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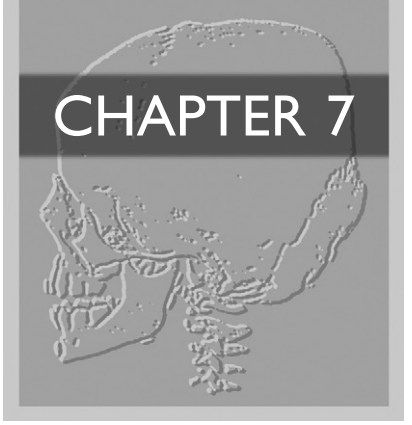

CLINICAL CONDITIONS

This chapter addresses some clinical conditions than can be effectively treated using manipulative procedures. Such treatment, however, is not intended to replace definitive medical or surgical treatments. Rather this therapeutic option is offered as a gentle, alternative, non-invasive approach, with essentially no untoward side effects, to be employed as a first line of therapy. Patients respond quickly to a specific technique when it is appropriately applied following a precise diagnosis.

It is the responsibility of the practitioner to ensure that underlying pathologies requiring more aggressive medical or surgical treatment have been ruled out or appropriately addressed. It should also be borne in mind that when a patient fails to respond to treatment as anticipated, they should be completely reassessed. If the osteopathic practitioner does not see a significant functional change in the patient's condition by the third to fifth treatment, the probability of misdiagnosis is likely.

For each clinical condition, the specific features of the condition are addressed. For the basic treatment protocols and descriptions of individual procedures, see Chapters 5 (Examination of the patient) and 6 (Treatment of the patient).

7.1 IMBALANCES OF THE AXIAL SKELETON

Congenital muscular torticollis and plagiocephaly are the most frequently occurring asymmetries of infancy. Brachial plexus injury, fracture of the clavicle, pectus excavatum and carinatum, scoliosis, kyphosis and vertebral somatic dysfunctions are other commonly encountered conditions with structural and functional consequences that can be addressed with osteopathic manipulative treatment.

TORTICOLLIS

Torticollis may be subdivided into congenital torticollis and congenital muscular torticollis (CMT). The patient with congenital torticollis presents at birth with their head tilted toward the involved side and rotated toward the opposite side. They commonly have associated medical conditions, such as osseous malformations, basilar impression or atlantooccipital anomalies, and neurologic disorders such as Arnold–Chiari malformations.[1] These underlying conditions should be diagnosed and appropriate treatment initiated before osteopathic manipulation is considered.

The patient with CMT, on the other hand, is symmetric at birth, and the asymmetry develops in the first weeks of life. CMT presents with a tight

sternocleidomastoid (SCM) muscle, causing the child's head to be tilted toward the side of the tight muscle and rotated in the opposite direction. A mass, or fibromastosis colli, can eventually be palpated within the muscle. The incidence of CMT ranges from 0.3 to 1.9%, but is as high as 3.92% when cases are diagnosed sonographically.[2] CMT occurs more often among boys than girls.[2,3] Some authors report a higher incidence on the right side,[1,4] whereas others report that it is more frequently encountered on the left.[2] Primiparity, assisted delivery[2,3] and breech presentation[3,5] frequently appear in the birth history. The larger the infant, birth body length and shoulder width, with more associated delivery trauma, the higher the incidence of CMT.[2]

Multiple theories to explain the etiology of CMT have been proposed. Van Roonhysen (1670) postulated abnormal uterine pressure as a cause of torticollis.[4] Pommerol, in the 19th century, attributed the unilateral shortening of the SCM to abnormal fetal position.[6] These 'intrauterine' theories have also attempted to explain the presence of several other deformities present at birth. Many authors have commented on aberrant constraint of the infant within the uterus and the association between torticollis, plagiocephaly, bat ears, scoliosis and congenital hip dislocation,[7–10] or hip dysplasia.[3]

Other theories link CMT to birth trauma. The high incidence of breech presentation associated with CMT may document the role of birth trauma in its occurrence. Others suggest that intrauterine torticollis predisposes the infant to breech presentation or forceps delivery.[11] It has been proposed that birth trauma with injury of the SCM and a resultant hematoma, which is then replaced with fibrous tissue, causes CMT. Histologic studies have, however, failed to support this proposition. Proposed compressive arterial occlusion as an etiology[12] has also been dismissed. Vascular compromise does not occur because there is abundant arterial and venous supply to the SCM that follows no regular or segmental pattern and has multiple anastomoses.[13]

Among more recent theories, CMT is proposed as the sequela of an intrauterine or perinatal compartment syndrome.[14] A bilateral imbalance of structures responsible for control of head posture may play a role – for example, as may be the case for the interstitial nucleus of Cajal, a neural integrator for head posture.[15]

Because the torticollis is not present at birth but appears later, it is possible that it results from improper handling of the child or incorrect positioning as in a car seat. Resultant dysfunction of the occipito-atlanto-axial joints may, therefore, occur and has been proposed as a cause for CMT.[16]

Several clinical variants are observed. The SCM mass is not always present.[17] Smaller masses may be found on the occipital bone below the superior nuchal line. When an SCM mass is palpable, usually in the first 2 months of life, it appears well circumscribed within the muscle, is located in the midportion of the SCM and ranges in size from 8 to 15.8 mm on maximal transverse diameter and from 13.7 to 45.8 mm in length (measured with ultrasound).[18] This mass typically disappears during the 1st year of life without any correlation with the resolution of the CMT.[3]

Although the findings of head tilt toward the involved side with head rotation toward the opposite side are consistent, the amount of rotation can differ between subjects. Contractures of the semispinalis capitis and the splenius capitis are sometimes present. CMT is probably the consequence of several concomitant factors. The problem may begin with a faulty intrauterine position that weakens the SCM, making it vulnerable to birth trauma and thus creating the dysfunction.

Osteopathic practitioners use their knowledge of anatomy to develop a rationale for treatment. The SCM arises, by two heads, from the sternum and clavicle (Fig. 7.1.1). The medial (or sternal) head has its origin on the upper part of the anterior surface of the manubrium sterni and the lateral (or clavicular) head arises from the anterior surface and superior border of the medial third of the clavicle. Initially, the two heads are separated from one another. They gradually join, below the middle of the neck, to form the body of a thick, rounded muscle. The SCM is inserted into the lateral surface of the mastoid process by a strong tendon and into the lateral half of the superior nuchal line of the occipital bone by a thin aponeurosis.

The two heads of the SCM consist of different types of fiber, the sternal head being more tendinous and the clavicular head being composed of fleshy and aponeurotic fibers. The clavicle is more mobile than the sternum and is subject to significant stress during the birth process, which may explain a different strain being put on the two parts of the SCM. A distinction between 'sternal torticollis' and 'clavicular torticollis' has been proposed.[19]

The insertion of the SCM covers the occipitomastoid suture; therefore, SCM tightness should be released whenever attempting to treat this suture. Conversely, somatic dysfunction between the temporal and the occipital bones will affect the SCM. The jugular foramen – located at the anterior end

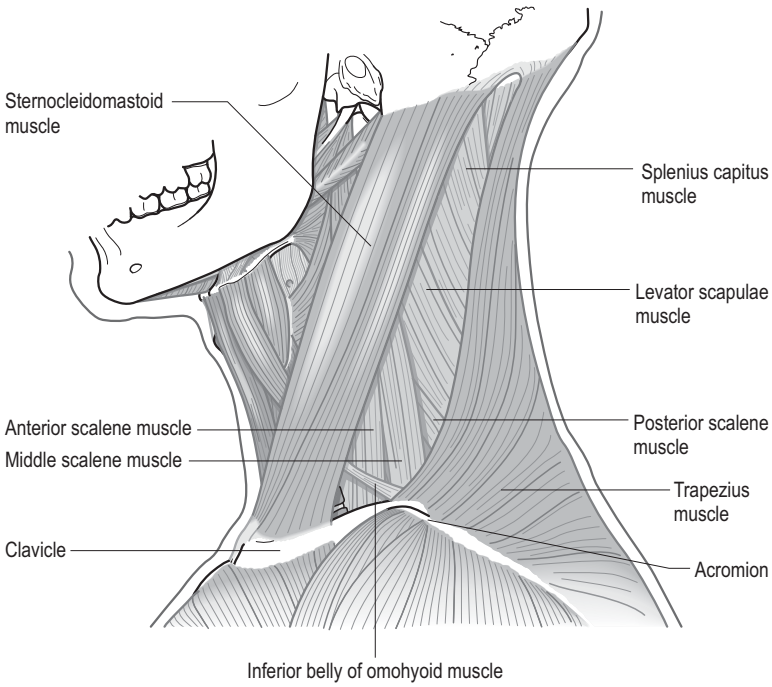

Figure 7.1.1. *Sternocleidomastoid muscle.*

of the occipitomastoid suture, between the petrous portion of the temporal bone and the occiput – will also be affected. The jugular foramen contains the inferior petrosal sinus and the sigmoid sinus that unite to form the internal jugular vein. It also contains the glossopharyngeal (CN IX), the vagus (CN X) and the accessory (CN XI) nerves. CN XI provides the motor supply to the SCM, its proprioceptive fibers passing through branches from the anterior divisions of the second and third cervical nerves to innervate the SCM. Thus, Sutherland stated: 'You will probably find the source of the torticollis to be entrapment neuropathy of the eleventh cranial nerve at the jugular foramen.'[20] Jacquemart and Piedallu, in 1964, recommended that osteopathic manipulation for CMT be directed at somatic dysfunction of the occiput and upper cervical spine.[16]

The mastoid process is not fully developed in the newborn and is totally covered by the tendon of the SCM from its apex to its superior border. The development of the mastoid process is linked to the traction of the SCM; asymmetric sidebending and rotation of the head, as in CMT, will cause the mastoid processes to develop asymmetrically. Cephalometric analysis demonstrates that, left untreated, persistent torticollis can lead to skull and facial asymmetry.[21] When it occurs, the cranial base deformation appears early, the changes being more significant in the posterior cranial fossa, whereas the facial deformity will develop later in childhood. Both the cranial base and facial deformities tend to increase with age.[22] Furthermore, the asymmetric function of the neck muscles stresses the mechanisms of postural control. Abnormal sensory input to the CNS and a sense of instability occurs that has to be compensated for with vision.[23] This can affect the infant's developing visual function. To prevent these sequelae, osteopathic procedures should be employed to address CMT as early as possible.

Physical examination and treatment

Observe the child for spontaneous rotation and sidebending of the cervical region. A good way to evaluate the range of motion in rotation and sidebending is to have the baby visually follow a toy moved in the directions to be tested. The child can also be held in the arms of the practitioner, facing the parents. The practitioner then pivots to the right and to the left, holding the infant in such a way that, in order to continue to observe their parents, the child must actively turn their head first to the left in response to the practitioner's pivoting right, and then to the right in response to the practitioner's pivoting left. Having the parents participate in this procedure allows them to understand the extent

of the child's restriction of motion; repeating the procedure after treating the child allows one to evaluate the effect of the treatment.

The size of the SCM mass (when present) and the tension of the muscle fibers should be evaluated. Palpate the infant to identify membranous, myofascial and interosseous somatic dysfunction, particularly in the upper thoracic spine, pectoral girdle, cervico-occipital area and cranium (temporal bone, occiput, occipitomastoid suture and jugular foramen). Treatment should use indirect principles. Because the mass associated with CMT often develops at the conjunction of the two heads of the SCM, in order to release the dysfunction the bones to which the muscle is attached (clavicle, sternum, occiput and temporal bone) should be balanced. Indirect myofascial release of the SCM may be employed. If the infant is treated early, before a dysfunctional pattern becomes engrained, osteopathic manipulation can rapidly alleviate the asymmetry of SCM.

The caregiver should be taught an active positioning program to be employed at home. For example, approaching the infant from the side opposite the rotation during daily activities, such as feeding and playing, head turning and lengthening of the SCM can be facilitated. The infant should be placed in a sleeping position that avoids reinforcement of the SCM shortening. It is improper to attempt to accomplish this by propping the infant's head with a pillow in such a way as to lengthen the SCM. Although this may appear to hold the head in the desired position, it induces a stretch reflex in the tight muscle, maintaining the shortening.

Gentle stretching exercises that induce active SCM stretching can be taught. These should be employed to rotate the head toward, and sidebend the neck away, from the side of the tight SCM. Such exercises are indicated until a full range of movement has been obtained.

PLAGIOCEPHALY

The term plagiocephaly – derived from the Greek *plagios* (oblique) and *kephalê* (head) – indicates distortion of the head and refers clinically to cranial asymmetry. Cranial deformations have been (and still are) produced intentionally, depending on the period and country, as signs of distinction, beauty, health, courage, freedom and nobility. The oldest known example is from Iraq, *c*.45 000 BC, and the earliest written reference is from Hippocrates, around 400 BC (*Airs, Waters, Places*), describing the Macrocephales who practiced head deformation.[24] Intentional plagiocephaly was obtained by pressure applied to an infant's skull, either through manual molding or with boards, pads or stones. Different deformities were induced depending on the methods employed.[25] The use of cradleboards and the wearing of headdresses are examples of other traditions that also resulted in head deformation.

Non-intentional plagiocephaly can be associated with premature sutural closure or craniosynostosis. Premature synostosis of one or several cranial sutures may be the result of genetic or metabolic conditions.[26,27] The fused suture does not allow bone growth and the shape of the skull reflects this anomaly: a brachycephaly develops when the coronal suture fuses, a dolichocephaly or a scaphocephaly when the sagittal suture fuses, and a trigonocephaly when the metopic suture fuses. Unilateral synostosis of the lambdoid or coronal suture (Fig. 7.1.2) results in a posterior or anterior plagiocephaly, respectively. Although any suture may be involved in synostotic plagiocephaly, true lambdoid synostosis with posterior plagiocephaly rarely occurs[28] and represents only 3.1% of all synostoses.[29] In the general population, the incidence of craniosynostosis has been estimated to be as low as 1 in 2100–3000 live births;[27,30] however, it is imperative that the diagnosis of synostotic plagiocephaly, which is most specifically accomplished by radiologic means, be made when the condition is present.

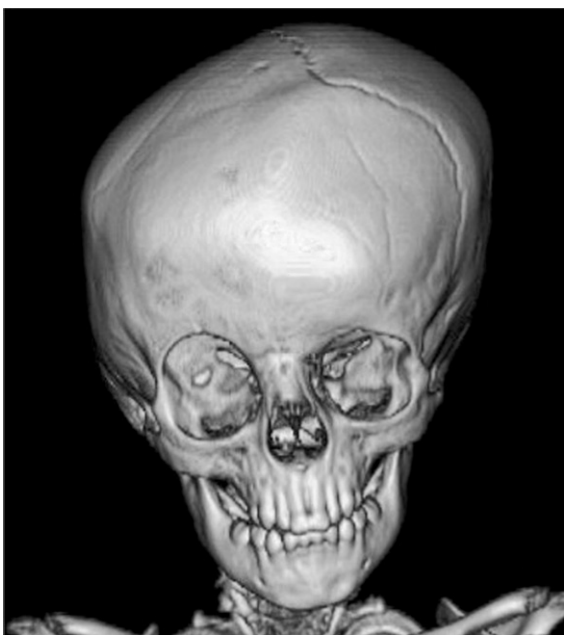

Figure 7.1.2. *Synostosis of right coronal suture.*

Children presenting with craniosynostosis should be monitored closely by a pediatric neurosurgeon for signs and symptoms of increased intracranial pressure. Treatment may require surgery, particularly for severe cases.

Non-synostotic plagiocephaly (NSP), also referred to as functional plagiocephaly, must be differentiated from craniosynostosis. The prevalence of NSP is estimated to be as high as 9.9% of all children under the age of 6 months.[31] NSP is identified as either frontal[32] or occipital[33] (Figs 7.1.3 and 7.1.4) depending on the site of the deformation. In 1992, the 'Back to Sleep' campaign was instituted in the USA for prevention of sudden infant death syndrome (SIDS). Parents were encouraged to put infants to sleep in the supine position. Following this, a shift occurred in the location of the deformity of NSP, from frontal plagiocephaly being more frequently encountered before the 'supine' directive, to occipital plagiocephaly more commonly encountered now.

Etiologies

NSP results from a variety of extrinsic and intrinsic factors affecting the infant before, during and after birth. These various factors may be isolated or may exert a cumulative effect. Associated risk factors for NSP include premature birth,[34] firstborn,[31,35] prolonged labor, unusual birth position,[35] use of forceps and vacuum extraction.[36] Male gender is also a risk factor because fetal distress during labor, with a consequently higher incidence of operative delivery, is more common with males than with females.[37]

Before birth, NSP may result from abnormal constraint on the fetal head in the intrauterine environment, as with a unicornuate uterus, uterine fibroids or oligohydramnios.[9] Multiple births are another risk factor, with deformational plagiocephaly being frequently encountered in these children.

Extrauterine constraints placed on the fetus can be responsible for NSP. This may be the result of pressure from neighboring abdominal organs. With athletic mothers, increased tonus of the abdominal muscles can compress the uterus back against the spine. Lack of abdominal muscle strength, on the other hand, will produce an increase in the already increased lumbar lordosis that is very common in late pregnancy. The increased lordosis is associated with anatomic flexion of the sacrum that displaces the sacral promontory forward, with resultant external pressure on the uterus.

During the birth process, the uterus contracts regularly to allow the descent of the fetus through the birth canal. Prolonged periods of uterine contraction may increase the mechanical forces applied to the infant's head. When the head enters the pelvic cavity, anatomic extension between the sacrum and the pelvic bones normally occurs to

Figure 7.1.3. *Occipital non-synostotic plagiocephaly.*

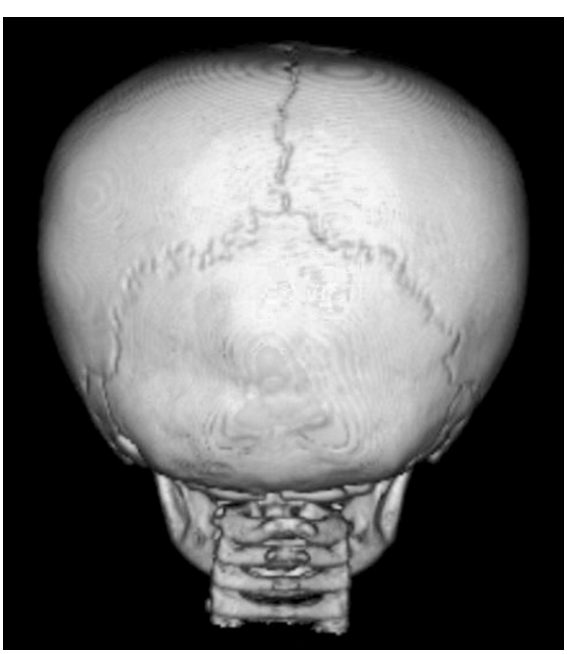

Figure 7.1.4. *Occipital non-synostotic plagiocephaly.*

increase the diameters of the pelvic outlet. Dysfunction of the maternal sacroiliac joints can result in a reduction of that increase and further constraint on the infant's head.

In the left occiput-anterior position – the most frequent birth presentation – the right side of the infant's occipital bone is in contact with the maternal pubic symphysis while the left frontal bone is compressed against the sacrum (Fig. 7.1.5). Asynclitism further increases the pressure of the infant's head against the pelvic bones with resulting occipital flattening on the right and frontal flattening on the left. The reverse – occipital flattening on the left and frontal flattening on the right – would result from the left occiput-posterior position. At the end of the descent, the head contacts the pelvic floor and turns in such a way as to position the occiput under the pubic symphysis. In the left occiput-anterior position, the right side of the occiput and the occipitomastoid area can be exposed to greater pressure. Later, during expulsion, compressive forces are applied on the occiput by the pubic symphysis.

After birth the pressure of the mattress on the infant's head is thought to contribute to occipital flattening.[38] Sleeping habits affect the cranial shape. When infants consistently sleep supine, as in Asiatic countries, posterior flattening of the skull occurs.[35]

Babies should be able to turn their head symmetrically to both sides. Asymmetric cervical rotation should be considered anomalous[39] and, if present, should be resolved by 12 weeks of age.[40] Although a preferred rotation of the head to the right side is present at birth in 59% of infants, this does not seem to be related to the fetal position. Often, infants may demonstrate asymmetric preferential motions, but a preferred motion should be differentiated from somatic dysfunction. Concern should arise when a rotational dysfunction is present. Early restriction of cervical motion is an identified risk factor for positional plagiocephaly,[41] particularly if the child is less active, staying in the same position to the point where an area of alopecia develops on the scalp. Upper thoracic and cervical restrictions, and SCM imbalance or torticollis, very often accompany plagiocephalies.[34,42,43] Furthermore, when the flattening has developed, it reinforces the preferential positional pattern with automatic positioning of the head on the flat area.

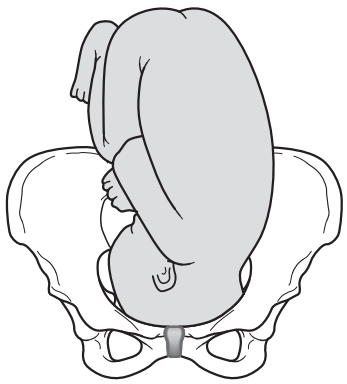

Figure 7.1.5. *Left occiput-anterior (LOA) presentation.*

Clinical aspect

By definition, plagiocephalic children have irregularly shaped heads. Since infants now sleep supine because of the 'Back to Sleep' campaign, the most commonly encountered forms of plagiocephaly are posterior, with either medial or lateral occipital deformity.

When compressive forces are applied to the squamous portion of the occiput, usually in the area of the lambda (superior angle of the occiput), the posterior portion of the head is flattened and assumes a brachycephalic shape. In severe cases, the flattening can demonstrate a slight depression, usually near the superior part of the occiput.

An asymmetric plagiocephaly results when dysfunctional rotation of the head is associated with the compressive forces. Because of the rotation, pressure from the weight of the infant's head on the mattress is asymmetric, and the occipitoparietal area on the side toward which the head is chronically rotated becomes flattened, while the other side develops excessively. This results in occipital flattening on one side and occipital bossing on the other. Anteriorly, the skull demonstrates frontal bossing on the same side as the occipital flattening, with frontal flattening on the opposite side. When the head is seen from above, it has a 'parallelogram' shape (Fig. 7.1.6).

The parallelogram cranial shape is commonly described in allopathic medical literature[28,33,44–47] as well as in osteopathic medical literature.[48–50] This pattern is also referred to as 'cranial obliquity'.[51] In the cranial concept, the parallelogram cranial shape is associated with a pattern of lateral strain in the sphenobasilar synchondrosis (SBS) (Fig. 4.10). Shearing between the posterior part of the body of the sphenoid, which is displaced laterally, and the anterior part of the occiput, displaced in the opposite direction, is lateral strain of the SBS and is found in children with NSP.[36] This results in the

parallelogram shape of the head, with the basilar portion of the occiput displaced away from the side of the posterior flattening.

The relationship between the cranial bones is disturbed, as described in lateral strain of the SBS, but this is not the only dysfunction associated with NSP. The compressive forces responsible for NSP can affect the relationship between different portions of the cranial bones, producing intraosseous dysfunctions. The occiput and temporal bones are the most common bones that demonstrate intraosseous dysfunction in the presence of NSP. At birth, the occipital bone is composed of four parts and each of the temporal bones of three parts. When occipital flattening occurs, the deformity is obvious in the squamous portion of the occiput where the compression has occurred; however, the compressive forces have also been directed against the non-visible parts of the occipital bone, i.e. the anterior intraoccipital synchondrosis that lies between the two parts of the condylar facets. When such a compression of the occipital condyles occurs, it most often occurs asymmetrically.

Figure 7.1.6. *Asymmetric posterior plagiocephaly.*

The hypoglossal canals are located bilaterally in the anterior intraoccipital synchondroses, between the basiocciput and the bilateral exocciputs. They contain the hypoglossal nerves that provide the motor supply to the tongue. Compressive force on one side can have an impact on tongue motor function, with resultant problems such as suckling difficulties.

Asymmetry of the cranial base can also affect the shape of the foramina therein, with potential for various entrapment neuropathies and vascular compressions. The jugular foramen, the right usually being larger than the left, contains the glossopharyngeal nerve (CN IX), the vagus nerve (CN X), the accessory nerve (CN XI) and the inferior petrosal and sigmoid venous sinuses. A broad spectrum of functions depends on these structures. Compression of CN IX is associated with altered sensation of the pharynx, fauces, palatine tonsil, pharyngotympanic tube (PT) and the posterior third of the tongue. Disturbances of CN X result in a wide range of symptoms including dysautonomia, colic and regurgitation. According to Magoun, 'the occipitomastoid suture and the jugular foramen should be considered as of significance with "pukey"

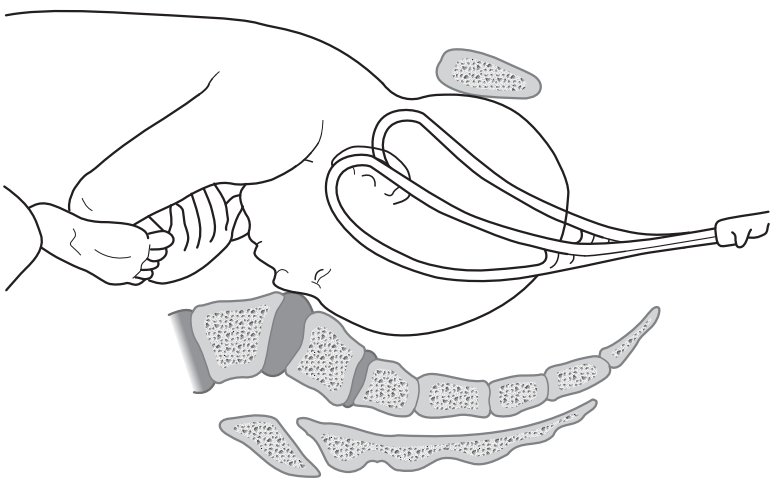

Figure 7.1.7. *Forceps delivery can be a risk factor for the development of asymmetric phlagiocephaly.*

babies'.[52] Compression of CN XI can compromise the motor supply to the upper and middle portions of the trapezius and to the SCM. The trapezius receives a portion of its motor supply from CN XI; the SCM depends mainly on CN XI. CN XI can also affect swallowing through its impact on the pharyngeal constrictor muscles that receive their nervous supply from the cranial portion of the accessory nerve.

With the resultant asymmetry in the cranial base, a difference in tension between the two SCM muscles is palpable. There are multiple myofascial attachments on the cranial base that can be similarly affected, resulting in a multiplicity of dysfunctional patterns in distant areas. Asymmetric tensions in the trapezius and semispinalis capitis muscles can cause thoracic spine dysfunction, asymmetries of the stylopharyngeus and stylohyoideus muscles can affect the pharynx, and the styloglossus muscle can dysfunctionally influence the tongue.

The compressive forces applied to the fetal head also affect the temporal bones. When occipital flattening occurs on one side, the ipsilateral temporal bone and attached ear are moved forward and the mastoid portion is compressed. This deformation can be demonstrated with computerized tomography of the skull base. The long axis of the petrous portion of the temporal bone is displaced in the direction of the coronal plane,[53] thus placing the temporal bone in a position resembling external rotation.

Embedded in the petrous portion of the temporal bone are the bony portion of the PT and the vestibular apparatus. Compressive forces affecting the temporal bone may increase the risk of otitis. The bony portion of the PT, the vertical portion of the tensor veli palatini and the mastoid air cell system have been found to be smaller than normal in children with secretory otitis.[54]

Temporal bone dysfunction may have further implications. Children with NSP present with a higher risk of auditory processing disorders, which is thought to lead to subtle problems of cerebral dysfunction later at school.[55] These problems include language disorders and learning disabilities, as well as attention deficits.[56] Plagiocephalic children also have a higher incidence of sleep disorders.[36]

NSP infants have been described as less active when sleeping on their backs than non-NSP infants, who move actively, turning their head and torso when developmental landmarks are achieved.[56] On the other hand, infants who sleep supine do not need to use their upper trunk and shoulder girdle muscles as much. Consequently, there is a delay in acquisition of early motor milestones.[57] Tripod sitting, creeping and crawling are particularly delayed, which contributes further to weakening of the core muscles. Scoliosis has been associated with plagiocephaly where sleeping supine was identified as a causative factor.[51,58,59] In The Netherlands, where the supine sleeping position has been encouraged since 1989, approximately 2.4% of 2–3-year-old children have cervical restriction of motion and/or plagiocephaly.[31] Interestingly, the habit of always bottle feeding a child on the same side seems to contribute to the pattern of asymmetric cervical rotation. Alternating right- and left-sided bottle feeding is consequently an imperative for every baby, particularly NSP infants.

The facial skeleton adapts to the cranial base asymmetry. Facial asymmetries and facial disharmonies are associated with NSP.[44,60,61] On the side of the occipital flattening, the maxillary bone develops less well, with less distance separating the nasion and the temporomandibular joint. This reduction is proportional to the amount of posterior occipital deformation.[62] The growth of the neurocranium and the viscerocranium occurs at different rates. Consequently, compensatory developmental disorders of the viscerocranium will appear later in childhood than the original NSP, and the association, therefore, is not commonly recognized.

Physical examination and treatment

It is a common observation that infants will sleep preferentially with their head turned toward one side, and that will be the side of the occipital flattening of the NSP. Daily activities such as difficulty nursing or accepting bottle feeding bilaterally will also reflect dysfunctions because of difficulty in turning the head to both sides. Associated disorders such as regurgitation, colic or sleep disorders are also often present. Otitis media can be part of the picture if the child is over 3 months of age.

While taking the history, the infant's appearance, posture and range of motion are studied. When observing the child, the osteopathic practitioner assesses what dysfunctional mechanics are involved in the present NSP. Where is the dysfunction? Is it only in the cervical spine or are other areas involved? What is the primary dysfunction? Three mechanisms are quite frequent: occipitocervical somatic dysfunction, thoracic somatic dysfunction and cranial somatic dysfunction:

1. Is there a pattern of restricted cervical spinal rotation that has obliged the child to lay their head on one side that explains the head flattening? If the head moves freely in all

directions, no cervical spinal dysfunction is present.
2. Is there a pattern of thoracic somatic dysfunction? Does the child rotate their head with a movement involving only one side of the pectoral girdle, indicative of thoracic dysfunction? In this case, asymmetry of the movements of the arms may also be present.
3. Is there a cranial dysfunction, such as a lateral strain of the SBS that explains the parallelogram shape of the head?

These questions should be kept in mind in the following examination.

Observe the relationship between the head and the pelvis. In the presence of a 'total body' dysfunctional pattern, the pelvis and the head of the child are rotated in opposite directions on the vertical axis of the spine. Palpation and motion testing will confirm these observations and treatment should be applied accordingly.

Evaluate the skull. Infants usually do not present with thick hair as encountered in most adults, making observation of the neurocranium easier. Look for any bald spots that indicate chronic contact between that area of the head and the bed during sleep. Look for posterior and anterior flattening; in the presence of posterior plagiocephaly these are usually on opposite sides. This results in a parallelogram-shaped head that is easier to see from above when the child is held on the practitioner's or parent's lap.

Observe the child's face and frontal bones. A pattern of compressive forces applied directly to the frontal bone, with no occipital deformity, would indicate a frontal dysfunction. Frontal flattening opposite to the occipital flattening is consistent with a pattern of lateral strain of the SBS. Symmetry of the frontal bone with occipital deformity on one side is an indication of synostotic posterior plagiocephaly, where the forehead may be symmetric or flattened on the side of the occipital flattening.[63]

When a frontal dysfunction is present, this may affect the eyes. Observe the size and shape of the orbital cavities, as well as the ocular bulbs. The orbital diameter is the distance between the superior medial and inferior lateral angles of the orbit. An increase in the orbital diameter results in an orbital cavity that appears to be wider than it is high and is associated with a cranial flexion–external rotation pattern. Conversely, if the orbital cavity appears to be narrower, this is associated with a cranial extension–internal rotation pattern. Frontal dysfunction, in turn, affects the rest of the facial bones and is particularly important when problem solving dysfunctions of the nasal bones and the maxillae.

Observe the positions of the ears. They are very indicative of the positions of the temporal bones. In cases of NSP, the ear located on the side of the occipital flattening is displaced more anteriorly than the contralateral ear. If the ear is displaced more posteriorly on the flattened side, further diagnostic investigation is warranted because the displacement might be a sign of synostotic plagiocephaly.[29] This sign, however, is not an exclusive indicator and synostotic plagiocephaly may also present with an anterior ear position.[28,63]

Observe the size and shape of the ears. Usually, on the side of the occipital flattening of NSP, the ear may, for example, have been compressed against the uterine wall and, therefore, may be smaller. In this instance, expect to find intraosseous dysfunctions of the temporal bone on that side. This is the ear that the child will usually rub and that eventually may present with an otitis media.

After observation, the osteopathic practitioner gently palpates (i.e. caresses) the child's head, looking for depressions, bossing and irregularities of contour. Sutures are palpated for ridges, overlapping or irregularities in shape. A thick ridge over a suture calls for attention, because it may be a sign of a synostotic suture. Flattenings are the result of compressive forces. Tissues are palpated to evaluate for tissue texture abnormality and increased tenderness. Palpation of osseous tissues gives a sense of density that might be different between the two sides of the suture; increased tenderness is usually proportional to the strength of the compressive forces. Posterior occipital muscles are evaluated by palpation for tension and asymmetry.

Although the vault and the back of the head, the parietals and the squamous portion of the occiput are quite accessible to palpation, palpation of the frontal and facial bones necessitates a little more patience and delicacy in order to avoid disturbing the child. The base of the skull is not directly accessible to palpation for structure. The lateral parts of the occiput or exocciputs and the basilar part, as well as the sphenoid bone and the petrous portions of the temporal bones, should be assessed by palpating for function. To visualize these areas correctly while palpating them, knowledge of anatomy is of paramount importance.

Motion testing will confirm the findings of palpation and observation. Dysfunctional head rotation is a treatment priority. It is of considerable importance

that the child leaves the office with a freer range of motion and an increased ability to turn their head. If not treated, the dysfunctional rotation will maintain the NSP. Look for any somatic dysfunction of the occipitocervical junction and the cervical and thoracic spine, and treat utilizing indirect principles. Structure will follow function, and if bilateral rotation is recovered, the traction of the muscles that insert on the occipital squama will help to reshape the flattened areas.

Membranous patterns of the cranial mechanism should be assessed and dysfunctions balanced. Particular attention should be given to the poles of attachment of the dura, the falx cerebri on the occipital squama and the tentorium cerebelli on the superior borders of the petrous portions of the temporal bones.

Motion of the SBS, occipitomastoid and lambdoid sutures should be assessed and treated accordingly. Check the frontal bones and their relationships with the facial bones. Any dysfunction should be treated.

Special attention should be directed at the cranial base, with the assessment and treatment of the compressed occipital condyles and compressed jugular foramina, when present. Molding procedures can be applied, with intraosseous balancing of the occiput, temporal bones, frontal bones, sphenoid and parietal bones as dictated by the patient's needs.

Advice to caregivers

Very often parents will comment on the fact that the flattening was not present at birth and say that they do not understand why NSP has developed. To succeed in the treatment of plagiocephaly, it is worthwhile explaining the mechanism of NSP and stressing the importance of parental participation in the following months. Explain that the initial asymmetry may have been present at birth in a very subtle way that has been exacerbated by a persistent positional preference. Explain that it is of paramount importance to encourage the child in activities that promote bilateral cervical rotation, as well as promoting sleeping positions that avoid pressure on the already flattened area. Successful treatment depends on this. Proper sleeping position may be obtained by elevating one side of the bed approximately 5 cm (2 inches). This can be accomplished by placing a rolled bath towel beneath the entire length of the mattress, on the side of the occipital flattening. This will encourage the infant to turn their head in the other direction. Toys and other attention-getting objects, soliciting the rotation of the child's head when lying supine, should be placed on the side opposite to the occipital flattening.

Encourage play in the prone position. It can be explained to the parents that activities in the prone position stimulate the posterior axial musculature. Muscular extension of the neck results in traction on the squamous portion of the occiput, helping to create a round head.

To stimulate the child's curiosity and open them to the world, while encouraging them to turn their head symmetrically left and right, carry the child facing forward, held in the midline of the parent's chest. One parental hand should support the child's bottom while the other hand contacts the front of the child's torso, holding them against the parent's chest. The prolonged use of a car seat or other similar such carrying device should be discouraged because it tends to maintain the child in a chronic position, usually that of the dysfunctional asymmetry.

Thumb sucking should be discouraged because the child assumes an asymmetric position while preferentially sucking one thumb. If oral gratification is necessary, an orthodontically shaped pacifier should be employed. Encourage the parents to gently caress the child's head bilaterally, behind the ears, in the areas over the occipitomastoid sutures and over the superior nuchal line of the occiput. This will help to reduce any dysfunctional tenderness.

SCOLIOSIS

Scoliosis is a lateral deviation of the spine. A structural scoliosis is a spinal deformation that is not totally reducible, while a functional scoliosis is totally reducible. The diagnosis of scoliosis is confirmed by radiographic analysis. A functional scoliosis is usually associated with pelvic or postural asymmetry, a difference in the length of the lower limbs or vestibular or visual disorders. A structural scoliosis presents a spinal deformation in the three planes of space involving sidebending, rotation and flexion or extension of the vertebrae. The side of convexity of the spinal curve defines the scoliosis – for example, a left scoliosis consists of a spinal curve that is convex on the left side.

Cobb's angle is a measurement of the degree of scoliosis between the most tilted vertebrae above and below the apex of the curve. This angle is obtained by measuring the angle of intersection between lines drawn perpendicular to the top of the most superior vertebra, and the bottom of the most inferior vertebra, of the curve.

A scoliosis is identified as idiopathic when no recognizable pathology explains its origin. It is identified as secondary in the presence of spinal anomaly

or neuromuscular dysfunction. The classification of scoliosis is based on the age of the patient when the spinal curvature is first identified: congenital scoliosis is present at birth, infantile scoliosis is diagnosed under the age of 3 years, juvenile idiopathic scoliosis between 3 and 10 years, and adolescent idiopathic scoliosis (AIS) between 10 years and the end of skeletal growth.

Etiologies of idiopathic scoliosis

The cause of idiopathic scoliosis has not been established, but seems to be a multifactorial interaction of environmental and genetic factors. Studies have shown scoliosis as a single-gene disorder that follows the simple patterns of Mendelian genetics.[64,65] Traits can be dominant or recessive. Genetic links exist, with 3% of parents and 3% of siblings having a scoliosis.[64] Further, a correlated incidence of scoliosis has been described in twins.[66] Older maternal age is a risk factor for greater progression of the curve of AIS.[58] Maternal age of 27 years or more at the time of the child's birth is associated with a higher incidence of AIS, whereas paternal age has no significant effect.[67]

Changes in the extracellular matrix of the connective tissues (e.g. collagen distribution and elastic fibers) have been found among patients with scoliosis, but most researchers do not consider these changes to be the etiology of the deformity.[68] Similarly, a change in muscle fiber composition or chronic muscle overuse might explained the hyperintense signal intensity shown on MRI in the multifidus muscle on the concave side of the scoliosis associated with greater degrees of curve severity.[69] Myopathy involving impaired calcium pump activity is among other proposed etiologies that require further study.

The progression of AIS is associated with increased calmodulin (calcium-binding receptor protein) levels in platelets, with a possible modulation of the calcium-activated calmodulin by melatonin.[68] The level of melatonin has been proposed as a predictor for progression of spinal curvature in idiopathic scoliosis.[70] Melatonin is produced and released by the pineal body at night, and its production is inhibited by environmental light. It is proposed that a deficiency of melatonin could disturb equilibrium and postural mechanisms.[71]

Neurologic origins have also been proposed as etiologies of scoliosis. Hearing-impaired children who have a high incidence of vestibular dysfunction have significantly less idiopathic scoliosis than children with normal hearing, resulting in the suggestion that idiopathic scoliosis has a neural etiology.[72] A sensory input deficiency of the spatial orientation system, involving visual and vestibular dysfunction, is believed to cause motor cortex and axial posture control disturbances.[73,74] Another hypothesis is that developmental disorders in the central nervous system, followed by asymmetry of the spinal rotators and other trunk muscles, results in AIS. A less efficient postural regulation system with a diminished quality of standing stability has been demonstrated in some scoliotic patients.[75]

Developmental disorders have also been considered at the level of the spinal cord. Differences between the growth of the vertebral column and the growth of the spinal cord – 'uncoupled neuro-osseous growth' – either with asymmetric nerve root tension or with reduced growth of the cord, are suggested etiologies for scoliosis.[76] More rapid growth of vertebral bodies that occurs through endochondral ossification, as well as slower circumferential growth of the vertebral bodies and pedicles that occurs through membranous ossification, is observed in AIS patients.[77] Furthermore, the length of the vertebral canal is shorter than the length of the vertebral column. This leads to the hypothesis that the thoracic spine is tethered by a tight spinal cord, with a resultant diminution of the kyphosis. This, in turn, results in a displacement of the spinal cord to one side of the vertebral canal with an associated sidebending of the spine and lastly a rotation of the vertebral bodies to allow for their growth while the vertebral canal stays in the midline.[78] The base of the vertebral canal remains at a right angle to the sagittal plane of the patient, in its original position, and does not follow the rotation of the vertebral body.[78] The tension of the core link – the strong dural membranes covering the cord, between the pelvic and cranial bowls[79] – may play a role in this mechanism. The relationships between the different parts of the craniosacral mechanism, therefore, should be considered, and any dysfunction of the dura and/or the vertebral ligaments should be treated.

Biomechanical factors can affect spinal alignment. Sidebending–rotation and torsion of the SBS, for example, can modify the level position of the orbits through the sphenoid and thereby modify the occipital neutral position, again altering balance mechanics in the spine below.

Pelvic obliquity is considered a cause of imbalance in the axial skeleton. Pelvic obliquity has been associated with unequal leg length. If this imbalance appears early in the growth process, it will result in abnormal asymmetric weight-bearing pressures on

the vertebrae. A 'dangerous triad' of joint laxity, delayed growth and persistence of asymmetric overloading of the spine has been described in rhythmic gymnasts who develop scolioses.[80] This group shows an incidence of scoliosis of 12%, compared to 1.1% in the general population of the same age group. Pelvic obliquity has also been associated with the 'molded baby syndrome', where intrauterine molding determines a vertebral curve, having a sacral tilt inferiorly on the side of the spinal convexity. Mechanical compressive forces acting on the infant during the prenatal, perinatal and postnatal periods have been proposed as an etiology for scoliosis. More breech presentations have been found among infants developing scoliosis during the first 6 months of life.[58] Plagiocephaly is frequently associated with infantile scoliosis.[58,81–84] A facial and cranial distortion, always on the side of the curve of the scoliosis and linked to the intrauterine position, has been described.[85]

McMaster noted that the scoliosis was rarely present at birth and, like plagiocephaly, develops very often within the first months of life. He proposed an explanation for the association between plagiocephaly and scoliosis, as follows. Infants prefer to turn toward their right side when in a supine position. Light but asymmetric pressures from the mattress on the skull as well as on the growing spine can create asymmetries, particularly when applied over a prolonged time or during a critical period of growth. With chronic right rotation of the head, the back of the head will flatten on the right, allowing growth of the skull on the left. The thorax follows the same pattern, the left side expanding freely backwards with a left rotation of the thoracic vertebrae.[83]

Infantile scoliosis is sometimes found in association with imbalance of the occiput or with dysfunction of the SBS. Intraosseous dysfunction of the occiput can produce asymmetry of the occipital condylar parts, resulting in altered balance mechanics in the spine below. The compressive forces applied to the occipital bone at the time of delivery have been described and suggested to be the causes for future compensatory scoliotic curves.[59,86] Thus, the child may prefer to sidebend their head slightly to one side and, as time goes on, with potential new injuries and therefore more difficulties for compensation, the adolescent will develop a scoliosis. Ventura et al. state: 'Even small deformities present at, or soon after, birth may get worse in infants whose connective tissues do not have a potential for recovery.'[84] To this we could add one or more of the components described above – the etiology is multifactorial.

Congenital and infantile scoliosis

Congenital scoliosis may be associated with neurologic pathology or a vertebral structural defect (failure of formation and/or segmentation), as well as associated abnormalities of the head, neck, pelvis and hips. A thorough medical examination should be performed to rule out these disorders. Hemivertebra is the most common anomaly that causes non-idiopathic congenital scoliosis[87] and is sometimes associated with posterior midline cutaneous abnormalities. Neural axis abnormalities have been demonstrated in 21.7% of otherwise asymptomatic patients with infantile scoliosis.[88] Congenital scoliosis is more common in females than in males, occurring in the ratio of 1.27:1.[87] Among children with congenital spinal anomalies, 30–60% have other anomalies located most commonly in the genitourinary tract, cardiovascular system, spinal cord or cervical spine.[89]

Since the time of Hippocrates, congenital and infantile scolioses have been described as potentially the result of mechanical factors operating during fetal life.[9] These scolioses are more common in males, presenting more often as a left thoracic curve.[83,85] Some resolve spontaneously, while others progress. Harrenstein in 1930,[90] quoted by Mehta,[91] stated: 'Spontaneous correction does occur without treatment but at the moment it is not possible to distinguish between the two at the time of the diagnosis.' Thus, in order to differentiate between progressive and non-progressive congenital and infantile idiopathic scoliosis, Mehta proposed a method of measurement of the rib–vertebra angle (RVA).[91] In an anteroposterior radiograph, the RVA is 'the angle formed between each side of the apical thoracic vertebra and its corresponding rib'. The RVAs are equal on a normal spine and a gap of 2–4 mm normally separates the head of the rib and the upper corner of the adjacent vertebra. Mehta states that the most progressive infantile scoliosis presents an RVA difference between the two sides equal to or greater than 20°. When this radiographic measurement is repeated after 3 months, the RVA remains unchanged or increases with a progressive scoliosis, whereas it decreases with a resolving curve. Furthermore, with time, in the anteroposterior radiograph, a progressive scoliosis demonstrates overlap of the rib shadow with the

upper corner of the vertebra. The relationship between the rib and the vertebra and the tissue response to any stress during growth periods is of great importance.

Curve progression is the major concern in every type of scoliosis, and the differentiation between progressive and non-progressive congenital scoliosis and infantile idiopathic scoliosis has been confirmed through sequential comparisons of RVAs over time.[84,92] When spontaneous resolution occurs in non-progressive infantile scoliosis, it does so between the ages of 12 and 18 months.[84] Spontaneous resolution of infantile idiopathic scoliosis varies between 17%[93] and 92%.[94] Therefore, it is appropriate to detect the scoliosis at its earliest stage and to treat all affected babies within the first 6 months of life.[95] It is typical to employ a wait-and-watch approach to the progressive category because orthopedic treatment is complex and difficult to initiate before the age of 18 months.[96] Osteopathic procedures, on another hand, may be begun immediately, with potentially good results being obtained in cases of congenital and infantile idiopathic scoliosis.

Physical examination and treatment

Observe the baby for spontaneous positioning and areas of restricted mobility. Look for positional asymmetries of the torso, head and neck, arms and legs. Look also for a bulky back on the convex side of the thorax and creases in the skin laterally on the concave side. Skin creases are a sign of fixed scoliosis.[97] Observe the baby for clumsy movement in the maintenance of head position and in general coordination.

Palpate the infant to identify membranous, myofascial and interosseous somatic dysfunction contributing to the visually observed functional restrictions. The infant may be treated utilizing indirect principles to release any restriction of motion, particularly in the pelvis, upper thoracic spine, ribs, sternum, thoracic diaphragm, pectoral girdle, cervico-occipital area and cranium. Intraosseous dysfunctions – most commonly encountered in the sacrum, lumbar and thoracic vertebrae and occiput – may be addressed using molding procedures.

At home, following treatment, it is important to avoid movements and positions that will reinforce the scoliotic pattern. In considering daily activities, such as feeding and play, and when putting the infant to sleep, encourage the parents to position the infant correctly and to solicit movement from the infant that promotes symmetry. A child should sleep on their back to prevent SIDS, but should play in the prone position to develop the vertebral musculature of tonic posture.

Prevention is the best therapy. Osteopathy, as a non-invasive treatment, facilitates the spontaneous recovery process or regression as quickly as possible. This allows the child to progress through the developmental milestones of infancy without interference from a dysfunctional musculoskeletal system.

Juvenile and adolescent idiopathic scoliosis (AIS)

Scoliosis is present between 10 and 16 years of age in 2–4% of children.[98] These patients differ from those with infantile scoliosis in that there is predominance among females and for the curvature to be a right thoracic curve.[83,85] The vertebral rotation is associated with a 'rib hump' on the side of the convexity, with most curves convex on the right in the thoracic area and on the left in the lumbar area. Cobb's angle is sometimes debated as a true assessment of the scoliosis. AIS is a three-dimensional deformity of the spine, with morphologic changes in the trunk and rib cage. Vertebral rotation should be considered in the evaluation. The presence of severe pain or neurologic symptoms would be atypical for idiopathic scoliosis and should raise concern for spinal cord pathology.

Idiopathic scoliosis, if left untreated, increases in adult life. The period of puberty should be considered as a high-risk interval, and regular screening is recommended. A significant correlation between growth in height and progression of Cobb's angle has been found, with a possible increase until 2.5 years after menarche.[99] Current studies have indicated that the younger the patient at the time of diagnosis by pubertal or skeletal maturation landmarks, the greater the chance of curve progression. A curve measuring less than 30° at skeletal maturity is least likely to progress, whereas curves measuring 30–50° may gain another 10–15°.[65] Thoracic curves deteriorate most, followed by thoracolumbar curves and double curves.[100]

Less than 25% of adolescent idiopathic scoliosis resolves without treatment.[101] Orthopedic treatment with a cast or a brace might be indicated to limit the progression of the curve and to employ surgery to correct the scoliosis. Bracing for at least 23 hours per day appears to be optimal for interdicting

progression of the curve.[102] Watchful waiting is suggested as an alternative treatment to bracing because bracing does not decrease the incidence of surgery and often results in adverse psychological effects.[103]

Manipulative treatment associated with exercise has been shown to stop curve progression in AIS.[104,105] Exercise may be used to effectively reverse the signs and symptoms of scolioses and to prevent progression of spinal curves in children and adults.[106] As scoliosis is a risk factor for the impairment of physical wellbeing and quality of life, treatment is important.

Physical examination and treatment

The physical examination begins with observation of the patient standing. Observe placement of the feet for asymmetries. From behind, note the level of the iliac crests and the symmetry of the pelvis and waist triangles. Leg length discrepancy as a contributing factor should be considered. Further, observe the relationship between the pelvic and pectoral girdles, and look for shoulder and scapular asymmetries. Elevation of the right shoulder compared to the left is associated with a thoracic curve convex on the right. Note the position of the patient's head. A vertical line dropped from the external occipital protuberance should fall in the middle of the intergluteal crease. The patient is next observed in profile for increased or decreased thoracic kyphosis, forward head posture and lumbar lordosis.

The Adam's forward bending test is employed to help identify scoliosis. The child bends forward, holding palms together with arms extended. The examiner looks from behind and from the side, along the horizontal plane of the back, to detect an asymmetry as a rotational deformity or 'rib hump' (Fig. 7.1.8). This deformity is associated with spinal curves and may be further delineated with radiographic evaluation. With the child remaining forward bent, observe the distance between tips of the fingers and the ground as an indication of general spinal flexibility.

The palpatory examination of the patient with AIS is directed at the identification of membranous, myofascial and interosseous somatic dysfunction. The patient may be treated utilizing indirect principles to address any identified dysfunction. Begin by treating the area of greatest motion restriction. This will often be in the upper thoracic and craniocervical regions. The presence of proprioceptive sensory endings in the ligaments and fascia of the upper cervical area contributes to postural balance. Primary high cervical somatic dysfunction can consequently impact postural balance. Somatic dysfunction in the upper thoracic region can result in compensatory dysfunction in the upper cervical spine. Once these areas have been treated, dysfunction of the thoracic spine, ribs, lumbar spine, sacrum and pelvis should be addressed.

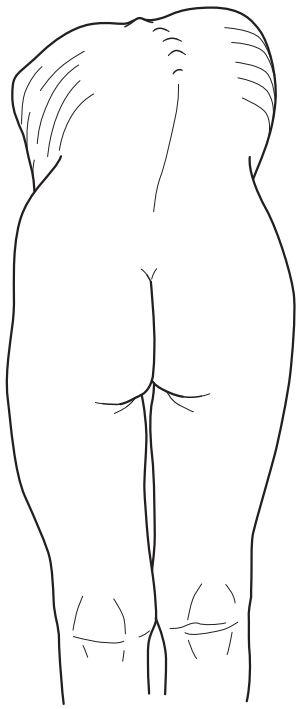

Figure 7.1.8. *The Adam's forward bending test allows the examiner to detect an asymmetry as a rotational deformity or 'rib hump'.*

Normalization of the diaphragm, to increase vital capacity, should always be part of the treatment of scolioses. Dysfunction of the thoracic diaphragm, because of the attachments of the crura on the lumbar spine, affects the mobility of the spine and may be linked to dysfunction in the pelvic and cranial diaphragms through the core link.

Any dysfunctional pattern present in the skull should be treated. Dysfunction in the occiput can affect the proprioceptive input from the craniocervical region. The vestibular apparatus located inside the petrous portions of the temporal bones contributes to balance mechanics and symmetric muscle tension. Dysfunction between the sphenoid, occiput and temporal bones should, therefore, be treated. By virtue of the fact that fusion of the SBS does not typically occur before late adolescence, there remains mobility in the base of the skull of younger AIS

patients, making them particularly receptive to cranial treatment.

Cranial dysfunction may also affect the scoliotic patient through the reciprocal tension membranes. Dysfunctional tensions in the dura have been proposed as an etiology of scoliosis.[76] Equilibration of intracranial and intraspinal membranes should, therefore, be employed. In younger patients where active bone growth is still present, intraosseous dysfunctions – most commonly encountered in the sacrum, lumbar and thoracic vertebrae and occiput – may be addressed using molding procedures.

Exercises are an important component of the treatment protocol. A properly employed exercise program should teach the patient to breathe effectively, increasing vital capacity and enhancing thoracic cage mobility. It should facilitate the establishment of the fullest range of motion possible and the development of symmetry of movement, particularly spinal rotation, while strengthening the core musculature and stabilizing the spine. It should allow the patient to develop proprioception and establish good postural habits.

KYPHOSIS–LORDOSIS

Kyphosis is an increase of the spinal curve in the sagittal plane that results in a greater than normal posterior convexity (anterior concavity). An increased kyphotic curve is encountered more often in the thoracic spine, where it produces a rounded upper back, or 'humped back'. In the cervical and lumbar spines, normal curves present with posterior concavity or lordosis. Under dysfunctional circumstances, the lumbar and cervical curves are reversed and become kyphotic. At the lower portion of the spine, a kyphosis can be ascribed to the sacrum, when its usual posterior convexity is increased.

A thoracic kyphosis of >45° is considered pathologic. Congenital kyphotic deformities are infrequent and can be caused by a failure of formation of the vertebral body or a failure of segmentation, for which the treatment is surgical.[107] In the infant, tumor of the spine is also a potential cause of kyphosis that requires specific medical attention.

In the juvenile period, Scheuermann's disease is a cause of kyphosis resulting from an alteration of the vertebral development. Wedging of the vertebral bodies, the posterior height being greater than the anterior height, produces the kyphotic deformity. Boys are more frequently affected and the resultant back pain might be the trigger for an X-ray where the diagnosis is made. Irregularities in the endplates of the vertebrae can be observed, particularly at the level of the lower thoracic and upper lumbar spine. Scheuermann's disease may also be associated with scoliosis.[108]

Because kyphosis is a spinal deformity, it should not be confused with poor posture. When examining an infant who is seated without support, it is normal to find a kyphosis of the thoracic and lumbar spine. Proprioception and muscular tone develop with age to maintain adequate sagittal balance. Sagittal spinal curves change as a child grows.[109,110] The thoracic kyphosis is more pronounced in males,[111] at a mean age of 12.8 years.[112]

More often, the exaggerated kyphotic curve is associated with dysfunctional posture. Juvenile and adolescent kyphosis can be the result of poor posture, as a compensatory pattern to an extension dysfunction elsewhere. Anterior displacement of the occipital bone on the superior articular surface of the atlas will project the chin forward, and the ensuing postural compensation will result in an increased thoracic kyphosis. This pattern is commonly found in individuals who demonstrate oral breathing.

When encountered in a child, an apparent kyphosis may also be the result of protraction of the pectoral girdle. In this case, the thoracic curve is not fixed in the kyphotic position and spinal backward bending can be achieved on demand, although lack of flexibility is common. The child is usually shy, and an extension–internal rotation pattern may be present, either at the level of the pelvis or at the level of the SBS, the temporal or occipital bones. A thoracoabdominal diaphragmatic dysfunction is very often associated with diminished thoracic flexibility and reduced vital capacity. The areas of the diaphragmatic attachments onto the inferior portion of the sternum and adjacent ribs might be the causative dysfunctional agents.

