pittsburgh, I have inferred that anxieties or fears are highly variable among patients and their families, and may represent problems.

While there are many ways one might choose to deal with fears, depending on their degree of incapacitating or bothersome effect, I would like to describe one procedure now used by psychologists at the University of Buffalo. Gale and Ayer (1969) reported a treatment for dental fears called *reciprocal inhibition.*

As a first step, the clinician teaches the patient how to achieve a level of deep relaxation. Next, the clinician and patient together construct a list of anxiety-arousing stimuli or stimulus situations. The list is then ordered from least to highest in anxiety arousal level. The stimuli might be, for example, the building housing the dental office, the sign on the office door, the waiting room, or the dental chair.

The next step is to have the patient relax and then visualize the least anxiety-producing stimulus. When relaxation can be maintained with the image of that stimulus, the next higher one is visualized, and so on. The visualizations

have been found to be a surprisingly good substitute for the actual stimuli, and may be all that is needed to enable the patient to face the actual anxiety-producing stimuli without fear.

Because of our particular professional training, there are probably few of us who will practice reciprocal inhibition, or systematic desensitization, as it is also known; but further information about the procedure can be found in books by Joseph Wolpe (1958, 1969).

In a sense, the desensitization procedure boils down to removing some of the excess emotional baggage that the individual has acquired in response to the influences of his environment. Since such a large part of what we acquire has noxious components, I would like to pursue some additional research in the area of stress and anxiety. Once again, the setting will be the dental operatory.

A major difficulty for psychological stress research in a dental setting has been to get a reproducible set of independent variables. One cannot do the same kind of cavity preparation on each subject, whether he needs it or not, so Corah and Pantera (1968) chose to devise a videotaped simulation of a specific dental operation. First, a television camera was positioned at the point in a dental chair where a patient's eyes would normally be. Then the dentist proceeded as authentically as possible to inject anesthetic, to drill, to use a hand instrument, and to place and to carve a Class I amalgam restoration. The camera recorded these procedures as a patient would view them.

To add additional realism, appropriate sounds, as they would be heard by a patient, were dubbed in. These sounds were recorded by placing a miniature microphone in the ear of a patient who did, in fact, receive such a restoration. Thus, the final tape contained the sights and the sounds, very nearly as they would be perceived by a patient.

Then subjects who were measurably different on other dimensions could be studied for differential response to the standard stimulus conditions. For instance, Corah (1969a, b) also developed a four-item anxiety scale (DAS) that he later used with the video simulation procedure. He found that subjects who previously scored high on the DAS reported more discomfort while viewing the videotape than subjects who had scored low in the test. He also indicated that all subjects reported more discomfort when exposed to the simulation than when exposed to a control tape on how to build a boat.

Corah's work is exciting because it not only offers an important improvement in experimental control of stimuli, but also because it has a reasonable degree of face validity for a dental setting.

There is another procedure in which videotapes might be a useful evaluation tool. Two of our first-year dental students at the University of Pittsburgh have designed and built a small device that contains two lights and two switches. The switches are put under the control of a child patient. One switch, which is easily depressed, turns on a small, green jewel light; the other switch, with greater resistance to closure, turns on a larger, red light. The patient is instructed to press the green light switch only when he feels discomfort but is

not too bothered by it. To the dentist the green light means caution, but it also means to continue the dental treatment. The patient is instructed that, should the discomfort become too great, he should press the red light switch. The red light will lead to the dentist's stopping. In brief, the device provides a communication system. Importantly, it also has some additional good qualities. It gives the patient a degree of control which probably has more immediacy and clarity than the usual procedures. It may also provide a distraction that might help the patient to remain occupied with some specific behaviors that are compatible with treatment, instead of, let's say, pulling at the dentist's hands or pushing him away. The latter behaviors are obviously incompatible with continued effective treatment. At this point the efficacy of this communication system has been demonstrated only in part. Research necessary to establish the value of the system will probably include videotape recordings. Our department is certainly interested in stimulating this kind of activity in dental students.

I am delighted about the research just described—not because this is earth-shaking research, but because this is an indication of motivation to change, motivation to improve. Students initiated the idea. Others of us are having the opportunity to interact with these students to help them develop their ideas and to help them learn some additional research skills. We believe that this is one direction dental education should be taking.

