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Review Article

The Babkin Reflex in Infants: Clinical Significance and Neural Mechanism

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ABSTRACT

BACKGROUND: There have been very few studies concerning the Babkin reflex—opening of the mouth and flexion of the arms in response to stimulation of the palms. We attempted to clarify the clinical significance and neural mechanism of the reflex through systematic review. **METHODS:** Searches were conducted on Medline, Embase, and Google Scholar from their inception through August 2012. **RESULTS:** In normal term infants, the Babkin reflex can be elicited from the time of birth, becomes increasingly suppressed with age, and disappears in the great majority by the end of the fifth month of age. A marked response in the fourth or fifth month of age and persistence of the reflex beyond the fifth month of age are generally regarded as abnormal. On the other hand, because there are some normal infants showing no response during the neonatal period or early infancy, the absence of the response during these periods is not necessarily an abnormal finding. **CONCLUSIONS:** Infants with these abnormal findings should be carefully observed for the appearance of neurological abnormalities including cerebral palsy and mental retardation. It is most likely that the Babkin reflex is mediated by the reticular formation of the brainstem, which receives inputs from the nonprimary motor cortices. On the basis of the hand–mouth reflex, more adaptive movement develops as control of the nonprimary motor cortices over the reflex mechanism in the reticular formation increases. Soon it evolves into the voluntary eye-hand-mouth coordination necessary for food intake as the control of the prefrontal cortex over the nonprimary motor cortices becomes predominant.

Introduction

In 1953, Babkin[1,2] observed a peculiar reflex connecting the hands and mouth areas in newborn infants, first called the hand–mouth reflex in the Russian literature. Later in 1958, Lippmann[3] named the reflex Babkin'schen reflex in his German article, while Lesný[4] designated it as the palmomandibular sign in 1960. Because a reflex substantially identical to the Babkin reflex has been observed in human fetuses as early as 14 weeks' gestation, this reflex appears to be phylogenetically very old and ontogenetically very primitive.[5] However, studies concerning the Babkin reflex have been surprisingly few, especially in the English literature. Therefore, pediatricians in general are not as familiar with the reflex as other well-known primitive reflexes such as the Moro and grasp reflexes. This review deals mainly with the clinical significance and neural mechanism of the Babkin reflex but also discusses the phylogenetic meaning of the reflex and the difference between it and other similar reflexes (i.e., the palmomental reflex and jaw-opening reflex).

Elicitation of the Babkin reflex

The Babkin reflex is elicited by an examiner by simultaneously pressing his or her thumbs against both palms of an infant lying on a flat surface in the supine position.[2] Tactile stimulation without pressure and nociceptive stimulation are both ineffective. The predominant response in the reflex is opening of the mouth, which is often associated with flexion of the forearms and head and closing of the eyes. If the subject's head faces laterally in the starting position, it may turn to midline when the stimulus is provided.[3] The response is to some degree dependent on the strength of

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the applied pressure, with sudden, strong pressure being recommended for a good response of the reflex.[3] Unilateral pressure may evoke a weak reaction,[6,7] with turning of the face toward the hand stimulated.[6] In addition to the palms, areas where the response may be elicited include the forearms[4,8] and infraclavicular space near the sternal bone.[9] There is some indication of dependence of the response on the state. It tends to be positive in the waking state but negative in the drowsy or sleep state, although it exhibits wide variation, especially in small preterm infants.[10] The Babkin reflex can be by far more easily elicited before feeding than after feeding and cannot be elicited during feeding.[3,11]

Clinical significance

Responses in normal infants

The study of the Babkin reflex in normal term infants has been undertaken by several researchers. Their results are fairly consistent regarding the times of the appearance and disappearance of the reflex. Babkin examined 1420 newborns in a maternity ward and followed the change in response in 210 of them until the age of 1-2 years after discharge.[2] He found that the reflex could be elicited from the time of birth, but was suppressed by the third or more frequently by the fourth month of life. Lippmann[3] could elicit the response in 278 of 311 (89.4%) newborns examined and followed the change in response after discharge in 50 of them. He observed that the response began to decrease during the first month of life, disappeared in the majority by the end of the third month, and could not be elicited after the end of the fourth month. Some authors also reported that the Babkin reflex disappears by the fourth month of life.[12-14]

On the other hand, Futagi et al.[11] reported that in 85 term infants the response was elicited in 84.6% of them during the first month of life and could still be elicited in 9.9% of them during the fifth month of life but could not be elicited thereafter. Pedroso and Rotta[8] described that the reflex could be elicited in all of 33 term infants during the first 2 months of life but had disappeared by the end of the fifth month in the great majority, with a few exceptions still exhibiting the response until the end of the sixth month.