An increased thoracic kyphotic curve is usually compensated for by an increase in the lumbar lordosis. Kyphosis and lumbar lordosis generally compensate each other. A correlation between these two spinal curves has been found in most age groups.[113]

The cervical and lumbar regions are normally lordotic. Hyperlordosis is an increase of the lumbar lordosis and is considered pathologic. It can be associated with other conditions, such as developmental dysplasia of the hip or neuromuscular disorders. There may be a family history of hyperlordosis, but it can also follow trauma, commonly from

athletic activities, particularly highly competitive sports, during periods of growth. Adolescents may also present with hyperlordosis as a consequence of a developmental spondylolisthesis. With this disorder, studies have shown an increase of hyperlordosis and sacral inclination, but a decrease of thoracic kyphosis.[114]

The degree of lumbar lordosis is correlated with sacral position. Sacral anatomic extension (craniosacral flexion) is normally associated with decreased lordosis; sacral anatomic flexion (craniosacral extension) is associated with increased lordosis. The global amplitude of the vertebral curves, cervical lordosis, thoracic kyphosis and lumbar lordosis changes with growth, but the association with the position of the sacrum is constant under normal conditions.

The relationship between the cranial and the pelvic bowls, through the core link, is a fundamental principle within the cranial concept. Cranial flexion is associated with sacral craniosacral flexion, and cranial extension is associated with sacral craniosacral extension. The vertebral anteroposterior curves are decreased when flexion of the cranial base is present; conversely, there is an increase of the lordotic or kyphotic curves with extension of the cranial base.[115]

Physical examination and treatment

Early detection of kyphotic and lordotic curves is important for successful treatment. The child should be considered from a total body approach and the posture of the whole body should be evaluated in the standing position:

- Observe the pattern of weight-bearing mechanics.
- Observe the feet for a pattern of inversion or eversion. A pattern toward eversion of the feet, and eventually flat feet, is consistent with increased sagittal curves and cranial extension–internal rotation.
- Observe the knees. Genu valgum is consistent with increased sagittal curves and cranial extension–internal rotation.
- Observe the pelvis for an increase of anterior tilt, with the sacrum in craniosacral extension.
- Observe for pelvic asymmetry and any difference in the greater trochanter and innominate crest heights.
- Observe the spine for an increase of the anterior–posterior curves. A pattern involving all of the curves might be the consequence of a cranial or sacral dysfunction with extension and internal rotation. A pattern of increased curvature limited to a portion of the spine may be associated with a dysfunction within the curve, in an adjacent spinal curve or adjacent junctions between the spinal AP curves.
- Observe the pectoral girdle for protraction or an associated asymmetric pattern. A difference in shoulder heights is common. This suggests somatic dysfunction in the thoracic spine with associated sidebending and rotational components.
- Observe the position of the head in relation of the rest of the body, in both the frontal and the sagittal plane. Forward displacement of the head is often associated with somatic dysfunction of the craniocervical or upper thoracic vertebrae.

Next, observe the child while they are moving. If necessary, have the child demonstrate active flexion–extension, sidebending and rotation of the spine to confirm previous observations. Muscles may show a difference in tension between anterior and posterior groups. Hyperlordotic children will present with increased tension in the hamstrings and hip flexors, while at the same time their abdominal muscles will lack tension.

Tests of listening are performed on the innominates, the sacrum, the lumbar and cervical vertebrae and the cranium. The treatment of any identified dysfunctional areas should follow using indirect principles. The postural response to effective manipulation is almost immediate. You should be able to see improvement in the posture of the child after the first treatment.

Simple exercises may be recommended, particularly if poor posture is present. Pelvic tilt is useful, as are stretching the hamstrings and proprioceptive exercises to increase body posture awareness. The child should be encouraged to judiciously practice athletic activities, such as swimming and tai chi, which will strengthen and balance the core muscles and improve flexibility and coordination.

Advice should be given that appropriately addresses daily living conditions. For example, the patient should avoid carrying a backpack on one shoulder; rather, they should carry it using both shoulders. They should avoid reading and writing on a flat horizontal surface and should work instead on a surface that is tilted approximately 20° to limit cervicothoracic flexion.

Once the problem has been effectively addressed, the child may then be treated as needed, but they should be followed on a regular basis, at least annually, until they have stopped growing.

PECTUS EXCAVATUM AND CARINATUM

Pectus excavatum is a deformity of the anterior thoracic cage in which the sternum is depressed in a concave shape, whereas in pectus carinatum the sternum is protruded in a convex shape. These deformities may or may not be associated with genetic disorders or with scolioses. Decreased thoracic cage compliance and reduced vital capacity may be present, although the heart and lungs develop normally. Pectus excavatum and pectus carinatum are present at birth, but the parents usually do not become aware of the deformity until it becomes more apparent with growth. Severe cases often result in significant psychological impact, usually in early adolescence.

Pectus excavatum is frequently associated with an SBS extension pattern. This results in internal rotation of the paired structures, specifically the pectoral girdle. These individuals may also demonstrate direct mechanical derangement of the internal fascial structure of the thoracic cage and intraosseous dysfunction of the ribs and sternum. A diaphragmatic dysfunction is almost always associated with this condition. In pectus carinatum, similar mechanisms exist but with a tendency for SBS flexion.

Physical examination and treatment

The evaluation of the patient commences by examining the interrelationship between the sternum and the thoracic spine, and between the sternum and the occiput. Next, examine all myofascial structures attached to the sternum, including the pectoral girdle and the diaphragm. The anterior abdominal wall should be evaluated for dysfunctional tension, and, if present, contributory mechanics should be sought out in the lumbar spine, sacrum and pelvis. Visceral abdominal dysfunction should also be considered.

Treatment consists of myofascial release applied to identified dysfunctions. If possible, molding procedures directed at the sternum should be employed simultaneously with the myofascial release modalities to enhance the efficacy of both. The application of these procedures should be done in synchrony with, and with the intent to enhance, the inherent motility of the body. The younger the patient when treatment is initiated, the greater the potential for positive outcome.

VERTEBRAL SOMATIC DYSFUNCTION

Vertebral somatic dysfunction in infants and children can be found at any level of the spine. It will, however, be more commonly encountered in the lumbar, upper thoracic and cervical regions. It usually results from the day-to-day physical activities and traumas of childhood. In younger children, dysfunction in the cervical region will often present as cervical pain and, eventually, as torticollis. In the lumbar region, somatic dysfunction may remain quiescent for a protracted period, in time manifesting through a somatovisceral mechanism as abdominal pain. In older children and adolescents, the initial complaint from vertebral dysfunction is usually localized or referred musculoskeletal pain. Because of the young patient's ability to compensate for somatic dysfunction, any vertebral somatic dysfunction should be thoroughly evaluated to rule out a viscerosomatic origin.

The mechanics of vertebral somatic dysfunction manifest in children and adolescents is the same as that encountered in adults, showing the coupled relationships between flexion–extension, sidebending and rotation as described by Fryette.[116] However, because of the flexibility of the soft tissues in these young patients, dysfunctional barriers are more compliant, lending to the application of indirect techniques in their treatment.

Somatic dysfunction may also exist as a reflex manifestation of visceral dysfunction and disease. Although the precise locations of viscerosomatic reflexes in infants and young children have not been specifically reported, it is reasonable to anticipate locations similar to those in adults. The facilitated state of the segmental spinal cord in the presence of visceral input can, in turn, result in a somatovisceral response. A listing of viscerosomatic locations as they have been reported in the osteopathic literature is summarized in Box 7.1.1.

Because of the growth potential of these patients, vertebral somatic dysfunction can exert disproportionate impact on their developing posture as well as on the viscera through somatovisceral reflexes.

Box 7.1.1

VISCEROSOMATIC REFLEXES

The following locations are summarized from a review of the osteopathic literature.[117–123]

- *Eyes, ears, nose, and throat*: The sympathetic reflex is T1–T5. The trigeminal nerve is the final common pathway for both sympathetic and parasympathetic innervation of the upper respiratory tract. The muscles of mastication, commonly the temporalis muscles, receive motor innervation from the trigeminal nerve and serve as the somatic component for the upper respiratory tract sympathetic and parasympathetic reflexes. An additional reflex site is occiput–C2. This results from a reflex between the trigeminal nerve and upper cervical nerves.[123]
- *Heart*: The sympathetic reflex is T1–T5, left-sided greater than right. The parasympathetic reflex is vagal, occiput, C1, C2.
- *Lung*: The sympathetic reflex is bilateral from T1 to T4. Conditions involving both lungs result in bilateral reflex findings. Conditions involving one lung result in a reflex on the same side as the involved lung. The parasympathetic reflex is vagal, occiput, C1, C2.
- *Gastrointestinal tract*:
  - The parasympathetic reflex from the gastrointestinal tract proximal to the mid-transverse colon is vagal, occiput, C1, C2; the parasympathetic reflex from the distal half of the transverse colon to the rectum is sacropelvic S2–S4
  - The esophagus has a right-sided sympathetic reflex from T3 to T6
  - The stomach has a left-sided sympathetic reflex from T5 to T10
  - The duodenum has a right-sided sympathetic reflex from T6 to T8
  - The small intestine sympathetic reflex is bilateral from T8 to T10
  - The appendix and cecum sympathetic reflex is from T9 to T12 on the right
  - The ascending colon sympathetic reflex is from T11 to L1 on the right
  - The descending colon to rectum sympathetic reflex is from L1 to L3 on the left.
- *Pancreas*: The sympathetic reflex may be left-sided or bilateral and is T5–T9. The parasympathetic reflex is vagal, occiput, C1, C2.
- *Liver and gallbladder*: The sympathetic reflex is right-sided from T5 to T10. The parasympathetic reflex is vagal, occiput, C1, C2.
- *Spleen*: The sympathetic reflex is left-sided from T7 to T9.
- *Kidney*: The sympathetic reflex is on the same side as the involved kidney, from T9 to L1. The parasympathetic reflex is vagal, occiput, C1, C2.
- *Urinary bladder*: The sympathetic reflex is bilateral from T11 to L3. The parasympathetic reflex is sacropelvic, S2–S4.
- *Ovaries (and testes)*: The sympathetic reflex is on the same side as the involved organ from T10 to T11.
- *Adrenal glands*: The sympathetic reflex is on the same side as the involved gland from T8 to T10.

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7.2 APPENDICULAR IMBALANCE

Upper extremity

FRACTURES OF THE CLAVICLE

The clavicle is the bone most frequently fractured during the birth process. Clavicular fractures occur in about 1.6% of all vaginal deliveries[1,2] and 0.5% when considering all live births.[3] Both males and females are affected equally, with equal left versus right-sided incidence.[3] There is, however, a slightly increased incidence of right-sided fractures in left occiput-anterior deliveries.[4]

Reported risk factors for clavicular fractures include increased duration of the second stage of labor, increased birth weight and neonatal length (macrosomia), instrumental delivery[2] and shoulder dystocia.[3] In cephalic presentation, the compression of the infant's anterior shoulder against the maternal symphysis is responsible for the trauma.[4] Direct pressure or torsion applied to the clavicles to facilitate delivery can also result in fracture.

Complete or incomplete greenstick fractures are most frequent. They present with edema, crepitus, a palpable bony bump and tissue texture changes over the fracture site. Decreased or absent movement of the affected arm is present, as may become apparent when eliciting the Moro reflex. Asymptomatic or incomplete clavicular fractures may not be initially identified until after discharge from the hospital. Usually, the caregiver will notice that the child demonstrates irritability with discomfort and pain when putting the child's arm through the sleeve of a garment or when lifting the child by holding them under their arms. They are also liable to report that the child cries when positioned on the affected side.

Shoulder dislocation, humeral fracture and brachial plexus injury are part of the differential diagnoses. The diagnosis is confirmed radiographically. Associated complications, such as Erb's palsy, are present in 11.3% of newborns with fractures of the clavicle,[2] although clavicular fracture may actually reduce the potential nerve injury from traction on the brachial plexus.[4]

Usually no orthopedic treatment is necessary for asymptomatic and incomplete clavicular fractures. When the neonate presents with pain or discomfort, the affected arm may be immobilized by pinning the sleeve to the front of the shirt for 7–10 days.[5] A large callus typically forms at the fracture site within a week, and recovery is usually considered to be complete. Osteopathic procedures may be employed to assist the recovery process and address the dysfunctions usually associated with a fracture of the clavicle.

The clavicle is of importance because of its myofascial attachments. It serves as a junction between the fasciae of the thorax, arm and neck. The investing layer of the deep cervical fascia completely surrounds the neck. Superiorly, it is attached to the external occipital protuberance and the superior nuchal line, the mastoid processes, zygomatic arches and the inferior borders of mandible. It splits to surround the trapezius and sternocleidomastoid muscles. Inferiorly, it is attached to the manubrium of the sternum, the acromion and the spine of the scapula.

The clavicle is the link in the fascial continuity between the investing layer of the deep cervical fascia and the fascia of the thorax and the arm. The clavipectoral fascia attaches on the clavicle, as does the deltoid fascia. The deltoid fascia is in continuity with the brachial fascia. The clavicle, therefore, plays an important role in the equilibrium of the fascia of the thorax, arm and neck, and should be balanced, as should the myofascial structures attached to it.

Forces applied during the delivery that are great enough to fracture the clavicle also affect the neck and upper thoracic vertebrae of the neonate. Therefore the osteopathic practitioner should evaluate these structures and treat any dysfunctions accordingly, using indirect principles.

Furthermore, because of pain from the fractured clavicle, the child will prefer to lie on the opposite side, thereby fostering the development of asymmetric fascial tensions. These asymmetries can, in turn, induce the child to select a chronic position of comfort, long after the clavicle has healed. The chronic asymmetric positioning can then predispose the child to the development of plagiocephaly.

Fractures of the clavicle that occur during childhood are usually the result of rough play or athletic activities. In childhood, the forces that result in clavicular fractures are usually violent, most often involving impact on the hand with the arm extended or impact on the shoulder. In 75% of cases the site of fracture involves the medial third of the bone. The standard orthopedic treatment reduces the displacement at the fracture site by maintaining the shoulder in an upward and backward position with a bandage or plaster.

Physical examination and treatment

Osteopathic procedures applied to the older child follow the same anatomic principles as for the infant, i.e. to alleviate myofascial imbalances and upper thoracic and cervical dysfunction. The acromioclavicular junction may demonstrate somatic dysfunction; it should, therefore, be evaluated and treated following indirect principles. It is of importance to allow for normal function of the growing upper extremity. Acromioclavicular dysfunction is the source of many adult shoulder disorders.

BRACHIAL PLEXUS INJURY

A brachial plexus injury occurs most commonly as a result of a difficult birth, fetal malpresentation,[5] shoulder dystocia, macrosomia[6] or assisted vaginal delivery.[7] Fracture of the clavicle(s) or humerus,[8] shoulder dislocation, torticollis, hematomas of the sternocleidomastoid muscle or paralysis of the diaphragm may be associated with injury of the brachial plexus.[5]

A commonly believed etiology of brachial plexus injury is excessive traction on the fetal head during birth. In vaginal delivery, during the attempt to deliver the anterior shoulder, the applied downward traction can damage the brachial plexus. This theory, however, is questionable because, in almost half the cases of brachial plexus injury, delivery of the shoulders occurs without difficulty. Therefore, an in utero, atraumatic theory is also proposed.[8] When asymmetry and diminished movement of the arm are observed on the fetal ultrasound, a vulnerable plexus may be injured without traction during delivery.[9]

The brachial plexus is formed by the union of the anterior divisions of the lower four cervical nerves and part of the anterior division of the first thoracic nerve. In addition, the fifth cervical nerve frequently receives a branch from the fourth cervical, and the first thoracic a branch from the second thoracic. The plexus extends from the inferior aspect of the side of the neck to the axilla. The fifth and sixth cervical nerves unite to form the upper trunk; the eighth cervical and first thoracic nerves form the lower trunk, while the seventh cervical nerve runs out alone as the middle trunk. These three trunks pass beneath the clavicle and split into the anterior and posterior divisions. The plexus is attached to the first rib and to the coracoid process by the costocoracoid membrane and is subject to any force that disturbs the relationship between the cervical vertebrae, the first thoracic vertebra and ribs, the clavicle and the scapula (Fig. 7.2.1).

Most of the time brachial plexus injury is unilateral and immediately recognizable. Brachial plexus injury can affect different spinal nerve roots and is identified as follows:

- *Upper type*, with involvement of C5 and C6, or Erb–Duchenne palsy that affects muscles of the shoulder and elbow. The child presents with adduction of the upper extremity and internal

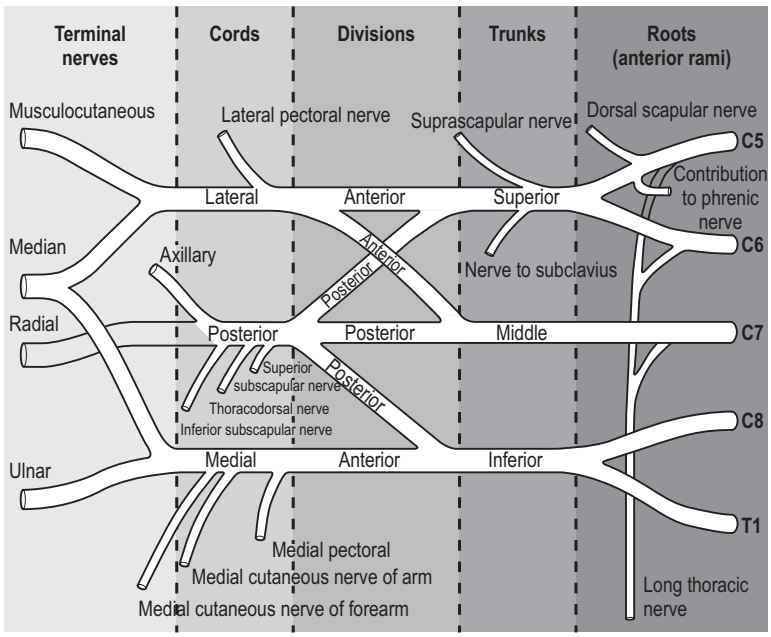

Figure 7.2.1. *Branches of the brachial plexus.*

rotation of the shoulder, but grasp remains intact. It represents most of the brachial plexus paralyses[10] and is considered to have a good prognosis.

- *Lower type*, with involvement of C7, C8 and T1, or Klumpke's palsy that affects muscles of the forearm and hand. The child presents with a paralysis of the hand and wrist. The presence of an ipsilateral Horner's syndrome (anhidrosis, miosis and ptosis) indicates an involvement of the sympathetic fibers associated with an intraspinal avulsion of the root of T1.
- *Whole arm type*, with involvement of C5–T1, with no movement of the upper extremity and often associated with sensory loss.

Extreme lateral flexion and traction of the head may be responsible for the stretch applied to the brachial plexus. The injury results in anything from a mild edema or hemorrhage within the affected nerves, to tearing of the nerve(s) that could be as extensive as to produce a total avulsion of the complete plexus. The C5 and C6 spinal nerves are located in the sulcus nervi spinali of the transverse processes. In that location they are strongly attached by various fibrous slips as extensions of the prevertebral fascia and surrounding structures attached to the spinous processes, and are, therefore, more likely to be ruptured.[11] The C8 and T1 spinal nerves may, more often, be subject to avulsion.[11]

Neuronal injury associated with brachial plexus injury may be of different degrees of severity:

- Neuropraxic lesions are failure of conduction without the axon having been affected, and are reversible.
- Axonotmetic lesions involve disruption of both the myelin sheath and the axon, but with the surrounding neuronal elements kept intact. Wallerian degeneration of the axon distal to the injury occurs.
- Neurotmetic lesions are total sectioning of the nerve with its myelin sheath and supporting connective tissue.
- Avulsion is a separation of the plexus from the spinal cord.

Obstetric brachial plexus palsy occurs in 0.4–2.5 per 1000 live births with an upper root (C5–C6) involvement in 50% of cases; C5, C6 and C7 involvement in 25%; and the whole plexus in 25%.[6,8,12] Neuropraxic and axonotmetic lesions have better prognoses.

Physical examination and treatment

The diagnosis is made by physical examination. The Moro reflex is asymmetric and the biceps' deep tendon reflex is absent. The grasp reflex is, however, normal. The child should be moved very gently: the injuries are painful and the tissues very fragile. Initial treatment is usually conservative with regular

assessment. If the patient fails to show significant improvement by 3 months of age, surgical opinion should be sought.[13]

Physical therapy, consisting of gentle passive mobilization, may be employed to maintain range of motion and prevent contractures while the infant is recovering active motion. Specific motor training can be initiated within the first 2 weeks, with facilitation of active movement. The persistent neurologic deficits may result in the development of internal rotation and adduction contractures of the arm. Gentle stretching of internal rotators should be performed to reduce this risk while avoiding reinforcement of forearm supination.

Osteopathic procedures should be employed as early as possible in the treatment of brachial plexus injury. Traumatic forces may have injured the brachial plexus, but other areas, such as the upper thoracic spine, the first rib, the cervical spine, clavicle and all of the myofascial components of the thoracic outlet, have been stressed as well. Dysautonomia may also be present because somatic dysfunction of the cranial base and occipitoatlantal region can affect the parasympathetic tone through the vagus nerve, while somatic dysfunction in the upper thoracic spine can affect sympathetic nervous function. Direct compression of the venous and lymphatic drainage of the brachial plexus, as well as somatovisceral reflexes, should be considered when attempting to facilitate nerve regeneration. Osteopathic procedures aim to promote fluid, electrolyte and metabolic exchange within the tissues to facilitate drainage of edema and to prevent or reduce tissue scarring. Treatment is intended to optimize nerve regeneration and prevent the development of muscular imbalance. Recuperation of the common motor deficits, such as the absence of active external rotation, flexion and abduction of the shoulder, and function of the biceps should be addressed to minimize bony deformities and joint contractures.

The neonate with possible brachial plexus injury may be examined on the treatment table. Observe for spontaneous movements of the head, trunk, pelvis and limbs. Check for subtle facial palsy that may be found as a concomitant result of birth trauma. Inspect shoulders and limbs for deformities. Evaluate range of motion of every joint of the affected limb. Palpate for tissue texture changes in the upper extremities. Look for signs of shoulder instability such as a palpable or audible click during movement. Palpate for tissue texture changes in the suboccipital, neck and upper thoracic areas. The connective tissues are responsible for the maintenance of shape against both external and internal stresses.[14] Mechanical forces contribute to the development and evolution of the extracellular matrices found in the connective tissues.[15] As tissue texture changes follow trauma, osteopathic procedures should help in resetting structure and function of traumatized connective tissues.

Evaluate, through tests of listening, the function of the humerus, scapula, clavicle, sternum, upper thoracic spine, first ribs, cervical spine and craniocervical joints. This method of assessment is of particular value with this type of pathology because it is so gentle. Anatomic visualization is, as always, important. As you evaluate the patient, visualize the different layers of soft tissue: fascia superficialis, cervical fascia, costocoracoid membrane, sternocleidomastoid and scalene muscles. Evaluate and visualize the different bones involved. Listen to the inherent motions in order to define the dysfunctional area. Study the relationship between the shoulder and the vertebral column. For instance, in the case of a Klumpke's palsy, place the pad of the fingers of one hand on the spinous processes of C6, C7, T1 and T2, and place the other hand on the ipsilateral shoulder. Listen, and look for dysfunctional motion. One vertebral segment may be more dysfunctional than others. The relationship between the humerus and the shoulder should also be balanced. Treat the dysfunctions you identify by applying indirect principles.

Improvement should be rapid and most cases have a favorable prognosis. Injuries involving the fifth and sixth cervical nerve roots have the best prognosis, whereas lower plexus and total plexus injuries have a poorer prognosis. Significant deficit persisting after 3 months should be explored surgically. Prognosis is excellent if antigravity movement of biceps and shoulder abductor is present by 3 months of age. Assessment may be performed by testing the biceps' strength in a supine position while simultaneously palpating the muscle. Bicipital activity should not be confused with flexion of the elbow obtained by the action of the supinator muscle. Surgery is considered by some authors when antigravity movement of the biceps is not present by 3 months of age.[16] Surgery is considered to be justified by others when an initial involvement of the C7 nerve root is present, with a birth weight above the 90th percentile and there is only poor elbow flexion at 6 and 9 months of age.[12]

SHOULDER DYSPLASIA

The plasticity of the glenohumeral joint in the newborn makes it subject to shoulder dysplasia and,

in more severe cases, dislocation. These disorders may be compared to similar disorders occurring in the hip joint – hip dysplasia and hip dislocation – and in some cases the etiology may be similar.

Intrauterine forces applied to the fetus can result in shoulder dysplasia. During the latter part of pregnancy any compression of the upper fetal torso within the uterus may affect the glenohumeral joint and the surrounding soft tissues.

Stresses during the birth process can also contribute to the development of shoulder dysplasia. Increased duration of the second stage of labor, greater than 2 hours, has been described as a factor contributing to shoulder dystocia.[17] Additionally, macrosomia that results in a size discrepancy between the fetal shoulders and the maternal pelvic inlet can, in severe cases, lead to significant neonatal morbidity including asphyxia and trauma, particularly to the brachial plexus. A large fetal trunk, or increased bisacromial diameter, prevents the rotation of the fetal shoulders into the oblique pelvic diameter during delivery.[18] As a consequence, certain obstetrical maneuvers may have to be employed to alleviate the impaction of the fetal shoulders within the maternal pelvis.[19] As stressful as such a disproportionate relationship between the fetus and the maternal pelvis can be, it does not always result in overt trauma to the brachial plexus. It can, however, cause injury to the shoulder that can lead to shoulder dysplasia. This type of injury to the shoulder can occur even from the stresses of an otherwise normal delivery.

Dysplasia of the shoulder may also result from postpartum conditions. The presence of dysfunctional asymmetries in the newborn, congenital muscular torticollis, non-synostotic plagiocephaly and brachial plexus injuries may contribute to abnormal development of the glenohumeral joint. Contractures of the muscles of internal rotation in neonatal brachial plexus palsy are responsible for posterior dislocation of the humerus from the glenoid fossa,[20–22] requiring orthopedic repair.

Physical examination and treatment

Milder cases of dysplasia without dislocation may be treated using osteopathic procedures. If untreated, these glenohumeral dysfunctions can increase with age, becoming the cause of adult scapulothoracic problems. Therefore, the scapula and the glenohumeral joint should be evaluated at birth for any signs of dysplasia, as precisely as the pelvis is evaluated. Particular attention should be directed at the identification of signs of shoulder instability, such as a palpable or audible click during movement. Observe and compare the size and shape of both shoulders. The examination may reveal asymmetry in the number of skin folds in the proximal part of the arms. Observe the freedom and range of motion of both shoulders. Note any restriction, stiffness of movement and asymmetry in movement, particularly external rotation and abduction of the arm. The malposition of the humeral head may result in an apparent difference in the length of the arms, with the arm on the side of the dysplasia appearing to be shorter. Compare the anterior and posterior aspects of the shoulders and look for any posterior fullness that may be indicative of a posteriorly displaced humeral head, necessitating orthopedic attention.

Treat any somatic dysfunction identified using indirect principles. Stabilization of the humeral head in the glenoid fossa may be facilitated when myofascial procedures are applied to the periarticular muscles of the shoulder.

NURSEMAID'S ELBOW

Nursemaid's elbow, also called pulled elbow, is a radial head subluxation that occurs in younger children when traction is applied suddenly to their hand or forearm. This commonly occurs when an adult is attempting to lift a child up by pulling upward while holding the child's hand. Traumas such as falls or when the infant initiates rolling over are other possible causes of this condition.

The head of the radius articulates with the radial notch of the ulna and the surrounding annular ligament. Under normal conditions, the annular ligament encircles the head of the radius with a certain amount of tension that maintains the contact with the radial notch. The normal movements of the head of the radius, within the ring formed by the annular ligament and the radial notch of the ulna, are anterior and posterior motion. Pronation is associated with posterior radial motion and supination with anterior radial motion.

In nursemaid's elbow a subluxation of the developing radial head from the annular ligament of the elbow joint occurs. A combination of pronation and traction on an extended elbow causes a proximal slip of the annular ligament over the top of the radial head with resultant interposition of some fibers of the anterior joint capsule between the two bones.[23]

Physical examination and treatment

The most common symptoms of nursemaid's elbow are immediate pain and an inability to move the arm. The child will have a partially flexed elbow

with pronation of the forearm. Most of the time anxiety is also present.

The intention of osteopathic treatment is to restore motion between the head of the radius and the radial notch of the ulna and surrounding annular ligament. Ligamentous articular strains may be balanced utilizing indirect principles that apply perfectly to this condition. Balancing the annular ligament and the radial collateral ligament of the elbow may prevent recurrence of a posterior radial head subluxation. The caregiver should be told to avoid pulling or lifting their child by the arms or hands.

Lower extremity

A frequent complaint in an osteopathic practice is intoeing, i.e. the child's feet turn in when walking or running. Malposition of the feet, developmental dysplasia of the hip and toe walking are among the other complaints encountered with infants, while sprains are more frequently encountered with the older children and teenagers.

DYSFUNCTIONS OF THE FEET

Metatarsus adductus

Metatarsus adductus is an adduction of the forefoot that occurs in 1 : 5000 live births.[24] The classic view of it resulting from intrauterine positioning is debated, since genetic factors may contribute.[25] Sleeping in the prone position also seems to promote it. This is a frequent cause of intoeing during the first year of life and is more frequently encountered on the left side.[24] Normally, in the neutral position, the heel-bisector line drawn through the midline axis of the hindfoot passes through the forefoot at the second web space. In cases of metatarsus adductus, the line passes lateral to the third toe. Therefore, an angulation exists medially between the forefoot, or metatarsals, and the hindfoot. Sometimes a transverse crease is present on the medial side of the foot and the lateral border of the foot is convex (Fig. 7.2.2).

Metatarsus adductus associated with an inversion of the foot is named metatarsus varus and adduction of the first metatarsal is metatarsus primus varus. Metatarsus adductus is frequently associated with internal tibial torsion. Metatarsus adductus associated with retracted equinus – the inability to dorsiflex at the ankle – is indicative of a diagnosis of clubfoot.

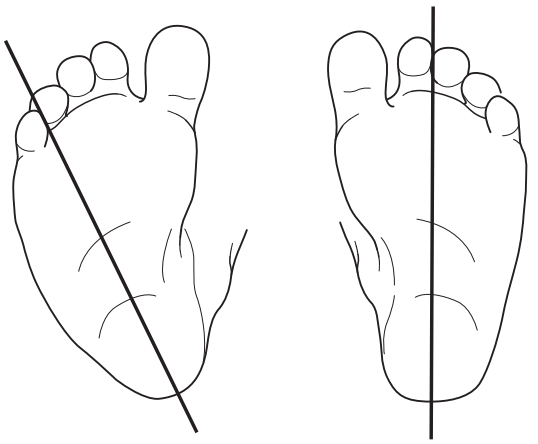

Figure 7.2.2. *Metatarsus adductus of the right foot.*

Dysfunctions of the feet might not seem grave, but left untreated they will lead to postural dysfunctions and compensatory dysfunctions of the feet, with difficulty wearing shoes and the development of bunions and hammer toes. A group of children with metatarsus varus, followed an average of 7 years, showed that 10% maintained a moderate, although asymptomatic, deformity and 4% demonstrated residual deformity and dysfunction (stiffness).[25] The opinion is that metatarsus adductus left untreated will persist into adulthood in 4–5% of cases.[26,27] Furthermore, some cases only appear to be clinically improved because of a compensatory pronation of the midtarsal joints and rearfoot.[28]

Metatarsus adductus is classified according to its flexibility. Normally, an infant should extend and abduct the foot when being tickled (e.g. with a toothbrush) along the lateral border of the foot, particularly over the fifth metatarsal head. The inability of the infant to react in such a way is indicative of metatarsus adductus. The physician should consider the total body approach and treat any dysfunctional mechanics, particularly of Lisfranc's joints (tarsometatarsal) following the principles of functional procedures. The parents should be encouraged to stimulate abduction of the forefoot, using a toothbrush or similar stimulus, as described above. In more severe cases it should be proposed that stretching exercises be practiced several times a day (e.g. at each diaper change). The calcaneus is maintained between the thumb and index finger, while the forefoot is gently pulled into a corrected position, holding the correction for 10 seconds and repeating the process about five times. It should be stressed that this exercise should be done properly, without creating a valgus of the hindfoot. If these treatments fail, and also in cases of

severely rigid feet, a series of casts are used to gradually straighten out the deformity.

Congenital idiopathic talipes equinovarus (CTEV)

CTEV, or clubfoot, is a complex deformity of unknown pathogenesis with several etiologic hypotheses that range from genetic to intrauterine factors.[29] The head–body angle of the talus (declination angle) which normally increases after the 16th week of gestation has been found to be decreased in CTEV, associated with hypoplasia of the talus.[30] More recently, studies indicate that talar deformity is not the primary lesion, but follows loss of spatial orientation of the deltoid and spring ligaments and tibialis posterior tendon insertion, with contracted soft tissues.[31]

CTEV is a relatively common congenital deformity that occurs with geographical differences ranging from 0.5 : 1000 live births in Japan to 7 : 1000 in the South Pacific (1.2 : 1000 live births among Caucasians).[32] About 50% of the cases are bilateral. The male-to-female ratio for affected children is 2.5 : 1.[32,33]

Clubfoot deformity presents with different components: hindfoot equinus (inability to dorsiflex), hindfoot varus and metatarsus adductus. The flexibility of the deformity is important to determine the degree of severity. Classic treatment consists of manipulation of the foot followed by casting.[34] Generally, casting is attempted for 3 months; if unsuccessful, surgery is planned.

Osteopathic procedures should be employed as early as possible for best results. Every bone of the hindfoot – the calcaneus, talus, navicular and cuboid bones – should be evaluated and treated to release any dysfunctional relationships between them and to equilibrate the soft tissues surrounding them. The deltoid and plantar calcaneonavicular (spring) ligaments are of particular importance and should be balanced with gentle fascial release procedures.

Pes cavus

Pes cavus, or high-arched or hollow foot, should first make you think of ruling out an underlying neurologic disorder as the primary etiology. Anterior pes cavus, where both the medial and lateral longitudinal arches are high, is benign. Medial pes cavus is more severe, often with claw foot deformity of the toes. The two may be differentiated by dorsiflexing the foot. In the presence of medial pes cavus the claw foot deformity of the toes increases with dorsiflexion of the foot.

The position and range of motion of the hindfoot bones, particularly the talus, should be evaluated. Hollow foot is often associated with a flexion–external rotation pattern of the craniosacral mechanism.

You must differentiate total flat foot or total hollow foot from partially flat foot or partially hollow foot. In the latter, only the posterior portion of the longitudinal arch is involved, resulting from an imbalance of the subtalar, calcaneocuboid or cuneocuboid articulation.

Pes planus (flat feet)

A rigid pes planus is a pathologic flat foot, also named tarsal coalition, in which one or more of the tarsal bones that should have a joint between them become fused. In infants the fused joints are cartilaginous and are still relatively flexible. Thus the condition is typically not symptomatic before adolescence.

Physiologic pes planus is a loss or reduction of the longitudinal arch (Fig. 7.2.3) that can be reestablished when the child stands on their toes. Physiologic pes planus is flexible and often associated with generalized ligamentous laxity. In a sample of primary school children, 2.7% demonstrated flat feet, and being overweight was shown to increase the prevalence of the condition.[35] Because of ligamentous laxity and/or obesity, the child's ankles cave in.

The plantar calcaneonavicular ligament plays an important role in maintaining the arch of the foot (Fig. 7.2.4). It supports the head of the talus and is part of the astragalonavicular joint. Any dysfunction of the plantar calcaneonavicular ligament affects the head of the talus that tends to be displaced downward, medially and forward by the weight of the body. The tibialis posterior, an inversion muscle, lies directly below the plantar calcaneonavicular ligament and participates considerably in maintaining the longitudinal arch of the foot. In cases of dysfunction of these structures, the foot becomes flattened, expanded and turned laterally.

This condition has been suggested as a cause for tarsal tunnel syndrome.[28] It has also been suggested as contributing to back and knee problems later in life, but no evidence supports this contention. Flat feet and dysfunction of the spine are, however, very often two components of the same problem, where postural mechanics are involved. Flat foot is usually

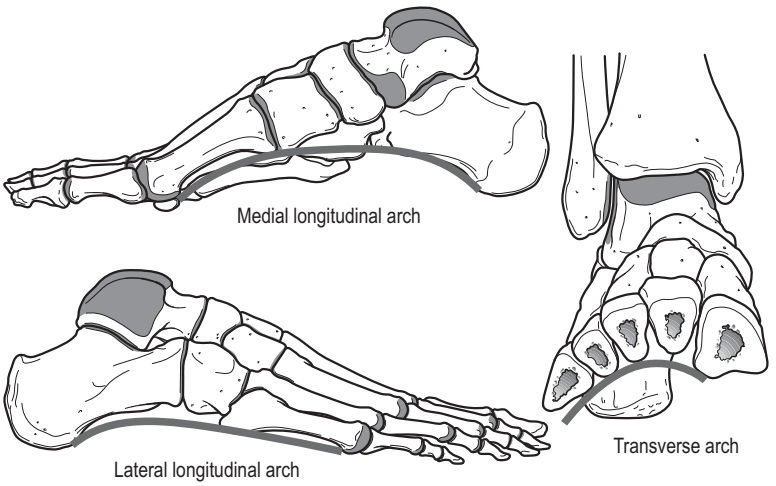

Figure 7.2.3. *Arches of the right foot.*

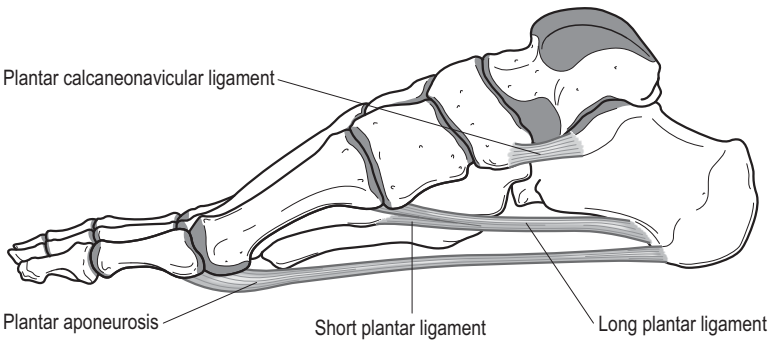

Figure 7.2.4. *Support for the arches of the foot.*

associated with an extension–internal rotation pattern of the craniosacral mechanism.

The treatment of physiologic pes planus with orthotic devices is controversial.[36] Insole arch supports diminish some of the muscular activity that maintains the arch and tend to weaken the muscles.[35] Two studies suggest an association between wearing shoes at an early age and flat feet.[37,38] This stresses the importance of allowing the feet to grow and develop without constraint. Parents should be reassured about their concerns regarding the child's appearance and gait. This condition tends to improve between 2 and 6 years of age.[39] To promote optimal growth without dysfunctional mechanics, the osteopathic practitioner should consider the global posture of the child. Check for rearfoot valgus by assessing the position and freedom of the calcaneus and talus. Check also for internal rotation dysfunction of any of the tarsal bones. Encourage the child, through their parents, to maintain a healthy lifestyle, to go barefoot as much as possible and not to become overweight. Physical activities that strengthen foot inversion, such as walking on the lateral borders of the feet or picking objects with the toes, should be recommended.

Calcaneovalgus

Positional calcaneovalgus, the result of intrauterine malposition, is a flexible dorsiflexion of the ankle with a mild subtalar joint eversion. It is frequently associated with external tibial torsion and has the appearance of flat feet. Treatment follows the principles of functional procedures to address the subtalar dysfunction.

Sprains and strains

Sprains and strains are common in the pediatric population. Young athletes are particularly vulnerable. Activities that involve jumping and landing, as in skateboarding, often result in such injuries. These

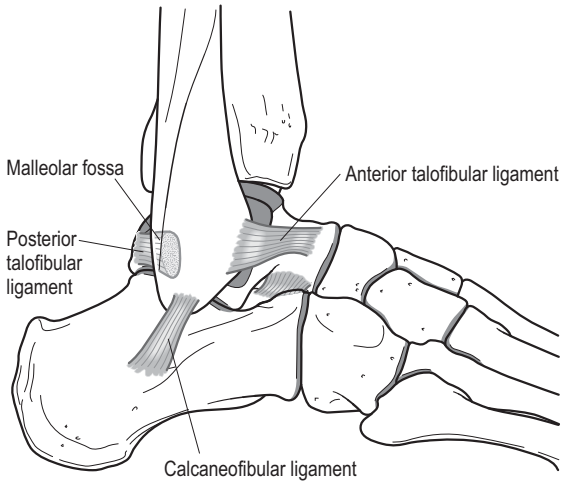

Figure 7.2.5. *Lateral view of the ankle joint.*

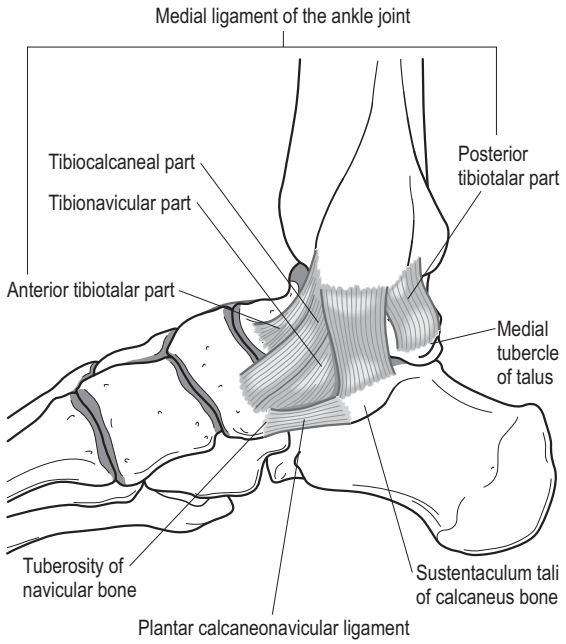

Figure 7.2.6. *Medial view of the ankle joint.*

injuries are, however, not limited to young athletes. Young children can sustain sprains and strains with activities of daily living, such as ascending or descending stairs. These injuries may be overlooked because of the child's tendency to get up and resume activity unless severely injured; however, if not properly addressed, such injuries can be the source of functional asymmetry and somatic dysfunction, often with sequelae in other anatomic areas at a later date. Following a foot or ankle sprain that causes excessive or prolonged midfoot pronation, abnormal patellofemoral mechanics may result.[40]

Traumatic twisting of the forefoot, most often inversion, commonly causes ankle injuries, often resulting in, but not limited to, sprains or strains involving the tibia, fibula and talus. It should be remembered that these stresses may also result in specific dysfunctions between the fibula and talus, the talus and calcaneus, the talus and navicular bone, and the calcaneus and cuboid, as well as dysfunctions between any of the other adjacent tarsal and metatarsal bones (Figs 7.2.5, 7.2.6).

On physical examination, the acutely injured ankle presents with pain and swelling and, with more severe injuries, ecchymosis. Ecchymosis indicates possible ligamentous tears or bony fracture necessitating radiographic evaluation. Further, if the subject is not willing to bear weight on the injured ankle, or if significant edema is present, radiographic evaluation is also appropriate to evaluate the extent of injury.[41]

Once bony fracture has been ruled out, soft tissue injuries may be treated with osteopathic manipulation by employing indirect principles. Under these circumstances, the patient should experience no aggravation of discomfort during the treatment procedure and will often feel a significant reduction of pain and swelling following the intervention. Post-treatment reduced weight bearing and avoidance of stressful activities should be recommended. Immobilization of the injured area with strapping methods should be considered for adolescents and individuals likely to be involved in weight-bearing activities. When there is no more pain for a week, a rehabilitation program can be organized to work the injured ankle in full range of motion with progressive resistance exercises. Exercises with a balance board, to strengthen proprioception, function and coordination, are also indicated. The patient places the foot on the board and first does flexion–extension movements, followed by rotation of the ankle around the ball (Fig. 7.2.7).

DYSFUNCTIONS OF THE LEGS

Tibial torsion

A thorough history and physical examination should be performed to rule out diagnoses such as cerebral palsy, which can present with rotational misalignment of the legs. Internal tibial torsion is said to be

Figure 7.2.7. *Rehabilitation includes exercises with a balance board.*

the result of intrauterine positioning or from the child's habit of sitting on their feet. This is often noticed by the parents between 1 and 2 years of age and is a common cause of intoeing in children under 3 years of age. Internal tibial torsion is more often bilateral; when unilateral, the deformity most commonly affects the left side.[24] Parents complain that their child is clumsy, trips and falls easily, although intoeing in athletes has been suggested as beneficial in activities like sprinting.[42] About 90–95% of all torsional deformities resolve spontaneously by maturity.[43] When it does not resolve, however, dysfunctional rotation results in improper alignment of the lower limb and is associated with arthrosis of the hip, knee and ankle.[44]

External tibial torsion is usually diagnosed later and demonstrates a tendency to increase with age. It is associated with conditions of the extensor apparatus as unstable patellofemoral joints and Osgood–Schlatter disease.[45,46]

Examination of rotation of the tibia is best done with the child in the prone position with their knee flexed to 90°. This allows measurement of the foot–thigh angle, the angle formed between the long axes of the femur and the foot.

The osteopathic practitioner should consider a total body approach with specific attention to intraosseous and myofascial tensions in the lower limbs. The relationships between the fibula and the tibia, as well as between the tibia and the femur and the tibia and the talus, should be balanced. Osteopathic procedures directed at functional alignment and balance of the lower extremity improve function and should reduce stressful compensatory patterns that may later result in patellar tendonitis and arthritis.

Femoral torsion

Femoral torsion is defined by the angle between the femoral neck axis and the transcondylar axis of the distal femur. Femoral torsion can be internal (femoral anteversion) or external (femoral retroversion) and results in the knees pointing toward or away from each other, respectively. A normal femur is anteverted, i.e. the femoral head and neck are rotated anteriorly with respect to the femoral condyles. Babies have 30° of femoral anteversion. This decreases by about 1.5° per year to reach 10° in adult life.[33] Femoral anteversion is also a very common cause of intoeing in children under 3 years of age, the child being obliged to internally rotate the femurs in order to re-center the femoral heads in the acetabula. Observation of the child's gait allows one to differentiate between intoeing that is the result of internal tibial torsion as compared to femoral anteversion where the patellae are positioned more medially on the knees. The child trips and falls frequently and does not like to sit with their legs crossed, preferring to sit in a 'W' position (Fig. 7.2.8). Parents note that the child's shoes are very quickly worn out in an asymmetric pattern. Studies in adults in whom the condition remained uncorrected found a correlation between femoral anteversion and arthritis of the knee.[47]

Normally, hip range of motion shows greater amplitude in medial rotation than in lateral rotation. Abnormal femoral anteversion can be predicted (±2 SD from the mean) if the difference between medial and lateral rotation is 45° or more.[48]

The osteopathic practitioner should consider a total body approach and release intraosseous and myofascial tension in the lower limbs. The pelvis should be diagnosed and treated if necessary, as well as the coxofemoral joint. Somatic dysfunction of the innominate, particularly during periods of growth, is significant because of the influence it can have on the position of the femur.

Figure 7.2.8. *Sitting in a 'W' position.*

Genu varum and genu valgum

Genu varum and genu valgum – also known as bowlegs and knock-knees, respectively – are common angular deformities of the lower extremities in children. All babies are born bowlegged. Between the ages of 2 and 3 years the bowlegging gradually decreases, and by 3 years the average child is maximally knock-kneed. The knock-kneeing straightens minimally over the next several years and, by age 7 years, most children have reached the typical adult configuration, which is slightly knock-kneed.

Persistent genu varum is better tolerated functionally than valgus, which causes stress to the medial aspect of the knee joint with the subsequent development of pain. The wider the varus or the valgus, the greater the shear stress (lateral–medial forces) on the joint.

An angular deformity is not physiologic when it is asymmetric or painful and radiographs might be necessary. Bilateral varus is associated with craniosacral flexion of the sacrum, and bilateral valgus with a sacrum in craniosacral extension. Unilateral varus problems are very often associated with flexion–external rotation patterns on the same side at the level of the pelvis, the temporal bone or the occipital bone. Similarly, unilateral valgus problems are associated with extension–internal rotation patterns of these same areas.

Using indirect procedures from a total body perspective, treatment is directed at specifically diagnosed dysfunctional mechanics. The distance between the knees (with the ankles together) of children who have varus, or between the ankles (with the knees together) of children who have valgus, can be measured to follow the response to treatment.

The knee can also present minor strains. A commonly found pattern occurs when an increased medial femoral torsion is combined with excessive lateral torsion of the tibia. This is found more often as the result of physical activities, such as in skiing, when the ski is stuck in the snow, slightly abducted, and the rest of the body moves forward. The relationship between the tibia and the femur should be balanced to address these strains.

Patellar disorders

Congenital dislocation of the patella is rare and may be isolated or associated with other limb malformations.[49] Patellar instability is not a congenital condition, although anatomic configurations such as patella alta, trochlear dysplasia and ligamentous laxity are thought to participate in the instability. MRI permits visualization of the nonosseous components of the patellofemoral articulation in the child. The cartilaginous composition of the articulation provides less restraint to lateral movement of the patella that allows instability.[40] The trilaminar soft-tissue structures[50] surrounding the patella present interconnections with the fibers of the iliotibial tract, lateral hamstrings and lateral quadriceps retinaculum. Tightness in these structures has been suggested as causing excessive posterior and lateral pull, contributing to patellar instability, especially if the medial patellofemoral ligament is injured and cannot stabilize the knee.[40] These structures are linked to the pelvic bone, and recurrent dislocation of the patella can be associated with pelvic dysfunctions that interfere with the balance transmission of weight-bearing forces.

Osgood–Schlatter disease occurs mainly in athletic adolescent boys. It is suggested that a well-developed and inelastic quadriceps creates a traction apophysitis on the tibia, with the development of loose ossicles and elongation of the patellar ligament leading to patella alta.[51] Patients with Osgood–Schlatter disease also present with increased external tibial torsion that, in association with other factors, has been suggested to predispose to the onset of the disease.[46]

The osteopathic practitioner will consider a total body approach and release the pelvis, the hips, the knees and the patellofemoral articulations (Fig. 7.2.9). Myofascial release should be considered for the thigh and patella. Gentle and pain-free stretching exercises should be done at home on a regular basis and should address tension in the quadriceps, the upper and lower iliotibial tract, the hamstrings, the hip flexors, the hip abductors, the gastrocnemius and the soleus. A long-term maintenance program

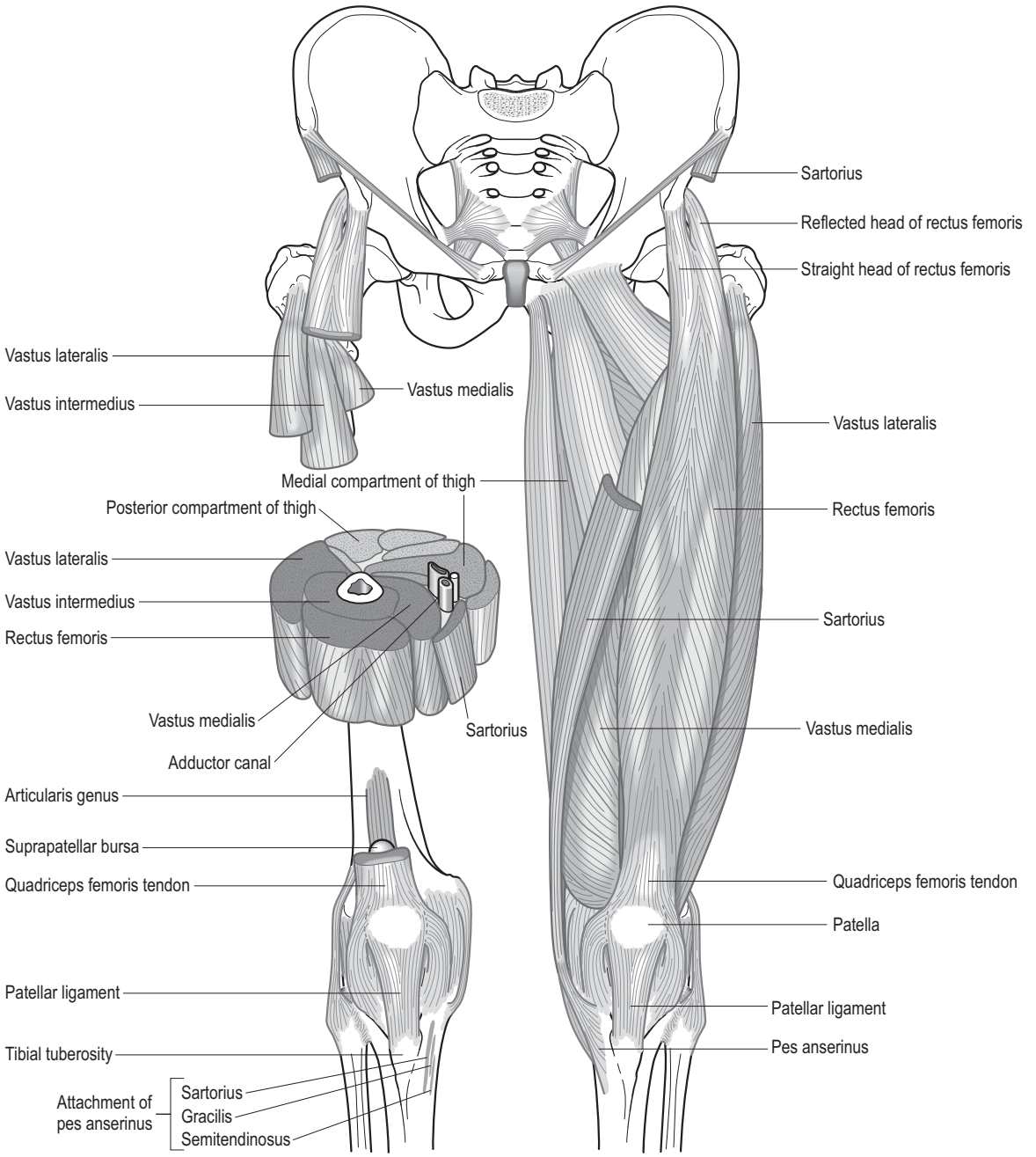

Figure 7.2.9. *Muscles of the thigh functionally and dysfunctionally uniting the pelvis and knee.*

should include strengthening in terminal knee extension in association with isometric exercises of the above muscles. Patellar knee sleeves are sometimes useful. They might have proprioceptive effects offering support. Patients feel less pain and the support provides some kind of reassurance.

DYSFUNCTIONS OF THE HIPS

Different terms describe hip dysfunctions. 'Developmental displacement of the hip' (DDH) is proposed in replacement for 'congenital dislocation of the hip' to stress the fact that the condition can occur prenatally or postnatally.[52] The different variants of the abnormalities of the hip joint include shallowness of the acetabulum and capsular laxity with resultant instability and propensity for dislocation. DDH refers to a deficient development of the acetabulum that could lead to subluxation and dislocation.

The femoral head remains well covered by the acetabulum in the early fetal period (between 6 and 20 weeks) and dislocation does not occur at this early time.[53] However, at birth, the human acetabulum is shallower than at any other time during development and is consequently vulnerable for hip instability.[54] Mechanical factors seem to play a role in neonatal hip instability. Moderate loading of the hips at 45° of flexion maintained for 3 hours has been shown to distend the articular capsule and to produce deformation and dislocation of the joint resembling that found in DDH.[55] Modifications in the pressure on the cartilaginous acetabulum are thought to interfere with normal bone growth.[56]

Uterine constraint is proposed by numerous authors as an explanation for the association between DDH and other deformations. Foot deformity,[57] congenital torticollis, congenital postural scoliosis[56,58] and plagiocephaly[59] are frequently associated with DDH. The sleeping position of the infant, with a preference to lie on one side (the 'side-lying syndrome'), has also been proposed as a contributing factor to DDH.[60]

Leg postures, associating extension and lateral rotation, critically predispose the infant to hip dislocation during fetal life and at birth. Interestingly, the newborn psoas muscle is totally relaxed in full abduction, flexion and lateral rotation. This muscle is always a lateral rotator of the hip, but exerts a much greater effect when the femur is abducted.[61] Extending the hip results in a levering action that is potentially critical if associated with other contributing factors such as acetabular or femoral anteversion and dysplasia.[62] Caution should, therefore, be exerted when moving the leg into combined extension and lateral rotation.

DDH occurs in 1:1000 live births and is more frequently encountered on the left side. Being female, first-born, having been carried or delivered in the breech position[57,63,64] and having a family history of acetabular dysplasia or ligamentous laxity are the main risk factors.[60]

Dislocation of the hip requires orthopedic attention. Clinical examination reveals asymmetries in the number of skin folds on the thigh and the inability to completely abduct the thigh when the knee and hip are flexed. The malposition of the femoral head may cause the leg on that side to look shorter than the other.

