Sucking Physiology

Chapter One from Color Atlas of Infant Tongue-Tie and Lip-Tie Laser Frenectomy

Alison Hazelbaker, PhD, IBCLC, FILCA

Tongue-tie impacts all three aspects of infant feeding: sucking, swallowing, and breathing. Optimal infant feeding depends upon the exquisite timing of these three aspects. As Wolf and Glass (1992) state:

> Three processes—sucking, swallowing, and breathing—are the cornerstones of infant feeding. Not only are these processes related functionally as they come together in bottle- and breast-feeding, but they are related anatomically. The anatomical structures responsible for sucking, swallowing, and breathing are physically in close proximity and overlap in function, with some structures having roles in the life-sustaining processes of both feeding and respiration. The close proximity of these structures, the interrelated nature of their functions, and the dual role some structures play in providing oxygen and nourishment to the body often underlie the feeding problems of infants. (p. 3)

To best understand how tongue-tie disrupts infant feeding, it is necessary to take a developmental journey, starting with embryology and ending at the end of the first postpartum month, when a particular infant's suck-swallow-breathe style has fully emerged.

Embryology

The primitive human being rapidly develops greater and greater sophistication as embryological days glide into weeks. The two ends of the curved tube that is the primitive embryo fold in on themselves. This embryonic infolding, occurring in week 4, allows the formation of various prestructures: the dorsal yolk sac is incorporated into the embryo as the primitive gut, the infolding at the newly formed head region becomes the oropharyngeal membrane, and the heart moves inferiorly, allowing a pit to form between it and the brain just above the first tissue (branchial) arch (Avery and Chiego, 2006). This pit soon becomes the stomodeum and, ultimately, the oral cavity. At its deepest, the oropharyngeal membrane forms the back of this cavity, only to rupture in week 5, making the oral cavity continuous with the tubular foregut. This newly formed structure eventually becomes the combined food passage and airway.

At the same time, arches form ventrally that give rise to the various tissues that comprise the face, mouth, and pharynx. The face and oral structures develop during embryonic weeks 4–7, courtesy of the tissue (branchial) arches derived from the neural crest cells that develop in the primordial pharynx floor. In one short week, the first of six pairs of branchial arches emerges and can be observed. The other five pairs emerge in the subsequent days and

weeks. These stacked, parallel arches play an important role in oral and facial development and contain muscle, nerve, blood vessel, and skeletal elements.

The muscles of mastication develop from the first branchial arch. In week 5, muscle cells begin their migration, arriving at their various destinations by week 6. They begin to replicate and differentiate into the masseters, medial and lateral pterygoids, and temporalis muscles. The trigeminal nerve (CNV)—the resident cranial nerve supplying the first branchial arch—innervates these developing muscles, the skin of the lower face, the anterior tongue mucosa, floor of the mouth, gingiva, and the mandibular teeth. This arch also gives rise to the maxillary artery and part of the external carotid artery.

The second of the pharyngeal arches, also known as the hyoid arch, develops into the lateroposterior tongue body, the facial muscles, the facial vessels, and aspects of the hyoid bone. Its resident cartilage, Reichert's cartilage, forms the basis for much of the stapes and contributes to the malleus and incus. In week 7, the facial muscles rapidly spread, sheetlike, into the facial area accompanying the branching facial nerve. This task is completed in the early fetal period.

The third branchial arch gives rise to the greater horns of the hyoid and its inferior body. The root of the tongue develops from this arch as well. The glossopharyngeal nerve (CN IX) supplies this arch and reaches out to supply the forming stylopharyngeal and upper pharyngeal constrictor muscles, as well as the posterior third of the forming tongue. Both the mesoderm and the neural crest tissues give rise to aspects of the carotid artery; the common carotid and, subsequently, the internal carotid form from the branchial arch artery, and the carotid body arises from the neural crest and is innervated by the glossopharyngeal nerve (see Figure 2-4).

