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AN ELECTROGLOTTOGRAPHIC AND ACOUSTIC ANALYSIS OF GLOTTAL ACTIVITY DURING SPEECH INITIATION IN STUTTERING

by

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ABSTRACT

Stuttering is recognized as a motor speech disorder with aberrant glottal activity being a salient characteristic. Current models of stuttering view this disorder as a dysfunction in the neuromechanism with involvement of specific neural structures, such as the basal ganglia. Within a framework of speech sensorimotor control as proposed by Van der Merwe (1997), the characteristics of glottal activity in stuttering seem to be associated with a motor programming disorder. Results of previous electroglottographic (EGG) and acoustic studies on vocal fold activity in people who stutter (PWS) showed abnormalities in the rate, range and muscle tone of vocal fold activities and difficulties in voice initiation. These results support the notion of motor programming involvement in PWS. It is therefore proposed that a description of vocal fold activity during a speech task may contribute to a better understanding of speech motor control in stuttering.

The purpose of this study was to determine differences in vocal fold activity in PWS and normal fluent speakers (NFS) during speech initiation. An EGG and acoustic analysis was conducted to investigate physiological and acoustically detected vocal fold activity in the two groups. The effect of different phonetic contexts on vocal fold activity was considered during the compilation of the speech material, namely: voiced without articulation (vowel), voiced with articulation ($C_{(v)}$), and voiceless with articulation ($C_{(vl)}$). The subject group comprised six PWS with stuttering severity levels ranging between very mild to severe. Six NFS were matched with the PWS with regard to gender and age. These six participants served as control group. Each participant orally read five wordlists consisting of 12 VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances. The EGG and acoustic recordings were made simultaneously. Only perceptually fluent speech samples were analyzed.

Results regarding *physiological vocal fold activity* showed the following: (a) Prephonatory vocal fold activity in PWS, compared to NFS, was mainly characterized by irregularities for VC, $C_{(v)}$VC and $C_{(vl)}$VC productions. (b) PWS had longer and more variable laryngeal onset time (L_xOT) values for VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances compared to NFS. (c) Motor complexity of the phonetic context affected the frequency of occurrence of disruptions in prephonatory vocal

fold activity as well as the L_xOT in both groups. For PWS, $C_{(v)}$VC and $C_{(vl)}$VC productions were more involved than VC productions.

The following results were obtained with regard to *acoustically detected vocal fold activity*: (a) Similar to NFS, the $C_{(v)}$VC and $C_{(vl)}$VC utterances of PWS were characterized by gradual voice onsets; VC productions showed a greater number of abrupt voice onsets; gradual voice onsets for PWS were associated with high frequencies of vocal fold irregularities in the period prior to voice onset. (b) Boths groups showed mean voice onset time (VOT) for initial voiced /b/ productions and initial voiceless /p/ productions within normal limits. (c) VOT range for /b/ productions was greater than for /p/ productions in NFS; this tendency was also seen in most PWS. (d) For PWS, VOT for /b/ productions were slower and overlapped with VOT for /p/ productions. The VOT range for /p/ productions showed consistency and a tendency toward a restricted VOT range. (e) As opposed to the NFS, VOT range values for /b/ and /p/ productions overlapped for most PWS; thus, VOT values for /b/ and /p/ productions did not indicate voice-voiceless contrasts for PWS.

The current findings on vocal fold activity during speech initiation in PWS were discussed within the framework of speech sensorimotor control with the emphasis on motor programming. From this perspective, deviant vocal fold activity in PWS may involve abnormal increase of muscle tone in the laryngeal structures or abnormaliteits in rate, velocity, and range of movements. Difficulties in the initiation of successive movements during the prephonatory period in PWS may also be associated with inefficient motor programming. The proposal that stuttering is a motor programming disorder could guide further explanations of speech motor control of PWS.

KEY WORDS: Stuttering, glottal activity, speech initiation, electroglottographic analysis, physiological vocal fold activity, acoustic analysis, acoustically detected vocal fold activity, motor programming, speech sensorimotor control.

OPSOMMING

Hakkel word beskou as ‘n motoriese spraakafwyking waarvan afwykende glottale gedrag een van die hoofkenmerke is. Resente teorieë beskou hakkel as ‘n disfunksie van die neurale meganisme met betrokkenheid van spesifieke neurale strukture soos die basale ganglia. Binne ‘n raamwerk van sensories-motoriese spraakbeheer soos voorgestel deur Van der Merwe (1997), kan die glottale gedragskenmerke van hakkel met ‘n afwyking in motoriese programmering geassosieer word. Resultate van vorige elektroglottografiese (EGG) en akoestiese studies oor stembandaktiwiteit in persone wat hakkel (PWH) het abnormaliteite in die spoed, die omvang en spiertonus van stembandaktiwiteit getoon, asook probleme met die inisiëring van stemgewing. Hierdie resultate steun die moontlikheid van ‘n disfunksie in motoriese programmering by PWH. ‘n Beskrywing van stembandaktiwiteit tydens spraakinisiëring by PWH mag daarom bydra tot ‘n beter begrip van spraakmotoriese beheer in hakkel.

Die doel van die studie was om verskille in glottale gedrag tussen PWH en normale vlot sprekers (NVS) gedurende spraakinisiëring te bepaal. ‘n EGG en akoestiese analise is uitgevoer ten einde fisiologiese en akoesties-waarneembare glottale gedrag by die twee groepe te ondersoek. Die effek van verskillende fonetiese kontekste op glottale gedrag is in aanmerking geneem by die samestelling van die spraakmateriaal, naamlik: stemgewing sonder artikulasie (vokaal), stemgewing met artikulasie ($K_{(sh)}$) en artikulasie sonder stemgewing ($K_{(sl)}$). Die groep proefpersone het bestaan uit ses PWH van wie die graad van hakkel van baie gering tot ernstig gewissel het. Ses NVS is met die PWH volgens geslag en ouderdom gepas. Hierdie ses deelnemers het die kontrolegroep gevorm. Elke deelnemer moes vyf woordlyste bestaande uit 12 VK, $K_{(sh)}$VK en $K_{(sl)}$VK uitinge hardop lees. Die EGG en akoestiese opnames is gelyktydig gemaak. Slegs perseptueel-waarneembare vlot uitinge is vir die data-analise gebruik.

Die resultate vir die *fisiologiese glottale aktiwiteit* was soos volg: (a) Prefonatoriese glottale aktiwiteit in PWH, in vergelyking met NVS, was hoofsaaklik deur onreëlmatighede vir VK, $K_{(sh)}$VK en $K_{(sl)}$VK produksies gekenmerk. (b) PWH het groter laringale aanvangstyd (L_xAT) waardes vir VK, $K_{(sh)}$VK en $K_{(sl)}$VK produksies getoon met meer variasie tussen die absolute waardes teenoor die NVS. (c) Motoriese kompleksiteit van die fonetiese konteks het die frekwensie van voorkoms van afwykende prefonatoriese glottale aktiwiteit asook die L_xAT by

beide groepe beïnvloed. By PWH het $K_{(sh)}$VK en $K_{(sl)}$VK produksies meer afwykende prefonatoriese glottale gedrag as VK produksies getoon.

Die volgende resultate is vir *akoesties-waarneembare glottale aktiwiteit* verkry: (a) In ooreenstemming met die NVS, het PWH geleidelike stemaanvang vir $K_{(sh)}$VK en $K_{(sl)}$VK produksies getoon; by VK produksies toon PWH ‘n hoër frekwensie van voorkoms van skielike stemaanvang; geleidelike stemaanvang vir PWH toon ‘n hoë frekwensie onreëlmatige glottale gedrag tydens die prefonatoriese periode. (b) Beide groepe se gemiddelde stemaanvangstyd (SAT) vir inisiële stemhebbende /b/ produksies en inisiële stemlose /p/ produksies was binne die normale omvangwaardes. (c) Die omvang van SAT vir /b/ produksies was groter as vir /p/ produksies in die NVS. Hierdie neiging is ook by meeste PWH waargeneem. (d) By PWH, was SAT vir /b/ produksies stadiger en het met die SAT vir /p/ produksies oorvleuel. Die omvang van SAT vir /p/ produksies het konstantheid getoon en ‘n neiging tot beperkte SAT omvang is vir PWS waargeneem. (e) In vergelyking met die NVS, het die SAT omvangswaardes vir /b/ en /p/ produksies vir meeste PWH oorvleuel; dus, SAT waardes vir /b/ en /p/ produksies het nie stemhebbend-stemlose kontraste vir PWH aangedui nie.

Die huidige bevindinge ten opsigte van glottale gedragskenmerke gedurende spraakinisiëring by PWH is binne ‘n raamwerk van sensories-motoriese spraakbeheer bespreek met die klem op motoriese programmering. Vanuit hierdie perspektief kan afwykende stembandaktiwiteit in PWH geassosieer word met ‘n abnormale toename in spiertonus in die laringale strukture of afwykings in rigting en spoed van beweging, en omvang van beweging toon. Probleme in die inisiëring van opeenvolgende spraakbewegings gedurende die prefonatoriese periode word ook met uitvalle in motoriese programmering geassosieer. Die voorstel dat hakkel ‘n afwyking in motoriese programmering is, kan verdere verklarings vir spraakmotoriese beheer in PWH rig.

SLEUTELWOORDE: Hakkel, glottale aktiwiteit, spraakinisiëring, elektroglottografiese analise, fisiologiese stembandaktiwiteit, akoestiese analise, akoesties-waarneembare stembandaktiwiteit, motoriese programmering, spraak sensories-motoriese beheer.

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"but those who hope in the Lord will renew their strength.
They will soar on wings like eagles;
they will run and not grow weary,
they will walk and not be faint."
Isaiah 40:31

CHAPTER 1: AN ELECTROGLOTTOGRAPHIC AND ACOUSTIC ANALYSIS OF GLOTTAL ACTIVITY DURING SPEECH INITIATION IN STUTTERING: ORIENTATION AND PROBLEM STATEMENT

1.1 INTRODUCTION

Stuttering is recognized as a motor speech disorder with aberrant laryngeal behaviour being an important aspect of the disrupted peripheral speech physiology (Wingate, 1970; Perkins, Rudas, Johnson & Bell, 1976; Freeman, 1979; Starkweather, 1982; Van Riper, 1982; Adams, Freeman & Conture, 1984; Denny & Smith, 1997). Many challenging questions still exist regarding the relationship between stuttering and laryngeal behaviour, for example: - Can the difference between the speech of persons who stutter (PWS) and normally fluent speakers (NFS) be associated with characteristics in glottal activity? What is the cause of abnormal vocal fold behaviour in stuttering? Are indications of slower initiation of glottal activity in PWS caused by poorly coordinated muscle activity preventing quicker movement of the vocal folds? Does a correlation exist between stuttering severity and aberrant glottal activity?

These questions illustrate the fact that the role of laryngeal behaviour in the breakdown of speech production in stuttering has not been indubitably clarified. Clarification of the nature of laryngeal characteristics of PWS is important for the interpretation of aberrancies in laryngeal behaviour, as well as for providing insight into the breakdown in coordination of phonation, respiration and articulation in stuttering.

Research findings regarding laryngeal coordination during fluent and stuttered speech of PWS and normally fluent speakers have great importance in forming a hypothesis about the relation between laryngeal behaviour and stuttering. Existing views suggest that aberrant laryngeal behaviour together with respiratory and articulatory deviancies form part of the symptoms of stuttering (Adams, 1974; Van Riper, 1982; Silverman, 1992). Perkins *et al.* (1976) even proposed that phonatory discoordination is the precipitator of associative articulatory and respiratory aberrancies in stuttering. However, whether some PWS's laryngeal systems are more subjected to mistimings than the articulatory system or the respiratory system during speech production, has yet to be proven. Detailed information on the acoustic and physiological

correlates of aberrant laryngeal movements during speech production in PWS will allow descriptions of the speech motor characteristics underlying the laryngeal discoordinations. Thus, descriptions of possible deviancies in initiation, timing control and duration of laryngeal coordination* in speech production, can be useful in constructing a theory of laryngeal stuttering behaviour as a breakdown in speech motor control.

In view of the continuous controversy regarding stuttering as a motor speech disorder (Kent, 2000), this study will be based on a comprehensive theoretical model of motor speech control to explain the characteristics of laryngeal behaviour in the speech production process of PWS. An example of a theoretically sound model of speech motor control that is gaining interest in the field of motor speech disorders, is the four-level framework for the characterization of pathological speech sensorimotor control proposed by Van der Merwe (1997). Based on the conceptualization of motor programming according to Van der Merwe's (1997) model, a motor programming disorder would result in an impairment of initiation of movement sequences, and specific spatiotemporal and force dimensions. It is of additional relevance to note that Borden, Baer and Kenney (1985) further assert that stuttering is characterized by difficulty in the initiation of voicing involving a breakdown in the coordination of respiratory, laryngeal and articulatory movements. Thus, in accordance with Van der Merwe's (1997) framework, it is subsequently proposed that stuttering might be associated with a dysfunction of motor programming.

1.2 PROBLEM STATEMENT AND RATIONALE FOR THE STUDY

Van Riper (1982:415) described stuttering as "a temporal disruption of the simultaneous and successive programming of muscular movements required to produce one of the word's integrated sounds...". Stuttering is thus viewed as a motor speech disorder characterized by temporal discoordination of the movements involved in speech. Several other theories also suggest that stuttering occurs as a result of a breakdown in the timing of speech motor movements, for instance, stuttering as a result of discoordination of phonation with articulation and respiration (Perkins *et al.*, 1976), stuttering as a sequencing and timing disorder (MacKay &

* In this study *laryngeal coordination with respiration and articulation* refers to intersystem coordination of phonation, respiration and articulation during speech production. Laryngeal behaviour is emphasized due to the nature of this study.

MacDonald, 1984), stuttering as a disorder of sensorimotor processing (Andrews, Craig, Feyer, Hoddinott, Howie & Neilson, 1983), stuttering as a temporal processing disorder (Kent, 1984), and stuttering as a disorder of movement production (Zimmermann, 1980*c*). Despite the fact that all of the hypotheses regarding stuttering as a failure in speech motor control agreed in principle that stuttering involves a mistiming of coordinated movements necessary for fluent speech production, the origin of this breakdown in motor timing was not clearly defined.

To view stuttering as a disorder of speech motor timing not guided by a narrowly defined hypothesis on the neural mechanisms of speech motor control, has direct implications for the interpretation of previous studies into the speech motor symptoms of stuttering. Studies investigating speech motor control in stuttering mainly focussed on peripheral (including physiologic, kinematic and acoustic) characteristics of stuttering and agreed that temporal coordination of respiratory, laryngeal and articulatory movements during the initiation of speech production is particularly difficult for PWS (Perkins *et al*., 1976; Borden *et al.*, 1985; Peters & Boves, 1988; Dembowski & Watson, 1991*a*). However, these observations were not only evident for stuttered speech, but also for fluent speech as well as certain non-speech tasks requiring laryngeal activity (Till, Reich, Dickey & Seiber, 1983; Bakker & Brutten, 1989; Dembowski & Watson, 1991*b*).

The possibility therefore exists that the breakdown in motor timing inherent in stuttering is not restricted to perceptual moments of stuttering but is dependent on the complexity of coordinative respiratory-laryngeal-articulatory movements. Interpretations of peripheral behaviours during speech production in PWS need to be based on all speech samples with reference to the complexity of the required movements. In previous studies, numerous investigators only included so-called fluent speech samples, and used different methods to elicit and identify the fluent utterances (Agnello, Wingate & Wendell, 1974; Zimmermann, 1980*b*; Metz, Samar & Sacco, 1983; Healey & Gutkin, 1984). Such an omission of relevant subject information and flaws in methodology impacts on the interpretation of speech motor features of stuttering, especially if results across studies are compared to formulate a hypothesis on stuttering as a motor speech disorder.