Barlow and Ortolani positive tests confirm the diagnosis and are performed with the child supine on a flat pad, placing the fingers on the baby's greater trochanters and the thumbs on the inside of the lower portion of the thighs and knees. The hips and knees are flexed to 90°. The Barlow test consists of adducting the legs and pushing down gently on the knees in an attempt to disengage the femoral head from the acetabulum. A 'clunk' will be felt as the femoral head dislocates. The Ortolani test relocates a dislocated hip and is performed by slowly abducting the thighs while maintaining axial pressure. The fingers on the greater trochanter exert a movement in the opposite direction to assist the return of the femoral head to the acetabulum. The examiner will again feel, and hear, a 'clunk.' Diagnostic ultrasonography may be carried out after 4–6 weeks and radiographs after 4–6 months; before these ages there is insufficient ossification for the tests to be diagnostic.

Different methods of treatment are proposed for DDH, with good results. Abduction devices (Pavlik harness) and traction followed by plaster immobilization are always done with special care because of the risk of aseptic necrosis of the femoral head. A program of home abduction–adduction exercises administered by the parents has been successful in infants with limited abduction and acetabular dysplasia without dislocation.[57]

Developmental dysplasia of the hip – insufficient depth of the acetabulum to accommodate the femoral head – in the infant can be associated with pelvic imbalance, such as intraosseous dysfunction of the innominates and sacrum, coxofemoral dysfunctions and eventually with craniosacral dysfunctions. The innominate bone needs to be in a neutral position in order to provide a satisfactory acetabular placement.

An internally rotated innominate results in a higher position of the acetabulum, leading to an apparently shorter leg on that side. This can be confused with the apparent inequity of leg length found in association with hip dislocation.

Osteopathic procedures attempt to balance the craniosacral mechanism of these children. The relationship between the sacrum and the occiput, the temporal bones and the innominates should be balanced.

Special care is given to myofascial structures responsible for pelvic tensions or pelvic asymmetry. Myofascial procedures should be applied to the periarticular muscles of the hip, in particular the iliopsoas, the adductors and the abductors, to release contracture of the joint and improve restrictions of motion in adduction or abduction.

The treatment of dysplasia is intended to stabilize the femoral head in the acetabulum and to allow the growth of a symmetric pelvis with a balanced sacrum, innominates and hips. Dysfunctional interosseous relationships between the sacrum and the innominates, and between the innominates and the femurs, should be identified and treated. Any dysfunctional intraosseous relationship between the ilium, ischium and pubes should be also balanced, and, if present, intraosseous dysfunction of the sacrum treated.

Clinical examination of these children should be repeated during the 1st year, with an annual follow-up, until full skeletal maturity. Dysplasia may result in early development of osteoarthritis of the hip.[65–68] Treatment should be directed at promotion of function and prevention of future degenerative changes.

GAIT DISORDERS

Toewalking

By the age of 4 years children should be walking with a heel–toe gait. After this age, toewalking is abnormal and can be due to an underlying neurologic disorder. A tight Achilles tendon may be present, but in other cases nothing will be found. Look for any extension dysfunction of the craniosacral mechanism. Balance the sacrum and the craniocervical junction. Release the posterior myofascial components of the spine and inferior limbs. Teach the child to walk heel–toe.

The child with a limp

Because serious medical and orthopedic conditions may be responsible for limping, any such underlying pathology should be ruled out before using manipulation as a primary treatment. For example, a limp or a waddling gait between the ages of 2 and 6 years might be associated with a congenital coxa vara, where the angle between the femoral neck and the femoral shaft is less than normal (<130°). Coxa valga is an increase in that angle (>135°) and, in this case, the child may present with increased internal rotation and adduction of the hips.

Observation of the child while walking allows one to determine what area is dysfunctional. It is better to have the child bare legged to best visualize the different components of the lower extremity. Observe the foot angle, the direction in which the child's feet point when they walk. Observe for tibial and femoral torsions and the movements of the hips. Determine which part is not following the global movement of the limb or if one side does not contact the floor in the same fashion.

The postural balance should be evaluated and any dysfunctional asymmetries treated. An asymmetry of leg length might be present as the cumulative result of several dysfunctions. For example, an innominate in external rotation on the right side, combined with an innominate in internal rotation on the left side, will give the appearance of a longer leg on the right side. Evaluate the range of motion of the different joints of the lower limbs and, utilizing appropriate manipulative procedures, treat any dysfunction identified.

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7.3 EARS, NOSE AND THROAT

OTITIS MEDIA

Otitis media (OM) is among the most common of illnesses affecting preschool children.[1] Almost every child experiences OM at least once before their third birthday[2] and 20% of children experience recurrent OM.[3] Its prevalence has increased considerably recently, resulting in an enormous economic burden to society.[4] Clinical classifications include acute otitis media (AOM) and chronic otitis media with effusion (COME).

AOM is a viral or bacterial infection, commonly secondary to an upper respiratory infection and usually occurring in young children from the age 3 months to 3 years. It presents with a sudden inflammation in the middle ear, fever, pain and irritability. An incomplete resolution of AOM or an obstruction of the pharyngotympanic tube (PT), also called the Eustachian or auditory tube, may lead to an effusion in the middle ear containing common pathogenic bacteria. Repeated episodes of acute symptoms are considered to be recurrent AOM.

COME is a chronic inflammation of the middle ear mucosa, with the retention of fluid within the middle ear space that lasts more than 3 months. It is a condition wherein irreversible changes have occurred, affecting the tympanic membrane, the PT or the middle ear.

The multitude of studies attempting to identify the causative factors of OM have provided multiple, and often opposite, results. This is probably because the etiology of individual cases of OM is often multifactorial, including genetic, environmental, nutritional and behavioral factors. Thus, the number of variables necessary for consideration makes such studies extremely difficult. The following have, however, been identified as risk factors for the onset of OM: genetic predisposition,[5,6] low birth weight,[7] male gender,[8] number of siblings,[9] day-care attendance,[10] not being breastfed,[1] use of a pacifier,[11,12] season of the year,[10,13] passive exposure to smoking[14] and low socioeconomic status.[15] On the other hand, breastfeeding, even for periods as short as 3 months, has been shown to reduce the incidence of OM in childhood.[16]

Anatomic factors should also be considered. The ear, particularly the middle ear, and adjacent structures provide a site where genetic, environmental, nutritional and behavioral factors can interact, resulting in the development of OM. The three parts of the ear – external, middle and internal (Fig. 7.3.1) – are related anatomically and functionally to the temporal bone. The external ear consists of the auricle (pinna) and external acoustic meatus. The auricle on the lateral aspect of the head, at the level of the temporal bone, reflects the global position of the temporal bone. A protruding auricle, for example, are often associated with external rotation of the homolateral temporal bone. The auricle functions to collect sound waves. The external acoustic meatus

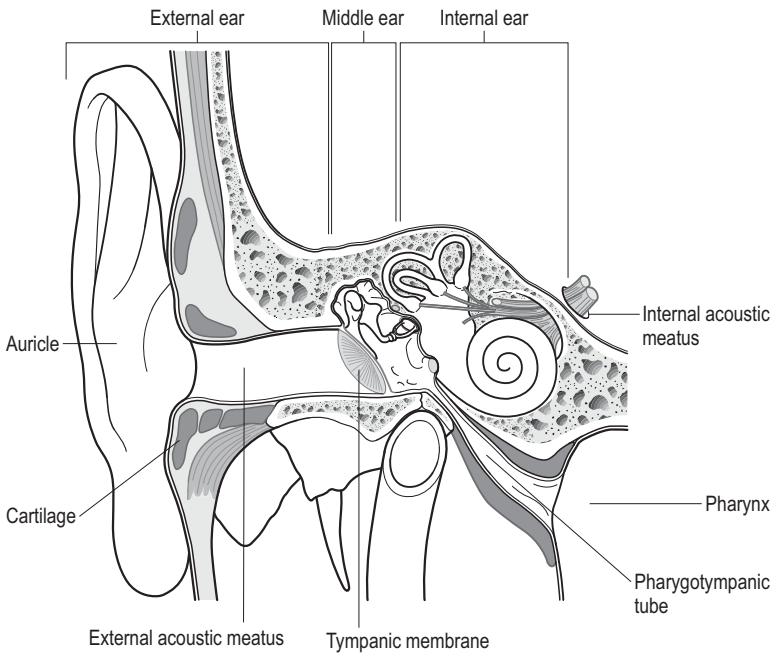


Figure 7.3.1. *Ear.*

terminates in the tympanic membrane. Its lateral part is membranous, continuous with the auricle. The medial part is surrounded by the squamous portion of the temporal bone above and the tympanic portion in front and below.

The middle ear or tympanic cavity, an air-filled space, is located between the tympanic membrane laterally and the lateral wall of the internal ear medially. It contains three bones, or ossicles – the malleus, incus and stapes – that transmit vibrations from the tympanic membrane to the cochlea of the internal ear. The tympanic cavity is open posteriorly to the mastoid antrum, an air sinus located in the petrous portion of the temporal bone, and to the interconnected mastoid air cells. Anteriorly, the tympanic cavity communicates with the nasopharynx through the PT. A mucosa covers the complete cavity, including its contents, the three ossicles and the two muscles (tensor tympani and stapedius), and forms the inner layer of the tympanic membrane. This mucosa is in continuity with that of the pharynx. The mastoid cavity, mastoid antrum and auditory ossicles are nearly completely developed at birth.

The internal ear consists of several bony cavities, the vestibule, the semicircular canals and the cochlea that form the bony labyrinth. It contains the membranous labyrinth with the organ of hearing (the cochlear duct) and the organs of balance (the semicircular ducts). The membranous labyrinth floats in the perilymph, the fluid filling the bony labyrinth. The structures that form the internal ear are also nearly completely developed at birth.

Most of the above structures of the ear can be found nesting within the petrous portion of the temporal bone. In diseases of the ear such as OM, as well as in balance and hearing disorders, this anatomic relationship confers great significance on the temporal bone and its function and dysfunction.

The temporal bone is formed by the squamous, petromastoid, tympanic and styloid parts. The petromastoid part develops in the cartilaginous otic capsule of the cranial base. The squamous and the tympanic portions are ossified from mesenchyme. The tympanic portion (the tympanic ring) unites with the squama just before birth.[17] Total fusion of the temporal bone, except the distal part of the styloid, is complete by the end of the 1st year. Nevertheless, the mastoid portion is completely flat at birth. The mastoid process, a postnatal petrous development, begins to develop with the growth of the mastoid air cells and because of the traction from the tendon of the sternocleidomastoid (SCM) muscle. The development of the mastoid process is dependent on the child's ability to lift their head (extend their cervical spine) and to rotate their cervical spine symmetrically. For this reason the prone position, recommended in the statement 'back to

sleep, prone to play' is important. In this case, function determines structure, and children with torticollis will present with asymmetry in the shape and size of the mastoid processes. Alternatively, infants with non-synostotic plagiocephaly may present with a flattening in the area of the occipitomastoid suture, where compressive forces inhibit mastoid development.

The expansion of the mastoid process is of particular significance. The mastoid air cells that develop inside the mastoid during the growth period are important components of the complex system that regulates and buffers the fluctuations of middle ear pressure.[18] The volume of the mastoid air cells is about 20 times the volume of the tympanic cavity. Often compared to an air reservoir, the mastoid cavity is an active space for gas exchange through its submucosal capillary network[19] (Fig. 7.3.2).

The submastoid cell structure in humans is histologically similar to that found in the pulmonary alveolar and nasal membranes, and, therefore, is suitable for gaseous effusion and diffusion. The production of gas within the tympanomastoid cavity keeps the internal pressure at the same level, or higher, than atmospheric pressure.[20] Swallowing allows gas to be expelled through the PT into the pharynx. This positive pressure gradient prevents bacteria from entering the tympanomastoid cavity.[20] The depth of the mastoid air cell system has been found to be shorter in children with secretory OM compared with healthy individuals.[21] Decreased mastoid pneumatization has been proposed as a prognostic indicator for chronic inflammation of the middle ear, as has poor outcome with OM when the mastoid is poorly pneumatized.[22]

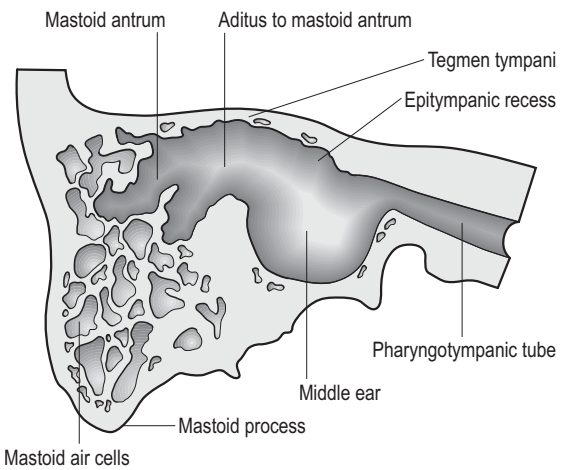


Figure 7.3.2. *Mastoid antrum.*

The mastoid cells connect to the tympanic cavity and through the PT to the nasopharynx. Both mastoid cells and PT are of paramount importance in the normal function of the ear and, consequently, the pathogenesis of OM. The PT connects the middle ear to the nasopharynx, balances pressure between the middle ear and ambient air, clears debris and secretions toward the nasopharynx and also protects the middle ear against nasopharyngeal secretions and noxious agents from the airways. It begins on the anterior wall of the tympanic cavity and extends forward, medially and downward to the nasopharynx posterior to the inferior meatus of the nasal cavity (Figs 7.3.3, 7.3.4). Because of these close relationships between the middle ear and the nasopharynx, OM, frequently described as a complication of rhinitis, may be considered to be a disease of the upper respiratory tract. Descriptive and functional anatomy of the nose and nasopharynx is discussed in 'Rhinitis' and 'Sinusitis' below.

The PT is shaped like two cones joined together at their apices. The posterolateral cone, shorter, approximately one-third of the PT, is osseous (protympanum), located inside the petrous temporal bone. It ends at the junction of the petrous and squamous parts of the temporal bone, immediately posterior to the foramen spinosum. The remaining two-thirds of the PT are fibrocartilaginous, partially fixed to the cranial base, in a furrow following the sphenopetrosal synchondrosis, between the petrous portion of the temporal bone and the posterior border of the greater wing of the sphenoid. The upper border of the cartilaginous PT is arched laterally and looks like a hook on transverse section. A fibrous membrane completes the tube. The tubal isthmus, where the PT diameter is smallest, joins the two cones of the PT. The cartilaginous portion has a greater vertical inclination than the osseous portion.

The length of the PT in the adult is approximately 35–45 mm. The PT in the newborn is approximately half its adult length and reaches approximately 98% of its adult length by 7 years of age. The ratio of the length of the cartilaginous and junctional portions of the PT to the length of the bony portion is 8:1 in infants and 4:1 in adults.[23] The PT in infants is not only shorter, it is also more horizontal, and, therefore, the clearance function is less effective. Additionally, when the bony portion of the PT of children with secretory OM is compared to that of healthy children, it is found to be even shorter.[21]

The PT connecting the middle ear and nasopharynx has been compared to the bronchial tree

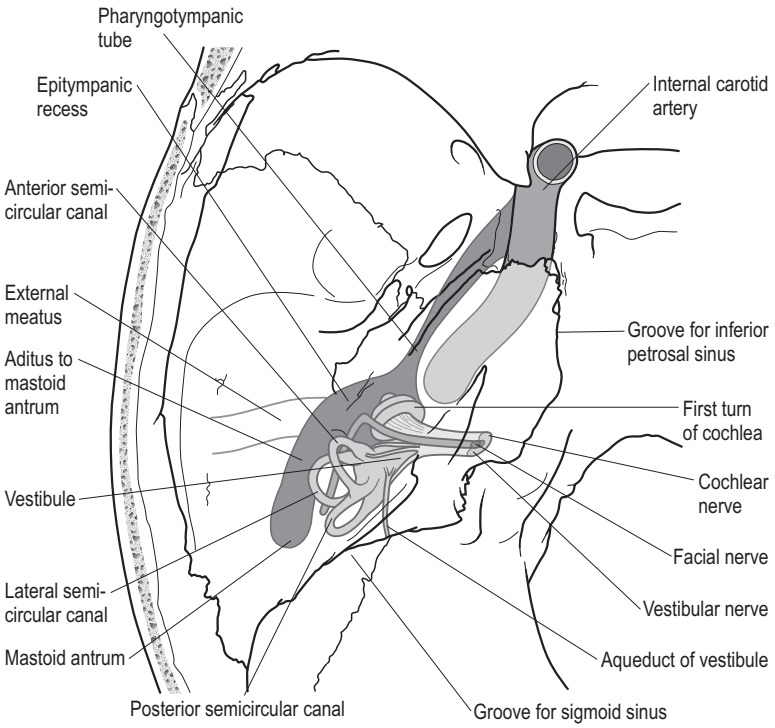
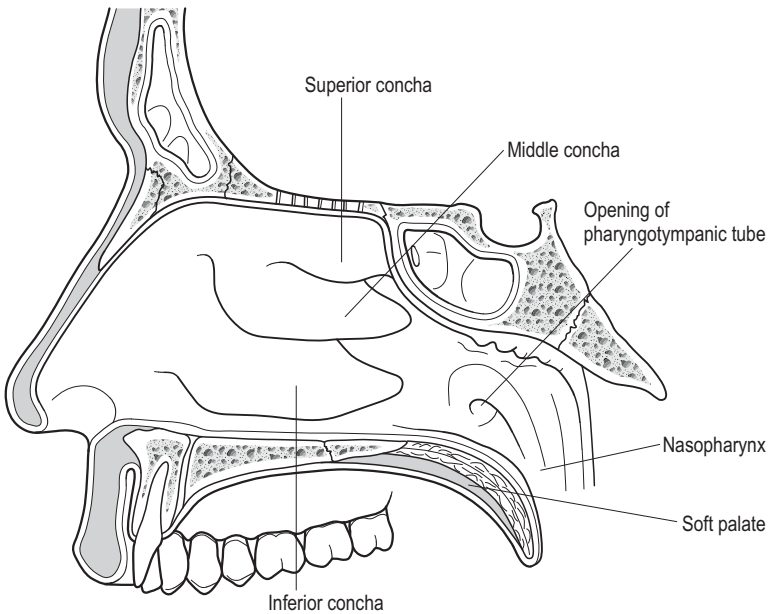


Figure 7.3.3. *Pharyngotympanic tube.*

Figure 7.3.4. *Opening of the pharyngotympanic tube in the nasopharynx.*



connecting the lung to the nasopharynx.[8] The mucosal lining of the PT contains mucus-producing cells, ciliated cells,[24] plasma cells and mast cells. Infants and children demonstrate an increase in the density and size of the folds in the tissues lining the PT and it has been suggested that this plays a role in protecting the middle ear.[25] The gas exchange through the submucosal connective tissue seems to be accelerated when the submucosal vasculature dilates and blood flow is augmented due to middle ear inflammation. Alternatively, gas exchange can be diminished when the mucosa thickens and submucosal tissue proliferates due to extended inflammation.[26]

In normal tubal function at rest, the PT is usually collapsed, fulfilling its protective role against retrograde infection from the nasopharynx.[27] The tensor veli palatini (TVP), the dilatator tubae (DT), the levator veli palatini (LVP) and the salpingopharyngeus muscles are all attached to the PT. The TVP arises from the scaphoid fossa, from the spina angularis of the sphenoid and from the lateral wall of the cartilage of the auditory tube. It then descends vertically, becomes tendinous and inserts onto the pterygoid hamulus, the lower extremity of the medial pterygoid plate of the sphenoid, and medially onto the posterior border of the hard palate to form part of the palatine aponeurosis. The DT is attached above to the PT, particularly to its membranous portion. It intermingles below with the TVP and rounds the pterygoid hamulus. Most authors agree that contraction of the TVP opens the PT lumen and therefore ventilates the middle ear;[24,28] this action is particularly significant for the fibers of the DT.[29]

The LVP arises from the medial lamina of the cartilaginous PT and from under the apex of the petrous portion of the temporal bone. It extends into the palatine velum, its fibers broadening to the middle line, where they blend with those of the opposite side. The salpingopharyngeus arises from the inferior part of the PT, is directed downward and blends with the posterior fasciculus of the pharyngopalatinus (Fig. 7.3.5). It contributes to PT opening during deglutition.

Functionally, the PT allows air to enter or exit the tympanic cavity during activities that normally

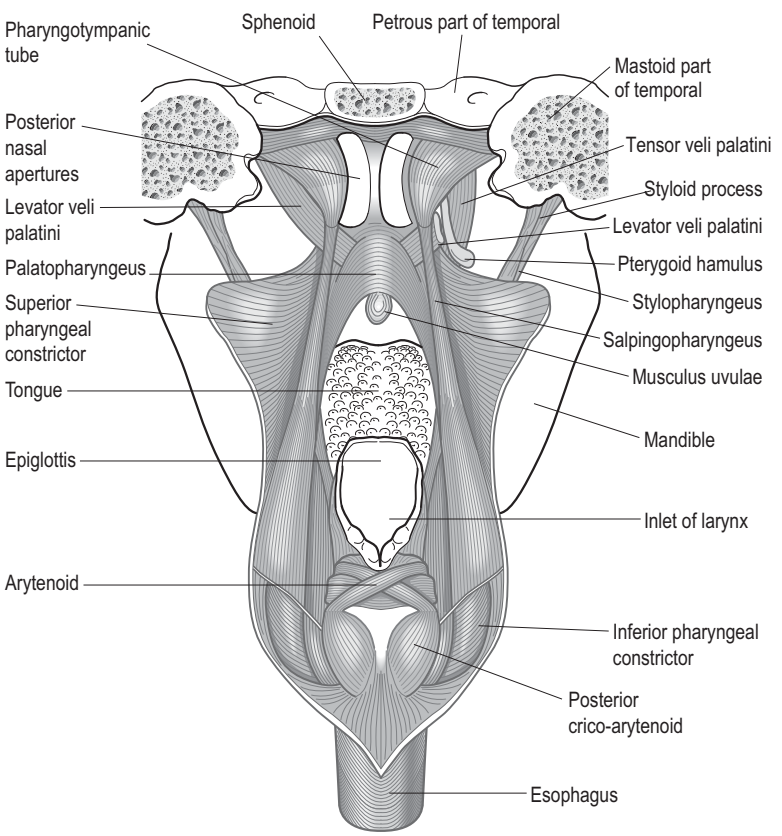


Figure 7.3.5. *Muscles of the palate.*

open it – for example, swallowing, crying or yawning. This balances the pressure gradient between the atmosphere and the tympanic cavity. When the mechanism of swallowing is dysfunctional, opening of the PT is not efficient. Sustained collapse follows with the development of negative middle ear pressure and retraction of the tympanic membrane. The subsequent potential for aspiration of nasopharyngeal secretions into the middle ear may result in OM. Furthermore, dysfunctional swallowing may also cause gastroesophageal reflux, another risk factor for OM. In addition, if present, the frequent use of a pacifier may encourage infantile deglutition, i.e. forward tongue thrust when swallowing. Although the use of a pacifier does not increase the incidence of respiratory infections, there is evidence that constant use affects the occurrence of AOM, possibly because of alteration in the pressure equilibrating function of the PT.[12]

Dysfunction of the PT may result in negative middle ear pressure that, in turn, impairs auditory sound conduction to the cochlea of the internal ear affecting hearing. PT dysfunction can also affect hearing through its impact on the tensor tympani muscle. The tensor tympani muscle is continuous with the TVP. It arises from the cartilaginous portion of the PT and the adjoining sphenoid, and inserts on the manubrium of the malleus.[24,29] Tensor tympani contraction draws the malleus medially, increasing tympanic membrane tension while pushing the incus and stapes medially against the fenestra vestibuli. This results in an increased intravestibular pressure that, under normal circumstances, serves to dampen violent noises. Consequently, dysfunction of the PT may be associated with both negative middle ear pressure and spasm of the tensor tympani with resultant disturbance of hearing.

Anatomic developmental delays such as immaturities of the PT and surrounding structures or of the neuromuscular system may result in dysfunctional opening of the PT in infants and children.[30] Most of the time, the dysfunctional opening of the PT improves with age as the base of the neurocranium and the viscerocranium develop. This too, however, can contribute to the complex interaction of phenomena that predispose the child to develop OM.

The base of the skull (Figs 7.3.6, 7.3.7) goes through significant developmental changes during the first years of life. Two critical phenomena participate in this development. First, the diverse stimulation produced by normal orofacial functions, such as suckling and swallowing, spurs the growth of structures, particularly the pterygoid processes in which the involved muscles insert. Secondly, but concomitant with the above, the progressive flexion of the cranial base, associated with the anteroposterior growth of the skull, contributes to positional changes of both the pterygoid processes, which become longer and more vertical, and the petrous portions of the temporal bones, which become externally rotated. Additionally, several changes occur in the viscerocranium, such as the increase in

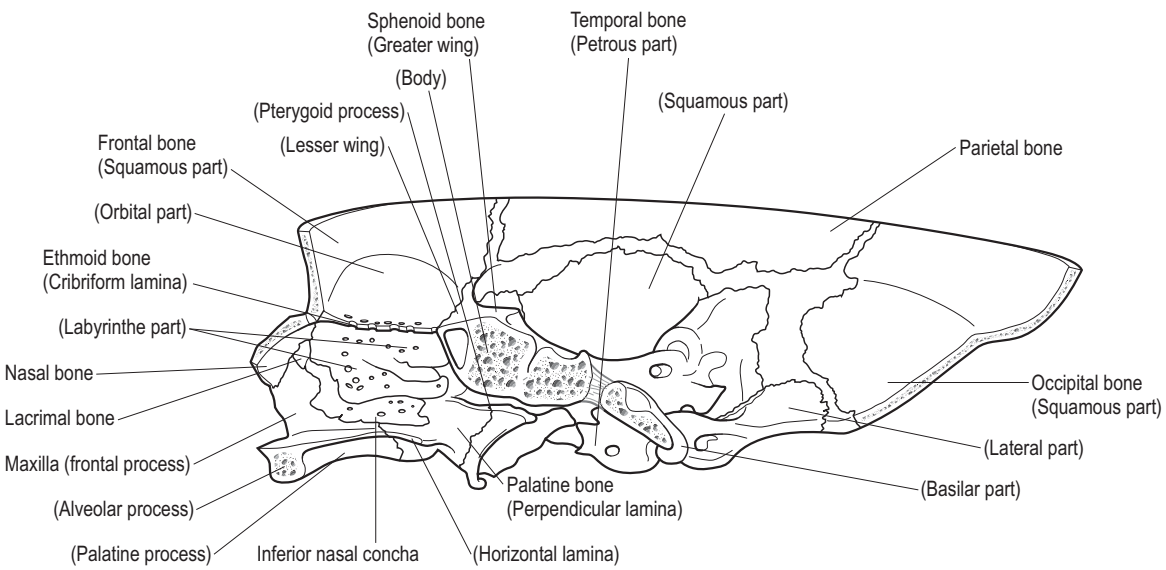


Figure 7.3.6. *Sagittal section of the cranial base at birth.*

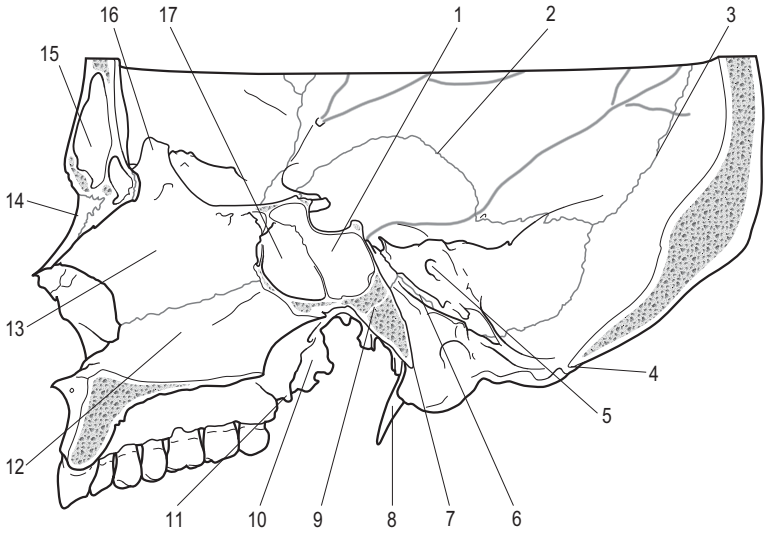


Figure 7.3.7. *Sagittal section of the cranial base in the adult. 1. Right sphenoidal sinus; 2. squamosal suture; 3. lambdoid suture; 4. posterior margin of foramen magnum; 5. internal acoustic meatus; 6. petro-occipital suture; 7. anterior margin of foramen magnum; 8. styloid process; 9. line of sphenobasilar junction; 10. lateral pterygoid plate; 11. pterygoid hamulus; 12. vomer; 13. perpendicular plate of ethmoid; 14. nasal bone; 15. frontal sinus; 16. crista galli; 17. left sphenoidal sinus.*

height of the vomer that accompanies the expansion of the nasal cavity. These developmental changes are concomitant with the development of the PT and its associated muscles. Therefore, any structural imbalance that develops in association with the development of the base of the skull or viscerocranium may adversely affect the ventilation and drainage of the ear.

A dysfunctional tongue posture may affect the tongue's pumping function on the palatine aponeurosis and, therefore, the associated PT ventilation that occurs during swallowing. Tongue posture adapts to oral development and PT function appears to be diminished in long-faced adenoidal children.[31] Additionally, children with significant overbites are found to be more predisposed to develop PT dysfunction.[32]

Body position also seems to influence PT opening. Evidence shows a correlation between the lateral recumbent position, where one ear is positioned downwards, and a lower PT opening function on that side.[20] This reinforces the need to avoid the repeated use of the same sleep position for infants.

Any cranial somatic dysfunction of the base of the skull may disrupt its developmental sequence as well as affecting tongue posture, with resultant impact on PT growth and function. This may be a cranial somatic dysfunction of the bony constituents of the cranial base, the occiput, sphenoid and temporal bones, or dysfunction of any component linked to the vascular supply and innervation of the myofascial structures involved in suckling and swallowing. The TVP and the tensor tympani are innervated by branches from the otic ganglion, located immediately below the foramen ovale, on the medial surface of the mandibular nerve (CN V_3). The glossopharyngeal nerve (CN IX) innervates the stylopharyngeus, and the accessory nerve (CN XI) innervates the palatal muscles through the pharyngeal plexus. They both exit the skull through the jugular foramen. The hypoglossal nerve (CN XII) innervates the intrinsic and extrinsic tongue muscles. It exits the skull through the anterior intraoccipital synchondrosis, the site of the hypoglossal canal of the occipital bone when ossification occurs. Consequently, efficient PT function requires equilibrium among surrounding bony structures, such as the temporal bones, occiput, sphenoid and mandible. It also requires that associated myofascial structures be free of dysfunction.

A dysfunctional PT creates a terrain where other risk factors are reinforced. Recurrent bilateral OM with effusion develops when poor PT function is allied with diminished immune status.[33] The allergic inflammatory response that often occurs in the nasopharynx also occurs in the middle ear,[34] and the prevalence of allergic rhinitis is significantly higher in children with OM with effusion than in healthy children.[35,36] The allergic inflammation in atopic children is not localized in one area only, but manifests itself in the middle ear on both sides, as well as in the nasopharynx, demonstrating totally the united airways concept.[37] Mucosal inflammation with release of histamine and other mediators following nasopharyngeal exposure to an allergen may, consequently, be responsible for PT obstruction and

dysfunction. Chronic allergic inflammation of the upper airway may lead to lymphoid hypertrophy with increased size of adenoidal and tonsillar tissue.[38] In such a case, with edema and inflammation of the posterior nasopharynx, the enlarged adenoids may obstruct the pharyngeal ostium of the PT. Tubal tonsil hypertrophy is a possible etiology for OM, when recurrence appears after adenoidectomy.[39]

The PT mucociliary apparatus contains components that have an important role in eliminating middle ear debris by moving it toward the nasopharyngeal orifice. Additionally, specialized epithelial cells express and secrete surface-active materials that appear to facilitate the muscular action of the PT opening and to protect the middle ear against infections.[3] Conversely, many viruses impair the mucociliary function of the PT epithelium and perturb the nasopharyngeal bacterial flora, increasing the adherence of bacteria to the epithelial cells.[40] Bacteria and respiratory viruses (e.g. the respiratory syncytial virus or influenza viruses) are common causes of middle ear infection.[40–42] Because of the connection between the upper and lower airways, the pathophysiologic site of origin is frequently the nasal pathway. Babies born in the fall begin their lives during the peak seasons for viral exposure and the development of respiratory infections, a risk factor for OM.[13] Additionally, impaired or decreased nasal mucociliary activity may also cause PT mucociliary dysfunction.[43] Thus osteopathic procedures that facilitate the clearance of secretions and the mucociliary action of the PT and upper airways are indicated. Blood flow to the region should also be improved.

Gastroesophageal reflux may also predispose to bacterial infection.[44] Possibly because of reflux, infants fed in the supine position demonstrate abnormal postfeeding tympanographic results compared to infants fed in the semi-upright position.[45]

Signs of AOM include fever, insomnia and the presence of pus in the middle ear with a tympanic membrane that appears bulging and erythematous when observed by otoscopic examination. Ear infections may be painful, causing irritability, rubbing of the affected ear, loss of balance and impaired hearing with lack of response to moderate sounds. Although it is generally thought that OM causes permanent hearing loss, this has not been demonstrated.[46] Transient mild to moderate hearing loss associated with OM has, however, been shown to cause delays in communicative development.[47]

Physical examination and treatment

Osteopathic considerations for the treatment of OM are directed at augmenting the body's defenses against infection and its recuperative power after infection is present. Mainstream medical interventions are often fraught with controversy. Because the infectious agents responsible for OM are both viral and bacterial, antibiotic therapy, although appropriate for bacterial infection, is not universally effective. Guidelines for determining when to employ antibiotics and other modalities are available.[2,4,48,49] The use of tympanostomy tubes is controversial.[50,51] Consequently, non-toxic interventions like osteopathic treatment, which appear to reduce the need for antibiotics, have been shown to be of potential benefit as adjuvant therapy for children with recurrent AOM.[52]

Diagnosis should begin with observation. Start with an overall evaluation of the child's posture. Look at the pectoral girdle, often protracted in patients with ENT infections. Observe the cervicothoracic junction, the cervical spine and its relationship to the skull for lack of mobility and vertical compression. These patients may demonstrate a shrugged shoulder posture, with the appearance of a shortened neck.

Observe the auricles of the ear bilaterally for deformity, asymmetry of position and relative external or internal rotation. The appearance of the ear follows the temporal bone which, in turn, affects the function of the PT. Examine the parietomastoid and occipitomastoid sutures bilaterally. Look for flattening or compression of the area. Because ear position reflects temporal bone position, asymmetry of the ears is often associated with asymmetry of cranial shape. Non-synostotic plagiocephaly has been shown to be associated with an increased incidence of OM. Enquire if the child repeatedly pulls at one ear. This will often occur on the side of compression of the parietomastoid and occipitomastoid suture.

Study the face. Open mouth facies are indicative of mouth breathing and nasal obstruction, predisposing to OM. Diagnosis and treatment of dysfunction in this area is discussed in Part 7.4, 'Mouth breathing'.

Next, perform a palpatory examination. Begin by evaluating the upper thoracic spine, ribs and pectoral girdle for somatic dysfunction. The viscerosomatic reflexes from the upper respiratory tract, including the ear, are to be found at level of T1–T4. Somatic dysfunction in this area results in increased sympathetic tone with vasoconstriction affecting the ears, nose and throat through somatovisceral reflexes. Mechanical dysfunction of the upper thoracic spine (T1 and T2), associated ribs, sternum and clavicles impairs lymphatic drainage from the head and neck. Further evaluate the remainder of

the thoracic cage and thoracoabdominal diaphragm which, when dysfunctional, can also impair lymphatic circulation. Utilizing indirect principles, treat any dysfunction identified in the above areas.

Examine the cervical region for somatic dysfunction. Pay particular attention to the occipitoatlantal and atlantoaxial articulations, to the myofascial structures for their relation to lymphatic nodes and vasculature, and to the SCM muscles that, when dysfunctional, impact the function of the temporal bone. Treat any identified dysfunction.

Evaluate the skull. Begin by assessing the cranial base, paying attention to the sphenobasilar synchondrosis and temporal bones. The articulations of the temporal bones should be examined. The occipitomastoid sutures are important for their impact on the contents of the jugular foramen: cranial nerves IX, X and XI. The petrobasilar suture and sphenopetrosal synchondrosis are often dysfunctional in the infant's skull. Dysfunction of these articulations may affect the petrous portion of the temporal bone containing the osseous part of the PT. Furthermore, the cartilaginous portion of the PT is located beneath the sphenopetrosal synchondrosis and free motion of the petrous portion of the temporal bone, in external and internal rotation, facilitates the clearance of secretion from the PT. Next, evaluate the temporal bones for intraosseous dysfunctions between the petrous, squamous and tympanic portions. Palpate for the cranial rhythmic impulse (CRI) at the level of the mastoid. Intraosseous mastoid cranial respiration may promote mastoid cell function. Examine the relationship between the mandible and the temporal bones. There is usually tenderness in the area. Any temporomandibular dysfunction can affect the mobility of the temporal bones and the myofascial structures of the anterior neck below. The PT is commonly cleared by the actions of swallowing and yawning. These actions can be impaired by dysfunction of the mandible and its relationship to the tongue and soft palate. Treat identified dysfunction.

Specific attention should be paid to the efficient clearance of secretions from the PT and mastoid cavities. This activity may be stimulated by the mastoid pump procedure and the Galbreath technique.

When possible, the rate and amplitude of the CRI should be monitored during the above procedures. Following the CRI during the mastoid pump enhances the efficacy of the procedure. The specific treatment of cranial dysfunctional patterns will augment the amplitude of the CRI, improving fluid mobility and affecting low frequency oscillations in autonomic nervous system (ANS) physiology.

Advice to caregivers

Counsel the caregivers to maintain a healthy lifestyle for the child. Maintain a regular sleep–wake cycle. Provide a balanced diet with adequate hydration and avoiding refined carbohydrate as much as possible. Bottle feed and nurse in a semi-upright position and never put the infant to bed with a bottle. As much as possible, limit pacifier use to moments when the infant falls asleep and try to eliminate its use after the age of 6 months. Avoid exposure to passive smoke. When bathing the infant, limit the amount of water entering their ears.

Caregivers should be instructed to lay the child on their side, with the problem ear up. They should then massage the mandibular region, applying gentle skin traction from the area anterior to the ear in the direction of the chin. This tends to open the PT and the position employs gravity to facilitate drainage. They can also gently caress around the ear, particularly over the mastoid region. These actions allow the caregiver to actively participate in the child's recovery. They sensitize the caregiver to the health status of the child and promote relaxation for the child. Encourage the caregiver to play with the child in a fashion that promotes mimicry of the production of sounds in the throat and the clicking of the tongue by pulling it quickly from the hard palate. All activities encouraging action of the myofascial structures connected to the PT will tend to open it and facilitate its drainage.

RHINITIS

Rhinitis is the inflammation of the nasal mucous membranes. Acute rhinitis may be the consequence of a viral infection, whereas allergic rhinitis is caused by an immune-mediated response to any one or more of a myriad of allergens. Other classifications include atrophic rhinitis and vasomotor rhinitis. Although these conditions are the result of differing etiologies, they are all affected by the presence of somatic dysfunction. It is an established osteopathic dictum that the body possesses the inherent ability to heal itself. The presence of somatic dysfunction can predispose the individual to develop rhinitis or interfere with the body's recuperative mechanisms. Knowledge of the anatomy and physiology of the nasal cavities and the mucosa lining their walls is absolutely necessary to understand the etiologies of nasal dysfunction and how osteopathic principles may be applied to promote health in this area.

The nose is divided by the nasal septum into two cavities, or fossae. The two nasal cavities open

anteriorly by way of the anterior naris, or nostril. They are continuous posteriorly by way of the posterior nasal apertures, or choanae, into the nasopharynx.

The nasal septum represents the medial wall of each nasal cavity. It is formed by the perpendicular plate of the ethmoid, the vomer and the septal cartilage (Fig. 7.3.8).

The roof of the nasal cavities is formed anteriorly by the nasal spine of the frontal bone and the two nasal bones. The cribriform plate of the ethmoid, with numerous perforations for the olfactory nerves, is located behind the nasal bones. More posteriorly, the anterior aspect of the body of the sphenoid causes the roof of the nasal cavities to slope downward. The sphenoidal sinuses open into the nasal cavities from above, on each side of the nasal septum (Fig. 7.3.9).

The floor of the nasal cavities is the upper surface of the osseous palate. The maxillary palatine processes form the anterior two-thirds, while the palatine horizontal plates form the posterior one-third (Fig. 7.3.10).

The lateral walls of the nasal cavities demonstrate numerous structures. They are formed anteriorly by the maxilla, posteriorly by the palatine bone and superiorly by the ethmoid labyrinth and lacrimal bone. The inferior, middle and superior nasal conchae (turbinates), the most central portion of this lateral wall, by virtue of their curled shape add a great amount of surface area to the nasal cavities. The space below each turbinate is referred to as a meatus (Fig. 7.3.11).

The nasal vestibules, just inside the nares, are the anterior-most aspect of the nasal cavities. The nares and vestibules are bounded laterally by the alar and lateral cartilages, and medially by the cartilaginous septum and the connective tissue septum, the columella (Fig. 7.3.12). The vestibule is lined with skin that contains sebaceous and sweat glands and coarse hairs (vibrissae) that assist in air filtration.

The nasal cavities are completely covered with a lining that varies histologically in different areas. At the anterior part of the nasal cavities, in the vestibules, the lining is continuous with the facial skin. Above, at the level of the upper border of the alar cartilages, the limen nasi defines the beginning of a lining formed by a non-keratinizing stratified squamous transitional epithelium that evolves further into a pseudostratified ciliated epithelium, the respiratory mucosa. This mucosa covers the remaining surface of the nasal cavities, except for the olfactory area that is covered with olfactory epithelium. This mucosa is also present in many other parts of the upper respiratory tract.

Several additional cavities communicate with the nasal cavities and demonstrate a continuum of the nasal respiratory mucosa. Each of the nasal cavities communicates directly with the nasopharynx

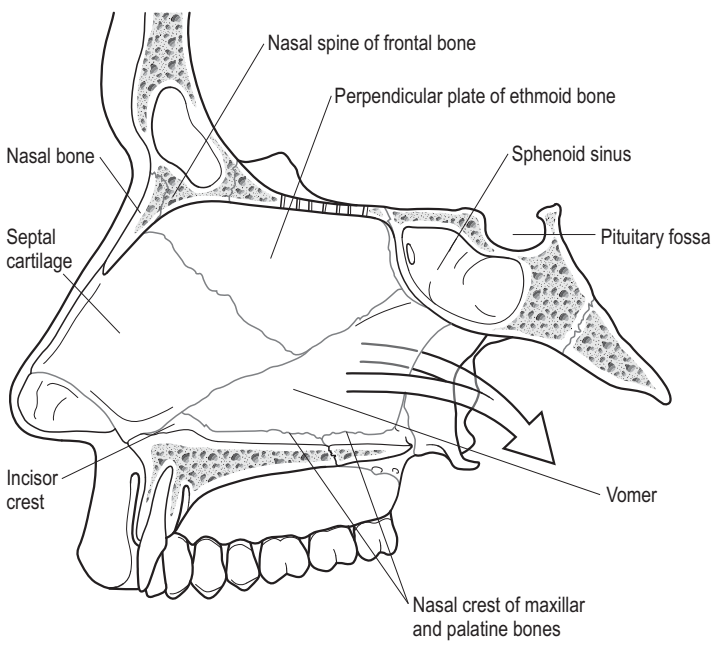


Figure 7.3.8. *The nasal septum, medial wall of the nasal cavity.*

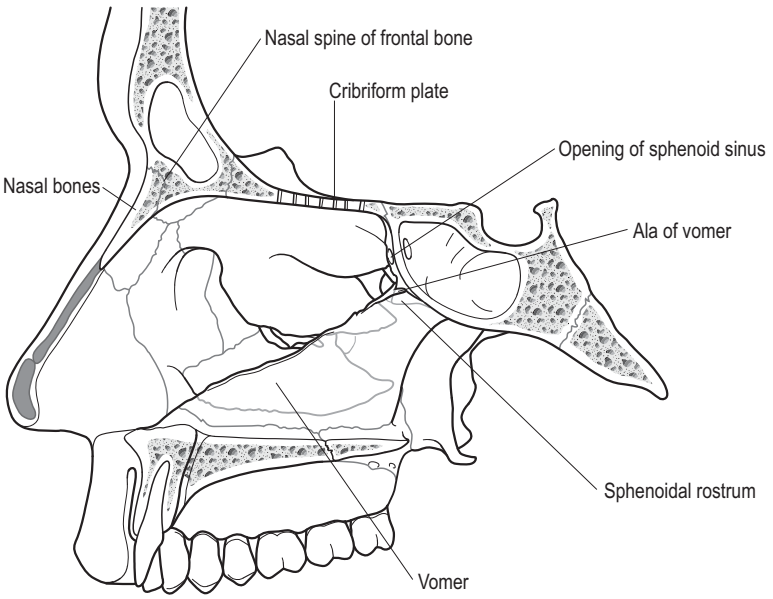


Figure 7.3.9. *The roof of the nasal cavity.*

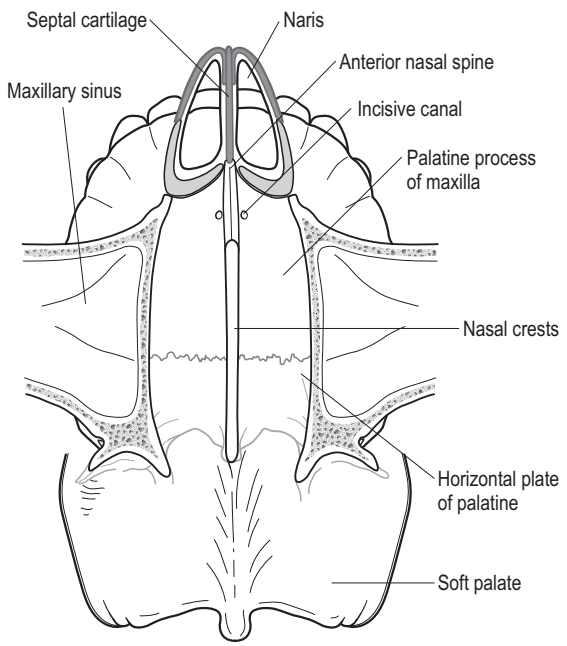


Figure 7.3.10. *The floor of the nasal cavity.*

through their posterior aperture or choana (Fig. 7.3.13). The nasopharynx communicates through the PTs with the tympanic cavities. The paranasal sinuses, i.e. maxillary, sphenoidal, frontal and ethmoidal, are also lined with ciliated and mucus-secreting respiratory mucosa and open into the nasal cavities. The nasopharyngeal mucosa is continuous above with the conjunctiva of the eyes through the nasolacrimal ducts.

Below, the nasopharynx continues to become the oropharynx, the laryngopharynx and the esophagus. The mucous membrane of the pharynx is continuous with that lining the mouth and larynx, as well as, through the trachea and bronchi, into the lungs. This continuum is a perfect example of the interrelationship between the different structures of the human body and exemplifies the concept of the body as a unit.

The respiratory mucosa plays a significant part in the physiology of the nose, as well as in its pathologies, as is the case in rhinitis. The mucosa acts as a selective barrier, essential for the defense of the airways against inhaled pathogens. The respiratory pseudostratified ciliated epithelium is formed by ciliated columnar or cuboidal epithelia with goblets cells, non-ciliated columnar cells and basal cells. Mast cells and migrating lymphocytes, mainly T cells, are also present. Under the basal lamina of this epithelium, the submucosa is adherent to the periosteum of the adjacent cranial bones and includes a fibrous layer with diffuse lymphoid tissue and a layer of mucous, seromucous and serous glands. An abundant mucous film is produced by these glands and by the goblets cells. Additional plasma exudation may occur, particularly in the presence of inflammatory states. This film gathers the particles and debris from the air that is inspired to sweep them away. Almost

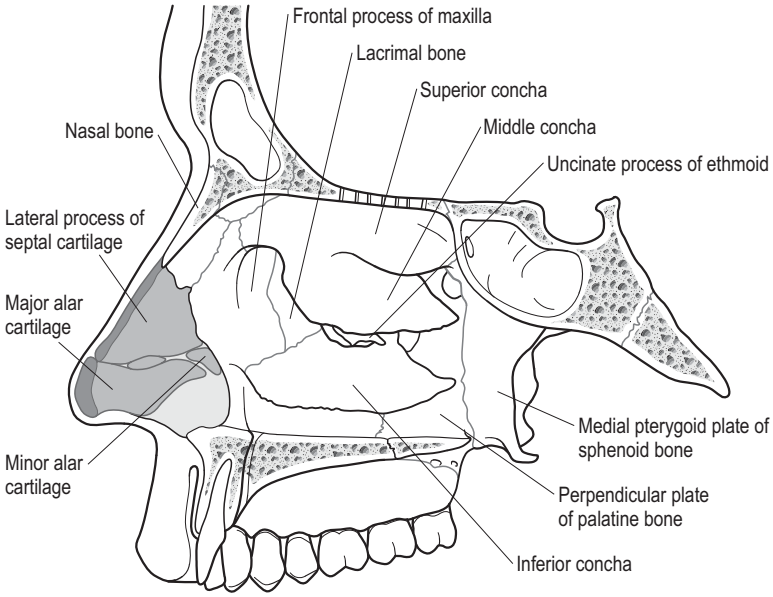


Figure 7.3.11. *The lateral wall of the nasal cavity.*

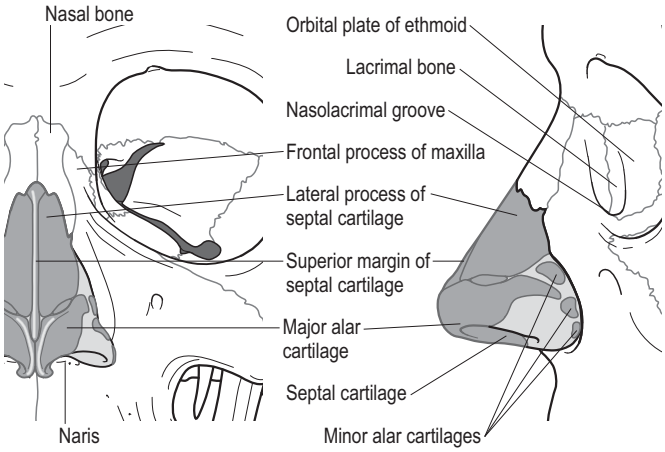


Figure 7.3.12. *The external nose.*

all particles greater than 5 μm and about 50% of those between 2 and 5 μm are collected. They end up either in the nasopharynx and oropharynx to be periodically swallowed, or in the anterior nasal vestibules.

Mucociliary clearance depends on the beating function of the respiratory cilia. They beat about 1000 times per minute.[53] The frequency of the mucociliary transport rate is subject to various influences, such as mucus viscoelastic properties, airway epithelia alkalization that appears to be a stimulator or airway epithelia acidification that decreases the rate.[54] Healthy function of the respiratory cilia results in constant motion of the mucous film. The nasal ciliary beating propels the mucus secretions posteriorly in the direction of the nasopharynx. Conversely, dysfunction of the drainage of the nasal respiratory epithelium leads to stasis and the accumulation of secretions within the nasal cavities. Cranial somatic dysfunction – particularly of the frontal bones, sphenoid, ethmoid, maxillae and vomer, with resultant loss of their inherent motility – is a possible cause of mucociliary stasis. Septal deviations are known to influence the dynamics of the nasal cavity and are often associated with a 'stuffy' nose. Histologic studies confirm this observation.

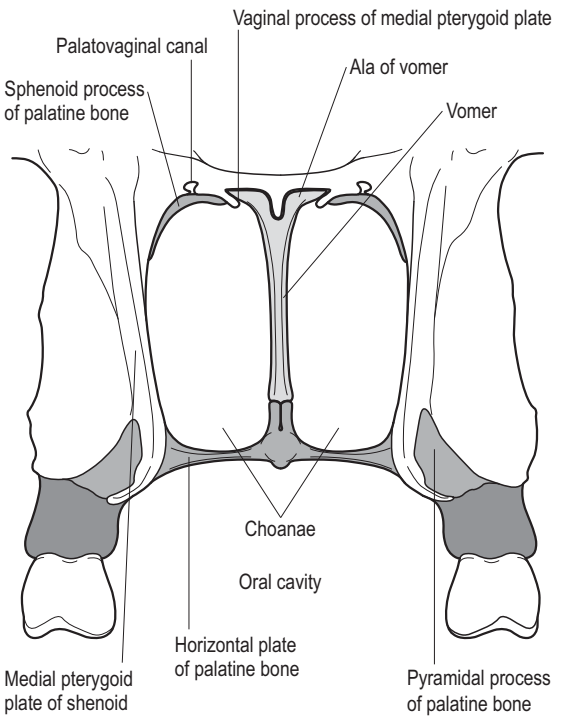


Figure 7.3.13. *The posterior nasal apertures or choana.*

Loss of cilia, increased inflammation and decreased density of the glandular acini are reported affecting the mucosa on the concave side of the septal deviation.[55]

The vasculature of the respiratory epithelium contributes significantly to the function and dysfunction of the nasal cavities. The nasal mucosa contains a profuse subepithelial capillary network that supplies nutrients and water that through its evaporation contributes to the conditioning of inspired air. The vascular supply also includes a number of different capacitance vessels, i.e. the veins, venules and cavernous sinusoids that modulate blood flow. Constriction and relaxation of these sinusoid vessels produce shrinkage or swelling of the mucosal surfaces that consequently regulates airflow and alters nasal patency. Most of the venous cavernous sinusoids are located on the inferior conchae, major sites for nasal congestion.

Air conditioning is a major function of the nose. On inspiration, the air contacts the nasal mucosa and is brought to the appropriately conditioned temperature and humidity. By virtue of their contour, the conchae, located on the lateral walls of the nasal cavities, provide an increased surface area for the flowing air to be in contact with the nasal mucosa. During expiration, some of the heat may be returned to the mucosa. When an individual is in a setting of 23°C, the nasal cavities warm the inspired air to 33°C.[56] The air is also humidified and this allows gas exchange within the alveoli of the lungs that takes place at 37°C and 100% relative humidity.[57] Therefore, nasal air conditioning requires large amounts of heat and water for conditioning the inspired air and the capacitance vessels seem perfectly designed to fulfill that need. They might also operate as a short-term reservoir, either for heat or for water. Special conditions (e.g. hyperventilation) call for these reserves in order to provide cooling and evaporation.[58] Under normal circumstances and normal vasculature, a healthy nose succeeds in warming and humidifying the inspired air in order to protect the lungs. Conversely, paucity in blood supply or moistening may reduce the efficiency of the air conditioning system of the nasal cavity.[56]

The nasal vasculature also contains an extensive system of arteriovenous anastomoses. This allows for the rapid passage of blood through the mucosa without reducing the nasal patency. Great amounts of arterial blood may flow through these anastomoses, providing heat exchange similar to hot water in a radiator.[53]

Brain cooling appears to be the result of several mechanisms including a possible nasal and paranasal convection process. This latter process involves the transfer of cool venous blood from the respiratory mucosa to venous structures of the brain, such as the superior sagittal sinus between the parietal bones or the cavernous sinuses on each side of the body of the sphenoid, where arterial thermoregulation may then occur. On both sides, the cavernous sinus drains venous blood from the skin of the face and from the nose and mouth areas, and is in intimate contact with the internal carotid artery.[59] The direction of flow in these sinuses is reversible.[17] Countercurrent mechanisms are suggested, where the arteriovenous anastomoses, present in the nasal vasculature, may also participate, allowing enhanced thermoregulation and brain cooling in hot conditions.[60] Changes in craniofacial morphology have been observed as adaptations to weather conditions. Wider nasal cavities and larger paranasal sinuses are considered to be adaptive mechanisms that, under hot conditions, offer more evaporating surface and consequently greater cooling capacity, thus protecting the brain.[61] Orofacial dysfunction may alter nasal breathing and consequently the above functions.

Body position also affects the nasal vasculature. The supine position increases vascular congestion, thus decreasing nasal patency and the ability of the

nose to condition cold, dry air. Conversely, the upright position decreases vascular congestion.[62] Consequently, it is appropriate to enquire if the patient experiences excessive increased nasal congestion when they lie down as it may result in snoring and sleep disorders.

