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Dysarthria

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Introduction

Dysarthria represents a neuromuscular disorder of speech. That is, it is a problem with speech pronunciation, which arises in relation to disorders of the central or peripheral nervous system that impairs the nerve pathways and muscle movements involved in the production of speech. If the disruption is so severe that no speech is possible it may be termed anarthria (though the francophone term 'anarthrie' is often used to mean what in English is termed apraxia of speech – see below). A distinction is made between acquired and developmental dysarthria. The former denotes problems arising from disorders that occur after speech has been fully learned. The latter covers dysarthrias associated with congenital and perinatal conditions that affect nervous system support for the speech musculature, or disorders acquired in childhood before speech is fully developed. The focus of this chapter is primarily on acquired dysarthria.

In its very mildest forms dysarthria may exist more as a subjective feeling of added effort required for speech on the part of the speaker, or a feeling of altered sensation around the mouth or face. Speech distortions may be scarcely, if at all perceptible to even a trained listener. It may only manifest itself at certain times or on certain tasks - e.g. when fatigued, under time pressure; on particularly complex sound combinations; or when attention to speech production is distracted by another complex task. In severest forms the speaker is totally mute (anarthric), or they can produce only completely unintelligible utterances.

This chapter looks at underlying causes of dysarthria and the variety of types of dysarthria that are said to exist. An overview of assessment practices is offered, emphasizing that a full understanding of dysarthria is gained only when one moves beyond a narrow impairment, underlying neuromuscular view of dysarthria, to encompass its effects on activities the person may engage in and its effects on their psychosocial well-being and participation in society. Treatment principles and general practices are also outlined.

The cause and course of dysarthria

To produce speech we need to be able to take in sufficient breath for each utterance, control expiration of air to last for the length of the utterance and closely coordinate expiration onset and termination with starting and stopping speech. The air driven from the lungs causes the closed vocal cords to vibrate (around 120-130 Hertz (cycles per second) for older men, 200-210 for older women). The vibration creates what we hear as voice. Appropriate variations in loudness, pitch and vocal quality (e.g. roughness, breathiness) depend on the balance between the tightness and pattern with which the vocal cords come together, the tension within the cords and the pressure of air from the lungs. What we hear as speech sounds, derive from the

ways in which movements of the velum (soft palate), tongue and lips shape the vocal note by shutting off, channelling and redirecting the air stream in different ways. Speaking thus entails balanced control of the muscles of the abdomen, diaphragm and chest wall (respiratory subsystem for speech); the intrinsic and extrinsic muscles of the larynx (laryngeal, phonation subsystem); the velum and pharynx (velopharyngeal, resonance subsystem); the tongue, lips and mandible (articulatory subsystem).

If the delicate balance between timing of breathing and speech, between air pressure from the lungs and vocal cord control, ability to raise and lower the velum efficiently and, to move and shape the tongue and lips is altered, voice and speech are correspondingly distorted – i.e., speech becomes dysarthric. Dysarthria arises when nervous system disturbances alter the normal generation, pattern and transmission of nerve impulses to muscles; this in turn affects the tone, power, coordination of movements of any or all of the muscles involved in producing voice and speech; and this in turn alters the range, rate, force, sustainability of articulatory movements.

Such impairments may stem from isolated cranial or peripheral nerve lesions that affect a particular muscle group (e.g. XII cranial nerve lesion (Umapathi, Venketasubramanian et al. 2000; recurrent laryngeal nerve lesion). They can stem from circumscribed lesions within the primary motor cortex or subcortical structures and pathways. Impairment can be associated with systemic changes that impact on multiple structures and/or pathways. Disorders of the neuromuscular junction (e.g. myasthenia gravis) and of muscles themselves (e.g. the muscular dystrophies) that affect speech production are usually considered under the umbrella of dysarthria too.

Dysarthria can be the sole symptom of a neurological condition. Other times it is part of a more wide-ranging disruption to communication, and in several etiologies is part of a highly complex picture involving motor planning as well as execution problems, alterations to sensation, and a whole range of autonomic and neuropsychological dysfunctions.