Thus, stuttering as a motor speech disorder is not confined to isolated perceptual disruptions

in the flow of speech (disfluencies). Utterances may be perceived as fluent or disfluent, depending on the extent to which difficulty in motor timing affects PWS's speech production. In other words, perceptual disfluencies may relate to the extent to which asynchronies affect articulatory, respiratory and particularly laryngeal behaviours during the initiation of speech production. Perceptual disfluencies, or surface symptoms in PWS seem to be an elaboration of subtle aberrancies (Martin & Haroldson, 1981). If this holds true, then describing the underlying speech motor variables involved in laryngeal-respiratory-articulatory discoordinations in PWS's speech, should enable the researcher to explain the perceivable speech deviancies. When studying laryngeal behaviour during speech production in PWS using instrumental analysis procedures, it is thus possible that perceptual observations of speech motor behaviours can be associated with the acoustic and physiologic results. For example, excessive expiration, sound prolongations and hard glottal attack as based on perceptual observations (Van Riper, 1982; Shipley & McAfee, 1992; Bloodstein, 1995) could be associated with delayed voice onset times in PWS (Healey & Gutkin, 1984; Borden, *et al*., 1985; Peters & Boves, 1988). However, perceptual descriptions of associative speech motor behaviour in stuttering do not explain the underlying aberrancies in speech motor control of PWS. The breakdown in this control should be investigated by an instrumental study of speech motor coordination. This study will therefore utilise electroglottographic and acoustic data of glottal activity during speech initiation in PWS in order to better describe specific laryngeal coordinatory activities in their speech production.

Despite the fact that definitions of stutter-free speech in previous research has varied, most of these studies have revealed what has been considered to be the primary symptoms of stuttering, namely abnormal coordinations in phonatory, respiratory and articulatory activity (Perkins *et al.*, 1976; Freeman & Ushijima, 1978; Shapiro, 1980; Van Riper, 1982; Baken, McManus & Cavallo, 1983; Healey & Gutkin, 1984; McClean, Goldsmith & Cerf, 1984; Borden *et al*., 1985; Bloodstein, 1987; Caruso, Abbs & Gracco, 1988). Findings such as these resulted in a growing interest in the discoordination of speech production processes and the quest for a detailed analysis of the aberrancies in PWS's speech production by means of instrumental studies; for instance acoustic, physiological, aerodynamic and kinematic studies (Dembowski & Watson, 1991*a*). It is therefore imperative to acknowledge that instrumental procedures have enabled researchers to identify physiologic (subacoustic) irregularities in PWS's speech production, which consequently lead to refreshed and insightful comparisons of PWS's stutter-free

speech with their disfluent speech, as well as PWS's fluent speech with that of normally fluent speakers. In broader terms, the use of instrumental measurements has thus impacted largely on the interpretation of physiological disfluent behaviours within the context of speech motor control, where stuttering has continued to be viewed as a motor speech disorder.

Instrumental studies has thus long proclaimed that phonatory behaviour is one of the prominent components of speech production characterized by aberrancies (Perkins *et al*., 1976; Borden *et al*., 1985; Perkins, 1986; Watson & Alfonso, 1987*b*; Bakker & Brutten, 1989; Peters, Hulstijn & Starkweather, 1989; Dembowski & Watson, 1991*b*). Different perspectives exist on the relation between vocal fold behaviour and stuttering. In this regard Starkweather (1982) has distinguished between two types of hypotheses: firstly, the "weak" vocalization hypothesis, which is said to explain laryngeal stuttering behaviour as being part of the symptomatology of stuttering together with abnormal respiratory and articulatory behaviour; and secondly, the "strong" vocalization hypothesis, which suggests that aberrant laryngeal behaviour is the underlying cause of stuttering and may result in further non-laryngeal stuttering behaviour. Examples of hypotheses depicting the role of vocalization in stuttering can be found in Wingate's (1976) review of fluency evoking conditions, Schwartz's (1974) theory on stuttering as a disorder of the vocal mechanism with reference to hyperexcitability of vocal reflexes, and Perkins' *et al.* (1976) view on discoordination of phonation with respiration and articulation in stuttering.

These hypotheses in part, formed the basis for perceptions regarding aberrant vocal fold activity in stuttering. Although numerous studies have focussed on the area of vocal fold behaviour in stuttering (Starkweather, 1982; Adams *et al*., 1984), existing data fail to support hypotheses stating that aberrant laryngeal behaviour is a causal factor of stuttering (Bloodstein, 1995). The lack of experimental support, however, does not imply that hypotheses, such as *stuttering as a discoordination in phonation with articulation and respiration* (Perkins *et al*., 1976), are to be disregarded, but rather it indicates the need for additional data on laryngeal behaviour and alternative interpretations of previous data.

It is not intended with this study to isolate aberrant laryngeal stuttering events from the intersystem coordination of laryngeal-respiratory-articulatory movements, necessary for the production of fluent speech. Acknowledging only irregularities in glottal activity in

stuttering could cause a distorted overemphasis on its discreet speech symptoms, and might thereby lead to misconceptions about the nature of vocal fold behaviour in stuttering. This is especially pertinent with regard to studies that have attempted to display the possible "role of the larynx" in stuttering (Adams *et al.*, 1984:100). These studies (Schwartz, 1974; Wyke, 1974; Parry, 1994) are characterized by many assumptions surrounding laryngeal behaviour as the causal factor of stuttering, but they lack sufficient evidence. The relative value of such studies for gaining insight into the breakdown in speech motor control underlying laryngeal discoordination during speech production in stuttering is therefore limited.

Hypotheses on stuttering as a breakdown in speech motor control are mainly derived from research on fluency-enhancement conditions such as rhythmic speech, masking, singing, chorus-reading, shadowing and prolonged speech (Wingate, 1976; Van Riper, 1982; Borden *et al.*, 1985; Gow & Ingham, 1992). According to Riley and Ingham (2000) the reduction in moments of stuttering under these conditions could be associated with reduced neurophysiological demands of speech motor control. Many studies on fluency-enhancing conditions indicate that the decrease in disfluencies might be attributed to changes in phonatory gestures (Colcord & Adams, 1979; Ingham, 1984; Stager & Ludlow, 1993). Previous results emphasize the contribution of changes in temporal aspects of laryngeal activity in the modification of stuttering (Colcord & Adams, 1979; Adams & Ramig, 1980; Healey & Adams, 1981; Ingham, 1984; Gow & Ingham, 1992). Stuttering may thus be influenced by modifying the sequencing and timing aspects of laryngeal behaviour during intersystem coordination (Van Riper, 1982; Perkins, 1986; Peters & Boves, 1988) which includes producing gentle vowel onsets, regulating the transglottic pressure and prolonging voicing, as well as a slower or altered speech rate (Stager & Ludlow, 1993). A possible explanation is that these changes in temporal aspects diminish stuttering due to simplification of the complex coordination of the speech production process (Adams & Reis, 1971; Andrews, Howie, Dozsa & Guitar, 1982). Simplification of laryngeal coordination, in particular, has been related directly to the reduction in stuttering (Perkins *et al.*, 1976). Therefore, based on the effects of effective rate, duration, timing and sequencing of complex laryngeal movements during phonatory-articulatory-respiratory interaction in reducing stuttering, these spatiotemporal variables possibly underlie coordinated speech movements and consequently fluency. If so, then a logic assumption would be that irregular changes in rate, duration, timing and sequencing of movements during speech production could result in a

breakdown in intersystem coordination, i.e. stuttering.

Literature on stuttering frequency suggests that motor complexity of the speech response may be a predisposing factor in the occurrence of stuttering (Perkins *et al*., 1976; Till *et al*., 1983; Peters *et al.*, 1989). Motor complexity is thus dependent on the movement requirements of a speech utterance (Perkins *et al*., 1976) that is mainly determined by the phonetic context (Van der Merwe, 1997). Manipulating certain contextual factors of the speech utterance, such as the sound structure (e.g. vowel-consonant *versus* consonant-vowel-consonant), the phonatory features (e.g. voiced consonant *versus* voiceless consonant), or the complexity of laryngeal coordination with articulation (e.g. /b/ *versus* /p/), places different demands on the speech production system. Thus, the degree of interdependency between the physiological activities of the respiratory, laryngeal and articulatory systems is a function of the phonetic context, which means the phonetic context determines the motor complexity of the laryngeal-articulatory-respiratory coordination (Watson & Alfonso, 1987*b*). It follows then that the more complex the coordination of speech production processes becomes, the more likely it is for aberrant laryngeal, respiratory and articulatory activities to occur.

Difficulties that PWS encounter in managing the complexity of temporal sequencing of speech movements are reflected in problems with voice initiation during speech production (Freeman & Ushijima, 1978; Shapiro, 1980; Van Riper, 1982; Borden *et al.*, 1985; Conture, Schwartz & Brewer, 1985; Peters & Hulstijn, 1987). The initiation of phonation is a complex process involving the adduction of the vocal folds, adjustment to numerous laryngeal muscles, and increase of subglottal pressure (Peters & Boves, 1988). The laryngeal, respiratory and articulatory processes must be spatially and temporally adjusted in preparation of voice initiation and then coordinated with one another to produce the targeted acoustic outcome. Difficulty with the initiation of phonation is regarded as a characteristic symptom of stuttering (Peters & Boves, 1988; Bakker & Brutten, 1989). Research has previously found several processes involved in starting phonation in PWS that differ from normally fluent speakers such as aberrant laryngeal muscle activity (Freeman & Ushijima, 1978), a slowness in preparing the vocal folds for phonation (prephonatory adjustment) (Watson & Alfonso, 1983), inappropriate vocal fold positioning (Conture *et al.*, 1985), and mistiming of laryngeal movements in coordination with respiration and articulation (Perkins *et al.*, 1976). Even PWS's perceptually fluent speech

indicates slower initiation of voicing than in normally fluent speakers (Hillman & Gilbert, 1977; Healey & Gutkin, 1984).

Interest in instrumental analysis of laryngeal discoordinations during intersystem coordination in stuttering has led to a number of studies in voice onset time (VOT), laryngeal reaction time (LRT), voice reaction time (VRT), voice initiation time (VIT), speech initiation time (SIT), and electromyography (EMG) involving laryngeal activity during speech production of PWS (Reich, Till & Goldsmith, 1981; Healey & Gutkin, 1984; Starkweather, Franklin & Smigo, 1984; Borden *et al.*, 1985; Peters & Hulstijn, 1987; Peters & Boves, 1988; Bakker & Brutten, 1989) *(see also Starkweather, 1982; Adams et al., 1984; Bloodstein, 1995, for reviews).* From these studies, three factors were subsequently specified as the primary areas of difficulty in PWS's speech: (a) the *initiation* of motor speech behaviour (Starkweather *et al.*, 1984; Peters & Hulstijn, 1987); (b) the *temporal coordination* between phonation, respiration and articulation during speech production (Starkweather, 1982; Borden *et al.*, 1985); and (c) the *duration* of the coordinative phonatory-respiratory-articulatory activities (Healey & Gutkin, 1984; Peters & Boves, 1988). These three factors correlate with the main determinants of fluency identified by Adams (1974), which he further describes as being dependant "upon the correct timing and the prompt smooth initiation and maintenance of air flow and glottal vibration" (Adams, 1974:37). The effects are said to result from synchronisation between subglottic pressure, glottal resistance and supraglottic pressure. It is thus proposed within this study that in order to develop an understanding into the nature of laryngeal behaviour during speech production in stuttering, the focus needs to be based on the three factors mentioned above that have been highlighted by previous research and literature.

Inferences about the physiology underlying PWS's speech initiation, especially the coordination of laryngeal and supralaryngeal articulations, were generally based on only acoustic evaluations of PWS's speech (Adams, 1981; Zebrowski, Conture & Cudahy, 1985; Borden & Armson, 1987). However, many physiologic characteristics of PWS's speech initiation are not perceivable without instrumentation that addresses disordered peripheral physiology directly; for example, irregularities in waveform signals of vocal fold contact, or delayed onset of voicing associated with abnormalities in prephonatory laryngeal movements (Dembowski & Watson, 1991*a*). Acoustic measurements contain information on voice production and the resonance

properties of the vocal tract (Kent, 1993*a*), thus reflecting the acoustic result of the interaction between phonatory, respiratory and articulatory behaviours in speech. When considering the complexity of the physiological speech events (including laryngeal activity) underlying the acoustic signal, it is clear that only a combination of instrumental measurements can give justification to the complexity of the speech production process. Using different instrumental measurements is therefore necessary as evidence of the movement dynamics underlying fluent and disfluent speech in stuttering. Applying different levels of measurement - acoustic and physiologic – can provide quality information on abnormalities in both voice and vocal tract function in PWS. Results from these measurements may subsequently develop a better insight into the underlying speech motor dysfunction of stuttering.

In this study, simultaneous recordings will be made of the electroglottographic (EGG) signal and the acoustic signal. The EGG signal is free from the influence of the resonant frequencies of the supraglottal vocal tract (Borden, Harris & Raphael, 1994) and can be used to make inferences about phonatory and vocal tract dynamics such as subglottal pressure changes. The acoustic signal reflects audible speech production and allows for inferences about supraglottal vocal tract activities. Valuable information about the temporal sequencing of PWS's respiratory, phonatory or articulatory movements during speech initiation may be obtained by analysing the acoustic signal in conjunction with EGG data (Dembowski & Watson, 1991*a*). Analysing the same speech event at different levels of measurement will thus make it possible to reveal the interactions that define coordination and thereby, derive information about physiological abnormalities in PWS's speech.

Based on the common factor among previous theories regarding stuttering as a motor speech disorder, i.e. a disruption in motor timing, and on its core physiologic symptoms, i.e. aberrant phonatory, respiratory and articulatory behaviour, the emphasis in studying stuttering should be on the initiation, timing and duration of laryngeal movements coordinated with the articulatory and respiratory systems. The assumption is therefore made that aberrant laryngeal characteristics in PWS would occur subsequent to failures in the coordination between laryngeal (phonatory) and respiratory behaviour, reflected by laryngeal onset time and laryngeal and articulatory behaviour, reflected by voice onset time (VOT). This study thus specifically investigates the thesis that a stuttering speaker experiences difficulty in initiating coordinated laryngeal

behaviour during speech production.

The coordination between laryngeal and subglottal (aerodynamics and respiratory function) behaviours is reflected by laryngeal onset time. Laryngeal onset time represents the temporal interval between initial closure of the vocal folds prior to release of subglottal pressure and the onset of vocal fold vibration. Laryngeal onset time reflects therefore on the coordination between respiratory and laryngeal adjustments during the preparation period before voice onset. Though, no study published on stuttering has specifically focused on measuring *laryngeal onset time*, laryngeal onset during speech initiation in PWS has been investigated by Bakker and Brutten (1989) and Peters *et al*. (1989). These studies compared laryngeal onset in PWS and normally fluent speakers by measurements of laryngeal adjustment time (Bakker & Brutten, 1989) and the post-laryngeal interval (Peters *et al.*, 1989). These findings along with other studies (Baken *et al.*, 1983; William & Brutten, 1994; Stager, Denman & Ludlow, 1997) indicate that PWS are slower in initiating laryngeal movements than normally fluent speakers, which suggest that PWS have difficulty in coordinating their vocal folds with respiration prior to vocal fold vibration, thus voicing.

Difficulties with respiratory-laryngeal coordination at the onset of speech are suggested to result in less controlled subglottal pressures and a greater flow of expiratory air (Baken *et al.*, 1983; Peters & Boves, 1988; William & Brutten, 1994; Peters & Starkweather, 1990). Instabilities in the control of respiratory-related pressures have been demonstrated to occur before the onset of perceptually fluent speech (Peters & Boves, 1988; Peters, Hietkamp & Boves, 1993). A delay in laryngeal onsets may be a combined effect of a relative lag in the preparatory postural adjustments of the vocal folds prior to phonation and the increase in subglottal pressure needed for vocal fold vibration. Longer latencies of vocal fold vibration may also be ascribed to delays in activating the expiratory air stream (Bloodstein, 1987) due to fixation or increased tension of the respiratory musculature (Watson & Guitar, 1986), or a difficulty in temporal coordinating respiration and phonation for the production of the required laryngeal responses (Perkins *et al.*, 1976). The intersystem coordination between phonation and respiration in PWS may be disturbed by involuntary movements and/or tremor in the laryngeal system (Smith, 1992), a general slowed rate in the preparation of movements (Webster, 1990; Williams & Bishop, 1992), or increased variability in time, pressure and flow of respiratory function during speech

(Denny & Smith, 1997). Studies investigating voice initiation in fluent utterances of PWS, also suggest inappropriate organization of laryngeal and respiratory movements during voice initiation, especially pertaining to the timing and variability of these movements.