The superior laryngeal branch of the vagus nerve serves the fourth branchial arch, from which arise the thyroid cartilage, the cricothyroid, the pharyngeal constrictor muscles, the palatopharyngeus, the levator veli palatini and uvular muscles, and the palatoglossus muscle of the tongue. The muscle cell migration proceeds posteriorly, then spreads simultaneously caudad and cephalad to form the continuous semi-tube that characterizes the fully formed throat. Its artery develops into the aortic arch and contributes to the right subclavian and brachiocephalic arteries.

The fifth arch lives a short life, disappearing almost as soon as it develops.

The cricoid and laryngeal cartilages and intrinsic laryngeal musculature arise from the sixth arch, whose nerve supply comes from the recurrent laryngeal nerve, a branch of the vagus. Its nutritional supply via the sixth artery later forms the pulmonary arteries and the temporary ductus arteriosus so important to fetal life. Postnatally, the ductus arteriosus becomes the ligmentum arteriosum.

Ectoderm coats the anterior surfaces of the arches and the interior surface of the second arch. The oral epithelium emerges from the ectoderm of the first and second arches. Endoderm lines the interior surfaces of arches three through six. The gastrointestinal tract and its glands arise from this primordial tissue. At the potent core of the arches, within the

mesoderm, lie the elemental tissues for the blood vessels, muscles, nerves, cartilages, and bones of the face, which will differentiate into these various structures in concert with one another.

The mandible is the first structure to develop as the medial ends of the mandibular prominences of the first branchial arch merge. As the mandible begins its formation, the maxillary prominences also make their move medially, followed closely by the medial nasal prominences. The surrounding mesenchyme proliferates to form the medial and lateral nasal prominences, which in turn become the primitive nasal sacs. This newly generated nasal cavity is separated from its sister oral cavity by the oronasal membrane, which ruptures at the sixth week and becomes the primitive choanae. From this point on, the nasal and oral cavities are directly communicating neighbors.

At first, the developing tongue occupies this space with little room for other structures. The cavity is bounded in front by the primary palate, which, as it develops and rises, becomes the permanent dividing structure between the nasal and oral cavities**.**

Number	Arch-Derived Structures	Groove-Derived Structures	Pouch-Derived Structu
I (Mandibular)	Trigeminal (V) nerve Muscles of mastication, anterior belly of the digastrics muscle, mylohyoid, tensor tympani, tensor palatine Malleus, incus, sphenomandibular ligament, Meckel's cartilage	External auditory meatus	Middle ear and eustachia
II (Hyoid)	Facial (VII) nerve Muscles of facial expression, stapedius, stylohyoid, posterior belly of the digastric muscle Stapes, styloid process, stylohyoid ligament, lesser cornu of hyoid, upper part of the hyoid body	Cervical fistula	
III	Glossopharyngeal (IX) nerve Stylopharyngeus muscle Greater cornu of the hyoid, lower part of the hyoid body	Cervical fistula	Palatine tonsil, thymus, i thyroid
IV	Vagus (X) nerve, Laryngeal muscles, pharyngeal constrictor muscles Laryngeal cartilages	Cervical fistula	
V	Spinal accessory (XI) nerve, Sternocleidomastoid and trapezius muscles		
VI			Superior parathyroi ultimobranchial bod

Table 1-1: Summary of structures that develop from pharyngeal arches, grooves, and pouches.

Tongue Development

The tongue organ forms from the lingual primordium, which arises from branchial arches one to four in weeks 4–5, each arch contributing specific aspects of the primordial structure. Mesenchymal proliferation of the pharyngeal arches generates multiple swellings on the floor of the mouth. Bordered by bilateral lingual swellings, the tuberculum impar rises up from the midline of the mandibular process. The lingual swellings merge with each other and with the tuberculum impar at the midline, forming a larger mass that gives rise to the mucous membrane of the anterior two-thirds of the tongue.

The mesenchyme of arches two to four form a copula (arch two-generated) and hypobranchial eminence (arches three- and four-generated) that merge to create a large mass. This posterior mass becomes the root of the tongue and its overlying mucosa. The sulcus terminalis, a V-shaped demarcation, forms the line between the mobile tongue body and the anchored tongue root.