The course of dysarthric symptoms varies according to the underlying aetiology. Dysarthric speech may be the first indication that something is amiss, a signal for more detailed investigations. It can be an early, inevitable and prominent feature of a disorder (e.g. motor neurone disease). In other disorders, dysarthria may be a late or rare manifestation. In selected conditions symptoms are reversible after treatment of the triggering cause (e.g. in certain metabolic or drug induced disorders). After stroke, head injury, tumour removal symptoms may show (for a time) some improvement, even if recovery is to a plateau below previously normal speech. In degenerative conditions the course may be slowly or rapidly progressive, with or without periods of remission or stepwise decline.

Are there different types of dysarthria?

Historically, dysarthria was viewed as a unidimensional phenomenon, with variations in the ear of the listener characterised by such technical terms as ‘hot potato speech’, ‘gravelly voice’! Pioneering work in the 1960’s (summarised in Darley et al. 1975) claimed there are distinguishable subtypes of dysarthria associated in a one to one fashion with lesions in different parts of the central and peripheral nervous system.

They recognised flaccid dysarthria linked to lower motor neurone lesions with characteristic hypotonia and loss of movement range, force, sustainability. Spastic dysarthria, with its typically slow, laboured, effortful speech, was described as the result of upper motor neurone

lesions. Given the rich crossed and uncrossed innervations of the speech articulators this was claimed to arise principally after bilateral lesions, though a unilateral form is acknowledged (Urban, Wicht et al. 1999; Urban, Rolke et al. 2006). Lesions of the cerebellum, in analogy to limb motor control disruptions, brought about ataxic dysarthria, with a corresponding dysmetric (over and undershooting) movements and dysrhythmic (dysfluent, uneven) flow of speech (Spencer and Slocomb 2007). In the taxonomy of Darley et al lesions of the basal ganglia may cause dysarthrias with either slow, reduced range of movement (hypokinetic dysarthria, as in e.g. Parkinson's disease) or by exaggerated, fast or slow dyskinetic movements (hyperkinetic dysarthria, as in e.g. Huntington's disease, chorea). Some etiologies may produce a mixed type dysarthria showing characteristics of different subtypes (e.g. in multisystem atrophy or multiple sclerosis).

There is evidence that indeed different clusters of speech characteristics exist associated with varying CNS lesion sites. These rest primarily on physiological and acoustic studies that have examined patterns of muscle function and elements of the sound signal and its stability and variability in terms of duration, rhythm, amplitude, pitch/frequency (e.g. Kent and Kim 2003; Weismer 2006; Yorkston 2007). This approach, combining perspectives from neuroanatomy, neurology and speech sciences has undoubtedly brought significant advances in our understanding of speech motor control and its breakdown in the dysarthrias. It has led to important clinical insights.

Whether the claimed different dysarthria types, that can be differentiated instrumentally, can be distinguished perceptually (i.e. with the naked ear), even by trained listeners, remains a moot point. Several groups have been unable to replicate Darley et al's findings concerning the perceptual features and distinctions between putative dysarthria types (Zyski and Weisiger 1987; Zeplin and Kent 1996; Weismer 2006). Nevertheless, in as far as some therapies are better suited to tackle increased rather than decreased tone, or dysmetria, and disco-ordination rather than tone and power problems, then at some level of analysis etiological differentiation is important. The model offered by Darley et al provides a valuable framework within which to understand these issues.

As well as so called acquired dysarthria, when disruption is to a speech system that has previously been fully developed and normally functioning, there is also congenital or developmental dysarthria associated with various developmental disorders, perinatal conditions or trauma and illness acquired before the onset or completion of speech development. For developmental dysarthrias, at least those associated with cerebral palsy and Worster Drought syndrome (congenital suprabulbar palsy), current taxonomies cover spastic, dyskinetic, ataxic and mixed subtypes.

Dysarthria also arises in association with a range of other neuromuscular, movement disorders. It can be present in dystonia, especially where neck, jaw, facial, lingual and laryngeal movements are involved. Spasmodic dysphonia (adductor and abductor laryngeal dystonia) is one of the more common dystonic disorders affecting speech (more strictly speaking voice). So called action dystonias (where abnormal movements emerge under specific conditions) have been described in association with a range of speech behaviors (Scolding, Smith et al. 1995; Laskawi and Rohrbach 2001; Ilic, Potter et al. 2005; Grillone and Chan 2006; Singer and Papapetropoulos 2006; Roy, Mauszycki et al. 2007; Schneider 2007). Tremor is a feature common to many neurological conditions and can cause dysarthria when the head, neck, palate, larynx, chest wall movements are implicated (Zadikoff, Lang et al. 2006; Whaley, Putzke et al. 2007). Dysarthria has also been described as a symptom

underlying other speech phenomena such as a foreign accent syndrome (Miller, Lowit et al. 2006).