Furthermore, the nasal vascular supply is under the influence of hormones, psychological stress and diverse substances (e.g. gases or inflammatory molecules) that once in contact with the nasal mucosa seem to produce vascular congestion with edema and plasma exudation. It should be noted that children and teenagers often report nasal vascular congestion as nasal obstruction.

An alternation of breathing between the two nares is known as the nasal cycle. It has been observed as early as 3 years of age, with the duration of a cycle ranging from 40 to 215 minutes.[63] Alternation of the side of nasal breathing has been associated with the central mechanism regulating the dominance of the cerebral hemispheres. Increased sympathetic activity in the nasal mucosa appears to be linked to greater sympathetic tone in the ipsilateral hemisphere and thus with decreased blood flow and mental activity in that hemisphere.[64]

Changes in the tone of the nasal vascular supply are regulated by the ANS. Parasympathetic nerves are vasodilator, sympathetic nerves are vasoconstrictor. Therefore, predominance of parasympathetic activity causes a vasodilatation and nasal congestion, whereas increased sympathetic activity produces a vasoconstriction that decreases nasal airflow resistance.

The preganglionic fibers of the cranial portion of the sympathetic nervous system originate from axons of somata in the lateral gray column of the upper thoracic spinal segments. The fibers enter the superior cervical ganglion adjacent to the second and third cervical vertebrae where they synapse. The postganglionic fibers ascend, following the course of the internal carotid artery, forming the internal carotid plexus.

The greater petrosal nerve, a branch of the facial nerve (CN VII), contains the preganglionic parasympathetic fibers traveling to the pterygopalatine (sphenopalatine) ganglion. Located deeply in the pterygopalatine fossa, between the pterygoid process and maxilla, anterior to the pterygoid canal, the pterygopalatine ganglion is one of the major peripheral parasympathetic ganglia. At the level of the foramen lacerum, the greater petrosal nerve is joined by the deep petrosal nerve from the internal carotid plexus (sympathetic) to form the nerve of the pterygoid canal (Vidian nerve). These fibers synapse in the pterygopalatine ganglion; the postganglionic parasympathetic fibers are secretomotor and supply the glands of the nasal mucosa.

Additionally, the nasal cavities are densely innervated by the sensory nervous system. Nerves are present in respiratory mucosa, particularly in the walls of the venous vessels and the gland acini. Glands are innervated by both parasympathetic and sensory nerve fibers. Sensory nerves are stimulated by mechanical, thermal or chemical stimulation and afferent fibers run in the trigeminal nerve. Sensory nerve stimulation instigates different reflexes, such as the sneeze reflex.[65] Nasal thermal stimulation, as occurs with inhalation of cold dry, dry or moist air, produces a nasopulmonary bronchoconstrictor reflex in normal healthy individuals, inducing changes in airway resistance.[66] Activation of temperature-sensitive nerve endings in the nasal mucosa generates this response and the decrease of airflow through the nose and trachea protect the lungs from insufficiently conditioned air.[66]

The ANS controls several aspects of nasal function, i.e. nasal secretions, mucociliary function, blood flow, microvascular permeability, release of inflammatory cells and nasal patency. The modulation and balance of nasal functions necessitate an interaction between the sympathetic and parasympathetic systems, as well as a well-tuned sensory nervous system. Dysfunction may lead to pathologic nasal syndromes. Because of the relationships between the sympathetic nervous system and the upper thoracic spinal segments, the second and third cervical vertebrae, and between the parasympathetic nervous system and the sphenoid, maxilla or palatine bones, somatic dysfunction of any of these vertebral and cranial areas can result in dysfunction of the ANS with impact on nasal function. Furthermore, because of the role of the trigeminal nerve, particularly the first and second divisions, in the sensory function of the nose, the temporal bone should be added to that list. Osteopathic procedures may be applied to balance the ANS and promote healthy nasal functions.

Unpaired and paired structures form the nasal cavities, as in the remainder of the skull. As such, during the PRM inspiratory phase, the midline unpaired structures of the nasal cavities (i.e. the sphenoid, ethmoid, vomer and septal cartilage) demonstrate cranial flexion and the paired structures (i.e. maxillae, palatine, nasal and lacrimal bones and conchae) externally rotate. In the reciprocal PRM expiratory phase, the midline structures move in the direction of cranial extension and the paired structures internally rotate. Therefore, in health, the

nasal cavities follow each cycle of the PRM, with a resultant widening of the cavities during flexion–external rotation of the inspiratory phase and narrowing during extension–internal rotation of the expiratory phase. Cranial somatic dysfunction very frequently follows asymmetric patterns. Thus, the nasal cavity will be wider on one side than on the other. This can be observed by nasoscopic examination, as well as by simply looking at the patient to note asymmetry in the facial features. It may also be observed by comparing the relative size of the nares. One side is usually more open than the other. The open side is the side of the external rotation, whereas the other side is associated with internal rotation. The patient often reports more nasal congestion on the smaller side and in cases of small children the mothers comment on the increase of nasal secretion on that side.

The alternation of cranial flexion and extension, with all cranial structures free to follow this movement, is necessary to ensure effective tissue perfusion of the nasal mucosa. It also promotes venous and lymphatic drainage of the nose, as well as the removal of secretions from the nasal cavities and sinuses. Under these circumstances, mucosal inflammation and hyperreactivity associated with rhinitis may be reduced.

In neurogenic inflammation of the upper airway mucosa, such as in chronic rhinosinusitis, sensory nerves are excited and mediators are released, including histamine, prostaglandins and various neuropeptides such as substance P.[67] They may then cause vasodilatation, vascular congestion, extravasation of plasma with edema, and recruitment and activation of inflammatory cells. Secretion from the submucosal glands may also be increased. These exaggerated sensory and parasympathetic defensive reflexes form the pathophysiologic basis of rhinitis.

Acute rhinitis, one of the symptoms of the common cold, is the result of a viral infection. Numerous viruses cause infections in the respiratory tract and any region of the tract may be inflamed – the nose, the paranasal sinuses, the throat, the larynx, the trachea and the bronchi. Acute rhinitis represents one of the most frequent upper respiratory infections.

Allergic rhinitis is considered to be the most common allergic airway disease, with about 10% of the population experiencing this condition.[53] Allergic rhinitis is common in children and most of the time this condition first develops during childhood or adolescence.[68] Typical behaviors are usually observed such as grimacing and picking of the nose. Older children are likely to blow their noses more often than younger children who present with constant clearing of the throat because of postnasal drip, frequent sniffing or snorting.[69]

Rhinorrhea, nasal obstruction, sneezing, itching of the eyes, nose and palate, and watery eyes are typical symptoms associated with allergic rhinitis. The disease results from exposure to various allergens including foods, pollens, molds, dust mites and animal dander. Two groups are described: seasonal allergic rhinitis, often the result of pollen exposure, and perennial allergic rhinitis, lasting for at least 9 months of the year.

Allergic individuals demonstrate a decreased capacity to warm and humidify inhaled air.[57] They also are prone to develop other diseases of the upper and lower respiratory tract such as sinusitis, otitis media with effusion and asthma that may complicate allergic rhinitis.[70] Although the reason why individuals develop allergic rhinitis is uncertain, a genetic predisposition to develop the allergic response has been suggested.[69] It is thought that these individuals probably have a greater sensitivity to allergens and are predisposed to develop mucosal inflammation and hyperreactivity.

A 'microflora hypothesis' has also been suggested. It is thought that the disturbance of the normal microbiota in the gastrointestinal tract, in part due to the use of antibiotics and dietary changes in industrialized countries over the past two decades, is a factor that may lead to modified airway tolerance to allergens and atopic disorders.[71] Genetics and microbiotic disruption would then be considered as predisposing factors, increasing an individual's susceptibility to develop airway hypersensitivity and allergy.

The nasal dysfunction associated with allergic rhinitis results in various symptoms. Nasal congestion with increased airflow resistance, particularly in the supine position, causes sleep-disordered breathing. It is a risk factor for snoring, affecting teenage males more frequently than females.[72] It is also linked to various systemic symptoms such as headaches, irritability and fatigue that diminish functional capacity. Thus, allergies are one of the main reasons for missed school days in the US.[73] School performance may be decreased because of inattention and decreased concentration.[74] Physical and emotional impairments associated with the allergic condition make it a whole body dysfunction.[75]

Because allergic rhinitis can affect the patient's quality of life to such an extent, and because of its economic impact, prevention and treatment are essential.[73] Osteopathic procedures may be seen

as a valuable complement to traditional medical treatment.

Physical examination and treatment

The examination for somatic dysfunction is begun by observing the global postural pattern and/or how the cervical and thoracic regions are, or are not, integrated into this pattern. The child should be observed from behind, from the side and from the front.

From behind, observe upper body postural mechanics. Look for cervical and thoracic sidebending, occipitocervical rotation and slumping of the pectoral girdle.

From the side, observe cervical and thoracic anteroposterior mechanics. There is often upper thoracic flexion with increased cervical lordosis. In this position, the head will very commonly be thrust forward with significant tension placed on the anterior cervical soft tissues. Observe specifically the submandibular myofascial structures and the position of the hyoid bone. The child with rhinitis may have to compensate by mouth breathing. As such, they may demonstrate the associated mouth breathing posture to a variable degree, depending on the chronicity of the condition. A double-chin appearance and the demonstration of a slack-jawed posture are indicative of chronic mouth breathing.

From the front, observe and confirm the sidebending and rotation observed from behind. Again look for the presence of mouth breathing and the associated orofacial characteristics. Children who mouth breath demonstrate a lack of tonicity of their facial tissues. The lower lip is typically everted and the tongue slightly protruded. Observe the relationship between the tongue and the teeth. Persistent protrusion of the tongue results in anterior displacement of the upper incisors with an eventual overbite. The child with allergic rhinitis will demonstrate puffiness of the facial soft tissues, particularly noticeable around the eyes, as well as darkening of the tissues beneath the eyes. The nasion is often recessed in the face. Because of the chronic lack of nasal breathing, the bony structures of the nasal cavities are small, resulting in narrowed nasal apertures. Children with persistent nasal congestion demonstrate an observable transverse crease in the skin across the lower third of the nose, at the junction between the nasal bones and cartilages. This develops as the result of repeatedly rubbing and pushing the tip of the nose vertically or laterally with their fingers or hand in response to nasal itching – the 'allergic salute'.

Following observation, the palpatory examination is best performed with the child supine. Begin by palpating the upper thoracic region for structure and employ the tests of listening to assess function, paying particular attention to the motion of the vertebrae and ribs. Examine the clavicles in similar fashion. Next, evaluate the cervical spine, with attention to the structural and functional relationships between the occiput, C1, C2, C3 and C4. Palpate the soft tissues in this area for the presence of edema. In acute upper respiratory conditions, the trigeminally mediated upper cervical reflex (occiput, C1) will result in acute tissue texture changes. Assess the anterior cervical soft tissues and midline structures with attention to the hyoid bone. Identify somatic dysfunction and treat it using the principles of indirect technique.

Examine the cranial base. Note the pattern demonstrated between the sphenoid and occiput. SBS compression and inferior vertical strain are often encountered in association with nasal dysfunction. Note the relationship between the occiput and temporal bone. Visualize how this relationship impacts the jugular foramina and consequently CN X. The functional status of the sphenoid bone exerts significant influence on the frontal bone and the facial bones below, and should be assessed. Also assess the frontal bone. Any dysfunctional motion restriction will result in diminished movement, and consequently diminished drainage, of the nasal cavities. In particular, dysfunctional frontal internal rotation causes the ethmoidal notch to be narrowed, restricting the movement of the ethmoid below. When evaluating the relationships between the sphenoid, frontal bone and the facial bones with the tests of listening, first assess the global motion of the region and then proceed to assess the individual bones and their interosseous relationships. During this assessment, localized motion restriction may be perceived that requires further identification through visualization. The following sutures are potential sites of interosseous dysfunctions: frontoethmoidal, frontomaxillary, frontonasal and sphenoethmoidal.

The relationships between the vomer and the sphenoid, ethmoid, maxillae and palatine bones, as well as the articulation between the two maxillae, should be evaluated. The nasal cartilages should be assessed in their relationship with the nasal bones and perpendicular plate of the ethmoid.

Following indirect principles, treat any dysfunctions found. Address the upper thoracic and cervical regions for their effect on the ANS. Treat cranial dysfunctions for their effect on the autonomic and sensory nervous systems, venous and lymphatic drainage, and to promote the production and drainage of nasal secretions.

Treatment of nasal structures will affect the total body through the cranial mechanism. The nasal septum consists of the vomer and the perpendicular plate of the ethmoid. Posteriorly, it is continuous with the sphenoidal sagittal septum that divides the body of the sphenoid into two sinus cavities. Posteriorly and superiorly, it is continuous with the falx cerebri and falx cerebelli. These structures combined constitute a vertical septum that separates the paired structures of the head and unites the viscerocranium and the neurocranium and, through the core link, the body below.

The nasal mucosa contains a rich supply of nerve endings and a dense network of microvasculature. For these reasons, it is highly sensitive. This should be kept in mind during the physical examination and treatment. It makes tests of listening and methods of treatment employing indirect principles the techniques of choice. Further, to follow the PRM, its rhythm and potency within nasal tissue, and to employ treatment when appropriate to enhance its potency, may help to modulate the autonomic dysfunction present in rhinitis, as well as help to reduce stasis and edema on a macro level in the mucosa and a micro level in the neuronal synapses.

Advice to caregivers

Recurrent and chronic rhinitis is commonly triggered or aggravated by environmental circumstances. As such, the caregiver should be provided with information as to potentially irritating conditions and substances that will allow them to identify and remove these triggers from the child's environment. Allergens should be sought out, identified and, if possible, removed. The most common allergens include pollens, foods, molds, dusts and animal dander. A detailed list of these substances may be found by doing an Internet search.

Conversely, dietary considerations and respiratory exercises may be employed to improve the function of the immune system. It has been suggested that disruption of the normal microbiota in the gastrointestinal tract contributes to decreased airway tolerance to allergens.[71] Refined foods should be avoided as much as possible, while a diet rich in fresh fruit and vegetables and antioxidants such as vitamins C and E should be recommended. A diet high in probiotics that promote the growth of beneficial bacteria (Bifidobacterium, Lactobacillus, Bacteroides) is recommended. This includes prebiotic carbohydrates such as inulin and oligofructose, plant carbohydrates that are not digestible in the small intestine but rather are fermented by bacteria in the colon.[76] Lactose intolerance should be considered.

Respiratory exercises are intended to enhance nasal breathing, promote mucus drainage and reduce vascular stasis in the nasal mucosa. They should be initiated in such a way that they can be successfully performed and yet avoid frustrating the child. Nasal congestion impairs nasal respiration and if the child is simply told to breathe through their nose, the difficulty of the experience usually limits the success of the exercise. The child will feel frustrated at best, and at worst may experience anxiety and a sense of suffocation. And they will stop doing the exercises.

Begin by explaining to the child the importance of nasal breathing. Explain that breathing through their nose cleans the air they breathe and gives them more oxygen in their blood, and that this will, in turn, enhance their performance in school and in sports.

Next, have the child breathe through their nose and become aware of the sensation of nasal airflow. Have them palpate the lateral aspects of the nasal cartilages as they breathe. Teach them to actively flare the nose by contracting the dilatator nostril muscles as they inhale. Palpating the nose during this process reinforces their awareness of nasal flaring. After 2 or 3 minutes of active nasal breathing the child should observe the difference in the sensation of nasal airflow. They may now be instructed to repeat the above process at home, exercising for 5 minutes at least three times daily. The child should also be taught to breathe using their thoracoabdominal diaphragm.

Successfully implementing these exercises not only allows the child to improve nasal respiratory function, it further teaches them a sense of control over their own respiration. This will reduce and eventually eliminate the sense of suffocation the child experiences when they attempt to breathe through congested nasal passages because, even thought they may experience nasal congestion, they will have been empowered to alleviate it.

SINUSITIS

It is estimated that children get an average of six to eight colds annually, and that 5–10% of all upper respiratory tract infections are complicated by sinusitis.[77] The application of osteopathic principles in the treatment and prevention of sinusitis in children should be utilized, as it is particularly efficient. It is based on an understanding of the anatomic and functional aspects of the nasal cavities and sinuses

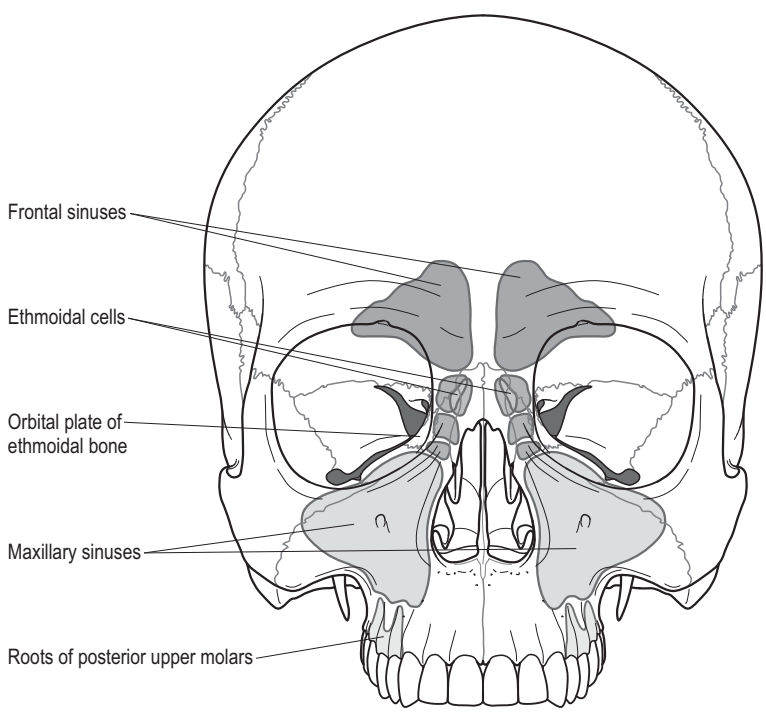
as part of the upper respiratory tract. A description of the nasal cavities, mucosa and its main characteristics has already been provided in 'Rhinitis' above. We shall, therefore, only consider the description of the paranasal sinuses and the pathophysiology that explains their dysfunction.

Many speculative theories exist concerning the function of paranasal sinuses. Some 2000 years ago, Galen hypothesized that they were 'porous bones' reducing the skull's weight. Since then, other theories have described the paranasal sinuses as shock absorbers, resonance chambers, air conditioning areas or the result of the evolutionary process and parts of the facial development.

There are four paranasal air sinuses associated with each nasal cavity: the ethmoidal, frontal, sphenoidal and maxillary sinuses, all of which open into the lateral walls of the nasal cavities by small apertures that differ from one individual to another. The lateral wall of the nasal cavity is formed anteriorly by the frontal process of the maxilla and the lacrimal bone; centrally by the ethmoid, maxilla and inferior nasal concha; and posteriorly by the vertical plate of the palatine bone and the medial pterygoid plate of the sphenoid. Three meatuses are located in this wall. They consist of three irregular passages directed anteroposteriorly – the superior, middle and inferior meatuses of the nose. The superior meatus is the smallest. Located between the superior and middle nasal conchae, it occupies the middle third of the lateral wall of the nasal cavity. The middle meatus is between the middle and inferior conchae. The inferior meatus is the largest of the three. It lies in the space between the inferior concha and the floor of the nasal cavity. Only the nasolacrimal duct drains into the inferior meatus in the anterior part of the nasal cavity.

The ethmoidal sinuses, on each side, are formed by 3–15 air cells, filling the ethmoidal labyrinth. They are divided into three groups: anterior, middle and posterior. The anterior and middle ethmoidal cells drain into the middle meatus, whereas the posterior ethmoidal cells drain into the superior meatus. The frontal sinuses are the highest. Each frontal sinus develops from an anterior ethmoidal cell that extends posteriorly along the medial part of the orbital roof and laterally above the internal part of the eyebrow at about 5 or 6 years of age. On each side, through the frontonasal duct and the ethmoidal labyrinth, the frontal sinus drains into the middle meatus. The maxillary sinuses are the largest. They form large pyramidal cavities within the bodies of the maxillae and drain into the middle meatus on each side. The sphenoidal sinuses are within the body of the sphenoid, their apertures being on the upper portion of the anterior walls of the sphenoidal body. The sphenoid sinuses drain into the superior meatus, near the roof of the nasal cavities (Figs 7.3.14, 7.3.15).

Figure 7.3.14. *Paranasal air sinuses.*



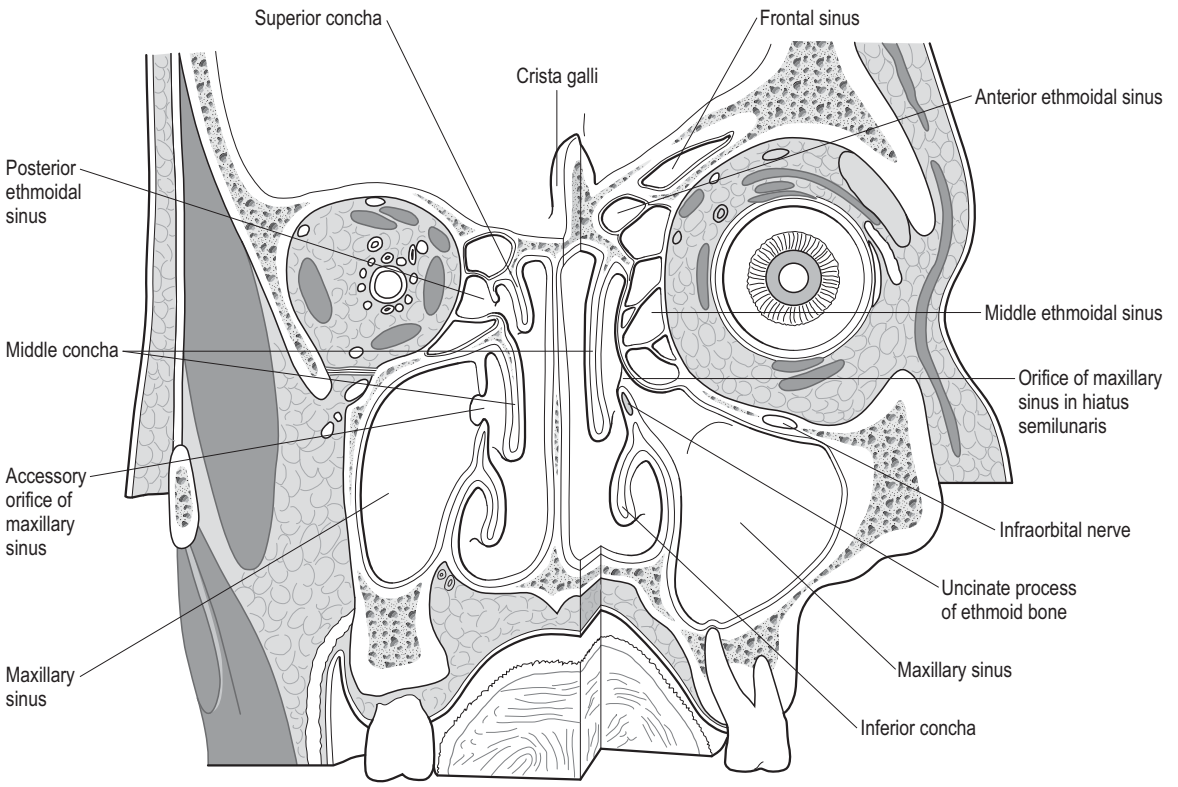


Figure 7.3.15. *Coronal section through the nasal cavity, from the posterior aspect. The plane of the section is more anterior on the right side.*

The pneumatization of the paranasal sinuses occurs at different rates, with a great deal of variation between individuals. The maxillary and sphenoidal sinuses are the first ones to develop in the fourth gestational month, followed by the frontal and ethmoid cells that appear in the sixth month. At birth, the parasinuses are quite small. The ethmoid cells measure 2–5 mm in diameter while the maxillary sinuses appear as furrows 7 mm in length and 4 mm in width.[78] The sphenoid sinuses are usually pneumatized around 5 years of age[79] and the frontal sinuses have developed to the extent that they are radiographically apparent by about the age of 6. Around this time, the growth of the cerebral mass slows down. The inner table of the frontal bone is stabilized while the outer table is still dragged forward by nasomaxillary growth. A space forms between both plates, where the frontal sinus expands. Mechanical forces associated with mastication and the actions of growth hormones are linked with the increase in the size of the sinus.[80] Sinusal development normally continues until late adolescence.

The paranasal sinuses are innervated by branches from the trigeminal nerve (CN V). The frontal and sphenoidal sinuses are innervated by branches from the ophthalmic nerve (CN V_1), the maxillary sinuses from the maxillary nerve (CN V_2), and the ethmoid cells from both the ophthalmic and maxillary nerves. As in the remainder of the nasal cavity, described previously, the paranasal sinuses are lined with a respiratory mucosa, ciliated and mucus-secreting, that is continuous with that of the respiratory tract.

Additionally, the paranasal sinuses seem to be an anatomic source for the excretion of nasal nitric oxide (NO). Mammals lacking paranasal sinuses (e.g. baboons) demonstrate lower exhaled concentrations of this molecule.[81] NO is involved in vasodilatation, neural transmission and immunologic activity, and appears also to participate in local host defense, even before allergens reach the respiratory mucosa. It may also regulate the mucociliary motility of the respiratory mucosa, thereby participating in the drainage of nasal secretions and the defense mechanisms of the mucosa. From the paranasal sinuses, NO may also play distal functions. Throughout normal nasal breathing, NO is constantly excreted into the upper airway, acting as an 'aerocrine' messenger.[82] It follows the airflow to the lungs

where it modulates pulmonary function through regulation of blood flow and oxygen uptake.[83] Nasal breathing, therefore, becomes crucial for these vital functions. When compared to oral breathing in healthy individuals, nasal breathing results in an improvement of arterial oxygenation[84] with a reduction of pulmonary vascular resistance. In this manner, NO would be an airborne messenger. It is of prime importance to improve nasal respiration in infants and children as soon as possible. Nasal breathing promotes healthier conditions in association with inspiration. Additionally, it stimulates the development of the maxillofacial skeleton and, therefore, of the nasal cavities.[85]

The paranasal sinuses are a common site for infection in children and adolescents. The most common sinusitis is maxillary, followed by ethmoidal and then frontal sinusitis. The tendency to develop sinusitis can be explained in part by anatomy. Proper ventilation is critical for preservation of sinus integrity. The maxillary sinuses are ventilated, but the ostium of each sinus is positioned high on the lateral wall of the nasal cavity. This encumbers gravitational drainage and probably predisposes patients to infections of the maxillary sinus. The frontal sinus is ventilated and because of the location of its ostium at its base, this sinus benefits the most from gravity. Sphenoidal sinusitis rarely occurs as an isolated infection, being found more often as a part of complete sinusal involvement.

Viral infections of the upper respiratory tract commonly result in an inflammation of the sinuses and nasal mucosa to produce the rhinosinusitis.[86] Bacterial infections with purulent nasal drainage are most often located in the paranasal sinuses.[79] Viral rhinosinusitis precedes about 80% of bacterial sinus infections while the remaining 20% most often follow allergic rhinitis.[77] Persistence of nasal symptoms, such as discharge or congestion, cough and headaches, particularly when awakening, for more than 30 days is defined as chronic sinusitis.

The sinus ostium is a common structure shared by all of the paranasal sinuses. Its function has been compared to that of the pharyngotympanic tube. They both permit drainage. The ostia of the sinuses allow drainage of the paranasal sinuses whereas the PTs drain the tympanic cavities.[87] Therefore, following the same principles as those used to treat otitis media, osteopathic procedures may be applied to affect the bones of the paranasal sinuses to improve their compliance and promote sinus drainage. Inter- and intraosseous techniques for the frontal bones, ethmoid, sphenoid and maxillae are very efficient in the treatment of sinusitis.

The normal size of the sinus ostia is approximately 2.4 mm.[88] The ostia are lined with mucosa and inflammation and swelling of that mucosa may decrease or occlude ostial patency and consequently the drainage of the paranasal sinuses. Obstruction of a paranasal sinus ostium will initially produce an increase of the pressure within the sinus. This is followed by intrasinusal gas absorption that consequently results in negative pressure within the sinus. This condition predisposes aspiration of bacteria-laden secretions into the ethmoid or maxillary sinuses from the nasal cavity, particularly when an individual sniffs or blows their nose.[87] Obstructed drainage generates stasis of mucus in the paranasal sinuses that, in turn, becomes an ideal culture medium for bacteria. More inflammation follows, with a self-perpetuating condition that leads to chronicity.

Additionally, somatic dysfunction may contribute to impaired nasal secretion. Parasympathetic stimulation results in vasodilatation and increased activity of the seromucous glands and goblet cells, with symptoms such as rhinorrhea and nasal congestion. On the other hand, increased sympathetic activity produces vasoconstriction and dryness of the nasal mucosa.[89] Cranial somatic dysfunction of the maxilla, palatine bone and sphenoid can affect the pterygopalatine ganglion and both parasympathetic and sympathetic supply of the nose and paranasal sinuses (see 'Rhinitis' above). Dysfunction of the cranial base and craniocervical junction will reflexly affect the trigeminal nerve and, through it, sympathetic and parasympathetic reflexes. Somatic dysfunction in the cervical and upper thoracic spine can affect sympathetic activity, as well as lymphatic drainage of the facial area. Once again, normal motion of skeletal structures, functional ciliary motion and autonomic regulation are required for a healthy upper respiratory system.

It should be stressed that cranial somatic dysfunction, although it may originate very early in life, may not manifest until years later. Nasal septal asymmetry may affect as many as 1% of newborns as the result of compression of the tip of the baby's nose during vaginal delivery.[90] Nasal suction bulbs or nasogastric tubes may also be traumatic to the nose. A nasal septal deviation may affect the middle concha and predispose to the obstruction of the osteomeatal area.[87] Cranial base dysfunction and vertebral somatic dysfunction may result from difficult labor. Obviously, for all these reasons, a whole body osteopathic evaluation and treatment of the newborn should be performed, including attention to the facial bones, particularly those of the nose. Left untreated, facial

somatic dysfunction may restrict full development of the paranasal sinuses. Furthermore, nasal obstruction will lead to mouth breathing and sleep-disordered breathing.[85] Sore throat and sinusitis may follow. Nasal obstruction is frequently associated with chronic maxillary sinusitis, adenotonsillar hypertrophy and otitis media as well as dental malocclusion and facial maldevelopment.[31]

Later in childhood and adolescence, somatic dysfunction can occur as the result of traumatic forces from physical bumps, falls, athletic strains and the like. The resultant dysfunction, depending on the direction of the traumatic force, may be established in the pattern of the individual's underlying postural balance or completely independent of it. Somatic dysfunctions of the facial bones and upper thoracic region are of particular consequence in the development and maintenance of sinus dysfunction.

The cranial respiration of the PRM differs from the thoracoabdominal respiration. However, they may entrain each other. This happens during states of relaxation, where the rate of the pulmonary respiration decreases to that approximating the rate of the PRM. Thereby, the two respirations combine their action to affect the entire body. This principle is particularly significant in the upper airway to promote movement of the nasal secretions, the gaseous contents of the paranasal sinuses, blood and lymph.

The normal cranial motion associated with the PRM consists of an inspiratory phase (flexion–external rotation) during which the paranasal sinuses as paired structures expand laterally and decrease in height. Conversely, during the expiratory phase of the PRM (extension–internal rotation), the sinuses decrease their lateral dimension and increase their height.

During cranial inspiration, the maxilla and the zygomatic bone move in external rotation, but at the same time a slight twisting occurs between them that contributes to the drainage of the maxillary sinus. The movements of all bones in association with the biphasic PRM may be described as the result of the combined movements in the three cardinal planes. The twisting between the zygomatic bone and the maxilla occurs predominantly in the sagittal plane. During external rotation, the zygoma demonstrates a component of anterior rotation, while the maxilla simultaneously demonstrates posterior rotation. The reverse occurs during internal rotation. This motion may be compared to the wringing out of a wet rag.

The zygomatic bones are an interface between the greater wing of the sphenoid, the maxilla, the frontal and the temporal bones. Their position is strategic and they play a key role in the balance of the face. The vomer is located between the sphenoidal body and the hard palate. Its inferior border articulates anteriorly with the intermaxillary suture between the palatine processes of the maxillae and posteriorly with the interpalatine suture between the horizontal plates of the palatine bones. The vomer rotates posteriorly during cranial flexion or inhalation, when the body of the sphenoid rotates anteriorly. Conversely, the vomer rotates anteriorly during cranial extension or expiration, when the body of the sphenoid rotates posteriorly. Accordingly, Sutherland stated: 'During inhalation the zygomatic bones and the vomer function somewhat like a plumber's plunger on the sphenoidal sinus and the maxillary sinuses.'[91] Every component of the facial skeleton is involved as part of the global functional pattern and should be assessed. Again according to Sutherland, even the smallest structures should be considered: 'See the turbinates on the side of the nose as they are in the living body, curling and uncurling during inhalation and exhalation.'[92]

The treatment of sinusitis is intended to promote the PRM. The osseous structures as well as the potency of the PRM should be considered.

Physical examination and treatment

Manipulative treatment of the somatic dysfunction associated with sinusitis should be employed in conjunction with standard medical treatments. The sooner it is initiated, the more rapid and successful the response. Failure to treat chronic sinusitis effectively will result in altered growth patterns of the viscerocranium. Nasal breathing will be impaired with concomitant malposition of the tongue and resultant dental malalignment.

Osteopathic examination and treatment of sinusitis are very similar to that of rhinitis. Examination is directed at the identification of somatic dysfunction that affects normal mucociliary clearance and impairs blood and lymphatic circulation, as well as ANS function. For this and the associated treatment discussion, the reader is directed to 'Rhinitis' above.

Manipulative treatment for sinusitis should focus on the reduction of mucosal edema, which will increase osteal patency. Procedures should be employed to drain the sinuses and provide symptomatic relief. Treatment should also include procedures that address sympathetic and parasympathetic somatovisceral reflex activity affecting the sinuses.

The sympathetic supply of the paranasal sinuses emanates from the upper thoracic spine. When

examining the child with sinusitis, it is all too easy to become focused on dysfunction of the viscerocranium and forget this significant area. Additionally, when working with children it is less intrusive to begin in an area away from the face. Perform tests of listening to evaluate the motion of the upper thoracic vertebrae and associated ribs. Because of its relationship with the trigeminal nerve, the occipitocervical region should also be assessed.

The rhythmic motion of the cranial bones under the influence of the PRM significantly facilitates sinus drainage. It is, therefore, appropriate to examine the global cranial pattern, looking specifically for dysfunction that reduces the motion of the sphenoid, ethmoid, vomer, palatine and zygomatic bones, maxillae and conchae.

Observe the face of the child, looking for puffiness in the nasal area. Look for asymmetry of the nares and asymmetric nasal respiration. Compare the degree of nasal alar flare with inspiration. In addition, inspect the nasal cavity, noting secretions, edema and erythema of the mucosa. Observe the child for open mouth posture that may be indicative of enlarged adenoids. Listen to the child's speech for hyponasality.

Utilizing tests of listening, evaluate the sphenoid and frontal bones. Proceed to assess the facial bones, paying particular attention to the ethmoid, maxillae and zygomatic bones. The function of the vomer, palatine bones and conchae should also be considered.

The zygomatic bones are easily accessible and their manipulation readily results in drainage of the maxillary sinuses. This procedure (see Chapter 6) is straightforward and easily mastered by the novice. Manipulation of the zygoma, in turn, affects the ipsilateral maxilla and greater wing of the sphenoid.

The motion of the sphenoid should be assessed. It exerts significant influence on the facial bones and on proper drainage of the sphenoid sinuses whose ostia are located in the upper portion of the anterior walls of the sphenoidal body. The ethmoid bone is a common site of dysfunction in children and adolescents, and should be assessed in its relationships with the frontal bones, sphenoid and maxillae. Proper motion of the ethmoid bone is necessary to facilitate the emptying of secretions from the ethmoid air cells.

Intra- and interosseous motion of the maxilla should be assessed because it is necessary for the effective drainage of the maxillary sinus. Additionally, in conjunction with the pterygoid process of the sphenoid and the palatine bone, it forms the pterygopalatine fossa where the pterygopalatine (sphenopalatine) ganglion is located. Any dysfunction of these bones may affect the ganglion and its effects on mucosal secretions.

The vomer's contribution is very important to the mechanism of the pumping action of the paranasal sinuses. Its assessment is often performed with one finger placed intraorally. When treating very young children, this procedure should be done only when absolutely necessary and then only with the greatest delicacy. The examiner should never attempt intraoral palpation of the vomer if the child is not completely cooperative. An alternative procedure is to employ visualization of the vomer while palpating the anterior edge of the nasal septal cartilage.

Treat any dysfunctional areas identified. Treatment is normally performed in continuity with assessment. Because indirect treatment procedures are used preferentially for children and adolescents, the effective treatment of a given area results in further relaxation of the patient, thereby facilitating the treatment of the next area to be evaluated and treated. Furthermore, when performing indirect techniques, tissue responses are continuously monitored, so that in acute conditions the patient's tolerance to the procedure is also continuously assessed. The tissues dictate the treatment; they guide your actions and determine the dosage.

The upper thoracic region should be treated for its sympathetic somatovisceral effects, to facilitate lymphatic drainage of the head and neck, and for functional reasons because it is the foundation on which the above structures rest. The occipitocervical junction should be treated for its reflex impact on the trigeminal nerve and its relationship to the cranial base. The sphenoid, frontal and facial bones should be treated for their direct effect on the paranasal sinuses.

Paranasal sinuses are intraosseous cavities and their drainage is dependent on the inherent motility of their respective bones: frontal, sphenoid, ethmoid and maxillae. As such, intraosseous dysfunctions of any of these bones can impact the associated sinus. Specifically pumping the individual bones may be employed to facilitate drainage of their sinuses. Positioning the patient contributes to the drainage of the paranasal sinuses by using gravity. Drainage of the sinuses is best performed as follows:

- drainage of the frontal sinus in the seated position
- drainage of the sphenoidal sinus in the seated position with the head of the patient bent forward
- drainage of the maxillary sinus in the supine position with the patient's head rotated to the

opposite side so that the sinus to be drained is up.

When treating the vomer, if the child resists the digital intraoral approach, the child's pacifier, if they have one, may be utilized. By allowing the child to actively suck on the pacifier the resultant alternation of intraoral pressure and tongue movement, pressing the pacifier on the roof of the child's mouth, may be employed to manipulate the vomer, while the practitioner works on the adjacent sphenoid and ethmoid bones.

Advice to caregivers

The caregiver should be encouraged to maintain a healthy lifestyle for the child, including a balanced diet with adequate hydration and the avoidance, as much as possible, of refined carbohydrate.

Respiratory exercises including nasal respiration and diaphragmatic breathing may be taught. Vocal activities such as humming can be beneficially employed to increase sinus ventilation.[93]

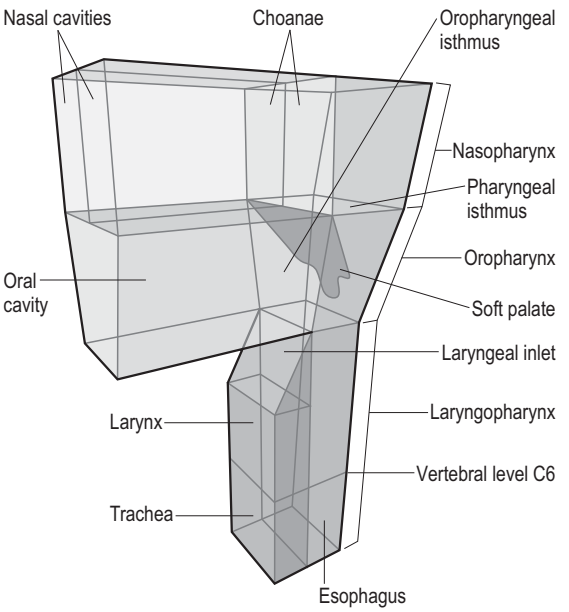


Figure 7.3.16. *Pharynx.*

PHARYNGITIS AND TONSILLITIS

The pharynx is a musculomembranous half-cylinder that connects the nasal and oral cavities with the larynx and esophagus. It extends from the base of the skull to the level of the sixth cervical vertebra where it joins the esophagus. The pharynx is divided into three portions: the nasopharynx located above the hard palate, the oropharynx that extends from the hard palate to the base of the epiglottis and the laryngopharynx from the base of the tongue to the larynx. The oropharynx can be further subdivided into the retropalatal or velopharynx from the hard palate to the caudal margin of the soft palate and retroglossal from the most inferior tip of the soft palate to the base of the epiglottis (Fig. 7.3.16).

The pharyngeal wall consists of an internal mucous layer, an intermediate fibrous layer and an external layer of skeletal muscle. In the superior part of the pharynx, the pharyngobasilar fascia is the thickest portion of the intermediate fibrous layer of the pharyngeal wall and is firmly attached to the base of the skull. The attachment forms an irregular U-shaped line. The anterior part inserts on the posterior margin of the medial plate of the sphenoidal pterygoid process. It then curves under the cartilaginous part of the PTs where it inserts onto the petrous part of the temporal bone and continues to the pharyngeal tubercle of the occipital basilar part to meet the attachment from the other side (Fig. 7.3.17).

The anterior part of the pharyngeal wall is not continuous; rather, it has multiple attachments to the medial pterygoid plate, the pterygomandibular raphe, the mandible, the tongue, the hyoid bone, and the thyroid and cricoid cartilages.

Six muscles contribute to constitute the pharyngeal wall. The bilateral superior, middle and inferior constrictor muscles constrict the pharyngeal cavity and, on each side, three longitudinal muscles – the stylopharyngeus, salpingopharyngeus and palatopharyngeus – elevate the pharyngeal wall and participate in swallowing (Figs 7.3.18, 7.3.19). The fibers of the three constrictor muscles fan out posteriorly into the median pharyngeal raphe, a fibrous band that is attached above to the pharyngeal tubercle of the occipital basilar part. The pharyngeal raphe descends to the level of the sixth cervical vertebra where it blends into the posterior wall of the esophagus. A thin retropharyngeal space filled by loose areolar tissue connects the pharynx with the cervical portion of the vertebral column and the prevertebral fascia covering the longus colli and longus capitis muscles.

The pharynx is the common route for air and food, and seven cavities communicate with it: the two nasal cavities, the mouth, the larynx, the two tympanic cavities and the esophagus (Fig. 7.3.20). The nasal cavities open posteriorly into the nasopharynx through the choanae. The oral cavity also opens posteriorly through the oropharyngeal isthmus

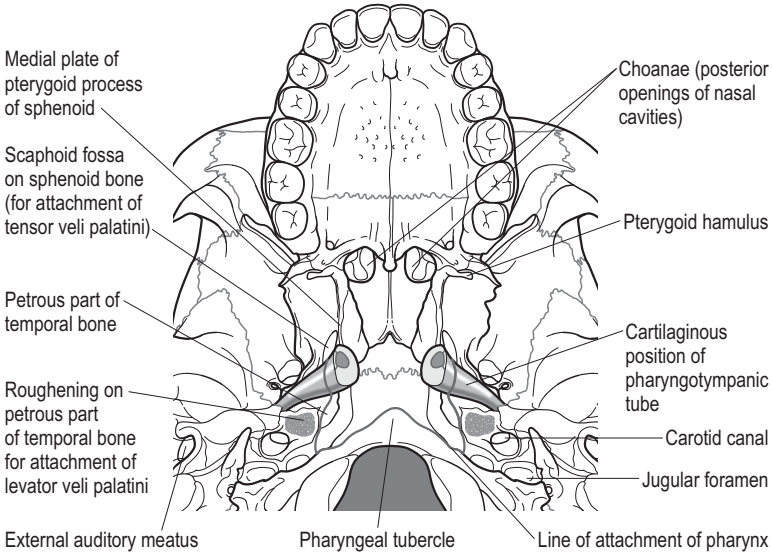


Figure 7.3.17. *Pharyngeal attachments to the base of the skull.*

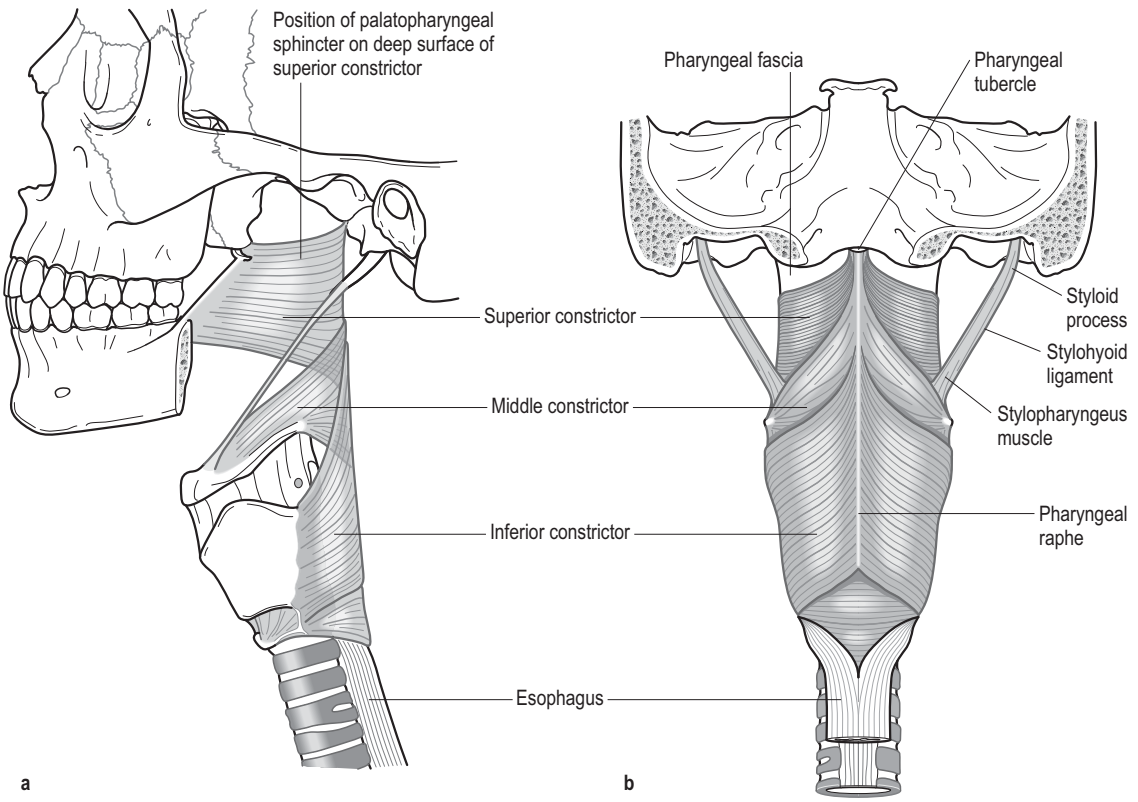


Figure 7.3.18 *Constrictor muscles of the pharynx: (a) lateral view; (b) posterior view.*

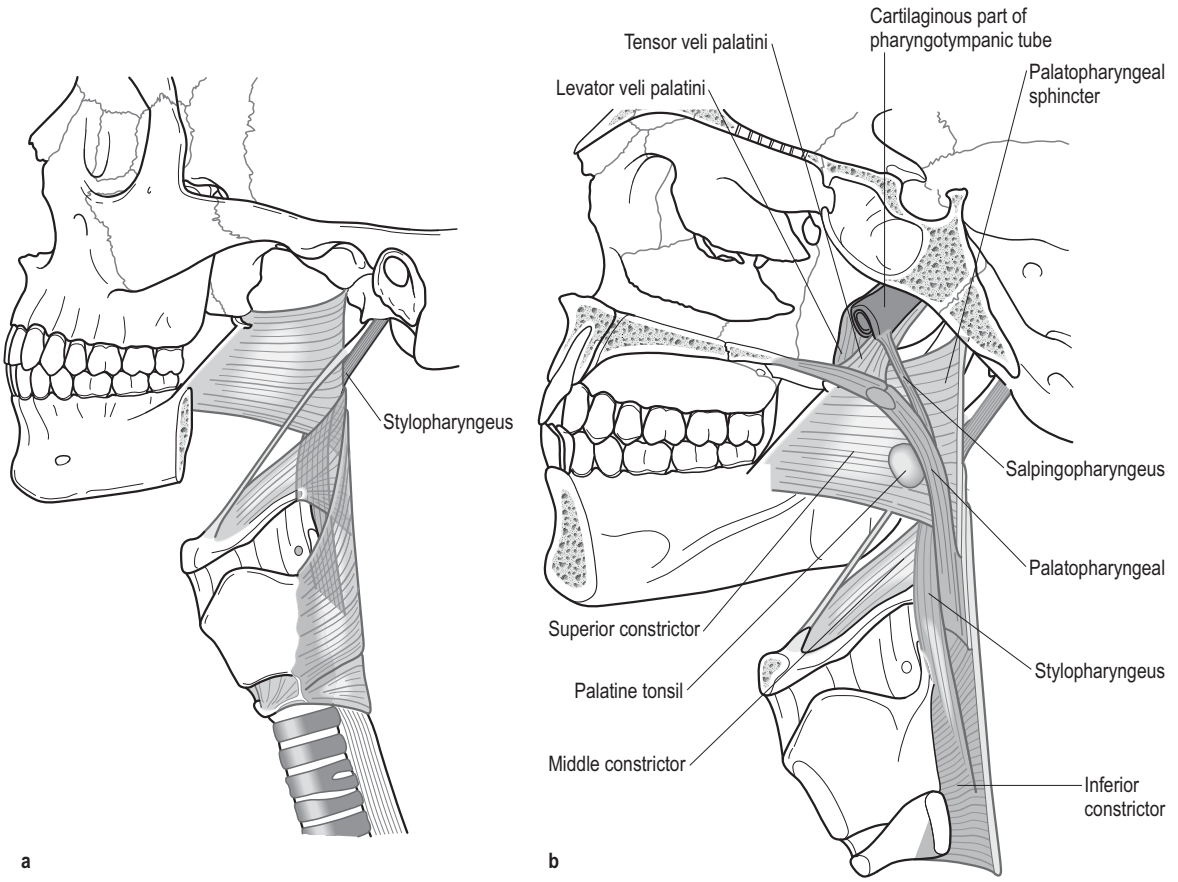


Figure 7.3.19. *Longitudinal muscles of the pharynx: (a) stylopharyngeus muscle; (b) medial view.*

and the larynx opens superiorly in the laryngopharynx through the laryngeal inlet. The PTs open laterally into the wall of the nasopharynx through the pharyngeal ostium located in the pharyngeal recess, or fossa of Rosenmüller.

The internal mucous layer of the pharyngeal cavity is continuous with that of the mouth and larynx and that lining the nasal cavities and PTs. It contains a large collection of lymphoid tissue, arranged in a circular orientation around the wall of the throat, the Waldeyer's tonsillar ring that represents the primary defense against pathogens at the entry of the upper respiratory and alimentary tract. The constituent parts of this defensive annulus are the nasopharyngeal, palatine, tubal and lingual tonsils, plus lymphoid tissue in the intertonsillar intervals.

The nasopharyngeal tonsil is located in the area of the nasopharyngeal roof and posterior wall, where the mucosa covers the inferior part of the sphenoidal body and the basilar part of the occipital bone. The palatine tonsil constitutes the major part of Waldeyer's ring. The paired palatine tonsils are located in the lateral wall of the oropharynx, in the tonsillar fossae, posterior to the base of the tongue between the anterior and posterior pillars, the palatoglossal and palatopharyngeal folds, respectively. They can be observed through the open mouth with the tongue depressed (Fig. 7.3.21). They are located slightly higher in the neonate and descend during the 2nd and 3rd years of age. The lingual tonsils are multiple lymphoid nodules situated on the posterior one-third of the tongue while additional small nodules beneath the mucosa of the PT form the tubal tonsils.

The nasopharyngeal tonsil increases in size in the first years of life to reach its peak around 6 years of age. Thereafter, it starts to involute until almost completely atrophied by puberty. When the nasopharyngeal tonsil is enlarged it is referred to as

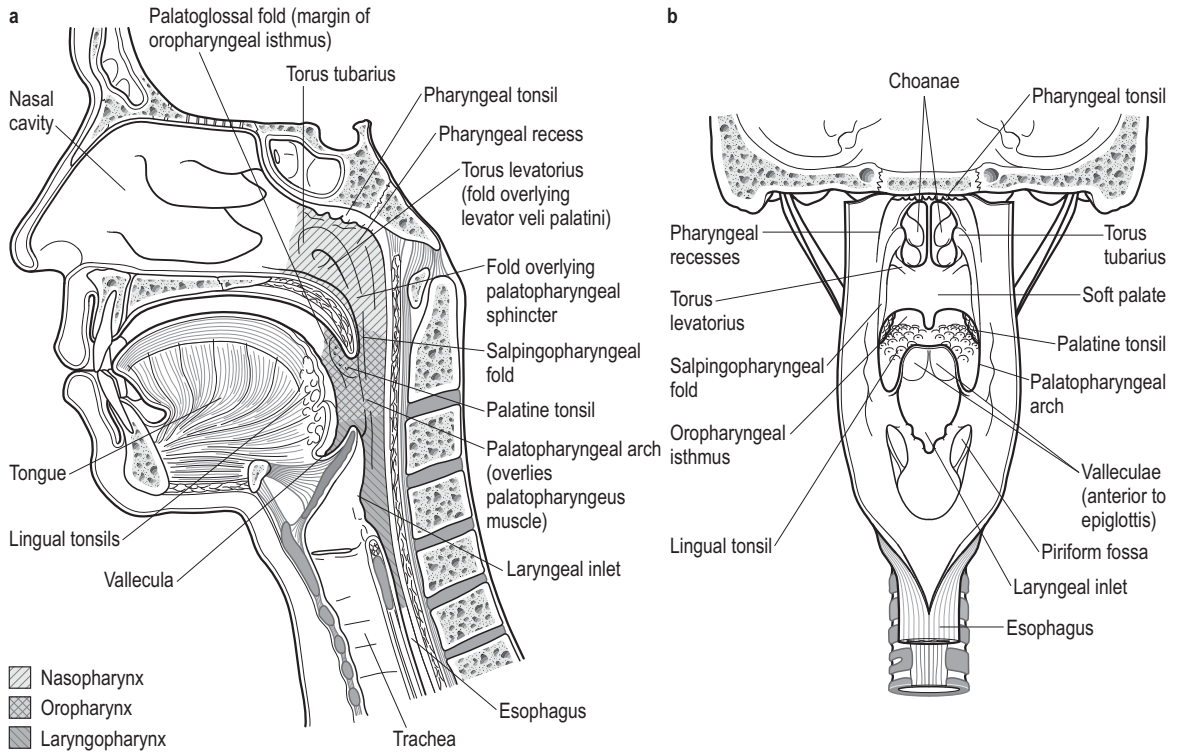


Figure 7.3.20. *Sagittal section of the oral and pharyngeal regions in the adult: (a) lateral view; (b) posterior view with the pharyngeal wall opened.*

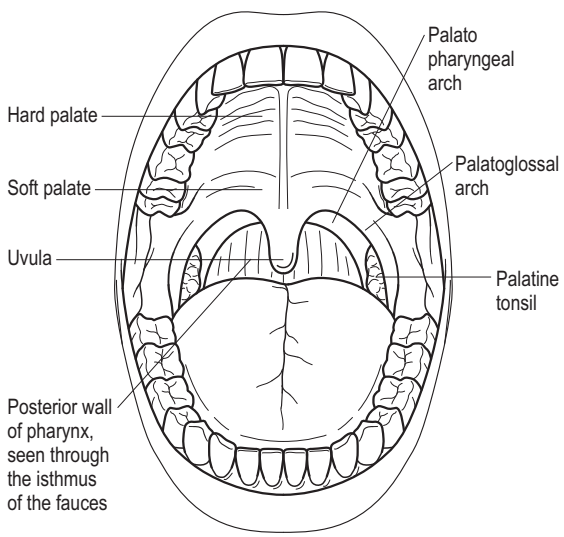


Figure 7.3.21. *The palatine tonsils located in the lateral wall of the oropharynx are observed through the open mouth, with the tongue depressed.*

adenoid or adenoids; 'tonsils' is usually the common name for the palatine tonsils.

Waldeyer's tonsillar ring is located at a strategic point where numerous antigens, both food-supported and airborne, first come into contact with the body. Thus, it plays an important role in the immune system as a site of antigen recognition and synthesis of antibodies, including IgE.[94] The nasopharyngeal and palatine tonsils are major sources of T lymphocytes that participate in cell-mediated immunity and B lymphocytes that produce immunoglobulins.[95] The nasopharyngeal tonsil also seems to participate in immune peripheral tolerance to harmless foreign antigens commonly inhaled or present in digested nutrients.[96]

Bacterial, or less frequently viral, infections are most often responsible for tonsillitis, where the tonsils may be acutely inflamed. Bacterial infections are often streptococcal, usually from group A streptococci, the most virulent species in humans. Differential diagnosis between viral and bacterial infection, based on physical examination alone, is difficult. Tonsillitis is characterized by sore throat and pain, particularly during swallowing, which may

involve the ears. Headaches, vomiting and high fever may be associated. It should be noted that bacterial infections are correctly treated with appropriate antibiotics and that in these instances the diagnosis and treatment of somatic dysfunction should be considered as adjunctive.

