1

Physical Structure and Function and Speech Production Associated with Cleft Palate

Martin Atkinson[1] and Sara Howard[2]

[1]University of Sheffield, School of Clinical Dentistry, Sheffield, S10 2TA, UK
[2]University of Sheffield, Department of Human Communication Sciences, Sheffield, S10 2TA, UK

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

Cleft Palate Speech: Assessment and Intervention, First Edition. Edited by Sara Howard, Anette Lohmander.

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 (Section1.5.1); 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 palatophayngeus 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.

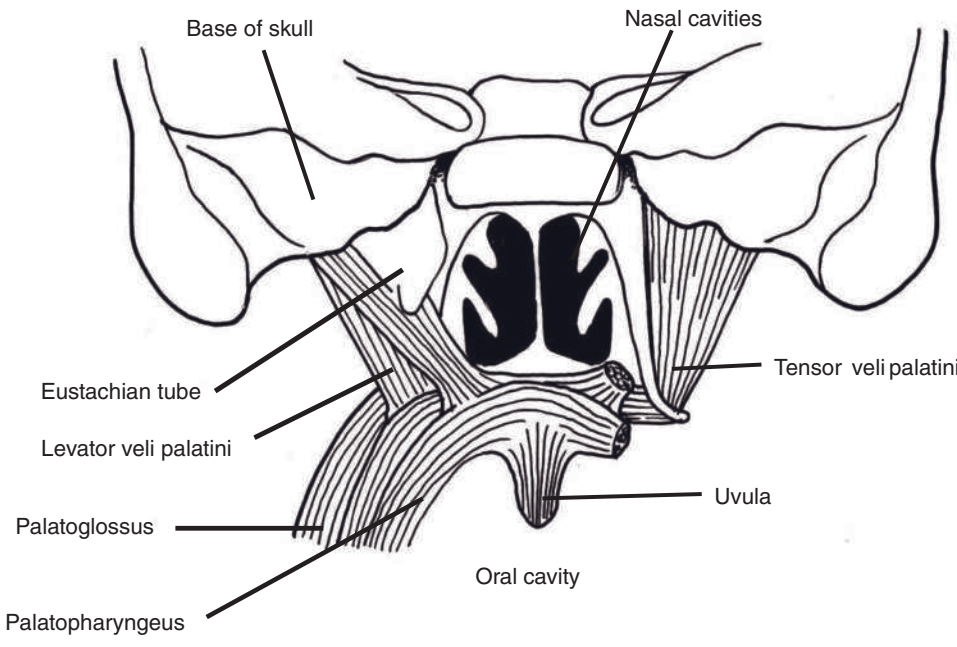

Figure 1.1 The muscles of the soft palate viewed from behind looking into the posterior nasal aperture. (Reproduced with permission from Atkinson & McHanwell, 2002.)

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).