What dysarthria is not

Regarding differential diagnosis dysarthria is distinguished from several other speech disorders with which it may be confused or co-occur. Dysarthria is a problem with the execution of movements to produce sound, with the medium, not the message. In this sense, it contrasts with aphasia, a disorder of language where problems entail understanding others or finding the words and grammatical structures to convey meaning to express ideas.

As a neuromuscular disorder dysarthria is a breakdown in neural transmission, not a difficulty in the planning of movements. The latter is caused by (speech) apraxia. There, tone, power, coordination; and hence range, rate, force of movements, are intact. The problem centres on being unable to assemble individual movement components into a concerted whole action (McNeil, Robin et al. 2008; Miller and Wambaugh in press).

Traditionally, apraxia of speech has been seen as a cortical level breakdown and dysarthria as a subcortical dysfunction. More recently a greater role of subcortical functions in speech planning has been established – in particular a role of the cerebellum in feedforward monitoring of control, synergistic action and even coordination of higher cognitive functions, and for the basal ganglia a role in initiation and switching of movements and modulation of activities of the frontal lobes (Paquier and Marien 2005; Spencer and Rogers 2005). Thus the previously sharp distinction between cortical and subcortical speech disruptions has become blurred, more complementary rather than mutually exclusive.

This is especially so in developmental speech disorders where the differentiation and independence of roles and pathways from each other has not emerged. This doubtless contributes to the debates surrounding the separation of developmental dysarthria and developmental apraxia of speech (also termed childhood apraxia of speech; American Speech-Language-Hearing Association 2007). Occasionally, individuals present with speech and voice distortions that sound dysarthric / dysarthrophonic, but are in fact psychogenic in origin or associated with various psychiatric states, (Duffy 2005; Aronson and Bless 2009). Speech-language pathology and psychological evaluation are required to exclude neurogenic factors and include psychogenic ones.

Assessment

Taking an ICF perspective of motor speech disorders (Hartelius and Miller in press) assessment divided into the examination of: i) the underlying impairment (alterations to tone, power, coordination of movements and consequent alterations to range, velocity, etc.); ii) the consequences speech changes have, for communication activities (e.g. intelligibility and naturalness of speech); iii) the impact real and perceived changes exercise on the speaker's participation in society and possible psychosocial impact on them and their family.

Concerning whether intervention is indicated as or not the prime focus will be to establish: i) if problems exist with intelligibility and acceptability of speech that affect the person's ability to go about their daily activities; ii) whether or not speech changes, even if they do not depress intelligibility, nevertheless, affect a person's ability or willingness to participate in their normal and desired roles. Importantly, the severity of the dysarthria, in terms of listener impressions of distortions to speech or voice or in terms of objective clinical assessments

does not relate in any straightforward way to the severity of impact dysarthria may have on the individual and their family (Baylor, Yorkston et al. 2005; Miller, Noble et al. 2006; Miller, Noble et al. 2008; Hartelius, Jonsson et al. in press). Mild speech or voice changes may exercise a devastating effect on one individual; apparently severe changes may not significantly perturb another.

Secondary focus is on underlying impairment to examine what the basis of lost sounds or sound contrasts might be and to determine appropriate therapeutic methods. Where the aim of assessment is to consider if changes to speech are present that indicate a neurological episode has occurred or that herald a degenerative condition, impairment assessments are more likely to constitute the prime focus of evaluation.

Intelligibility testing

This is best accomplished using diagnostic intelligibility tests (Kent, Weismer et al. 1989; Gentil 1992; Ziegler and Hartmann 1993; Hunter and Kempler 2004) to determine which sounds, and more crucially which sound contrasts, an individual has difficulty signalling and which losses have the greatest consequences for intelligibility. There are some issues around best methods for scoring such tests (Hustad 2006; Hustad 2008), but diagnostic testing still outweighs alternative ways of assessing. Rating scales or wordlists not selected for their minimal pair diagnostic capability to establish levels of intelligibility have no use in informing therapeutic content and in any case have been demonstrated to be markedly unreliable measures (Schiavetti 1992; Kreiman and Gerratt 1998). A measure sometimes gainfully employed is of comprehensibility (Yorkston, Strand et al. 1996; Hustad 2008). This refers to the ability of the listener to understand the acoustic signal given all the other clues (grammar, topic of conversation, physical context, etc) there may be to intelligibility.