There have been only a few studies on the Babkin reflex in preterm infants. Lippmann[3] examined 67 preterm infants weighing 1030-2500 g at birth and described that the reflex was clearly obtainable, but he gave no further details. Parmelee[10] studied 57 preterm infants who were examined at 2- to 4-week intervals while they were in the hospital, with a total of 179 tests being undertaken. He found that the tests were positive in 153 of 179 (85.4%). The most premature among the infants, with a birth weight of 580 g and gestation of 26 weeks, exhibited a strong response of mouth opening with rotation and flexion of the head. Futagi et al.[11] compared the change in response with age in 173 preterm infants of gestational ages ranging from 25 to 36 weeks with that in 85 term infants, according to the corrected age as to expected birth date. All of the subjects appeared to be normal at the time of the latest neurological examination with an average corrected age of 2.2 years. There was no age-related difference in the evolution of the response between the two groups throughout the first year of life.

Abnormal responses

Babkin[15] described that, in newborns with traumatic injury of the shoulder plexus, the reflex may be absent on the affected side. It may also be absent in infants with circulatory insufficiency in the brainstem, whereas in individuals with cerebral hemisphere lesions, the reflex is sometimes elicited after 4 months of age. Lesný[4] noted an infant aged 10 months with severe perinatal brain damage who showed the reflex by opening the mouth on pressure to the palms and the forearm. Kabátník and Trávník[12] reported that in infants with brain damage the reflex was still found at the age of 6 months, and in rare instances up to the age of 1 year. Holt[14] also states that elicitation of the reflex after 4 months of age indicates a cerebral lesion.

From the medical records, Futagi et al.[11] selected 22 children with cerebral palsy and 29 with mental retardation without motor disturbance who had been tested for the Babkin reflex during the first year of age and compared their responses with those in normal infants. The response was not different among the groups during the first 2 months of age. However, in the normal group, the response was rapidly suppressed thereafter and had disappeared by the end of the fifth month of age, whereas it was retained beyond the fifth month of age in the cerebral palsy and mental retardation groups. The incidence of a positive response from the sixth through the twelfth months of age was nearly constant, at about 40% for infants with cerebral palsy and about 20% for infants with mental retardation.

Later, Futagi et al.[16] described another six infants who had showed a marked response during the fourth to fifth months of age or a positive response after the fifth month of age, but without any other overt neurological sign at the time of examination. During the follow-up period, all of the subjects were eventually found to have a neurodevelopmental disorder (cerebral palsy in five and mental retardation in one), which suggests the predictive value of the reflex. In these two studies, the ages of the preterm infants were corrected as to expected birth date to compare the results with those in term infants.

Pedroso and Rotta[8] examined 50 children with neurological disorders, including cerebral palsy and mental retardation, aged between 9 months and 13 years, and compared the results with those obtained in 33 normal infants aged 6 months and below. They performed this study with the intention of proving a functional continuum of cutaneous reflexogenic zones for the mouth-opening reflex by stimulating various limb segments. Rapid compression was simultaneously applied on both sides of the upper limbs (palms, distal forearms, and central aspect of upper arms) and lower limbs (soles, lower legs, and central aspect of the thighs). In normal infants, within the first 2 months of life, they could elicit the reflex in all of the subjects with a stimulus to the palms, and in 90-100% of them for the forearms and about 20-35% of them for the upper arms, depending on age in months. However, mouth opening following compression of the lower limbs was never observed in normal infants, although it could sometimes be elicited in children with a neurological disorder.

