3.5 Auditory processing and physiological factors

Thus far we have looked at the case for an auditory disturbance based on disrupted laterality. However, there is limited evidence that stuttering may be associated with differences at a more peripheral level also. In an old but oft-quoted study, Shearer and Simmons (1965) compared electromyographic (EMG) activity in the reaction of the stapedius muscle to vocalization between a small group of people who stutter and control speakers. This tiny muscle contracts as a reflex action at the onset of vocalization (Borg & Zarkrisson, 1975).[2] Shearer and Simmons (1965) noted that, while the timing of stapedius contraction was consistent within the control group, the group who stuttered showed a greater delay in timing onsets. Further research has proved equivocal, though: Hall and Jerger (1978) found no difference between experimental and control groups with regard to the timing of stapedius contraction, but did find that the scale of contraction was reduced amongst those who stuttered. Subsequently, Hanley and Dorman (1982) found no differences between their two groups, although it is possible that this may be due to using a different method to activate the laryngeal nerves to those used in earlier experiments. A problem with all of these studies is that findings were based on a small number of subjects, and this, coupled with the range of findings, leaves many questions unanswered regarding a possible physiological basis to auditory disturbance.

Another source of data lies in the study of air and bone conduction, and the scientific phenomena that if two pure tones of identical pitch (frequency) and loudness (amplitude) are presented in opposite phases (180 degrees), then they will cancel each other out and no sound will be heard. An early study (Stromstra, 1957) subjected a group of people who stutter and a control group to two such tones, one presented through air and the other through bone (via the teeth). The subjects were then asked to manually vary the amplitude and phase until they no longer heard any sound. A significant difference was found between the two groups in the relative phasing of the air- and bone-conducted tones at 2000 Hz. In a related experiment, Stromstra (1972) involved similar groups of subjects adjusting amplitude and phase of two air-conducted tones presented at either ear until the sounds cancelled themselves out. The phase disparity at several frequencies was found to be twice as wide for the PWS group as for the controls. Again, small subject numbers call for cautious interpretation, but there is tentative support here for the notion that people who stutter have a reduced capacity to control and manipulate auditory signals.

3.6 Stuttering as defective auditory processing

So where does all this evidence lead us? Well, there is little doubt that, from a physiological, neurological and experiential perspective, the appearance of stuttering and changes to stuttering severity can be associated with auditory

processing variables. The cumulative effect of findings that stuttering can be increased and decreased, contingent on changes in auditory feedback, have led some authorities to believe that the disorder results, at least in part, from defective auditory processing. One such attempt was made in an innovative study by Harrington (1988), who contended that the basis for error-free ongoing speech output rested on the ability to accurately predict the rhythmic structure which pre-specifies the timing of the ongoing speech output from one stressed vowel to the next. Thus, (1) speech is perceived in terms of a rhythmic structure, based on the speaker's predictions regarding when the next stressed vowel will come; (2) the person who stutters makes faulty predictions as to the arrival-time of the next stressed vowel[3] due to a mistiming of auditory information; and (3) therefore stuttering results from the attempts to re-align the misperceived timing elements. So, in effect, the person who stutters is trying to correct an error which does not actually exist, and it is the attempt to resolve this perceived, but non-existent, timing error that results in the motor speech disruptions identified as stuttering. The argument followed that DAF is effective in increasing fluency amongst people who stutter because the artificial timing delay cancels out the asynchrony between the faulty misperception of when the next stressed vowel is due (see Figure 3.1.) The effects of DAF on normal speakers are also explained in this manner in the converse relationship. That is, under DAF, the late feedback leads the speaker to miscalculate the arrival of the next stressed vowel, which had been correctly predicted in the first place. The variety of motor-speech consequences of the incorrect vowel percepts, such as repetitions, prolongations and so on, are explained as various attempted repair strategies to rectify the misperceived rhythmic structure. The theory which stresses individual variability in these perceived timing relationships is potentially consistent with more recent knowledge that optimal delay settings on DAF devices vary from one speaker to another (Lincoln, Packman, & Onslow, 2010).

Although nearly 30 years old, this theory still appears innovative today. The problem with it now, as then, is that it is almost impossible to verify empirically, and equally that a production theory can account for this data as easily as a perceptually based one. As Borden (1988), in a counter argument, pointed out – stuttering occurs not because of a mismatch between the stutterer's perception regarding when the following stressed vowel is due and when it actually arrives, but because the production of the relevant timing units *is* disrupted. In other words, it is perceived as being late because *production* of that sound unit *is* late. It is also worth pointing out that Harrington's account would also not explain the more recently discovered fluency-enhancing effects of FAF. There may, though, be something of a stalemate here. Motor theories are not necessarily easier to verify empirically, either, but there is, as we saw in Chapter 2, growing support for stuttering as a problem of motor speech timing due to attenuation of signals in the basal ganglia/BG cortical projection circuitry.