Speech that is intelligible but that nevertheless is felt by speakers themselves or listeners to deviate from normal patterns, to sound abnormal, can act as a barrier to communication. Speech naturalness or acceptability can be gauged using types of rating scale (Southwood and Weismer 1993).

Participation and psychosocial impact

Assessments of communication related quality of life specific to dysarthria have only recently been developed. The PROMIS programme (DeWalt, Rothrock et al. 2007; Baylor, Yorkston et al. 2009) is developing item banks applicable to dysarthria. Some general scales exist that quantify narrower or broader domains of impact (Donovan, Kendall et al. 2008; Hartelius, Elmberg et al. 2008; Walshe, Peach et al. 2009). Other suitable methods have been adapted from related fields to dysarthria – e.g. semantic differential techniques (Miller, Noble et al. 2008), or have borrowed assessments that focus on particular aspects of performance – e.g. for voice (Bogaardt, Hakkesteegt et al. 2007; Karnell, Melton et al. 2007).

Impairment assessments

These examine the ability of the different speech subsystems to function to their normal capacity and, vitally, to operate in coordination with each other. The focus, therefore, is on the ability to take in sufficient breath; the ability to control expiration; to be able to produce a viable and functioningvocal note; to evaluate the production and balance of nasality in speech; to look at the ability of the tongue and lips to achieve and adjust necessary articulatory positions and sustain and coordinate movements compatible with intelligible speech.

Clinically, these are accomplished with tasks such as: ability to sustain expiration for as long as possible on 'ah' or 'sss', or blow bubbles through a straw at 5cm depth for 5 seconds (Hixon, Hawley et al. 1982); loudness, variability and sustainability of 'ah' sound and the ability to vary the pitch up and down. Perceptual assessments of voice (e.g. GRBAS, Karnell, Melton et al. 2007) may be used to judge voice quality changes. Diadochokinetic tasks are assessments of choice in testing individual articulators (e.g. repeat syllable 'ta' for tongue tip function, 'pa' for lip control) and coordination between articulators (e.g. repeat 'pa-ta' or 'ta-ka') (Ziegler 2002; Gadesmann and Miller 2008). In research contexts acoustic and physiological measures of respiration, resonance and articulation may supplement or replace clinical measures in characterising speech status (McNeil 2008).

An important aspect of assessment concerns the ability of the speaker to signal and the listener to perceive key variations in stress and intonation that contribute to intelligibility and acceptability. This is accomplished through assessment of prosody (cover term for the rhythm, stress and intonation dimensions of speech) (Kuschmann, Miller et al. in press).

Many earlier tests utilised nonverbal movements to gauge articulator impairment (e.g. stick your tongue in and out; blow out your cheeks). The utility of these tasks for informing speech functioning is in question (Clark 2003; Weismer 2006; Ruscello 2008). Currently, the consensus favours an emphasis on speech and speech-like tasks (e.g. diadochokinetic repetitions), avoiding nonverbal items.

Further assessment considerations

Communication does not take place solely through speech. Body language, nonverbal gestures, and facial expression all play a role. In many neurological conditions, these elements of communication are also affected. Furthermore, spoken communication does not occur in a vacuum. It always involves a context and at least one other interlocutor. Facets of the environment and qualities of the listener may also bear on communicative success. Thus a full evaluation of dysarthria should also encompass these features.

Treatment

The overarching aim will be to improve communication in order to enable as full as possible participation in the family and wider social life of an individual. Accordingly, intervention will entail programmes directed at personal communication and life goals, but formulated within the broader physical, sensory, cognitive, and social context occasioned by the neurological condition. Whilst elements of intervention may tackle impairment level performance, this is subordinate to minimising consequences for activity limitation and maximising opportunity for participation in communication. Spoken communication may be a desire, but on occasions partial or total reliance on alternative and augmentative means of communication may prevail (Beukelman, Fager et al. 2007).

