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Physical Structure and Function and Speech Production Associated with Cleft Palate

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1.1 Introduction

Speakers with a cleft lip and/or palate contend with unusual structure and function of the vocal organs from birth and physical abnormalities may persist after surgical intervention. (Surgery itself, for many individuals with a cleft, consists of a series of interventions over an extended period, so both structural and functional changes to the speech apparatus may be a feature of the entire period of speech development). These differences and changes may have a profound effect on speech production and speech development, and cleft lip and palate is one area where a significant proportion of the speech difficulties encountered (although not necessarily all) can be traced back in some way to an anatomical or physiological cause. This chapter explores some of the links between atypical vocal organ structure and function in cleft lip and palate, and those many and varied features encountered in speech production associated with cleft palate. Of course, some of these issues are also dealt with in other chapters in this book (Chapters 3, 5, 8, 10, 11 and 12), so the reader is directed, where appropriate, to seek further information from these chapters; this chapter, therefore, focuses on those issues not discussed.
elsewhere in the book. More detailed accounts of the physical structures and functions associated with speech production can be found in Atkinson and White (1992) and Atkinson and McHanwell (2002).

1.2 The Hard and Soft Palates and the Velopharynx

1.2.1 Anatomy of the Hard and Soft Plate

The palate comprises the rigid bony hard palate anteriorly and the mobile muscular soft plate (velum) posteriorly. The shape of the hard plate is variable but is usually a concave dome. However it may take on a V-shape with the apex superiorly, which narrows the hard palate. This configuration of the hard palate often accompanies a class II malocclusion (Section 1.5.1); as the upper dental arch is narrowed the posterior teeth cannot align along a curved dental arch but follow the V-shape, pushing the anterior teeth forward. The bony plate is formed from components of two pairs of bones; the palatine plates of the maxilla form the anterior two thirds and the horizontal plates of the palatine bones form the remainder. The bones are joined at sutures. A midline suture marks the line of fusion of the two halves of the palate during palatogenesis and terminates anteriorly at the incisive foramen, another landmark relating to the development of the palate. The sutures are, of course, covered in life by the mucosa lining the mouth. However, the site of the incisive foramen is marked by a small incisive papilla visible just behind the central incisor teeth.

The soft palate extends from the posterior border of the hard palate. Four pairs of muscles form the soft palate (Figure 1.1). The tensor veli palatini tenses the velum by exerting a lateral force; these muscles are tendinous within the soft palate and the other muscles are attached to the tendons. The levator veli palatini raises the soft palate. Note that the tensor and levator palatini attach to the Eustachian tube and open it when the velum is raised or tensed, so that fluid drains from the middle ear cavity and air pressure is equalised on the either side of the eardrum. These two muscles are often inefficient in the early stages of cleft palate repair so that the Eustachian tube does not open. Drainage of the middle ear is therefore poor, accounting for the high incidence of ‘glue ear’ in cleft clients. The palatoglossus and palatopharyngeus muscles depress the velum. The soft palate has a backward extension, the uvula which is very variable in shape and size.

1.2.2 Embryology of Palate

In the early embryo, the oral cavity is a slit between the frontonasal process that overlies the developing brain and the first pharyngeal arch. The first arch forms the mandible and associated structures but also the maxilla, including a large component of the palate. The palate develops between the fourth and twelfth week of pregnancy to separate the nasal and oral cavities. It develops from three components that change shape and position from their original location during subsequent growth and development and must fuse together to form the palate. A small triangular component, the primary palate, develops from the frontonasal process as the nasal cavities develop around the fifth week.
The primary palate forms the area behind the four upper incisor teeth only as far back as the incisive foramen. At six weeks, two palatine processes grow in from either side of the first arch. Logically they would be expected to grow horizontally but they actually grow downwards. The reason for this apparent peculiarity is that the tongue develops very early and fills the developing oral cavity, thus deflecting the palatine processes downwards. Around eight weeks, the mandible widens out and the tongue drops into its conventional position, thus no longer impeding the palatine processes. The palatine processes dramatically ‘flip up’ into a horizontal position. This change of orientation, palatal elevation, is not simply a consequence of tongue displacement but depends on the build up of hydrophilic (water binding) chemicals that make the processes turgid. At this stage the three processes are separated by quite wide gaps but over the next two weeks the processes grow and converge. Where they make contact, a chain of reactions is triggered within the epithelial cells covering the processes that kill the cells; this process is known as programmed cell death or apoptosis. The death of the epithelial covering allows the underlying tissues to fuse to complete the palate by twelve weeks post-fertilization. The complete palate is invaded by bone anteriorly to form the hard palate and by muscle posteriorly to form the velum; this process is usually complete by about fifteen weeks (Figure 1.2).