From these findings, they concluded that the mouth opening in response to stimulation of the lower limbs served as an early indicator of central nervous disturbance in children. They assumed that the enhancement of the response in children with a neurological disorder was caused by liberation from the inhibitory control by the higher brain mechanisms, which uncovered the functional continuum of reflexogenic zones for the mouth-opening reflex latently existing in all four limbs.[7,8]

No adult with a Babkin reflex has been reported, although a few patients with severe brain pathology showed the mouth-opening reflex triggered by painful stimulation of the face and limbs[17] or by visual stimulation with an object approaching the mouth.[18,19] It is speculated from imaging studies and clinical signs that the former reflex was mediated by the brainstem, which was released from the higher brain mechanisms because of a cross-sectional midbrain lesion, while the latter was mediated by the premotor cortex, which was released from the diseased prefrontal cortex.

In summary, a marked response of the Babkin reflex in the fourth to fifth months of age and the persistence of the response beyond the fifth month of age can generally be regarded as abnormal. On the other hand, there are some normal infants showing no response during the neonatal period or early infancy, as demonstrated in the studies of Lippmann[3] and Futagi et al.,[11] which indicates that the absence of the response during these periods is not necessarily abnormal. However, in none of the studies so far reported were the subjects properly categorized into subgroups according to their state and feeding conditions, as expressed as the time elapsed from the last feeding, to evaluate or compare the responses. When no response is obtained in neonates and early infants, it may be necessary to repeat tests to confirm the response under the optimal conditions for eliciting the reflex (i.e., in the awaking state with no gross movement and just before the next feeding).

Phylogenetic meaning

The Babkin reflex can be more easily elicited before feeding than after feeding, which implies that this reflex is closely related to food intake. Lower animals including amphibians and reptiles catch their food exclusively with the mouth, whereas in higher forms, the food is at first grasped with the forelimbs and only then seized with the mouth, as seen in mice, squirrels, and monkeys.[6] Thus, the Babkin reflex can be phylogenetically regarded as the rudiment of the hand–mouth association for prey in animals. In response to unilateral pressure to the palm, turning of the face toward the hand stimulated with mouth opening is also suggestive of the meaning of the reflex, because it signals the motor behavior when an animal is about to prey.[6,7] The sucking reflex inhibits the Babkin reflex.[3] This is an example of the hierarchical interrelation among primitive reflexes that have been phylogenetically developed for animals to successfully attain a purpose: the hand–mouth association for prey is not necessary during feeding, and sucking would be prevented if the Babkin reflex were elicited.[20]

On the other hand, the Babkin reflex can be ontogenetically regarded as the primitive framework evolving into the hand–mouth coordination for food intake in human infants.[2,6] The automatic hand–mouth reflex is transformed into a more adaptive hand–mouth association, which then evolves into the flexible voluntary hand–mouth coordination as the control of the higher brain mechanism over the lower reflex center increases.

Neural mechanisms

Reflexes in early human fetuses

Humphrey[5,21] described that at 14 weeks' gestation, tactile stimulation of the palms first elicits the mouth-opening reflex in human fetuses. However, the spinothalamic connections in fetuses first appear at 20 weeks' gestation, and the first functional axons appear during the cortical plate formation in the thalamocortical connections at 22 weeks' gestation.[22,23] These findings indicate that sensory signals provoked by stimulation of the body surface do not reach the cortex until after midgestation. Accordingly, the mouth-opening reflex in response to palm stimulation in early fetuses is apparently not mediated through the cortex, but rather through the brainstem and upper cervical segments of the spinal cord.

The earliest reported elicitation of a reflex response was in a 7.5-week human fetus ex utero.[5,22,24] This first reflex consists of only contralateral flexion of the head, with movement away from a tactile stimulus applied to the perioral region. With stimulation around the mouth, flexion of the trunk and pelvis are incorporated into the contralateral whole-body flexion by the end of 8 weeks, and the mouth opening appears only as part of the whole-body flexion from 8.5 through 9.5 weeks. The transition of negative (or avoiding) reflexes, movements away from a stimulus, to positive reflexes, movements toward a stimulus, begins to appear in the middle of the ninth week.[5,22]

However, the trigeminal nerve does not carry motor fibers that extend to the trunk region. Additional circuitry is required to explain the whole-body flexion in response to perioral stimulation in early fetuses. Flower[22] speculates that the reticular formation of the brainstem and the reticulospinal pathways are involved in this circuitry. The reticular formation consists of intimately interconnected neural networks, and neurons in this region receive input from most sensory and motor pathways, which are critical for sensorimotor integration. The reticulospinal pathways convey the descending signals from the reticular formation, which reach all spinal cord segments and terminate primarily at interneurons that control the function of alpha and gamma motor neurons.[25] Humphrey[21] also states that the reticulospinal pathways, which develop early embryologically and phylogenetically, undoubtedly participate in the reflexes as soon as the pathways extend further caudally.