Motivation is a subject alluded to only a few times so far in this paper. Yet, in the psychology of development, motivation must certainly be one of the most important facets of anyone's life. What is motivation? From where does it come? What does it do? How much of it is there? What kinds of things happen to it over time?

There are many more questions to be asked about motivation and little assurance that many of the answers are known. There is probably no reason to try to answer many of those questions at this time. However, to ignore motivation would be an error. Therefore, I will try to make a few statements, which I hope will be relevant to the purpose of this *Report*.

Correctly or incorrectly, it might be useful to assume that motivation is a hypothetical construct, a concept that exists in the sense of function but not in the sense of material. This assumption leads us to look at, to identify, and to state what conditions lead to what changes in behavior. It focuses upon Freud's idea of behavior having causes, but also calls for identification of those environmental events over which some control is exercised. In the Skinnerian sense, we recognize that behavior operates on the environment to generate consequences and that the direction of that behavior will be influenced by the consequences which follow it.

White (1961) has written an excellent article on motivation, in which he discusses the shortcomings of Hull's "drives" and Freud's "instincts" as sufficiently comprehensive explanations of behavior. White does not deny the importance of sex, hunger, thirst, and so on, as forces in the directing of behavior. However, as have others, he has tried to find better reasons for

behaviors such as touching, visually exploring, solving problems, manipulating, running, climbing, and playing. Each of these falls into a category of behaviors which interact in some way with the environment but which do not appear to depend upon any physiological, tissue need. For explaining these kinds of behaviors, White uses a concept called *competence.* He defines competence as "an organism's capacity to interact effectively with its environment" (p. 279).

If one attempts to reduce this functional kind of definition to a lower physiological level, one may derive speculative possibilities. One possibility is that any stimulation alters the level of activity of the central nervous system, either increasing or decreasing some over-all state. It is conceivable that any change from a given state, regardless of the actual source of stimulation, may be reinforcing to the organism. For instance, if the organism is understimulated and nervous system activity is at some particular level, any action which increases over-all stimulation will be reinforcing. On the other hand, if the level of stimulation is at a high level, which may be stressful to the organism, a reduction in stimulation will be reinforcing. Thus, in looking at consequences of behavior and assuming such a speculative system, one may infer that the electrical activity of consequences may be fed back through the nervous system to reinforce the behavior which generated the consequences, even though no apparent organic needs are identified.

One does not need the previous reductionistic argument to give strength to White's concept of competence. Merely observing people, especially children, can provide the impression that learning to manage the environment—whether walking, riding a bike, playing an instrument, learning to write, or any number of similar skills—has a rewarding quality, simply in the accomplishment. There seems to be reinforcement in achievement.

Assuming the last statement to be a fact clearly points to constructive things we can do to aid a healthy psychological development. In short, we can try to guarantee an environment in which those thousands and thousands of achievements can take place.

How nicely the concept of competence fits with a concept of motivation which depends upon the environmental consequences of behavior. How nicely both fit with a tendency to stress the influences of the environment upon psychological development. Perhaps they fit too easily, but such a simplistic view is helpful to me in trying to make decisions about behavior. It seems to help in dealing with my children and my students, and presumably, in other interpersonal interactions also.

But what about some other aspects of our influence upon behavior? Isn't planned influence an attempt to control, an attempt to manipulate? What kind of questions must be asked if one proposes intentional manipulation of people's behavior? These questions have led to considerable debate. Although a comprehensive discussion is not called for here, some differing points of view should be stated.

Rogers (1966) agrees with Skinner (1966) that man has always tried to

predict, to control, and to understand behavior, and that as a result of scientific development in the area of behavior the capacity to predict and to control is increasing. On the other hand, Rogers believes that men are free in some sense that will make complete scientific knowledge of man's behavior impossible. He sees man in a process of becoming something more than he is at any given moment, of being involved in a self-actualizing process which moves him to more challenging and more enriching experiences.

A key fact that must be considered in thinking of a given man's freedom of choice, however, is that he does not exist in a vacuum. He is, more often than not, interacting with other people and in many different kinds of situations. His behavior in these situations is being reinforced, positively or negatively, by the consequences which follow his behavior. He is being controlled, intentionally or unintentionally, by those consequences. He can indeed be a victim of someone's manipulation, and all the more so if he is unaware of what is happening and how it is happening.