The interarticulatory synchronization between articulatory and laryngeal functions is reflected by VOT. VOT is the interval between the release of the articulatory obstruction and the onset of vocal fold vibration (Lisker & Abramson, 1967). This interval represents the speakers' (in)ability to distinguish between voiced and voiceless stop consonants effectively. VOT requires precise coordination between the phonatory and articulatory systems (Abramson, 1977). However, the influence of breathing disturbances and variable subglottal pressures on VOT in PWS (Van Riper, 1982) must also be recognised; particularly where VOT has the potential for providing clues on the nature of speech motor control (Borden *et al.*, 1985). Studies on VOT in stuttering have focussed on the issue of temporal coordination of articulatory and laryngeal behaviour during speech initiation. Together with studies of vowel durations (DiSimoni, 1974; Colcord & Adams, 1979), VOT studies suggested that stuttering adults take longer to initiate speech production than do nonstuttering adults (Agnello, 1975; Hillman & Gilbert, 1977; Shenker & Finn, 1985). Thus, stuttering is generally characterized by slower initiations of movement as opposed to normal speech production (Starkweather, 1982; Van Riper, 1982; Adams *et al*., 1984; Bloodstein, 1995).

Explanations for the slower VOT posit that a failure to coordinate the release of the expiratory airstream with laryngeal movements or a problem with laryngeal posturing prior to onset of voicing may interfere with the initiation of voicing (Prosek, Montgomery, Walden & Schwartz, 1979; Adams *et al.*, 1984; Healey & Gutkin, 1984). Excessive laryngeal or articulatory tension may also interfere with the timing for voice onset. Temporal mismanagement between the different speech systems can result in imprecise sound production such as devoicing of voiced sounds (Dalton & Hardcastle, 1989; Shipley & McAfee, 1992) or aspirated stop sounds (Borden *et al*., 1985). Aspirated sound production refers to a short voiced stop that may be perceived as a long voiceless stop, due to slower voice onset during speech production. Whether this inaccurate sound production is an indication of an intended compensatory technique to sound production (i.e. easy onset, or a reflection of the primary nature of stuttering) may be answered by considering the consistency of the longer VOTs.

Previous VOT investigations have yielded contradictory results mainly due to using speech samples judged acoustically to be free of stuttering (Peters & Boves, 1988). The inconsistency of VOT results may be due to differences in the degree to which subacoustic moments of stuttering (covert blocks) were identified and separated from fluent speech samples. In other words, stuttered speech could have occurred physiologically but not acoustically (Alfonso, Watson & Baer, 1987; De Nil & Abbs, 1990).

Quantitative data reporting aberrancies in laryngeal activity of PWS, exist in abundance (Conture, McCall, & Brewer, 1977; Freeman & Ushijima, 1978; Reich *et al.*, 1981; Borden *et al.*, 1985; Peters & Boves, 1988; Bakker & Brutten, 1989; Dembowski & Watson, 1991*b*). The need is thus not to prove the occurrence of abnormal laryngeal behaviour, but rather to define its nature within a comprehensive, explanatory framework of speech motor control. In the absence of a theoretically sound model of speech motor control, previous interpretations of findings by researchers on deviant speech motor behaviour in stuttering have been fragmented. As a result, it can be expected that previous research would have failed to depict the precise nature of laryngeal behaviour in stuttering.

In order to address queries on deviancies in timing control and coordination of laryngeal behaviour in stuttering (Adams, 1974; Perkins *et al.*, 1976; Perkins, 1986) which are supposedly associated with a breakdown of motor programming (Van der Merwe, 1997), it is imperative to work from a theoretical framework based on normal speech sensorimotor control. This will allow for the identification and explanation of irregularities in PWS's speech motor behaviour (including laryngeal aberrancies) more comprehensively, thereby enabling a better definition of the nature of this breakdown in speech motor control. Thus, overcoming barriers to understanding the nature of stuttering (including laryngeal behaviour) depends on the knowledge of the neuromotor control underlying normal speech production (McClean, 1990).

Until recently motor speech disorders were defined as "disorders of speech resulting from neurologic impairment affecting the motor programming or neuromuscular execution of speech" (Duffy, 1995:5). Definitions such as Duffy's (1995) were based upon the traditional three-level speech production model for neurogenic communication disorders that only differentiated

among the classic neurogenic pathologies, i.e. aphasia, apraxia and dysarthria. According to this model aphasia was classified as a linguistic disorder, apraxia as a disorder on the planning-programming level and dysarthria was a disorder of the execution level. The three-level speech production model did not accommodate classification of other motor speech disorders such as stuttering and therefore limited our understanding of the nature of these disorders. Current development in the field of motor speech disorders supports the need for a coherent model of speech motor control (Ballard, Granier & Robin, 2000) in order to "put diagnosis, differential diagnosis, and management on a sound theoretical basis" (Van der Merwe, 1997:1). A more comprehensive theoretical framework of speech sensorimotor control that redefines the above-mentioned three levels into a four-level framework, is proposed by Van der Merwe (1997). Van der Merwe's (1997) framework differentiates between linguistic-symbolic planning, motor planning, motor programming and motor execution based on neurophysiological grounds.

According to Van der Merwe's (1997) model, aphasia is associated with a linguistic-symbolic problem, a motor planning problem may manifest as verbal apraxia, and problems in motor programming and motor execution are associated with different kinds of dysarthria. The motor programming level as depicted by Van der Merwe (1997), entails the selection and sequencing of motor programs for movements of the muscles of articulatory structures (including the vocal folds). Specifications of spatiotemporal and force dimensions such as muscle tone, direction, rate, range of movement and the mechanical stiffness of joints (postural fixation) required for coordinated movement patterns, are determined by the motor program (Van der Merwe, 1997). Thus given that the specifications for a particular level predict the motor speech symptoms in the presence of a breakdown of the indicated level then symptoms resulting from a motor programming disorder would probably indicate defects in speech rate, sound distortions and/or problems in the initiation of movement (Van der Merwe, 1997). Various research findings thus continue to support the notion that speech motor characteristics associated with motor programming disorders are evident in PWS's speech, e.g. abnormalities in laryngeal and articulatory muscle tone (Freeman & Ushijima, 1978), involuntary movements, excessive levels of laryngeal muscle activity (Freeman & Ushijima, 1978; Shapiro, 1980), slow initiation of laryngeal movements (Adams, 1985; Peters & Hulstijn, 1987; Bakker & Brutten, 1989), tremor-like movements of the face, tongue, lip, jaw, larynx and respiratory system (Zimmermann, 1980*a*; McClean *et al.*, 1984; Borden *et al.*, 1985; Smith, 1989), postural fixations of the

glottis (Conture *et al*., 1985), and a lack of coordination between articulation and onset of phonation (Hutchinson & Watkin, 1976; Healey & Gutkin, 1984; Borden *et al.*, 1985).

In her four-level framework for the characterization of pathological speech sensorimotor control, Van der Merwe (1997) related differential neurophysiological involvement of the various neural structures to neuromotor function. The assumption could be made that the affected level of neuromotor function should indicate the neural structure/s involved in a motor speech disorder such as stuttering. Hence, if stuttering is to be regarded as a motor programming disorder, the neural structures involved in stuttering should correspond with those associated with the motor programming as defined by Van der Merwe (1997). According to this proposed model, the neural areas involved in motor programming comprise the basal ganglia, the lateral cerebellum, the supplementary motor area (SMA), the motor cortex and the fronto-limbic system. Comparisons between atypical activation of neural structures in PWS, as indicated by research (Wood, Stump, McKeehan, Sheldon & Proctor, 1980; Finitzo, Pool, Freeman, Devous & Watson, 1991; Wu, Maguire, Riley, Fallon, LaCasse, Chin, Klein, Tang, Cadwell & Lottenberg, 1995; Fox, Ingham, Ingham, Zamarripa, Xiong & Lancaster, 2000), and those structures related to the different levels of speech motor control including planning, programming and execution (Van der Merwe, 1997) mostly confirm a dysfunction on the level of motor programming in PWS. Furthermore, Lebrun (1998) states that comparisons between speech characteristics of PWS and other motor speech disorders that indicate similar involvement of neural areas may be one of the main sources of information regarding the nature of stuttering.

A need for further investigation of the salient characteristic, abnormal laryngeal behaviour, of stuttering in order to define the nature of this behaviour has been thoroughly emphasized. An accurate description of the laryngeal characteristics of stuttering might provide a clearer picture of possible fundamental disparities that exist between speakers who stutter and normally fluent speakers, especially as reflected in speech initiation. Such descriptions will ultimately lead to a better understanding of the nature of breakdown in speech motor control underlying the symptoms of stuttering. This study therefore aims to provide information on the nature of vocal fold (laryngeal) behaviour during the initiation of speech production in PWS. This is achieved by defining: (a) characteristic vocal fold behaviour in laryngeal-respiratory-articulatory coordination during the preparatory period prior to speech production; (b) the temporal

coordination between laryngeal activity and respiratory activity, as well as the coordination between voicing and articulatory activities during speech initiation, based on the premise that a breakdown in coordination between the speech production systems can be measured as abnormalities in laryngeal onset time and VOT, respectively; and (c) the coordination of laryngeal behaviour with respiration and articulation as a function of the motor demands placed on the speech production system by a phonetic context. The relation between aberrant laryngeal behaviour and PWS's perceptually fluent speech will be indicated. The effect of stuttering severity on the laryngeal-respiratory-articulatory coordination during speech initiation will also be considered, since stuttering severity may reflect the extent of speech motor involvement (Watson & Alfonso, 1982).

Moreover, in order to develop a more comprehensive perspective of aberrant laryngeal behaviour in stuttering, the nature of its characteristics will be discussed with reference to a theoretical framework for the characterization of pathological speech sensorimotor control as proposed by Van der Merwe's (1997). This data will thus serve to contribute to a more accurate description of the nature of PWS's speech characteristics, which in turn will shed light on pathological speech motor control in stuttering.

The aim of this chapter has thence been to present and discuss the arguments underlying the rationale of the study, to define and discuss the terminology used in this study, and to provide an overview of the division and content of the subsequent chapters in this research project.

1.3 DEFINITION OF TERMS

In order to avoid confusion, misinterpretation and misunderstanding of the terms used in this study, it is necessary to provide a comprehensive concept analysis of relevant terms and definitions used.

1.3.1 Electroglottographic Analysis

Electroglottography (EGG) is a non-invasive method investigating laryngeal behaviour and conveys essential information about glottal activity. The electroglottogram is a correlate of

vocal fold contact area (Orlikoff, 1992). Data on vocal fold adjustments during speech initiation or on intersystem coordinative characteristics of prephonatory speech behaviour can be obtained by analysing the EGG waveform.

1.3.2 Acoustic Analysis

The acoustic analysis of speech contains information on voice production and the resonance properties of the vocal tract (Kent, 1993*a*). Spectral analyses can be used to obtain data on the acoustic properties of consonant and vowel productions. It reveals whether a speaker is capable of making acoustic contrasts that are needed for intelligible speech (Kent, 1993*a*). Information on acoustically detected vocal fold behaviour (in coordination with articulation and respiration) for consonant and vowel productions is obtained by the sound signal.

1.3.3 Glottal activity

Laryngeal activity is a function of laryngeal biomechanics. Glottal activity refers to laryngeal function (Borden & Harris, 1984). The biomechanics of the larynx is considered to be a mechanical interaction between the laryngeal musculature, vocal fold stand, subglottal pressure and glottal impedance (Titze, 1993). Those mechanical activities need to be well coordinated to ensure accurate and smooth laryngeal function during phonation. Thus, a defined temporal relationship exists between presetting of the phonatory musculature and the continuous readjustment of laryngeal muscle tone (Wyke & Kirchner, 1976). Any breakdown in the mechanical interaction prior to, or during phonation, can result in irregular, abnormal laryngeal activity.

Laryngeal activity *during speech initiation* includes the prephonatory phase before the audible vocalizations commence. Before the physiological voice onset (vocal fold vibrations) there is a prephonatory closure of ±170 ms that is followed by separation of the vocal folds without vibrating (Köster, Marx, Gemmar, Hess & Künzel, 1999). The onset or the prephonatory closure may differ in length depending on factors such as phonetic context and coordination of vocal fold adjustments with respiratory and articulatory activities.

1.3.4 Speech initiation

Speech initiation refers to the onset of speech production. The onset of physiological speech activity includes the prephonatory period before audible speech production. Speech initiation involves a coordinated interaction between three speech motor subsystems: the respiratory system which produces a slowly varying tracheal (subglottic) air pressure, a laryngeal system that includes valving actions to manage glottal flow resistance and a supraglottal vocal tract (articulatory system), that shapes the spectrum of the glottal flow pulses into an acoustic signal (Rothenberg, 1983). The spatiotemporal relationships between the speech motor systems need to be specified to enable smooth coordinated movements during speech initiation (Moore, 2004).

1.3.5 Stuttering

Stuttering is an involuntary breakdown in the ongoing fluency of speech, thus, an inability to maintain the connected rhythms of speech (Van Riper, 1982). It is important to point out that a disfluency in speech does not necessarily indicate stuttering. Overt stuttering refers to audible disruptions (repetitions, prolongations and/or blockages) in the utterance of speech elements, though perceptually fluent speech of PWS also indicates deviant motor behaviour. With regard to overt disfluencies, Perkins (1983*a*) stated that a normal disfluency is a result of linguistic uncertainty, a characteristic shared by all speakers. A stuttered disfluency presumably is a motor speech defect and is considered abnormal.

According to the standard definition of stuttering proposed by Wingate (1964:498), the following descriptions of stuttering behaviour can be added: “Sometimes the disruptions are accompanied by accessory activities involving the speech apparatus, related or unrelated body structures, or stereotyped speech utterances. These activities give the appearance of being speech-related struggle. Also, there are indications of the presence of an emotional state, ranging from excitement or tension to fear. The immediate source of stuttering is some incoordination expressed in the peripheral speech mechanism; the ultimate cause may be complex or compound.”

Stuttering as described in this study refers to developmental stuttering. Developmental

stuttering is identified by disfluencies that begin during early childhood, and which originate from an acquired neurologic injury (neurogenic stuttering). The cause of developmental stuttering is unknown, but does not exclude the possibility of neurologic dysfunction. Though many similarities exist between developmental stuttering and neurogenic (or acquired) stuttering (Rosenbek, 1984), these two conditions are not identical.

1.4 ORGANIZATION OF CHAPTERS

1.4.1 Chapter 1

This chapter serves to provide an orientation to the field of study and presents the rationale and motivation for the study. The unresolved factors in the investigation of stuttering as a motor speech disorder, namely timing, initiation and duration of laryngeal behaviour during intersystem coordination (including respiration, articulation and phonation) are highlighted. The foundation laid by vocalization theories in developing a hypothesis on the relationship between stuttering and laryngeal behaviour is mentioned. In addition, consideration is given to aberrant laryngeal activity in stuttering as a salient characteristic of deviant speech motor control. Furthermore, laryngeal discoordinations are regarded as the result of a breakdown in the timing control of coordinative movement of phonation, respiration and articulation during speech production.

Moreover, failures in previous studies to differentiate between perceptual and physiologic disfluencies, the need to use an acoustic and physiological instrumentation and careful selection of the speech material are discussed. Suggestions to overcome these difficulties in the current study are also made. The primary characteristic of stuttering, namely difficulty in voice initiation, is emphasized which further underscores the importance of measurements of laryngeal activity during speech initiation. Understanding the nature of vocal fold activity in stuttering within the context of motor programming as depicted by the four-level model for the characterization of normal and pathological speech sensorimotor control (Van der Merwe, 1997) is underscored. A discussion of relevant terms and concepts are provided, as well as a brief review of the chapters of the study.

