
2.1 Introduction

Diagnosis of oral motor disorders is a professional skill. Most orthodontists and other specialist dentists are familiar with the many types of oral motor behaviors and their dysfunctions, such as normal chewing, speech, improper bites, malocclusions of the teeth, and oral–facial imbalances, but have perhaps not thought too much about the underlying processes or mechanisms that regulate these behaviors and which may eventually provide practitioners with a rationale for correcting dysfunction. Thus, special interest is focused on the information–input–processing function of the brain that is necessary for giving a conscious experience and how it may be related to oral motor behaviors that underlie the structure of the mouth.

In this context, the basic goals of oral practitioners are to discover the patient's motor problem, understand the potential causes of the problem, the ways the central nervous system regulates motor behavior, and to recommend appropriate treatment. To accomplish these goals the diagnostician must have a basic knowledge of normal and disordered oral motor behavior. Since the clinicians must mediate between the patient and the dysfunction when providing corrective experience it is important that they can conceptualize the nature of oral motor behavior.

The information processing approach to motor control and learning provides a conceptual framework in which to study and understand oral motor behavior, for instance during chewing and swallowing, within the control of a mouth system with ever-changing behavior.

2.2 The Sensorimotor Cortex

The somesthetic cortex or neocortex (excluding the limbic cortex) integrates information received from the receptors throughout the body, including the mouth. The somesthetic cortex (postcentral gyrus, Brodmann's areas 1, 2, and 3) is organized vertically. Columns of neurons (module), from one to several neurons, are oriented vertically to the cortical surface. Many vertical cylinders of neurons constitute this area. The column of neurons or module in

the somesthetic cortex of the postcentral gyrus is the basic neuron circuit that in its elemental form comprises input channels (afferent fibers), complex neuronal interactions in the module, and output channels. The output channels are largely the axons of the pyramidal cells of the motor cortex of the precentral gyrus, which directly or indirectly through the pyramidal and extrapyramidal tracks, respectively, innervate the alpha motor neurons of skeletal muscles, including the trigeminal system. The postcentral gyrus, however, also has a role in motor activity, since the electrical stimulation of the gyrus may elicit movement at times. Similarly, the precentral gyrus is also a “primary sensory cortex” because it also receives input from the ventral posterior thalamic nucleus as does the postcentral gyrus [36].

The cerebral cortex is also organized into six layers or laminae of cells, arranged parallel to the surface of the brain, each of which contains different cell types that play different roles. Beginning at the cortical surface the layers are: (1) the plexiform layer or molecular layer, (2) the external granular layer or layer of small pyramidal cells, (3) the layer of medium-sized and large pyramidal cells or external pyramidal layer, (4) the internal granular layer or layer of small stellate and pyramidal cells, (5) the inner or deep layer of large pyramidal cells, and (6) the spindle cell layer or layers of fusiform cells or multiform layer [36].

Pyramidal cells of layer III project to other cortical regions (corticocortical connections), whereas pyramidal cells of layer V project to subcortical structures conveying motor commands to muscles. Layer V pyramidal cells are the largest. Layer III pyramidal cells are generally smaller. Nonpyramidal local circuit cells, which do not project out of the immediate cortical area, are smaller and more numerous than pyramidal cells. Some of these local circuit cells can be excitatory local relays, and others mediate local inhibition. One type of local circuit cell, the horizontal cell, interconnects cells across the module and is generally inhibitory. Minicolumns and modules are organized around the large pyramidal cells. The many excitatory local circuit cells relay information up and down the vertical column. In this manner, the large dendrite tree of pyramidal cells receives many inputs via local circuit cells [15].

Pyramidal cells receive many of their inputs onto dendrite spines. Spines are appendages of dendrites that increase their surface area, and compartmentalize the molecular machinery relevant to receiving inputs. As the cortical hierarchy increases, pyramidal cells receive more inputs. For example, pyramidal cells in the prefrontal cortex have 23 times more spines (and inputs) than the pyramidal cells in the primary visual cortex [15].

2.3

Oral Sensorimotor Integration

Oral sensory integration is a child’s ability to feel, understand, and organize sensory information from his/her mouth. Sensations flow into the brain through oral experiences, and the brain locates, sorts, and orders sensory stimuli into sensations. When the stimuli flow in a well-organized or integrated manner (patterned input) the brain can use those sensory data to integrate functions underlying sensation, perception, learning, memory, motor control of muscles and mentation. The connection between the capacity of an individual to integrate sensory data and the child’s social and emotional development should not be underestimated. How a child integrates information through the sensory system provides a basis of

the child's oral and body reality. When the flow of sensory stimuli is disorganized, life can be like a rush hour traffic jam. It is sensory integration that attempts to "put it all together" and that helps us make sense of who we are and understand the world around [78, 92].