Figure 1.1 The muscles of the soft palate viewed from behind looking into the posterior nasal aperture. (Reproduced with permission from Atkinson & McHanwell, 2002.)

From this brief outline of palatogenesis, it is clear that there are several stages where the processes may be disrupted. Essentially, the requisite building blocks may not develop
or may not grow sufficiently; the palatine processes may not elevate if the specific signals to build up the hydrophilic molecules are not given; the processes may not fuse if molecular signals do not trigger apoptosis or if there is any obstruction present. A palatal cleft may manifest anywhere along the Y-shaped lines of fusion between the primary palate and palatine processes (the arms of the Y) and the two palatine processes (the stem of the Y). It can vary from a cleft uvula to a complete bilateral cleft running along the whole extent of the Y and extending into the upper lip.

Figure 1.2 The development of the palate between 6 and 12 weeks post-fertilization. (a), (c) and (e) are sections taken through from the top of the head to the mandibular arch. (a) and (b) represent palate formation at about 6–7 weeks, (c) and (d) at 8 weeks as the palate elevates and (e) and (f) at 12 weeks when palatal fusion is complete. (b), (d) and (f) show the sequence of events viewed from the oral aspect of the developing palate. (Reproduced with permission from Atkinson & McHanwell, 2002.)
1.2.3 Velopharyngeal Structure and Function in Relation to Speech Production

Sell and Pereira (Chapter 8) and Sweeney (Chapter 11) provide detailed accounts of the effects of velopharyngeal (VP) problems on speech and on their assessment. Here only a brief account of the main speech production difficulties linked to VP difficulties is given. Because all known spoken languages contain both oral and nasal (and in some cases nasalized) sound segments, the ability to valve air appropriately through the oral and/or nasal cavities in close coordination with phonatory and articulatory activity is a vital component of successful speech production. Where inadequate structure or function of the soft palate and velopharyngeal port do not permit this, as is the case for a speaker with a cleft palate, speech problems are likely to emerge. Interestingly, speech production problems associated with VP insufficiency do not necessarily disappear following surgery and VP function may remain atypical into adulthood (Moon et al., 2007; Mani et al., 2010). Not only range of movement and the ultimate ability to create an adequate seal at the VP port, but also speed and timing of VP movements will affect airflow and resonance (Dotevall, Ejnell and Baker, 2001; Warren, Dalston and Mayo, 1993). Although Kuehn and Moller (2000, p. 351) note that ‘excessive nasality or hypernasality is probably the signature characteristic of persons with cleft palate’, Peterson-Falzone et al. (2005) state that difficulties achieving velopharyngeal closure can affect not only resonance, but also articulation and phonation, thus providing a reminder of the pervasive consequences of VP difficulties for speech production. Each of the five universal speech parameters proposed by Henningsson et al. (2008) for reporting on the speech of individuals with a cleft palate (hypernasality; hyponasality; audible nasal emission and/or nasal turbulence; consonant production errors; voice disorder) may be traced in some way or another to VP insufficiency.

1.3 The Tonsils and Adenoids

Because speakers with a cleft palate are particularly vulnerable to resonance problems, those structures which may impede velopharyngeal closure are of particular significance for these individuals. The tonsils and adenoids are two such structures, comprising aggregates of lymphoid tissue lying just under the mucosal lining of the pharynx. Lymphoid tissue is involved in defence mechanisms designed to fight bacterial and viral infections, acting as a first line of defence against pathogens entering through the nose or mouth. The paired tonsils (properly termed the palatine tonsils) lie just behind the palatoglossal arch (the anterior pillar of the fauces) that demarcates the junction between the oral cavity and pharynx, and immediately below the lateral attachments of the velum to the tongue and pharynx (Figure 1.3). The adenoids (the pharyngeal tonsils) lie on the posterior wall of the pharynx, behind the nasal cavities, at or just above the point at which the velum makes contact with the pharyngeal wall during elevation and closure.