1.4.2 Chapter 2

Perspectives on stuttering as a breakdown in speech motor control are outlined first. These views have emphasized two major speech motor characteristics in stuttering, i.e. difficulty in initiation of muscle movements, and a breakdown in the temporal coordination of successive movements. Such speculations about the nature of stuttering form the basis for theories on the core physiological symptoms of stuttering (particularly aberrant laryngeal behaviour). Secondly, theories focusing on the relation between stuttering and laryngeal activity will be reviewed. These theories can further be divided into two approaches (Starkweather, 1982): (a) abnormal laryngeal behaviour forms part of the symptomatology of stuttering together with articulation and respiration; and (b) aberrancies in laryngeal behaviour causes a breakdown in articulatory and respiratory activities. Most studies seem to support the former approach.

In order to explore this line of thought, aberrant laryngeal activity in stuttering is discussed, where laryngeal behaviour is defined with regard to its coordination with respiration and articulation during normal speech production, as well as its characteristics in the presence of a disruption of coordination in the speech of PWS. Physiologic and acoustic studies investigating these laryngeal activities in PWS are tabulated and discussed. The majority of these studies have indicated a disruption of the initiation of laryngeal behaviour in stuttering, however, the role of laryngeal behaviour in a breakdown of speech motor control is still unclear. This problem can be addressed by describing the nature of laryngeal characteristics in PWS within the context of a sound theoretical framework for the characterization of normal and pathological speech sensorimotor control as proposed by Van der Merwe (1997). The four levels of speech sensorimotor control are subsequently defined with emphasis on the level of motor programming, since stuttering is associated with a breakdown in motor programming (Van der Merwe, 1997).

Comparisons between the neural substrates associated with the different levels of speech sensorimotor control as depicted by Van der Merwe's model and those prominently involved in stuttering, illustrate similarities between stuttering and a speech motor disorder on the level of motor programming. Parallels between symptoms of stuttering and those of a motor speech disorder associated with a defect in motor programming (i.e. dysarthria due to a lesion in the basal ganglia and associated areas), strengthen the possibility that stuttering is related to a dysfunction in motor programming. Next, the salient characteristics of motor programming

disorders according to Van der Merwe (1997) are related to characteristics of laryngeal behaviour in stuttering.

1.4.3 Chapter 3

The research methodology of the empirical research is described in terms of the research aims, subject selection criteria and procedures, research design, measurement instruments and speech material, data collection procedure and finally the data analysis and processing procedures employed in this study.

1.4.4 Chapter 4

The results of the study are presented according to the various sub-aims. The period of speech initiation in PWS is studied with specific focus on the physiologic vocal fold activity and the acoustically detected vocal fold activity. Descriptive statistics will be used to show the effect of three phonetic contexts with varying voicing features (i.e. *vowel*-consonant, *voiced consonant*-vowel-consonant and *voiceless consonant*-vowel-consonant) on the initiation of speech in PWS. Comparisons are made between the group of PWS and the group of normally fluent speakers (between-group comparisons). Within-group comparisons are made for the individuals who stutter.

1.4.5 Chapter 5

The results in Chapter 4 are discussed with reference to the research aims. The results of the current study are compared to results of other studies and explained with reference to the underlying nature of stuttering. Results are also explained using Van der Merwe's (1997) theoretical framework for the characterization of pathological speech sensorimotor control.

1.4.6 Chapter 6

General conclusions are provided as well as an evaluation of the research methodology. The clinical implications of the study are discussed and recommendations for future research are

suggested.

1.5 SUMMARY OF CHAPTER 1

In this chapter, the rationale and motivation for the study are presented. The importance of studying laryngeal activity during speech initiation in PWS (as part of an intersystemic coordination dysfunction between respiratory and articulatory behaviour), is motivated by examining the contribution of pertinent research studies. Shortcomings in these studies and the need for further research is emphasized by high-lighting that limited research has focused on the analysis of temporal coordination and duration of laryngeal behaviour in stuttering. Finally, the terminology is explained and the chapters in this study are briefly outlined.

CHAPTER 2: LARYNGEAL BEHAVIOUR DURING SPEECH INITIATION IN STUTTERING

2.1 INTRODUCTION

The purpose of the current study is to gain insight into the laryngeal (or vocal fold) behaviour of persons who stutter (PWS) during speech initiation. Although articulation and respiration in stuttering indicate spatial and temporal discoordinations, many researchers believe that the onset of phonation is the key component of stuttering behaviour (Starkweather, 1982; Till *et al.*, 1983; Borden *et al.*, 1985; Bloodstein, 1987; Watson & Alfonso, 1987*b*; Dembowski & Watson, 1991*b*; the National Centre for Stuttering (*NCS*), 2002). This study will describe one of the most salient features of stuttering, namely abnormal laryngeal activity (Perkins *et al.*, 1976; Freeman & Ushijima, 1978; Starkweather, 1982; Bakker & Brutten, 1989), in a broader context as a disorder of speech motor control. The issue to be addressed is the underlying motor variables in vocal fold control responsible for the discoordination in phonation, which is, according to Perkins (1986), a causal factor in stuttering.

Motor control in stuttering relates to a number of significant questions concerning laryngeal behaviour: What are the possible causes for the abnormal voicing features, such as longer voice reaction times, in perceptually fluent speech? Are there certain speech motor characteristics (e.g. difficulty in voice initiation) other than the surface symptoms of stuttering, which distinctly and uniquely differentiate persons who stutter from normally fluent speakers? Are the disrupted laryngeal events in stuttering isolated from other speech behaviour? Is stuttering indicative of a disruption in the execution of laryngeal movements with respiration and articulation, or is it due to a dysfunction in the programming of these movements? Possible answers to these questions may be disclosed when contemplating stuttering within the context of a comprehensive theoretical framework for the characterization of pathological speech sensorimotor control.

Many theories on stuttering that focussed on speech motor control depicted a failure in motor timing control as the cause of this disorder (MacKay, 1982; Van Riper, 1982; Kent, 1984; MacKay & MacDonald, 1984; Peters & Hulstijn, 1987; Peters, Hulstijn & Starkweather, 1991; Hulstijn & Van Lieshout, 1998). However, the nature of this failure in speech motor control has been difficult to identify. The problem is further compounded by the lack of a

generally accepted and comprehensive account of speech motor control per se (Kent, 1998). Viewing stuttering as a disorder in motor timing of speech without identifying the nature of the underlying timing error, suggests that stuttering can be the result of any disruption in the rhythmic flow of speech. Such a general hypothesis does not allow for a differential diagnosis of stuttering from other speech motor disorders with dysfluencies (e.g. apraxia or dysarthria). These shortcomings emphasize the need to explain the nature of the breakdown in temporal programming in stuttering within a comprehensive model of speech motor control. A model of speech sensorimotor control that has received recognition from researchers in the field of motor speech disorders, is Van der Merwe's (1997) theoretical framework for the characterization of pathological speech sensorimotor control. This four-level framework differentiates between linguistic-symbolic planning (a non-motor phase), motor planning, motor programming and motor execution. According to Van der Merwe (1997:19) the core symptoms of stuttering "seem to fit in well with a disorder in programming of speech rate and repeated initiation." Van der Merwe (1997) has proposed that stuttering might be understood at the motor programming level. A comparison between the nature of a breakdown in motor programming as depicted in this model, and the physiological characteristics of PWS such as abnormal laryngeal activity (Adams *et al*., 1984; Bakker & Brutten, 1989; Peters *et al*., 1989; Dembowski & Watson, 1991*b*), will justify the perspective taken in this study, namely, to view laryngeal discoordinations within the paradigm of motor programming.

The following four aspects will be discussed in this chapter:

Firstly, previous theoretical approaches to speech motor control in stuttering will be reviewed. The importance of these approaches is two-fold, i.e.: (a) Existing theories provide us with notions about the inherent problems of stuttering, such as difficulty in initiation of motor programs (Van Riper, 1982), that form the origin of hypotheses about the relation between stuttering and its speech symptoms (including laryngeal aberrancies); (b) Shortfalls in theories that fail to explain the nature of the salient symptoms of stuttering, emphasize the need to work from a theoretically sound model of speech sensorimotor control which depicts normal as well as pathological speech production. A theoretical basis of speech sensorimotor control for normal and deviant speech production will enable one "to discover aspects of motor performance that distinguish the breakdowns *(in speech motor control)* from successful operation of the system" (Denny & Smith, 1992:1228).

Next, when proposing that stuttering is a disorder of speech motor control (Kent, 2000), the core symptom of stuttering, namely abnormal laryngeal behaviour (Starkweather, 1982), needs to be described within the context of this assumption. Some of the pertinent historical accounts of a breakdown in speech motor control, as they relate to characteristic laryngeal behaviour in stuttering, will be reviewed. This is followed by queries regarding the failure in speech motor control of PWS explained by these theories of laryngeal behaviour in stuttering.

Thirdly, the nature of laryngeal behaviour in stuttering is described with reference to its coordination with respiration and articulation. The breakdown in laryngeal coordination with respiration and articulation associated with stuttering is discussed against the background of the physiological aspects underlying fluent speech production. Physiological and acoustic studies that have focussed on the discoordination of laryngeal behaviour with respiratory and articulatory activities in PWS are reviewed and the need for further research is highlighted.

Finally, since control of laryngeal behaviour during respiration, phonation and articulation has long been a focus in research on speech motor dynamics in stuttering, the objective will be to interpret these behaviours within a model of speech motor control. A theoretical model of speech motor control that recent literature on speech motor disorders (Ballard *et al.*, 2000; Nijland, Maassen & Van der Meulen, 2003) refers to, is the four-level framework for the characterization of pathological speech sensorimotor control (Van der Merwe, 1997). Understanding the nature of the fundamental levels of speech sensorimotor control as depicted by Van der Merwe (1997), is essential to distinguish a breakdown in speech motor performance in PWS from that of other speech motor disorders. According to the proposed framework, stuttering is viewed as a disorder of motor programming (Van der Merwe, 1997). To develop the perspective of stuttering as a disruption at the level of motor programming: (a) specific features of motor parameters involved in stuttering (including laryngeal behaviour), such as a dysfunction in temporal coordination, initiation and duration of movements that indicate a breakdown in motor programming, will be reviewed; (b) a comparison will be drawn between speech motor characteristics of stuttering and those of speech motor disorders representing a dysfunction at the level of motor programming such as hyperkinetic dysarthria; (c) and, since knowledge of the neurological involvement in stuttering serves as a theoretical basis for the interpretation of pathological speech motor behaviours (Wood *et al.*, 1980; Finitzo *et al.*, 1991; Wu *et al.*, 1995; Fox *et al.*, 2000), the neural substrates involved in “normal” motor programming will be compared with results of brain imaging studies in PWS.

This discussion will provide a rationale for the study of laryngeal coordination with respiration and articulation during speech initiation in PWS and normally fluent speakers.

2.2 THEORETICAL PERSPECTIVES ON STUTTERING AS A BREAKDOWN IN SPEECH MOTOR CONTROL

A prominent approach to stuttering research concerns the involvement of speech motor control abilities in stuttering. The major shortcoming in previous speech motor conceptions about the nature of stuttering is the fragmentation of the proposed constitutional factors (e.g. abnormal sensorimotor feedback, mistiming of successive movements, neural dysfunction) underlying the breakdown in speech motor control. As a result, different theories on speech motor control in stuttering have originated that are, in fact, interrelated.

2.2.1 Stuttering as a Disorder of Movement

Zimmermann's (1980*b*) preliminary model of *stuttering as a disorder of movement* attributed disfluent behaviours to asynchronies in timing of respiration, phonation and articulation. According to his viewpoint speech motor execution generates a large amount of afferent activity. Afferent activity feeds back into the motor system and may influence speech motor control directly. Parameters such as displacement, velocity and duration of movement, and temporal and spatial coordinations of the articulatory movements determine the effect of the afferent feedback on neurophysiological processes. Although Zimmermann (1980*c*) mainly referred to the articulatory system, he implied that the laryngeal and respiratory systems are also affected by these parameters. Zimmermann (1980*c*) stated that speech structures normally function within a certain range of movement parameters and interact within a range of spatial and temporal values. Stuttering may occur when brainstem reflexes are bombarded with highly irregular feedback from PWS's variable *articulatory* movements, thus when the ranges of movement parameters are exceeded. The aberrant activation of brainstem pathways throws the articulatory system out of balance and a breakdown in speech behaviour occurs. This breakdown supposedly manifests as oscillations or static positioning of speech structures; in other words, stuttering behaviours are evident.

Thus, Zimmermann (1980*c*) claims that impaired motor (kinematic) feedback best accounts for the peripheral physiological behaviour of stuttering. From this perspective stuttering interruptions are viewed as physiologic motor speech events, which may manifest as

superficial speech symptoms such as prolongations or repetitions. Descriptive physiological data gathered from PWS's fluent and stuttered speech, such as proposed by Zimmermann (1980*c*), could contribute to speculations about neural mechanisms responsible for stuttered speech (Starkweather, Armson & Amster, 1987).

2.2.2 Stuttering as a Disorder of Timing

Van Riper (1982) and Kent (1984) amongst others have proposed that stuttering occurs as a result of inappropriate temporal patterns or sequences. Though both perspectives are based on the importance of preparation of movement sequences for fluency, the two viewpoints focus on different aspects of stuttering. Van Riper (1982) explained how difficulties in motor programming could be reflected in the speech symptoms of stuttering (respiration, articulation and phonation). Van Riper (1982) also described factors important for the stability of motor sequence patterns in normal speech production and considered these factors as possible indicators of a disruption of motor sequences in PWS. While Van Riper's (1982) view focused on the influence of temporal discoordination on peripheral motor activities, Kent (1984) elaborated on the cause of these mistimings in stuttering on a neurologic level. He associated aberrant speech motor activity with linguistic functions in PWS. Thus, Kent's (1984) perspective of temporal programming did not separate motor function from linguistic function. On that account, the two different approaches to stuttering as a temporal disruption are discussed separately.

2.2.2.1 Stuttering as a Breakdown in Timing Control of Speech Movements

Van Riper (1982:415) defined stuttering as "a temporal disruption of the simultaneous and successive programming of muscular movements required to produce one of the word's integrated sounds, or to emit one of its syllables appropriately or to accomplish the precise linking of sounds and syllables that constitutes its motor pattern." Van Riper (1982) summarized several sources of evidence to substantiate that the principal difficulty of stuttering lies in a disruption in temporal coordination of motor sequences of speech production. For example, asynchronies or delays have been observed in all of the speech muscles of PWS, not only during overt stuttering behaviour, but also during their perceptually fluent speech (Shapiro, 1980; Zimmermann, 1980*b*). Thus, the temporal coordination of voice, respiration and articulation is apparently disrupted during fluent as well as non-fluent utterances of PWS. Van Riper (1982) based his theory on the underlying temporary

changes that account for the fluency evoking effect of whispered and pantomimed speech in PWS. For example, slower speech rate and a high degree of conscious articulation may permit enhanced synchronization between speech production processes, or the absence of voice/airflow coordination in whispered speech results in simplified coordination, hence less motor sequences per time unit. Van Riper (1982) also commented on the strong effect rhythm has on stuttering, where fluency is enhanced when an individual who stutters speaks in time with a metronome or any other rhythmic stimulus (including visual, auditory or tactile) provided the rhythm is not abnormally fast.

According to Van Riper (1982) the successive programming* of muscular movements required for a specific utterance (also called motor patterning) is dependent on three principles: practice, the complexity of coordination, and the feedback systems used to monitor the movements. Firstly, the more often a motor pattern for a certain word or syllable or sound has been used correctly, the more stable it will be and less likely it will be disrupted. Conversely, if a word/syllable/sound has been stuttered on in the past, a disrupted motor pattern for that speech unit might have been learned (stabilized) and used in future. Van Riper's (1982) viewpoint on this aspect of motor learning, correlates with Kehoe's (1998) concept of maladaptive motor skills in stuttering which is based on Fitts' (1964, in Kehoe, 1998) three stages of motor learning. Also, the more complex the motor sequence or coordinative pattern of a speech unit, the more unstable it tends to be and might result in a disrupted motor pattern, i.e. stuttered speech. The last factor focuses on the role of auditory and proprioceptive feedback in speech. Van Riper (1982) poses that motor patterns for speech are initially monitored by means of auditory and proprioceptive feedback. Once the motor pattern has stabilized, speech can be monitored by proprioception alone. The motor pattern is regarded as stabilized if the auditory system doesn't feed back error signals anymore, thus the speaker is able to hear *what* he says as opposed to *how* he is saying it. The feedback principle states that "the more stable the motor sequence of a word, the more feedback distortion it can undergo before it becomes disrupted" (Van Riper, 1982:431). The implication that auditory feedback distortion has on PWS's speech is still speculative, but, as emphasized by Van Riper (1982), there is enough evidence indicating that auditory feedback plays an essential role in stuttering.