We humans don't like to be manipulated, especially if it involves threats or other aversive stimuli. We seem to be less disturbed by control over us that may result from praise or attention, especially from friends, family, peers, or the community. We may, in a sense, even encourage some degree of control when we see it as having some benefit to us, as in psychotherapy, speech therapy, and dental treatment.

Ignoring the kind of influential interactions that I have discussed in the last few paragraphs will not make any of them less effective or any less important to the psychological development of either ourselves or the others with whom we interact. I sometimes cringe at the thought of being under the control of some super behavioral scientists employed by an army or a government. At such times I take hope in the belief that there is protection against such an outcome. That protection is knowledge about the variables which control behavior. I believe that knowledge and ability to apply that knowledge offers the best protection for all of us. If there is a degree of free choice, it must surely reside in the options which knowledge may provide.

## REFERENCES


AZRIN, N., JONES, R. J., and FLYE, B., A synchronization effect and its application to stuttering by a portable apparatus. *J. appl. behav. Anal.*, **1**, 283-295 (1968).

BLACKERBY, P. E., Why not a department of social dentistry? *J. dent. Ed.*, **24**, 197-200 (1960).

BROOKSHIRE, R. H., Speech pathology and the experimental analysis of behavior. *J. Speech Hearing Dis.*, **32**, 215-227 (1967).

CORAH, N. L., Development of a dental anxiety scale. *J. dent. Res.*, **48**, 596 (1969a).

CORAH, N. L., Psychologic stress in a video-simulated dental restoration. *J. dent. Res.*, **48**, 444-447 (1969b).

CORAH, N. L., and PANTERA, R. E., Controlled study of psychologic stress in a dental procedure. *J. dent. Res.*, **47**, 154-157 (1968).

DAVIDSON, P. O., HARYETT, R. D., SANDILANDS, M., and HANSEN, F. C., Thumbsucking: Habit or symptom? *J. Dent. Child.*, **34**, 252-258 (1967).

DOLLARD, J., and MILLER, N. E., *Personality and Psychotherapy*. New York: McGraw-Hill (1950).