The integration of oral sensory and motor functions or behavior depends on the development of reflex circuits connecting the sense organs through the central nervous system to muscles. The sensorimotor integration function of the mouth can then be analyzed in terms of three basic functions:

1. An input transducer function. This includes all the sophisticated sensory receptors and their innervating sensory neurons, which are endowed with specificity, that is to say are able to transform definite kinds of external stimuli into complex electrical and chemical phenomena, depending on the nature and strength of the stimulus, and convey patterned action potentials (coded information) from the senses to the somesthetic cortex.
2. A control function carried through the central processing unit, the cerebral cortex, namely the postcentral and precentral gyri, which represent the primary somesthetic and motor cortex. The capacity to distinguish one sensory experience from another in the cortex is known as discrimination. Specifically, the cortical neurons must be capable of decoding the frequency of the action potential of each nerve transmitted to the brain in order to interpret the sensory messages of the receptors, and to store in memory information of the learned particular sensorimotor reflex experience.
3. An output transducer function. The human brain processes information in countless ways, most of which are poorly understood, if at all. When, however, there is stimulation of the oral motor areas in the cortex, movement of the mandible and tongue may result as a result of activation of trigeminal and hypoglossal motor nuclei, respectively [92].

By definition these three basic functions with relationships among them form a system, the oral sensorimotor integration system. There is no direct link between the oral input and output in the system. This means that the oral sensorimotor integration reflex function is an open loop. The motor output, however, has an influence on the sensory input. That is the movements of the mouth stimulate the sensory receptor input that guides oral function. Thus, the mouth's sensory experiences are generated principally by its own actions, and its actions are responsive to its sensory experiences [43, 65]. This means that the oral sensory system is functionally integrated with the oral musculature.

Similarly the receptors of the skin of the oral-facial region are functionally integrated with the underlying muscles of facial expression. The contraction of muscles stimulates the receptors through deformation of the skin [78, 93]. Granit [44] suggested that adjacent portions of the stimulated skin may be inhibitory to muscle action. This function may be locally regulated without reaching conscious states in the cortex. Thus, the skin has also established "local signs" for reflex action, and not only for conscious perception. This may imply that in certain circumstances the movements of the mouth may be initiated locally without reaching perception in the cerebral cortex. In this view, the oral mucosa, like the skin, may be able to deliver both general and localized messages. The general piece of information may be based on very large receptive fields, in which the afferent fibers overlap. The localized messages may be based on smaller receptive fields down to punctiform, which are innervated by a single afferent fiber and from which a certain type of reflex

response could be elicited. The central nervous system may correspondingly be organized to take care of large receptive fields, reaching the conscious level, and others of small receptive fields for local function at the brainstem level [44].

The strength of information processing performed by a cortical circuit depends on the number of interneuronal connections or synapses. Morphologically speaking, a typical neuron presents three functional domains. These are the cell body or “soma” containing the nucleus and all major cytoplasmic organelles, a single axon, which extends away from the soma and has cable-like properties, and a variable number of dendrites, varying in shape and complexity, which emanate and ramify from the soma. The axonal terminal region, which connects via synapses to other neurons, displays a wide range of morphological specializations depending on the target area [3].

Turning morphology into a functional event, the soma of neurons and the dendritic tree are the major domains of receptive (synaptic) inputs. Thus both the dendritic arborization and the axonal ramifications confer a high level of subcellular specificity in the localization of particular synaptic contacts on a given neuron. The extent to which a neuron may be interconnected largely depends on the three-dimensional spreading of the dendritic tree [3].

Dendrites are the principal element responsible both for synaptic integration and for the changes in synaptic strengths that take place as a function of neuronal activity [46, 47]. While dendrites are the principal sites for excitatory synaptic input, little is known about their function. The activity patterns inherent to the dendritic tree-like structures, such as in integration of synaptic inputs, are likely based on the wide diversity of shapes and sizes of the dendritic arborizations [48]. The size and complexity of dendritic trees increase during development [49], which has been, in turn, associated with the ability of the neural system to organize and process information, such as when animals are reared in a complex sensory environment [50]. Thus, dendritic size and branching patterns are important features of normal brain development and function.