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

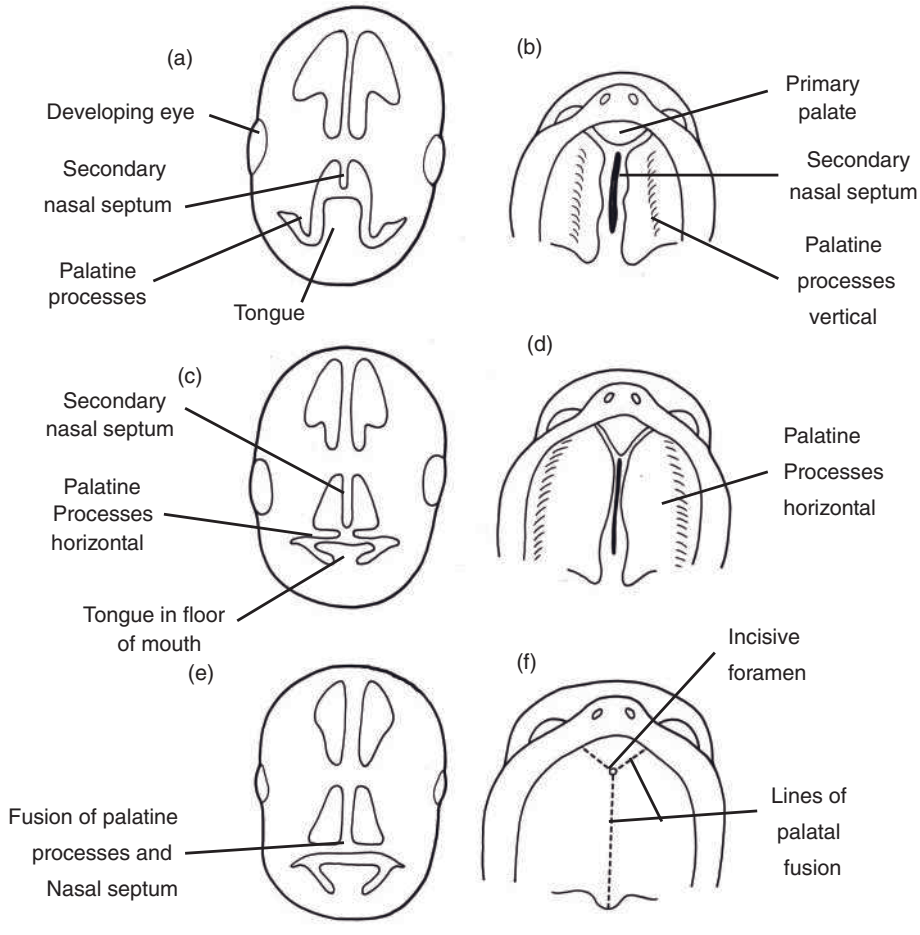

Figure 1.2 The development of the palate between 6 and 12 weeks post-fertilization. (a), (c) and (e) are sections taken through from the top of the head to the mandibular arch. (a) and (b) represent palate formation at about 6–7 weeks, (c) and (d) at 8 weeks as the palate elevates and (e) and (f) at 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.)

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.

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

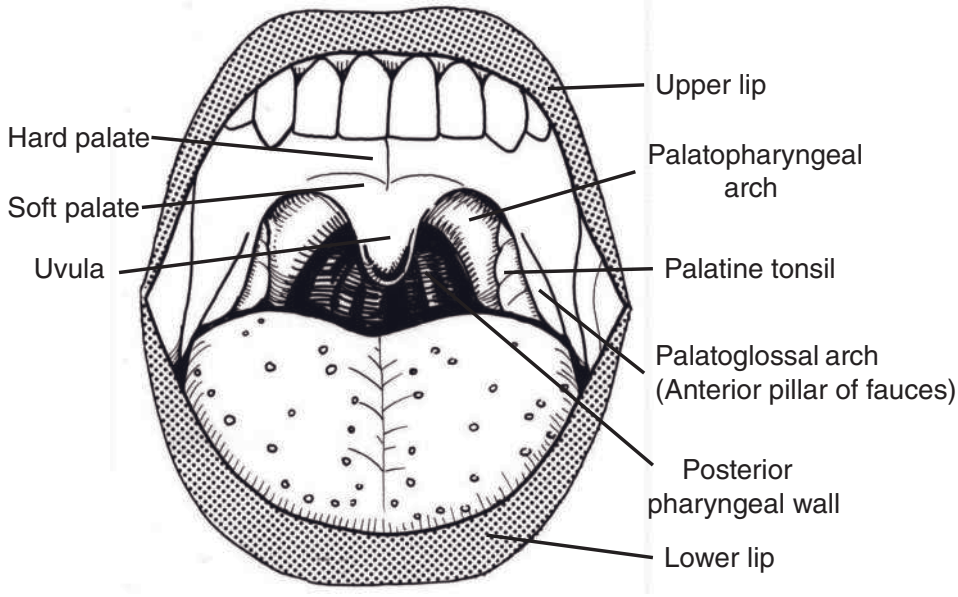

Figure 1.3 A view of the open mouth to show some of the important landmarks. Note the position of the palatine tonsils. (Reproduced with permission from Atkinson & McHanwell, 2002.)