Despite a body of evidence suggesting that altering auditory feedback can change fluency levels, defining stuttering as a consequence of disrupted

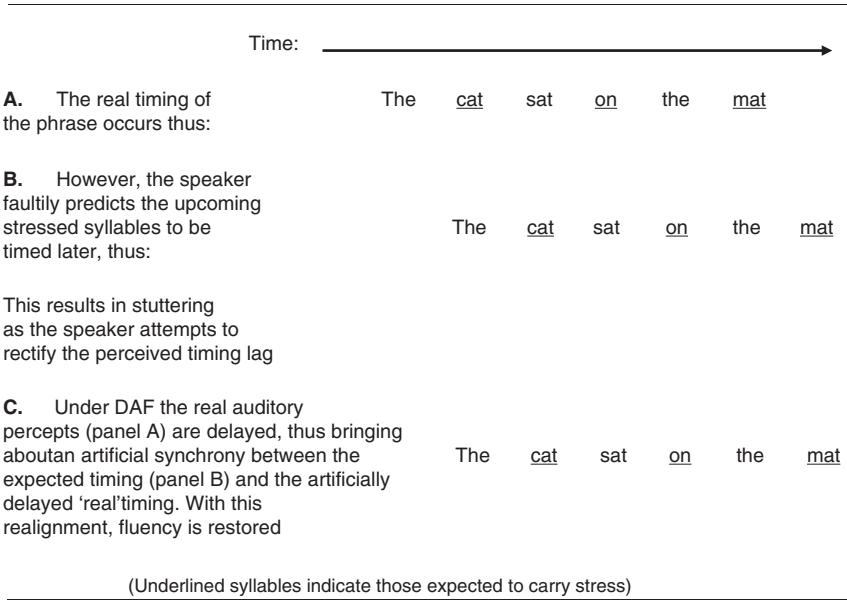

Figure 3.1 A schematic example of Harrington's model of stuttering as the misperception of linguistic rhythm.

The figure also demonstrates how introducing an auditory delay artificially restores synchrony between the speaker's auditory misperception and the 'real' timing of the next stressed vowel, thereby restoring fluency.

auditory feedback processing is far from universally agreed upon (Antipova et al., 2008; Postma & Kolk, 1992). Postma and Kolk (1992) argued against a theory of stuttering as defective auditory feedback following an experiment in which adults who stutter and control speakers had to detect self-produced speech errors while speaking under normal (no altered feedback – NAF) and white noise-masked feedback conditions. No significant differences were found between the two groups' ability to detect phonemic errors, leading the authors to conclude that error-monitoring skills function normally in those who stutter. They did, however, note that the subjects who stuttered detected fewer errors when asked to detect errors in the speech of others, but, as seen in Chapter 5, Postma and Kolk favour a production-based phonological processing explanation for the speech errors seen in stuttering.

3.6.1 DIVA\|GODIVA revisited

A rather different view on the relationship between motor and auditory feedback processes in stuttering has been proposed in Civier and Guenther's (2005) DIVA (Direction of Velocities into Articulators) and Civier et al.'s

(2013) GODIVA (Gradient Order Directions into Velocities of Articulators) models. As mentioned in Chapter 2, although essentially motor models, their premise is that the rapid and continuous nature of fluent speech production means that it cannot be controlled by a single feedback mechanism alone, but must also involve feed-forward projections that are mediated by an auditory feedback loop. The theory holds that PWS are prone to errors in these feed-forward mechanisms, leading to an over-reliance on auditory feedback, and it is this over-reliance that in turn leads to stuttering. As auditory feedback control is absent before a sound starts (a fact which can be problematic for some PWS who use altered feedback devices and experience silent blocks in word initial position), the model offers an auditory explanation for loci effects that are typically explained as consequences of motor or language variables (see Chapters 4 and 5, respectively): for example, why frequency of silent blocks are typically seen in sentence and word initial positions. The reduction in reliance on auditory feedback could explain how fluency increases under DAF; however, this does not explain the fluency-enhancing effects of FAF.