Most synapses, either excitatory or inhibitory, terminate on dendrites, so it has long been assumed that dendrites somehow integrate the numerous inputs to produce single electrical outputs [3]. It is increasingly clear that the morphological functional properties of dendrites are central to their integrative function [51, 52]. Branching in this context is essential to the increasingly complex ability of the neuron to respond to developmental complexity [48]. The dendritic cytoskeleton plays a central role in the process of ramification, filopodia formation, and more specialized neuronal activity such as long-term potentiation circuit dynamics, including memory formation, and the response to pathological conditions [3].

Synaptic contacts take place in dendritic spines, which are small sac-like organelles projecting from the dendritic trunk. Dendritic spines are more abundant in highly arborized cells such as pyramidal neurons and scarcer in smaller dendritic tree interneurons with few interconnections. The number of excitatory inputs can be linearly correlated with the number of spines present in the dendrite [3]. How information is processed in a dendritic spine, is still a matter of current study [6]. Dendritic spines contain ribosomes and cytoskeletal structures, including actin, and α - and β -tubulin protein. As in other parts of the neuronal structure, cytoskeletal components are highly relevant in the structure and function of the neuron. The axonal hillock contains large “parallel” bundles of microtubules. Axons are also located with other cytoskeletal structures, such as neurofilaments, which are more abundant in axons than in dendrites. The axon hillock is the region where the action

potential is generated. The dynamics in cytoskeletal structures are central to our contention that information can be processed and “delivered” to the synaptic function by changes in the cytoskeletal structures [3].

The recently advanced new learning and memory model of Penrose and Hameroff [113] suggests that information processing and storage of memory occurs in the microtubules and microtubule-associated proteins of the subsynaptic zone of dendrites, which gives rise to the spine. Microtubules in the subsynaptic zone are capable of storing important information concerning where spines were originally located, and of using that information to determine where synapses will reappear following learning or enriched experiences [39–41].

The primary receptive area of the cortex integrates the more complex aspects of touch, deep sensibility, pain, and temperature. These perceptions include the appreciation of the location and position of a body part (proprioception), the localization of the source of pain, temperature, and tactile stimuli, as well as the comparison of these sensed modalities with those formerly experienced through memory [36]. Ablation of the postcentral gyrus is followed by loss of the finer and more subtle aspects of sensory awareness. For example, when an object is handled with the eyes closed, the subject can feel it but cannot appreciate its texture, estimate its weight or temperature. Difficulty is also experienced in appreciating the position of the body or body parts in space [36].

The sense of taste is claimed to be located at the base of the precentral and postcentral gyri, slightly rostral to the primary somesthetic cortex or in the cortex immediately adjoining the insula area [42].

In sum, as we view the development and function of the mature mouth we are more aware that the mouth is essentially an information-processing system, in which the higher faculties of the brain, namely learning, memory, motor control of muscles and reasoning, are chiefly concerned with the choices involved in getting food, and the development of the computational activities of mastication, speech, taste, etc, through learning and memory of the oral sensorimotor integration function of the cerebral cortex. Any animal would starve to death if the effect of food was limited to its action on the surface of the mouth. Thus, the enormous significance of feeding acquired by experience and learning becomes evident.

2.4

Sensory Integration Dysfunction

All the basic five sensory systems need to be working simultaneously and cooperatively for acquisition and performance of higher skills. In fact, if any one system does not work properly either by itself or in conjunction with the others, a sensory dysfunction of some kind may result. The degree of sensory dysfunction, whether it manifests as merely a “sensory issue” or mild, moderate, or severe sensory dysfunction, is highly dependent on which senses and/or sensory systems are impaired and to what extent. Again, the sensory systems actually operate in conjunction and cooperation with one another, so the resultant behavior or sensation is probably the outcome of accessing more than one system [79].

Often a child with sensory integration dysfunction will be categorized as immature and possible spoilt. The new social, cognitive, and motor demands of the school setting often

create even more confusion, anxiety, and chaos for the child who has sensory integration dysfunction [79]. Now, let us explore some of the systems that may indicate sensory integration dysfunction.

2.4.1

Dysfunction in the Proprioceptive System

This refers to an inefficiency in the proprioceptive system that may cause a child to have difficulties both at school and at home. Since the proprioceptive system gives feedback from muscles and joints, it supports holding a pen or a pencil correctly, staying properly seated in a chair, and learning to use a knife, fork and spoon correctly. This system also supports learning to walk, opening and closing a jar or a door, using playground equipment, and chewing [79].

2.4.2

Dysfunction in the Tactile System

A dysfunction in the tactile system may manifest in a variety of ways. A child may be actively defensive and not want to be touched, bumped, or hugged, or to touch certain textures or wear certain types of clothes. An atypical tactile system may also cause a child to crave certain tactile sensations and experiences, such as never being able to keep their hands to themselves, constantly touching and feeling the people and/or things around them. In other words, the tactile system greatly influences relationships, fashion sense (clothing choice), and school performance [79].