(Maryn *et al.*, 2004). 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

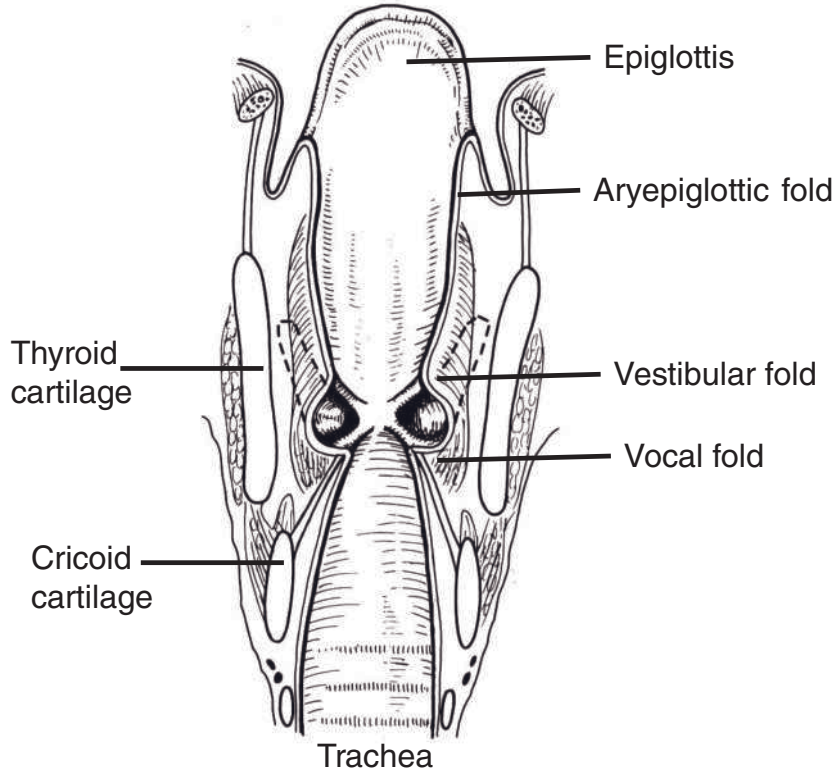

Figure 1.4 A schematic diagram of the larynx viewed from the posterior aspect. The larynx has been opened to present a clearer view of the positions of the vocal and vestibular folds. Note also the position of the aryepiglottic folds forming the lateral borders of the laryngeal entrance. (Reproduced with permission from Atkinson & McHanwell, 2002.)

1

Physical Structure and Function and Speech Production Associated with Cleft Palate

Martin Atkinson[1] and Sara Howard[2]

[1]University of Sheffield, School of Clinical Dentistry, Sheffield, S10 2TA, UK
[2]University of Sheffield, Department of Human Communication Sciences, Sheffield, S10 2TA, UK

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

Cleft Palate Speech: Assessment and Intervention, First Edition. Edited by Sara Howard, Anette Lohmander.

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 (Section1.5.1); 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 palatophayngeus 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.