The tongue muscles arise from the myoblasts generated in the bilateral occipital myotomes concurrently with the other muscles of the orofacial and pharyngeal areas. The muscle cell masses migrate forward into the newly formed oral cavity floor, where the lingual primordium has begun to develop. The nerve cells that form the hypoglossal nerve migrate together with the myoblasts. Both infiltrate the lingual primordium in week 5 and push the lingual primordial tissue to develop into the tongue mass of differentiated and mature striated muscle, fully completing their developmental task in gestational week 9.

The ectoderm surrounding the still-developing tongue grows downward, allowing the tongue to separate from the floor of the mouth. This ectodermal tissue then degenerates to form the lingual sulcus and frees the tongue body for movement while gently anchoring it to the remaining surrounding oral and pharyngeal tissues. Incomplete degeneration of this tissue and the mesodermally derived muscular and connective tissue, which it envelops, leaves tissue behind that restricts the tongue's mobility to one degree or another. What we now call tongue-tie is the incomplete degeneration of tissue at the apex of the tongue (what we refer to as the "tongue blade"). Ankyloglossia results from a complete failure of downward ectodermal growth and subsequent apoptosis, preventing the tongue from differentiating from the mouth floor and partial ankyloglossia, or tongue-tie, results from fractional failure of apoptosis due to some genetic or epigenetic event.

The complex innervation of the tongue organ owes its origin to the multiple generating locations of the organ. The anterior sensory innervation arises out of the generative first branchial arch, which is innervated by the trigeminal nerve. The fifth cranial nerve therefore serves the sensory needs of the anterior two-thirds of the tongue. The posterior one-third of the tongue's sensory innervation arises from the same generative third arch that is innervated by the glossopharyngeal nerve. Motor coordination comes from the hypoglossal nerve, which arises from the occipital somites, as do the myoblasts, which form the tongue musculature.

Maxillary Lip Development

Week 6 is an exciting and busy time for the embryo's developing mouth. The maxillary and nasal prominences seriously begin their migration toward one another, pushed into their journey by enlargement of the brain. This brain enlargement gently forces the eyes to the front of the face and compels the maxillary and nasal prominences to move toward the midline of the soon-to-be-completed face. As the maxillary prominences move medially, the lateral nasal prominences enlarge and reach toward both the maxillary prominences and the medial nasal prominences. At the same time, the medial nasal prominences push toward one another, fuse in the midline, and enlarge distally and caudally. Subsequently, cell adhesion molecules should cause these three structures to fuse at the areas where their edges come into contact. If not, clefts develop at the fusion sites.

The frenum of the upper lip arises from the medial nasal prominence mesenchyme toward the end of this developmental phase, anchoring and stabilizing the newly formed upper lip to the developed gumline residing just behind. The maxillary frenum, part of the nose, is composed of collagen and elastin fibers and may also contain a few muscle fibers. This frenum is highly vascular and contains myelinated nerve fibers.

The migration-fusion process of the mouth sphincter takes five weeks from start to finish. At the end of the process, 76% of newborns possess a papillary insertion phenotype that will recede and become smaller as the process of dentition ensues.

Fetal Development of Sucking and Swallowing

Sucking and swallowing mature at different rates in the fetal period. They have been observed in fetuses as early as 12½ weeks gestation, but they are not well-coordinated as a sequence at this very early stage of development. A coordinated pattern of sucking and swallowing emerges between weeks 32 and 35. At about week 34, a stable rhythm asserts itself, enabling an infant who may be born at this age to oral feed without much compromise to caloric intake. This pattern reaches full maturity between weeks 39 and 40. At birth, the infant is fully capable of coordinating sucking with swallowing with breathing. If the infant cannot accomplish this complex sequence with ease, something has failed to mature or has disrupted synchronization.