EVANS, R., Social and behavioral sciences research–1962-1966. *J. Amer. dent. Ass.*, **74**, 1500-1511 (1967).
EVANS, R. I., ROZELLE, R. M., LASATER, T. M., DEMBROSKI, T. M., and ALLEN, B. P., New measures of effects of persuasive communications: A chemical indicator of toothbrushing behavior. *Psychol. Rep.*, **23**, 731-736 (1968).
FISCHER, H., Finger, tongue and lips habits in children. *J. dent. Pract.*, **45**, 35-37 (1969).
FLANAGAN, B., GOLDIAMOND, I., and AZRIN, N., Operant stuttering: The control of stuttering behavior through response-contingent consequences. *J. exp. Anal. Behav.*, **1**, 173-177 (1958).
FLANAGAN, B., GOLDIAMOND, I., and AZRIN, N., Instatement of stuttering in normally fluent individuals through operant procedures. *Science*, **130**, 979-981 (1959).
GALE, E. N., and AYER, W. A., Treatment of dental fears by reciprocal inhibition. *Int. Ass. dent. Res. Abstr.*, 185 (1969).
GOLDIAMOND, I., The maintenance of ongoing fluent verbal behavior and stuttering. *J. Math.*, **1**, 57-95 (1962).
GOLDIAMOND, I., Stuttering and fluency as manipulable operant response classes. In L. Krasner and L. P. Ullman (Eds.), *Research in Behavior Modification: New Developments and Implications*. New York: Holt (1965).
HANSON, M., Some suggestions for more effective therapy for tongue thrust. *J. Speech Hearing Dis.*, **32**, 75-79 (1967).
HARYETT, R. D., HANSEN, F. C., and DAVIDSON, P. O., Chronic thumbsucking. A second report on treatment and its psychological effects. *Amer. J. Orthodont.*, **57**, 164-178 (1970).
HOLLAND, A., Some applications of behavioral principles to clinical speech problems. *J. Speech Hearing Dis.*, **32**, 11-18 (1967).
JANIS, I. L., and FESHBACK, S., Effects of fear arousing communications. *J. abnorm. soc. Psychol.*, **48**, 78-92 (1953).
KELLER, F. S., *Learning: Reinforcement Theory*. (2nd ed.) New York: Random House (1969).
LATTAL, K. A., Contingency management of toothbrushing behavior in a summer camp for children. *J. appl. behav. Anal.*, **2**, 195-198 (1969).
MARTIN, R. R., and SIEGEL, G. M., The effects of response-contingent shock on stuttering. *J. Speech Hearing Res.*, **9**, 340-352 (1966a).
MARTIN, R. R., and SIEGEL, G. M., The effects of simultaneously punishing stuttering and rewarding fluency. *J. Speech Hearing Res.*, **9**, 466-475 (1966b).
MORE, D. M., The dental student: Approaching graduation–1962. *J. Amer. Coll. Dent.*, **29**, 115-208 (1962).
QUIST, R. W., and MARTIN, R. R., The effect of response contingent verbal punishment on stuttering. *J. Speech Hearing Res.*, **10**, 795-800 (1967).
ROGERS, C., Some issues concerning the control of human behavior. In R. Ulrich, T. Stachnik, and J. Mabry (Eds.), *Control of Human Behavior*. Glenview, Ill.: Scott, Foresman (1966).
SHAMES, G. H., and SHERRICK, C. E., A discussion of nonfluency and stuttering as operant behavior. *J. Speech Hearing Dis.*, **28**, 3-18 (1963).
SKINNER, B. F., Some issues concerning the control of human behavior. In R. Ulrich, T. Stachnik, and J. Mabry (Eds.), *Control of Human Behavior*. Glenview, Ill.: Scott, Foresman (1966).
STEBBINS, W. C., Auditory reaction time and the derivation of equal loudness contours for the monkey. *J. exp. Anal. Behav.*, **9**, 135-142 (1966).
STEBBINS, W. C., and MILLER, J. M., Reaction time as a function of stimulus intensity for the monkey. *J. exp. Anal. Behav.*, **7**, 309-312 (1964).
WECHSTEIN, M., Practical applications of basic psychiatry to dentistry. Chapter 5 in L. N. Hollander, *Modern Dental Practice: Concepts and Procedures*. Philadelphia: Saunders (1968).
WHITE, R. W., Motivation reconsidered: The concept of competence. In D. W. Fiske and S. R. Maddi (Eds.), *Functions of Varied Experience*. Homewood, Ill.: Dorsey (1961).
WOLPE, J., *Psychotherapy by Reciprocal Inhibition*. Stanford, Calif.: Stanford Univ. Press (1958).
WOLPE, J., *The Practice of Behavioral Therapy*. New York: Pergamon (1969).
WRIGHT, L., NUNNERY, A., EICHEL, B., and SCOTT, R., Application of conditioning principles

to problems of tracheostomy addiction in children. *J. consult. couns. Psychol.*, **32**, 603-606 (1968).
YULES, R. B., and CHASE, R. A., A training method for reduction of hypernasality in speech. *Plast. reconstr. Surg.*, **43**, 180-185 (1969).