Although the tonsils do not generally have any effect on articulation, resonance or voice, they may enlarge considerably if they become infected. This, in turn, may cause hypernasality, by obstructing velopharyngeal closure, and has also been linked to the fronting of target velar consonants, by restricting space in the rear of the oral cavity.
Where a tonsillectomy is performed, significant improvements in speech and voice usually follow (Mora et al., 2009), without any great risk of velopharyngeal inadequacy (Peterson-Falzone, Hardin-Jones and Karnell, 2010). Compared with the tonsils, the effect of the adenoids on speech production is less clear-cut, due to the fact that for all speakers the adenoids change over time, both in size and in location relative to the other vocal organs. They grow very rapidly after birth to reach their maximum size at about five to six years of age, thereafter decreasing, and they shift from a vertical to a horizontal orientation. Peterson-Falzone, Hardin-Jones and Karnell (2010) provide a reminder that the adenoids are crucial for velopharyngeal (VP) closure in young children, and Maryn et al. (2004) suggest that this is so significant that ‘veloadenoidal closure’ should be added as a fifth category to the different types of VP closure proposed by Skolnick et al. (1975). As developmental structural changes take place very gradually, children usually accommodate to them without problems and there is no effect on speech production. However, for children with a submucous cleft or borderline VP inadequacy, the presence of the adenoidal pad may have been critical to achieving adequate VP closure and in these children the normal decrease in size may result in resonance problems. Conversely, enlarged adenoids may cause hyponasality and open mouth breathing, and in some cases therefore surgery may be indicated. However, the sudden structural changes brought about by an adenoidectomy may then cause hypernasality, as the child fails to adjust to the increased velopharyngeal port space (Witzel et al., 1986).
1.4 The Larynx

The larynx plays a key role in speech production, acting as it does as an articulator (for sounds like \([h]\) and \([ʔ]\)), as an airstream initiator (in the production of ejectives and implosives), and as the source of phonation, both at the segmental level, for voiced-voiceless segmental contrasts, and at the level of voice quality and overall vocal settings (Laver, 1994). For normal voicing, the tensed vocal folds vibrate in the egressive airstream. The vocal folds are attached anteriorly very close to the midline of the inner aspect of the thyroid cartilage and posteriorly to the vocal processes of the widely spaced arytenoids cartilages thus forming a V-shaped glottis with the point of the V anteriorly (Figure 1.4). For phonation, the vocal folds must be approximated to build up subglottal pressure and tensed. Approximation (adduction) is achieved by sliding the arytenoid cartilages together on the upper rim of the cricoid cartilage. Tension is created by tilting the anterior part of the cricoid upwards towards the thyroid cartilage (‘closing

![Figure 1.4](image)

**Figure 1.4** A schematic diagram of the larynx viewed from the posterior aspect. The larynx has been opened to present a clearer view of the positions of the vocal and vestibular folds. Note also the position of the aryepiglottic folds forming the lateral borders of the laryngeal entrance. (Reproduced with permission from Atkinson & McHanwell, 2002.)
the visor’). The cricoid pivots about two thirds of the way back so that the posterior lamina bearing the arytenoids is carried backwards and downwards, thus extending the distance between the anterior and posterior attachments of the vocal folds; the folds are lengthened, tensed and thinned. Tilting the arytenoids forward on the cricoid cartilage has the opposite effects. Because the backward and forward movements are produced by different sets of muscles, they can be varied independently to produce changes in pitch and loudness.