3.7 Summary

Evidence from a range of approaches and methodologies shows that stuttering is associated with disrupted auditory processing. Fluent speech requires the integration of both sensory and motor processes, so it is not surprising that, since motor speech is implicated in stuttering, there will be implications for auditory processing too. Whether stuttering can be regarded as a disorder of auditory processing, however, is another matter, and it is interesting to note that there may be some unusual right-sided bias for auditory processes in audition, just as there may be for motor speech production in older children and adults who stutter. Some of the fluency-altering effects of altered feedback noted in this chapter can potentially be explained in other terms – whether by motor explanations (Harrington, 1988) or perhaps even potentially psychologically based or distraction factors (choral reading or shadowed speech, where there is an external stimulus) – that might also influence self-perception in a rather different manner to the idea that the fluency comes about simply because it is perceived as exogenous. That said, it is difficult to ignore the strength of the findings which show that fluency is improved through attenuation of feedback of the speech signal (hearing impaired, masking). The effects, too, of AAF as a therapy tool away from the clinic and in many cases without resulting in speech rate changes suggest there is a genuine effect here (also see Chapter 15).

Key points

- The deaf population is the only one in which stuttering is under-represented.
- There is a range of evidence that points to the notion that stuttering is associated with disrupted auditory processing, although the exact nature of this disruption remains obscure.

- Auditory processing for speech may be a product of right hemisphere processing amongst older children and adults who stutter.
- DAF and FAF can have dramatic fluency-enhancing effects for some people who stutter, yet others remain DAF and FAF negative, for reasons which are currently unknown.
- Dichotic listening provides one testable method of determining hemispheric dominance for linguistic decoding and findings lend tentative support to the idea that auditory processing, too, might be a product of the right hemisphere, at least in some people who stutter.
- One of the biggest issues faced is that auditory processing is just one part of the communication chain, and does not occur in a vacuum. Both production and perception theories must allow for the fact that one is affected by the other (as seen in the criticism of Harrington's theory of linguistic rhythm and auditory feedback).

Notes

1 Subsequent findings on time delay settings have varied widely (see Chapter 15).
2 McCall and Rabuzzi (1973) argued that this is not merely reflexive action, but rather that the muscle contraction occurs as an integral and centrally mediated part of the vocalization process.
3 The significance Harrington places on the perception of stressed vowels is based on Ohman's seminal account of the nature of consonant and vowel relationships (e.g. Ohman 1967).

Suggestions for further reading

Harrington, J. (1988). Stuttering, delayed auditory feedback and linguistic rhythm. *Journal of Speech and Hearing Research*, 31, 36–47.

Yes, this is an old reference, but aside from the theoretical implications, this thought-provoking paper still provides a well-explained introduction to the links between perception and production aspects of speech processing in stuttering.

Howell, P. (2011). *Recovery from stuttering*. Hove: Psychology Press.

Howell's book covers a lot of theoretical ground but, in particular, along with Kalinowski, Howell has been a pioneer in the cause of reintroducing the field to the effects of altered auditory feedback since the mid-1980s. The chapters that implicate auditory processing are very well worth reading.

4 Motor speech control and stuttering

4.1 Introduction

Speaking is perhaps the most complex fine motor skill that humans can perform. The production of fluent speech requires the translation of abstract linguistic units into constantly changing movement sequences of the motor speech subsystems subserving articulation, phonation and respiration. Although a unitary definition of stuttering currently evades researchers, its primary expression lies in some incoordination within and between these three major motor speech subsystems. We see this with articulatory struggle, visible and auditory difficulties in commencing or terminating phonation and in spasmodic and poorly-timed breathing for speech. In other words, stuttering presents as a motor speech disorder. But to acknowledge that it presents as a motor control problem is not the same as concluding that stuttering is, in essence, a motor speech disorder. The speech of all but those with the most severe stuttering is of an output that is only intermittently interrupted by aberrant motoric activity. In fact, as we will come to see, findings show that while we may perceive nonstuttering speech as fluent, there is nonetheless evidence for consistent abnormal motor speech activity, even in the absence of observable stuttering.

4.1.1 Motor speech and linguistics: a proviso

The purpose throughout this book is to treat stuttering as a problem where systems are integrated, and the interrelationship between motor speech and linguistic factors in stuttering is one of the most interesting but also one of the most awkward to deal with. It is an issue we will return to in both this and the following chapter, which considers stuttering from a linguistic perspective. For the moment, we begin by examining the evidence for a dyscoordination hypothesis amongst the three motor speech subsystems that serve speech production, namely, those of respiration, phonation and articulation.