## CONFERENCE ATTENDANTS

*James Amerman, M.A.*, University of Illinois.
*Herbert J. Arkebauer, Ph.D.*, University of Nebraska.
*Ronald Baken, Ph.D.*, Teacher's College, Columbia University.
*Robert W. Blakeley, Ph.D.*, University of Oregon Medical School.
*Patricia Blanton, Ph.D.*, Baylor University College of Dentistry.
*H. Harlan Bloomer, Ph.D.*, University of Michigan.
**Arend Bouhuys, M.D., Ph.D.*, Yale University School of Medicine.
*John F. Bowman, D.D.S.*, University of Florida College of Dentistry.
*Holly Broadbent, D.D.S.*, Case Western Reserve University School of Dentistry.
*Meribeth A. Bunch, M.S.*, University of Southern California School of Dentistry.
*Kenneth R. Bzoch, Ph.D.*, University of Florida.
*Robert Canetta, Ph.D.*, University of Maryland.
*Dwight Castleberry, D.D.S.*, University of Alabama School of Dentistry.
**Richard M. Cole, Ph.D.*, Lancaster (Pennsylvania) Cleft Palate Clinic.
*William Davidson, D.D.S.*, University of Connecticut School of Dental Medicine.
*David Ross Dickson, Ph.D.*, University of Pittsburgh.
**Samuel G. Fletcher, Ph.D.*, University of Alabama in Birmingham.
**Richard M. Flower, Ph.D.*, University of California San Francisco Medical Center.
*Edward J. Forest, D.D.S., Ph.D.*, University of Pittsburgh School of Dentistry.
*James E. Fricke, Ph.D.*, American Speech and Hearing Association.
*Tanya Gallagher, M.A.*, University of Illinois.
*Jacob Geller, D.D.S.*, University of Texas, Austin.
*Stuart I. Gilmore, Ph.D.*, Louisiana State University.
*Frederick R. Greenberg, Ph.D.*, University of Southern California School of Medicine.
*John M. Grewe, D.D.S.*, University of Maryland School of Dentistry.
*Patti Grubb, Ph.D.*, Ohio State University.
*Marvin Hanson, Ph.D.*, University of Utah.
**Egil P. Harvold, L.D.S., Ph.D.*, University of California San Francisco School of Dentistry.
**Tryphena Humphrey, M.D.*, University of Alabama in Birmingham.
*Roy A. Koenigsknecht, Ph.D.*, Northwestern University.
*Charles R. Kremenak, D.D.S.*, University of Iowa College of Dentistry.

*Participant presenting a paper.

*Seymour J. Kreshover, D.D.S., M.D., Ph.D.*, Director, National Institute of Dental Research, U.S. Public Health Service.

*Donald G. Kruper, Ph.D.*, School of Dental Medicine, University of Pittsburgh.

*Norman J. Lass, Ph.D.*, West Virginia University.

*Lucie I. Lawson, Ph.D.*, University of California San Francisco School of Dentistry.

*Steve Leeper, D.D.S.*, University of Nebraska College of Dentistry.

*Mary Mann, Ph.D.*, State University of New York at Buffalo.

*Richard Mathewson, D.D.S.*, University of Oregon Dental School.

*Robert McCabe, M.S.*, University of North Carolina Dental Research Center.

*William Merow, D.D.S.*, West Virginia University School of Dentistry.

*Benjamin Moffett, Ph.D.*, University of Washington School of Dentistry.

*Karlind Moller, Ph.D.*, University of Minnesota.

*Hughlett L. Morris, Ph.D.*, University of Iowa.

*Melvin L. Moss, D.D.S., Ph.D.*, Columbia University School of Dental and Oral Surgery.

*Robert E. Moyers, D.D.S., Ph.D.*, Center for Human Growth and Development, University of Michigan.

*Robert Musselman, D.D.S.*, Louisiana State University School of Dentistry.

*James E. Nation, Ph.D.*, Cleveland Hearing and Speech Center.

*J. Douglas Noll, Ph.D.*, Purdue University.

*John M. Palmer, Ph.D.*, University of Washington.

*H. T. Perry, D.D.S.*, Northwestern University Dental School.

*G. R. Powers, Ph.D.*, University of Connecticut.

*Norman Scharf, DD.S.*, State University of New York at Buffalo.

*Sidney I. Silverman, D.D.S.*, New York University.

*Mike Spiedel, D.D.S.*, University of Minnesota School of Dentistry.

*Clark D. Starr, Ph.D.*, University of Minnesota.

*Harold S. Stocker, D.D.S.*, Wayne State University.

*Gwenyth Vaughn, Ph.D.*, Veterans Administration Hospital, Birmingham.

*Jesse J. Villarreal, Ph.D.*, University of Texas, Austin.

*Dale Wade, D.D.S.*, Ohio State University College of Dentistry.

*Nann A. Wickwire, D.D.S.*, Baylor University College of Dentistry.

*Frank Wilson, Ph.D.*, Washington University.

*Harris Winitz, Ph.D.*, University of Missouri.

*Frank Winter, D.D.S.*, Washington University School of Dentistry.

*W. R. Zemlin, Ph.D.*, Speech and Hearing Research Lab, University of Illinois.