The vestibular folds lie about 2 mm above the vocal folds but are attached more laterally on the arytenoids, so are not normally in the direct egressive air stream. They also lack direct muscle control and therefore cannot be adjusted like the vocal folds. Forced over-contraction of muscles that adduct the vocal folds can tilt the apices of the arytenoids inwards to approximate the vestibular folds and bring them into the air stream. Adduction of the apices of the arytenoid cartilages will also approximate the aryepiglottic folds. The aryepiglottic folds pass from the lateral rim of the epiglottis to the apices of the arytenoids and contribute to the oval profile of the laryngeal entrance. The epiglottis is made of elastic cartilage and moves passively. The flimsy aryepiglottic muscles in the folds cannot overcome the elasticity of the epiglottis but alter the outline of the laryngeal entrance from an oval to a slit during swallowing to minimize the risk of aspiration of foodstuffs. Coupled with extreme adduction of the cartilage apices, these folds could possibly approximate sufficiently to allow some degree of phonation.

Peterson-Falzone, Hardin-Jones and Karnell (2010, p. 240) comment, ‘It is clear ... that phonation disorders are more common in this population than in individuals without clefts’, and Cavalli (Chapter 10) provides an extensive discussion of the voice problems associated with a cleft palate, illustrating how this is largely due to muscle tension disorders, possibly as a compensation for velopharyngeal problems (Chapter 10). There is evidence that laryngeal structures not usually involved in phonation may be recruited in such circumstances (Kawano et al., 1997). In the review of the literature provided by Van Lierde et al. (2004) the following voice problems were noted as having been found in the speech production of individuals with cleft palate: breathiness, hoarseness, harshness, aphonia, pitch problems, reduced intensity (‘soft voice syndrome’) and limited vocal range. As with many speech production features associated with cleft palate, many of these can be seen as stemming from compensatory behaviours aimed at reducing or disguising hypernasality and audible nasal emission. Where vocal fold vibration is associated with the production of specific sound segments (to make distinctions between voiced and voiceless sounds) this does not appear to be a particular problem for speakers with a cleft palate.

1.5 The Jaws, Dentition and Occlusion

In discussing the effect that variations in oral and dental structure might have on speech production in the cleft population, it is important to note that wide variation is also a feature of speakers without a cleft (Beck, 2010). Within the cleft population, it is also the case that many individuals develop and produce speech normally (Peterson-Falzone, Hardin-Jones and Karnell, 2010). These two observations support the long-documented assertion that speakers are able to compensate successfully for variation in the speech apparatus and that unusual oral structure does not inevitably imply unusual speech
production (Fairbanks and Lintner, 1951; Nishikubo et al., 2009). Nevertheless, there is also a significant, if equivocal, literature linking some of the misarticulations produced by speakers with cleft lip and palate to atypical palatal morphology, occlusion and dentition: at issue here appears to be the degree to which speakers adapt to unusual structure and function.

1.5.1 The Jaws

The anatomical and dynamic relationships between the mandible and maxilla and the upper and lower teeth are important to ensure correct function for both speech and non-speech activities. In typical speech development, jaw movements appear to stabilize during speech production at an earlier point than either labial or lingual movements (Green, Moore and Reilly, 2002), and this stability is critical for speech production. As Cheng et al. (2007, p. 353) suggest ‘the jaw provides the foundation necessary for the acquisition of more specialized motor skills’, including those of the lips and tongue. Coordination of mandibular and labial movements typically appears to emerge earlier than jaw–tongue coordination and although these are both broadly achieved in infancy, refinement of control and coordination continues throughout early childhood, and perhaps even into adolescence (Green et al., 2002; Smith, 2006). Coordination between the mandible and the tongue is particularly important for tongue-tip sounds, which, of course, have frequently been noted as being especially vulnerable in the speech production of individuals with a cleft palate.

Spatial relationships between the jaws and teeth are also important and are often compromised in individuals with a cleft palate. In a class I dental occlusion, the maxillary arch supporting the upper teeth is wider than the corresponding mandibular arch (Figure 1.5a). The maxilla is positioned such that the upper anterior teeth protrude beyond the lower teeth by about 2 mm when the teeth are brought into occlusion. They also overlap the lower teeth by a similar amount in a vertical dimension. A class II occlusion occurs if the maxilla is too far forward with respect to the mandible: note that this misalignment may be due either to an oversized maxilla or to a reduced mandible (Figure 1.5b). When the converse applies and the mandible is anterior to the maxilla, a class III occlusion is produced in which the lower anterior teeth protrude beyond the upper anterior teeth: this is the most frequently occurring malocclusion in the cleft population (Figure 1.5c).