Pharyngitis, an acute inflammation of the pharynx, is usually the result of a viral infection, although it may be bacterial. The pharyngeal mucous membranes may be inflamed with purulent exudates. Sore throat and pain during swallowing are also present, associated with fever, cervical adenopathy and leukocytosis.

Susceptibility to infections differs between individuals. Host genetic components that adjust immune responses to pathogens seem to play an important role.[97,98] Nevertheless, the peritonsillar location is the most common of head and neck space infections in children in 49% of cases.[99] The American Academy of Otolaryngology–Head and Neck Surgery currently proposes adenoidectomy as a guideline after '3 or more infections of tonsils and/or adenoids per year despite adequate medical therapy'.[100] Adenoidectomy is currently one of the most common operations performed on children in the United States.[39,101]

Besides bacterial and viral infections, allergies such as allergic rhinitis are considered to be common risk factors for adenoid hypertrophy.[102] The continuity between the internal mucous layer of the pharyngeal cavity, the nasal cavities, the mouth, the larynx, the tympanic cavities and the esophagus explains the interrelationship and diversity of clinical presentations in allergic conditions.

Increased immune activity results in hypertrophy of Waldeyer's tonsillar ring, in particular at the level of the nasopharyngeal tonsil. Chronic nasal airway obstruction may follow a condition quite common in childhood that leads to a persistent mouth-open posture and mouth breathing. Adenotonsillar hypertrophy has also been associated with a myriad of symptoms such as obstructive sleep disorder syndrome, nocturnal snoring, rhinosinusitis, hyponasal speech and impairment of the ability to smell.[103] Furthermore, hypertrophy of the nasopharyngeal tonsil or adenoids predisposes the individual to recurrent otitis media or otitis media with effusion, in part because of the diminution of the patency of the orifices of the PT, located just laterally to the nasopharyngeal tonsil.

Tonsillectomy and adenoidectomy in children is reported to improve symptoms associated with obstructive sleep apnea syndrome, such as snoring and restless sleep, as well as behavioral, emotional and neurocognitive difficulties.[104] These procedures are also associated with improvement of the nasal cavity geometry by reducing the venous stasis and congestion of the inferior turbinate present in adenotonsillar hypertrophy.[105]

Nasopharyngeal tonsil hypertrophy and the associated mouth breathing are usually believed to impact craniofacial development. Experiments conducted in primates have established that a persistent mouth-open posture and associated oral respiration leads to dental malocclusions such as cross-bite.[106] Mouth-breather children may present with a narrow, elevated palate and a decreased nasopharyngeal space. In such cases, they may demonstrate a receding chin (retrognathia) with crowding of the maxillary and mandibular teeth, and increased lower anterior vertical face height. Following adenoidectomies and establishment of nasal breathing, changes are reported, with a more anterior growth of the mandible, improvement of retrognathia and diminution of lower anterior vertical face height.[107]

The commonly used terminology 'adenoid' facies to describe longer lower face heights, open mouth and more retrognathic mandibles has led people to believe that adenoid hypertrophy was solely responsible for mouth breathing and associated disorders. This is an overly simplified conclusion. Once again, a multifactorial approach to diagnosis and, consequently, treatment may be necessary.

A study of children 12 years after adenotonsillectomy showed that upper airway narrowing during sleep was still present, although some resolution of sleep disturbance was obtained 6 months postoperatively.[108] Not all children who snore demonstrate adenotonsillar hypertrophy and such hypertrophy is not the only cause of sleep apnea. Pharyngeal collapsibility is implicated with anatomic predisposition[109] such as changes in the longitudinal tension within the pharyngeal airway.[109] Although adenoidectomy is considered to be an effective treatment for children who are mouth breathers, recurrence of breathing difficulties in these individuals occurs and has been attributed to their craniofacial anatomic pattern.[110]

In upper airway narrowing, sleep apnea, mouth breathing and adenotonsillar hypertrophy, the craniocervical membranous, myofascial, ligamentous and interosseous somatic dysfunction may contribute to the pharyngeal dysfunction. The upper part of the pharynx is attached to the sphenoid, the temporal bones and the occiput. Through the prevertebral fascia the pharynx is linked to the cervical spine and movements of the cervical spine are associated with changes of pharyngeal size: cervical flexion decreases

oropharyngeal size; cervical extension does the opposite.[111–113]

Any dysfunction of the cervical spine or of any of the structures on which the pharynx is inserted can impair normal pharyngeal function. Movement is necessary to mobilize body fluids, particularly lymph, and somatic dysfunction affecting the structures surrounding lymph nodes and vessels is associated with impaired motion and can thus interfere with lymph flow. Lymphatic vessels from the pharynx drain into the deep cervical lymph nodes through the retropharyngeal, paratracheal and infrahyoid nodes. The retropharyngeal nodes consist of a median and two lateral groups located in front of the lateral masses of the atlas. They are positioned between the pharyngeal and prevertebral fasciae and drain the nasopharynx and PT, as well as the two upper cervical joints.

The deep cervical lymph nodes are beneath the SCM muscle. Among them, a large node surrounded with several small ones forms the jugulodigastric group that receives drainage from most of the lymphatic vessels from the tonsil. The tonsils differ from the lymph nodes in that they do not receive afferent lymphatic vessels. Small lymphatic vessels organize as efferents from the tonsils that traverse the superior constrictor muscle before draining to the jugulodigastric nodes. Located against the posterior belly of the digastric muscle, these nodes swell during tonsillitis and may be palpable in front of the anterior border of the digastric muscle, below the mandibular angle.

The vessels that supply the pharyngeal wall come from the external carotid artery. The tonsillar branch of the facial artery (external maxillary) is the main blood supply to the palatine tonsil. The veins of the pharynx drain through the pterygoid plexus in the infratemporal fossa and into the facial and internal jugular veins.

Physical examination and treatment

Because the pharynx is so intimately linked to the cervical spine and the cranial base, as well as associated muscles and fasciae, optimal pharyngeal function necessitates that these areas are unimpaired. The upper thoracic spine is the anatomic origin of the sympathetic supply to the pharynx and, consequently, along with associated ribs, should also be considered when addressing pharyngeal dysfunction and disease. Finally, the functional freedom of the clavicles, thoracic inlet and cervical spine, together with associated soft tissues above, is necessary to facilitate lymphatic drainage of the pharynx.

Having performed a total body structural examination to identify the global postural pattern and its relationship to the pharyngeal complaint, with the child in a supine position, it is appropriate to begin the local examination by evaluating the cervical and upper thoracic regions. First, palpate for tissue texture abnormalities in the paravertebral muscles and superficial soft tissues, looking for areas of muscular tension and subcutaneous edema. Similarly, palpate the anterior and lateral aspects of the neck. Observe and palpate the location of the hyoid bone and larynx that should be in the midline. Palpation of the anterior neck structures should be done with great delicacy to prevent irritating already inflamed tissues and because this is an area of increased sensitivity, particularly in infants who were born with a nuchal cord. Assess the anterior and lateral cervical musculature, paying specific attention to the SCM muscle because of its relationship to the deep cervical lymph nodes. Follow the SCM inferiorly to its attachment on the clavicles and assess clavicular motion.

Evaluate the cervical vertebrae, noting the alignment of the spinous processes. Clinical experience has shown a strong association between pharyngitis and cervical articular somatic dysfunction that, when treated, appears to prevent recurrent pharyngitis. Next, evaluate the upper thoracic vertebrae and associated ribs.

Assess the functional status of the cranial base, noting the relationships between the occiput, temporal bones and sphenoid. The pharynx is suspended beneath the skull and tone of the pharyngeal musculature is impacted by cranial dysfunction. For proper function, the pharyngeal muscles require precise interrelationships between their origins and insertions. Dysfunctional mechanics that affect these relationships will impair function of the pharynx and associated Waldeyer's tonsillar ring.

The pharyngeal tonsil is located directly beneath the cranial base, at the level of the sphenobasilar junction. As such, freedom of motion of the cranial base may facilitate lymphatic drainage of the adenoids.

Mandibular function should also be examined. Dysfunction of the mandible may impact anterior cervical myofascial function, contribute to impaired lymphatic drainage of the jugulodigastric node and participate in chronic mouth breathing.

Apply indirect principles to treat any identified articular dysfunction of the spine, ribs and clavicles. Myofascial release techniques may be employed to address pharyngeal, anterior cervical and spinal muscular dysfunctions. Normalizing the cranial and

thoracoabdominal diaphragms and the thoracic inlet may be employed to promote lymphatic and venous circulation. Following the inherent motility of the PRM may enhance all of these interventions. This slow, gentle rhythm is soothing to the child and is integral in the maintenance of homeostasis.[114]

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7.4 RESPIRATORY DYSFUNCTIONS

MOUTH BREATHING

Humans may breathe through their noses, mouths or intermittently through both. It is commonly held that, between birth and at least 2 months of age, infants breathe exclusively through their noses. This belief has, however, been challenged and some authors propose that infants are 'preferential nasal breathers' rather than 'obligate nasal breathers'.[1] In older children and adults, under normal conditions and at rest, the nasal respiratory route is used, and oronasal breathing occurs typically when a higher degree of ventilation is necessary, as during exercise.

In order to breathe through the nose, the nasal airway (nostril, nasal cavity and nasopharynx) should be patent. Nasal obstruction consists of partial or complete blockage of one or more of these components of the air passages. In the newborn, the posterior nasal aperture may be blocked by choanal atresia. Because of the infant's preferential nasal breathing route, such total nasal obstruction is a medical emergency.[2]

In the young infant, and later in life, several other factors can cause nasal airway obstruction. 'Stuffy nose' or rhinitis is a common cause of nasal obstruction in young infants that results in mouth breathing (see p. 224). Viral upper respiratory tract infections, foreign body, deviated nasal septum, hypertrophy of the inferior turbinates and nasal polyps are other factors that can also predispose to mouth breathing and produce an open mouth posture. Nasal endoscopy may be necessary to assess children with severe nasal obstruction that may require surgical repair.[3]

Tonsillar hypertrophy is believed by many to be the main cause of nasal obstruction in children. The pharyngeal tonsil, or adenoids, is a large collection of lymphoid tissue located beneath the roof of the nasopharynx, at the level of the SBS. Enlargement of the pharyngeal tonsil commonly occurs as the result of frequent bacterial or viral infections and can obstruct the nasopharyngeal route so that mouth breathing is the only possible alternative. 'Tonsils' is the common name for the palatine tonsils, which are lymphoid tissue located laterally on the oropharyngeal walls, just posterior to the base of the tongue.

Children with enlarged adenoids are described as having 'adenoidal facies' (long faces). They share common features with other mouth breathers, having low body weight and short stature, circles around their eyes, receding chins, small mouths, dry, large lower lips and short upper lips that are held apart from one another. They also tend to have multiple allergies and to demonstrate a specific postural configuration, the most commonly described feature of which is the anteriorly displaced or extended position of the head.[4]

Efficient respiration is the result of multiple intricate neurophysiologic processes and several anatomic structures contribute to this complex system. The extended, or forward, head posture may follow nasal obstruction as a solution to compromised nasal breathing. Experimental studies have shown that cervical extension increases maximum oropharyngeal airway size.[5] Therefore, children with nasal obstruction will spontaneously tend to assume the extended or forward head posture.

An extended head position is associated with an anterior displacement of the condyles of the occiput on the superior articular surfaces of the atlas, while the squamous portion of the occiput is lowered. The distance between the occiput and dorsal arch of the first cervical vertebra has been shown to be decreased in mouth breathers.[6] The condyles of the occiput are convex and the superior articular surfaces of the atlas are concave. Extension of the head for the shift from nasal to mouth breathing results in anterior and ascending displacement of the occipital condyles on the superior articular surfaces of the atlas. This causes the horizontal line of sight of the orbits to be angled upward, with resultant shift of the visual field and the need for postural compensation. One way to accomplish this, which is commonly observed in mouth breathers, is to increase the thoracic kyphosis. Another way – possible with young children when the synchondroses of the cranial base are still patent – is to increase the amount of flexion in the cranial base. Such an increase of cranial base flexion is present in mouth-breathing children.[7] Interestingly, an increase in cranial base flexion in primates has been shown to result in a decrease of the anteroposterior length of the nasopharynx and shortening of the anteroposterior length of the mandibular ramus.[8] The decrease of the anteroposterior length of the nasopharynx reinforces the tendency for mouth breathing, while the shortening of the anteroposterior length of the mandibular ramus correlates with the receding of the chin (retrognathia) that is observed in children who are mouth breathers.

Adenoidectomy is considered to be an effective treatment for children with enlarged adenoids who are mouth breathers. However, recurrence of breathing difficulties in these children has been observed and is attributed to their craniofacial anatomic pattern.[7] An extended craniocervical junction, or any dysfunctional pattern in the surrounding myofascial structures, may, therefore, be considered a primary dysfunction that could, in turn, predispose the child to chronic mouth breathing. This is confirmed by clinical observations that some children present with chronic mouth breathing secondary to nasal airway obstruction, while others tend to be mouth breathers without any obvious obstruction of the nasal airway.

Mouth breathing has also been correlated with an inferoposterior displacement of the hyoid bone and an anteroinferior positioning of the tongue.[4,7,9] The hyoid bone, a 'U-shaped' bone with an anterior convexity, is not directly articulated with any other skeletal structures. Through its myofascial attachments it acts as an interface between the tongue, the pharynx, the larynx and the skull and thorax. Therefore its position is influenced by dysfunction of any of these related structures and it can, in turn, exert influence on them. In mouth breathers, both the hyoid bone and the tongue are displaced to a position lower than normal.

Chronic mouth breathers often demonstrate premature molar eruption. Mouth breathing also influences the growth of the mandible, resulting in anterior mandibular rotation[9] and increase of the gonial angle between the ramus and the body of the mandible.[7] Consequently, the vertical height of the lower face of mouth breathers is usually increased, with a resultant open bite that is an augmentation of the vertical dimension separating the jaws.[9]

Nose breathing performs several functions of importance, such as warming and humidification of the inspired air, facilitation of arterial oxygenation[10] and regulation of pulmonary function.[11] Nasal obstruction is linked to obstructive sleep apnea in children as well as in adults. Therefore, nasal obstruction is a significant dysfunction and osteopathic procedures can often be applied to improve the patency of the nasal airway. Preventive treatment should be carried out for infants and toddlers to ensure normal development of the cranial base and craniocervical junction.

Physical examination and treatment

Mouth breathing is present with and without nasal obstruction and the child should be evaluated to determine if nasal obstruction is present. In severe cases, nasal endoscopy and medical treatment may be necessary. Hyponasal speech usually indicates an obstructed airway. A simple way to check for nasal patency is to ask the child to breathe, at least 10 times, through their nose while keeping their mouth closed. The child should be able to perform this test without becoming short of breath. Difficulty in achieving the test may be associated with rhinitis or chronic sinusitis. Successfully completing the test indicates that the prognosis for a positive response to manipulative treatment is good.

An alternative method to assess nasal patency is to hold a mirror under the nostrils of the subject. Normally patent nasal respiration will fog the mirror. If nasal obstruction is not demonstrated, the child should be examined to define somatic dysfunction possibly responsible for mouth breathing. Identified somatic dysfunction should be treated and then procedures to establish nasal breathing should be taught to the child.

The objective of treatment is to obtain optimal posture, improve function of the cervical and thoracic spine, balance the cranial base and its relationships with the hyoid bone and mandible, and facilitate nasal passage patency. As the pharyngeal tonsil is located immediately below the cranial base, at the level of the sphenobasilar junction, augmentation of the motion of the cranial base may increase tonsilar lymphatic drainage.

When treating the child it is best to begin by addressing their global standing posture. Observe the relationship between the head and the remainder of the body. Look for any asymmetries of cervical rotation and sidebending. Look for an exaggerated position of cervical extension or flexion and for increased thoracic kyphosis and lumbar lordosis.

Next, with the child in the supine position, palpate for tissue texture change in the suboccipital area and in the cervical and thoracic spine below. Palpate the soft tissue in the submandibular area; look for any lack of tonicity of the genioglossus muscles. Palpate for texture change in the tissues surrounding the mouth and the nose.

Assess the motion of the suboccipital area, cervical and thoracic spine. The cranial base, sphenobasilar synchondrosis and sphenoid should be evaluated next. The motions of the frontal bone, ethmoid and facial bones, particularly the maxillae, are considered next. Identify any membranous myofascial dysfunction that might also be present and treat accordingly, following indirect principles.

Teaching the child activities for the rehabilitation of normal breathing habits should complete the treatment. The child should be trained to breathe

through their nose. They should be taught to control the nasalis muscle that compresses the nasal aperture with its transverse part and laterally opens the nostril with its alar part. With their index finger and thumb bilaterally contacting their nose, lateral to the nasal ala, they can be taught to feel the expansion of the nares when breathing. Next, they can be encouraged to open their nostrils more dynamically during inhalation while palpating the resultant nasal expansion. Encouraging them to smell pleasant odors such as flowers or favorite foods may further increase awareness of nasal function. Small children may be taught to intermittently flare and relax their nostrils by telling them to move their noses as a rabbit does, while keeping their mouths closed, but not so tight as to purse their lips by constricting the orbicularis oris muscle.

SLEEP APNEA

The upper airway is a very complex area that participates in several physiologic functions as diverse as deglutition, vocalization and respiration. Multiple anatomic structures take part in the mechanics of respiration. Consequently, more than one specific site of the upper airway can be blocked in obstructive disorders such as sleep-disordered breathing (SDB) where the different parts of the pharynx, the base of the tongue and the esophagus may be involved.[12] In SDB, several factors may be present along with the obstructive phenomenon. These include abnormalities or diseases of the paranasal sinuses, tonsilses, soft palate and tongue, as well as obesity.[13–15] A review of the development and relationships between the multiple constituents of the upper respiratory system is helpful in understanding the dysfunctional patterns of the region.

The respiratory system consists of the larynx, trachea, bronchi, lungs and pleura. The larynx is situated between the trachea and the root of the tongue. It forms the lower part of the anterior wall of the pharynx and is the organ of voice. In the infant, during the first 2 months of life, the larynx is located in a high position and, throughout both deglutition and respiration, contact is maintained between the epiglottis and the soft palate.[16] By approximately 6 months of age, however, this contact is maintained only during deglutition and separation occurs during respiration. The larynx progressively descends from a high position in the neck at the level of C1–C3 during the first 2½–3 years of life to a lower position in the adult, where it is located between the upper border of C4 and the upper border of C7.[16]

During the first 2 years of life the descent of the larynx is associated with important changes in the relationships of the structures that constitute the pharynx. In the newborn and the very young infant, the tongue is located totally within the oral cavity. As the larynx descends, the posterior part of the tongue is drawn posteriorly and inferiorly to participate in the constitution of the superior part of the anterior wall of the pharynx, i.e. the oropharynx.

The pharynx is shaped like a funnel, having a length of about 12.5 cm. The upper wide end of the pharynx is tipped forward and consists of the oral and nasal cavities, whereas the lower part, at about the level of C6, is continuous with the esophagus. Superiorly, it is attached above to the base of the skull on the posterior borders of the medial plates of the pterygoid processes of the sphenoid bone, on the petrous parts of the temporal bones and on the pharyngeal tubercle of the occipital bone. Laterally, the pharynx is connected to the styloid processes of the temporal bones and posteriorly to the cervical spine and the prevertebral fascia that envelops the longus colli and longus capitis muscles. The anterior portions of the pharynx are attached to the medial pterygoid plates of the sphenoid bone, the pterygomandibular raphes, the mandible, the tongue, the hyoid bone, and the thyroid and cricoid cartilages.

The pharyngeal wall consists of two groups of muscles, the constrictor muscles and the longitudinal muscles, grouped according to the arrangement of the muscle fibers. The three constrictor muscles – the inferior, middle and superior – function to constrict the pharyngeal cavity. The longitudinal muscles are the paired stylopharyngeus muscles that connect the pharynx with the styloid processes of the temporal bones, the salpingopharyngeus muscles that connect the pharynx with the cartilaginous part of the pharyngotympanic tubes and the palatopharyngeus muscles that connect the pharynx with the soft palate. The three longitudinal muscles elevate the pharyngeal wall. The pharyngeal muscles are innervated by the vagus nerve (CN X), except for the stylopharyngeus muscles that are innervated by the glossopharyngeal nerve (CN IX). The pharynx consists of three parts: the nasopharynx into which the choanae of the nasal cavity open, the oropharynx that constitutes the posterior portion of the oral cavity and the laryngopharynx into which the superior portion of the larynx opens. The soft palate is located between the nasopharynx and the oropharynx.

The soft palate is a determining factor in the establishment of the breathing route. It is compared to a curtain hanging from the posterior border of the hard palate, i.e. the posterior borders of the two palatine bones. The soft palate extends downwards and backwards between the mouth and pharynx. It consists of muscular fibers, aponeurosis, vasculature, nerves, adenoid tissue and mucous glands enclosed in a fold of mucous membrane. Its posterior surface is convex and is continuous with the floor of the nasal cavities. Its anterior surface is concave and is continuous with the roof of the mouth (Fig. 7.4.1).

The position of the soft palate determines the route of breathing. It acts as a valve hanging over the oropharyngeal isthmus. In its more horizontal position it separates the nasopharynx from the oropharynx, its posterior tip being closer to the posterior wall of the pharynx, facilitating the oral route of breathing. When it is depressed against the base of the tongue, the oropharyngeal isthmus is closed and the nasal breathing route is made possible. When respiration is through both mouth and nose, the position of the soft palate is shown to be between the tongue and the posterior pharyngeal wall.[13]

The soft palate is under the control of five pairs of muscles. The levator veli palatini (LVP) and tensor veli palatini (TVP) arise from the base of the skull. The LVP originates from the apex of the petrous part of the temporal bone and from the cartilage of the PT and spreads in the palatine velum to blend with the muscle of the opposite side. The TVP arises from the scaphoid fossa of the medial pterygoid plate, from the spina angularis of the sphenoid and from the lateral wall of the cartilage of the PT. It descends vertically and then turns around the pterygoid hamulus to insert into the palatine aponeurosis. The musculus uvulae is related to the uvula. The palatopharyngeus arises from the pharynx and the palatoglossus from the tongue. All the soft palate muscles are innervated by the vagus nerve (CN X) except the TVP that is innervated by the mandibular nerve (CN V_3).

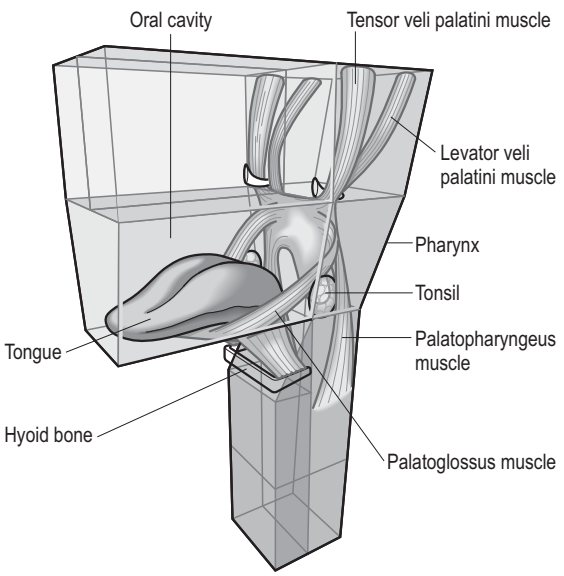

Figure 7.4.1. *Soft palate.*

The TVP muscles tense the soft palate; the LVP muscles elevate the soft palate and, therefore, close the nasopharynx. The palatopharyngeus muscles depress the soft palate and participate in the closing of the oropharyngeal isthmus. The palatoglossus muscles depress the soft palate caudally and ventrally and elevate the root of the tongue. The function of the soft palate is supposed to be actively determined, with dominant activity of the palatoglossus muscle when a subject is breathing quietly, and more activity from the LVP muscle during forced expiration.[13] Some activities such as breathing exercises or singing require both the nasal and oral breathing routes and the position of the soft palate consequently adapts to the situation.

Normal breathing relies on a patent nasal and pharyngeal airway. This patency depends on neural regulatory mechanisms as well as normal anatomic structures. The neural regulation is in part under the control of reflexes mediated through the trigeminal nerve (CN V) or the vagus nerve (CN X). The role of the vagus nerve is of paramount importance in the preservation of pharyngeal airway patency and any dysfunction of the upper cervical spine, in particular at the level of the jugular foramen, should be considered when airway patency is functionally compromised.

Considering the anatomic structures, the soft palate has been found to be the most common site of obstruction in infants presenting with significant obstructive sleep apnea (OSA).[17] Furthermore, an increase of cranial base flexion has also been demonstrated in mouth breathing or SDB children.[7] One can assume that, because of the insertion of the soft palate muscles, a dysfunction of the cranial base would lead to modified soft palate mechanics. The soft palate occupies a strategic position, an intersection between the muscles arising from the base of the skull and the muscles connected to the tongue and the pharynx. Considering these relationships, it is logical that any disturbance in the anatomic features influencing the soft palate, the tongue and

the pharynx may also play a role in the development of SDB.

Adenotonsillar hypertrophy is described as being the most common cause of OSA in children; its importance correlates with the dimension of the adenoids.[18] OSA, the result of airflow obstruction, occurs in older children as well as in infants, where symptoms may include apneic spells.[17] Pharyngeal collapse is involved and it is proposed that airway muscle dysfunction[19] or anatomic alterations predispose to that collapse.[20] Loss of longitudinal tension within the pharyngeal airway is suggested as being responsible for the collapsibility.[20] Subtle abnormalities in upper airway neuromuscular function or structure are proposed additions to the etiology of adenotonsillar hypertrophy.[21,22] The craniocervical junction is under great stress in the infant and dysfunction of the pharynx, which is attached to the base of the skull and to the cervical spine and prevertebral fascia, may be associated with any somatic dysfunction of the craniocervical junction and cranial base.

The position of the cervical spine is correlated with pharyngeal changes: cervical flexion reduces oropharyngeal size; cervical extension increases it.[5,23,24] Studies have demonstrated that patients with obstructive SDB present a narrower or more obstructed oropharynx and laryngopharynx than controls, particularly during sleep.[15] Besides the effect of cervical posture, alteration of reflexes due to sleepiness may play a role. When present, nasal obstruction also disturbs nasopharynx reflexes, mediated through the trigeminal or vagus nerves, which may result in decreased patency of the oropharynx.[25]

Multiple sites are considered as potential causes for SDB. In view of the intricate anatomic relationships of the soft palate, the pharyngeal wall, the tongue and the mandible, any dysfunction of one affects the associated structures and their positions. Biomechanical interactions between the tongue and lateral pharyngeal walls have been described[26] and displacement of the mandible also affects oropharyngeal size.[5] Mouth opening reduces oropharyngeal size.[27] The modified mandible position affects the function of the genioglossus, one of the tongue muscles that protrude the anterior part of the tongue out of the mouth. This, in turn, increases upper airway collapsibility, the position of the tongue being a strong determinant of the patency of the oropharynx. This explains why mouth breathers present with snoring and SDB.[7]

Alterations in the electromyographic activity of the geniohyoid and genioglossus muscles follow experimental nasal obstruction in monkeys with affected mandibular growth as a result. Similar mechanisms are believed to exist in humans[27] where mouth breathing in children, associated with SDB, may cause developmental facial abnormalities.[25] Maxillary and mandibular protrusions are smaller in children with OSA and the position of the hyoid bone is lower.[28] Mouth breathing generates the development of a craniofacial type that includes an increased value of the anterior facial height and an open bite.[29] The increased respiratory effort that these children must exert is responsible for functional impairment and eventually structural changes in their craniofacial anatomy. OSA is also associated with poor sleep quality, failure to thrive, impaired daytime psychomotor performance, irreversible developmental delay[17] and enuresis.[30] Osteopathic procedures should consequently be employed at the earliest possible time to avoid the establishment of dysfunctional patterns and their sequelae.

Physical examination and treatment

Begin by assessing for, and treating, any dysfunctional imbalances in the global postural pattern, particularly the craniocervical junction and upper aspects of the thoracic region including clavicle, sternum, upper thoracic vertebrae and associated ribs. Next, attention should be paid to the mechanics of the hyoid bone, including the infra- and suprahyoid muscles because of their relationship with the pharynx and tongue.

The synchondroses of the cranial base, the occipitomastoid sutures and the relationship between the sphenoid and temporal bones should be considered next. Any dysfunction of the temporal bones should be addressed in the treatment of SDB because of the connection between the longitudinal muscles of the pharynx and the styloid processes of the temporal bones. Evaluate the temporomandibular joint, the mandible and surrounding myofascial elements.

The inferior part of the pharynx is continuous with the esophagus that connects the pharynx to the stomach. Possible stomach as well as tracheal visceral dysfunctions may affect the pharynx, either through mechanical imbalance or through disturbed vagal reflex; therefore, they should be treated if present.

Teaching the child activities that strengthen and tone the soft palate and tongue can complete the treatment. Vocal exercises employed in singing are beneficial. The tongue, particularly the genioglossus muscles, can be strengthened by having the child alternately attempt to touch the tip of their tongue up to the tip of their nose and down to their chin.

BRONCHIOLITIS

Bronchiolitis is an acute viral infection of the lower respiratory tract that affects infants and young children. In Europe, Australasia and North America, an average of 3% of all children born every year present with bronchiolitis.[31] Respiratory syncitial virus (RSV) and influenza A virus are the most important viral causes of lower respiratory tract infection in young children. Infections with influenza viruses B and coronavirus may also be common.[32] Influenza viruses are highly contagious and are responsible for epidemics presenting various degrees of severity, although only a small proportion of children infected with the virus develop severe disease. Risk factors are childcare attendance, exposure to environmental pollutants, school-aged siblings, congenital abnormalities of the airways and neuromuscular disease.[33]

RSV is so named because, in tissue culture, it grows as a giant syncytia, a mass of protoplasm containing several nuclei. Multiple genotypes of RSV cocirculate every year,[34] along with an important variability in influenza virus occurrence from year to year.[32] The infecting virus first establishes in the upper respiratory tract and then spreads to the medium and small bronchi and bronchioles, resulting in inflammation of the epithelium with edema and bronchial obstruction that manifests principally during expiration. Air is trapped within the alveoli and hyperinflation of the lungs follows. Respiratory distress appears with tachypnea and tachycardia. Expiration is difficult and prolonged, and when the infant presents with severe tachypnea, breath is rapid and short, with poor air exchange. Wheezing, crepitus and fever may be present.

RSV is an important childhood pathogen in infants younger than 24 months of age.[35] Once infected, an infant does not develop complete immunity and recurrence of infection is common. Pneumonia and bacterial superinfections of the respiratory tract are frequent complications.[32] RSV may be severe in infants under 3 months of age and is an important cause of hospitalization for acute lower respiratory tract infection in infants and young children.[36]

Bronchiolitis, particularly when due to RSV, may be a precursor of the later development of asthma. Allergic rhinitis also exacerbates bronchial inflammation and may be a risk factor for the development of asthma.[37] Young children who have more than three episodes of infectious bronchiolitis, and those with a family history of asthma who have more than two episodes of infectious bronchiolitis, may also be predisposed to asthma. Widespread respiratory viruses like RSV are also possible factors in the cause of acute otitis media in young children.[38,39] Consequently, preventive considerations should be applied.

The primary defense against common pathogens of acute lower respiratory infections is Waldeyer's (circumpharyngeal) tonsillar ring, a collection of lymphoid tissue in the mucosa of the nasopharynx. It consists of the pharyngeal tonsil, the palatine tonsils and the lingual tonsil that are the multiple lymphoid nodules located on the posterior part of the tongue, plus small other nodules in the PT and lymphoid tissue in the intertonsillar intervals. The nasopharyngeal tonsil increases in size in the first years of life, are largest at 6 years and atrophies by the time of puberty.

The efferent lymphatics of these mucosa-associated lymphoid tissues start in plexuses surrounding every lymphoid follicle and drain through the retropharyngeal lymph nodes or directly into the upper deep cervical nodes. The retropharyngeal nodes consist of three groups, two of which are located on either side, anterior to the lateral masses of C1, following the lateral borders of the longi capitis.

Somatic dysfunction of the cervical spine, the frontal bone, the maxillae, the ethmoid bone, the nasal bones and the zygomatic bones can alter the function of the upper respiratory tract as a primary defense against common pathogens. Dysfunction of the thoracic cage and diaphragm can impair efficient return of lymph to the general circulation and the ciliary clearance current of mucus in the bronchial tree. Thus, somatic dysfunction can contribute to the creation of a fertile environment wherein pathogens can thrive. It has been shown that osteopathic manipulative treatment (OMT), particularly the lymphatic pump, results in decreased morbidity and mortality in patients with influenza.[40,41]

The ANS is of prime importance in the regulation of bronchial secretion and its dysfunction may facilitate bronchiolitis. The sympathetic postganglionic fibers between T2 and T4 stimulate bronchial and bronchiolar dilatation and decrease fluidity of the secretions. The pulmonary branches of the vagus nerve are motor to the muscles fibers of the bronchi and bronchioles and are consequently bronchoconstrictor. Thus, somatic dysfunction of the upper thoracic, upper cervical and cranial regions can affect the lower respiratory tract through somatovisceral reflex action.

Physical examination and treatment

The objective of osteopathic treatment in the acutely ill, non-emergent child with bronchiolitis is

to stimulate the expectoration of mucus, reduce air trapping and promote homeostasis by balancing the ANS and enhancing the venous and lymphatic drainage of the lungs. Furthermore, it is important to reduce somatic dysfunction that can predispose the child to the recurrence of the illness.

Observe the chest and the way the child is breathing, paying particular attention to the mobility of the ribs and sternum. Children with respiratory obstruction may demonstrate suprasternal, infrasternal, subcostal and intercostal retraction when breathing. On percussion, the chest is hyperresonant. Prolonged expiration, wheezing, and fine moist crackles may be observed at auscultation. It is important to auscult the lungs before and after the osteopathic treatment.

Using indirect principles, restore thoracic spine and rib motion. Thoracic pumping may be used to loosen mucus, stimulate expectoration and decrease bronchial obstruction. The thoracic diaphragm and thoracic inlet should be evaluated and treated as findings dictate. Rib raising and sternal molding may be applied to further mobilize the thoracic cage, increase ventilation, loosen mucus and stimulate expectoration.

Sympathetic activity should be balanced by treating any dysfunction of the upper thoracic spine (T2–T4). Parasympathetic tone can be normalized with treatment of the suboccipital area. Using indirect principles, treat any cervical somatic dysfunction that is present. Improve lymphatic drainage from the upper deep cervical lymph nodes with soft tissue techniques applied to the cervical myofascial structures.

Observe the child's face. The frontal bone, the maxillae, the ethmoid bone, the nasal bones and the zygomatic bones form the upper respiratory tract. Look for asymmetries of these structures and for any tissue texture change. Palpation and motion testing will confirm these observations and treatment should be applied accordingly. It is imperative that the child is breathing through their nose, so any dysfunction that impairs nasal respiration should be addressed.

Following manipulative treatment it is important to re-evaluate the child. Re-evaluate the musculoskeletal areas treated and auscult the lungs again to check clearance of the secretions and progress of airflow.

It is important to maintain hydration of the respiratory tract. The caregivers should be advised to encourage consumption of fluids and to maintain sufficient humidification of the child's environment.

ASTHMA

Asthma is the most frequently encountered chronic disease in childhood. Allergic disorders and asthma in childhood have increased in prevalence in many countries over the past 20–30 years.[42,43] Asthma is characterized by chronic inflammation leading to airway hyperreactivity and recurrent reversible airflow obstruction.

A multifaceted interaction of genetic and environmental factors appears to cause asthma. A genetic predisposition seems to exist[42,44,45] and the risk is greater if both parents present with the disease.[46] In the predisposed host, immune responses to different exposures such as allergens and air pollutants may trigger pathogenic inflammation.[47]

Children from lower socioeconomic groups more often present with asthma, rhinitis and allergic sensitization, especially to food allergens.[48] Different factors may explain this susceptibility, the quality of food being one of them. Evaluation of different diets demonstrates the asthma preventive effect of dietary management for children with a family history of asthma. Of demonstrable benefit are diets with increased anti-inflammatory 'n-3' polyunsaturated fatty acids (omega-3 polyunsaturated fatty acids), alone[49] and in combination with house dust mite allergen prevention.[50] There is also some evidence that dietary omega-3 polyunsaturated fatty acid supplementation during pregnancy and early childhood may potentially reduce infant atopy and asthma.[51,52]

The controversial 'hygiene hypothesis' was developed in the late 1980s to explain the high prevalence of allergic diseases and asthma in industrialized countries.[53] The attention to hygiene in these countries is associated with reductions in microbial exposures and decreased incidence of infectious diseases. Microbial encounters in infancy and early childhood stimulate the development of the immune system and the 'hygiene hypothesis' states that atopic disorders are the consequences of the lack of early life infections.[54] Alternatively, the use of antibiotics in the 1st year of life may increase the risk of asthma.[55]

Opposite to the hygiene hypothesis, however, there is evidence that the pathogenesis of asthma may include early exposure to viruses and bacteria.[56,57] A high frequency of respiratory tract infections in the 1st year of life is a predictor of asthma between the ages of 6 and 8 years.[46,58] Elevated IgE levels at 6 months are also a predictor.[46] Alternatively, the association of atopy with asthma is controversial and the onset of eczema during the 1st

year of life is not always found to be associated with the later development of asthma in childhood.[46]

A clinical association between rhinosinusitis and asthma is strongly suggested. Furthermore, effective treatment of rhinosinusitis has a positive effect on concomitant asthma.[59] Elements that contribute to the concept of 'united airway' disease include the dissemination of postnasal drip of inflammatory cells into the lungs. A vascular circulatory route with the migration of inflammatory cells to the lungs is another suggested pathway to explain the connection.

Another possible link between the upper and lower airways is through the nervous system, with naso-pharyngo-bronchial reflexes involving the trigeminal and the vagus nerves.[60] Upper airway inflammation may have an effect on receptors in the nose and pharynx. Afferent (sensory) fibers from these receptors participate in the constitution of the trigeminal nerve that connects with the dorsal vagal nucleus in the brainstem through the reticular formation. The vagus sends parasympathetic efferent fibers to the bronchi to preserve bronchial muscle tone and modulate bronchospastic responses. In asthmatic children, bronchoconstriction and mucus secretion is increased due to augmented parasympathetic nerve activity.[61] A neurogenic inflammation activated by inflammatory mediators and environmental irritants along the neural reflex pathway may be the cause of a neuronal dysfunction.[62]

The osteopathic principle of holistic integration of the different parts of the body applies perfectly to this hypothesis where an initial body reaction is followed by distant manifestations. The theory of 'one airway – one response' states that the common histopathology in both upper and lower airways results in a global allergic inflammation of the whole airway.

The total body allergic response is also illustrated through interactions between the respiratory system, the skin and the gastrointestinal tract. The lung and the gut are part of a unified mucosal system. The circulation of cells of the blood, from the bone marrow and the mucosal lymphoid tissue explain a possible interaction between these different areas and allergens.[63] The intestine is one of the most significant immune organs of the body. The composition of its microflora differs between infants with and without atopy, and the differences are verifiable before the occurrence of some clinical manifestation such as asthma.[64]

Alternatively, there is a link between the mode of obstetrical delivery and the maturation of the humoral immune system. Infants delivered by cesarean section demonstrate a delay in intestinal colonization.[65] The initial stimulation by the gut microflora may possibly be more significant than that of a sporadic infection and there is evidence of a relationship between cesarean section delivery and increased occurrence of atopic asthma.[66]

There is another connection between the lung and the guts with asthmatic patients. About 50–80% of adults and children with asthma present with gastroesophageal reflux – reflux of gastric contents into the esophagus – which may not be clinically obvious.[67] Intracellular acidification diminishes the ciliary beat frequency of the epithelial cells of the human tracheobronchial apparatus.[68] This point is significant in asthma, as well as in other respiratory dysfunction, as a factor contributing to decreased mucociliary clearance.

The lungs and esophagus are both innervated by the vagus and upper thoracic distribution of the sympathetic nervous system. Autonomic dysfunction may explain symptoms related to both gastroesophageal reflux and asthma.[69] Under normal conditions, the parasympathetic nervous system through the vagus sustains bronchial muscle tone while sympathetic fibers evoke bronchodilatation. Sympathetic nerve fibers also innervate the bronchial and gut-associated lymphoid tissue that seems to be essential in neuroimmune interactions.[70]

The parasympathetic visceral sensory system collects internal information that, in turn, influences emotions as much as emotional states impact autonomic function.[71] The influence of stress is potentially negative on neuroimmunoregulation.[70] In asthmatic patients, there is evidence that stress experienced prenatally or in the 1st years of life may participate in the development of asthma.[72,73] Increased psychological stress may impact respiratory illnesses in children and contribute to immune deregulation. There is evidence that stress facilitates susceptibility to infections[74] and may be associated with the development of asthma. Very often, asthmatic children present with anxious facial expression.

Most of the time, an asthmatic reaction is triggered by exposure to numerous environmental agents. Asthmatic children have hyperresponsive or hyperreactive airways. Various stimuli such as dust mites, pollutants and tobacco smoke produce an exaggerated bronchoconstrictor response with sensations of shortness of breath and chest tightness. There is evidence that the pathogenic development occurs early in the lungs, producing architecturally altered lungs later in life.[47] Treatment, therefore, should be initiated as soon as possible.

Asthma presents in different forms. The patient may demonstrate prodromal symptoms such as itching over the upper part of the chest and associated dry cough. This can be followed by episodes of dyspnea, tachypnea and tightness in the chest with wheezing and coughing that result from exposure to allergens, air pollution[75] or exercise. In asthmatic patients, a bronchoconstrictor response follows nasal inhalation of cold air.[76] Other patients present with chronic coughing and wheezing, associated with shortness of breath and decrease of vital capacity. Anxiety may occur related to the sensations of shortness of breath and chest tightness.

Physical examination and treatment

Osteopathic considerations for the treatment of asthma, although directed as a whole body intervention, are specifically focused on somatic dysfunctions of the thoracoabdominal diaphragm, thoracic cage, upper thoracic spine, cervical spine, sacrum, cranium and face. The goals of treatment are to encourage expectoration of mucus, reduce the mechanical impact of somatic dysfunction, enhance the recuperative effect of balanced sympathetic and parasympathetic tone, and facilitate the arterial, venous and lymphatic components of tissue perfusion. Because signs and symptoms are often observed first by the parents, these children may present to the osteopathic practitioner before the diagnosis of asthma has been formally made. It must be stressed that asthma is a potentially life-threatening condition, and although the treatment of somatic dysfunction can greatly benefit the patient,[77,78] the need for other methods of medical management should never be dismissed. The earlier somatic dysfunction is effectively addressed, the better the possible outcome. Manipulative treatment should begin with the area most easily accessible without distressing the child. The sequence of treatment is determined by the patient's acceptance of the intervention. Older children are commonly tolerant and treatment may be begun on the treatment table. For younger children, it is often easier to begin with the evaluation and treatment of the upper thoracic cage because this can be done with the child seated, even in the caregiver's lap. After a trusting physician–patient relationship has been established, the child may then be transferred to the treatment table for further treatment.

With the child seated or supine, observe the upper thoracic cage, looking for decreased compliance to respiratory excursion, i.e. tension of the scalene, trapezius and sternocleidomastoid muscles in the region of the supraclavicular triangle. Evaluate sternoclavicular motion and flexibility of the sternum for dysfunction. Articular motion of this region becomes mechanically discrete as the skeletal structures become more developed around 7 years of age. Palpate the thoracic spine and ribs for somatic dysfunction, paying attention to the area from T2 to T4 because of viscerosomatic input and somatovisceral impact with the lungs in this region. Utilizing indirect principles, treat identified somatic dysfunctions.

Following this, with the child, if possible, in the supine position, evaluate the lower thoracic cage and thoracoabdominal diaphragm. Observe the mechanical pattern of respiration. Asthmatic children tend to demonstrate forced expiration. Palpate for lower thoracic cage compliance, comparing the inspiratory and expiratory phases of respiration. Greater resistance will typically be appreciated during the expiratory phase and the child will often manifest shallow, rapid respiration. Palpating the lower thoracic cage bilaterally, evaluate for general tension and asymmetry in the excursion of the thoracoabdominal diaphragm. Assess the lumbar spine for dysfunction that can impact the diaphragm through the diaphragmatic crura. Examine the sacrum and pelvis to identify dysfunctional mechanics that can affect the asthmatic through the core link. Treat identified somatic dysfunction with indirect procedures. Entraining the movement of the manipulative treatment with the patient's breathing allows the practitioner to follow and gradually augment the amplitude of respiratory excursion.

Next, evaluate the cervical spine and myofascial structures of the neck. After general screening for dysfunction, attention should be directed at the upper cervical spine because of the viscerosomatic and somatovisceral vagal influence of the area. Treat identified dysfunction.

Examination can now proceed to the cranial base. Using your preferred hand placement, evaluate the motion of the SBS. Anecdotally, children with asthma and eczema seem to present frequently with SBS compression and decreased amplitude of the CRI. Assess the relationship between the occiput and temporal bones for compromise of the jugular foramen with its potential to interfere with vagal function. Evaluate the temporal bones where part of the tentorium cerebelli attaches. Dysfunction at this level affects the respiratory breathing pattern. An external rotation of one temporal bone tends to limit the freedom of expiration. Examine the relationship between the sphenoid, frontal and facial bones. The bones of the face – ethmoid, lacrimal, maxillary and nasal bones, which contribute to the structure of the

nasal airway – are suspended beneath the frontal bones. Additionally, the ethmoid bone articulates posteriorly with the sphenoid. It is not uncommon to encounter compression between the frontal bones and the bones of the face. Treat specifically identified dysfunctional patterns. It is important that the nasal airway is unobstructed because of the effect of nasal respiration on inspired air and thoracopulmonary function. The interrelationship between rhinosinusitis and asthma further stresses the importance of appropriate function of the facial bones in asthmatic children. Treatment of the cranial mechanism with attention to the inherent rhythm augments the CRI with a resultant total body effect.

Following manipulative treatment, various activities can be prescribed to facilitate the results of the intervention. For small children the caregiver should be encouraged to regularly gently stroke the thoracic paravertebral region, particularly T1–T5, bilaterally.

The child can be encouraged to perform expiratory activities like blowing soap bubbles. Older children can be taught breathing exercises to improve lung function to increase vital capacity, facilitate the clearing of airway secretions and enhance the quality of life. Begin by encouraging the child to breathe slowly and deeply, employing the thoracoabdominal diaphragm and with the least possible utilization of the accessory muscles of respiration – scalene, sternocleidomastoid, trapezius and abdominal wall muscles. They should learn to breathe on demand with particular attention to control of expiration. Further, they should be taught to hold their breath in the most relaxed possible way. This allows them to experience apnea without anxiety. As they become experienced in these activities, they can be encouraged to practice this method of respiratory relaxation at the first perception of an asthma attack. Because controlled breathing is an integral part of singing, they may benefit from participation in a choral group.

Teach them to maintain good posture. A simple procedure is to have the child walk with a book balanced on the top of their head. Finally, children and adolescents with asthma should participate in regular physical activity.[79,80]

Diet considerations should be initiated. Daily intake of fresh fruit and vegetables should be recommended. Processed sugars and foods that increase gastric acidity should be limited. Dairy products (e.g. ice cream) which increase mucus production should be consumed moderately and preferably before 5 p.m. to facilitate gastric emptying before bedtime and avoid gastroesophageal reflux.

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7.5 DISORDERS OF THE EYES

LACRIMAL DUCT OBSTRUCTION

Approximately 20% of infants demonstrate symptoms of imperfect lacrimal drainage during their first months of life. Most of the time spontaneous resolution takes place and by their 1st birthday only about 0.7% of infants still present with the condition.[1] Congenital nasolacrimal duct obstruction (dacryostenosis) may result from an abnormality in the lacrimal drainage system or from an infection.

The diagnosis of congenital nasolacrimal duct obstruction is clinical. It is based on a history of epiphora (tearing), mucopurulent discharge, or both, in the presence of non-inflamed conjunctiva, usually affecting only one eye and occurring in the first few weeks of life. The condition may be continuous or intermittent and crusting on the lid margins is common. Parents will often report that the eyelids are stuck together on the child's awakening or that the child has a persistently watering or sticky eye. Digital pressure applied medial to the eye over the lacrimal sac will produce increased discharge from the excretory puncta.

There is much debate regarding a standard medical approach for the management of congenital nasolacrimal duct obstruction.[2] The availability of multiple therapeutic approaches for any given condition indicates that none of them works particularly well in all cases. The list of procedures that are employed as standard to correct symptomatic nasolacrimal duct obstruction includes probing and irrigation, intubation with a silicone tube, balloon dilatation of the nasolacrimal duct and infracture of the inferior turbinate.[3] Osteopathic manipulation offers an alternative approach and, since it is benign, it should be attempted before more aggressive procedures are employed. The earlier the infant or child is treated, the better the chance of rapid resolution of the obstruction. Additionally, a good mental image of the anatomy of the nasolacrimal duct and lacrimal drainage system, on the part of the practitioner, will provide the basis necessary to improve the efficacy of manipulation.

The lacrimal apparatus consists of several parts that develop simultaneously. The lacrimal gland secretes the tears and the excretory ducts (lacrimal canaliculi) deliver the fluid to the surface of the eye, while the lacrimal sac and the nasolacrimal duct collect and transport the fluid into the nasal cavity.

The lacrimal gland consists of two portions: the superior orbital part and the inferior lacrimal part. The superior orbital part is located in the lacrimal fossa, in the superolateral part of the orbit, on the medial side of the zygomatic process of the frontal bone. Shaped like an almond, it is connected to the periosteum of the orbit and rests on the levator palpebrae superioris and the lateral rectus. The inferior lacrimal gland is separated from the superior by a fibrous septum and projects into the lateral part of the upper eyelid. Although the lacrimal gland reaches full development at about 3–4 years of age, the production of tears in infants is similar to that in adults.[4]

On each eyelid, the lacrimal canaliculus originates at a lacrimal punctum, a minuscule orifice on the medial margins of the lids. The superior canaliculus is the smaller and shorter. First it goes up, then turns at an acute angle to meet the lacrimal sac, while the inferior canaliculus descends, then turns upward toward the lacrimal sac (Fig. 7.5.1). At their angles the canaliculi are dilated and form ampullae. Their mucous lining is covered by stratified squamous epithelium, positioned on a basement membrane. The external wall consists of a layer of skeletal muscle fibers, continuous with the lacrimal part of the orbicularis oculi.

The lacrimal sac is the upper dilated end of the nasolacrimal duct. It is located in a fossa formed by the lacrimal bone, the frontal process of the maxilla and the lacrimal fascia. It measures from 12 to 15 mm in length and extends to form the nasolacrimal duct. Its superficial surface is sheltered by the lacrimal fascia (an extension of the orbital periosteum) and by the medial palpebral ligament. Its deep surface is crossed by the lacrimal part of the orbicularis oculi, which is attached to the posterior lacrimal crest on the lacrimal bone. The lacrimal sac is lined by a mucosal membrane continuous with the conjunctiva through the lacrimal canals and with the nasal cavity through the nasolacrimal duct.

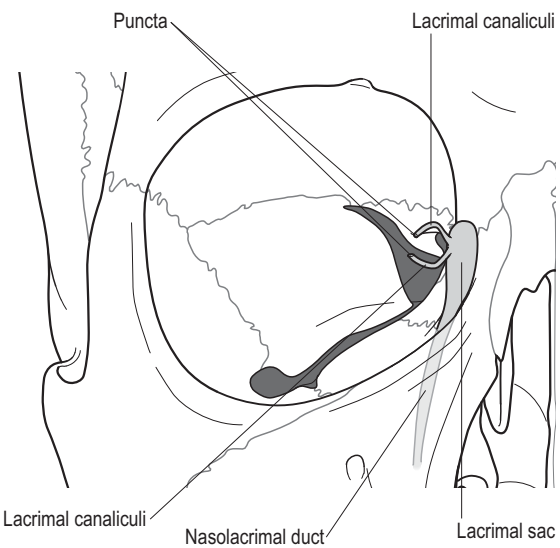

Figure 7.5.1. *The lacrimal sac.*

The relationship with the orbicularis oculi is of particular interest. This muscle surrounds the circumference of the orbit, with osseous attachments on the frontal bone, the frontal process of the maxilla and the lacrimal bone. When it contracts, as during 'blinking', compression of the lacrimal sac occurs through the lacrimal part of the muscle, pushing fluid into the nasolacrimal duct to drain into the inferior meatus of the nasal cavity. During muscular relaxation, fluids are drawn into the canaliculi and the expanded lacrimal sac.[5]

The nasolacrimal duct extends from the lacrimal sac caudally to open in the inferior nasal meatus. Both ends of the duct are wider than its middle portion, where it is enclosed in an osseous canal formed by the maxilla, the lacrimal bone and the inferior nasal concha. It is directed downward, backward and slightly laterally.

The nasolacrimal duct is formed embryologically from ectodermal cells enclosed between the maxillary and lateral nasal processes. During the 3rd month of gestation a canal appears in the center of this epithelial cord. It will develop progressively in a cephalocaudal direction from the 6th month of gestation until birth. In the third trimester of gestation the lower portion of the duct opens into the inferior meatus of the nasal cavity to constitute the nasolacrimal duct, while the epithelium from the nasal cavity invests the duct in a caudocephalad direction. A mucosal fold – the valve of Hasner – is located just above the nasal opening of the nasolacrimal duct. Total canalization of the epithelial cord may fail to occur with the persistence of membranous tissue that should normally disappear at birth or in the first days of life. In congenital nasolacrimal duct obstruction, the lower part of the duct may be closed at birth by fusion of the mucosa covering the nasal folds. The resultant obstruction is usually observed in infants at about the 10th or 12th day of life.

Nasolacrimal duct obstruction is frequently associated with dysfunction of the bones forming the osseous canal in which the nasolacrimal duct is located, i.e. the maxilla, the lacrimal bone and the inferior nasal concha. It frequently follows

compression of the frontal bone and may also be associated with an inferior vertical strain of the SBS. Consequently, the relationships between the frontal bone, maxilla and lacrimal bone are affected, potentially modifying the patency of the nasolacrimal duct. This type of compression occurs frequently during pregnancy, difficult labor or delivery and appears to be a prediposing factor. Furthermore, dysfunction of the frontal bone, maxilla and lacrimal bone changes the relationship between the points of attachment of the orbicularis oculi, predisposing to muscular dysfunction.

Physical examination and treatment

When treating nasolacrimal duct obstruction, the delicacy of the intervention necessitates that the infant moves as little as possible. This may be accomplished by arranging to see the infant at a time that coincides with their nap time. The best approach is to have the caregiver arrive with the infant about 30 minutes before the scheduled appointment. Place the infant and caregiver in a quiet examining room and allow the infant to have their bottle or to nurse. Allow sufficient time thereafter for the infant to go to sleep, preferably in a supine position on the examining table.

Once the infant is asleep, quietly commence treatment. First observe the frontal bones. Often the frontal bone on the side of the lacrimal duct obstruction will be found to be lower than the other side. Look next at the area of the nasion, nasal bones and frontal processes of the maxillae, noting asymmetry and compression of these structures. The forces commonly involved in lacrimal duct obstruction are most often vertical compression between the frontal and nasal bones, sometimes with sidebending and rotation of the facial block beneath the frontal bone, with the obstruction on the side of the facial concavity.

Confirm the observations with tests of listening, paying close attention to the frontal bones, nasal bones, maxillae and lacrimal bones. Keep a mental picture of the minute details of the area to help this process. Define areas of restricted motility and employing the inherent forces of the PRM and its rhythm, utilize the most delicate indirect treatment procedure. The treatment procedure should be so gentle as not to awaken the infant.

Advice to caregivers

Encourage the caregiver to regularly clean the secretions from the eye to avoid the development of an infection. To promote drainage into the nasolacrimal duct, the caregiver can milk the lacrimal sac by intermittently applying gentle digital pressure medial to the punctum of the eye in a superior to inferior direction.

Actively playing with the child to encourage facial expression stimulates the function of the orbicularis oculi. If the child is old enough, have them blink tightly and make faces. Contracting the orbicularis oculi promotes drainage by placing pressure on the lacrimal sac.