Sources disagree about the exact timing of the emergence of sucking. Arvedson and Brodsky[1] report that "true sucking begins around the 18th–24th [gestational] week." They define **sucking** as marked by a backward and forward movement of the tongue, with emphasis on the backward movement. According to Wolf and Glass,[2] sucking has been observed *in utero* between weeks 15 and 18. They describe these mouthing movements as immature sucks. Sperber[3] asserts that intrauterine thumb-sucking occurs as early as week 16. This activity builds upon the muscular movements of the lips and tongue seen early in the fetal period.

Herbst[4] suggests that the mouthing movements seen at week 24 may be a form of nonnutritive sucking. At weeks 27–28, random mouthing activities can be visualized. Several texts note a stronger burst-pause sucking pattern beginning to emerge by week 32. This more rhythmic activity matures into the burst-pause rhythmicity of the suck-swallow-breathe triad.[5]

The pharyngeal swallow, an early motor response of the pharynx, first occurs between weeks 10 and 11 of gestation.[4] By week 12, this swallowing can be observed.[1] At this time, the fetus begins to drink its amniotic fluid, which lays down the meconium in the gastrointestinal tract. It is possible that the action of taking in amniotic fluid is a rehearsal for postnatal suck-swallow-breathe coordination. By week 40, a full-term fetus is capable of swallowing 450 ml of amniotic fluid (about half of the total fluid volume) in a single day. During pregnancy, an imbalance in amniotic fluid circulation can be an indicator of an anatomical or physiological swallowing problem.

At 37 weeks gestation, the fetus possesses an intact suck–swallow–breathe mechanism that forms the foundation for later feeding efforts, including a voluntary swallow. The maturation that occurs in the first month postpartum actually represents stylistic preferences or responses influenced by the infant's environment: rate of milk flow, hunger and satiety levels, breast size and shape, bottle-teat type, and other factors. The infant's ability to respond to environmental cues indicates the amount of volitional control built into the suck–swallow–breathe mechanism.[5]

What Goes Wrong?

Avery and Chiego[6] indicate that the face and palate are the most likely areas to malform; their development depends upon exact timing and is therefore extremely vulnerable to external disruptions. According to Arvedson and Brodsky,[1] tongue abnormalities are relatively uncommon, especially those that interfere with feeding and swallowing. However, the dramatic incidence of tongue-tie, both as a stand-alone condition and accompanying malformation, underscores its significance. Most writers and researchers consider tongue-tie a minor anomaly, but one of note.

Tongue-tie is caused by insufficient apoptosis of the tongue during the stage of differentiation of the tongue from the floor of the mouth. "Apoptosis, or programmed cell death, is the fragmentation of a cell into membrane bound particles, which are then eliminated by phagocytosis by specialized cells."[7] Programmed cell death during the embryonic period eliminates obsolete or purposefully transient tissues to make way for ever-increasing differentiation and complexity.

The typical programmed cell regression under the tongue during embryogenesis leaves a small segment of tissue we identify as the normal lingual frenulum—an extension of the lingual oral connective tissue that stabilizes the tongue organ by gently connecting it to the floor of the mouth throughout life. Failure of cell destruction can occur to one degree or another during this regression stage, leaving variable amounts of tissue behind and creating the erratic and confusing presentation of tongue-tie.

Tissues are most susceptible to defective development when they begin to differentiate in embryonic weeks 2–8. A defect in the development of a group of cells is considerably less damaging than a defect in an organ or organ system. The smaller and less complex the development, the less extensive the problem created, as long as that structure does not play a foundational role in the development of higher-order and more complex structures. This may

be why abnormalities in the other frena of the mouth are rare compared to anomalies of the tongue organ.

Abnormalities of the mandibular, maxillary, and buccal frena have as yet to be fully quantified. Most, but not all, are linked with syndromes. Causes of prominent, short, tight, or otherwise abnormal oral frena are as yet unknown but are likely to be multifactorial, as are cleft lip and palate.