* According to Van Riper (1982) "*programming*" refers to the presetting or preparation of sequential motor acts.

2.2.2.2 Stuttering as a Temporal Programming Disorder

Kent (1984:296) presented a theoretical perspective of *stuttering as a temporal programming disorder* in which he posed that PWS and normally fluent speakers differ in their capacity "to generate fine temporal programs that are necessary for motor regulation, for efficient auditory perception, and for language expression". According to Kent (1984:283) a temporal program can be defined as "a time plan or pattern useful for both perceptual processing of sequential patterns and for the regulation of motor sequences". These temporal programs are seen as time structures of action which specify the timing and nature of feedback information most suitable to accomplish motor goals.

Kent (1984) also included the influence of genetic factors on stuttering in his perspective. According to Kent (1984) anomalous hemispheric functions could be linked to genetic factors. The inability to create proper timing programs may stem from the fact that an individual who stutters' left hemisphere is not as developed as his right hemisphere (Kent, 1984). Since the left hemisphere is more adept at generating temporal programs for fine motor control of verbal output, a stuttering speaker may be lacking the skill of timing regulation at the speed or frequency needed for speech. The central timing device that regulates the left hemisphere aspects of speech production, must also integrate timing between rapid left hemisphere segments and the slower prosodic elements from the right hemisphere. Posing that the right hemisphere is involved with expressing emotion, it was thought that stuttering may occur due to inefficient timing regulation of linguistic and paralinguistic speech processes or that speech processes are vulnerable to interference by right hemispheric activity due to increased emotion. Thus, stuttering may be associated with a breakdown in the simultaneous processing of the different temporal ranges of information from the left and right hemispheres. Perkins, Kent and Curlee's (1991) theory of neuropsycholinguistic function in stuttering elaborated on Kent's (1984) assumption by hypothesising that speech involves linguistic and paralinguistic (such as expression of emotion and communicative intent) components. These are processed separately by different neural systems and converge on a common output system. Stuttering occurs under abnormal time pressure[*], when these components are not integrated in synchrony (Perkins *et al.*, 1991).

[*] Time pressure as defined by Perkins *et al.* (1991), is the pressure to begin, continue or accelerate an utterance.

2.2.3 Conclusion: Theories on Speech Motor Control in Stuttering

The foregoing theoretical assumptions indicate the presence of some specific breakdown in the coordination and temporal control necessary for fluent speech production in PWS's speech production. A disruption in motor timing seems therefore to be inherent to stuttering and can be expected to affect all levels of speech production (Adams, 1974; Van Riper, 1982; Perkins, 1986). This suggests that the phonatory mistimings characteristic of PWS's speech could affect the coordination of respiratory and articulatory movements (Perkins *et al.*, 1976). However, the underlying cause of the timing deviancy in the speech of an individual who stutters is uncertain. In order to understand stuttering and its characteristics from a speech motor control perspective, it is important to: (a) define the effects of motor speech variables such as timing on stuttering behaviour (including abnormal laryngeal activity); (b) differentiate stuttering from other motor speech disorders with dysfluencies, e.g. aphasia or apraxia.

In reviewing these perceptions on stuttering being a breakdown in speech motor control, several shortfalls have been identified. Firstly, the relation between phonologic (non-motor) functions and motor functions in speech motor control is indistinct (Kent, 2000), resulting in misconceptions about the role of linguistic and motor functions in the speech production of PWS. Secondly, the nature of the speech motor characteristics of stuttering is not adequately defined within the context of speech motor control, due to a lack of clear differentiation between levels of motor function. Also, many perceptions regarding a failure in speech motor control are primarily based upon peripheral physiologic behaviour, which prohibit direct inferences about causal factors based on a neurophysiologic level. Another shortfall is that views such as Zimmermann's (1980*c*) failed to account for perceptual (surface) symptoms. The underlying motor control defects should provide an explanation for the surface symptoms of stuttering, since stuttering is perceptually identified but thought to originate from underlying disturbances in speech motor control.

Perspectives on stuttering as a disorder of speech motor control have led to questions surrounding the relation between speech motor control and stuttering symptoms. Aberrant laryngeal behaviour, which is regarded as the core peripheral symptom of stuttering (Adams *et al.*, 1984; Kehoe, 1998), received special interest.

2.3 THE ROLE OF LARYNGEAL BEHAVIOUR IN STUTTERING

The relationship between laryngeal behaviour and stuttering has long been proclaimed (Wingate, 1969; Adams, 1974; Schwartz, 1974; Perkins *et al.*, 1976; Starkweather, 1982; Adams *et al*., 1984; Pool, Freeman & Finitzo, 1987; Bloodstein, 1995; Kehoe, 1998). "This attraction announces in no uncertain terms the conviction of investigators that somehow the larynx plays a key role in stuttering, presumable more fundamental than articulation and respiration" (Perkins, 1986:82). However, the implications of aberrant laryngeal function in stuttering are far from clear, since any vocal response (such as throat clearing) is dependent on the interrelated activity of the respiratory, laryngeal and oral musculature and not an exclusive function of the larynx (Bloodstein, 1995).

Starkweather (1982) described two types of vocalization hypotheses concerning the relation between stuttering and laryngeal behaviour. Firstly, the "weak" vocalization hypothesis which explains laryngeal stuttering behaviour as a symptom of stuttering together with articulatory and respiratory stuttering behaviour (Starkweather, 1982). Thus, the entire vocal tract is thought to be involved in stuttering. The second hypothesis is the "stronger" vocalization hypothesis which suggests that laryngeal behaviour is primary to stuttering and may precipitate other non-laryngeal stuttering behaviour (Starkweather, 1982). It does not, however, imply that laryngeal behaviour is directly related to the cause of stuttering (Starkweather, 1982). Nevertheless, whether stuttering causes abnormalities in laryngeal behaviour or whether stuttering is influenced by abnormal laryngeal behaviour is still a controversial issue in stuttering literature.

2.3.1 The Relationship between Stuttering and Laryngeal Behaviour: Vocalization Hypotheses of Stuttering

Different perspectives on the relationship between stuttering and laryngeal behaviour exist. Examples of such vocalization hypotheses are those offered by Schwartz (1974), Wingate (1976), Perkins *et al.* (1976) and the *NCS* (2002). These perspectives help to enhance our understanding of laryngeal behaviour in context of speech motor control. Moreover, the vocalization hypotheses failed to establish a cause-effect relationship between stuttering and laryngeal behaviour. This emphasized the need to explain distinctive laryngeal symptoms in stuttering within a framework of speech motor control.

2.3.1.1 Stuttering as a Disorder in Voicing

2.3.1.1.1 The Relationship between Stuttering and Voicing Conditions: Wingate's Review of the Conditions that Enhance Fluency

Wingate (1986) posed that stuttering is a problem in making phonetic transitions, especially the voiced-voiceless transitions. Disfluent speech is described as a failure to actualize the vowel. In constructing a perspective on phonatory aberrancies in stuttering, Wingate (1976) reviewed conditions that enhance fluent speech. The fluency enhancing conditions* (FEC) discussed by Wingate (1976) can be divided into two groups according to their nature: (a) conditions concerned with rhythmic stimulation, as in metronomically paced speech, singing, choral speaking and shadowing (Table 2.1.1 and Table 2.1.2); and (b) conditions involving changes in auditory feedback, such as hearing loss, masking noise and delayed auditory feedback (Table 2.1.3). According to Wingate (1976) FEC reduce speech rate, resulting in vowel lengthening. Hence, longer vowel duration during slower speech is the speech variable responsible for enhanced fluency. Lengthening of consonants is thought to be the result of lengthened vowel production (Wingate 1976). Wingate (1986) concluded that the reduction in stuttering due to FEC, is the result of changes in the manner in which the stuttering speaker would ordinarily use his voice. A discussion on Wingate's review on FEC and other approaches to these conditions is included in Tables 2.1.1 to 2.1.3.

*For the purpose of this study fluency enhancing conditions will be abbreviated as FEC.

TABLE 2.1.1: WINGATE'S REVIEW ON THE EFFECTS OF FLUENCY ENHANCING CONDITIONS ON STUTTERING: THE EFFECTS OF METRONOMICALLY PACED SPEECH AND SHADOWING ON STUTTERING

WINGATE'S REVIEW ON FEC	DISCUSSION	OTHER INTERPRETATIONS
Metronomically Paced Speech		
Wingate (1976) dismissed the following reasons to account for the metronome effect: (a) the metronome distracts PWS from their own speech; (b) PWS speak slower when pacing their speech to a metronome and a slower speech rate promotes fluency; (c) the effect of intermittent noise masking by the metronome; (d) the metronome caused PWS to speak more regularly. Wingate (1976) acknowledged that pacing speech to a rhythmic stimulus is induced by a significant amount of regularity. The individual who stutters paces his speech by timing the stressed syllables to coincide with beats of the metronome. This is accomplished by "placing emphasis on syllable nuclei" (Wingate, 1976:181). Syllable nuclei are considered to be primary vowels, which are the main phonatory component of speech. Wingate (1976) assumed that the rhythm effect reduces stuttering because it causes PWS to emphasize vowels. In addition, Wingate (1976) mentioned the reduction of stress contrast in paced speech, which can contribute to fluency. With reduction of stress contrast, the need for changes in the speed of articulatory movement, the tonal aspect (fundamental frequency) and intensity (Hartelius, Wising & Nord, 1997) also reduce. The result is that all syllables are emphasized, including normally unstressed syllables. Furthermore, Wingate (1976) suggested a reduction in consonant articulation during metronomically paced speech.	Wingate (1976) assumed perfect regularity to explain the effect and claimed there was irregularity of rhythm in the speech of PWS as they spoke to the beat of a metronome. He did not consider that PWS's speech is more regular when paced to a metronome as opposed to normal conditions. Though, he noted that PWS can be trained to pace their speech to a metronome, which will regularize their speech. Trained metronome effect, however, does not produce immediate and continuous fluency the way the untrained syllable-timed effect does (Starkweather, 1982). Nevertheless, the regularity factor induced by the metronome can increase fluency. Wingate's hypothesis that the rhythm effect depends specifically on louder or longer vowels, are queried (Starkweather, 1982). An increase in vowel duration is thought to reflect slowed speech rate (Hutchinson & Navarre, 1977). The effect of rhythm on stuttering may thus be dependant on the reduction in speech rate. A reduction in rate includes other speech characteristics beside vowel lengthening, for example: (a) consonants as well as vowels may be lengthened (Klatt, 1974 in Starkweather, 1982); (b) speech is physiologically simplified due to fewer speech movements per unit of time; (c) coarticulatory overlap is decreased (Stevens & House, 1963 in Starkweather, 1982).	• Azrin, Jones and Flye (1968 in Bloodstein, 1995) reasoned that a rhythmic beat produces a synchronization effect on vocal responses. • Adams and Reis (1971) explained paced speech (where all syllables are stressed) as a reduced need for phonatory adjustments. Coarticulatory interaction may be reduced as a result of pronouncing all syllables as stressed and thereby reducing the durational variability of the syllables. • Starkweather (1982) posed that the effect of pacing speech on stuttering can be due to the increased regularity of timing (by external control of the time interval between stressed syllables) or the decrease in variation between stress levels of syllables - which suggests reduced variation in rhythm. The control of temporal aspects of speech, such as segment duration and syllable duration, can be an indication of improved coordination of speech movements, hence, better coarticulation (Hawkins, 1984). Thus, in paced speech efficient rhythmical flow of speech is facilitated by simplification of phonatory, respiratory and articulatory adjustments needed for speech production (Perkins et al., 1976).
Shadowing		
According to Wingate (1976) the speaking rate is slower when an individual who stutters directly repeats the verbal message of a lead speaker.	Shadowed speech is perceived to be less intelligible (Sergeant 1961 in Van Riper, 1982), meaning consonants and vowels might be produced differently to give speech a mumbled quality (Starkweather, 1982).	• Both Starkweather (1982) and Van Riper (1982) speculated that choral speaking and shadowing might involve alterations of timing and the provision of a timing model or template which reduces the complexity of the speech task. In other words, certain temporal aspects of motor speech control needed for speech execution are simplified. However, Starkweather (1982:6) stated that "the mechanism by which this might be achieved remains unclear."

TABLE 2.1.2: WINGATE'S REVIEW ON THE EFFECTS OF FLUENCY ENHANCING CONDITIONS ON STUTTERING: THE EFFECTS OF CHORAL SPEECH AND SINGING ON STUTTERING

WINGATE'S REVIEW ON FEC	DISCUSSION	OTHER INTERPRETATIONS
Choral Speech		
Wingate (1976) rejected distraction, reduced communicative responsibility, auditory masking and pace setting (syllabic regularity) to explain induced fluency when reading in unison with a model speaker. Wingate (1976) claimed that in choral speech PWS modify their vocalization, slow down their speech rate by prolonging syllable durations and increase its loudness. These changes in phonation are thought to produce fluency.	Wingate's position (1976) received support from Ingham and Carroll (in Van Riper, 1982). Though other researchers including Adams and Ramig (1980) and Van Riper (1982) found that changes in rate and loudness did not account for the "fluent" speech of PWS during choral speech. These researchers suggested that pace setting or external timing control might play a role in choral speech, hence fluency (Adams & Ramig, 1980; Van Riper, 1982).	• Adams and Ramig (1980) demonstrated that choral speaking could improve fluency when setting a pace in the absence of rate reduction. Schilling (1965, in Starkweather, 1982) found that fluency was evoked where the model speaker only whispers or pantomimes, while Barber (1939, in Van Riper, 1973) indicated that fluency was improved where the model speaker reads different material than the stuttering speaker. In accordance to Adams and Ramig's (1980) finding, these remarks by Schilling (1965, in Starkweather, 1982) and Barber (1939, in Van Riper, 1973) support the view that PWS's timing improve when speaking in unison.
Singing		
Wingate (1976) ascribed the effects of singing on stuttering as related to changes in timing and intonation of vocalizations, i.e. lengthening of the vowels, a slower speech rate and increasing vocal intensity. "In singing, a person intentionally emphasizes vocalization to express a pattern of stresses centering on the syllable nuclei" (Wingate, 1969:678). Consonants are also lengthened according to Wingate (1976), though not as much as the vowels.	Wingate (1969) points out that it is not the regularity of the rhythmic beats in singing which accounts for the fluency, because the syllables of the lyrics cannot be timed to the beat. Starkweather (1982) challenged this view by stating that the syllables, especially the stressed syllables, are generally located with regard to the music's rhythm. Thus, the syllables of a song may be said to be regularized, because the rhythm determines the timing of each syllable of the song (Starkweather, 1982).	• Starkweather (1982) states that singing causes lengthening of the steady-state positions of speech portions (vowels, silences, semivowels, and nasals) in which there are, compared to normal speech. These steady-state positions involve fewer variations in frequency and intensity than normal speech. These factors suggest that the demands for speech motor control is simplified during singing, since it involves reduced durational variability of speech segments, simplification of the coordinatory activities between articulatory, phonatory and respiratory; reduced coarticulatory interaction, and reduced demands on motor timing control. • The decrease in stuttering during singing suggested to Allan (1970) that stuttering may involve a disturbance of the precise temporal relations that normally exist between voluntary presetting of the phonatory musculature and continuous reflex adjustment of the tonus of this musculature.