2.4.3

Dysfunction in the Vestibular System

A problem or inefficiency in the vestibular system may cause difficulty with balance, coordination and motor planning. This may manifest in the child as clumsiness and uncoordinated fine motor activities, such as eating, drinking, using writing utensils etc. The vestibular system will play a key role in the ability of the child to participate successfully in sport, in the gym class or on the playground, and in various activities at school and at home [79].

2.4.4

Possible Sensory Signs and Symptoms

Let us now explore a wide range of behaviors and symptoms that may indicate some level of sensory dysfunction. This list is not meant to be used for diagnostic purposes, but rather to create awareness of some of the sensory difficulties a child may experience over the course of development and for which specialist help might be needed.

- Young children may be overly sensitive to touch. Touch is interpreted as a nociceptive stimulus and therefore working in their mouth is very difficult. For the same reason these children dislike bathing, brushing their teeth and other self-care activities. These children may be oversensitive to pain.
- Young children may be underactive to touch. These children exhibit oral hyperactivity by constantly eating or chewing. These children may be “chewers”—they chew shirts, blankets, toys, toothpaste etc.
- Children who do not tolerate the taste of certain foods, and therefore vomit or gag easily, and always have the same lunch; i.e., they are very picky eaters. This suggests gustatory sensory dysfunction.
- Children who do not know when their mouth is full and so stuff their meal into the mouth. This behavior suggests proprioceptive, tactile sensory dysfunction.

Traditionally, sensory integration has been the exclusive realm of the occupational therapist. Now this is starting to change and, although it is still the occupational therapist who will do much of the formal, standardized testing for sensory dysfunction, other specialists are beginning to recognize sensory integration dysfunction, including orthodontists, make referrals for further evaluations, and incorporate a “sensory approach.”

2.5

Oral Experience Changes the Brain

After centuries of debate about body and soul and the nature of man, we now understand that the brain’s architecture gives rise to mind, emotions, and personality. The tragedy of Alzheimer’s disease and dementia, which are accompanied by personality fragmentation, bear undeniable witness that the brain is the basis of mind. Neuroscientists widely accept that cognition and consciousness are correlated with the physiological behavior of the material brain, and that the matter that comprises brain gives rise to its functions, in particular higher cognition and consciousness, through integration with the environment [33].

Sensory systems, such as the mouth, transmit sensory information to the brain through the sensory pathways. An oral sensory pathway begins in a sensory organ, usually makes synaptic contact in the thalamus, and then relays to layer IV of the primary somesthetic cortex through a series of corticocortical circuits. Change in the brain begins when the new experience activates the oral sensory pathway conveying information to the brain. These records are established through growth changes in the pathways and in the cerebral cortex [15].

The initial response of the cortex to a peripheral stimulus is the evoked potential. Immediately afterwards there is a change in the frequency of firing in the module, resulting in the signal becoming more sharply defined by elimination through inhibition of all the weaker excitatory stimuli. As a consequence the stimulus can be more precisely located and evaluated by the cortex. In fact inhibition that sharpens the neuronal signal occurs at each relay station in the pathway. Each relay station also gives the opportunity to modify the coding of the messages transmitted from the receptor organs [37].

Neurons in the pathway generally communicate with each other using chemical messages called neurotransmitters. Glutamate is the main excitatory neurotransmitter, which transmits sensory information along the sensory pathways throughout the central nervous system. Glutamate is produced in the signal-transmitting presynaptic neuron by proteins related to synthesizing, packing and releasing the neurotransmitter from the presynaptic nerve terminal. Glutamate traverses the synaptic gap and alters the membrane potential of the signal-receiving postsynaptic neuron. If the signal is strong enough, or if it is accompanied by sufficient additional input, the receiving neuron will generate an action potential that will reach another neuron in line. Thus, the sequence of events repeats itself [15].

Glutamate mediates excitatory sensory input to the cerebral cortex where many of these presynaptic terminals contact dendrite spines by binding to NMDA, AMPA, or kainate receptors located on the surface membrane of the spines. For example, glutamate binds to the NMDA receptor within few milliseconds, altering the ion conductance across the membrane. Electrically charged calcium ions (Ca^{2+}) enter the neuron and stimulate calcium-activated proteins inside the cell. The activated proteins initiate polymerization and depolymerization cycles leading to breakdown and then rebuilding of the microtubule protein, tubulin, as well as of microtubule-associated protein 2. These changes occur in the subsynaptic zone of dendrites lying beneath the spine which gives rise to the spine [15]. Thus, NMDA receptors mediate breakdown and then rebuilding of glutamate synaptic sites on the spines in response to information input, for instance, of oral experience. In this view, oral sensory information changes the conformational state of neuron cytoskeletal proteins, i.e., changes the brain through learning and memory of new experiences. Evidence suggests that memory is hard-wired in dendritic cytoskeletal structures [54–56]. Accordingly, the NMDA receptor is key to brain neuroplasticity [33].