Dental malocclusions arise when the teeth themselves are malpositioned within dental arches that show a normal relationship. For example, prolonged thumbsucking may displace the upper anterior teeth anteriorly, producing a class II occlusion and/or an anterior open bite. In cleft palate, the teeth are often severely malpositioned as the fusion lines described above pass between the position of upper lateral incisors and canines. The upper dental arch is often narrow, producing a high vaulted palate as the dental arches tend to collapse inward. In many syndromic presentations of cleft palate, there are associated developmental anomalies in the size and position of the maxilla and mandible producing skeletal malocclusions. Repositioning of the teeth and/or jaws can be achieved by orthodontic intervention and/or maxillofacial surgery (Chapter 5).

Johnson and Sandy (1999) suggest that the presence, absence or malpositioning of individual teeth do not appear to have a significant effect on speech sound articulation,
probably because of individual speakers’ abilities to compensate for minor structural variation, although Chait et al. (2002) suggest that absent lateral teeth may result in misarticulated sibilant fricative production. A factor which is, however, implicated in sound segment misarticulation is anterior open bite (AOB). Bernstein (1954) was one of the first to note an association between AOB and misarticulation in children’s speech, and a specific link to misarticulated fricative and affricates, together with a generally advanced lingual setting for speech has been identified using electropalatography by Suzuki et al. (1981), Hiki and Itoh (1986) and Cayley et al. (2000).
Malocclusion, however, has also frequently been associated with misarticulation. In Class II and III occlusal relationships, where the maxilla and mandible fall into atypical alignment, the active and passive articulators will consequently also be misaligned. Thus, for example, in a Class II occlusion the bottom lip may fall into a closer vertical relationship with the upper incisors, or even the alveolar ridge, than it does with the upper lip; conversely in the case of a Class III occlusion, the top lip may naturally oppose the lower incisors rather than the bottom lip. Similar misalignments between the tongue tip and blade and the upper teeth and alveolar ridge will also exist. Although it is clear that such minor differences could be compensated for by quite small articulatory adjustments, where the speaker does not actively compensate, atypical articulatory strictures are likely to occur. Some of the unusual places of articulation included in the Extensions to the IPA (Duckworth et al., 1990), including reverse labiodental, labioalveolar, linguolabial, reflect these articulatory patterns in some speakers with cleft lip and palate (Chapter 7). Although Hassan, Naini and Gill (2007, p. 2543) caution that ‘there is no clear evidence directly relating malocclusions to speech discrepancies’, a series of studies nevertheless point to the potential vulnerability of dental, alveolar and postalveolar segments (particularly the sibilant fricatives) in both Class II and Class III occlusion (Laine, Jaroma and Linnasalo, 1987; Giannini et al., 1995; Laitinen, Ranta and Haapenen, 1999). Where the alignment of maxilla and mandible is corrected by orthognathic surgery some of these misarticulations have been shown by both perceptual and instrumental studies to improve spontaneously and quite rapidly (Vallino, 1990; Wakumoto et al., 1996) although for some speakers these changes may not be maintained a year after surgery (Lee et al., 2002).

Nishikubo et al. (2009) question the link between malocclusion and misarticulation, suggesting, on the basis of 3-D palatal imaging, that a more significant predictor of misarticulation in cleft speakers may be the general size and shape of the oral cavity (Chapter 3). Once again, a number of studies seem to support this view, linking relative length and/or shape of palate to various misarticulations of alveolar segments (Hiki and Itoh, 1986; Laine, Jaroma and Linnasalo, 1987; Okazaki, Kato and Onizuka, 1991). It may, however, be not so much structural as sensory anomalies which impact on articulation in speech production associated with cleft palate. Hardcastle (1975) demonstrated changes in lingualpalatal contact patterns for sibilant fricatives in typical speakers where lingual sensation was decreased by application of anaesthesia and, more recently, Premkumar, Venkatesan and Rangachari (2010) have related the misarticulations associated with anterior open bite and tongue-thrusting with an overall reduction in intra-oral sensation. It might also be suspected that any reduction in sensation consequent on post-surgical scarring in the region of the alveolar ridge and hard palate might also affect articulatory patterns in speakers with a repaired cleft palate.