TABLE 2.1.3: WINGATE'S REVIEW ON THE EFFECTS OF FLUENCY ENHANCING CONDITIONS ON STUTTERING: THE EFFECTS OF HEARING LOSS, MASKED NOISE AND DELAYED AUDITORY FEEDBACK (DAF) ON STUTTERING

WINGATE'S REVIEW ON FEC	DISCUSSION	OTHER INTERPRETATIONS
Hearing Loss		
Wingate (1976) noted that speech in the presence of a hearing loss, involved a slower rate with lengthened vowels and poorly articulated consonants.	Starkweather (1982) believed that the effect of a slower speaking rate, rather than the lengthened vowels as proposed by Wingate (1976) might be responsible for the induced fluency.	• The observations that a significantly lower incidence of PWS in the hearing impaired population (and the fluency-inducing effects of masking noise and DAF on stuttering) suggest a link between auditory feedback and speech production (Rosenfield & Jerger, 1984). In other words, specific sensorimotor processes may be involved in stuttering. Unfortunately, an inquiry of the connection between feedback and speech production in stuttering goes beyond the parameters for discussion of the current study.
Masking Noise (Masked Auditory Feedback)		
Fluency evoked by masking noise can be described as the result of the PWS not being able to hear his voice. Wingate (1976) contended that the changes masking noise induce on the PWS's speech, are the result of the Lombard effect.	The possibility exists that both consonants and vowels are lengthened as rate is reduced in an effort to improve intelligibility during masking noise (Starkweather, 1982). There is no evidence to support Wingate's assumption (1976) that the effect of reduced stuttering are exclusively attributed to changes in vowels.	• Most PWS increase the loudness of their speech (vocal intensity) under masking noise (Mallard & Webb, 1980; Martin, Siegel, Johnson & Haroldson, 1984). • In normally fluent speakers, the Lombard effect causes fundamental frequency, vocal intensity and word duration to increase and a reduction in speaking rate (Rivers & Rastatter, 1985; Van Summers, Pisoni, Bernacki, Pedlow & Stokes, 1988). These results indicate changes in vocalization, aerodynamics variables (Stager & Ludlow, 1993), coarticulation as well as speech rate.
Delayed Auditory Feedback (DAF)		
DAF as a fluency enhancing condition means that fluency is induced by the method of feeding back the stuttering speaker's voice electronically with a slight delay. Wingate (1976) posed that fluency is enhanced under DAF, because it reduces PWS's speaking rate. A decrease in speaking rate is the main effect of DAF, which implies increased vowel duration following Wingate's theory.	Wingate's (1976) viewpoint is argued by the fact that the DAF effect does not pertain solely to vowel duration.	• Martin and Haroldson (1979) found that the influence of DAF on stuttering frequency is related to two independent factors, namely: (a) the change caused by noise, which relates to the fluency effects of masking noise; and (b) the change caused by rhythmic pacing, which is associated with the effects of paced speech on fluency. Stager and Ludlow (1993) however, did not find an increase in vowel intensity during DAF as would be expected under masking conditions (Lombard, 1911 in Harrington, 1988). • Based on research by MacKay (1968) and Agnello (1970), the extent of modification to speech production due to DAF, is parallel to the duration of the sounds and syllables being produced. Thus, during DAF, an increase in fluency would be associated with longer sound and syllable durations. • Perkins et al. (1976) speculated that the effect of DAF on stuttering might be ascribed to a slower speaking rate that promotes phonatory synergy with articulatory and respiratory processes, hence fluent coordination of the speech system movements. • Based on the observation that DAF can produce stuttering in normal speakers, Van Riper (1982) hypothesized that DAF might cause a disruption in the organization and execution of motor sequences of speech. • Harrington (1988) proposed that in stuttering there might be a conflict in the actual time of production of the vowel and the predicted time of perception of the vowel. The expected time of production is early with respect to actual time of production. DAF seems to eliminate the asynchronies or mismatching. • In correlation with Wingate (1976), Stager and Ludlow (1993) found that vowel duration increases and speech rate decreases under DAF in normally fluent speakers. The explanation offered implicates that timing variables of speech segments are altered during DAF, which promotes fluency.

2.3.1.1.2 Indications of a Breakdown in Speech Motor Control in Stuttering according to Wingate's Hypothesis

Valuable information regarding the role of laryngeal behaviour in stuttering might have been derived from work on the effects of fluency enhancing conditions on phonation (Perkins *et al*., 1976). The effect of the majority of FEC on speech was related to changes in the (usual) manner of phonation, in other words, fluency was established by manipulating phonation (Borden *et al.*, 1985; Kehoe, 1998). The possibility remains that FEC somehow "lead to a reduction of uncertainty in the temporal pattern of speech motor control" (Kent, 1983:254). Kent's (1983) view on FEC relates to the effective *planning time* hypothesis (Perkins, Bell, Johnson & Stocks, 1979) which depicts stuttering as being a function of (effective) planning time for phonetic-voice-onset coordinations. Thus, if the duration of planning time increases to coordinate phonetic-voice-onset, stuttering will decrease. In other words, FEC simplify the complex motor planning required for coordination phonation with articulation and respiration (Perkins *et al.*, 1976).

In the following discussion three aspects of Wingate's (1976) vocalization hypothesis are emphasized: (a) the primary speech variables which are modified by the fluency enhancing conditions according to Wingate (1976), i.e. vowel duration and speech rate, are discussed in terms of its effects on speech motor control; (b) the reason why whispered speech as a FEC could not be explained by Wingate's theory; (c) lastly, fluency enhancement effects are related to modifications in speech motor control.

a. Vowel Duration and Speech Rate as Variables of Speech Motor Control

Wingate (1976) summarized the common feature in fluency-evoking conditions as vowel lengthening due to reduced rate of speech (excluding metronomically paced speech). This observation was to validate his view that the change in the manner of phonation could inhibit stuttering (Wingate, 1970). Stager and Ludlow (1993) support Wingate's (1976) assumption that all FEC indicate increased vowel durations. However, these researchers refer to lengthening of vowels as a *timing variable*. If stuttering is associated with an underlying failure in timing control of speech movements (Van Riper, 1982), then speaking conditions that require reduced demands on timing control may contribute to fluency. Vowel lengthening is only one aspect of reduced rate (timing control) (Starkweather, 1982). Other speech characteristics related to a slower speaking rate may also explain fluency enhancement, e.g. consonant lengthening (Klatt, 1974 in Starkweather, 1982), fewer

speech gestures per time unit (Starkweather, 1982), decreased coarticulatory overlap (Stevens & House, 1963 in Starkweather, 1982), simplified coordination of phonatory, articulatory and respiratory gestures (Perkins *et al.*, 1976; Van Riper, 1982), increased flexibility in the timing of stressed syllables (Folkins, Miller & Minifie, 1975) and changes in aerodynamic variables (Stager & Ludlow, 1993; Stager *et al.*, 1997).

Speech rate can be regarded as an indicator of underlying motor control organization (Adams, 1990). Speech rate is determined by two major factors: (a) the speed with which the articulators move, and (b) the extent to which adjoining gestures overlap, i.e. coarticulation (Gay, 1978). If one of these factors is slowed in a FEC, the other can compensate, within certain limits, for it to maintain control. A slower rate may enhance fluency by providing the stuttering speaker with more preparation time for voice onset coordinations (Perkins *et al.*, 1979), thereby reducing kinematic interaction and coarticulation (Wieneke, Janssen & Belderbos, 1987). Reduced rate of coarticulation may acoustically result in vowel lengthening (Meyer, 1993). Considering that stuttering speakers have increased latencies before initiating movement and appear to be less precise in the temporal patterning of a movement sequence (Borden, 1983; McMillan & Pindzola, 1986; Webster, 1986), it is possible that a slow rate includes more variables than only vowel duration (Kent & Adams, 1989). Regularized speech, such as paced speech, may enhance fluency by requiring fewer rate changes within a specific time interval. Thus, changes in rate and sequencing of speech movements may enhance fluency, since they reduce the motor demands placed on speech motor control.

b. Whispering as a Fluency Enhancing Condition

Wingate's (1976) vocalization hypothesis fails to address whispering as a FEC. In fluency enhancing conditions on which Wingate's theory was based, vocalization is enhanced and confirmed by lengthened vowel duration (which is the result of slow speech rate). Although whispering *does* induce a change in the manner of vocalization, it reduces phonation or vocalization to a large extent. Since PWS are fluent during whispered speech, even when they increase their speaking rate (Perkins *et al.*, 1976), prolonged vowel duration or slow speech rate can not be used as an argument to explain this FEC. Possible explanations for the effect of whispered speech on stuttering, are: (a) reduced coarticulatory interaction and simplification of phonatory-respiratory-articulatory coordinations (Perkins *et al.*, 1976), and (b) a change in aerodynamics and fewer gestures per unit of time (Starkweather, 1982). Aerodynamic variables, such as airflow, are aspects of speech that relate to respiration,

phonation and articulation and may reveal discoordination of these speech production systems.

c. Speech Motor Variables influenced by Fluency Enhancing Conditions

The predominant speech variable that enhances fluency in a PWS is timing. Aspects of speech production that are affected by FEC such as regulation, initiation and duration of movements, rate of movement, postural control and sequencing of coordinated speech movements are all interrelated with timing control. These factors correspond with spatiotemporal specifications for motor programming in speech production (Van der Merwe, 1997).

In explaining the effects of these speech variables on evoking fluency, many researchers (Perkins *et al.*, 1976; Andrews *et al*., 1982; Van Riper, 1982; Perkins, 1986) claimed that the increase in time for planning/programming of movement specifications was the reason for improved fluency. Perkins *et al*. (1979) suggested that FEC provide increased "*planning*" time, which may be accomplished by: (a) slowing in transitions; (b) requiring fewer coordinations or voice onsets; (c) an increase in the predictability of voice initiations; and/or (d) reduced grammatical complexity. Increased pausing or decreased articulatory rate also suggests increased time for "*planning"* of speech execution (Andrews *et al*., 1982). Timing of motor activities is either increased, which manifests acoustically as a slower speech rate, or regularized to an anticipated rhythm by these conditions. Van Riper (1973:71) ascribed the fluency evoking influence of rhythmic, timing and rate effects to "its facilitation of the timing of the basic synergies of respiration, phonation and articulation which appear to be disrupted in stuttering, and the timing involved in the coarticulation of the syllable and the motor patterns of the word." Thus, it is possible that temporal patterning and regularization of speech movements determined by an external timing mechanism (such as rhythmic stimuli), somehow simplify the complex motor demands required for coordinating phonation with respiration and articulation.

Finally, Wingate (1976) may have been biased in isolating vocalization as the primary factor that influences FEC. A review of FEC points out that validated explanations of its effects in reducing stuttering would be a key factor in understanding the underlying breakdown in speech motor control of this disorder. The focus on the peripheral motor control and timing characteristics of the laryngeal system in stuttering, was supposedly motivated by Wingate's (1976) review of FEC (Reich *et al*., 1981). Van Lieshout, Hulstijn and Peters (1996)

stated that stuttering might be better understood in terms of motor control strategies than in terms of motor control deficits. Thus, PWS's ability to assemble motor programs and prepare muscle commands is thought to differ from fluent speakers (Van Lieshout *et al.*, 1996). More research is needed to define the underlying speech motor control strategies in FEC. Research of this nature may reveal possible correlations between FEC and speech motor variables, such as the extent of coarticulatory interaction, duration of onset of movements, timing and sequencing of speech movements, speech rate and regulation muscle activity levels and complexity of coordinatory speech movements.

2.3.1.2 Stuttering as a Disorder of the Vocal Reflexes

2.3.1.2.1 Schwartz's Theory on Stuttering and the Airway Dilation Reflex (ADR)

Schwartz (1974) hypothesised that stuttering is caused by a hyperexcitability in the mechanoreceptors of the larynx, reflexively triggering increased tension in the laryngeal muscles. The theory of stuttering as the cause of laryngeal spasms is based on the airway dilation reflex (ADR). The ADR is regarded as an abrupt opening of the glottis in reaction to rising subglottic air pressure, due to an airway obstruction or need for more air. The glottis is abducted by contraction of the posterior cricoarytenoid (PCA).

According to Schwartz (1974) the mechanoreceptors of the larynx activate ADR function in response to a build-up in air pressure as required for speech. For speech production purposes the subglottal pressure is greater than in relaxed breathing. Thus, to achieve phonation, the PCA contraction needs to be inhibited. When the PCA contraction is not inhibited, the glottis would stay open due to the ADR, which is activated in reaction to the additional air pressure needed for speech. Alternate inhibition (for voiced sounds) and disinhibition (for voiceless sounds) of the PCA contraction are common in normal speech. These changes in the excitation of the PCA contraction are controlled by supra-medullary control centres that apparently regulate the medullary ADR.

Under stress, disinhibition of the ADR results, when supra-medullary control is lost and the need for additional air rise. Following Schwartz's theory (1974:173), stuttering blocks occur when "psychological stress produces a requirement for supratidal volumes and a tendency to misinterpret the air pressures required for speech as an airway obstruction." In response to the ADR, the larynx opens abruptly. Schwartz (1975:140) stated that "very often,

voluntary, anticipatory abduction precedes the ADR as an attempt to cope with the response of the PCA". The individual who stutters thus might try to compensate* for the disinhibition of the ADR by forcefully contracting the laryngeal musculature, which results in hypertense phonation or the blocking of phonation. Other compensatory hypertense postures might also be present, such as involuntary movements of the lips, tongue and jaw. Respiratory activity or movements of the limbs and face, as well as avoidance reactions are regarded as learned behaviours to voluntary compensate for the laryngeal problem.

Refining this theory, Schwartz (*NCS*, 2002) developed an approach to stuttering based on the sources of tension on the vocal folds. Schwartz focused exclusively on the tension that directly precedes the speaking act. The increased tension of the vocal folds during the prespeech state can, under certain circumstances and given a certain physiological predisposition, lead to the onset (and maintenance) of stuttering. Five sources of immediate prespeech tension on the vocal folds have been identified, three of which relate to the act of speaking, i.e. prephonatory tuning, speed stress and anticipatory stress. Prephonatory tuning is the ability to tense the vocal folds in preparation to sound production. This increased tension ensures that speech starts with the correct pitch and intensity. Vocal fold tension is also dependent on the speaking rate. The quicker the start of speech, the greater the tension needed. Higher initial speeds are thought to require the activation of more muscle fibres (that is, more tension) prior to the movement. Anticipatory stresses pertaining to sounds, words and speaking situations increase muscle tension throughout the body, including the vocal folds. Prespeech tension on the vocal folds is the result of these sources.

Schwartz (*NCS*, 2002) submits that individuals with the predisposition to become PWS have "the tendency to lose varying amounts of inhibitory control over their vocal folds and that this occurs only when they intend to speak under conditions of sufficient vocal cord tension." This model indicates the existence of a threshold for stuttering. If the stressors are simultaneously present, the threshold level might be exceeded and stuttering occurs. In other situations when limited stressors are present, an individual who stutters' vocal fold tension might be below the threshold and the person is "fluent".

Schwartz (*NCS*, 2002) motivates the concept of stuttering threshold by indicating that during childhood, development in linguistic-cognitive skills leads to an increase in sentence length.

* Compensatory in this context refers to an attempt to release the abducted larynx.

Increased sentence length is expected to result in a faster speaking rate of the first word. Coinciding with the increase in speed stress, internal rules governing intonation and syllable stress also develop. Intonation and syllable stress are seen as the prime shapers of prephonatory tuning of the vocal folds. Thus, there are changes in both speed stress and prephonatory tuning in speech-language development. If the individual experiences a loss of inhibitory control, it may trigger heightened tension of the vocal folds that prevents smooth initiation of vocal fold vibration. The laryngeal nerves provide information to the brain regarding the position and tension on the vocal folds. When initiation of vocal fold vibration is obstructed, a "characteristic pattern of sensory impulses is being produced by the tension and position receptors. That is, a unique pattern of sensory information is occurring, a pattern that only occurs when the folds assume a particular position, and the tension levels within and around the vocal folds reach a critical level. Also, the pattern must be bilateral, that is, the impulses must be coming from both sides of the voice box, signalling both sides of the brain. It is this characteristic bilateral sensory pattern which is the trigger for stuttering." (*NCS*, 2002:2). Stuttering is therefore regarded as a set of learned responses to a distinctive pattern of nerve impulses from the vocal folds (*NCS*, 2002).