Similarly, glutamate binds to AMPA receptors and opens sodium (Na^+) channels across the membrane. Sodium enters the neuron, increasing the sodium concentration inside the cell. This is likely to initiate polymerization of tubulin, the building protein block of microtubules. If the sodium concentration becomes too high, however, tubulin depolymerization will occur [15].

Thus, brief oral sensory experiences, for instance the taste of a substance or the exploration of an object in the mouth, generate fleeting electrical impulses in the neurons that innervate the stimulated receptors. The impulses make the transmitting neurons release molecular signal keys that selectively interact with specific receptor locks on the surface of the target neurons, eliciting biochemical changes and biological responses in targets underlying sensation–perception, learning–memory, and motor activity depending on the stimulus [15, 38, 58].

The signal from taste may also reach through local cortical circuits the hippocampus memory center and excite large specialized groups of neurons there that fire in a characteristic synchronized pattern that represents memory of learned experience [17, 57]. The memory mechanisms are then built into the rules governing the architecture of the cerebral cortex, in other words are built as changes in brain structure [38, 58]. Subsequently, the hippocampus sends a tract to hypothalamus that regulates the consumatory action pattern of the individual.

Similarly, when we chew food or gum the oral information is integrated in the sensorimotor cortex. The motor cortex then sends action potentials down to the alpha motor

neurons of the trigeminal nuclei, through the pyramidal and extrapyramidal pathways, which in turn make the masticatory muscles contract. The transmitting neuron releases neurotransmitter signal, acetylcholine, at the relay station, which stimulates electrically the receiving neuron. Acetylcholine binds to specific receptors of the receiving neuron and strengthens the synapse. In this way, the strengthening of synapses through the signal molecules allows the neurons to hold electrical conversations for enough time to evoke, for instance, a muscle contraction or a taste experience [2].

Thus, molecular signals (glutamate, acetylcholine, dopamine, etc – hundreds of them) govern the function of the brain and mind, and increase the ability of neurons to communicate with each other through the strengthening of synapses. Not surprisingly then brain signals and receptors play a critical role in a variety of dysfunctions and diseases and treatment procedures. Signal derangement results in a wide variety of neuropsychiatric diseases including depression, dementia, and Parkinson's and Alzheimer's diseases [2].

Fascinating experiments have provided insights into how experience induces brain changes. For example, rats were taught to navigate through a complicated maze, while the electrical activity of their hippocampal memory neurons was monitored. As the rats learned to navigate the maze successfully, the neurons fired with a characteristic (unique) synchronized pattern that represented memory of the learned task [59].

In another experiment, maze-learning rats were stressed with a mild electric shock to the tail. A control group received no electric shock. The group that received the shock stressor learned faster and retained the knowledge longer than the control group. Moreover, in the stressed rats neuronal connections were strengthened more efficiently than in the control group [60, 61]. Accordingly, it has been suggested that some degree of stress may promote some forms of learning, through alertness and attention of brain states that foster synapse strengthening and learning. In this view, neuroscience may provide new strategies for learning and teaching in school. Memory abilities are subject to change and hence and a little stress may sharpen memory through strengthened synapses [2].

Hebb [16] suggested that synapses in the brain are strengthened in accordance with how often they are used (stimulated), enhancing then the input and the processing of information, which in turn contributes to the richness of sensory experiences. Hebb speculated that strengthened synapses bind the neurons together into "ensembles." If individual neurons represent different features of a scene, for example, the ensemble recreates an image of the scene in its entirety, like the pieces of a puzzle. In this view, strengthened synapses may associate for instance, visual, auditory, emotional and conceptual features of a memory that are encoded in the component neurons.

In addition, experimental studies by Bliss and Lomo [17] suggested that experiences strengthen synapses of only the specific pathways processing an experience. Experiences do not affect synapses that are not activated by the action potential. Specificity is then built into the process of transmitting information; the information is transmitted through activated (specific) pathways. Accordingly, learning and memory are also specific processes, since they use specific pathways.