1.6 Symmetry: Structure and Function

Studies have identified significant structural asymmetries in the face (Bugaighis et al., 2010) and the palate and oral cavity (Kilpeläinen and Laine-Avala, 1996) (although not the mandible; Kurt et al., 2010) in individuals with a cleft palate, and structural asymmetry has, in turn, been linked to misarticulation (Nishikubo et al., 2009). At the same time, functional asymmetries of labial and lingual articulation have been noted in cleft
speakers and speakers with other speech impairments (Howard, 1994; Gibbon, 2004; Cheng et al., 2007). Nevertheless, it is important to note that there is no simple relationship between structural asymmetry and speech production. Minor asymmetries in lingual behaviour are common in typical speech production (Marchal et al., 1988), but it may be the extent of asymmetry which is significant in the perceptual impression of misarticulation (Howard, Clark and Whiteside, 1994).

1.7 The Tongue

The tongue could be described as a bag of muscles, and lingual function, dependent on these muscles, is notoriously challenging to explain (Atkinson and McHanwell, 2002). It is usually straightforward to predict the action of a given muscle, by looking at the bony points to which the two ends of the muscle are attached and anticipating what action contraction of the muscle will have on any joint that the muscle passes over. This is not the case with the tongue: one of the two sets of lingual muscles, the extrinsic muscles, only has one bony attachment, with the other end anchored to other muscles in the tongue. The other set, the intrinsic muscles, has both attachments within the tongue itself (Figure 1.6).

Anatomical textbooks usually suggest that the extrinsic muscles alter the position of the tongue, drawing it towards the anchorage of the extrinsic muscle in question on the mandible or skull, and the intrinsic muscles alter the shape of the tongue. A moment’s thought tells us that this is a vast, if convenient, oversimplification. Theoretically, it may be possible to alter tongue position without a change in shape, but it is impossible to alter shape without altering the position of the tongue relative to other articulators. The tongue is, therefore, the best example of the axiom that muscles act as groups rather than as individual entities. This emanates from the processing and control of motor activity in the brain. The brain thinks in terms of overall patterns of activity and sequencing of muscle contractions to produce a given complex movement; the rate, range and force of contraction of the individual muscles required to produce the movement is then superimposed on the overall patterning and movement sequence. When students studying anatomical specimens of the tongue encounter it for the first time, they are usually very surprised by its large size; it is much bigger than they have predicted. The oral, and most mobile, part of the tongue accounts for only about half of the organ; the posterior pharyngeal part is the other ‘unseen’ half.

Surprisingly, the tongue is usually unaffected by developmental disturbances affecting the face, palate and jaws, even the most severe, as its embryological origins are quite different (Atkinson and McHanwell, 2002) and, consequently, lingual structure and function is generally considered to be unaffected by a cleft palate (Peterson-Falzone, Hardin-Jones and Karnell, 2010). For a small number of individuals with a specific genetic abnormality, however, a cleft palate may be associated with ankyloglossia (tongue tie) (Braybrook et al., 2002), thus potentially affecting the successful elevation of the tongue-tip for alveolar and dental sounds, although how much ankyloglossia affects articulation remains unclear (Suter and Bornstein, 2009). In general, though, the atypical lingualpalatal contact patterns and overall lingual settings reported in the speech of individuals with cleft palate are deemed to be adaptive behaviours which may be linked to velopharyngeal inadequacy and its primary effects on speech. Thus, electropalatog-
The muscles of the tongue. (a) shows the extrinsic muscles and their attachments. (b) shows two views of the intrinsic muscles and their relationship to the extrinsic muscles as seen in a longitudinal section (A) and a cross section of the tongue (B). (Reproduced with permission from Atkinson & McHanwell, 2002.)