Abnormal development results from genetic and environmental factors and is usually a byproduct of both. The anomaly of tongue-tie has a clear genetic influence mediated by a mutated gene (or genes). How the gene(s) mutates has yet to be quantified. The role of environmental teratogens as a mediating factor of tongue-tie is poorly understood but may influence mutation.

Physiology of Infant Suck

The tongue organ resides in an oral "box" of sorts, limited in its range of movement by the borders created by the mandible, the maxillae, the palate, the cheeks, and the mouth floor. This arrangement, coupled with the horizontal orientation of the cranium, the gentle slope of the oropharynx, and the closely apposed structures of the soft palate and epiglottis, optimally controls a liquid bolus of milk to protect the airway and coordinate the swallow with the breath.

To create a safe and efficient swallow, 31 muscles must be coordinated by six cranial nerves and several cervical nerves by way of a complex interaction of signaling in both the brain stem and cerebral cortex. The complexity of coordinating swallowing and breathing accounts for the recruitment of so many nerves and muscles.

The trigeminal nerve carries sensory information from the mandible and from the various touch and pressure receptors of the hard palate and oral mucosa to modulate sucking and chewing action. Taste sensation is carried through the facial and glossopharyngeal nerves. Motor output occurs as a result of the action of the trigeminal nerve, which controls the jaw movements, and the hypoglossal nerve, which controls the tongue muscles.

After mouth opening occurs, the tongue extrudes to either accept the food or to latch on to the breast nipple or bottle teat. During sucking, the anterior tongue stabilizes the breast or bottle teat in the mouth and the remaining edges create a seal around it. Progressive contractile movements then initiate a sequence of pressure changes that move the bolus progressively from the high positive pressure area (anterior tongue against palate) to the low pressure area (dropping posterior tongue), where a vacuum has been generated. As the posterior tongue rapidly drops, the anterior tongue remains in contact with the hard palate, slightly changing the degree of positive pressure exerted by it. The milk then moves from the high pressure to low pressure area via a push-pull force.

To assist this intraoral pressure, the lateral tongue remains in contact with the buccal fat pads and the soft palate at its posterior border. As the dorsum of the tongue elongates and drops to create space in the posterior oral cavity, the soft palate quickly moves anteriorly,

followed by a rapid swing in a backward and upward direction. The soft palate closes off the nasopharynx, rapidly increasing the oral-pharyngeal space, which pulls the bolus backward. The posterior tongue then serves as a ramp that moves back to touch the pharyngeal wall, thereby mechanically dumping the bolus into the pharynx.

At the same time the dumping occurs, the hyoid bone moves upward toward the base of the tongue, pulling the larynx up and assisting laryngeal vestibule closure. The epiglottis, a cartilaginous structure that projects from the tongue-base, gracefully drops over the laryngeal aditus, specifically over the aryepiglottic folds, in part as a result of the upward and anterior movement of the hyoid. The epiglottic drape over the laryngeal vestibule shields it from the oncoming bolus as it opens the velleculae—wedge-shaped spaces formed by the anterior wall and lateral musculature of the pharynx and the lateral edges of the epiglottis—to the dropping bolus.

Next, the posterior-inferior tilt of the epiglottis over the laryngeal vestibule diverts the bolus unilaterally or bilaterally. When the bolus splits roughly in half, each half drops into the ipsilateral pyriform sinus and then rejoins its mate just before entering the open esophagus.

The push-pull originated by the tongue's progressive contractile forces continues in the pharyngeal phase of swallowing. The bolus makes its progressive and rapid descent toward the esophagus with help from the pharyngeal constrictor muscles and their progressive contractile movements, which generate positive pressure behind or above the bolus and negative or low pressure in front or below the bolus.

The rising larynx becomes smaller in diameter, making its vestibule smaller as well. The closure of both true and false vocal folds further ensures that the dropping bolus will not enter the airway. The laryngeal shift anteriorly and superiorly places traction on the upper esophageal sphincter, signaling it to relax and allowing an esophageal "gape" to occur. The now-open upper esophagus receives the reunited bolus, initiating the fourth phase of the swallow. The bolus's journey requires only one second from the initiation of phase two through the end of phase three, thanks to the combined efforts of the vagal branches and the glossopharyngeal and hypoglossal nerves.