4.2 Respiratory control and stuttering

In order to initiate speech, an egressive airstream must be available, and for speech to be fluent and properly coordinated, airflow must be controlled with

precision. There is a solid body of literature dating back many decades which implicates problems of respiratory control during moments of stuttered speech with regard to fixations of respiratory muscle systems (Henrickson, 1936; Murray, 1932) and loss of control of subglottal air pressure during stuttering (Johnston, Watkin, & Macklem, 1993; Peters, Hietkamp, & Boves, 1993; Zocchi et al., 1990). A substantial body of evidence has also noted that muscle groups which normally work reciprocally to ensure effective breathing have, instead, been found to operate antagonistically. (The interpretation of muscle antagonism in fluent speech is a somewhat contentious issue, though, and we will return it below.) Nevertheless, many people who stutter report spasmodic abdominal and thoracic muscle activity coincident with moments of stuttering, and a comparatively common complaint is that this sense of disrupted breathing persists even prior to fluent speech (often accompanied by a fear of impending speech breakdown).

There seems to be some support for these reports in controlled studies. Adams (1974) and Agnello (1975) both found that people who stutter exhibited higher levels of intra-oral air pressure during nonstuttered speech than nonstutterers, whilst Hutchinson and Navarre (1977) found a similar effect during stuttered episodes. Even prior to a fluent episode, abnormal subglottal air pressure has been identified (Peters & Boves, 1988; Peters, Hietkamp, & Boves, 1995). So, the picture we get from these studies suggests disrupted motor control at a respiratory level. But it is of course the complexity of the interaction of the various systems that presents us with the most challenging issues. We will shortly see that this is indeed the case for respiration and its relationship with phonation.

4.2.1 Therapeutic relevance

Many therapeutic programmes allow for the readjustment of breathing patterns, and a considerable number encourage breathing from the diaphragm (see Chapters 12 and 13). The rationale for this varies somewhat from programme to programme, but it is often in an attempt to provide increased control over exhalation and to divert attention away from clavicular (upper chest) breathing, which is often seen in stuttered speech in combination with excessive upper body tension. The reader may also be interested to learn about the McGuire and Starfish programmes and the Valsalva hypothesis – all approaches that have disrupted breathing as the sole target of vocal tract 'retraining' (see Chapter 15).

4.3 Laryngeal control and stuttering

4.3.1 Voice onset

Voice onset time refers to the time lag between the sudden opening of two articulators following a plosive (or 'stop') consonant, and the time taken to

initiate periodic vocal cord vibration subsequently. Findings seem to demonstrate two trends. First, most adults who stutter tend to show longer voice onset times than their normally fluent peers, and, second, that more generally, control of VOT is less well constrained in the speech of those who stutter, resulting in a greater range of VOT when compared with those who do not stutter (Agnello & Wingate, 1972; Hand & Luper, 1979; Hillman & Gilbert, 1977; Ward, 1990; Zimmerman, 1980b). However, some studies have failed to find statistically significant differences between such groups (Cullinan & Springer, 1980; Viswanath & Rosenfield, 2000). Viswanath and Rosenfield (2000) found that, rather than delaying VOT, their group of adults who stuttered displayed plosive pre-voicing across a number of different linguistic contingencies, including rate and consonant identity, thereby demonstrating negative voice onset time. This 'full voicing' can sound somewhat similar to the 'continuous voicing' sometimes used as a fluency-enhancing component in some fluency-shaping approaches to therapy (see Chapter 13). This is a strategy that requires continuous vocal fold vibration, even through phonemes which would normally be voiceless.

The lack of clarity in these findings has been highlighted in a more recent study (Arenas, Zebrowski, & Moon, 2012), which examined the establishment of stable voicing onset (leading up to the plosive) and offset (following plosive release) in a group of 14 preschool-age children who stutter and similarly aged controls. They found no difference between the two groups' coordination of laryngeal and respiratory systems for either onset or offset. More studies are needed to determine the stability of this finding, but the fact that, during fluent speech at least, these parameters can be stable in children who have only recently started to stutter could be taken as tentative support for the idea that the lack of stability in studies with adults might reflect possible adaptation of vocal tract control parameters on behalf of the speaker, rather than some underlying and possibly innate motor control fragility. It is worth noting that these empirical observations implicating abnormal motor speech control close to stuttering onset mirror recent findings from brain imaging studies which find that brain function in structures regulating motor speech control are similarly implicated amongst children who have just begun to stutter.

4.3.2 Voice initiation time

Voice initiation time (VIT) is also known as acoustic reaction time, and is similar to VOT in that it describes a period of time that precedes periodic vocal cord vibration, but differs in that the phonatory response, usually either a speech sound or a nonsense syllable, is initiated as quickly as possible in response to an external stimulus: this might be a verbal command ('go!'), a nonlinguistic auditory stimulus (buzzing tone) or visual stimulus (presentation of stimulus light). A number of studies have found people who stutter to exhibit slower VIT responses (Adams & Haydn, 1976; Cross & Luper, 1979; Cross, Shaden, & Luper, 1979; Haydn, 1975; Starkweather, Hirschmann,