STRABISMUS

Strabismus – the deviation of the alignment of one eye in relation to the other – is a condition frequently encountered in infants and children. It is a very complex subject and its complete discussion is beyond the focus of this text. However, the treatment of somatic dysfunction can prove very beneficial in certain presentations of the condition. Strabismus must be taken seriously and significant underlying pathologies, such as congenital cataract and retinoblastoma, ruled out. It is imperative that, on presentation, every case of strabismus, no matter what the age of the patient, has a thorough ocular examination, including cornea, lens, retina and optic nerve, as well as the neurologic status of the eye and extraocular muscles (EOM). Treatment of strabismus should be initiated at the earliest possible time to avoid loss of the ocular stimulation that normally contributes to the maturation of the visual system.

The eyeball – the organ of sight – is contained in the skeletal cavity of the orbit, which provides a protective space for the eyeball and associated structures, i.e. fasciae, eyelids, conjunctiva, lacrimal apparatus and EOM. The eyeballs start to develop from neuroectoderm of the lateral aspects of the forebrain as a pair of diverticulae at approximately the 22nd day of gestation. At the 25th day, two optic vesicles are formed. Around the 4th week, they invaginate and create the optic cups in which mesenchymal and vascular tissues enter the globe. The different parts of the future eyeball and surrounding orbital cavity are intimately interrelated. At the beginning of the fetal period – the end of the 2nd month of gestation – EOM are present, surrounding the eyeball. Growth will continue with a significant correlation between gestational age and fetal eye biometry, including lens, orbital diameters, circumferences and surfaces.[6]

Surrounding the eyeball, the constituents of the orbital cavity consist of the frontal, lacrimal, palatine and zygomatic bones, ethmoid, maxilla and

sphenoid. They develop in membrane and are quite responsive to the growth stimulation of the eyeball. Thus, the orbital cavity is growing as a result of the increase of the volume of the eyeball and the activity of the EOM, with more and more elaborate eye movements. From this time up to 5 years of age, the eyeballs will continue to grow, acting continuously as a growth stimulator for the skeletal cavity of the orbit. At birth, the orbit height is already 55% of its adult height. At 3 years of age it is 79%, while at 7 years of age it is about 94%, nearly its adult size.[7]

Evidence of the importance of this stimulating factor is demonstrated by conditions like microphthalmia, where the development of the eyeball does not occur correctly, or when an individual is enucleated in early childhood. Underdevelopment of the orbital cavity is typically associated with these conditions. An anophthalmic bony orbit may be 14.5% smaller when compared to the other orbit.[8] The concept that function affects structure, one of the basic osteopathic principles, is perfectly illustrated in this instance.

At the same time, structure affects function. In this case, the structures forming the orbital cavity may affect the ocular function of sight, associated vascular and neurologic aspects, and extra- and intraocular muscular activity. Therefore, it is appropriate to consider the protective case for the eye, i.e. the orbital cavity, and to see how its osseous components play a part in the etiology of ocular dysfunction.

The orbits are located in the upper and anterior part of the viscerocranium. They are shaped like pyramids, with their apices and long axes directed backward and medially. Each orbit consists of a roof, a floor, a medial and a lateral wall, a base and an apex. The roof is concave, directed downward and slightly forward. The orbital plate of the frontal bone forms most of the roof, while the lesser wing of the sphenoid forms its posterior part. Therefore, there is a suture on the roof of the orbital cavity between the frontal bone and the lesser wing of the sphenoid. The lacrimal fossa for the lacrimal gland is located laterally on the orbital surface of the frontal plate. Medially, below and behind the end of the supraorbital margin of the frontal bone, is the trochlear fovea for the attachment of the cartilaginous pulley of the superior oblique muscle. This feature is of particular interest in understanding ocular dysfunction, since the frontal bone is frequently under stress from fetal positioning, difficult labor or trauma sustained by young children, such as a fall on the head. Although most of this anatomy is not directly palpable, the supraorbital margin of the frontal bone is completely accessible and its position should always be evaluated in strabismus.

The floor of the orbit is directed upward and laterally. It consists mostly of the orbital surface of the maxilla; behind that and medially, the orbital process of the palatine; and in front and laterally, the orbital process of the zygomatic bone (Fig. 7.5.2). The maxilla articulates with both the palatine and the zygomatic bones. The lacrimal notch is located anteriorly, on the medial border of the maxilla, and provides the superior opening of the nasolacrimal canal. On this border the maxilla articulates with the lacrimal bone and the ethmoid's orbital plate behind. A depression situated just lateral to the lacrimal notch is the location of the origin of the inferior oblique muscle. The posterior border of the maxilla forms most of the anterior edge of the inferior orbital fissure, discussed further below.

The medial wall of the orbit is formed anteriorly by the frontal process of the maxilla, the lacrimal bone, the orbital plate of the ethmoid and a tiny part of the body of the sphenoid in front of the optic foramen. The lacrimal groove for the lacrimal sac is located anteriorly. It is limited behind by the posterior lacrimal crest, from which the lacrimal part of the orbicularis oculi arises. Three vertical sutures – the lacrimomaxillary, lacrimoethmoidal

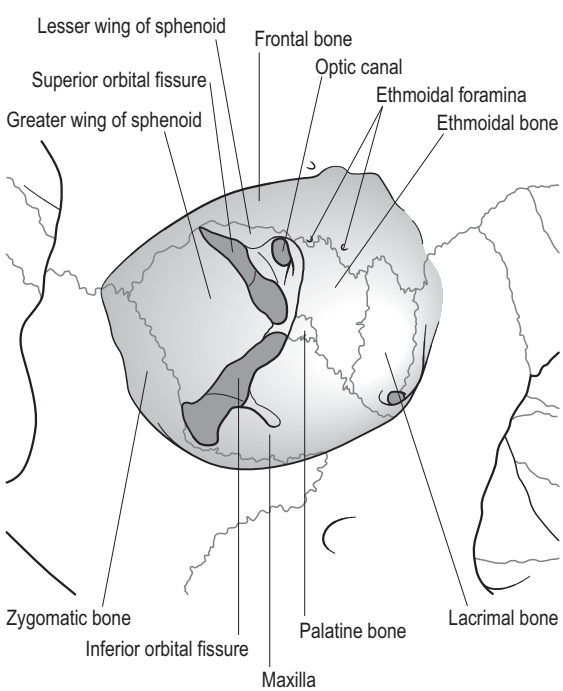

Figure 7.5.2. *The bony orbit.*

and sphenoethmoidal – are present, while the frontomaxillary, frontolacrimal, and frontoethmoidal sutures are situated between the superior border of the medial wall and the orbital roof.

The lateral wall of the orbit is directed medially and forward. It consists of the orbital process of the zygomatic and the orbital surface of the greater wing of the sphenoid. The sphenozygomatic suture unites them. This is another site of particular interest since the zygomatic bone is easily palpable. Through palpation of the zygoma one can visualize, indirectly assess and treat the less accessible greater wing of the sphenoid. The sphenozygomatic suture ends below at the anterior end of the inferior orbital fissure. The upper end of the sphenozygomatic suture meets with two other sutures, creating a sutural crossroads for consideration in the treatment of any ocular dysfunction. Anteriorly, the frontozygomatic suture can be observed, and posteriorly the suture between the frontal bone and the greater wing of the sphenoid. Just under the frontozygomatic suture, on the orbital process of the zygomatic bone, is a tubercle for the attachment of the levator palpebrae superioris' aponeurosis.

The inferior orbital fissure lies between the lateral wall and floor of the orbit, posterior to the zygomaticomaxillary suture. It communicates with the pterygopalatine and infratemporal fossae, and transmits the infraorbital vessels, the maxillary nerve (CN V_2) and the ascending branches from the pterygopalatine ganglion.

The superior orbital fissure separates the roof and lateral wall of the orbit in its medial portion. The oculomotor (CN III), the trochlear (CN IV), the ophthalmic division of the trigeminal (CN V_1) and the abducent (CN VI) nerves enter the orbital cavity through this fissure, accompanied by some filaments from the cavernous sympathetic plexus and the orbital branches of the middle meningeal artery. Additionally, the superior ophthalmic vein drains into the cavernous sinus through this fissure.

The apex of the orbit corresponds to the medial end of the superior orbital fissure, close to the origin of the EOM, just below the optic foramen. The cylindrical optic canal forms by surrounding the optic nerve and ophthalmic artery where the two roots of the lesser wing of the sphenoid join the sphenoidal body.

Through the optic canal and superior orbital fissure, the cranial cavity communicates with the orbital cavity. The cranial dura mater lines the internal surface of every cranial bone, with a firm adhesion at the sutures, and extends outside the cranial cavity through foramina and fissures, forming tubular sheaths for the cranial nerves as they leave the neurocranium. Thus, the endosteal layer of the cranial dura mater is continuous through the superior orbital fissure with the orbital periosteum. In addition, a tubular dural sheath from the meningeal layer of the dura surrounds the optic nerve as it passes through the optic canal. This dural layer blends with the ocular sclera and adheres intimately to the common annular tendon of the four recti muscles. There is an anatomic continuity between the dura and the lining and structures of the orbital cavity, such as the eyeball and the EOM.

The seven extraocular, or extrinsic, muscles include the levator palpebrae superioris, superior rectus, inferior rectus, medial rectus, lateral rectus, superior oblique and inferior oblique muscles (Fig. 7.5.3). They control the movements of the upper lid and eyeball. All the EOM are tied together in a complex fashion by fascial sheaths. There are also intrinsic muscles within the eyeball that are responsible of the shape of the lens and size of the pupil.

The levator palpebrae superioris arises from the lesser wing of the sphenoid, above and in front of the optic foramen, from which it is separated by the origin of the rectus superior. From a narrow tendon it soon broadens into a flat, triangular shape that ends anteriorly in a wide aponeurosis. The superficial fibers are prolonged forward, piercing the orbicularis oculi muscle, to insert on the deep surface of the skin of the upper eyelid. The deepest fibers blend with an expansion from the sheath of the rectus superior. Some fibers also attach to the upper margin of the superior tarsus and are referred to as the superior tarsal muscle.

A thickening of the sheath of the levator palpebrae superioris is referred to as the superior transverse ligament of Whitnall. It extends laterally and medially to insert in the orbital walls just behind the superior orbital rim. Medially, it attaches to the trochlea of the superior oblique muscle and to the frontal bone. Laterally, it is fixed to the capsule of the lacrimal gland and to the frontal bone.

When the levator palpebrae superioris contracts, it raises the upper eyelid. It is innervated by a superior branch of the oculomotor nerve (CN III). Interestingly, the superior tarsal muscle is innervated by postganglionic sympathetic fibers from the superior cervical ganglion. Therefore, a complete ptosis reflects the loss of oculomotor function, whereas a partial ptosis is associated with loss of sympathetic supply.[5]

The four rectus muscles arise from a fibrous ring – the common tendinous annulus of Zinn – that extends across the superior orbital fissure and

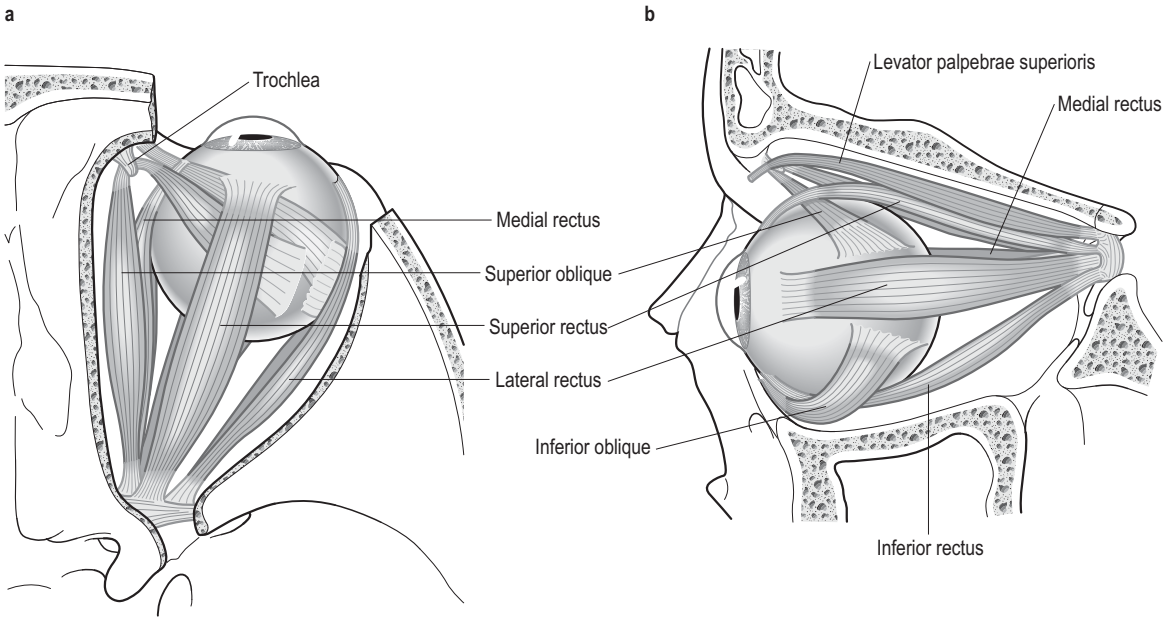

Figure 7.5.3. *Muscles of the eyeball: (a) superior view; (b) lateral view.*

surrounds the upper, medial and lower margins of the optic foramen, where it adheres strongly to the tubular dural sheath surrounding the optic nerve. The common tendinous annulus consists of two parts. Superiorly, the tendon of Lockwood gives origin to the superior rectus muscle, part of the medial rectus and the upper fibers of the lateral rectus; inferiorly, the tendon of Zinn gives origin to the inferior, medial and lateral rectus muscles. At their origin, the rectus muscles are tightly set in Zinn's annulus and start to separate at about 8 mm anterior to the optic canal.[9] The medial rectus is the broadest while the lateral rectus is the longest. The four rectus muscles go forward and insert through tendinous expansions into the sclera, the superior and inferior recti passing anterolaterally.

The superior oblique muscle originates from the body of the sphenoid, above and medial to the margin of the optic foramen and origin of the superior rectus. It passes forward, ending in a round tendon, which goes through a fibrocartilaginous ring or trochlea attached to the frontal bone. From that point, the tendon turns backward, laterally and downward beneath the superior rectus to insert into the sclera, behind the equator of the eyeball in its superolateral posterior quadrant.

The inferior oblique muscle originates from the orbital surface of the maxilla, just posterior to the orbital rim and lateral to the lacrimal groove. It traverses the floor of the orbit in a lateral, backward and upward direction, and inserts into the inferolateral posterior quadrant of the eyeball.

The EOM produce mobility of the eyeball with extreme amplitude in all directions (Fig. 7.5.4). This is because the type of attachment they demonstrate to the periorbita – an interlocking of tendinous and muscular fibers – provides them with a strong anchor.[10] The contraction of the EOM rotates the eyeball according to their insertions and the orientation of their fibers. However, in all cases it should be remembered that these muscles function together and not as isolated entities. The ocular movements of individual muscles can be simplified as follows:[5]

- The superior and inferior recti adduct the eyeball, in association with elevation and intorsion from the superior rectus and depression and extorsion from the inferior rectus.
- The medial rectus adducts the eyeball while abduction is the result of contraction of the lateral rectus.
- The superior oblique is considered to act from the trochlea.
- Both the superior and inferior oblique abduct the eye, with a component of depression and intorsion from the superior oblique and elevation and extorsion from the inferior oblique.

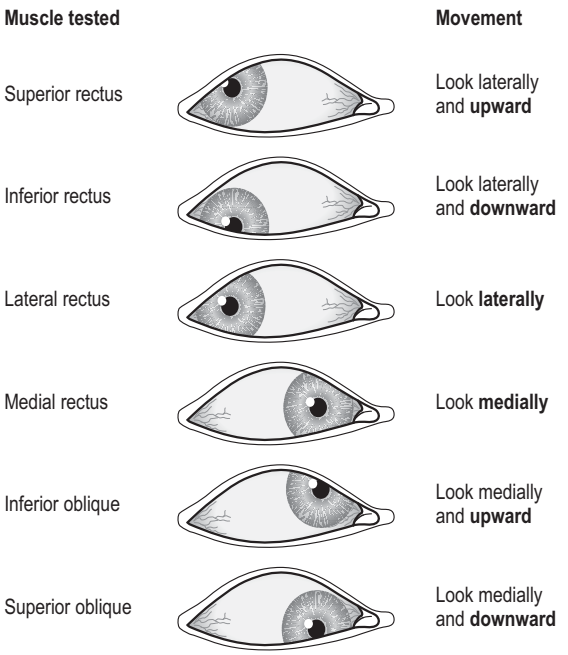

Figure 7.5.4. *Action of muscles of the eyeball.*

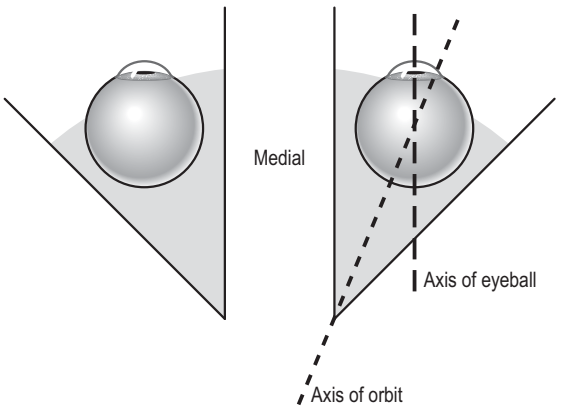

Figure 7.5.5. *Axes of the eyeball and orbit.*

It should be noted that while the longitudinal axis of the orbit deviates laterally in a posterior to anterior direction, the axis of the eyeball approximates the sagittal plane (Fig. 7.5.5). Therefore, muscular activity between medial and lateral muscles is not equal. At birth, infants tend to demonstrate intermittent ocular misalignments. This is associated with lack of maturity in visual function. It should disappear by 4 months of age when orthotropic ocular alignment and sensory binocularity should be present.[11] Transitory esotropic misalignments are usually considered to be typical in infants. However, if the condition is permanent with a fixed restriction of any of the EOM movements, or if it persists after 6 months of age, further evaluation is necessary.[12] In any case, osteopathic examination and treatment are indicated to balance the bony components of the orbit and their relationship with other parts of the skull. Dysfunction can also manifest through myofascial and membranous components. Entrapment neuropathy can result. Evaluation and treatment of these components are indicated to promote the best possible muscular activity and ocular function.

Although the study of EOM function is complex, it is imperative that it includes the orbital connective tissues that sheath the muscles. These tissues have long been recognized, the capsule of Tenon being described in 1806.[13] This capsule is a fascial sheath completely covering the eyeball. It extends from the optic nerve to the corneoscleral junction and separates the eyeball from the orbital fat. It is perforated by the tendons of the EOM on which it reflects to form tubular sheaths. Tenon described this capsule as a 'muscle pulley'.[13] After much controversy, this theory is again accepted, and further studies have established that each rectus and inferior oblique muscle passes through a pulley formed by a ring or sheath of collagen, elastin and smooth muscle that is located close to the equator of the eyeball, in Tenon's capsule.[14] In this manner, the rectus and the inferior oblique muscles have their paths constrained by pulleys that serve as functional origins for the muscles, in a fashion similar to that of the trochlea for the superior oblique muscle.[15] Thus, the position of a pulley insertion relative to the eyeball affects the forces of the EOM, and a translation of the eyeball of 1.5 mm modifies the pulling direction of the rectus muscle by 6°.[16] These pulleys are under active muscular control, allowing for constant ocular adjustments.[14] Conversely, their instability and modification of location are associated with ocular dysfunction.[16]

The EOM connective tissue sleeves that act as pulleys are firmly attached to each other and, through extensions, to the orbital walls. Those from the lateral and medial rectus are anchored to the orbital tubercle of the zygomatic bone and posterior to the lacrimal crest of the lacrimal bone, respectively. They are referred to as check ligaments. This point is of great significance. The zygomatic bone should not be overshadowed as a site of insertion for the EOM sheaths by the sphenoid on which the common tendinous annulus of Zinn inserts. In subjects with

strabismus or other ocular misalignments, the consideration of the orbital bones on which the EOM insert should include the zygomatic bone. Its location makes it vulnerable to being struck during the course of normal childhood activities. This may, in turn, affect the diameter of the orbit and the functional balance of the EOM, particularly the lateral rectus. Another site of importance is the trochlea for the superior oblique muscle on the frontal bone. Frontal dysfunctions are often found in infants, with one side lower than the other resulting in frontal trochlear asymmetry. This may be seen with a superior–medial deviation of the eye encountered with dysfunction of the superior oblique muscle. Dysfunction of the maxilla can also influence visual activity through its effect on the insertion of the inferior oblique muscle. Through these complex interactions the orbit functions as a unit and each part, when dysfunctional, is a potential site for muscular instability and resultant ocular dysfunction.

The somatic components of ocular dysfunction also involve oculomotor supply. The oculomotor nerve (CN III) exits the brain medially to the cerebral peduncles. It traverses the dura at the top of the clivus, enters the cavernous sinus and courses forward in the lateral wall of the cavernous sinus where it receives sympathetic fibers from the internal carotid plexus and connects with the ophthalmic division of the trigeminal nerve (CN V_1). It then divides into two branches that enter the orbit through the superior orbital fissure. The general somatic efferent fibers of the oculomotor nerve innervate nearly all of the EOM. The superior division innervates the superior rectus and the levator palpebrae muscles, whereas the inferior division innervates the medial and inferior rectus and inferior oblique muscles. The general visceral efferent fibers of CN III are part of the parasympathetic ANS and supply the sphincter of the iris that regulates the size and shape of the pupil and the ciliary muscle that modulates the shape of the lens.

The trochlear nerve (CN IV) is the only cranial nerve to emerge from the dorsal surface of the brainstem. It curves around the midbrain, pierces the dura between the lesser and greater circumferences of the tentorium cerebelli, enters the lateral border of the cavernous sinus, receives sympathetic fibers from the internal carotid plexus and traverses the superior orbital fissure to the orbit. It carries somatic efferent fibers to the superior oblique muscle.

The abducent nerve (CN VI) emerges from the brainstem between the pons and medulla oblongata. It pierces the dura covering the clivus and passes over the ridge of the petrous apex of the temporal bone through an osteofibrous canal underneath Gruber's petrosphenoidal ligament. It then enters the cavernous sinus where it receives sympathetic fibers from the internal carotid plexus and the orbit through the medial end of the superior orbital fissure and within the annulus of Zinn. CN VI carries somatic efferent fibers to the lateral rectus muscle.

Additionally, the EOM contain proprioceptive receptors that provide input as to the position and movement of the eye in the orbit.[17] This contributes to the control of eye's movements and facilitates central control of the direction of gaze and the relationship of the child to their environment. These afferent fibers appear to travel with the motor CN III, IV and VI before joining the ophthalmic branch of CN V to enter the CNS. Afferent input from these receptors not only affects static eye position but can also modify linear visual tracking, saccadic eye movement and the vestibulo-ocular reflex.[18]

Each of these nerves is subject to intracranial entrapment from osseous compression, membranous tension, ligamentous pull or the pressure of edema from venous congestion.[19] The anterior attachments of the tentorium cerebelli are a site with particularly great potential to cause such neurologic dysfunction. The ganglion of the trigeminal nerve may become entrapped by the tentorium in the trigeminal cave formed at the apex of the petrous part of the temporal bone. Bilaterally, the anterior fibers of the tentorium twine on each side as the fibers of the lesser circumference attach to the anterior clinoid processes and the fibers of the greater circumference attach to the posterior clinoid processes. Between these two attachments, the fibers of the tentorium cerebelli are oriented horizontally and contribute to the formation of the roof of the cavernous sinus. CN III and IV pierce the dura at this level to enter the cavernous sinus. Any dural membranous strains in these areas can result in entrapment neuropathy and each bone on which the tentorium cerebelli attaches should be considered as potentially critical in the development of ocular dysfunction. The petrosphenoidal ligament is another significant site under which CN VI may be compressed. Furthermore, as the nerve bends sharply in its course over the petrous ridge, it is, therefore, vulnerable to the changes in position of the petrous portion of the temporal bone.

Adequate arterial supply and drainage are necessary for healthy nervous function. Edema and stasis encumber this function, as is the case with a jugular foramen dysfunction and impediment of the drainage from the cavernous sinus, resulting in venous congestion. This, in turn, will affect the cranial nerves passing through the cavernous sinuses to the

orbits. The same rationale applies to the superior orbital fissure. Intraosseous dysfunction in infants between the greater and lesser wings of the sphenoid, or narrowing of the fissure because of greater wing dysfunction with surrounding structures, such as the temporal bone, impairs venous drainage or impulse conduction of the nerves passing through the fissure.[19] Additionally, lymphatic stases outside the skull can also entrap nerves at their foramina of exit.[19]

Multiple theories exist concerning the cause of non-paralytic or concomitant strabismus, including sensorimotor, anatomic and mechanical or muscular origins.[10] Prematurity[20] and difficult labor are considered to be risk factors. A supranuclear developmental abnormality in the CNS is thought to be the cause of strabismus occurring in the first 6 months of life. Impaired vision and amblyopia may also cause strabismus.

It is abnormal for an infant or child to have strabismus and, if present, a thorough ocular and neurologic examination by a specialist should be performed. Osteopathic procedures may, however, be applied to provide balance to the musculoskeletal and nervous systems. Such balance improves the self-healing capacities of the body and contributes to the success of other treatments. Osteopathic treatment should be initiated at the earliest possible time because the prognosis of strabismus is correlated to the time when the strabismus first appears and the time when treatment is initiated. Additionally, early treatment may positively impact the vestibulo-ocular reflexes and thereby the child's posture.

Physical examination and treatment

Young children and infants are most likely to present for the treatment of strabismus. As such the following treatment description must be appropriately adapted to fit the age of the patient. The following is not intended to treat organic pathologies of the eye; it is directed at problems of functional balance.

The examination and treatment for the eye should address different levels of anatomic dysfunction. These include osseous, myofascial, neurologic and vascular dysfunction and the intrinsic structure of the eye. Observation and palpation are directed at determining on which of these levels treatment should focus. Observe the relationship between the eye and the functional pattern of the skull. If the functional pattern of eye position and movement is consistent with the dysfunctional pattern of the skull, the focus of treatment should be on the cranial dysfunction. If eye position and movement is not consistent with the pattern of the skull, treatment should focus directly on the eye, EOM and associated fascial structures.

Start with observation of the bony orbit, relative to how it fits the global pattern of the skull. Look at the shape of the face and skull of the child to determine if a global pattern of cranial flexion–external rotation, extension–internal rotation, sidebending–rotation or torsion is present. Observe the orbital diameter, the distance between the superior medial and inferior lateral angles of the orbit. It is increased with cranial flexion–external rotation, resulting in an orbital cavity that is wider. Cranial extension–internal rotation decreases the orbital diameter, with a resultant orbital cavity that is narrower. Look for asymmetry between the visible constituents of the bony orbits, specifically the frontal bones, zygomae and maxillae.

Look at the eyes and observe for difference in size and shape. Cranial flexion–external rotation is associated with a prominent eyeball and an almond-shaped eye. In cranial extension–internal rotation, the eyeball is less prominent, with a smaller, rounder shaped eye. Epicanthus – a vertical fold of skin covering the medial portion of the eye – may give the impression of esotropia. Active motion testing will, however, demonstrate normal function of the eye.

Next, observe the spontaneous movements and neutral resting position of the eyes. Note the direction of gaze, the presence of esotropia or exotropia. The easiest way to evaluate the ocular movement of younger children is to hold a toy or some interesting object in front of the child to catch their attention. Move the object horizontally, vertically and in both diagonals, and observe the movement of the child's eyes as they follow the moving object. Note any asymmetric movement of the cervical spine that can be employed to compensate for the absence of ocular movement. This may be a sign of amblyopia that requires further evaluation. If possible, assess both eyes together and each eye separately by covering one eye with the child's or caregiver's hand and pretending to play 'peek-a-boo'. Comparatively note the speed and ease with which the eyes move to follow the object. Convergence may be determined by observing as the toy is brought closer to the infant's face. This procedure tests the actions of the EOM and normal responses are illustrated in Figure 7.5.4.

Determine if a correlation exists between the dysfunction of the EOM and the pattern of cranial dysfunction. The two most commonly encountered types of strabismus that are amenable to cranial

manipulation are the consequence of dysfunction involving the superior oblique muscle and the lateral rectus. A superior–medial–oblique deviation of the eye resulting from dysfunction of the superior oblique muscle may be associated with ipsilateral dysfunction of the frontal bone affecting its relationship to the muscle at the trochlea. Medial deviation of the eye resulting from dysfunction of the lateral rectus may be associated with ipsilateral temporal bone or sphenoid dysfunction that affects CN VI as it passes beneath the petrosphenoidal ligament.

Palpate for function to confirm the above observations. Tests of listening should be performed with specific attention to the anatomic structures associated with the eyes in order to define the dysfunctional area responsible for ocular deviation. Areas of specific interest include the bones on which the EOM are attached: the sphenoid, frontal, maxillae and zygomae. Assess the anatomic areas of neurovascular passage to the orbital cavity. These include the superior orbital fissure between the greater and lesser wing of the sphenoid and the cavernous sinus for their contents. Also evaluate the apex of the petrous portion of the temporal bone and the sphenoid for their relationship to the petrosphenoidal ligament. In actual practice, these assessments and the treatment of identified dysfunction flow seamlessly into one another.

Diagnostic palpation of the sphenoid, and to a lesser degree of the frontal bone, is complex because the movements of the EOM are transmitted to these bones. These additional movements on top of the biphasic PRM create multifaceted palpatory sensations that can be confusing to the inexperienced examiner. Consequently, it is desirable to evaluate the child when they are quiet with minimal eye movement. This occurs most readily when the child is sleeping. If the examiner is skilled with indirect procedures, the act of palpation will often soothe the child enough to bring them to a quiet state.

Complete examination of the sphenoid is somewhat difficult because of the limited surface area of the bone that is available for direct palpation. The areas of contact over the lateral-most aspects of the membranous greater wings, because of their flexibility, transmit an incomplete representation of the movement of the body of the sphenoid. For this reason, the lightest touch must be employed when evaluating the sphenoid and even then information about the body must be obtained indirectly. To assess the sphenoid body and associated lesser wings more effectively, visualize the relationship between the frontal bone and the sphenoid behind. The body and lesser wings of the sphenoid are in continuity with the orbital plates of the frontal bone. When palpating, one hand can be placed transversely on the frontal bone with the thumb and middle finger contacting the greater wings on either side. The movement of the lesser wings and body of the sphenoid is transmitted through the orbital plates of the frontal bone. This provides a method of augmenting the palpatory sensations from the body of the sphenoid through the frontal bone.

Similarly, the sensation of movement of the greater wings of the sphenoid may be enhanced through the zygomatic bones and their relationship with the greater wings at the sphenozygomatic sutures. These visualization procedures may be employed during both tests of listening and treatment, and require that the bones being palpated in association with the sphenoid are themselves free of somatic dysfunction.

In infants and younger children, intraosseous dysfunctions of the frontal bone and sphenoid should be considered. Listen to the movement between the two halves of the frontal bone. Palpate for deformation affecting the trochlea of the superior oblique muscle. Visualize and listen to the synchondroses between the greater wings and the body of the sphenoid.

Listen to the relationship between the greater and lesser wings at the superior orbital fissure, visualizing the neurovascular content, and try to promote the inherent forces of the PRM in the area. Similarly, assess the cavernous sinus because of its relation with the nerves and venous drainage of the eyes. Visualize the dura that constitutes the walls of the cavernous sinus and its relationship to the remainder of the reciprocal tension membranes.

Finally, if the child permits, and while utilizing the gentlest of indirect methods, palpate the globes of the eyes. Evaluate the tone and quality of the myofascial structures surrounding the eyeballs. Identify the functional point of balance between the EOM.

Treat any dysfunction as identified above, using indirect principles. Only after any osseous, membranous and fascial dysfunctions have been treated, and the EOM have been balanced, employ myofascial rehabilitation. In the patient with ocular deviation, the EOM provide dysfunctional proprioception to the CNS. The child should learn to experience the sensations of the normal range of ocular movements. Older children can be taught to move their eye while the practitioner gently applies digital contact intended to guide the eye through the normal range of ocular movements. In this exercise, particular attention should be directed at establishing awareness of the sensation of eye movements in

the directions that they are deficient. The process of myofascial rehabilitation is accomplished with greater ease once pre-existing osseous, membranous and fascial dysfunctions have been treated.

ASTIGMATISM, MYOPIA, HYPEROPIA

A description of the anatomy of the bony orbit and the EOM has already been provided in 'Strabismus' above. We shall, therefore, only consider the description of the eyeball and somatic dysfunction as they relate to the pathophysiology of astigmatism, myopia and hyperopia.

The eyeball occupies the anterior part of the cavity of the orbit. It is surrounded by the EOM and embedded in the fat of the orbit. Two spheres of different diameters form the eyeball. The anterior cornea is smaller, transparent, bulging outward and represents approximately one-sixth of the eyeball. Posteriorly, a larger opaque sphere constitutes the rest of the eyeball.

Anatomically, the eyeball may be described as being formed by three tunics or walls that are, from front to back, the fibrous tunic, the vascular pigmented tunic and the nervous tunic, and by internal components or refracting media, the aqueous humor, the vitreous body and the crystalline lens.

The outside fibrous tunic of the eyeball is formed by the sclera and cornea. In the front, the sclera is continuous with the cornea at the sclerocorneal junction. The sclera is a dense and firm membrane that preserves the shape of the eyeball. It is covered with a fascial membrane, the capsule of Tenon, posteriorly from the optic nerve to the corneoscleral junction anteriorly. This fascial membrane reflects onto each of the EOM tendons as they perforate Tenon's capsule to attach on the sclera. Posteriorly, the sclera is pierced by the nervous filaments of the optic nerve (CN II), forming the lamina cribrosa sclerae. The sclera is continuous with the fibrous sheath of CN II and, therefore, with the meningeal layer of the dura mater. Several small apertures exist around the lamina cribrosa sclerae for the ciliary vessels and nerves. The central artery and vein of the retina pass through CN II.

The cornea projecting in front of the sclera is almost circular, with its width slightly greater than its height. Its anterior convexity demonstrates varying degrees of curvature throughout life and between individuals. A rapid change in the corneal curvature occurs during the first 2–4 weeks of life. This rate of change then decreases considerably after the 8th week.[21] The corneal curvature usually stabilizes at approximately the end of the 1st year of life.[5] Its coating of tears constitutes the most important site of refraction of the eyeball.

The middle tunic of the eyeball is a vascular pigmented layer consisting of the choroid posteriorly and the ciliary body and iris anteriorly. The choroid is a thin, highly vascular membrane that constitutes approximately the posterior five-sixths of the globe. Its outer surface adheres firmly to the sclera while its inner surface is attached to the retina. This layer provides a significant percentage of the vascular supply to the retina.

The ciliary body consists of the ciliary processes and the ciliary muscle. The ciliary processes are circularly arranged as a ruche behind the iris surrounding the lens. They are continuous at their periphery with the layers of the choroid and anteriorly with the periphery of the iris. Posteriorly, they are connected with the zonule that is the suspensory ligament of the lens. The ciliary muscle plays an important role in accommodation. It adjusts the shape of the lens in order to change the focus of the eye. When the ciliary muscle contracts, it draws forward the ciliary body, thus reducing tension in the fibers of the suspensory ligament. This results in a relaxation of the lens' capsule with resultant increased convexity of the lens. The parasympathetic stimulation of fibers from CN III produces contraction of the ciliary muscle.

The iris is a thin, circular, contractile disk, located between the cornea and lens, and continuous peripherally with the ciliary body. The iris differs in color among individuals, and at birth is light blue because of a lack of pigment. Iris pigmentation is well developed at 6 months of age. The iris divides the space between the cornea and lens into an anterior and a posterior chamber that communicates through the center of the iris, the pupil. Pupillary dilatation or constriction results from the contraction of the dilator or sphincter pupillae. Responses to light and accommodation produce the two dominant pupilloconstrictor reflexes. Axons from preganglionic parasympathetic fibers course with CN III to the ciliary ganglion, located behind the eyeball. From there, postganglionic fibers supply the smooth muscle of the iris; this may result in pupillary constriction. Inhibition of the tonic activity of the oculomotor system in the midbrain Edinger–Westphal nucleus leads to pupillary dilatation. Extreme pupillary dilatation is referred to as mydriasis, extreme constriction as miosis.

The nervous tunic, i.e. the retina or inner layer of the walls of the eyeball, is truly a sensory extension

of the brain. During embryogenesis, the optic vesicles develop from the lateral aspects of the forebrain. When they invaginate, forming two optic cups, the inner walls of the cups become the retinal sensory mesh and send axons back to the optic stalk. On activation of the receptors of this sensory stratum, brain activity starts the visual sensory processing. Light and images of external objects are received on the retinal receptors, stimulating a chemical reaction and action potentials transmitted through the optic nerve to the visual cortex within the occipital lobe.

The vascular supply of the retina comes partly from the choroid that is in contact with the external layer of the retina. Internally, the retina lies in contact with the vitreous body. The thickness of the retina varies, its thickest portion being located in the back of the eye, near the central retinal area or macula lutea. The fovea centralis, the center of the macula lutea, is a point of maximum vision and could be considered as the posterior point of the eyeball's axis. It is approximately 3–4 mm lateral and 1 mm inferior to the center of the optic disc, a point where the retina is insensitive to light, referred to as the blind spot. The macula continues to develop until 16–18 weeks after birth. Fully developed visual acuity is normally established by 2 years of age.[22]

Light passes through various refracting media in the eye, the aqueous humor, the lens and the vitreous body. The aqueous humor fills the anterior and posterior chambers of the eyeball. It is secreted into the posterior chamber by the ciliary processes. It flows into the anterior chamber through the pupil and is absorbed into the scleral venous sinus between the iris and cornea. The lens is situated directly behind the iris and in front of the vitreous body. It is a transparent, biconvex structure surrounded by a capsule and is connected to the ciliary muscle through the suspensory ligament. It measures approximately 6 mm in diameter at birth and from the first years of life offers excellent refractive power. As the child grows, more layers are added on the lens periphery, and around 14 years of age it reaches its adult size. From then on, progressively, it will loose its hydration and thereby its flexibility and refractive power. Accommodation, normally occurring when tension of the lens' capsule decreases, becomes more difficult. This leads, for most individuals in their fifties, to the need for glasses to compensate. The postrenal (vitreous) chamber forms about four-fifths of the eyeball and is filled with the vitreous body (vitreous humor) that is situated between the retina and the lens. It is transparent, of a gel-like consistency, and is composed of about 99% water.

When light enters the eye, the cornea and crystalline lens normally focus the rays of light through accommodation specifically on the retina. When properly functioning, this is referred to as the emmetropic state and requires that normal ocular growth occurred resulting in normal eye biometry.

The central point of the anterior curvature of the eyeball is referred to as the anterior pole; the central point of the posterior curvature is the posterior pole. A line joining the two poles is referred to as the optic axis. The optic axes of the two eyeballs are almost parallel, approximating in the sagittal plane, while the longitudinal axes of the orbits are directed forward and laterally (Fig. 7.5.5). Therefore, for each eye, these two axes are aligned in different directions. The optic nerves enter the orbit with the ophthalmic artery, through the optic canal, and follow the direction of the axes of the orbits. This explains why the optic disc is medial to the fovea centralis in the center of the macula lutea. The transverse and anteroposterior diameters of the eyeball are slightly greater (24 mm) than its vertical diameter (23.5 mm). At birth the anteroposterior diameter is about 17.5 mm and at puberty is between 20 and 21 mm.

Ocular growth and refraction are dynamic processes evolving during infancy and childhood, well through adult life. Color vision develops rapidly after birth and in most individuals some color vision is usually present at 3 months of age.[23] Although difficult to evaluate in infants, normal visual acuity is thought to be present by 2 years of age.[22] Even though complete anatomic and functional maturity of the visual system is reached around 10 years of age, many important milestones occur during the first 2 years of life. For these reasons, when examining and treating infants and children, an osteopathic practitioner should pay close attention to any somatic dysfunction that may potentially alter the normal shift of the refractive state of the eye to emmetropia. The sites of dysfunction with potential for alteration of the visual system are the different bony components of the orbit, the eyeball, EOM and surrounding fascial and dural sheaths. Because the vestibulo-ocular reflexes link the individual's posture with ocular function, the axial skeleton and postural balance of the child should also be addressed.

Any disturbance in the visual components may alter vision, usually asymmetrically. If this occurs during the developmental process, the resulting absence of stimulation in the visual part of the cortex will cause partial loss of sight, or amblyopia. This could be associated with an important difference of visual acuity between both eyes. Normally

orthotropic ocular alignment and sensory binocularity should be present by 4 months of age.[11] If there are signs of misalignment and lack of sensory binocularity, further evaluation should be carried out.

Emmetropization allows a distant object to be projected as a focused image on the retina without accommodative effort.[24] This is possible when the optical power and the axial length of the eye are balanced. Normally, in the emmetropic eye with relaxed accommodation, objects are focused on the retina. If the point of focus falls behind the retina, increased accommodation is necessary to bring the point of focus on the retina. This occurs with hyperopia and has been associated with decrease of the axial length of the eyeball. Conversely, in myopia, the point of focus falls in front of the retina and this is associated with increase of the axial length of the eyeball (Fig. 7.5.6). Some compensation usually occurs in these conditions, since eyes with decreased axial length are associated with a more rounded cornea, whereas increased axial length is associated with flattening of the cornea.

Typically, infants demonstrate hyperopic eyes that tend to improve with ocular growth and, by the age of 5–8 years, they are emmetropic. By age 6 years, only a few children are myopic, but between the ages of 6 and 7 years, myopic children have the fastest progression of all age groups.[25] Myopia is a common condition affecting approximately 4.4% of Caucasian children, 13.2% of Hispanics and demonstrates the highest prevalence in Asians (18.5%).[26] This condition and its progression are influenced by

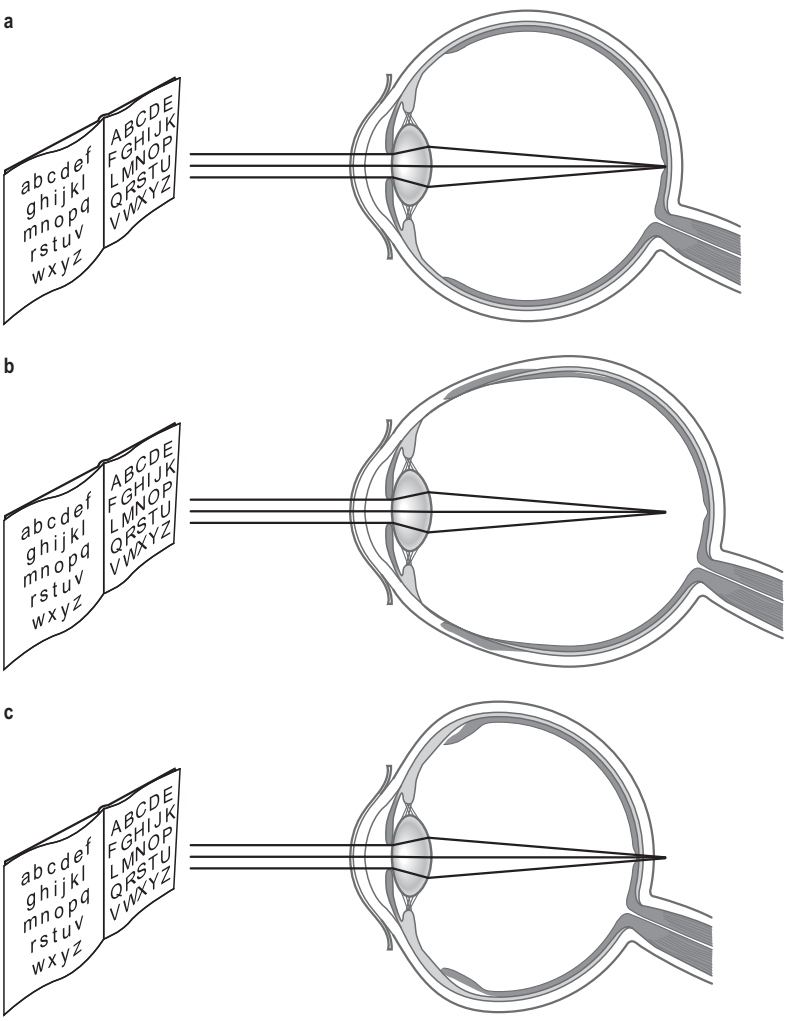

Figure 7.5.6. *Accommodation: (a) in the emmetropic eye, objects are focused on the retina; (b) in myopia, associated with increase of the axial length of the eyeball, the point of focus falls in front of the retina; (c) in hyperopia, associated with decrease of the axial length of the eyeball, the point of focus falls behind the retina.*

various factors such as demography and the environment[27].

The etiology of myopia seems to be multifactorial, with a genetic component since myopic individuals are more likely to have myopic parents.[29] A greater reading exposure in childhood has often been suggested as a contributing factor and is especially associated with myopia in Asian children, more often encountered around 7–9 years.[30]

Intrinsic ocular factors are also associated with myopias. Greater dimensions are present in myopic eyes than in emmetropic eyes when the vitreous chambers are measured along the axial direction, i.e. the optical and visual axes.[31,32] Heavier newborns with large heads have longer optical axial lengths, deeper vitreous chambers and flatter corneas. However, refraction is maintained and satisfactory, suggesting the presence of accommodative mechanisms in the cornea that compensate for the longer axial length.[24] Myopia usually develops because of a lack of coordination between postnatal growth and the refractive power of the eyeball components. There is some probability that the CNS influences the development of myopia.[28]

Unequal curvature(s) along the meridians of the refractive surfaces of the eye – cornea, anterior or posterior surfaces of the lens – result in astigmatism. Thus, when light enters the eye, instead of having the cornea and the crystalline lens focusing the rays of light at a single point on the retina, they are spread out as a line in one direction or another. The orientation of the axis of corneal astigmatism is affected by the mode of delivery. Infants delivered vaginally when compared to those delivered by cesarean section demonstrate a higher frequency of with-the-rule astigmatism, with the greater curvature of that astigmatism tending to be in the vertical meridian.[33] These astigmatic corneas usually flatten significantly by the age of 6 months.

Because astigmatism, myopia and hyperopia are associated with anatomic variations in the biometry of the eye, any form of treatment that may affect the refractive power and shape of the eye is desirable. This is particularly important in children less than 1 year of age, when the eye is still growing and the ocular reflexes are not totally developed. Prevention of the ocular complications of myopia, such as retinal detachment and glaucoma, may be facilitated by relaxation of tension in surrounding ocular structures. Addressing somatic dysfunction of the upper thoracic and cervical vertebrae may further assist relaxation of ocular tension. For these reasons, osteopathic procedures may be applied directly to the eyeball itself or the structures surrounding the eyeball including the orbit and its myofascial contents. Procedures may also be employed to affect the control of refractive mechanisms through the impact that the ANS has on the intrinsic ocular muscles.

Physical examination and treatment

The osteopathic structural examination of the eye and orbit for the patient with astigmatism, myopia and hyperopia is essentially the same as that described above for strabismus. The patient should be thoroughly evaluated for dysfunction affecting the component bones of the orbit, the EOM and associated fascial sheaths. In addition, because these ocular conditions involve changes in the shape of the eyeball, the physical evaluation of the individual with myopia or hyperopia should specifically include the search for any somatic dysfunction that affects the axial length of the eyeball. For the patient with astigmatism, somatic dysfunction responsible for changes in orbital shape and tension of myofascial structures that affect the curvatures along the meridians of the refractive surfaces of the eye should be identified.

Cranial flexion–external rotation is associated with a decrease of the orbital depth, whereas extension–internal rotation is associated with increased orbital depth. For these reasons, potentially contributory craniosacral somatic dysfunction should be sought out and treated. This includes dysfunction involving the SBS and bony components of the orbit as well as all components of the craniosacral mechanism, including, but not limited to, the sacrum and pelvis. A posture in which the child projects their head forward may be the result of poor sight. On the other hand, dysfunction affecting the posture, because of its effect on the cervical myofascial structures, particularly those attaching to the base of the skull, will tend to impair the inherent motility of the skull and potentially the components of the orbit. A craniosacral somatic dysfunction of the sacrum in flexion will produce a decrease of spinal AP curves and a tendency toward cranial flexion. This can, in turn, favor decreased orbital depth that is particularly significant in infants and young children at a time when ocular growth is occurring. As such, a complete postural assessment is appropriate when examining a child with ocular dysfunction.

It is also appropriate in these individuals to begin working on the global postural pattern because vestibulo-ocular reflexes link posture with ocular function. Using an indirect approach, begin with the treatment of postural dysfunction.

Because of their action on the PRM, the use of indirect techniques will relax the patient. The

treatment of ocular dysfunction requires the most delicate touch and as the patient relaxes it becomes easier to treat the eyes.

After addressing the global postural pattern, proceed to the examination of the neurocranium and viscerocranium. Assess the SBS with tests of listening, then progress to observe the size and shape of the orbit. Look at the relationship of the eyeball to the orbit. Prominence of the eyeball is associated with a wider orbit and a pattern of cranial flexion–external rotation, whereas a recessed eye is associated with a narrower orbit and cranial extension–internal rotation. Also observe the patient in profile bilaterally, comparing the amount of corneal curvature.

Determine if the dysfunctional mechanics identified at the SBS is consistent with the pattern of orbit and eyeball. If so, treat the SBS; if not, look for dysfunction in the orbit. (*Note*: In infants the state of development of the frontal bone with small brow ridges causes the eyeballs to appear as if they are bulging.)

Next, assess the different bones that constitute the orbit, particularly the frontal bone, zygomae, maxillae and sphenoid. The effect of cranial dysfunction on the depth and shape of the orbit is significant in astigmatism, myopia and hyperopia. The apex of the orbit is situated at the medial end of the superior orbital fissure between the lesser and greater wings of the sphenoid, near the origin of the EOM and inferior to the optic foramen. Thus, the length and direction of the orbital axes are influenced by the sphenoid. Dysfunctions of the sphenoid that can affect the orbit include dysfunction of the SBS, dysfunction between the sphenoid and the bones articulating with its wings and intraosseous dysfunction. With cranial flexion the apex of the orbit moves forward, thus decreasing the AP orbital diameter, whereas in extension the apex is displaced posteriorly, increasing the AP orbital diameter. With SBS torsion or sidebending–rotation, on the side of associated external rotation, the AP orbital diameter is decreased and on the side of associated internal rotation it is increased. Intraosseous dysfunctions of the sphenoid have variable effects on the orbit and eyeball. They must be identified and treated as early as possible, sphenoid ossification being essentially complete by 1 year of age. Dysfunction of the frontal bones, zygomae and maxillae, either individually or in conjunction with the sphenoid, can affect the shape of the orbit and, when present, should be treated using indirect principles.

An extension of the dura surrounds the optic nerve and is attached to the optic canal. Intraosseous dysfunction of the sphenoid can affect the cylindrical optic canal that is formed where the two roots of the lesser wing of the sphenoid join the sphenoidal body. The dura also extends through the superior orbital fissure and blends with the orbital periosteum. Consequently, membranous strain can affect the sphenoid and through the sphenoid the shape of the orbit. Dural membranous dysfunction should thus be sought out and treated at the youngest possible age.

To ensure autonomic balance for the orbital contents, the rhythmic motility of the orbit and neurocranium is necessary. Change of the AP diameter of the orbit is associated with altered tension of the intraorbital soft tissues with resultant stasis, edema and compression. This, in turn, will affect the function of nervous structures, in this case CN III and the ciliary ganglion. Treatment of regional dysfunction, ensuring the rhythmic flexion–extension of the PRM in the orbit, fissures and foramina, provides a pumping action mobilizing the extracellular fluids surrounding the nerves and facilitating their function.

The sympathetic supply to the orbit originates in the upper thoracic spine and, through the upper cervical ganglia, reaches the ciliary ganglia via the carotid plexus. Somatic dysfunction affecting these structures in the upper thoracic and cervical spine and the cranial base should be sought out and treated to ensure optimal sympathetic function.

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7.6 DIGESTIVE TRACT DYSFUNCTIONS

SUCKING AND SWALLOWING DYSFUNCTIONS

Sucking and swallowing are complex activities that are partially conscious and partially unconscious. They require the participation of many structures, including a significant percentage of the cranial nerves, and should be considered in the context of a sensory–motor complex that begins to develop well before birth.

Swallowing appears in utero after 11 weeks[1] with movements of the fetal tongue visible after 14 weeks. At that time, fetuses suck their thumbs reflexively in response to oral stimulation from the digital extremities.[2] Protrusion of the upper lip appears after the 17th week of gestation and protrusion of the lower lip at the 20th week. The real activity of sucking is seen at the 24th week. After that time, sucking and swallowing become more and more developed and appear as coordinated movements around 33 weeks.[1] At term, the fetus swallows about 500–900 ml of amniotic fluid per day. This daily exercise stimulates the maturation of oral structures, including the tongue, lips, soft palate and pharynx, and, therefore, prepares the fetus to perform the orofacial functions that will be vital at birth.

Sucking is one of the primitive reflexes that are totally present at birth in term infants. In the first days of life, oral feeding is almost entirely reflex, apparently without suprabulbar activity for rooting, latching, sucking and swallowing.[3] These reflexes are functionally important in infants to ensure successful ingestion. The primitive sucking reflex lasts for about a year, and then becomes more

difficult to obtain because of central nervous system maturation and the development of voluntary motor activity.

The tongue is one of the major participants in the process of sucking. It is a muscular structure with intrinsic and extrinsic muscles. The intrinsic muscles allow the precise movements necessary for sucking, swallowing and speech. They are the superior longitudinal, inferior longitudinal, transverse and vertical muscles. The extrinsic muscles originate from other parts of the skull and hyoid bone, and consist of four paired muscles:

- the genioglossus muscles originate from the mandible, attach to the hyoid bone and blend with the intrinsic muscles of the tongue
- the hyoglossus muscles originate from the hyoid bone and insert into the tongue
- the styloglossus muscles originate from the styloid processes of the temporal bones and insert into the lateral part of the tongue
- the palatoglossus muscles originate from the soft palate aponeurosis and insert into the lateral part of the tongue.

A midline fibrous sagittal septum divides the tongue into two halves and attaches to the body of the hyoid bone. Additionally, on the undersurface of the tongue, the frenulum linguae – a vertical fold of mucous membrane – connects the tongue to the floor of the mouth. As such, although anchored at its root, the tongue is mobile, allowing the different functions of sucking, suckling, latching, swallowing and eating, and later speech.

In infants, the tongue is totally contained in the oral cavity, although it is quite wide in proportion to the container and protrudes beyond the alveolar gum pads. However, its development is far from being complete and its position will go through many changes before the end of adolescence. One of the biggest changes occurs in the first years of life, as the child grows, the posterior third of the tongue descends following the larynx.[1]

When evaluating tongue malposition in children, the bony attachments of the tongue muscles should be evaluated for somatic dysfunction. Dysfunction of the mandible, the temporal bones through the styloid processes and the hyoid bone may potentially interfere with the position of the tongue. One of the most common lingual dysfunctions is forward and inferior displacement of the tongue. This predisposes to malocclusion and mouth breathing. Mouth breathing and a low posture of the tongue are also associated with an inferoposterior displacement of the anterior convexity of the hyoid bone.[4–6]

To ensure the lingual mobility necessary in the infant for feeding and the development of proper speech, the frenulum linguae should not be too short. Ankyloglossia, or tongue-tie, is a congenital anomaly characterized by an unusually short frenulum linguae. Breastfeeding is more difficult in the presence of ankyloglossia, and children have more difficulty pronouncing lingual and sibilant sounds such as T, D, Z, S, TH, N and L.[7] Furthermore, the tongue affects the growth and development of the maxillae and mandible and, therefore, the teeth in the alveolar processes, acting as a natural orthodontic appliance 'for better or for worse'.[8] Its position should be unencumbered as early in life as possible in order to play a satisfactory functional role. As such, clipping of the frenulum (frenuloplasty) may be necessary in association with osteopathic procedures to ensure a correct tongue placement.

Sucking has been classified in two categories: nutritive and non-nutritive sucking (NNS).[9] Nutritive sucking results in the delivery of milk and exhibits a slower rate of sucks that are continuous, whereas NNS consists of a series of bursts of rapid sucks, approximately twice the rate of nutritive sucking, followed by rest periods. During breastfeeding, before the initiation of sucking, the tongue protrudes beyond the lower gum and then retracts, repeating this protrusive and retrusive displacement of the tongue to draw the nipple into the mouth. If the newborn recognizes the odor of the breast's nipple, frequent sucking is initiated that, in turn, will stimulate the mechanism of lactation. The mother's milk odor results in increased sucking and stronger pressure than when the infant is fed formula or water.[10] Successful sucking is produced by a peristaltic wave in the medial portion of the tongue moving from the front to the back of the tongue.[11,12] This is the result of successive contraction and relaxation of the genioglossus and transverse muscles of the tongue. At the same time, a repetitive thrust of the mandible participates in the compressive action on the nipple to produce milk. Sucking results from the combined actions of the masseters, orbicularis oris and buccinator muscles, as well as from the muscles of the tongue and the mandible, in particular the lateral pterygoid muscles. This activity of the pterygoid muscles is important. At birth the pterygoid processes of the sphenoid are not fully developed, and because the activity of sucking requires a significant effort from the infant, the stimulation produced contributes to their development.