Similarity of reflexes between human fetuses and experimental animals

Shimamura[26] demonstrated that, in intact chloralose-anesthetized cats and decerebrated cats, there are four reflex systems, all mediated by the bulbar reticular formation: the spinobulbocranial, spinobulbospinal, craniobulbocranial, and craniobulbospinal reflexes. He applied electrical stimulation to the cutaneous afferent nerves in all forelimbs and hindlimbs, which elicited the reflex motor outflows from all cranial nerves except the first, second, and eighth cranial nerves. Impulses that entered the spinal cord ascended the cord bilaterally and reached the bulbar reticular formation, which provided the signals to motor nuclei of the cranial nerves to produce the bilateral motor effects (the spinobulbocranial reflex). Similarly, the spinal afferent volleys ascended the spinal cord and reached the medulla oblongata, where they were relayed and decussated, and some of the impulses then reentered the spinal cord to produce spinal motor outflows (the spinobulbospinal reflex). He also demonstrated that sensory afferent stimulation of the trigeminal nerve yielded bilateral motor responses of the cranial nerves (the craniobulbocranial reflex) and of the spinal cord at all levels (the craniobulbospinal reflex). In experiments with cats, it has been proved that the descending signals of the spinobulbospinal reflex travel through the reticulospinal pathways, originating from the bulbar reticular formation.[27,28] It has also been demonstrated that the spinobulbospinal reflex, which has been most extensively studied among the four reflexes, can be evoked in all mammals including humans.[29,30]

The reflexes in human fetuses observed by Humphrey[21] are closely akin to those in cats demonstrated by Shimamura[26] in terms of the sites of stimulation, response, and mediation. The similarity indicates that there may be a common basis of the reflex systems in human fetuses and experimental animals. This speculation is also supported by the widely accepted view that the reticular formation is phylogenetically ancient, representing a primitive nerve network on which more anatomically organized and functionally selective connections have developed during evolution.[31] It seems possible in human fetuses that in response to perioral stimulation, the earliest contralateral head flexion is evoked by the cranioreticulocranial reflex through the accessory nerve that supplies the sternocleidomastoid and cervical parts of the trapezius, and by the cranioreticulospinal reflex through the upper cervical nerves that supply other neck muscles. The subsequent flexion of the trunk and pelvis is evoked by the cranioreticulospinal reflex through the spinal nerves that supply erector muscles, and the mouth opening is caused by contraction of the jaw-opening muscles, which is evoked by the cranioreticulocranial reflex through the cranial nerves (trigeminal, facial, and hypoglossal nerves) that supply suprahyoid muscles and by the cranioreticulospinal reflex through the first three cervical nerves that supply the infrahyoid muscles.[21]

Reflex mechanism of the Babkin reflex

Humphrey[5] described that by 12.5 weeks, the reflexogenous cutaneous zones for mouth opening have spread to include the volar surfaces of the forearms and the soles of the feet. At 14 weeks, the close hand–mouth association has been observed following palmar stimulation. Mouth opening with tongue protrusion occurred with turning of the face toward the hand stimulated, which she suggested as the fetal forerunner of the Babkin reflex. The reflex systems for the Babkin reflex in infants are probably the same as those for the hand–mouth reflex in fetuses. On application of pressure to the palms, the mouth opening is evoked by the spinoreticulocranial reflex through the cranial nerves that supply the suprahyoid muscles and by the spinoreticulospinal reflex through the upper cervical nerves that supply the infrahyoid muscles. The closing of the eyes is evoked by the spinoreticulocranial reflex through the facial nerves, the flexion of the forearms is evoked by the spinoreticulospinal reflex through the cervical nerves, and the rotation and flexion of the head are caused by muscle contraction induced by the spinoreticulocranial reflex through accessory nerves and by the spinoreticulospinal reflex through cervical nerves.

Both the wide reflexogenic zones of the Babkin reflex distributed on the cutaneous surfaces of all four limbs[7,8] and the wide variety of the responses including atypical ones as reported by Santos et al.[9] could be well explained according to the assumption that the reflex center lies in the reticular formation that integrates sensory inputs and motor outputs among the signals from or toward the cranial and spinal nerves.