Thus, the communicative connection between neurons, the synapse, and its strengthening by electrical impulses and chemical signal molecules through use, is critical in regulating oral information input and motor output. If the synapses in the activated pathways are strong more information is relayed and processed in the cortex, resulting in learning,

memory, perception, and motor control of muscles. On the other hand, if the synapses are weak, less information is communicated [2].

Neurophysiological studies indicate that the sensory nerves are not high fidelity recorders of the peripheral stimulus, because they accentuate certain stimulus features and neglect others. In addition, the central nervous system, which does the processing of the information, is never completely trustworthy, allowing distortions of quality and measures. Because of this there is general agreement that sensation is an abstraction, not a replication, of the real world. The more specific is the sensory stimulus perceived by the brain, the more specific is the motor pattern [62].

While the central nervous system needs continuous stimulation of all sensory modalities during childhood to maintain the strength of synapses in order to increase the input and processing of information, which is the key to sensation, perception learning, memory and motor control of the mouth, on the contrary idleness of the mouth decreases the strength of synapses and inactivates the sensory pathways to the brain. Hence, very little information is entering the brain and the cerebral cortex is suffering from sensory deprivation [63]. Under these conditions the cortex will reduce the input to the motor cortex controlling the motion of the mouth and the voluntary masticatory movements, leading to oral dysfunction and to delayed maturation of oral functions [64].

The neural events pertaining to the normal or abnormal function of the mouth are registered in the cerebral cortex at all stages of the development of the mouth. This ability of the brain for self-regulation of its structure and function, according to oral sensory information-processing and learning–memory capacity, is called plasticity of the brain, and is reflected point-to-point in the anatomical representation of the mouth in the sensory and motor maps of the cerebral cortex. The cerebral maps are continually modulated by the various sensorimotor functions of the mouth reflecting experience-driven changes in the brain [43, 65]. Thus, the normal oral sensorimotor functions, such as speech, chewing, swallowing, taste, etc., will be seriously disturbed without normal sensation and perception of oral structures, without normal jaw and tongue movements, and without adequate secretory activity of the salivary glands [66].

In this view, mastication of food might be an important motor activity, contributing to significant sensory input and to structural and functional development of the brain. Similarly, any dental or orthodontic procedure that restricts the movements of the mouth for a long time may affect the normal sensory input and the integration of sensorimotor functions in the cortex.

Kawamura [66] suggested that both life experience and emotion contribute to individual variation in oral sensorimotor functions and in particular to the masticatory function. Bosma [67] suggested that the infant organizes and governs its sensorimotor world of experience in relation to its own current needs and purposes. In doing so the infant is generating its own neurological future. This reaction of the infant to external influences is not a passive one. Instead the child is striving toward the outer world, and this is an instinctive involuntary phenomenon. When this striving is satisfied, i.e., when it causes, for instance, a movement, the movement is a reflex in the full sense of the term. There is no doubt that complete dependence on this instinctive striving is responsible for the extreme mobility of infants and children, which constantly pass from exercise of one nerve to that of another. It is then this mobility that ensures the normal development of the sense organs, including the oral senses, resulting in

organized motor behavior. Thus, the first condition for maintaining the structural and functional integrity of nerves and muscles is adequate exercise of these organs [68].

2.6

Role of Reticular Formation in Oral Sensorimotor Functions

The reticular formation is believed to regulate or control the sensory input to the brain. The feedback information supplied between the sensory input and motor output must be of the right amount if the operation of the sensorimotor function of the mouth is to be regulated for optimum results. Too much feedback or central modification of the sensory input may mean sensory overload for the brain, which can be catastrophic leading to wild oscillation in oral motor behavior. Conversely, reduced sensory input may mean sensory deprivation of the brain, again deranging oral motor behavior. Thus sensory overload, sensory deprivation and the central disposition of the individual may reduce the arousal reaction of the cerebral cortex for attention, awareness and learning, leading to unconscious reduction of interaction of the individual's mouth with the external environment through changes in the reticular formation and the ascending reticular activating system (ARAS) [63, 64].

There are four major neurophysiological mechanisms dealing with the organization and functioning of the central nervous system, all related to reticular formation; these are discussed in the following sections.

2.6.1

Descending Reticular Control

The descending reticular control mechanism has been implicated in the facilitation and inhibitory roles of the reticular formation. Specifically, the reticular formation at the midbrain level has been found to maintain a facilitatory or augmenting influence on motor reflexes. However, the more caudal portion of the reticular formation in the pons and medulla has been found to have an inhibitory role, keeping motor activity under control [64].