2.3.1.2.2 Implications of Schwartz's Theory on Stuttering and the Airway Dilation Reflex (ADR)

The main reason for rejecting Schwartz's (1974) original theory is the lack of evidence to support his view that stuttering is caused by the malfunctioning of a single laryngeal muscle, the PCA (Starkweather, 1982; Van Riper, 1982). Focussing on discoordination and mistiming in laryngeal functioning alone as the core of stuttering, when these disruptions are also evident in respiration and articulation is the primary objection to Schwartz's view (*NCS*, 2002). Other arguments against Schwartz's theory include: (1) Inadequate description of the physiology of respiration, in regulating subglottic pressure for inhalation and exhalation, is presented (Freeman, Ushijima & Hirose, 1975); (2) Substantial evidence is needed to determine whether the neural drive for the laryngeal behaviour prior to the speech act is subcortical or reflexive (Starkweather, 1982); (3) No indication is given if fluent speech might also have deviancies during prespeech behaviour; (4) The influence of emotional state on the occurrence of stuttering is suggested, but restricted to the relationship between psychological stress and stuttering (Zimmermann & Allen, 1975); (5) The influence of multisystem coordinations on prespeech laryngeal activity is not indicated. Generally speaking, the circumstantial evidence for reflex control of the vocal tract during

phonation and speech is quite meagre. Direct experimental tests of such reflex regulation that could have revealed strong reflex control of laryngeal behaviour have failed to do so (Davis, Bartlett & Luschei, 1993). Such studies, however, do not rule out more subtle reflex effects that might be important for fine control (Davis *et al.*, 1993).

However, Schwartz's (1974) description of stuttering does imply difficulties in speech motor control. These speech motor difficulties may include characteristics such as: (1) a breakdown in reflexive stabilization adjustments to facilitate postural control of the larynx; (2) irregularities in the coordination of voluntary movements of phonation and respiration; and (3) excessive and sudden muscle contractions of laryngeal muscles interrupting the initiation of speech movements.

The aspects of Schwartz's (1974) theory in need of further review pertaining to stuttering are: the suggested influence of involuntary reflexive actions on voluntary, goal-directed movements due to involvement of neural control, and the laryngeal involvement in the discoordination between phonation and respiration. The latter aspect will be discussed following the review of Perkins *et al.*'s (1976) perspective on discoordination of laryngeal behaviour with articulation and respiration.

2.3.1.2.3 Conclusive remarks on Stuttering associated with Involuntary or Reflexive Motor Behaviours

Schwartz's (1974) description of vocal arrest during moments of stuttering is similar to that of spasmodic aphonia, which is a neurologic voice disorder associated with lesions of the so-called extrapyramidal motor system* (Aronson, 1990). Pool *et al.* (1987) suggested that stuttering, as well as spasmodic aphonia should be classified as *vocal motor control disorders*, due to the common dysfunction in fine control of volitional phonation which is related to extrapyramidal and cerebellar involvement (Lastovka, 1995; Abwender, Trinidad, Jones, Como, Hymes & Kurlan, 1998). The basal ganglia and cerebellar control circuits are crucial in the maintenance of synchrony of speech movements, especially their fine coordination and regulating reflexes (Duffy, 1995; Zraick & LaPointe, 1997). These control circuits are important for postural control and maintaining the tone for voluntary, skilled movements (Duffy, 1995), and their roles are predominantly inhibitory (Mitchell & Mayor,

*The term "extrapyramidal motor system" is still being used in the literature and refers to the subcortical control circuits of the basal ganglia (including the limbic system) and cerebellum (Murdoch, 1990).

1983; Duffy, 1995). If the basal ganglia system is unable to inhibit autonomous reflexes, these involuntary movements will interfere with normal coordination. The movement disturbances characteristic of damage to the basal ganglia system are mainly abnormal involuntary movements (such as tremor, excessive and forceful muscular activities) and disturbances of muscle tone (such as sudden muscle contractions) (Murdoch, 1990; Zraick & LaPointe, 1997). Instrumental acoustic and physiological studies of stuttering have revealed similar movement disturbances such as excessive levels of muscle activity (Freeman & Ushijima, 1978; Shapiro, 1980), tremor-like activities of speech musculature (Freeman & Ushijima, 1975; McClean *et al.*, 1984; Smith, 1989; Lastovka, 1995), abnormal excitation in reflex arcs (Zimmermann, 1980*b*; Smith & Luschei, 1983), and postural fixation of vocal tract (Conture *et al.*, 1977). These observations reflect an important aspect regarding voluntary movements, i.e. that damage to the basal ganglia system would cause a disturbance of movement without (significant) paralysis. The correlation between the characteristics of basal ganglia disorders and muscle activity during stuttering suggests a link with involvement of the basal ganglia system.

The above-mentioned perspective is strengthened by brain-imagery research indicating that stuttering is associated with neural structures of the basal and cerebellar control curcuits, e.g.: supplementary motor area (Wu *et al.*, 1995; Fox *et al.*, 2000), thalamus (Pool & Finitzo, 1989 in Watson & Freeman, 1997), basal ganglia, especially the corpus striatum (Wu *et al.*, 1995), cerebellum (Fox *et al.*, 1996; 2000) and substantia nigra (Wu *et al.*, 1995). According to Van der Merwe's (1997) model of speech sensorimotor control, these neural structures, especially the basal ganglia, supplementary motor area (SMA) and cerebellum, are associated with motor programming. Involvement of these three areas may affect initiation, temporal synchronization, timing, preparation of complex movements and regulation of movement (Van der Merwe, 1997). Inadequacies in all of these motor programming functions (Van der Merwe, 1997) are associated with stuttering as indicated by the effects of FEC on stuttering.

Thus, the evidence from brain-imagery studies, supported by peripheral acoustic and physiological research indicate sudden, excessive movements with abnormal tones in speech musculature of PWS may be indicative of involvement of the basal ganglia and cerebellar control circuits in stuttering.

2.3.1.3 Stuttering as a Discoordination of Speech Production Systems

2.3.1.3.1 Perkins, Rudas, Johnson and Bell's hypothesis of Stuttering as a Discoordination of Laryngeal Behaviour with Articulation and Respiration

Perkins, Rudas, Johnson and Bell (1976) proposed that stuttering is a function of complexity of phonatory coordinations with articulatory and respiratory processes. According to Perkins *et al*. (1976) who studied the effects of simplifying the complexity of phonatory and respiratory coordination for speech on stuttering, it seems likely that reduction of stuttering consistently follow physiological simplifications of laryngeal adjustments in speech production. As seen in Table 2.2, the phonatory process, as opposed to articulation and respiration, seems to permit progressive simplification of motor coordinations. Thus, the rate and number of vocal fold adjustments to be coordinated can be systematically altered to reduce the complexity of vocal tract movements without sacrificing speech intelligibility, the rate and number of distinctive articulatory and respiratory movements necessary for speech production would appear to remain constant regardless of respiratory or articulatory adjustments.

Perkins *et al.* (1976) further proposed that complexity of phonatory adjustments could trigger discoordination of speech movements that can result in the audible prolongations, repetitions and hesitations of stuttering. However, physiological details of how motor discoordinations manifest as perceptual stuttering behaviour are not specified.

TABLE 2.2: THE EFFECT OF PROGRESSIVE SIMPLIFICATION OF PHONATORY COORDINATION ON SPEECH PRODUCTION (*PERKINS, RUDAS, JOHNSON & BELL*, 1976)

PROGRESSIVE SIMPLIFICATION OF PHONATORY COORDINATION WITH RESPIRATION AND ARTICULATION	Increased simplification in voice production requirements	*Voiced Condition Severity*	PHYSIOLOGICAL ADJUSTMENTS DURING SPEECH PRODUCTION
	I Complex coordination for normal speech production	*Vocalized speech*	• Vocal folds are abducted and adducted for voiceless and voiced sounds, respectively. • Voiced and voiceless sounds are often produced alternatively in a phrase. This means that adduction and abduction of the vocal folds can occur at the same rapid rate as articulatory movements. • Pitch and loudness adjustments must be made for each voiced sound. • Precise vocal fold adduction as well as rapid vocal fold adjustments of effective mass, elasticity and viscosity are required for these prosodic adjustments.
	II Simplified coordination of speech movements	*Whispered speech*	• The number and rate of physiological adjustments are reduced. Whispered speech requires sufficient adduction of the vocal folds to generate turbulence form airflow through the glottis. Vocal fold resistance is higher than subglottal pressure to prevent vibration. Thus, a voiceless breathstream is generated. • Pitch and loudness adjustments are probably determined by respiratory adjustments of subglottal pressure or by adjustments of the glottis or both. • No adductory or abductory movements are needed between phrase initiation and termination since all sounds are produced voiceless.
	III More simplified coordination of speech movements	*Lipped or silent articulated speech*	• A further simplification of phonatory and respiratory movements. • The breathstream is generated independently of speech requirements. • The vocal folds do not play a role in speech production during lipped speech. • Prosodic and voiced-voiceless requirements are eliminated. • Phonation and respiration need not be coordinated with articulation.

The influence of motor complexity on stuttering was demonstrated by Perkins *et al.* (1976), specifically the effects of laryngeal adjustments on stuttering. These researchers found a reduction in stuttering as the phonatory complexity was physiologically simplified by using voiced, whispered, and lipped speech conditions (Table 2.2). Studies comparing soft and hard voice onsets support the notion that simplified laryngeal coordination induces fluency (Uys, 1970; Vaane & Janssen, 1978 in Bloodstein, 1995). The motor complexity of laryngeal adjustments that was simplified had a direct effect on stuttering frequency and lead to the assumption that a cause–and-effect relationship may exist between phonatory complexity and stuttering. Furthermore, physiological discoordination is thought to be associated with stuttering severity. According to Perkins *et al.* (1976) a breakdown in coordination is more likely to occur in severe stuttering than in mild stuttering as evidenced by the fact that silent articulation was more effective in reducing stuttering behaviour in severe stuttering than mild stuttering.

The effect of simplification in phonatory adjustments could not be explained by an increase of voluntary control (i.e. conscious effort or/and deliberate articulation). Perkins *et al.* (1976)

noted that under such circumstances articulatory rate would decrease, which was in contrast with their observation that speaking rate accelerated progressively from voiced to whispered and from whispered to lipped speech. Thus, PWS seemed to increase their speaking rates as the complexity of laryngeal adjustments was simplified.

Perkins *et al.* (1976) reasoned that the complexity of the phonatory process is related to stuttering. The focus on how laryngeal adjustments affect stuttering did not exclude the possibility that complexity of respiratory and articulatory movements may also play a role in speech motor discoordinations in stuttering. Perkins *et al*. (1976) viewed speech production as a complex process of phonatory, articulatory and respiratory coordinations. The complexity in speech coordination means a change in glottal resistance alters the balance of subglottic, supraglottic, and transglottic pressures (Perkins *et al*., 1976). The observation by Uys* (1970) that English speakers stuttered more on initial consonants than vowels underscores this concept. The lower articulatory complexity of English vowels might account for the difference in initial vowel versus consonant productions. Respiratory and articulatory adjustments could therefore accompany changes in the complexity of phonatory movements. Thus, when a physiological disfluency occurs, it involves a breakdown in the coordination of all the speech production systems and not only phonation. The cause of stuttering, according to Perkins *et al.* (1976), is the problem of coordinating phonation with articulation and respiration which is reflected in the integrated coupling of the speech production systems. The discoordination hypothesis however, does not explain if the complexity of articulatory and respiratory adjustments might trigger discoordinations in speech production.

2.3.1.3.2 Implications of Perkins, Rudas, Johnson and Bell's Discoordination Hypothesis

Perkins *et al.* (1976) described stuttering as a physiological peripheral discoordination. A possible breakdown in the underlying activities of neuromotor control is implied by this hypothesis. The suggestion of a dysfunction in neuromotor control related to stuttering needs further discussion. Perkins *et al.* (1976) assumed that discoordination in speech production derives from problems in phonatory coordination with articulation and respiration. This

* Uys (1970) compared Afrikaans-speaking PWS and English-speaking PWS in their vowel and consonant productions. The Afrikaans speakers stuttered on more initial vowels than the English speakers. Uys (1970) ascribed this to the fact that Afrikaans vowels are more like consonants in their manner of articulation, thus requiring more complex coordinations in speech production.

discoordination hypothesis (Perkins *et al*., 1976) failed to specify the underlying neuromotor control of complex laryngeal activity. Denny and Smith (1997) proposed research into the neural control of these discoordinations in speech production of PWS. According to Denny and Smith's (1997), the phonatory-respiratory-articulatory discoordination of speech production in stuttering can be viewed within a framework of neural control.

Denny and Smith (1997) described three neural centres or controllers that are active during speech production: (a) the dominant *speech motor controller* of voluntary speech behaviours, (b) *the metabolic respiratory controller*, and (c) the *emotional vocalization* and the *periaqueductal gray matter (PAG).* The functions of the different neural centres are summarized in Table 2.4. The normal processes by which respiratory, emotional-vocal and speech motor control are integrated may be impaired in PWS (Denny & Smith, 1997). According to this perspective, neural centres that should react to inputs related to phonatory or articulatory aspects of speech may be receiving competing inputs from the neural circuitry that regulates metabolic breathing, emotional vocalization or both (Denny & Smith, 1997). The influence of these neural centres on speech production in stuttering is presented in Table 2.3.

Denny and Smith (1997) suggested that the speech controller and PAG are capable of affecting the metabolic respiratory controller. The influence of the speech controller on the PAG is unknown, as well as the interaction between the speech and metabolic respiratory controller. The PAG and metabolic respiratory controllers have the potential to interfere with the central speech motor controller in PWS, the reasons offered are: (a) both the PAG and the metabolic respiratory controller can influence the entire speech production musculature due to their autonomous function; and (b) both controllers are associated with emotional expression.

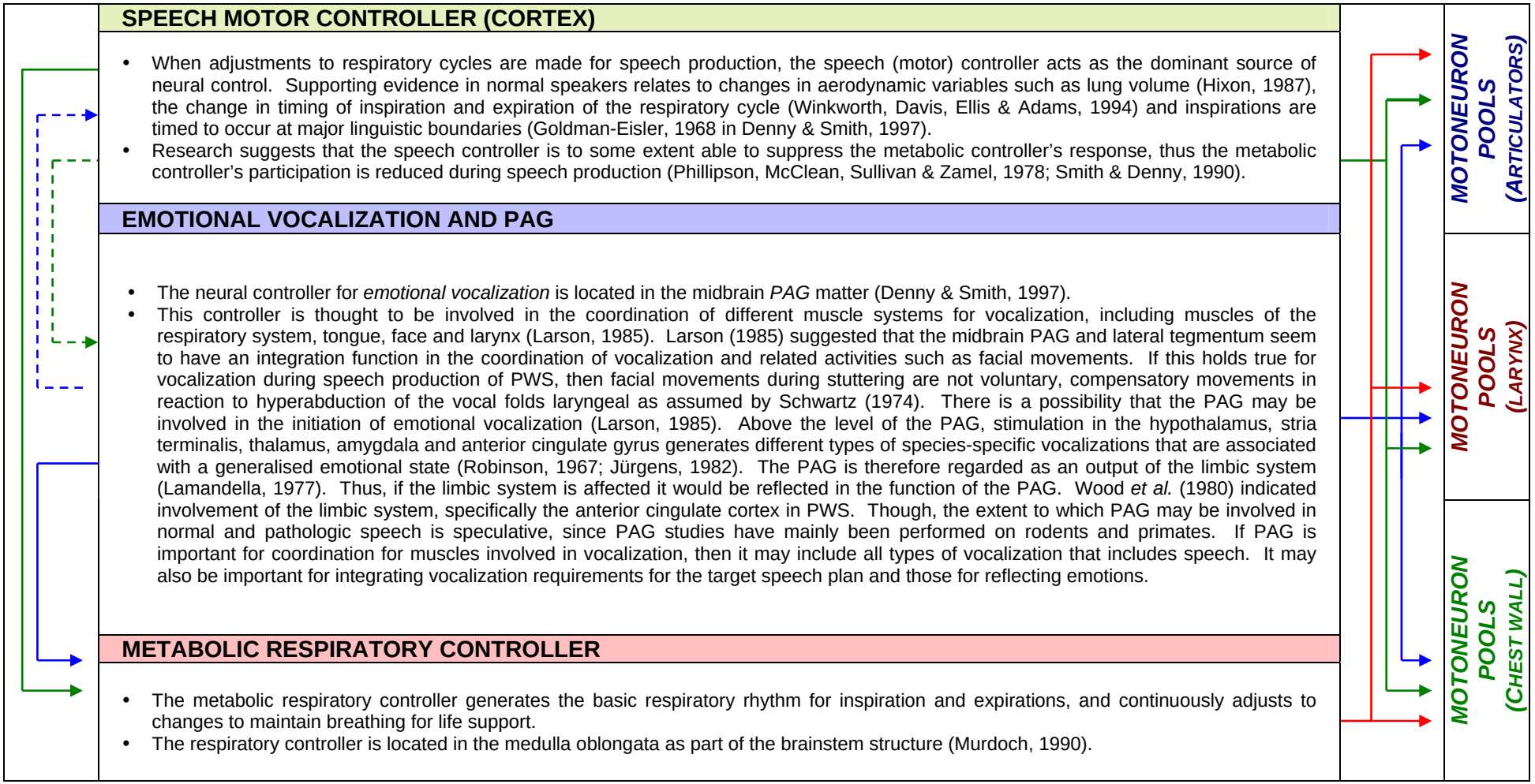
From the above discussion, it can be suggested that a deficiency of one neural controller can contribute to asynchrony in speech motor movements causing stuttering behaviours. Stuttering might be associated with asynchrony or mismatching between the voluntary control of goal-directed movements and autonomous control of motor movements (Denny & Smith, 1997). Some evidence of higher levels of autonomic arousal associated with stuttering exists (e.g. Edgren, Leanderson & Levi, 1970; Ickes & Perce, 1973; Chmelova, Kujalova, Sedlackova & Zelany, 1975; Peters & Hulstjin, 1984). More research of the interactions among motor systems that coordinate respiration, emotional vocalization and speech in

both normal and stuttering speakers are needed to support Denny and Smith's hypothesis (1997). Denny and Smith's (1997) viewpoint is congruent with theories on stuttering stating the primary problem of stuttering as a lack in temporal sequencing of movements (Van Riper, 1982; Kent, 1984) and underscores the need to interpret the motor discoordination in stuttering in terms of neural functioning.