In this final phase of swallowing, the now peristaltic contraction of the esophagus propels the bolus downward toward the stomach, continuing the push-pull of the changing pressure gradient aspects of the previous three phases. The upper esophageal sphincter (or cricopharyngeal sphincter) closes after the bolus moves through at the proximal end of the "food tube." The lower esophageal sphincter (or cardiac sphincter) opens to receive the bolus and admit passage into the stomach. Along its length, as the bolus enters the top of the tube, first the muscles are inhibited, then a proximal-to-distal excitation, caused by the vagus nerve, initiates the cephalocaudal movement of the bolus.

The action of the tongue organ plays a critical role in successful completion of each suck-swallow-breathe cycle. A too-tight-too-short lingual frenulum compromises this well-coordinated action in several ways.

Functional Deficits Created by Tongue-Tie

As indicated above, tongue movements inside the oral "box" remain somewhat limited. The tongue can bunch, hump, retract, thrust, flatten, lateralize, lift, and peristalse. All these movements are designed to safely move either liquid or solid foods in the mouth toward the oropharynx for the swallow. Tongue-tie reduces the tongue's range of motion, forcing it to protect the airway by using the less productive movements in its repertoire—humping, retracting, flattening, and thrusting. Tongue-tied babies have difficulty lifting the tongue away from the mouth floor, thereby compromising intraoral pressure changes. Tongue-tie also compromises peristalsis.

Babies may compensate for the difficulty they have in maintaining a seal by tensing the jaw, thereby keeping the tongue more closely approximated to the breast or bottle teat. This tense jaw further compromises tongue motion and can create nipple pain and damage to the mother during breastfeeding. Conversely, the tongue may remain relatively flat, compromising seal and peristalsis and resulting in inadequate milk transfer.

In either situation, the tongue fails to set up a protective swallow and a tongue-tied baby may have to protect the airway via overutilization of the protective gag and cough reflexes. Tongue-tie puts the infant at serious risk of aspirating. A breastfed baby's lungs most likely will not be compromised, as breastmilk is benign even when aspirated. Formula feeding, on the other hand, poses a risk of lung compromise.

Babies who have difficulty feeding will often withdraw in one of a number ways: They may vigorously begin to feed but then slow the pace of their sucking to reduce threat to the airway. They may move minimally to ensure that only small amounts of milk enter the pharynx at one time. They may pop off the breast as a means to protect the airway. Or they may refuse to feed altogether.

Many other structural problems that affect the mouth floor, tongue, and throat manifest in the same compromises, making differential diagnosis essential. Not all babies with tongue-motion compromise have tongue-tie. Therefore, tongue motion deficits alone cannot be used as diagnostic criteria for tongue-tie.

We must also be careful of the assertion that "lip-tie" causes breastfeeding problems. There is, as of the date of publication of this textbook, no basis in the scientific literature for the condition, treated surgically, that some practitioners refer to as "maxillary lip-tie." The article that originally mentioned lip-tie as a cause of breastfeeding problems contained no data,[7] and no data exists that demonstrates the relationship between infant maxillary frena and feeding. Some anecdotal reports claim that a papillary insertion phenotype maxillary frenum causes problems with the lip seal at breast; many of these reports also claim that there is a statistically significant positive correlation between tongue-tie and lip-tie. There is no data to back up these claims. Further, reports in the literature of such a correlation failed to control the treatment variables: none of the babies in the cohorts had lip-tie revision without concomitant lingual revision to serve as a control.[8–10]

More research on the morphology and function of various maxillary frena phenotypes must be done before we can ethically justify surgery for this anecdotal concern. With that said, maxillary lip-tie may exist in infants. Some children appear to have difficulty with a variation of the papillary insertion phenotype that fails to recede with growth and development. The dilemma is, we do not yet know how to predict which babies will grow up to have such issues.

Chapter 1 References

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