2.6.2

Ascending Reticular Activating System

The reticular formation and the ARAS are both necessary for arousal of the brain for attention, perception and conscious learning. Without the collaboration of the ARAS the oral sensory messages cannot be projected from the reticular formation to the cortex and hence, they cannot be elaborated and decoded, resulting in no discrimination of the senses. Thus, the state of excitation and adjustment or adaptation level of the reticular formation as it monitors both incoming sensory and outgoing motor messages, as well as the adaptation of the ARAS, become part of the process of arousal of the brain, for learning ability and habit formation [63, 64].

These changes of the reticular formation and of the ARAS depend upon the ebb and flow of activity in the afferent and efferent systems. The strategic location of the ARAS at the cross roads of the sensory input and motor output systems of the cranial nerves, including the trigeminal input and output pathways, permits the ARAS to sample and monitor all such activities. In doing so it becomes adjusted to certain levels of activity and its own response is projected to the cerebral cortex to exploit aspects of its information-processing capabilities, such as perception, learning, memory, motor planning, etc. The regulation of mature oral sensorimotor functions derive from the cerebral cortex through the ARAS. The sensory-input/processing function of the cortex determines the level of arousal of the brain for attention and awareness. For example, if the ARAS is activated experimentally by electrical stimulation, the cortex shifts from sleep to waking in the encephalogram. Thus, the ARAS is involved in the sleep and waking rhythm of the organism, which is controlled by the cerebral cortex through the neural activity of the sensory-input/information-processing function [63, 64].

When the ebb and flow of activity in the afferent and efferent systems is restricted, compensatory adjustments are made within limits in the reticular formation and the ARAS. When this fails, for instance in sensory overload or sensory deprivation, the cortical factors are not under the control of the ARAS. When this happens persistently, perception is disrupted, attention gives way to distractability and interest to boredom. Behavior, including oral motor behavior, becomes disorganized. This seems to be reflected in either a pervasive inhibition of sensory stimuli or a marked facilitation of oral reflexes with excessive motor activity of a highly stereotyped type and nonadaptive nature, which in turn may lead to oral habit formation [64].

In this view, early sensory deprivation of the brain or early growth and development in an impoverished environment, one with diminished heterogeneity and a reduced set of opportunities for manipulation and discrimination, not only rob the organism—mouth of the opportunity for constructing sensory models of the environment in order to deal efficiently with them, but it also prevents the utilization of sensory modalities to extract and evaluate useful information from the environment [69, 70]. This may result in impairment of sensorimotor functions through the disruption of cortical space and time quality organization (perceptual changes), as well as deterioration in learning ability in association with a decline in reasoning (cognitive changes) [64]. This disorientation of oral motor behavior is often associated with a reduction in taste, poor adaptive capacity of the occlusion of the teeth and a decline in function of the mouth. In general irritability and restlessness of the mouth are coupled with anxiety [63, 69].

Sensory deprivation may occur experimentally with unvarying sensory stimulation of the cortex by reducing both the amplitude and the rate of change of the stimuli to a monotonous level, at which the sensory receptors adapt and stop firing resulting in sensory deprivation of the brain, which is characterized by disruption of the capacity to learn or even to think [71, 72]. This breakdown of the sensory–perceptual–motor and learning–memory references, by which the organism—mouth guides its correction strategies in perceiving, becoming cognizant, reacting and manipulating the environment [69], and guides the ontogenetic development of the body [73], may make it increasingly difficult in early postnatal development for the oral sensorimotor functions to structure a normal anatomical mouth [43, 65].

Specifically, since normal perception depends partly on normal motor activity [16], and normal motor activity generates normal patterned sensory input [58], resulting in normal

development of the brain [33], then it is likely that the early deviation of the mouth from the normal sensory-input perceptual function of the cortex will affect the brain structure and its cognitive state. These changes in turn will be reflected in the neurological development of the mouth. In this view, Bosma [43, 65] suggested that the developing central nervous system guides the development of the anatomical mouth and the position of the teeth. Conversely, the mouth is developmentally inscribed at all stages upon the maturing brain and the neural networks of the brain are modulated by the oral sensory experiences.

2.6.3

Corticoreticular Control

It is established functionally that there are interconnections between the cortex and the reticular formation. This means that arousal and alerting of the brain are not solely dependent upon peripheral sensory influx, but may equally well be produced by cortical activity [64]. In this view, imagination of past or present stimuli has the potential to excite the reticular formation and the ARAS, which in turn produces a suitable state of activity in the cortex. The cortical factors now not under the control of ARAS may play a more prominent role. The effect tends to be facilitation of stimuli, which will favor cortical restless hyperactivity [15, 64]. Recently, it has been shown that the motor cortex is activated as if sensory input had occurred in anticipation of a motor activity [74]. This shows that we experience sensory feedback before the sensory input occurs [15].