Therapy is set up with carefully staged goals, to challenge but not defeat the individual; and follows principles known to foster motor, cognitive and social learning. Therapists may use imitation, unison talk and action, peer pairing, watch and listen, guided imagery, as appropriate, to support advances. They will employ a range of tailored exercises and techniques to target specific aspects of speech production.

If assessment showed breathing for speech to be a problem, therapy attends to the prerequisites for efficient breath capacity before proceeding to breath control practice. Chief preconditions cover: stable sitting and/or standing posture to facilitate unimpeded expansion of the abdomen and thorax; head and neck (upright, central, stable) posture that does not impede mouth opening, inflow of air or quality of voice and speech output; attention to airway health to manage effects of e.g. chest infections, on lung capacity.

Therapy may be direct to help the individual adjust and maintain their own posture, or indirect through creating prosthetic postural support or seating adjustments. Frequent airway infections require medical management, as well attention to possible sources of infections (e.g. dysphagia, poor oral health; both commonly coexistent with dysarthria). A secondary cause of poor breath support may be an insufficient velum, in which case management targets this too (below).

Work on breath control itself aims to effect normal patterns of breathing, through attention to inspiratory and expiratory capacity and control and suppression of maladaptive patterns. Speech-language pathologists employ a variety of exercises to achieve this. Speakers can monitor their inspiration through cognitive, attentional cues (think: big breath in); through awareness of tactile-kinaesthetic feedback of rib and abdomen expansion; and by instrumental visual feedback from high- or low- tech airflow monitors.

Once sufficient breath support is available one source of possible voice difficulties is excluded. More specific targets around voice production will be to create a vocal note loud enough for listeners to hear, that can be sustained long enough to complete an utterance, where voice quality is not so distorted as to mask the articulatory message, and is acceptable and appropriate for the context. Excessive or unpredictable swings in loudness or pitch are addressed. Again a range of programmes and techniques is available, some with good or promising evidence of efficacy (Ramig, Sapir et al. 2001; Yorkston, Hakel et al. 2007; Speyer 2008).

Resonance disturbances in dysarthria typically concerns reducing excess nasality. First line treatments are via behavioural methods (Yorkston, Spencer et al. 2001; Dworkin, Marunick et al. 2004). Several prosthetic interventions are available if behavioural methods fail, or to supplement them (e.g. palatal lifts, Karnell, Hansen et al. 2004), nasal obturators (Hakel, Beukelman et al. 2004). Continuous positive airways pressure treatment has been piloted as potentially effective (Cahill, Turner et al. 2004). Surgical intervention may be a last port of call for selected candidates if hypernasality is a serious and intractable problem.

Once adequate breath support, viable voice and balanced resonance are available attention turns to intelligibility. Intervention may be direct, with articulation work aimed at retraining missing sounds; re-establishing lost contrasts and/ or stabilization of sounds where inconsistent production undermines intelligibility. Mandibular control may occasionally be the subject of therapy if it lies too open (overtaxing suboptimal lip and tongue movements), shows excessive tremor or dyskinetic movements or is held too closely shut to permit efficient inspiration.

Work may be indirect via modifications to speaking overall that have been shown to improve intelligibility. Two approaches here are rate control (Yorkston, Hakel et al. 2007; Hustad and Weismer 2007) and overemphasizing movements, creating maximum effort in executing articulatory gestures (DeThorne, Johnson et al. 2009). Rate control will be particularly

pertinent in ataxic dysarthria where the problem lies with coordination of movements across the vocal tract rather than difficulties attaining target range, force or velocity for individual articulators.

An issue parallel to the use of nonverbal vs verbal tasks or not in assessment applies to intervention. The same arguments pertain, with a consensus favouring similar conclusions (Clark 2003; Weismer 2006) that speech, not nonverbal movement, is best for practising speech, or at least that there is a lack of well designed studies to definitively settle the argument (McCauley, Strand et al. 2009).

Recalling that communication entails more than just speech and more than one person, it always occurs in context and elements of the context may support or hinder communication, it is equally important to address ways in which variables external to or interacting with the person with dysarthria can be manipulated to achieve successful exchanges.

Conclusion

This overview has given a broad indication of the nature and variety of dysarthria, as well as highlighted several practical and theoretical issues pertaining to assessment and management. The coverage will, it is hoped, be sufficient for many purposes. The references given will help readers delve into further detail where necessary.

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