At approximately the 10th month of life, a new sucking pattern appears. It resembles that of later years, as when sucking from a straw, and demonstrates less movement of the tongue.[13]

Reflexes are of paramount importance in the actions of the oral cavity. They facilitate various vital functions, including suckling and swallowing, but also breathing, eating and drinking. Sucking elicits a swallowing reflex in the infant by stimulating the lips and the oral cavity. This reflex results from sensory input that induces a series of motor responses. It is mediated through neural receptors located on sensory fibers, the afferent pathway of sensory fibers, central synaptic connections and interneurons, and the efferent pathway composed of motoneurons or autonomic postganglionic neurons innervating the effector organ. In a somatic reflex, the effector is the striated muscle innervated by the alpha motoneuron. In an autonomic reflex, the effector includes a peripheral neuron extending its postganglionic axon to visceral structures. In sucking and swallowing, the sensory receptors – the tongue, gums, palate and pharynx – are located in the oral cavity. The sensory input passes through the superior laryngeal nerve, trigeminal nerve (CN V) and glossopharyngeal nerve (CN IX) to the brainstem, in the nucleus tractus solitarius (NTS) and adjacent reticular formation. From there, second order neurons engage brainstem deglutitive pharyngeal and laryngeal motoneurons in the nucleus ambiguus and in the facial, trigeminal and hypoglossal nuclei. The palate and palatopharyngeal muscles respond, separating the mouth from the nasal cavity to prevent passage of food from the oral cavity to the nasal cavity during the swallowing process.[14]

The integrity of the infant's oral cavity is of significance when considering the development of orofacial reflexes. The concept that structure affects function, and vice-versa, is perfectly illustrated in this area. The different structures that constitute the oral cavity evolve as the infant is developing, thus producing changes in function that will, in turn, further affect those structures. Conversely, malposition of any of the structures contributing to the oral cavity, such as lingual malposition, will affect function and consequently viscerocranial development in infants and children.

Breastfeeding differs from bottle feeding. Breastfed infants have better control of the amount of milk obtained than bottle-fed infants. Bottle feeding is greatly influenced by the force of gravity, with higher volumes resulting in faster flow of milk. In order to control this increased fluid flow, the newborn tends to adapt with a lingual and mandibular posture that is less protrusive. This decreases muscular participation and bottle-fed infants demonstrate a reduction of masseter muscle activity.[15] The positive effect of muscular traction on bony development and thus on orofacial development[16] is decreased and may, in turn, contribute to malocclusion.[17] The type of nipple employed during bottle feeding, i.e. its material, density and location of holes, requires different adaptive patterns from the infant. Most of the time, the root of the tongue does not come up to the tip of the nipple, as is the case with breastfeeding, but rather demonstrates a more posterior position.

Sucking is considered to be a determining factor in orofacial morphogenesis. It influences mandibular growth through the traction of myofascial attachments. The upper head of the lateral pterygoid muscle inserts into the developing condyle of the mandible. Active sucking as produced by breastfeeding is considered to be of paramount importance in mandibular development. It contributes to the change from the retrognathic mandibular posture seen in infants to a more centered position at 1 year of age.[18,19] During breastfeeding, the shape of the breast and the sucking pressure of the infant's tongue against their palate has a modeling effect on the palate. The pressure of the tongue on the anterior portion of the palate produces a pattern of cranial flexion–external rotation. The rhythmic sucking pressure on the infant's palate results in a pumping action that may contribute to the balancing of their skull and cranial mechanism. Additionally, breastfeeding seems to have a positive effect on orofacial morphogenesis,[20] and a protective effect for the development of posterior cross-bite in deciduous dentition.[21]

Human milk is a complex fluid that contains more than 200 components and is highly effective for the health and wellbeing of the infant.[22] Breastfeeding provides multiple benefits, such as decreased gastrointestinal and respiratory infections in childhood,[23] in particular otitis media, as well as protection against atopic disease.[24,25] It also seems to reduce pain in infants[26] and to enhance cognitive and visual development, as well as to have a potentially positive impact on the mother's health.[22]

Breastfeeding is thus recommended as the optimal source of nutrition for infants during the first 6 months of life. Although thought of as a natural process, it is not always easy, and some infants demonstrate difficulties. Such is the case for preterm infants, or infants with developmental anomalies such as micrognathia or ankyloglossia. Nursing difficulties may also be related to developmental anomalies of the mother's breast or variations of

nipple size or shape that reduce the effectiveness of sucking. When developmental anomalies are not present, but nursing difficulties persist, an evaluation for somatic dysfunction should be performed. The oral structures, including the mandible, should be assessed, and the function of the tongue should be checked. The intrinsic muscles of the tongue are all innervated by the hypoglossal nerve (CN XII), as are all the extrinsic muscles, with the exception of the palatoglossus muscle which is innervated by the vagus nerve (CN X). The hypoglossal nerve leaves the skull through the hypoglossal canal, situated in the anterior intraoccipital synchondrosis, between the basiocciput and the exocciput. The hypoglossal nerve may be subject to compressive forces during a difficult labor, with consequent impact on tongue motor function and resultant suckling difficulties.

Feeding is a highly complex activity in young infants since it requires the integration of sucking, swallowing and breathing. Normally, coordination of the buccopharyngeal functions takes place by 35 weeks postconceptional age,[14] although reflex swallowing occurs when sucking stimulates the lips and the oral cavity. A spoon of food placed on the tongue of term infants will also produce a reflex where the tongue pushes against the spoon.[1] It is only around the 4th or 5th month of age that infants open their mouth when a spoon is brought before them or touches their lips, and approximately 2 weeks later that they use their tongue to move food to the back of the mouth to swallow.[27]

The mechanism of deglutition is divided into oral, pharyngeal and esophageal stages. The oral stage occurs when the milk or food is in the mouth and when the child tastes, plays, experiments with the food and forms a bolus to be pushed into the pharynx and esophagus. At that time, the anterior part of the tongue presses against the hard palate, while in infants the buccinator muscles participate with resultant suction. Next, the tongue propels the bolus in the back of the mouth into the pharynx and the swallowing reflex is elicited. Multiple receptors around the opening of the pharynx are stimulated by the bolus, resulting in sensory impulses conducted to the swallowing center via CN V, IX and X. Consequent motor activity is triggered by CN IX and X.

During this pharyngeal stage, while the tongue propels the bolus posteriorly, the hyoid is displaced anteriorly, the larynx superiorly and anteriorly toward the base of the tongue, and the epiglottis moves to cover the superior opening of the larynx. The displacement of the epiglottis and the approximation of the vocal cords of the larynx combine to prevent the passage of food into the trachea. Thus, the bolus passes into the pharynx, dividing around the epiglottis while the soft palate moves against the posterior pharyngeal wall to close the nasopharyngeal entrance. The stylopharyngeus muscles draw the sides of the pharynx superiorly and laterally. This stretches the opening of the esophagus, and the bolus, always under the influence of gravity and contractions of the pharyngeal constrictors, enters into the esophagus. This third stage of the mechanism of swallowing – the esophageal phase – is totally under the influence of the ANS via the vagus nerves and the cervical and thoracic sympathetic ganglia.

Several differences in the coordination of swallowing are observed between infants and adults. Human infants, like non-human primates, demonstrate the ability to breathe and suckle simultaneously with swallowing phases between respirations (Fig. 7.6.1). They will keep this ability until approximately 2 years of age when the neuromuscular system matures. At that time, the larynx and tongue have completed their descent and the posterior third of the tongue is more vertical, forming the upper anterior wall of the pharynx.[28] The larynx and hyoid bones are involved in the descent, with separation of the epiglottis away from the uvula of the soft palate. The tip of the epiglottis is located at the level of the first cervical vertebra at 4 months of age and at the level of the third vertebra between 12 and 18 months of age.[29]

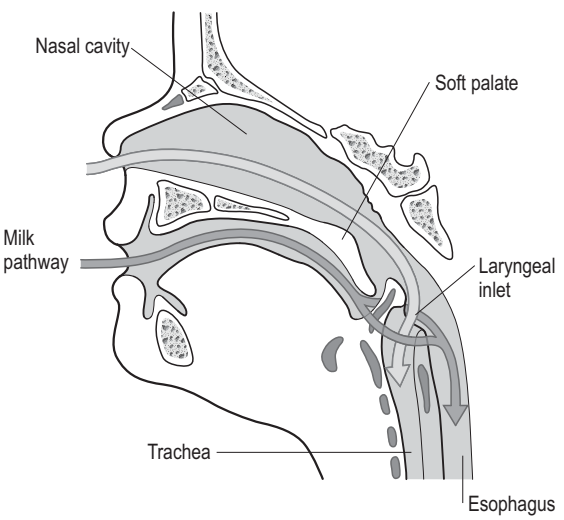

Figure 7.6.1. *Swallowing in a newborn child.*

In infants, swallowing is immature, different from that of the adult and characterized by an important anterior displacement of the posterior pharyngeal wall. During immature swallowing, the tongue protrudes over the lower lip with active contraction of the facial muscles, in particular the orbicularis oris and mentalis muscles. With progression of time, the teeth erupt in surroundings that can have an influence on their development, as much as they can influence the adjacent structures. Therefore, with maturation, the tongue rests lower in the mouth and is located between the teeth. Now, during swallowing, the tip of the tongue is posterior to the incisors, the lips are sealed and facial muscles relaxed. Infantile or immature swallowing should disappear around 2–3 years of age, and the positioning of the tongue against the palate will contribute to the development of a broad palate and a well-developed facies.

The act of swallowing is complex, involving the lips, jaw, tongue, palate, pharynx, larynx and esophagus, and each of these structures may be affected by somatic dysfunction. Conversely, the functions of sucking and swallowing are of paramount importance for the development of structures to which they are related. Oropharyngeal development is influenced by the functional posture of the tongue, lips and jaws.

The pharynx is attached superiorly to the base of the skull, the pterygoid processes of the sphenoid bone, the petrous portions of the temporal bones and the pharyngeal tubercle of the occipital bone. Laterally, the pharynx is connected to the styloid processes of the temporal bones and posteriorly to the cervical spine. Consequently, any dysfunction of the cranial base, cervical spine, hyoid bone and mandible should be considered when evaluating dysfunctional swallowing. Craniocervical dysfunctions are very frequently associated with an anterior displacement of the tongue, predisposing the individual to tongue thrusting. Furthermore, because CN IX and X trigger the motor activity of swallowing, the relationship between the occiput and temporal bones should be assessed for its effect on the jugular foramina.

The process of maturation of swallowing is not always established, leading to swallowing dysfunction or immature swallowing. Many individuals swallow with their tongues barely touching the palate or with their tongues between their teeth, in a dysfunctional manner. Swallowing occurs between 1500 and 2000 times a day, and, when dysfunctional, the thrusting force applied on the incisors with every swallow results in labial tilting. The child or adolescent who swallows immaturely also demonstrates some contraction in the orbicularis oris and mentalis muscles, failure to contact the molars and tongue protrusion when swallowing.[30] Furthermore, they have a narrow, high, palatal arch, and thus do not benefit from the pumping action of the tongue on the palate, and the palatine aponeurosis, which is suggested as being necessary for aeration of the pharyngotympanic tube (Eustachian tube).[31]

Physical examination and treatment

Begin by observing the face, specifically noting the proportion between the upper, middle and lower thirds of the face. The lower two regions are proportionately smaller in newborns than in older children, adolescents and adults. Observe the tonicity of the lips and note whether or not pursing is present. Observe the tonicity of the perioral musculature.

Have the child open the mouth and observe the position of the tongue; note any protrusion and if the tongue is at rest or demonstrates slight muscular activity. Assess the length of the frenulum linguae. It may be too short, inserting near the tip of the tongue, sometimes giving the tongue a heart-shaped appearance due to it.

Observe the movements of the tongue and, if the child is old enough, have them protrude the tongue to note its mobility. Ask them to pretend they are going to touch the tip of their nose with their tongue to observe its undersurface. With severe ankyloglossia, the tongue will tend to curl under during protrusion and will not be able to protrude because the tip is tied down by the short frenulum.

In infants, test the suck response by introducing a cotted finger, with the palmar surface in the direction of the hard palate, into the infant's mouth and allow them to suckle. The infant should respond by latching tightly. Feel coordination and strength of tongue movements.

Note any oral habits, such as tongue sucking, thumb or finger sucking, pacifier sucking or nail biting. When present, observe the position the child assumes when sucking a finger, thumb or pacifier and note in what direction the object being sucked is oriented. These positions will usually be taken to reinforce a dysfunctional pattern. For example, placement of an index finger relatively vertically in the mouth is associated with a narrow, high, palatal arch. Thumb sucking with the index finger curled over the nose is consistent with a cranial pattern in extension because the ethmoid bone is placed in anterior rotation as during extension.

Utilizing tests of listening, assess the cranial base, noting the movement of the occiput and the temporal bones. Intraosseous dysfunction of the occipital bone at the anterior intraoccipital synchondrosis will compress CN XII in the hypoglossal canal, while interosseous dysfunction between the occiput and temporal bones can affect the jugular foramen, entrapping CN IX and X.

Also examine the temporal bones because they affect the position of the mandible and because of the insertion of the styloglossus muscles on the styloid processes. Assess the mandible, temporomandibular joint, hyoid bone and cervical spine. These areas should be balanced to ensure satisfactory function of the associated myofascial structures.

Patient education and advice to caregivers

Patient education should be approached in three steps:

1. Instruct them to consciously keep their tongue in their mouth.
2. They must learn to swallow with their lips closed.
3. While keeping their lips closed, they must learn to swallow with the tip of their tongue resting against the palate behind their upper incisors.

An exercise to accomplish this is to place a small piece of food between the tongue and palate and keep it there while attempting to swallow several times. This may be repeated up to the point where swallowing with the tip of the tongue resting against the palate becomes an automatic reflex.

In young children with minor ankyloglossia, have them perform exercises to mobilize the tongue as much as possible in order to stretch the frenulum. Stress that mispronunciation often associated with baby talk may appear to be cute, but that it is actually dysfunctional and should not be encouraged. Insist, when speaking, that the child repeats incorrectly articulated sounds as correctly as possible to improve tongue mobility. Have them play sound-making games where they repeat certain sounds that cause the tongue to make a clicking noise against the palate.

Be aware that functional asymmetries are often associated with repetitive asymmetric activities, such as unilateral bottle feeding or thumb sucking. Educate the caregiver and patient to lose these habits. The use of a pacifier should be limited as much as possible. Suggest that pacifiers should be used only during the first 10 months of life, when the need for sucking is strongest.[32]

REGURGITATION AND GASTROESOPHAGEAL REFLUX

Gastroesophageal reflux (GER) is the retrograde flow of gastric contents into the esophagus and above. It is rather frequent during infancy and is often considered as 'physiologic' under 2 months of age with a benign prognosis in the majority of cases. However, it is a problem of considerable concern for the parents that ranges from minimal regurgitation with the bringing up of small amounts after feeds, to significant vomiting with failure to thrive. Because the latter may be indicative of a serious condition, it requires further medical evaluation.

GER is one of the most recurrent symptomatic clinical disorders affecting the gastrointestinal tract of infants and children, and regurgitation in infancy is a complaint in 5.8% of children seen in osteopathic practice.[33] Daily regurgitation is present in 50% of infants in the first 3 months of life, with a peak incidence at 4 months.[34] Most of the time, the condition resolves before the end of the 1st year and only 5% of infants still bring up their food between 13 and 14 months of age; however, when the condition is present for at least 90 days before 2 years of age, children are predisposed to demonstrate GER symptoms into middle childhood.[35] There is no association with gender,[35,36] nor with the mode of feeding the infant, breast or bottle; nevertheless, a genetic component seems to be present and a correlation exists between maternal symptoms of GER and the occurrence of regurgitation in infancy and GER in middle childhood.[35] Additionally, there is a stronger prevalence of GER among Caucasian infants.[36,37]

Normal gastroesophageal function is a complex mechanism where the relaxation and contractility of the lower esophageal sphincter are important factors. The esophagus is a tube connecting the pharynx and stomach, with muscular sphincters – the upper and lower esophageal sphincters. The upper portion of the esophagus (the cervical esophagus) consists of skeletal muscle; the remainder (the thoracic esophagus) consists of smooth muscle. After swallowing, the bolus of food passes through the pharynx to enter the esophagus. A primary wave of contraction starts in the pharynx and travels along the whole length of the esophagus, reinforced by secondary waves in the body of the esophagus. These peristaltic waves assist gravity to propel the bolus through the esophagus and into the stomach. Normally, at the gastroesophageal junction (GEJ), the lower esophageal sphincter (LES) relaxes to allow the bolus of liquid or food to pass into the stomach.

In GER, the GEJ is a critical site. The LES is formed by circular smooth muscle surrounding the terminal esophagus. The LES is under tonic contraction that relaxes during swallowing as well as following esophageal distension by a bolus of liquid or food. Additionally, the crura of the diaphragm participate in this sphincteric mechanism. The esophagus traverses the diaphragm through an opening in the right diaphragmatic crus, the esophageal hiatus, which is located between the central tendon and the hiatus aorticus. The two vagus nerves (CN X) also traverse the diaphragm through the esophageal hiatus. At this hiatus, one crus, most often the right crus, surrounds the esophagus forming an external sphincter; however, the fibers from the crus are not directly connected to the esophageal wall. Rather, fibers coming from the transversalis fascia, and thus continuing to form a fascia under the diaphragm, pass into the esophageal hiatus and surround the esophagus to blend into its walls 2–3 cm above the GEJ. Some of the elastic fibers of the fascia extend into the esophageal submucosa.[1] This connection between the diaphragm and esophagus is called the phrenoesophageal ligament. This ligament simultaneously allows GEJ mobility, permitting the displacement associated with respiration and swallowing, while providing restriction of upward movement of the esophagus. Additionally, during pulmonary inspiration, the diaphragmatic esophageal hiatus contracts in response to increased intra-abdominal pressure.[1]

The fascial relationships between the esophagus and the diaphragm must be balanced when addressing dysfunction of the GEJ. The transversalis fascia is a vast layer of fascia lying between the peritoneum and the abdominal walls, in continuation below with the pelvic fasciae, behind with the thoracolumbar fascia and above with the fascial sheet covering the undersurface of the diaphragm. The phrenoesophageal ligament, described above, consists of fibers extending from this layer to form the cone surrounding the GEJ. Furthermore, the stomach is connected to the diaphragm through the gastrophrenic ligament, i.e. the portion of the greater omentum that extends from the greater curvature of the stomach to the inferior surface of the diaphragm. From a functional point of view, the diaphragm must be balanced to allow satisfactory GEJ function.

The smooth muscle of the LES generates a tonic pressure at the GEJ that is the major barrier to reflux of gastric contents into the esophagus. Thus, reflux of acidic fluid backward, out of the stomach and into the esophagus, is normally controlled at the GEJ. At birth, neuromuscular activity and esophageal and LES peristalsis are undeveloped, resulting in frequent retrograde spilling out of gastric contents. The achievement of full development of the LES occurs as the infant matures, with the pressure at the LES reaching adult levels between 3 and 6 months of age. At the same time, the average capacity of the stomach at birth, i.e. around 30 ml, will rise to 100 ml at the end of the first month and will reach 1000 ml in the adolescent.

In order to act as a protective barrier against reflux from the stomach, pressure in the GEJ has to be higher than in the adjacent stomach or esophagus. Inhibitory and excitatory factors influence the tone of the LES. As mentioned above, a bolus of liquid or food initiates the relaxation of the sphincter in response to esophageal distension. This mechanism is under the control of intramural plexuses of the enteric nervous system. The motor fibers start in the dorsal motor nucleus of the vagus and the nucleus ambiguus, and the neural release of nitric oxide and vasoactive intestinal peptide among others by postganglionic fibers triggers the relaxation of the LES. Additionally, neurological activity from the dorsal motor nucleus of the vagus and nucleus ambiguus is also sent to the crura of the diaphragm.[14] Thus, both the esophageal and crural musculature are inhibited.

Embryologically, both the esophagus and stomach develop from the foregut. The dilatation of the foregut for the stomach starts in the sagittal plane at 4 weeks of gestation. Several spatial changes occur before the stomach reaches its final position. Because growth is greater along the dorsal border of the stomach, the greater and lesser curvatures are formed. This is followed by rotation, with the greater curvature moving to the left and the lesser curvature moving to the right. The two vagus nerves follow this rotation. Thus, the right vagus becomes posterior and supplies the dorsal part of the stomach, while the left vagus is anterior and supplies the ventral part of the stomach. Some torsion of the stomach occurs between the lower part of the esophagus and the pyloric canal, which are consequently no longer in the same plane. This has been suggested as a risk factor for GER.[38] This developmental process results in what is felt when the stomach is palpated. The identification of a strong torsional sensation in this region of the gut is an indication for osteopathic treatment that should be directed at the alleviation of the identified torsional dysfunction.

The etiology of GER is multifactorial, with often more than one factor being present in children with this condition. However, a transient LES relaxation (TLESR) as the result of a vasovagal reflex appears to be frequently associated with GER, producing a

complete relaxation of the LES without pharyngeal swallowing.[39,40] Other factors for GER include ineffective esophageal motility or delayed gastric emptying.[41] A high fat diet can contribute to an increase in the number of episodes of TLESR.[42] Additionally, in infants, gravitational and positional factors associated with the supine position may exacerbate the reflux. Most infants spend more time in this recommended position since the 'Back to Sleep' campaign to prevent sudden infant death syndrome (SIDS). The return of the gastric acid contents into the esophagus predisposes the infant and the child to esophagitis; for these patients, crying becomes common.[43]

Acid reflux into the esophagus can reach the upper esophagus, the pharynx and larynx, and may possibly be aspirated into the trachea. Chemoreceptors in the mid or upper esophageal mucosa are stimulated, resulting in reflex respiratory inhibition, hypertension and bradycardia. Approximately 1% of infants demonstrate reflux-induced apnea with airway closure or laryngospasm.[44] The severity of these reactions may lead to SIDS.[43] Bronchospasm may also occur and, in turn, accentuate the symptoms of asthma, although GER does not seem to increase the risk of developing asthma.[45] Recurrent bronchitis or pneumonia is frequent and can be caused by repeated pulmonary aspiration of acid reflux. ENT problems such as nasal obstruction are sometimes related to GER, with secondary inflammation and narrowing of the posterior nasal apertures.[46,47] With premature infants, the most common complication of recurrent GER is failure to thrive.

In children and adolescents, GER is frequently associated with abdominal pain in the midepigastrium, eventually substernal heartburn and possible chronic irritative coughing. Furthermore, regurgitation of gastric contents into the oral cavity is responsible for dental erosion and dental caries.[48]

The vagus nerve (CN X) is essential in the control of LES tone. It contains both motor and sensory fibers, with a course and distribution more widespread than any of the other cranial nerves, passing through the neck and thorax to the abdomen. About 80% of vagal fibers are afferent fibers and may be general somatic, general visceral or special afferent. They provide significant sensory input from the viscera. In infants, children and adolescents with GER, there is a definitive dysfunctional viscerosomatic and/or somatovisceral component. This may, in turn, lead to altered behavioral responses. Possible dysfunction affecting CN X in its extensive course may facilitate GER. Critical sites are the jugular foramina and the passages through the diaphragm at the esophageal hiatus. Additionally, the stomach and esophagus may demonstrate visceral dysfunction in relation to somatic dysfunction in the surrounding structures, i.e. diaphragm, fasciae and ligaments.

The osteopathic approach to GER consists, therefore, of addressing any dysfunction that can impede the vagus, balancing the stomach and esophagus together and in their relationships with surrounding structures – the diaphragm, fasciae and ligaments.

Physical examination and treatment

Inspect the abdomen, noting its shape, contour and movement with respiration. The abdomen should be rounded and dome-shaped in infants because of the lack of abdominal muscular tone. Observe for abdominal asymmetry, noting areas of tension under the inferior border of the thoracic cage, in particular under the xiphoid process. Note if the umbilicus is centered or displaced or if it is protruded. In children and adolescents, observe the inside of the mouth for dental caries.

Examine and treat the craniocervical junction, with particular attention to the relationship between the occiput and temporal bones because compression of the jugular foramen can impact the exit of CN X. Check and balance the cervical area (C3, C4, C5) because of the phrenic nerve and its action on the diaphragm. Examine and treat identified somatic dysfunction affecting the thoracolumbar junction because of its potential to affect the crura of the diaphragm.

Evaluate and treat the diaphragm, paying particular attention to the freedom and symmetry of its excursion and its attachment around the xiphoid process as well as its anterior costal attachments. Balance the fasciae at the level of the LES by balancing the area below the xiphoid process with the diaphragm.

Balance the relationship between the stomach and diaphragm. The stomach itself demonstrates curling and uncurling movements, similar to those that created its curvatures. Palpation should reveal a balance between these curling and uncurling movements. A predominance of one phase can be treated using indirect procedures. Balance the torsional relationship between the stomach and the esophagus. Address somatic dysfunction related to both viscerosomatic and somatovisceral reflexes: occiput, C1, C2 for the vagus; upper thoracic for the esophagus; midthoracic for the stomach.[49]

Advice to caregivers

Infants should have small and frequent feeds, and should be fed in a semi-upright position. Feeding

should occur under calm circumstances. Instruct children and adolescents to eat slowly and to avoid swallowing air. They should eat and drink small amounts to prevent distension of the stomach. They should also be instructed to avoid the consumption of fatty foods, caffeinated beverages and chocolate. Lifestyle changes such as avoiding eating before bedtime and, if appropriate, weight loss should be encouraged.

COLIC

The term colic is derived from the Greek, *kblikos*, meaning in relation to the colon. In the list of the most frequent gastrointestinal (GI) dysfunctions encountered in infants, colic is second only to regurgitation. It occurs in 10–40% of infants[50–52] and is one of the most common complaints for which parents seek professional advice.

The colicky infant presents with abdominal distension, frequent gas emissions, apparent abdominal pain, irritability and excessive crying. The mechanisms that explain this disorder are not entirely understood. They may be discussed in anatomic or physiologic terms, reflecting dysfunction at one or more levels of the brain–gut axis. Additionally, because the ontogeny of the GI system is under multiple influences – genetic heritage, intrinsic development, endogenous regulatory mechanisms and the environment[53] – the etiology of colic is commonly multifactorial. Thus, various etiologies have been proposed, including GI diseases such as allergy to cow's milk protein, lactose intolerance and intestinal hyperperistalsis, as well as neurodevelopmental dysmaturation or psychological difficulties in the parent–child relationship.

On average, it is estimated that infants cry for 2.2 hours per day during the first 2 months of life, with a peak around 6 weeks of age.[54] Crying time then slowly decreases to less than 1 hour a day by 12 weeks of age. The 'rule of three' proposed by Wessel et al. in 1954 is usually the reference employed to identify colicky children. These children are said to cry for more than 3 hours per day, more than 3 days per week and for longer than 3 weeks.[55] Colic usually starts in the 1st month of life and may persist until 3 or 4 months of age. Crying is commonly concentrated in the late afternoon and evening, occurs in prolonged bouts and is unpredictable and spontaneous. Colicky children are described as crying without identifiable cause, fussing and hard-to-soothe, although being otherwise healthy infants, well fed and showing no signs of failure to thrive. Individual variation may be present in the cries, such as duration or intensity, associated fussing or the resulting parental distress, adding complexity to the definition.[56] Colic cries convey acoustic information that the infant is highly aroused or distressed.[57] During a crying episode, the infant usually positions their legs flexed over their abdomen, in association with a hard distended abdomen, gurgling noises from the abdomen (borborygmi), sometimes gas and regurgitation, as well as facial grimacing and clenched fists. This presentation is indicative of a GI etiology for colic. When evaluating the colicky infant, several aspects of GI development and associated neuro–hormonal factors should be considered.

Bacterial colonization of the sterile GI tract of the neonate starts quickly on delivery. A flora develops, resulting from microbial and host interactions. The main factors that influence this developing microbial population are the maternal intestinal flora, the use of antibiotics by the mother during pregnancy and when nursing, the mode of delivery (vaginal or cesarean) and the mode of feeding (bottle feeding or breastfeeding). The bacterial flora in breastfed infants is composed typically of bifidobacteria, whereas in bottle-fed infants anaerobic bacteria as well as aerobic species are present.[58] It is also recognized that the flora of breastfed infants includes far fewer species that are liable to be pathogenic.[59] The developing bacterial flora usually becomes stable by the end of the first week of life. By 2 years of age the infantile flora resembles that of the normal adult. This process is one of the main components, along with host cells and nutrients, that form the complex ecosystem of the intestine.

The intestinal lumina are lined by a very large surface of mucosa that is the principal interface with the external environment. Besides its function of digestion and absorption of the nutrients contained in food, the intestinal mucosa forms a protective barrier against foreign substances and potentially pathogenic microorganisms from the external environment. The mucosal barrier consists of cellular and stromal components covered by a mucus layer. Within the mucosal barrier various secretions form a viscoelastic gel, creating a site for bacteria–bacteria interactions. Thus, the microbiota play an important role in the development of the endogenous barrier mechanism in the gut and are involved in the maturation of humoral immunity in humans.[60]

The intestine can be considered the primary immune organ, housing more than 70% of all immune cells.[25] The gut-associated lymphoid tissue (GALT) develops through innate and acquired immunity and consists of organized and diffuse

lymphoid tissues that constitute the aggregated lymphoid follicles (Peyer's patches) and the mesenteric lymph nodes. This arrangement permits rapid response to any challenge in order to maintain the integrity and protective functions of the immune barrier at the GI mucosal surface. The GALT contains B lymphocytes, implicated in the synthesis of secretory antibodies of the immunoglobulin A (IgA) class, and T lymphocytes. In this ecosystem, the equilibrium of the microbiota is important in the maintenance of homeostasis and intestinal immune responses. It is also of paramount importance in the prevention of food allergies, and it is during the first 2 years of life, when the infantile flora is not well diversified, that food allergies and enteropathies are more apt to be established.

Particular circumstances in infants, such as prematurity, cesarean section delivery and antimicrobial treatment, delay intestinal colonization and its important immunostimulating effect.[60,61] On the other hand, breastfeeding is considered to confer beneficial effects on the microbiota, explaining its protective role against atopic disease, although it does not appear to have a protective effect on the development of colic.[62] Differences in the gut microbiota of breastfed colicky infants have been shown to exist when compared to non-colicky, and Lactobacilli are present less than in controls.[63]

That breastfed infants may suffer from colic might be explained by the fact that breast milk contains intact proteins similar to those of cow's milk and that colic often appears to be related to a food allergy, particularly to cow's milk protein.[64,65] This exposure would trigger the production of specific IgE to milk protein and may be considered a manifestation of an allergic predisposition. This is, however, subject to debate because no data support the hypothesis that infantile colic leads to an increased risk for subsequent allergic disease or atopy,[66] although more irritability during the 7th week and colic-type crying during the 12th week has been observed in children with atopic disease at 2 years of age.[67] Furthermore, lactose intolerance also contributes to infantile colic.[68] Incomplete lactose absorption encourages bacterial growth with resultant flatulence and cramping. As the etiology of infantile colic is multifactorial, cow's milk protein elimination may be an effective treatment for infantile colic,[64] and for colicky breastfed infants a maternal diet free of cow's milk may be considered.[69] Other maternal dietary modifications during breastfeeding should include reduction of cruciferous vegetables, onion and chocolate, foods that are associated with colic symptoms in infants.[70]

Hyperperistalsis is another theory included in the GI disorder etiology to explain abdominal cramping and colic. Intestinal smooth muscle normally generates rhythmic peristaltic activity. Movements of the intestine with the regulation of gut motility are the result of complex neural and muscular interactions that take place at several levels and under the influence of neurotransmitters and hormones.[71] The innervation of the gut consists of intrinsic and extrinsic nerves. Ganglionated and non-ganglionated plexi, part of the enteric nervous system (ENS), form the intrinsic innervation, while the vagus, splanchnic and pelvic nerves form the extrinsic innervation. The intrinsic afferent neurons project within the myenteric and submucous plexuses and information from activated sensory receptors reaches the CNS through vagal and spinal afferent nerves. The gut interacts constantly with the CNS through either somatic or autonomic neurons (brain–gut axis). Consequently, the CNS modulates the gut motor activity through the ANS (sympathetic and parasympathetic), maintaining the normal rhythm of activity in the GI tract as well as adjusting autonomic output to accommodate any external challenge. The release of acetylcholine by the parasympathetic fibers increases gut motility, whereas the release of norepinephrine, somatostatin and neuropeptide Y by sympathetic fibers decreases the motility. Multiple factors, such as emotion, stress, nervousness, fasting or eating, can also influence the activity of the ANS. Additionally, communication occurs between the different parts of the GI tract through myogenic and neurogenic signals along the gut, and through reflex arcs transmitted via autonomic neurons.

Individual differences are present in the functioning of the CNS. Accordingly, an increased sensitivity to stimuli, such as distension of the gut, is demonstrable in patients with functional GI disorders,[72] and excessive crying in colicky infants may be the result of hypersensitivity in the perception of gut stimuli or excessive afferent responses to these stimuli.[73] Genetic susceptibility to functional GI disorders may exist. Supporting this theory, a correlation is found between high responsiveness of the newborn during neurobehavioral assessment in the first days of life and crying behavior at home.[74] Additionally, intestinal hypersensitivity may alter motility of the GI tract by increasing intestinal afferent–efferent reflexes.[75]

Cranial osteopathy offers a therapeutic option for these patients. Osteopathic treatment of colicky infants has been demonstrated to decrease crying and increase sleep time.[76] Somatic dysfunction affects the

ANS. The resulting state of facilitation, in turn, increases the perception of either mechanical or chemical intestinal stimuli, and visceral hypersensitivity or dysfunction follows. Somatic dysfunction can involve any osseous, articular, ligamentous, membranous, fascial, muscular, visceral and vascular component associated with the GI tract. Vagal viscerosensory neurons have their cell bodies in the inferior ganglion located under the jugular foramen. The spinal sensory input takes place through perivascular nerves passing through the prevertebral ganglia to the dorsal horn of the spinal cord, and these neurons have their cell bodies in the dorsal root ganglia. Pain perception is thought to be mediated essentially by the spinal innervation.[77] The craniocervical junction (parasympathetic), the thoracic and thoracolumbar spine (sympathetic) and sacropelvic region (parasympathetic) may be sites of somatic dysfunction resulting in somatovisceral dysfunction. Treating these areas may contribute to balance the gut function of the colicky infant.

Changes in internal sensory states, acting in a bidirectional manner between the viscera and the brain, are obviously related to interoception and visceral hypersensitivity, and are considered as the key pathogenetic factors underlying the emotional state present in subjects with functional GI disorders.[78] Stress, in a broad sense, as with any menace to one's individual homeostasis, may come from the external or internal environment.[78] As such, visceral hypersensitivity, milk allergy or somatic dysfunction may be stressful, and colicky infants are quite often described as demonstrating difficult temperament.

An infant who is crying is expressing distress without indication of its origin. This can be a significant source of stress for parents and caregivers. They respond differently to these cries according to cultural and sociodemographic factors that, in turn, may affect the amount of infant crying. Responses of mothers to infant crying diverge from putting the child to bed, to holding and carrying them, riding around in the car, rocking or swaddling.[79] Globally, first-born babies are usually reported as crying more excessively, and the Western caregiving style is associated with a higher incidence of reported crying.[79,80]

Touching, holding and caressing a child results in positive effects on the emotion regulation and stress reactivity in the infant.[81] Maternal care facilitates the development of central corticotropin-releasing factor (CRF) systems which regulate the expression of behavioral, endocrine and autonomic responses to stress.[82] It is well established that infant–maternal contact is of paramount importance in mediating infant emotional reactivity. Gut–brain peptide cholecystokinin (CCK) and endogenous opioid analgesic agent production are increased following contact.[81] This contributes to the development of attachment through the 'interactive regulation of biological synchronicity between organisms'.[83] Under normal conditions, this relationship between mother and infant contributes to the wellbeing of both.

Conversely, infantile irritability can prove to be a major source of distress to caregivers. In extreme circumstances the irritability of the child can prove so stressful as to create an impulse to shake the child.[73] A potential psychosocial etiology for colic in the 1st year of life is associated with pre-existing maternal anxiety.[62] If the caregiver is stressed, the child will cry more, and the caregiver will be further worried by their incapacity to help the child. Furthermore, a baby is frequently described as crying excessively when the crying distresses the parents. Infants' cries can also be indicative of a behavioral problem resulting from a less than optimal parent–infant interaction. This complex interrelationship between the child and caregiver has led to a behavioral hypothesis for the etiology of colic where interventions such as modifying parental responsiveness, using motion and sound to calm the baby, and reducing stimuli have been suggested.[64] These caregivers do not need to be told that they are bad parents; they need to be supported and managed with understanding. The circumstances must be discussed and the caregiver given the opportunity to vent their anxiety and frustration. Behavioral adaptations, when appropriate, should be encouraged. Maternal smoking may contribute to the disorder.[84]

One hypothesis for colic suggests that it is the result of transient developmental dysmaturation based on the fact that infantile colic often stops after 5 or 6 months.[85] It is important, nonetheless, to treat these children and help the parents, because persistent behavioral difficulties, including crying, sleeping or feeding behavior in infancy, are precursors of hyperactivity or behavioral problems and academic difficulties in childhood.[86]

Physical examination and treatment

It is important to perform a thorough physical examination to rule out other causes for persistent crying to ensure that there is no organic cause for the crying. Once this has been done, osteopathic manipulation may be employed to reduce the somatic afferent load of somatovisceral reflexes into the CNS as well as to alleviate the mechanical impact that somatic dysfunction can have on the GI tract.

Treatment is appropriate not only for infants, but also for children and adolescents, because colic may persist later in life as a functional GI disorder.[87]

The osteopathic component of the examination should begin with observation of the infant's posture. The infant may position themselves with their legs flexed to their chest. If the patient is a child, they might present with an apparent increased lumbar lordosis that occurs when bowel dysfunction creates a distended abdomen with consequent relaxation of the abdominal muscles. Observe the abdomen to see if it is tense with bowel distension from flatulence. Note the power and rhythm of the inherent motility of the PRM in the head and throughout the body, particularly noting the abdomen. Somatic dysfunction should be sought out, especially in the regions of, but not limited to, the posterior neurocranium; the occipitocervical junction and upper cervical spine; the thoracic spine, ribs and upper lumbar spine; the thoracoabdominal diaphragm, the anterior abdominal wall and the sacrum and pelvis.

Treatment should employ indirect principles and manipulation should be directed at somatic dysfunction, when identified, involving the temporo-occipital relationship for its impact on the jugular foramen, and the occipitocervical junction and upper cervical spine for their effect on the vagus and parasympathetic somatovisceral reflexes. Somatic dysfunction of the thoracic spine, ribs and upper lumbar spine may be treated to affect sympathetic somatovisceral reflexes. In acute cases, practice inhibition in the lumbothoracic area. Avoid active massage of the abdomen which can be irritating. Dysfunction of the thoracic spine, ribs and thoracoabdominal diaphragm should be addressed for its impact on the lymphatic and venous drainage of the contents of the abdomen. Diaphragmatic, abdominal wall and pelvic dysfunction should be treated to alleviate the impact of dysfunctional fascial tensions on the GI tract. Dysfunction of the thoracoabdominal diaphragm is related to the function of the mesenteric plexus. Sacropelvic somatic dysfunction may be treated to affect pelvic splanchnic parasympathetic somatovisceral reflexes. Using indirect principles, release the periumbilical area and intestine. In every treatment procedure pay attention to the inherent motility of the PRM as manifest throughout the body. Treating this mechanism affects the ANS and probably facilitates tissue perfusion, reducing congestion.[88]

Patient education and advice to caregivers

The caregiver should be encouraged to create an environment of comfort, calm and relaxation for the infant. Gently caressing the frontal area of the neurocranium will often facilitate this. Obtaining relaxation of the infant will also have a calming effect on the caregiver(s) that will, in turn, further relax the infant. This relaxation will often make it easier to obtain such a state of calm in the future and will also demonstrate to the caregiver(s) that it can be obtained. Avoid, however, active massage of the abdomen which can be irritating.

Dietary considerations may be employed to improve the function of the GI and the immune system. Breastfeeding should, if at all possible, be encouraged. The mother should pay attention to her diet, avoiding cow's milk, cruciferous vegetables, onion and chocolate.

In colicky children, lactose intolerance should be considered. A diet rich in fresh fruit and vegetables and antioxidants such as vitamins C and E should be recommended, while refined foods should be avoided as much as possible.

CONSTIPATION

Constipation is the chief complaint in 3% of all pediatric outpatient visits and defecation disorder is present in 10–25% of children referred to pediatric gastroenterologists.[89] Constipation in children is usually defined as abdominal pain, difficulty or pain when passing stool, with the passage of feces that are either large and too hard or in small pebble-like pieces, with difficulty defecating and a frequency of two times or less per week.

The first intestinal discharge, meconium, is passed in healthy newborns within 24 hours.[90] After that, the mode of feeding determines the frequency and quality of stools. Bottle-fed infants demonstrate less frequent stools than breastfed infants, who have soft yellow stools up to five times a day. However, breastfed infants may go for 3 or more days without defecation. Weaning – the commencement of nourishment with food other than milk – usually occurs between 4 and 6 months of age and results in firmer feces. Although the frequency of bowel evacuation varies from one infant to another, it is generally admitted that a frequency of less than one stool a day before 6 months of age, and three times or less per week after 6 months, may be considered as pathologic. In preschool children, constipation is present when the child has less than two stools per week.

Most of the time, constipation is functional, without any objective evidence of an underlying pathologic condition and a thorough history and physical examination are sufficient to make the

diagnosis. It is, however, important to rule out failure to thrive or endocrine, metabolic or structural disease such as Hirschsprung's disease. Children presenting with this latter condition, characterized by the total absence of ganglion cells in Meissner's and Auerbach's plexuses, suffer from constipation with massive colonic dilatation proximal to the segment of affected bowel. The onset, however, occurs at birth with delayed passage of meconium and children suffering from the disease demonstrate poor growth.[91]

Functional constipation has also been described as functional fecal retention, voluntary withholding, psychogenic megacolon or idiopathic constipation. It affects boys more often than girls, contrary to the adolescent and adult populations where women suffer more often from constipation. Another difference between children and the adult population is the presence of fecal incontinence in children, which is not present in adults with functional constipation.[91]

During the first months of life, infants may present with dyschezia, i.e. difficulty in defecation. In this case they experience severe problems when trying to defecate and strain and scream during prolonged endeavors. This behavior may last up to 20 minutes, until they successfully pass stools that are usually soft or liquid. The cause is hypothesized to be the failure to coordinate the augmentation of intra-abdominal pressure with the relaxation of the pelvic muscles.[89] Painful defecation quite often results in chronic fecal retention with fecal impaction (the immovable collection of compressed or hardened feces in the colon or rectum) and resultant fecal soiling (the passage of liquid stool around the impaction). More than 50% of school-aged children suffering constipation have a history of painful defecation before 36 months of age.[92] Toddlers may succeed in avoiding defecation for several days. The evacuation that then occurs is often painful and may be associated with bleeding, thereby strengthening the behavior of fear and retention. Fecal retention, in turn, is associated with subsequent abnormal contraction of the anal sphincters (anismus) and contraction of the pelvic floor during attempted retention. The child develops a retentive posture with contraction of the gluteal muscles. With overstretching of the rectal wall and muscle fatigue in the pelvic floor, incompetent anal function results in spontaneous relaxation of the sphincters, with consequent fecal soiling with soft or liquid stool. Fecal soiling often follows constipation facilitated by rectal distension.[93] Constipated children demonstrate several associated symptoms including irritability, abdominal cramps and decreased appetite. Most of the time, children with constipation have a withholding type of behavior and very often feel ashamed. They demonstrate more behavioral problems than children who are not constipated; however, these accompanying symptoms disappear immediately following the effective treatment of constipation.[94]

The role of psychological and emotional components in the etiology of defecation disorders is subject to debate. It is uncertain which problem comes first: the emotional disorder or the defecation disorder.[94]

Toilet training is normally initiated between the ages of 18 months and 3 years. When conducted in an overly coercive and stressful fashion, the associated stress has been proposed as a cause of fecal retention. However, when hard bowel movements or painful defecation are present in association with stool toilet training refusal, constipation should be considered because the first episode of constipation in children usually occurs before stool toilet training refusal.[95] Familial environmental factors, or added parental anxiety because they want their child to be able to go to school, may add a psychological factor to a physical predisposition to constipation.

A decrease in colonic peristalsis has been proposed as a cause of constipation in childhood. It is well recognized that reduction of physical activity and reduction of fluid intake in adults can be associated with constipation. Because children tend to be normally physically active, inactivity is not a major cause of constipation for this population. However, food allergy, particularly cow's milk allergy with cow's milk protein hypersensitivity, seems to be associated with constipation in children.[96,97]

Constipation may certainly be multifactorial. The behavioral approach does not explain every case of constipation. Furthermore, children with constipation demonstrate a higher incidence of bladder disorders such as urinary incontinence, bladder overactivity, dyscoordinated voiding, large bladder capacity, poorly emptying bladder, recurrent urinary tract infection and vesicoureteral reflux.[98] This constitutes a syndrome, an aggregate of associated symptoms and signs that may be addressed by an osteopathic approach. Most cases of functional constipation respond well to osteopathic manipulative procedures.

Defecation is a complex process that involves a mixture of voluntary and involuntary actions. It is triggered by the excitation of anorectal mechanoreceptors sensitive to distension of the rectum. It is followed by coordinated voluntary activity of the abdominal and pelvic musculature and involuntary relaxation of anal sphincters. As the fecal mass

moves, the pelvic floor muscles relax in order to allow alignment of the rectum with the anal canal. Defecation can be inhibited by voluntary contraction of the external anal sphincter and pelvic floor muscles.

Normally, the anal canal is occluded by the internal and external anal sphincters. Additionally, the puborectalis muscle, the medial part of the levator ani muscle, contributes to the loop surrounding the anorectal junction by mixing some of its fibers with the deep part of the external sphincter of the anal sphincters. Additional fibers of the levator ani muscle join the conjoint longitudinal coat that surrounds the anal canal between the internal and external canal. Behind the rectum, the pubococcygeal fibers of the levator ani muscles attach to the anterior surface of the coccyx.

The internal anal sphincter has an autonomic innervation: the sympathetic fibers are from the hypogastric plexus and the plexuses located around the superior rectal artery; the parasympathetic fibers are from the pelvic splanchnic nerves (S2–S4). The external sphincter has a voluntary motor supply that comes from the inferior rectal branch of the pudendal nerve (S2–S3) and the perineal branch of the fourth sacral nerve.

Physical examination and treatment

If the child is old enough to be standing and walking, observe their postural mechanics, noting particularly the degree of abdominal protrusion, thoracolumbar mechanics as they relate to psoas muscle mechanics, the degree of lumbar lordosis as it relates to abdominal protrusion and psoas muscle mechanics.

With the patient supine, observe the abdomen, noting its contour and possible distension. Palpate the abdomen, looking for tension in the abdominal wall and for palpable stool. In many cases of chronic constipation, stool will be palpable throughout the colon. Define stool consistency and assess the quantity of the rectal fecal mass by looking at the height of transabdominally palpable stool above the pelvic brim. Diagnostic digital rectal examination should be performed gently to avoid perpetuation of dyschezia. Maneuvers that result in rectal stimulation produce potentially noxious sensory experiences and should be discouraged.[89]

Specifically look for somatic dysfunction affecting the thoracolumbar junction that can be associated with psoas muscle dysfunction and sympathetic somatovisceral reflexes. Examine the sacrum, coccyx and pelvis for dysfunction that can affect the pelvic floor and also be the source of parasympathetic somatovisceral reflexes.

The general medical treatment approach includes dietary changes with behavioral modification techniques (cognitive and behavioral interventions such as toilet training, which diminishes phobia and provides positive reinforcement through a rewards system). This approach is often combined with prolonged courses of laxatives. Treatment is usually successful, but may take up to 6–12 months.[93] When somatic dysfunction has been identified, the correct osteopathic treatment can result in significantly faster results with a resolution of constipation, often in one or two treatments.

Therefore, treat any somatic dysfunction as identified. Treat somatic dysfunction of the sacrum for its relation with pelvic splanchnic nerves and the pudendal nerve. Release sacroiliac joints and surrounding myofascial structures. It is important to remember that the sacrum is not completely ossified in infants and children and that intraosseous dysfunction may be present between the different sacral segments. In the infant the sacral molding procedure often provides good results. Treat somatic dysfunction of the coccyx, paying attention to its relationship to the insertion of the levator ani muscles. Treat somatic dysfunction of the thoracolumbar area for its somatovisceral effect on the sympathetic output to the intestine. With hypersympathetic drive, the bowel becomes less active and may result in constipation. It is also important to be sure that child's posture is balanced, without dysfunctional tension at the level of the psoas for its relationship with the sympathetic chain near the psoas muscle origin.

Patient education and advice to caregivers

It is important to increase the amount of fluid that the child drinks every day. Infants may be given fruit juices such as prune and pear which contain fructose and sorbitol for their mild laxative effect. If the child is old enough to be eating solid foods, increase bran cereal and fruits and vegetables that are high in fiber. Encourage the consumption of meals at regular times. If the child is relatively sedentary, as tends to occur these days with the playing of computer games, encourage increased physical activity.

Tell the parents to watch for infrequent, difficult or painful defecation that heralds the recurrence of constipation. When identified, they should respond quickly, encouraging the child to go to the bathroom more frequently and modifying the child's diet and physical activities.

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7.7 OROFACIAL DYSFUNCTIONS

ORAL CAVITY AND OROFACIAL FUNCTIONS

In a holistic approach to health care, any one area of the body is linked directly or indirectly to all the other areas of the body. Consequently, observation of the oral cavity not only provides information about the mouth, it also provides knowledge as to many other aspects of the individual. The oral cavity provides access to the posterior nasal cavity, pharynx, esophagus and lower respiratory tract. It contains an ecosystem that reflects the overall health status of the individual. Dental occlusion reflects the functional balance of the musculoskeletal system, both locally and at distant areas of the body. Muscular tone, facial expression and orofacial habits mirror the individual's psychoemotional status. The oral cavity is also an area that allows communication with others through facial expression and speech. It is the major point of entry for nutrition and hydration and a secondary portal for respiration. Optimal function of the area is essential for the very survival of the individual.

Oral cavity

The main osseous structures of the oral cavity are the mandible, maxillae and palatine bones. The latter two bones are part of the viscerocranium and as such their growth and development extend through adolescence. This process is under genetic influence as well as epigenetic factors in which orofacial functions play a significant part. The long period through which development occurs allows extensive opportunities for dysfunction to be established. Thus, a thorough understanding of the structure and function involved in this evolution, combined with attentive observation of the infant and growing child, is imperative to identify somatic dysfunction, the effective treatment of which is necessary to promote health and balance.

The oral cavity, the mouth, is surrounded by the lips and cheeks and consists of two parts: the oral cavity proper and the vestibule. The oral cavity proper is limited anteriorly and laterally by the alveolar arches, teeth and gums, and above by the hard and soft palate; posteriorly, it communicates with the pharynx through the oropharyngeal isthmus. It includes the tongue, which is totally contained in the oral cavity in infants. As the child grows, only the anterior two-thirds remain in the oral cavity while the posterior third descends following the larynx.[1] The vestibule is located between the lips and cheeks externally, and the gums and teeth internally. It connects with the outside through the oral fissure between the lips.

The entire oral cavity is covered by mucosa, starting at the labial margins and consisting of three portions: the lining and the masticatory and specialized mucosae.[1] These portions demonstrate different properties according to their location. The oral mucosa is in continuity with the pharyngeal mucosa at the oropharyngeal isthmus.

Several bones within the viscerocranium define the oral cavity. The maxillae are the first to come to mind; together with the mandible, they are among the largest of the facial bones. Nevertheless, all of the components that constitute the skeletal

framework of the oral cavity must be taken into consideration. Myofascial structures attached to this framework affect it and are, in turn, affected by it. Besides the mandible and maxillae, these bony boundaries include the paired palatine and temporal bones, and unpaired sphenoid and hyoid bones.

The maxilla consists of a body with zygomatic, frontal, alveolar and palatine processes. Bilaterally their bodies contain the maxillary sinuses, the largest of the paranasal sinuses. Although these cavities are large in the adolescent, at birth they are small furrows 7 mm in length and 4 mm in width.[2] Consequently, in the infant, the vertical diameter of the maxilla is smaller than the transverse and anteroposterior diameters.

The maxillary body is shaped as a pyramid, having four surfaces: anterior, posterior (infratemporal), superior (orbital) and medial (nasal). The lower part of the anterior surface demonstrates multiple eminences above the roots of the teeth. Several facial muscles insert on the anterior surface of the maxilla and their traction during orofacial activities such as sucking and chewing contributes to the development of the maxilla. The depressor septi nasi muscle arises above the eminences of the incisor teeth. A slip of the orbicularis oris muscle is attached on the alveolar border below this incisive fossa, and the nasalis muscle is attached superiorly. The levator anguli oris muscle arises from the canine fossa, lateral to the incisive fossa. Above the canine fossa is the infraorbital foramen, the anterior end of the infraorbital canal, which transmits the infraorbital vessels and nerve. Above the foramen on the margin of the orbit is attached part of the levator labii superioris. The nasal notch medially limits the anterior surface of the maxilla and nearby the nasalis and depressor septi muscles are attached. Bilaterally the two nasal notches join to form a pointed process, the anterior nasal spine.

Laterally, the convex infratemporal surface of the maxilla forms the inferior part of the infratemporal fossa. The maxillary tuberosity forms the lower part of this surface and on its medial side articulates with the pyramidal process of the palatine bone. Inconstantly, it articulates with the lateral pterygoid plate of the sphenoid and gives origin to a few fibers of the medial pterygoid muscle. Just above this is the pterygopalatine fossa, with a groove for the maxillary nerve.

The maxillary orbital surface forms part of the floor of the orbit. Medially, behind the lacrimal notch, the margin articulates with the lacrimal bone, the ethmoid's orbital plate and the palatine's orbital process. Posteriorly, it constitutes the posterior border of the inferior orbital fissure. Anteriorly it forms part of the orbital margin, which is continuous medially with the frontal process and laterally with the zygomatic process. Anteriorly and medially, the inferior oblique muscle originates just lateral to the lacrimal groove.

On the nasal surface is the maxillary hiatus, the large opening of the maxillary sinus. The superior border of the maxillary nasal surface articulates with the ethmoid and lacrimal bones. The inferior meatus of the nasal cavity is located below the maxillary hiatus and behind a surface for articulation with the perpendicular plate of the palatine bone. A groove crosses this surface, running obliquely downward and forward, and forms, with the palatine bone, the greater palatine canal. Anterior to the maxillary hiatus a deep groove forms part of the nasolacrimal canal.

The zygomatic process of the maxilla is triangular and is situated at the convergence of the anterior, posterior and orbital surfaces. The frontal process forms part of the lateral boundary of the nose projecting posterosuperiorly. It gives attachment to part of the orbicularis oculi and levator labii superioris alaeque nasi. Its medial surface forms part of the lateral wall of the nasal cavity. The upper border articulates with the frontal bone, the anterior border with the nasal bone and the posterior border with the lacrimal bone.

The alveolar process is very thick, being broader behind than it is in front. It contains eight deep cavities for the roots of the teeth. These cavities vary in size and depth: the cavities for the canine teeth are the deepest; the cavities for the molars are the widest. The alveolar processes of the maxillae articulated together form the alveolar arch. The buccinator muscle arises from the lateral surface of the alveolar process, as far forward as the first molar.

The palatine process projects horizontally and medially from the nasal surface of the maxilla. It constitutes an important part of the floor of the nasal cavity and the roof of the mouth. Its inferior surface is concave and forms, with the palatine process of the opposite side, the anterior three-quarters of the osseous plate of the palate. In young skulls, a fine linear suture, the incisive suture, may be observed. It extends from the incisive fossa, behind the incisor teeth, to the space between the lateral incisor and canine teeth. The small part in front of this suture forms the premaxilla (os incisivum) that contains the sockets of the incisor teeth. The two palatine processes join to form the median intermaxillary palatal suture. The margins are sometimes raised and form a prominent palatine torus. The medial border

of the superior surface of the palatine process forms a ridge, the nasal crest. With the opposite side, it forms a groove for the vomer. The posterior border is articulated with the horizontal plate of the palatine bone (Figs 7.7.1, 3.5).