Glossography has revealed a range of unusual lingual-palatal contact patterns in speakers with cleft palate, including retracted articulation of alveolar and postalveolar consonants, lateralization, palatalization, broad and undifferentiated patterns with overuse of the tongue dorsum, and double articulations, all of which may stem to some extent from articulatory strategies for the prevention or masking of hypernasality and nasal emission (Gibbon, 2004; Howard, 2004; Chapter 12). The retracted patterns of articulation and the tendency to make greater than normal use of the tongue dorsum in segmental
articulation seem also to be linked to a frequently observed habitual lingual setting in cleft speakers, whereby the body of the tongue is generally held in an elevated and somewhat retracted position throughout speech production, a feature which it has been argued also aids in elevating the velum and thus contributing to the overall mechanism of velopharyngeal closure (Lawrence and Philips, 1975).

1.8 The Lips

The lips are formed by muscles covered on the outside by non-hairy skin and on the oral side by oral mucosa. A circular muscle, the orbicularis oris, surrounds the oral cavity and is the basis of the structure of the lips. Although anatomically this muscle appears to be a single entity, in reality it is a very complex structure formed by eight semi-independent parts, two in each quadrant. It is these subdivisions that enable the exquisitely precise labial movements in speech in particular, but also in other oral activities. Orbicularis oris is one of a group of subcutaneous muscles known collectively as the muscles of facial expression. Other members of this group exert traction on the upper lip and lower lip, working in concert with orbicularis oris to alter the shape and position of the lips during articulation.

Studies of motor activity in speakers with a repaired cleft lip suggest that there are some significant functional differences from typical non-cleft speakers. For the production of bilabial segments, where the upper and lower lips need to move together to form a stricture of complete closure, differences are reported for both lips. Electromagnetic articulography (EMA) studies of lip movements during speech production suggest that there is more variability in movement patterns in speakers with a cleft lip, and that this variability increases with increases in the linguistic complexity of the utterances examined (Rutjens, Spauwen and van Lieshout, 2001). Van Lieshout, Rutjens and Spauwen (2002) also identified patterns of reduced upper lip movement in speakers with a repaired cleft lip, which complements Trotman, Barlow and Faraway’s discussion (2007) of compensatory activity found for the lower lip in bilabial closure tasks. In other words, where lip surgery has taken place, the motor effects are not confined to the site of surgery but spread out over the whole labial system. A further finding which may have significance for the development of speech patterns in speakers with a cleft lip is that of reduced sensation in the upper lip (Essick et al., 2005), once again not only confined to the side with the cleft.

A short and/or tight upper lip, consequent on lip surgery, may affect lip closure for bilabial sounds and also the lip-rounding necessary for vowels such as [u] and [u] (Peterson-Falzone et al., 2005). Interestingly, even where normal labial activity for bilabial sounds is achieved, some speakers with a cleft lip and palate exhibit other unusual articulatory activity during the production of bilabials, in the form of complete lingual-palatal closure patterns concomitant with the lip closure (Gibbon and Crampin, 2002), a pattern which is not found in typical speech production (Gibbon, Lee and Yuen, 2007) and which may, therefore, be a further compensatory pattern associated with the speech production of individuals with a cleft palate.

Although it is generally supposed that speakers with an isolated cleft lip are less likely to have speech output problems than those with a cleft lip and palate or an isolated cleft palate, it cannot be assumed that they will not have any difficulties. A study by Vallino,
Zuker and Napoli (2008) suggests that both articulation and language problems occur at significantly higher rates in individuals with an isolated cleft lip than in the non-cleft population, although they also note, perhaps unsurprisingly, that resonance is generally not a problem.

1.9 Summary: Compensations Across Systems

Although information has been presented here on different articulators and vocal organs and their effect on speech production associated with a cleft palate in a sequence of separate sections, the point cannot be overstated that in speech production no organ or system ever functions in isolation. This is amply illustrated in the literature, where reports of the interrelationship between different subsystems, and of compensations within and across subsystems, serve as a reminder of the complexity of speech production associated with cleft and underscore the heterogeneity of the population of individuals with a cleft lip and/or palate. Different speakers find different solutions to the challenge of producing speech sounds in order to interact with others. It is hoped that this account gives a flavour of the varied features encountered in the speech of individuals with a cleft palate and of some of the reasons for these features, but there will always be a new speaker to be encountered who will offer a slightly different solution to the challenge of being intelligible in the face of structural and functional difficulties.

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