Because the nonprimary motor areas (premotor cortex and supplementary motor cortex), which are involved in the planning and programming of movements, project to the reticular formation,[32-35] these cortices appear to regulate the area that mediates the Babkin reflex in the reticular formation of the brainstem. The premotor cortex receives somatosensory input from the primary sensory cortex and the visual input from the occipital cortex, both via the posterior parietal cortex (association area).[25] The integration of these inputs in the premotor cortex is involved in the control of associated motor behaviors such as reaching and grasping under visual guidance.[25] The premotor cortex is also involved in the eye–mouth association when one opens the mouth to an object approaching the mouth.[18,19] Thus, the eyes-hand-mouth association, which is essential for feeding and food intake, is principally regulated by the nonprimary motor areas.

On the basis of the automatic hand–mouth reflex, more adaptive associated movement develops as the control of the nonprimary motor areas over the reflex mechanism of the brainstem increases. An infant opens its mouth practically every time it grasps an object that has just come in contact with its hand, especially at the moment when he or she draws it toward the mouth.[2] Soon, the associated movement evolves into the selective voluntary eyes-hand-mouth coordination for food intake as the control of the prefrontal cortex over the nonprimary motor cortices becomes predominant.

The Babkin reflex is elicited in neonates and early infants as a result of insufficient control of the reflex center in the brainstem by the immature higher brain mechanisms, including the nonprimary motor areas, but it disappears with age because of the increased inhibition accompanying brain maturation. Some brain lesions will reduce the inhibitory control and lead to prolonged retention of the reflex in infants.[36-38]

Afferent and efferent pathways

Sudden strong pressure to the palms is adequate for eliciting a good response of the Babkin reflex. Ghazni et al.[39] demonstrated in healthy adults that perpendicular pressure to the skin of the dorsal surface of the hand with a 15-g von

Frey filament elicited significant neural activity in the area of the pontine reticular formation, but not with a 2-g filament, as detected by functional magnetic resonance imaging. This finding suggests that the production of neural excitement in the reticular formation on application of pressure to the skin depends on its intensity, although they interpreted the result as a pain response induced by the strong pressure.

Sensory impulses elicited by pressure to the palms ascend largely through the median nerves to reach the lower cervical cord, where they are relayed and decussated, and then the anterior spinothalamic pathways take over the pressure sense and convey it to the thalamus.[40] On the other hand, spinoreticular neurons in the spinal cord respond to input from the skin or deep tissues that is elicited not only by noxious but also by innocuous stimulation.[40] The spinoreticular pathways project to many nuclei of the pontomedullary reticular formation, which modulate the spinal motor function.[31] Although the ascending pathway from the spinal cord of the Babkin reflex is unknown, the collaterals to the reticular formation of the spinothalamic pathways and the spinoreticular pathways may be involved in the reflex arc, because they are the primary ascending pathways from the spinal cord to the reticular formation.[41] The density of cutaneous sensory receptors parallels the level of ascending signals elicited by stimulation. The greatest density is found on the tongue and palmar surface,[42,43] which explains why the palm is the most effective area for eliciting the Babkin reflex.[8]

Mainly based on animal experiments, it has been documented that there are connections from the reticular formation to motor nuclei of the cranial nerves including those of the trigeminal, facial, hypoglossal, and accessory nerves that are related to the mouth-opening and head flexion of the Babkin reflex.[31,44-50] The reticulospinal pathways that develop earlier in fetuses than the cortical structures, including pyramidal cells, would carry the neural excitement to the spinal cord to elicit the body reactions of the reflex.[51]