2.6.4

Centrifugal Afferent Control and Central Inhibitory Mechanisms

According to most neurologists the centrifugal influences serve the purpose of making the receptors or pathways more capable of sending information or of preventing the normal deluge of sensory impulses from reaching the higher central nervous system levels. In this view, the centrifugal afferent control system can reduce the sensory input if the motor response output becomes excessive. The descending fibers from the cortex terminate in excitatory synapses with interneurons, which in turn act through presynaptic or postsynaptic inhibition on the neurons of the ascending affected pathways. The effect of centrifugal afferent control is generally inhibitory. It is exerted at the synaptic relays, as well as at the receptors themselves, tending to suppress the amount of information transmitted to the cortex [64]. Under centrifugal afferent control there is a marked reduction in the total mass of the sensory input, while the “useful” or “significant” information is preserved or even enhanced. Thus, selectivity or filtering is an important consequence of these inhibitory mechanisms. It is of interest that the centrifugal control mechanisms are learned with early sensory experience [64].

The importance of inhibitory reflex mechanisms in the normal motor maturation, is seen in the early postnatal period. For example, if one gives a two-year-old child a pencil, it holds it tightly. It is part of the educational program of the school to weaken and inhibit the grasp reflex, so that the child will be able to hold the pencil and write with it. Thus the major portion of the education that human beings receive during the preschool years, in

school and in later life, consists in the development of inhibitory nerve fibers and inhibition mechanisms in order to overcome reflexes. A new-born child cries when it is hungry. It must get used to eating at meal times. Thus, one of the first things a child learns is to suppress the reflex to cry when hunger pangs appear. Then a child is taught to inhibit the bladder and intestinal reflexes, and to let them function only at certain times. This is the beginning of training for life, and this is how it continues for ten, twenty, thirty years under the constant motto “develop inhibitory fibers,” so that training may be said to be almost equivalent to the possession of inhibitory mechanisms, and the ability to control oral and body reflexes, drives and animal instincts in social behavior by means of them [37, 75].

Childhood is generally characterized by extremely extensive reflex movements arising in response to relatively weak (from the adult point of view) external sensory stimulation. Gradually, however, during development one or two groups of muscles separate from the mass of other muscles and the movements become more limited. Having become more limited, the movements acquire a definite character. It is in this limiting process that the inhibitory mechanisms take part [37]. For example, the normal coordination of the contraction of the muscle groups involved in mastication and deglutition require central inhibition of the neurons of the trigeminal nuclei when the deglutition function is initiated, and conversely central inhibition of the glossopharyngeal motor neurons, which innervate the pharyngeal muscles of deglutition, when the masticatory muscle group is activated [76].

Thus, by exerting presynaptic and postsynaptic inhibition, the cerebral cortex is able to block the synapses in the sensory pathways and hence, give the opportunity for an inhibitory action to occur, that sharpens the neural signals by eliminating the weaker and irrelevant excitatory stimuli. As a consequence, the stimuli can be more precisely located and evaluated by the brain [37].

In fact, a tactile stimulus, for instance, not only produces a sensation but at the same time produces an inhibition of the surrounding cutaneous or mucosal area [44]. Thus, the concept maintained in telephone engineering—that we have a simple input and output—does not hold in the sensory systems, since every stimulus or input produces definitely two phenomena. There is a usual output as found in telephone systems, but there is also an inhibition in the surrounding area of the stimulus produced by the output [77].

In this view, the brain makes a simplification by blocking certain excitatory inputs in order to protect the masticatory apparatus from the many reflexes that would normally arise from all structures had been stimulated separately during mastication. This complex interaction of excitatory and inhibitory influences upon the oral tissues can limit the forces developed during chewing. For example, when crushing of a food bolus is desired, a protective mechanism involving a cortical loop may be called into play and periodontally induce inhibition of jaw-closing motoneurons, which outweighs the excitatory influence, resulting in cessation of jaw-closing muscle activity during a masticatory cycle [78].

It is also interesting to note that when we are very intensely occupied, for instance in carrying out some action or in experiencing or in thinking out some action, the cerebral cortex can block the synapses in order to protect itself from being bothered by stimuli that can be neglected. Accordingly, in the heat of combat, severe injuries can be ignored by the individual by suppression of the pain pathway to the brain. Thus, we can account of afferent anesthesia, of hypnosis or yoga or acupuncture by the cerebral cortex inhibiting the sensory pathways [37].

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