The maxilla ossifies in a mesenchymatous sheet. The number of ossification centers is debated and between two and four such centers may appear during the 7th week of fetal life. They form a premaxilla (os incisivum) and a maxilla that start to unite at the beginning of the 3rd month of development. The identification of the junction between these two parts as a suture is also debated,[3] and information is lacking to demonstrate its role as a growth site.[4] A line or cleft, however, may be observed in the anterior part of the palate until the middle decades of life.[1] This site – be it suture, line or cleft – is of consequence in osteopathic practice because it provides a hinge-like location where intraosseous maxillary somatic dysfunction can develop. Such dysfunction is commonly found in infants and children as the result of activities such as thumb sucking and from falls where the area is injured. If not treated, this dysfunction will have a significant impact that can only increase as the structures grow. Orofacial dysfunctions such as malocclusion or speech disorders may follow.

The maxillary sinus, described as a small furrow at birth, reaches its full size after the second dentition. As such, the size of the maxillary body at birth is small, the teeth sockets located almost at the level of the floor of the orbit. The relatively small length of the maxillary vertical dimension, when compared to that of the adult, gives the infant the appearance of having large eyes. Augmentation in volume of the maxillary sinus and development of the alveolar processes will contribute to an increase in the vertical dimension of the maxillary body (Fig. 2.13).

The maxilla articulates with nine bones including the frontal, ethmoid, zygomatic, nasal, lacrimal, inferior nasal concha, palatine, vomer and the opposite maxilla. The articulation with the orbital surface or with the lateral pterygoid plate of the sphenoid is inconstant.

On the other hand, the mandible articulates only with the two temporal bones. It consists of a curved horizontal body and two perpendicular portions, the rami. It is the largest and strongest bone of the face and contains the alveoli for the roots of the lower teeth. Its shape and position determine the positional arrangement of the lower teeth and as such it contributes to the relationship between the occlusal surfaces of the maxillary and mandibular teeth when they are in contact.

The mandibular body has two surfaces and two borders. In the midline of the external surface is a small ridge, the remnant of the line of fusion between the two halves of the mandible at the symphysis menti. This ridge divides inferiorly to surround the mental protuberance. The mentalis muscle and a small portion of the orbicularis oris are inserted below the incisor teeth on either side of the ridge. Laterally, the depressor labii inferioris, depressor anguli oris and platysma muscles are attached.

The internal surface is concave and the paired superior and inferior mental spines are situated on either side of the symphysis menti. The genioglossi muscles have their origins on the superior spines and insert on the lingual fascia beneath the mucous membrane and on the hyoid bone. They depress and protrude the tongue. If their origin on the mandible is dysfunctionally positioned, their leverage will be altered and consequently their effect on the tongue will, in turn, be dysfunctional. The geniohyoid muscles insert on the inferior mental spines, and the

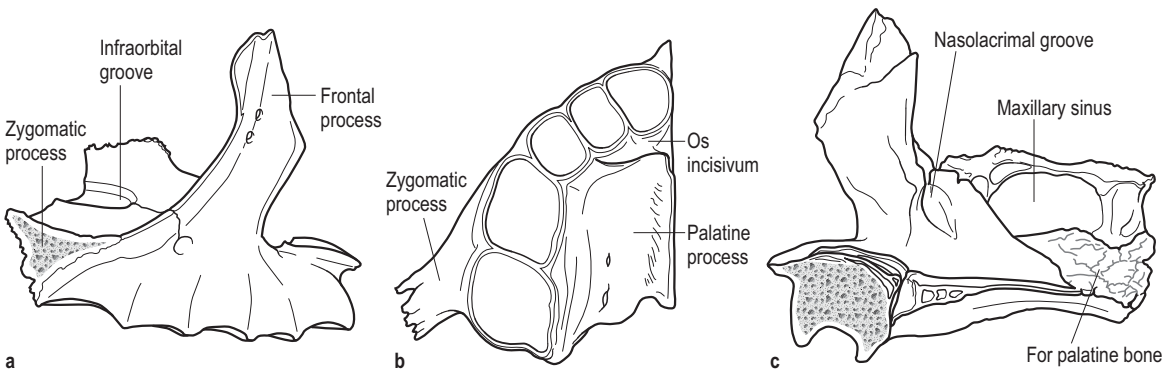

Figure 7.7.1. *Right maxilla at birth: (a) lateral aspect; (b) inferior aspect; (c) medial aspect.*

anterior belly of the digastric insert below the mental spines, on either side of the midline.

On either side, an oblique line, the mylohyoid line, runs from a point inferior to the mental spine upward and backward to the ramus behind the third molar tooth. It gives attachment to the mylohyoid muscle, the lower-most part of the superior constrictor of the pharynx, and the pterygomandibular raphe. The superior constrictor, pterygomandibular raphe and buccinator are intimately joined together to the mandibular periosteum. Furthermore, they form a continuous band that unites the orofacial structures with the cranial base and cervical spine (Fig. 7.7.3). Thus, postural imbalances in the axial skeleton can affect the orofacial structures, potentially leading to orofacial dysfunction and malocclusion.

The upper or alveolar border contains 16 cavities for the roots of the teeth. On either side, the buccinator muscle is attached on the outer lip of the superior border, as far forward as the first molar tooth. The buccinator has its origin posteriorly on the lateral aspect of the maxilla and oblique line of the mandible, the pterygoid hamulus and pterygomandibular raphe (Figs 7.7.2, 7.7.3). It inserts anteriorly on the angle of the mouth, the middle fibers of the muscle crossing in the region of the modiolus, the lower set passing to the upper lip, the upper set to the lower lip.[5] The modiolus located near the corner of the mouth represents a convergence of several muscles of facial expression and, as such, observation of this area is highly indicative of the psychoemotional state of the individual. The fibers of the buccinator muscle are interspersed with more horizontal portions of the orbicularis oris muscle. The buccinator flattens the cheek, retracts the angle of the mouth and plays an important role in mastication in conjunction with the tongue and orbicularis oris muscle to form a belt on each side of the oral cavity, preventing food from accumulating in the oral vestibule.

The quadrilaterally shaped mandibular ramus consists of two surfaces, four borders and two processes. The lateral surface is flat and gives attachment to the masseter muscle. The masseter muscle is attached above on the inferior border of the anterior two-thirds of the zygomatic arch and the medial surface of the zygomatic arch. It inserts inferiorly on the lateral surface of the ramus and the coronoid process of the mandible. It elevates the mandible and its function is of paramount importance in the development, growth and maturation of the mandibular condyles and fossae. A reduction of masseter muscle activity in bottle-fed babies has been observed[6] and, with the loss of chewing behavior, may predispose to chewing/swallowing disorders and malocclusions.[7]

Located on the medial surface of the mandibular ramus, approximately in its center, is the mandibular foramen leading to the mandibular canal. It contains the alveolar nerve and vessels. A sharp spine on the anterior margin of the foramen, the lingula (Spix spine), provides attachment for the sphenomandibular ligament. This site is of particular interest in mandibular kinematics because mandibular motion

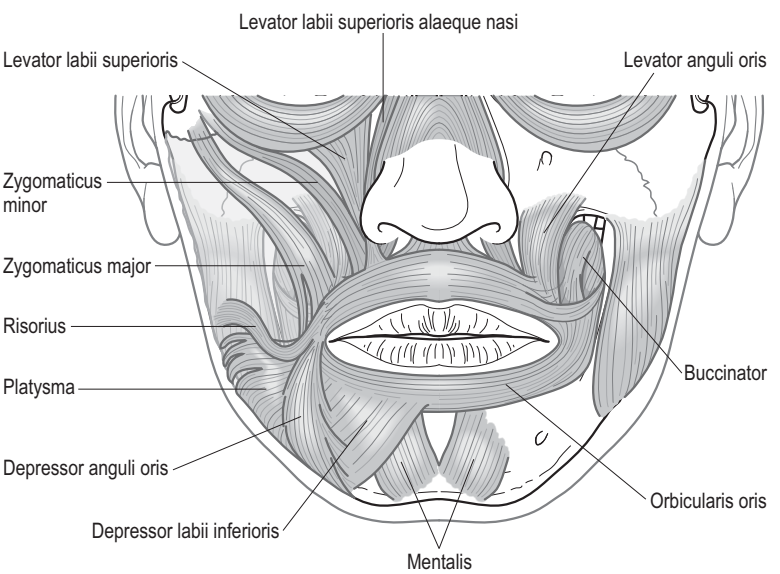

Figure 7.7.2. *Orofacial muscles.*

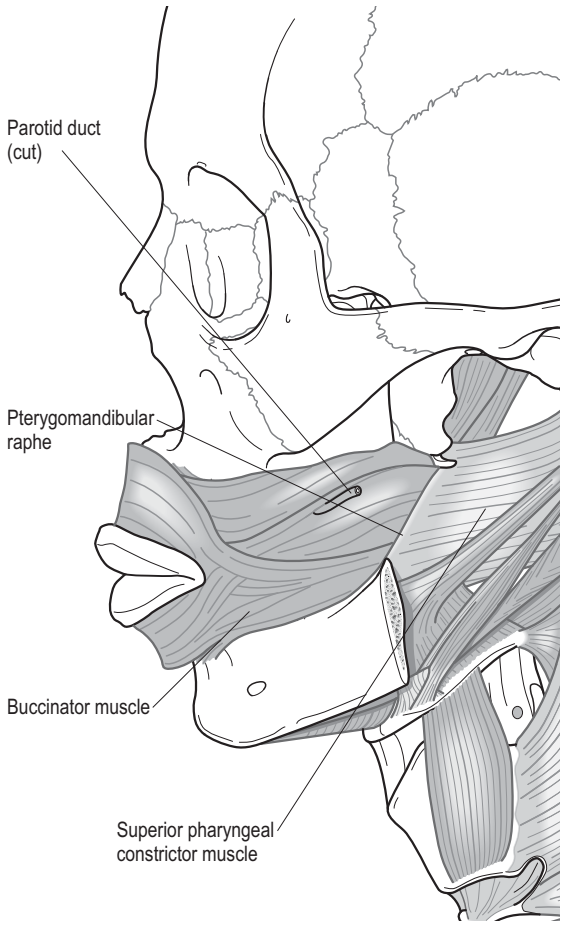

Figure 7.7.3. *Buccinator muscle.*

is centered on a point located near the lingula. The vascular–neural bundle is, therefore, protected from injury caused by normal mandibular motion.[8]

The medial pterygoid muscle inserts below the mandibular foramen, on the medial surface of the mandibular angle. Thus, the mandibular angle is affected by the action of the masseter laterally and medial pterygoid medially. Traction from these muscles dynamically influences the growth of the mandible. This influence has to be balanced unilaterally between the masseter and medial pterygoid, as well as bilaterally.

The coronoid process is a thin, flattened piece that forms the anterior limit of the mandibular incisure. The temporalis and masseter muscles are attached on its lateral surface; the temporalis also covers part of the medial surface and anterior border of the ramus. The temporalis muscle has its origin on the temporal fossa and functions to elevate the mandible and close the jaw. Its posterior, almost horizontally oriented fibers are the primary retractors of the protruded mandible. These muscles determine chewing action and, when dysfunctional, can have significant impact on the mandible.

The condylar process provides an articular surface with the disk of the temporomandibular joint (TMJ). Its long axis is slightly oblique and directed medially and posteriorly. The neck that unites the head or condylar process to the ramus has the pterygoid fovea (anteriorly) for the attachment of the lateral pterygoid muscle. This masticatory muscle has two heads: the inferior head has its origin on the lateral plate of the pterygoid process; the superior head originates on the infratemporal crest and adjacent greater wing of the sphenoid. In addition to its insertion on the mandible, some of its fibers join the articular disk and capsule of the TMJ. It acts to protrude the lower jaw and also contributes to the opening of the mouth. Unilateral pterygoid contraction deviates the chin laterally, enabling grinding motion for chewing. This muscle is important in the development of children's orofacial structures, where unilateral functional patterns will stimulate growth in an asymmetric fashion. Alternatively, structural asymmetries may, in turn, prevent the individual from having symmetric functional patterns.

Ossification of the mandible occurs in the fibrous membrane covering the outer surfaces of Meckel's cartilages. These cartilages develop bilaterally in the first pharyngeal arches. The primitive TMJ starts to organize during the 7th week of development, concomitantly with the emergence of muscular activity in the masticatory apparatus. At birth the mandible consists of two parts separated by a cartilaginous symphysis menti not yet ossified. The body, although rather underdeveloped, is much larger than the rami. It consists of relatively thin cortices with tooth buds almost totally occupying its volume.[9] Each side contains the sockets of the two incisors, the canine and the two deciduous molar teeth. The angle between the body and the ramus is obtuse (175°), and the coronoid process, of rather large size, projects above the level of the condyle. At birth, the TMJ is slack. The mandibular fossa of the temporal bone in which the condylar process of the mandible is located is almost flat, providing little stability.[10] During the first years of life growth will occur through bony deposition and resorption. Bone deposition occurs on the mandibular labial side, whereas resorption occurs on the lingual side, thus allowing for elongation of the mandible and more space for the deciduous dentition.[11] The two halves of the mandible

join during the 1st year, although a line of separation may still be seen in the beginning of the 2nd year. Concomitant development of mastication stimulates growth of the alveolar and subdental portions of the mandibular body, allowing in turn more powerful traction from the masticatory muscles. During the first 3 years of life, mandibular bicondylar width grows rapidly, in synchrony with cranial base growth. It is the fastest growth period of the mandible. With the development of permanent dentition, the angle becomes less obtuse – 140° at about the 4th year. Crown formation of the permanent teeth, and their following eruption, is a time of possible occlusal instability, lasting up to 10–12 years of age when the majority of the permanent teeth have erupted. After puberty, the mandibular angle is 120° (Fig. 7.4.4).

Development of the teeth, like the development of the other components of the craniofacial complex – jaws, dental arches, tongue and myofascial structures – is under the influence of genetic and environmental factors. This development is largely influenced by the surrounding structures of the cranium, particularly the temporal bones with which the mandible articulates, and the sphenoid and hyoid bones that are part of the skeletal framework of the oral cavity.

At the root of the zygomatic process, in the squamous portion of the temporal bone, is the mandibular fossa, with which the condylar process of the mandible articulates. This fossa, also called the glenoid cavity, is a deep hollow in the adult but is almost flat in the infant. A disk separates the mandibular condylar process from the temporal glenoid cavity, and a fibrous capsule and synovial membrane surround the joint. The position of the condylar process of the mandible is greatly influenced by the position of the mandibular fossa of the temporal bone. The relationship can be potentially dysfunctional if one or several components of the TMJ demonstrate somatic dysfunction. In the cranial concept, during cranial external rotation, the mandibular condyles move somewhat posteromedially, following the mandibular fossae of the temporal bones, and the chin recedes. Concomitantly, the mandibular angles move laterally. The opposite occurs during cranial internal rotation when the mandibular fossae of the temporal bones move anteriorly. As a result, the mandible moves anteriorly, with the chin becoming more prominent.

The deep cervical fascia and stylomandibular ligament also contribute to the relationship between the temporal bones and mandible. The deep fascia of the neck is divided into an external or investing layer and a pretracheal layer. The former surrounds the neck and encloses the trapezius and sternocleidomastoid muscles. Above, it fuses with the periosteum along the superior nuchal line of the occipital bone, on the mastoid and styloid processes of the temporal bone and the complete base of the

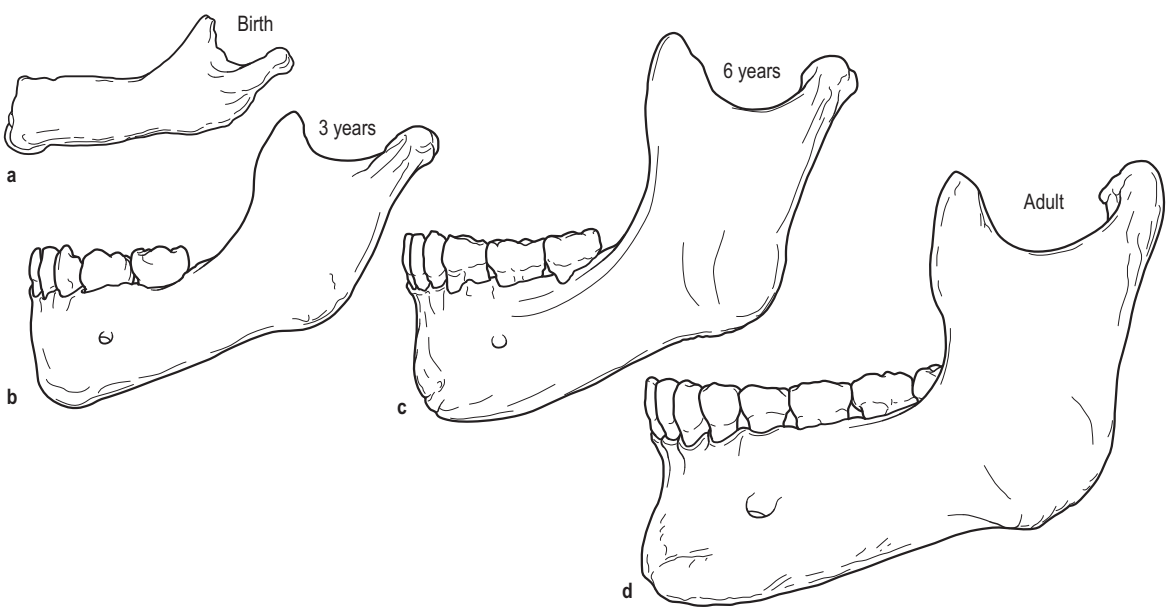

Figure 7.7.4. *(a–d) Development of the mandible.*

mandible.[1] The stylomandibular ligament, a condensation of the deep cervical fascia, extends from the tip of the styloid process of the temporal bone to the posterior border of the angle of the mandible. This fascia links the mandible to the base of the skull. Thus, dysfunction of the cranial base may affect the mandible, and vice versa. It is of great significance in the first years of life, when structures have not yet completed their growth. As such, torticollis or plagiocephaly, if untreated, may predispose to somatic dysfunction of the mandible and viscerocranium, leading to malocclusion.[12]

As it belongs to the cranial base, the sphenoid also influences the mandible through its muscular and ligamentous relations. The sphenomandibular ligament is of particular importance. This fibrous band, a remnant of Meckel's cartilage, runs from the spine of the sphenoid's greater wing to the lingula of the mandible. It constitutes the primary passive support of the mandible, with the area of the lingula acting as an anchor. An artificial axis passing through the two lingulae may be proposed, around which motions of the mandible occur. These motions include depression of the mandible during opening of the mouth and elevation during closure, as well as protraction and retraction. The mandible may be compared to a swing hanging under the sphenoid's greater wings. This comparison is particularly valid in infants and young children, where the TMJ is quite loose and the sphenomandibular ligament well defined.

Other links between the mandible and sphenoid include the bilateral pterygomandibular raphe and pterygoid muscles joining the pterygoid process to the mandible. The pterygomandibular raphe, also referred to as the pterygomandibular ligament, is a thickening of the buccopharyngeal fascia. It separates and gives origin to the buccinator muscle anteriorly and the superior constrictor of the pharynx posteriorly. The pterygomandibular raphe is attached superiorly to the pterygoid hamulus, the hook-shaped inferior extremity of the medial plate of the pterygoid process. Below, it is attached above the mylohyoid line behind the third molar tooth.

Indeed, multiple influences exist between the cranial base and the facial bones, potentially affecting the orofacial structures. The human profile and the position of the mandible have been correlated postnatally with the basicranial shape.[13] Furthermore, facial morphology is more related to variation of the lateral part of the basicranium than to flexion occurring in the midline.[14] Epigenetic factors play a significant role in this process. This role is largely fulfilled by several orofacial functions that start as early as the first weeks of development. It is hypothesized that, around 8 weeks of development, early mandibular movement participates in the differentiation of the primary mandible from Meckel's cartilage.[15]

Orofacial functions

Consideration of orofacial function is of great significance in the understanding and treatment of any orofacial disorder or malocclusion. To a large extent, orofacial function, interconnected with the musculoskeletal apparatus in a complex system, contributes to the formation of the oral cavity and viscerocranium. Deglutition, sucking, ventilation, facial expression, mastication and speech are the sequential events that, through dynamic processes, constantly influence the growth and development of the orofacial components.

For most of these activities, the tongue plays a central and vital part. In feeding, it moves food through the oral cavity for chewing and then to the pharynx for swallowing. In respiration, its position, relative to the posterior pharyngeal wall, determines the dimensions and shape of the airway for air to flow between the palate and the posterior tongue. With the larynx, the tongue contributes to the production of sounds and speech. Finally, the tongue seems to play an important proprioceptive role that, in turn, functionally or dysfunctionally regulates all of these activities.[1]

The tongue consists of a mass of muscle covered by mucous membrane, where the papillae of the gustatory organ are located. The lingual musculature is divided into intrinsic and extrinsic muscles. The intrinsic muscles, which are totally contained inside the tongue, are the bilateral superior and inferior longitudinal, the transverse and the vertical lingual muscles. Conversely, the extrinsic muscles extend outside the tongue and consist of the genioglossus, hyoglossus, styloglossus and palatoglossus muscles. The lingual musculature matures quite early in life, transforming the tongue into a powerful growth stimulator for the surrounding structures.

At birth, the infant tongue is totally intraoral and its extrinsic muscles are short. In the infant, the tongue fills the oral cavity, contacting the soft and osseous palates, the cheeks and lower lip. Although mobile, the tongue is solidly anchored at its base on the mandible by the paired genioglossi and on the hyoid bone by the genioglossi and hyoglossi muscles. In addition, the lateral surfaces of the tongue are suspended from the soft palate by the palatoglossi and from the styloid processes of the temporal bones

by the styloglossi muscles. The fibers of these muscles blend with the superior portion of the tongue.

As in other primates, the larynx of the newborn is positioned high, with the epiglottis in direct contact with the soft palate. During the first years of life, in association with the growth of the cervical spine, the hyoid bone migrates from the level of C1–C2 at birth to C3–C4 after puberty.[16] The posterior third of the tongue follows this caudal migration, consequently stretching its superior attachments. This contributes to change the orientation of the soft palate from horizontal in the infant to more vertical in the adult.[17] Although the positional changes occur mainly during the first years of life, the reciprocal influences, from the cranial base and hyoid bone acting on the tongue and soft palate, should be kept in mind, no matter the age of the patient.

Normally, at about 4 or 5 years of age, after the descent of the larynx and tongue, the tongue rests lower in the mouth.[18] At this time it forms part of the floor of the oral cavity and part of the anterior wall of the oropharynx.[1] It is contained inside the mandibular arch and the functions of swallowing, sucking, ventilation, facial expression, chewing and phonation should be performed without difficulty. Dysfunction develops when lingual mobility and function are impaired. This may be the result of somatic dysfunction affecting the cranial base, hyoid bone or mandible, or accommodation of these areas to dysfunction in more distant body areas.

Tongue-tie, or ankyloglossia, is a partial or complete adhesion of the tongue to the floor of the mouth. Because of abnormal shortness, the frenulum linguae tethers the tongue to the floor of the mouth, impairs lingual mobility and, in severe cases, prohibits its extension beyond the lower gum. Although it may present as part of several craniofacial syndromes, in most cases the child is perfectly healthy.[19] Ankyloglossia is, however, associated with up to 12.8% of serious breastfeeding problems.[20] It is also a potential etiology for speech disorders[21] and dental problems,[22] such as diastasis between the lower incisors due to the lingual pressure.[23] Furthermore, impaired lingual mobility is related to difficulty with intraoral toileting and later with the playing of wind instruments.[23] Although surgical treatment (frenuloplasty or tongue-tie division) remains debated, in specific indications it is reported to improve difficult breastfeeding and to protect the maternal nipple,[24] and to alleviate speech disorders and improve tongue mobility.[25]

Swallowing, or deglutition, is one of the first orofacial functions to appear in utero and has been observed after 11 weeks, when the child reflexively swallows amniotic fluid.[1] At birth, new habits develop with feeding, allowing the evolution of the pattern of deglutition. In the infant, sucking and swallowing are the results of a pumping action in the hyolingual complex, with a rhythmic tongue thrust, the tip of the tongue showing regularly between the alveolar processes. Musculoskeletal growth and maturation of the neuromuscular system result in the development of a more mature or adult swallowing pattern. At around 2–3 years of age, at rest and during swallowing, the apex of the tongue is normally located on the palate, behind the upper incisor teeth. When swallowing, the lips are closed, but the perioral musculature, particularly the orbicularis oris and mentalis muscles, is relaxed. Lingual pressure applied against the palate contributes to the development of a broad palate and well-developed maxillae. The tongue works as a natural orthodontic appliance 'for better or for worse'.[26]

In the cheeks, the muscular bands formed by the buccinator and superior constrictor muscles, joined together through the pterygomandibular raphe, bilaterally act to constrain the tongue within the oral cavity. Thus, functional balance or dysfunction of the cervical spine and cranial base, the sites of origin of the superior constrictor, contributes significantly to lingual posture. In addition, because the tongue is anchored on the mandible, it affects and is affected by mandibular position and growth. When mature or adult swallowing does not develop successfully, immature or atypical swallowing will continue.

Chewing forces also contribute to maxillofacial growth. First, mastication occurs purely in the sagittal plane, with the mandible moving up and down, and demonstrating propulsion and retraction. Then, progressively, an alternating unilateral chewing pattern appears, with diagonal movements of the mandible.[8] The masticatory muscles develop in response to this demand. At around 9 months of age, a child can chew soft food and keep it in their mouth; 1 year later they can chew more solid foods.[27] As such, their diet should include food that requires sufficient chewing to allow satisfactory occlusal force per chew that can, in turn, stimulate periosteal growth. Symmetric chewing is a requisite for adequate muscular stimulation of the digastric, temporal and pterygoid muscles. The traction of these muscles will, in turn, stimulate bone growth. At this time cranial balance is necessary to facilitate the establishment of symmetric masticatory patterns that are learned and practiced throughout the rest of life,

making early identification and treatment of somatic dysfunction essential.

Besides swallowing and chewing, ventilation, speech and facial expression also contribute to the development and growth of the maxillofacial structures. Ventilation, as a vital function, is present from birth onwards. In the first months, the infant is normally a nasal breather. At this time, observed respiration should be unencumbered. It is imperative that airflow be quiet and through both nares equally. Dysfunctional nasal respiration will impact the development of the orofacial structures, potentially leading to mouth breathing.

Establishment of the route of respiration is partly under the control of the soft palate. This muscular fold, suspended from the posterior border of the bony palate, extends inferiorly and posteriorly into the oropharynx. Under normal circumstances the soft palate and tongue act in apposition to close the oropharyngeal isthmus; conversely, when the soft palate rises and contacts the posterior pharyngeal wall, the nasopharynx is closed. Thus, the position of the soft palate determines the route of respiration and regulation of airflow through the nose or mouth. The soft palate is united with the tongue through the palatoglossi muscles and with the pharynx through the palatopharyngei muscles. For optimal function of all of the structures that contribute to upper airway respiration, somatic dysfunction affecting the sites of origin of these muscles should be identified and treated. For optimal breathing, the cranial base, hyoid bone and mandible should be balanced. It is well established that an inferoposterior displacement of the hyoid bone and an anteroinferior positioning of the tongue are correlated with mouth breathing.[28–30]

Normally, the tongue demonstrates large movements in all three planes of space.[31] In the production of sounds and speech, the tongue must change shape and the dorsum of the tongue must contact the palate. Shortening of the base of the tongue results from anterior displacement of the hyoid bone, whereas lengthening of the base of the tongue results from its posterior displacement. By employing the activity of the lingual muscles, speech contributes to the overall function and development of the orofacial cavity.

Although the hyoid bone has no direct articulation with other skeletal structures, it is an interface between the mandible and tongue above and the upper thoracic area below. Consequently, its position and motion are influenced by changes occurring at the level of the mandible or anywhere in the thorax. A hyolingual complex – a 'kinetic chain' – may be described,[32] part of a more global oropharyngeal complex where every structure is interdependent with others as part of the system.

Physical examination and treatment

Begin by noting the relationship between the head and the remainder of the body. The skull should be centered above the spine. Note any tension in the posterior, lateral and anterior (submandibular) cervical musculature. Divide the viscerocranium into three regions: frontal, nasal and buccal. Observe the relationship between these three regions and the relative harmony between them. In newborns, the lower two regions are proportionately smaller than in older children, adolescents and adults because the paranasal sinuses are not yet developed and the teeth have not erupted.

Observe facial expression and orofacial functions such as breathing, sucking (in infants) and swallowing. Look for coordination of the tongue and orofacial musculature, the rate of sucking if the patient is a baby, and possible tongue sucking or tongue thrusting. Note the ease of swallowing without any concomitant inappropriate muscular contraction if the patient is a child. If they are old enough to respond, ask them to describe the location of the tip of their tongue during swallowing; normally it should contact the hard palate behind the upper incisors.

Observe the resting respiration. Nasal patency of airflow can be assessed easily with a wisp of cotton held adjacent to each nasal aperture or look for fogging with exhalation on the convex side of a cold metal spoon or a cold mirror.

Observe the midline of the face, the metopic suture, nose and symphysis menti. All of these landmarks should be aligned in a straight line

Observe the maxillae and compare size and shape: in external rotation the maxilla appears wider; in internal rotation it appears narrower.

Observe the position of the mandible. Note its relationship with the maxillae, particularly its centric position. Observe the location of the gnathion; newborns are normally retrognathic. With the infant, an open mouth posture with tongue forward may be observed with dysfunction of the cranial base, mandible or hyoid bone.

With children, observe the mouth area and the lips for symmetry and tonicity. The upper lip reflects the functional pattern of the maxillae, whereas the lower lip reflects the mandible. Look for the capacity to keep the lips closed and in soft contact. There should be no tension, in particular in the orbicularis oris or mentalis muscle, and no protrusion, retraction or pursing of the lips. Note any abnormal

perioral muscle function or nervous habits such as finger and nail picking or nail biting.

Compare the nasolabial sulci for depth and obliquity. Increased sulcus depth is associated with external rotation of the ipsilateral maxilla and/or the zygoma, whereas decreased depth is associated with internal rotation of these bones.

Look inside the mouth. Assess the position and function of the tongue. Note the presence of a large frenulum linguae. If the child is old enough to voluntarily open the mouth, look at the relative position of the tongue within the oral cavity. It should be inside the mandibular arch and should not cover the lower teeth. Again, if possible, have the child slightly protract their tongue to look for the presence of dental imprints on the lateral aspects of the tongue, unilateral or bilateral, indicating lingual malposition and/or dental malalignment. With the tongue protracted, look for tongue deviation. Assess the mobility of the tongue, looking for limitation of movement. Dysfunctional tongue posture is often accompanied by decreased tone and eversion of the lower lip.

Observe the teeth as to position, dental attrition and progression of dental development according to the child's age. Observe clenching of the teeth and observe the occlusion of the teeth. The upper and lower midline between the incisors should be in alignment. Normally, the upper incisors should slightly override the lower incisors and upper molars should rest on the lower molars. Note misalignment or protrusion of the upper or lower incisors. Note any crowding of the teeth. If asymmetric crowding is present, look to see if there is ipsilateral cranial internal rotation. Note the potential association with impaired ipsilateral nasal breathing and/or dysfunctional mastication.

Observe the shape and symmetry of the palate: a lower, flattened palate with everted teeth is associated with external rotation; a high arched palate with inwardly directed teeth is associated with internal rotation.

Observe the mandible. It should appear balanced under the cheeks. If not, differentiate between asymmetry of position and asymmetry of size and shape. Asymmetry of mandibular position is associated with asymmetry of the temporal bones: external rotation of the temporal bone results in posterior displacement of the mandibular fossa; internal rotation results in anterior fossa displacement. The chin will be displaced toward the side of temporal external rotation.

If asymmetry of mandibular size and shape is noted, look for a potential cause. Structural asymmetry may result from intraosseous mandibular dysfunction, dental malalignment or asymmetric orofacial function, such as mastication occurring only on one side.

Note the consistency between orofacial findings and the cranial pattern. If they are in concordance, a cranial osteopathic approach may be indicated. Palpation for function and treatment of identified dysfunction should follow. When treating orofacial problems, don't forget to check and treat somatic dysfunction of other areas, frequently the cranial base, craniocervical junction and upper thoracic area. Cranial procedures directed at release of the global membranous strain pattern, the SBS, vault and facial bones to ensure optimal freedom of movement are anecdotally associated with facilitation of the teething process. It is also important to remember that all tooth buds are present in the maxillae and mandible, even though not yet erupted. Any finger or object placed inside the mouth of a child can act as an orthodontic device and move the teeth. Therefore, it is inappropriate to employ force when examining the patient or using osteopathic manipulation to treat children's orofacial somatic dysfunction. The earlier osteopathic treatment of orofacial dysfunction is initiated, the better. Results may be enhanced when osteopathic treatment is employed in conjunction with appropriate orthodontic treatment.

MALOCCLUSION AND OROFACIAL DYSFUNCTIONS

Dental development begins in the 3rd month of intrauterine life and ends at approximately 25 years of age. Throughout this period profound occlusal events occur, resulting in potential sources of stress for the orofacial structures. To understand the evolution of the occlusal mechanism, it is important to begin with the infant and to follow the eruption and arrangement of the teeth along the dental arches.

During the first months, the absence of teeth allows total freedom in the displacement of the mandible and the child can experiment and discover objects surrounding them by bringing everything to their mouth. The lower central incisors are normally the first to appear between 6 and 8 months of age. When the upper central incisors emerge, the first occlusal relationship appears, associated with new constraint for mandibular mobility. The upper and lower lateral incisors are almost completely erupted at around 14 months, the time when the first molars appear. Just before the end of the 2nd year of life,

the central and lateral incisors and the first molars are in occlusion, the canines have erupted and the second molars are beginning to emerge. In the 2nd year of life, the 20 deciduous teeth are present, with occlusion between the incisors, canines and first molars. At the same time, the child's orofacial functions have matured and their food is becoming more solid. Mouthing (active oral interaction with the environment by sucking different objects) is still quite frequent up to 3 years of age. Several explanations – from a means of exploring their environment to proactively exposing the naive GI tract to environmental antigens – have been offered for this behavior.[33] Whatever the reason, mouthing effectively contributes to dental attrition, thereby eliminating any dysfunctional occlusal contact and facilitating functional adaptation. Smoothing of the occlusal surfaces results in proprioception important in establishing functional balance of mandibular motion patterns. Conversely, any dysfunctional occlusal contact will result in dysfunctional mandibular motion patterns and stimulate abnormal mandibular and maxillary growth.[34] It will also prevent the establishment of alternating unilateral mastication.

The teeth develop in sockets in the alveolar parts of the maxillae and mandible. They are each held in their respective socket by a periodontal ligament. This anchors the teeth solidly while still allowing micromovements. It is also the site for periodontal innervation that is of paramount importance in the development and control of orofacial praxis. Proprioceptive input from the periodontal ligament in association with proprioception from the TMJ and surrounding myofascial structures provides constant information to the CNS. This allows the individual to adjust to the challenges of mastication as well as to global postural mechanics.[35]

Deciduous teeth are small, with thin enamel coverage, allowing rapid wearing out. On the other hand, permanent teeth are the hardest of all tissues in the body. Each consists of a crown and a root, meeting at the cervical margin. Dentine forms most of the tooth, with a central pulp cavity ending in a pulp chamber and canal. The side of the tooth in contact with the lips and cheeks is the labial or buccal surface while the side in contact with the tongue is the lingual or palatal surface. When the jaws are brought together the teeth meet, or occlude; thus, dental occlusion occurs. Occlusion is qualified according to the respective positions of the teeth. A centric occlusion takes place when the relation of opposing occlusal surfaces of mandibular and maxillary teeth provides the maximum contact, or intercuspation. In such circumstances, the mandible is in centric relation to the maxillae.

Infants usually demonstrate a retrusive mandibular position. When the first teeth erupt, the relationship will tend to show a horizontal protrusion, or overjet, of upper incisors beyond the lower incisors. However, at this early age tongue thrusting will result in a functional edge-to-edge repositioning of the teeth. In the first years of life, during eruption of the deciduous teeth, changes in the orofacial osseous and myofascial structures normally allow sufficient space for the teeth to emerge. Typically, by approximately 6 years of age, there is occlusion between all deciduous teeth that already gives a good idea of the occlusal pattern of the future permanent teeth. The overjet and overbite relationship should have resolved in order to allow for freedom of mandibular diduction, which is only possible if the mandible can slide forward slightly. This sequence occurs as the result of an alternating chewing pattern, enhanced by chewing the solid foods found in the correct type of diet.

It is important for the osteopathic practitioner to observe the oral cavity and teeth in order to identify their eruptive pattern, position, occlusal contacts and any asymmetric wear that may be associated with cranial somatic dysfunction. In addition, any asymmetry of function that predisposes the child to mouth and then chew using only one side, as might occur with a neurologic impairment, a child with one handicapped upper extremity or simply a child with a torticollis, will result in asymmetric wear of the occlusive surfaces of the teeth and eventually asymmetric mandibular growth.

The permanent incisors are significantly wider than the deciduous incisors that they have replaced by approximately 8 years of age. There might be a tendency for overjet or overbite, either positive or negative. While a horizontal protrusion of upper incisors beyond the lower ones is named overjet, a vertical overlap is named overbite (Fig. 7.7.5). At this age, the child's temporary canines, the two temporary molars and the first permanent molars are in occlusion. Around 10 years of age, occlusion exists between the permanent incisors, the temporary canines and the first permanent molars. At 12 years of age, occlusion is typically present between the permanent incisors, permanent canines and first permanent molars.

In mixed dentition, if the child has developed an asymmetric pattern of chewing, with a tendency to chew only on one side, they may wear out the teeth in a way that will not allow functional and symmetric development. Unsatisfactory occlusal contact

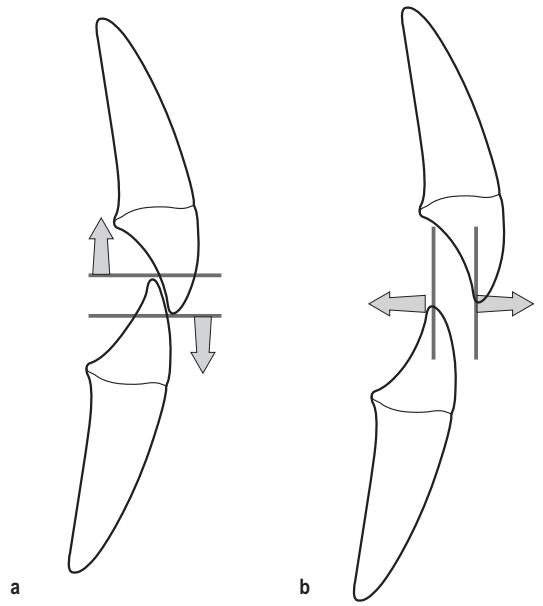

Figure 7.7.5. *Occlusal relationships: (a) overbite is the vertical overlap of the upper incisors over the lower incisors; (b) overjet is the horizontal protrusion of the upper incisors beyond the lower incisors.*

will create points of resistance that will, in turn, control and guide the movement of the mandible in asymmetric patterns. The masticatory, perioral and craniocervical muscles, as well as the highly elaborate TMJ structures, result in engrams of the repetitive dysfunctional pattern. If no change occurs, this will be the permanent pattern ingrained in the CNS. Thus, it is important to recognize asymmetric patterns and to identify and treat any underlying somatic dysfunction. Because patterns of function become fixed as the child ages, for the best therapeutic outcomes the cranial osteopathic approach should be employed at the earliest opportunity.

At the end of the 19th century, Edward Angle proposed that a definitive nomenclature was necessary in orthodontia as in anatomy.[36] He felt that the term malocclusion was far more expressive than 'irregularities of the teeth' to describe not only the relationship between the maxillary and mandibular arches, but also of the individual teeth to one another. As a result, a classification was born, referred to as Angle's classification.

Angle's classification is based on the mesiodistal relationship of the permanent molars. In dentistry, distal means away from the median sagittal plane of the face, following the curvature of the dental arch, while mesial means proximal. Three classes of dental arrangement are described. Class I identifies the normal relationship of the jaws, where the mesiobuccal cusp of the maxillary first molar occludes in the buccal groove of the mandibular first molar. In the individual with normal dentition and centric occlusion, the lower incisors bite against the lingual surfaces of the upper incisors, the crowns of the lower incisors being covered in their superior third. In addition, from the canine teeth backwards, each lower tooth is slightly in front of its upper fellow. Classes II and III categorize variations of malocclusion. In Class II, all the lower teeth occlude distal to normal, wherein the distobuccal cusp of the maxillary first molar occludes in the buccal groove of the mandibular first molar. In addition, this class is further subdivided into Division 1, labioversion of maxillary incisor teeth, and Division 2, linguoversion of maxillary central incisors. This may occur unilaterally of bilaterally. Class II is the most frequently encountered type of malocclusion. In Class III, all the lower teeth occlude mesial to normal, wherein the mesiobuccal cusp of the maxillary first molar occludes in the embrasure between the mandibular first and second permanent molars. Class III may further be classified as a unilateral condition.

The origin of malocclusion is highly debated, again with multifactorial origins proposed. Besides the genetic aspect of malocclusion, in particular for Class III,[37] malocclusal patterns are felt to result from an imbalance between intrinsic forces such as from the tongue and lips and extrinsic forces such as stressful orofacial habits. A large number of studies have considered the influence of epigenetic factors such as the orofacial functions of deglutition, sucking, ventilation, mastication and phonation (see below). In fact, it is more the orofacial dysfunctions and parafunctions or orofacial habits that are the source of malocclusion. Edward Angle said: 'Orthodontic treatments are very unlikely to succeed, if the functional disorders are still going on.'[38]

Class II malocclusion represents a heterogeneous collection of conditions with malocclusion that may be the result of purely skeletal or combined skeletal and dental origin; they are, however, more often purely dental in nature.[39] The teeth and alveolar bone constitute an interface between opposing forces and pressures, primarily from muscular function – the perioral musculature of the lips and cheeks on the one hand and intraoral forces from the tongue on the other. Pressures are applied to the teeth with varying distribution according to the oral function involved.[40] Under appropriate circumstances these pressures should affect the teeth from multiple, yet

balanced, directions. When dysfunction results in chronically repeated patterns of activity, limiting the directions that these muscular pressures are applied to the teeth, dental malocclusion can be the result.

The development of Class II malocclusion has also been correlated with non-nutritive sucking habits such as thumb sucking.[41–45] In the early years of life the premaxillae may be easily pushed forward by non-nutritive sucking, pushing the upper incisors along with them. Thumb sucking creates an anterior opening between the dental arches, which facilitates the forward displacement of the tongue that occurs during tongue thrusting. It consequently results in dysfunctional forward placement of the tongue. This will, in turn, promote maxillary prognathism because, with every deglutition, which occurs approximately 1500 times per day, the tip of the tongue will move forward between the teeth, applying pressure to the premaxillae and upper incisors, pushing them forward.

For similar reasons, the sucking of a pacifier is also commonly associated with malocclusion, followed by the practice of sucking fingers.[46] Prolonged pacifier habits result in changes to the dental arches with a prevalence of posterior cross-bite and increased amount of overjet.[47] Until the age of 2–3 years the risk of developing a dysfunctional occlusion may be reduced proportionally to the reduction of time that the child uses the pacifier, and under these circumstances the dental arches should be regularly evaluated.[48] Significant maxillary prognathism has also been correlated with persistent digit sucking habits.[48,49] Among other sucking habits, a trend toward association of bottle feeding with the need for orthodontic treatment has been found.[50] On the other hand, breastfeeding seems to have a positive effect on orofacial morphogenesis,[51,52] with a protective effect for the development of posterior crossbite in deciduous dentition.[45]

Not only the mode of feeding but also the type of food is associated with malocclusion. There are suggestions that changes in diet and food processing are associated with variations in facial size and shape.[53] The prevalence of occlusal disorders in subjects born in the 1950s has been found to be lower than in individuals born in the 1990s. It is thought that this is probably because of dietary habits resulting from the increased consumption of processed foods.[54] At the same time, there is an increased prevalence of allergies that result in mouth breathing. There is a 3% incidence of mouth breathers in individuals born in the 1950s and 21% in those born in the 1990s.[54]

When mouth breathing is associated with nasal obstruction, it results in an inferior position of the mandible. In addition, the tongue is maintained lower in the oral cavity (Fig. 7.7.6). This posture is associated with compensations in the perioral musculature, such as hypotonicity, as well as compensations at the level of the vertebral spine. Mouth breathers tend to assume an extended or forward head posture.

Respiratory dysfunctions are also associated with malocclusion. Premature molar eruption is often present in chronic mouth breathers. Mandibular growth is affected, with resulting anterior mandibular rotation[29] and an increase of the mandibular angle.[30] Thus, the vertical dimension of the lower face is increased, with resultant open bite.[29] The term 'adenoidal facies' (long faces) describes infants with an open mouth, a short upper lip and prominent and crowded anterior teeth. Lip hypotonicity – decreased tonicity of the orbicularis oris muscle – is typically found in patients with Class II, Division 1 malocclusion.[55] In this presentation, hypotonicity of the orbicularis oris muscle is balanced by compensatory contraction in the mentalis muscles. It is this aspect that can be observed in patients with 'adenoidal facies' syndrome. In addition, the maxillae are narrow and the palate high arched with concomittant increase in the mandibular angle; thus the face appears longer.[56] These individuals also demonstrate a lack of development of the masseter muscles as well as of the maxillary bones.[57]

Alternatively, hypertonic and bulbous masseter muscles with reduced facial height are correlated with a tendency to clench the teeth.[57] These findings are commonly identified in association with cranial somatic dysfunction involving the temporal bones and/or TMJs. The cranial concept lends itself well to the understanding of functional orofacial disorders. Children who have dysfunctional extension of the SBS will present with a long narrow head. The resultant internal rotation of the maxillae is associated with a palate that is narrow and high arched. This orofacial cranial pattern fails to provide sufficient space for the teeth in the dental arches, predisposing the child to dental crowding. In young children, Class II malocclusion may be found in association with an intraosseous dysfunction between the premaxilla and the maxilla. The premaxilla can be pushed forward or backward as the result of stress patterns occurring during intrauterine life or at the time of delivery. Postpartum behaviors, such as thumb sucking, can also push the premaxilla forward, while a fall forward striking the face, particularly the

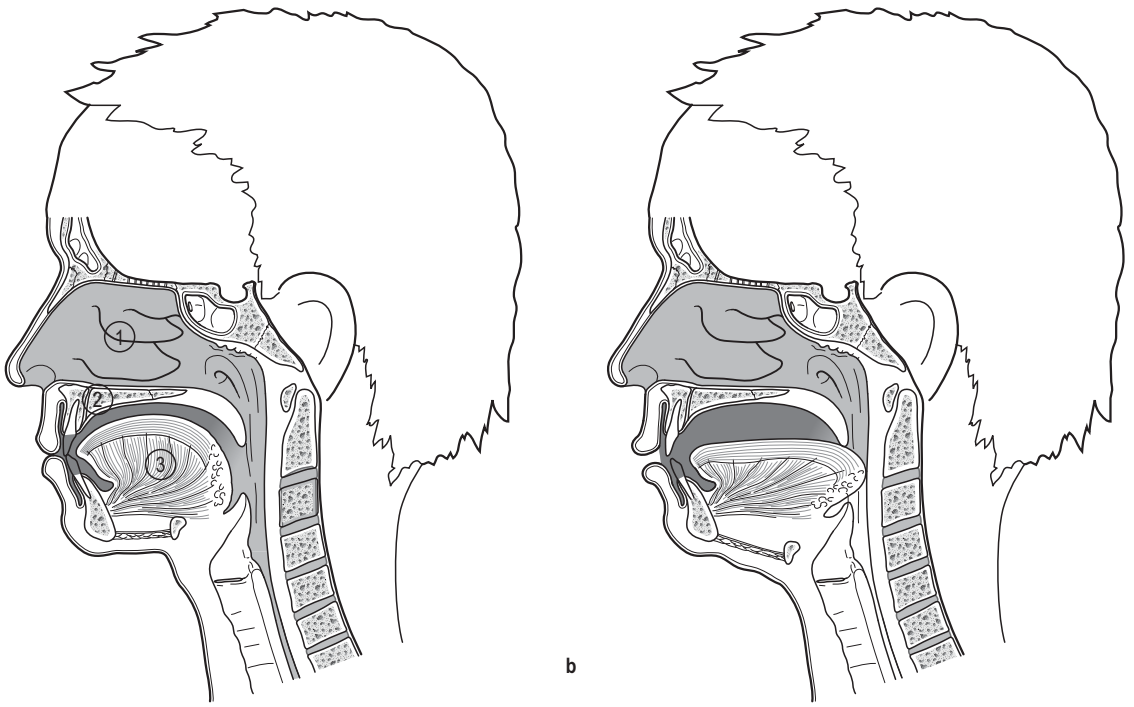

Figure 7.7.6. *The position of the tongue. (a) Functional position: note the lips are closed, the dorsum of the tongue is molding the palate, the oropharynx is patent. (b) Dysfunctional position: note the absence of labial contact, the displacement of the tongue posteroinferiorly and the decrease of the lumen of the oropharynx. 1. nasal cavity; 2. hard palate; 3. tongue.*

upper incisors, will push the premaxilla in a posterior direction.

Cranial maxillary dysfunction can be the beginning of a sequence of events that act to reinforce each other. Maxillary dysfunction can reduce nasal patency and predispose to mouth breathing. It can affect the dental arch with potential consequences for occlusion. Maxillary dysfunction can also trigger a compensatory pattern in the position and development of the mandible.

The mandible itself may demonstrate several types of dysfunction. Similar to maxillary dysfunction, mandibular dysfunction may result from strains or traumas. It can also occur as accommodation to internal or external rotation dysfunction of the temporal bones. Temporal bone dysfunction may be unilateral or bilateral, with the mandible moving anteriorly on the side of temporal internal rotation or posteriorly on the side of temporal external rotation. The resultant mandibular displacement will, in turn, affect occlusion.

In fact, any facial strain or trauma may impact the symmetry of the occlusal pattern. The area of the nasion – the junction between the frontonasal and internasal sutures – as well as the frontomaxillary sutures, may suffer compression from intrauterine pressures or as the result of a difficult delivery. The nasal bones should be attentively evaluated. With the maxillae, they are suspended from the frontal bone and as such will demonstrate restriction of movement following any trauma of the frontal bone. Furthermore, an impact on the frontal bone may result in a decrease in the vertical height of the maxillae with potential intraosseous dysfunction and resultant compromise of nasal respiration. Discrepancy in size between the maxilla and mandible may result from insufficient growth of these structures, potentially producing overjet or overbite.

Positioning of the child during the first months of life influences the growth pattern of the cranial bones. Non-synostotic plagiocephaly is associated with chronic sleep position and is most often visible in the first months of life.[58] It has also been shown that when a child sleeps in the same position for months, changes in the dental arches are observed.[59] Deformation of the face, however, because the growth of the viscerocranium continues later in life than the growth of the neurocranium, is not

commonly observable by the non-trained examiner before several years of age. Facial asymmetries are, however, frequently associated with plagiocephalies.[60–62] With asymmetric occipital deformation, compensatory changes may occur in the maxillae, with resultant mandibular asymmetry.[63]

The relationship between the neurocranium, cranial base and viscerocranium has been studied extensively. An increase in anterior facial height is observed in subjects maintaining a forward-bent head position; a decrease is associated with a backward-bent head posture.[64,65] Changes in the position of the hyoid bone are observed in Class II malocclusion where the hyoid bone is higher and more forward relative to the mandible.[66] The posture of the cervical spine changes in response to changes in the occlusal plane as well as to imbalance between right and left masticatory muscles.[35] Modifications of the cranial base are linked to Class III malocclusion where a decrease in the angle between the clivus (surface from the dorsum sellae of the body of the sphenoid to the foramen magnum) and the cribriform plate of the ethmoid has been observed.[13]

Physical examination and treatment

The child's occlusal pattern should be assessed. If abnormal occlusion or malocclusion is identified, the somatic dysfunction that may be responsible should be sought out and treated. The etiology of malocclusion is multifactorial and osteopathic procedures may be employed when cranial somatic dysfunction contributing to the establishment of the malocclusive pattern is present. To be effective, these procedures should be employed at the earliest possible age. Cranial manipulation is in no way intended to be a replacement for standard orthodontic treatment. However, orthodontic treatments are less likely to be fully successful if somatic dysfunction affecting the child's occlusal pattern persists.

When examining the child with malocclusion, the principles of examination discussed above in 'Oral cavity and orofacial functions' apply. In addition, when looking for contributory somatic dysfunction, the practitioner should pay attention to the standing postural mechanics and their impact, particularly on the upper thoracic and occipitoatlantal areas. Even in cases where orthodontic treatment has been initiated, the child's posture should be addressed. The identification and treatment of dysfunctional postural mechanics resulting in functional balance of the orofacial complex will accelerate the effect of the orthodontic treatment. Furthermore, orthodontic treatment in itself serves as a source of stress, affecting the overall mechanics of the body. Thus, the release of existing dysfunctional mechanics will provide comfort to the child, making the orthodontic process more tolerable, as well as facilitating the effect of the orthodontic procedure.

With the child seated, it is appropriate to observe the occlusal pattern. Have the child actively open their mouth while observing for displacement of the symphysis menti. This motion normally should follow a straight vertical path, without a distorted pattern. Next, have the child move their mandible laterally to the right and left sides to compare ease of movement and degree of displacement bilaterally. If appropriate, give the child some chewing gum to observe the presence or absence of an alternating masticatory pattern. Observe and note the presence of a dominant side to the masticatory pattern. Check the presence of immature swallowing by observing and asking the child to describe the location of the tip of their tongue within their mouth during swallowing. Assess the musculature of the lips and cheeks by placing your thumbs inside the child's mouth, such that their palmar surfaces are in contact with the inside of the cheeks. Ask the child to close their mouth and swallow, and with your thumbs appreciate the strength of contraction of the buccinator muscles.

Next, with the child lying supine, palpate the clavicles, sternum and hyoid bone, assessing their myofascial attachments. Go on to assess the myofascial structures of the upper thoracic and cervical spine for somatic dysfunction, noting the relationship between the occiput and atlas. Palpate the myofascial structures of the skull, paying attention to the orofacial muscles and TMJ area for dysfunctional asymmetric tension. Check the TMJs and proceed with tests of listening. Evaluate the cranial base, looking for patterns of predominant cranial flexion or extension, as well as torsion, sidebending–rotation, compression and strain patterns. By placing one hand on the frontal bone with the index and middle fingers controlling the greater wings of the sphenoid and the other hand cradling the mandible, note the balance between the mandible and greater wings of the sphenoid. Listen to the different bones of the skull that may be involved in the malocclusion, paying particular attention to the temporal bones, sphenoid, frontal bones and maxillae, looking for intra- and interosseous cranial somatic dysfunctions. Treat all identified somatic dysfunction using indirect principles. Intraosseous molding may be considered if the child is young enough.

All of these relationships are often reciprocal and illustrate the integration of all parts of the body, local and distant, into a united system. Osteopathic treatment may be employed to address the somatic dysfunction that is found in association with orofacial dysfunction. When indirect principles of treatment are respectfully employed, these procedures may be used to treat from the very youngest patient on through adolescence. The earlier the dysfunction is recognized, the younger the patient and the more plastic the tissues, the greater the possibility to affect the structure and to re-establish satisfactory functional patterns, thereby activating the body's inherent ability to heal itself.

Patient education and advice to caregivers

Involve the parents or caregivers; empower them to help the child to succeed in dealing with the dysfunction. Take the time to explain to the child why it is necessary to breathe through the nose and eat slowly. Encourage the child to promote correct function, such as nasal breathing and learning how to blow their nose. Encourage the child to practice activities that promote breathing, such as singing with good articulation of the words.

Young children with dysfunctional tongue mechanics or with minor cases of ankyloglossia can perform exercises to mobilize the tongue and to stretch the frenulum as much as possible. Encourage the parents or caregivers to correct the child when there is mispronunciation. It may appear to be cute, but is actually dysfunctional. The child should repeat incorrectly pronounced words as correctly as possible to improve tongue mobility. Infants can be encouraged to play sound-making games by repeating sounds that cause the tongue to make a clicking noise against the palate.

Teach efficient swallowing without tongue thrusting by instructing the child to consciously keep their tongue in their mouth and to learn to swallow with the tip of their tongue resting against the palate behind their upper incisors. To practice this, the child can hold a small piece of food between the tongue and palate while attempting to swallow several times. This exercise should be repeated until the act of swallowing with the tip of the tongue resting against the palate becomes automatic.

Insist on alternate unilateral mastication and that the child takes sufficient time to chew their food thoroughly. Those children who are old enough should be encouraged to eat food that is as unrefined as possible. Soft refined junk food often does not require sufficient chewing.

Caution the parent or caregiver to watch for functional asymmetries that are associated with repetitive asymmetrical activities, such as unilateral bottle feeding or thumb sucking. Instruct the parent or caregiver and the patient, when old enough to understand, to avoid these activities or perform them in a fashion that alternates sides.

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