Palmomental reflex and jaw-opening reflex

The palmomental and jaw-opening reflexes are cutaneous reflexes similar to the Babkin reflex in terms of the hand–mouth association or the mode of response (mouth opening). The palmomental reflex, first described by Marinesco and Radovici,[52] is usually elicited by stroking the thenar eminence in a proximal to distal direction using a sharp object. The response consists of contraction of the ipsilateral, contralateral, or both mentalis muscles. The diagnostic value of the palmomental reflex is considered to be limited, because the reflex is easily elicited in healthy adults,[53] although there are great discrepancies in the literature about the incidence of the reflex, ranging from 2.5% to 50% of healthy adults between 20 and 60 years of age.[54] In children, Parmelee[55] reported that the palmomental reflex was present in 88.8% of 50 newborns, including 43 preterm infants, whereas Marti-Vilalta and Graus[53] found that it was present in 25% of 100 newborn infants born at term who were examined between the second and fifth days of life. Marx and Reschop[56] described that the reflex was observed in 13.2% of 30 infants from 1 to 21 days of life, in 3.3 % of 30 infants from 22 days to 1 year of age, and in 6.6% of 30 children from 2 to 3 years of age. On the other hand, in the study by Little and Masotti,[57] the reflex was observed in 66% of 120 normal children and in 81% of 16 cognitively impaired children under the age of 3 years. The clinical value of the reflex in children is also limited because of its high incidence in normal subjects and large discrepancies among the studies, as in adults.

The threshold of the palmomental reflex seems to be very low compared with other primitive reflexes, because the incidence of the positive response is considerably high even in normal adult subjects. This is in striking contrast to the Babkin reflex: no adult with the reflex has been reported. Marx and Reschop[56] found that the palmomental reflex is more easily elicited after feeding than before feeding in newborn infants, which is precisely the opposite of the Babkin reflex. From these findings, it seems that the palmomental and Babkin reflexes are substantially different entities.

The reflex arc of the palmomental reflex has not been fully elucidated.[58-60] Caccia et al.[60] obtained electrical responses with surface electrodes on the mentalis muscles caused by stroking stimulation of the palmar and dorsal surfaces of the hands in 23 normal adults. In 11 of the subjects, two ipsilateral responses, an early and a late one, were observed, with average latencies of 30.4 and 112.3 ms, respectively. They also recorded the cortical-evoked potentials from the contralateral scalp with the C3-C4 vs Fz configuration on mechanical and electrical stimulation of the skin surface of the thenar eminence. The average latencies of the mechanical and electrical cortical-evoked potentials were 29.2 and 27.2 ms, respectively. Based on these findings, they hypothesized that there are two pathways for the evoked impulses to reach the motor nuclei of the facial nerves: through the short-(paucisynaptic and mediated by the brainstem) or long-loop (trans-thalamocortical) circuit.

The jaw-opening reflex in response to intraoral electrical stimulation was first described by Sherrington[61] in decerebrated cats. Since his report, it has been demonstrated in most decerebrated, anesthetized, or awake experimental animals that perioral or intraoral electrical or mechanical stimulation can easily elicit the activation of jaw-opening muscles and the inhibition of jaw-closing muscles, bilaterally and simultaneously. This reflex has been regarded as an interesting means of exploring the neural bases of oral and facial functions and also as a useful experimental model for studies on nociception and antinociception.[62] In human adult subjects, however, elicitation of the reflex is not as easy as in animals, and some studies have shown that the jaw opening is elicited only on the inhibition of the jaw-closing muscles. The activation of the jaw-opening muscles could not be detected on myographic recording, even with the maximum intensity of nociceptive stimulation.[63,64]

Although animal experiments and clinical studies have implied that the jaw-opening reflex is modulated by the cell functions in the pontomedullary reticular formation,[64-67] the detailed regulatory systems of the reflex are still under investigation.[68,69] In contrast to a great number of reports on animal experiments, studies on human subjects have been limited in number,[63,64,70] and there are no available data for children as far as we are aware. It is unknown whether the jaw-opening reflex in infants is elicited only on the inhibition of jaw-closing muscles as it is in adults.

Conclusions

Although studies concerning the Babkin reflex have been few, there is agreement on the clinical usefulness of the reflex. Infants exhibiting a marked response at the fourth or fifth month of age and persistence of the reflex beyond the fifth month of age should be carefully observed for the appearance of neurological abnormalities. The Babkin reflex seems to be mediated by the reticular formation of the brainstem. On the basis of the hand–mouth reflex, more adaptive movement develops as the control of nonprimary motor cortices over the reflex mechanism in the reticular formation increases, which evolves into the voluntary eyes-hand-mouth coordination necessary for food intake as the control of the prefrontal cortex over the nonprimary motor cortices becomes predominant. The Babkin reflex and palmomental reflex may be different entities. There are currently no available data concerning the jaw-opening reflex in children.

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