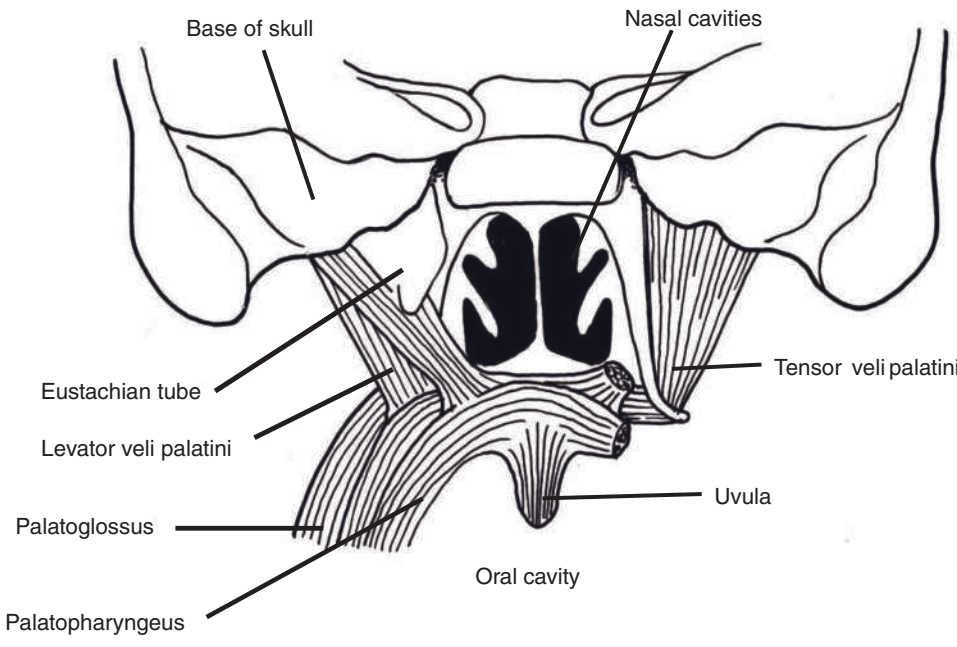

Figure 1.1 The muscles of the soft palate viewed from behind looking into the posterior nasal aperture. (Reproduced with permission from Atkinson & McHanwell, 2002.)

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).

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

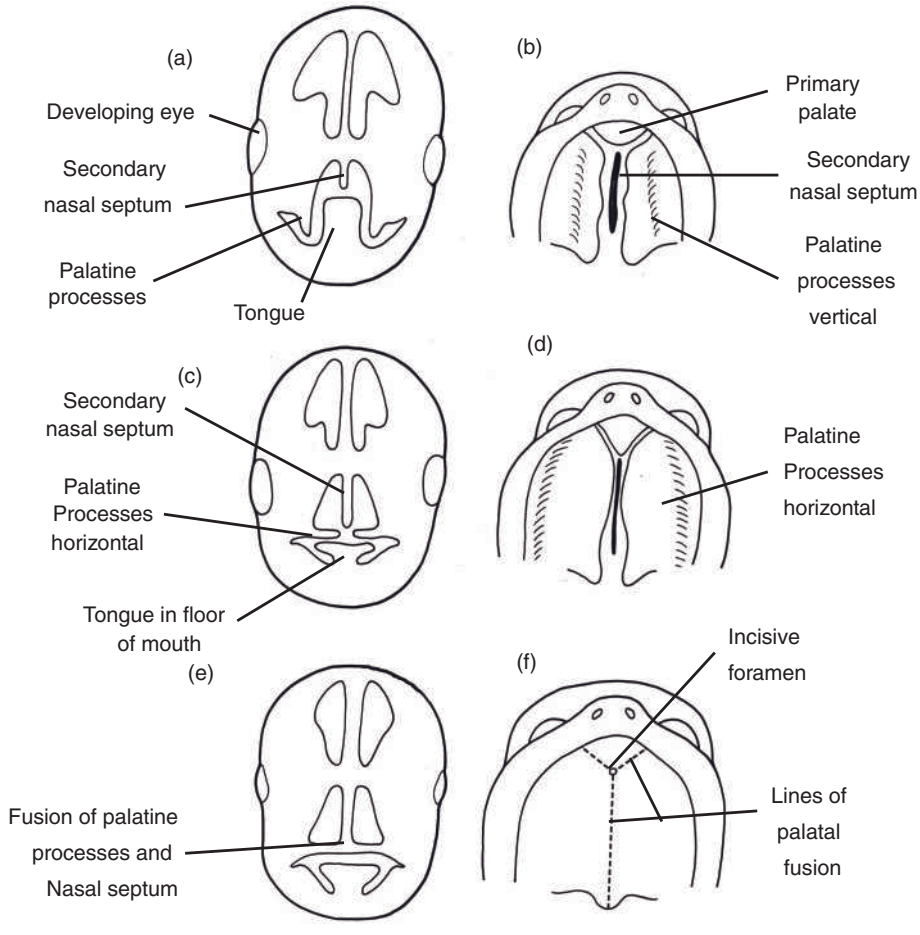

Figure 1.2 The development of the palate between 6 and 12 weeks post-fertilization. (a), (c) and (e) are sections taken through from the top of the head to the mandibular arch. (a) and (b) represent palate formation at about 6–7 weeks, (c) and (d) at 8 weeks as the palate elevates and (e) and (f) at 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.)

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.

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