Cleft Palate Speech

Assessment and Intervention

Edited by Sara Howard & Anette Lohmander
Cleft Palate Speech
Cleft Palate Speech: Assessment and Intervention

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Preface

This book emerged out of conversations which we, the editors, enjoyed over a number of years both at conferences and on visits to each other’s institutions in Sheffield, Gothenburg and, latterly, Stockholm. Observing current developments in research into speech production in cleft palate, we both recognised the need for a book which reflected the increasing breadth of the research being carried out across the world. Whilst important work was being undertaken in the more traditional areas of speech, there was a growing body of research, which recognised the potential of certain aspects of language, to contribute significantly to the field. We were also keen to recognise the importance of cross-linguistic and cross-cultural issues in cleft speech research. In addition, we wanted to broaden our focus to include both the speaker’s own and the listener’s perspective on communication. Thus we chose to use the WHO-ICF framework as a backdrop to all of the work contained in this book. Finally, we aimed to include current evidence of best practice (EBP) regarding both assessment and intervention. Our contributing authors were thankfully very receptive to these ideas, and thus the concepts of the WHO-ICF structure and EBP are given specific attention and have been regularly applied throughout the book.

For one of us, there was also a more specific stimulus for this book: coincident with its development, a set of postgraduate courses in cleft palate were being introduced at the University of Sheffield, and this book was designed, in part, with the needs of these students in mind. From this perspective it can be seen as a companion text to Watson, Sell and Grunwell’s Management of Cleft Palate Speech. Where that book provides a picture of all aspects of the multidisciplinary care of individuals with a cleft, this book focuses specifically on speech, and on assessment and intervention for speech problems associated with a cleft. We have both learnt a lot from conversations with our postgraduate students, who come from all over the world, and hope that this book reflects that learning process and will, in turn, prove useful to all of our future students.

We have clearly been very lucky that such a strong and inspiring set of researchers agreed to collaborate with us on this project. It has been a pleasure and a privilege to work with them. And we have been lucky, also, in having a series of very supportive (and unflappable!) editors at Wiley-Blackwell, who guided us patiently throughout the process, with all its attendant hiccups and delays. Our families should get a mention, too, for their support and forbearance!

Sara Howard and Anette Lohmander
Part One

Speech Production and Development

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In this book we examine the nature and impact of speech difficulties associated with cleft. As with all developmental speech impairments, cleft speech problems have experienced a significant broadening of perspective over the last century. Following a long period when all children’s speech difficulties were seen as articulatory in origin, and as being wholly interpretable through a medical model (Macbeth, 1967), there has been a gradual but welcome transformation to the current position, where much more emphasis is placed on other potential areas of difficulty (including phonology, language, literacy and interpersonal communication and interaction, as well as psychological and psychosocial implications). Developmental speech impairment is thus now situated within a social context. This fits comfortably with developments over the last decade or so, which have sought to classify and consider speech, language and communication impairments using the ICF (the International Classification of Function, Disability and Health; WHO (World Health Organization), 2001). In this book we use the ICF throughout as a point of reference.

Even a glance at the structure and headings used by the ICF indicates how useful it can be for extending our understanding of the possible impact of a communication...
impairment associated with cleft palate. There are two main parts (‘Functioning and Disability’ and ‘Contextual Factor’) with subcomponents which include, for the former, Body Structures, Body Functions, and Activity and Participation, and for the latter, Environmental Factors and Personal Factors. Such is the value of this framework that in the United Kingdom the Royal College of Speech and Language Therapists, in its manual on commissioning and planning services for cleft palate and velopharyngeal impairment (VPI), provides a detailed description of the impact of a cleft which relates specifically to the ICF classification (RCSLT (Royal College of Speech and Language Therapists), 2009). The ICF provides what McLeod (2006) describes as as ‘biopsychosocial view of health’ and, thus, of communication impairment.

It is noteworthy, of course, that unlike many types of developmental speech impairment, cleft speech problems do, indeed, have a physical basis, and thus the ICF subcomponent Body Structures is relevant in a way which is not the case for most children with speech difficulties. Thus, we need to understand what the anatomical and functional constraints on speech production are likely to be, as well as being aware of how physical structure and function are likely to be affected, over the lifespan, and over the course of speech and language development, by surgical intervention. Chapters in the following section consider each of these issues and also reflect on current evidence for different methods of assessment and intervention. The ways in which speech development for a child with a cleft palate are likely to be similar to and different from speech development in children without a cleft is clearly a hugely important area, which is also addressed in this section.

To make clinical, diagnostic decisions and to plan effective intervention, we need to be able to distinguish between speech difficulties directly attributable to the cleft and its consequences (including the likelihood of hearing impairment), and the coexistence of more general phonological delay or disorder. Such diagnosis can only take place if we have detailed information about the typical course(s) of speech and language development for children with a cleft. The ICF component ‘Body Functions’ is relevant here, including, as it does, intellectual and cognitive function, and temperament and personality, as well as specific aspects of speech production, including articulation, voice, fluency and also hearing (McLeod and Bleile, 2004).

In seeking a wider, more holistic perspective on the impact of a speech impairment, the ICF can also help us to understand the effects of a cleft on a child’s ability to participate more broadly in social interaction, across different contexts, including vital areas such as education, family and social life. The ICF components remind us that a communication impairment is not just the property of an individual, but is constantly negotiated between different individuals, in different contexts: a child’s intelligibility, for example, will differ depending on when, why, where and with whom they are talking. As the title of McCormack et al.’s article (2010) eloquently puts it ‘My speech problem, your listening problem and my frustration …’. Later chapters in this book deal in detail with intelligibility and with the child’s ability to participate in society through effective use of communicate.

The second of the main parts of the ICF, Contextual Factors, encourages us to consider the impact of a cleft palate and cleft speech difficulties in terms of the systems, policies, services and attitudes existing in a particular society, country or culture that will exert an influence on the support a child is likely to receive. Taking this perspective, one can quickly see how the impact of a cleft could be very different in the developed versus
developing (minority versus majority) world, where infrastructure and attitudes may differ significantly. One of the chapters in the following section addresses this important issue. Personal factors, such as age, gender, race, character and general psychological resilience and well-being, will also need to be taken into account when considering the impact of a cleft. Some children with severe speech disorders will nevertheless prove remarkably resilient in the face of their difficulties, whereas others may need specific help to adapt to even mild speech problems (Nash, 2006).

The ICF, then, provides us with a framework which can extend our thinking about the impact of a speech impairment associated with cleft palate and encourage us to take a more holistic view of individuals thus affected (Ma, Threats and Worrall, 2008). The material we cover in this book endeavours to do just that.

References


1

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.
PHYSICAL STRUCTURE AND FUNCTION AND SPEECH PRODUCTION

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).

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 adenoidealctomy may then cause hypernasality, as the child fails to adjust to the increased velopharyngeal port space (Witzel et al., 1986).

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