TABLE 2.3: CHARACTERISTICS OF NORMAL SPEECH PRODUCTION AND STUTTERING ACCORDING TO *DENNY AND SMITH* (1997)

	CHARACTERISTICS OF NORMAL SPEECH PRODUCTION	***CHARACTERISTICS OF STUTTERING***
Neural Centres involvement in Speech Production	During normal speech production, excitation of both the metabolic breathing and periaqueductal gray matter (PAG) controller are reduced. The speech motor controller primarily controls respiratory and laryngeal functions and needs to be co-ordinated with the other two neural centres.	In PWS the metabolic respiratory and emotional-vocal centres may compete with the speech motor control system.
Speech Breathing	As opposed to relaxed breathing, normal speech breathing requires rapid changes in time, pressure and flow.	PWS have difficulty in controlling voluntary respiratory movements (Bloodstein, 1987). PWS generally indicate increased variability in time, pressure and airflow variables, suggesting that normal speech motor control be not adequately established.
Laryngeal Function	During breathing for life support, the larynx acts as a variable resistor, that regulates airflow in and out of the lungs. Laryngeal resistance is a determinant factor in the temporal coordination between the duration of expiratory flow and the time before the next inspiration (Berke, 1993). Davis *et al*. (1993) stated that the coordination of movements for these two purposes and the integration of reflexes that serve them require highly skilled regulatory physiology.	If the metabolic respiratory controller facilitates destabilizing inputs to speech in PWS, it would be expected that laryngeal function would be disrupted. In other words, inadequate respiratory control is a potential source of the aberrant laryngeal functioning. Denny and Smith (1997) posed that the inadequate synchrony among these neural control systems may thus cause instability in speech motor output in PWS. Other speech motor problems (including laryngeal behaviour) related to a lack of integrated neural control are difficulty in pre-setting of movement, initiation or coordination of movements, tremor and increased variability of movements (Denny & Smith, 1997).

TABLE 2.4: FUNCTIONS OF THE NEURAL CENTRES DURING NORMAL SPEECH PRODUCTION BY *DENNY AND SMITH* (1997)



--------- *Influence of metabolic respiratory controller* --------- *Influence of speech motor controller* --------- *Influence of emotional vocalization and PAG*

- - - - - *A dotted line suggests a possible influence on another centre*

2.3.1.4 Conclusive remarks on the Relationship between Stuttering and Laryngeal Behaviour

In summary, Wingate (1976), Schwartz (1974) and Perkins *et al.* (1976) associated stuttering with a voicing disorder involving underlying motor control factors. These vocalization hypotheses lead to the question whether phonation (thus, laryngeal behaviour) is inherently more "susceptible" to stuttering than the other speech production systems (Starkweather, 1982; Adams *et al.,* 1984; Perkins *et al*., 1976; Bloodstein, 1995). Several possibilities exist why phonation would seem to be the most complex speech production process to coordinate for fluent speech.

Firstly, voiced-voiceless phonatory coordinations are more complex than subglottic respiratory and supraglottic articulatory adjustments (Wingate, 1976; Perkins *et al.*, 1976). For instance, in faster speaking rates supraglottic articulation is achieved by overlapping movements of the different vocal tract structures at slower speeds (provided they remain within limits of producing intelligible speech) (Perkins *et al.,* 1976). No overlapping of laryngeal movements exists for abduction/adduction of the vocal folds for faster speaking rates. Thus, the laryngeal system has less variety of gestures available to it opposed to the articulation system. The complexity of laryngeal adjustments is therefore directly related to the rate of speech production (Perkins, 1986). Furthermore, the phonatory structures must move in synchrony with the articulatory structures to produce the target sound/syllable/word.

Secondly, comparing the anatomical features of the laryngeal system to the articulatory and respiratory systems, it is apparent that the laryngeal system is smaller. Nonetheless, the laryngeal system's function in speech production is still as complex as the functions of the other two systems. These structural and physiological differences between the speech systems suggest that the phonatory system must be controlled with greater precision than the articulatory and respiratory structures (Perkins *et al.,* 1976), especially pertaining to the timing of abductory/adductory movements.

A third possibility is that the interaction between laryngeal adjustments and aerodynamic forces to initiate and maintain vocal fold vibration seems to be more involved and dynamic than the interaction between the breath stream and the articulatory structures (Perkins *et al.,* 1976). The articulatory structures are positioned to shape the vocal tract that modulates the breath stream. Conversely, glottal vibrations are dependant on vocal fold mass,

elasticity and viscosity adjustments that are coordinated with subglottal and supraglottal pressures to maintain transglottic pressure and to produce the desired prosodic characteristics (such as pitch or loudness) in the utterance (Boone, 1983).

Present viewpoints on the nature of stuttering does not support claims for the "stronger" vocalization hypothesis, hence laryngeal causation of stuttering (Bloodstein, 1995; Kent, 2000). Findings do indicate, however, that laryngeal behaviours are an important symptom of the disrupted peripheral speech physiology that characterized stuttering (Freeman, 1979; Starkweather, 1982). Thus, the physiological aspects of laryngeal behaviour in PWS are an indicator of a breakdown in speech motor control (Perkins, 1986).

2.3.2 Laryngeal dynamics in Speech Initiation of Persons who Stutter (PWS)

Speech movements are responsible for producing on-target acoustic output, thus fluent speech. Underlying the actual production of speech is the accurate timing between the different movements of the speech production processes (including phonation, respiration and articulation) (Alfonso, 1990). In other words, intersystem synchronization is crucial for fluent speech production.

When considering motor speech coordination during normal speech production, it is inevitable to note that laryngeal function is indicated in phonatory, respiratory and articulatory coordinations. Laryngeal valving during speech requires both vocal fold abduction and adduction gestures to differentiate between voice and voiceless speech segments (Lisker & Abramson, 1967). In addition, phonated segments of speech require an appropriate laryngeal muscular set to maintain voicing and to enable modifications in intensity and frequency necessary for prosodic variations (Till, Crumley, Jafari & Law-Till, 1994). Combined with valving by the supraglottal articulators, laryngeal valving determines the airflow rate during speech (Till *et al*., 1994). Also, laryngeal valving functions are suggested to be involved during linguistically motivated pauses or prosody (Till *et al.*, 1994). Thus, laryngeal dynamics are important to intersystem synchronization for fluent speech.

Understanding the physiology of intersystem coordination between respiration, phonation and articulation in normal speech initiation, makes it possible to infer about the underlying physiology of aberrant speech production associated with stuttering. Asynchrony or

discoordination between the different speech production processes in stuttering has been reported by numerous instrumental studies (Perkins *et al.*, 1976; Van Riper, 1982; Adams *et al*., 1984; Bloodstein, 1995), especially pertaining to the initiation or onset of speech movements (Starkweather, 1982; Van Riper, 1982; Adams *et al*., 1984; Borden *et al*., 1985; Bakker & Brutten, 1989; Bloodstein, 1995). Though inappropriately timed movements or contacts may occur at any place of articulation and may involve respiratory activities, it is generally recognized that PWS's primary difficulty lies in the onset of phonation (Dalton & Hardcastle, 1989). The distinctive function of laryngeal movements in onset of phonation may be reason why laryngeal dysfunction has been perceived as the core symptom of stuttering (Kehoe, 1998). Whether deviant laryngeal behaviour results in difficulty with onset of phonation, or whether difficulty with initiation of voicing (due to an underlying breakdown in higher-order speech motor control) results in aberrant laryngeal movements is debatable. The fact remains that deviant laryngeal behaviour of PWS is associated with the breakdown in intersystem coordination during voice (and speech) initiation (Van Riper, 1982).

The current emphasis on laryngeal function in stuttering does not propose that a breakdown in the laryngeal dynamics is the source of motor speech discoordination, but rather it is directed to indicate that such a breakdown may shed some light on the suspected discoordination between phonation-respiration-articulation associated with stuttering. The motor speech skills that are predominantly influenced by discoordinations in speech production, include sequencing of movements, force, range, direction, rate and regulation of movements in the muscles responsible for respiration, phonation, articulation, resonance or prosody (Carter, Yorkston, Strand & Hammen, 1996). A breakdown in these skills is thus associated with discoordinations of the speech production processes. By studying laryngeal dynamics of PWS, such as the sequencing, duration or regulation of vocal fold movements during speech production, specific information on the breakdown in speech motor control may be revealed.

In the following discussion the *so-called* core symptom of stuttering – abnormal laryngeal behaviour – (Kehoe, 1998) will be explored in terms of its characteristics in intersystem (respiratory, phonatory and articulatory) coordination during speech initiation in PWS. For discussing laryngeal dynamics during voice/speech initiation, an arbitrary differentiation has been made between the onset of laryngeal activity relating to the coordination between

phonation and respiration, and the onset of voicing relating to the coordination between phonation and articulation. Prominent studies that investigated these aspects of speech production in PWS will be reviewed. Studies that used complimentary measurement (e.g. acoustic, physiologic, and/or kinematic) techniques to study intersystem coordination during voice initiation are included. Acoustic measurements reflect the acoustically detectable (perceptual) result of the speech task, while physiologic and kinematic measures are more direct ways of addressing specific aspects of speech movement itself (Dembowski & Watson, 1991*a*). Complimentary measurement techniques will also be used in the current study (see Chapter 3).

2.3.2.1 Laryngeal/Phonatory-Respiratory Coordination during Speech Initiation

2.3.2.1.1 Normal Phonatory-Respiratory Characteristics during Speech Initiation

The onset of phonation is preceded by a prephonatory phase. During this phase, preadjustments of the vocal folds (and glottis) in coordination with an increase in subglottal pressure take place as preparation for speech initiation. According to Wyke (1983) the prephonatory period consists of an inspiratory phase followed by an expiratory phase, each characterized by certain laryngeal-respiratory dynamics as indicated in Table 2.5.

TABLE 2.5: THE PREPHONATORY PHASE PRECEDING SPEECH INITIATION ACCORDING TO *WYKE* (1983)

	PREPHONATORY PHASE	
	Prephonatory Inspiratory Phase	**Prephonatory Expiratory Phase**
	Time →	
Description of the Prephonatory Inspiratory and Expiratory Phases	At the onset of the prephonatory inspiratory phase of the breath group*, that follows each speech sequence, the vocal folds are rapidly abducted and remain abducted until completion of inspiration necessary for the utterance to follow. These prephonatory activities take place within a relatively short, rapid time-interval. The prephonatory expiration phase is initiated next.	The vocal folds begin to adduct rapidly some 50-100 ms prior to changes in subglottic pressure (generated by the expiratory air stream) and before audible vocalization starts. Due to the time relations of the reciprocal prephonatory adjustment of adductor and abductor muscle activity, the tension, length and mass of the vocal folds (as well as the dimensions of the glottic aperture) are preset by between 150-500 ms before each audible utterance. This coordinated presetting of vocal fold muscular activity is thought to form the learned process of prephonatory tuning of the larynx.

* The function of respiratory patterning (i.e. syllables per breath group, breath group length, grammatical location of breaths and consistency of breaths) is to partition speech into meaningful units (Hammen & Yorkston, 1994). From an aerodynamic perspective, continuous speech is considered a series of adjacent breath groups (Till, Jafari, Crumley & Law-Till, 1994). Each breath group is composed of (a) inspiration, (b) valved expiration during production of speech and, optionally, (c) one or more pauses (Till *et al.,* 1994). The larynx performs important valving functions associated with each of these three partitions of breath group (Till *et al.,* 1994).

According to Shipp, Izdebski and Morrisey (1984) the preparatory adjustments before onset of vocal fold vibration can be attained by either one of two prephonatory strategies, i.e. respiratory breath holding or laryngeal breath holding. In the respiratory breath holding strategy the vocal folds are positioned in a somewhat abducted posture and the chest wall is fixed before speech initiation commences (Shipp *et al.,* 1984). The laryngeal breath holding strategy includes adducted vocal folds and an increase in the subglottal air pressure in preparation of speech initiation (Borden & Harris, 1984). Both of these strategies require precisely timed coordination between the phonatory and respiratory mechanisms for the onset of vocal fold vibration.

The presetting of motor sequences involving laryngeal-respiratory dynamics during the time interval prior to phonation is crucial for the onset of voicing. During this period the subglottal air pressure below the vocal folds must exceed the pressure above the folds to enable voicing (Borden & Harris, 1984). Phonation starts with the initiation of vocal fold vibration, which is the effect of interaction of the aerodynamic forces generated in the subglottic portion of the respiratory tract with the myoelastic forces found in the preset vocal folds (Wyke & Kirchner, 1976). Voice initiation requires thus the activation of both the phonatory and respiratory systems in concert to enable vocal fold vibration. In addition to active variation of subglottal pressure by the laryngeal-respiratory dynamics, passive variations in glottal-supraglottal airflow resistance can also occur due to a rapid articulatory closing gesture or by a supraglottal cavity volume change (Rothenberg, 1968; 1983). In other words, voice initiation is dependent upon active laryngeal-respiratory movements, but might be influenced by passive variations in airflow resistance.

The precise duration of the prephonatory period varies between individuals as well as with pitch and loudness of the speech sounds to be produced (Wyke, 1983). The time interval of the prephonatory phase also "decreases with increasing rapidity of the utterance of repetitive phonemes" (Wyke, 1983:73). This may indicate that the time needed to prepare the different movements for on-target speech production is lessened, suggesting that the role of voluntary control is subdued when a specific movement sequence is successively repeated (Kehoe, 1997*a*). According to Kehoe (1997*a*) the need for continuous peripheral feedback to update the preset movement sequence is subsequently reduced and movement control for that particular speech event becomes more automatic.

2.3.2.1.2 Abnormal Phonatory-Respiratory Characteristics during Speech Initiation of Persons who Stutter (PWS)

If initiation of vocal fold vibration for speech production is seen as a function of prephonatory vocal fold position and relative lung volume (Izdebski & Shipp, 1978), then any interference with the timing and coordination of the two systems will, most likely, lead to a breakdown in the initiation of phonation. A mismatch occurs when laryngeal resistance is too high for the subglottal expiratory pressure (Berke, 1993) or if subglottal pressure is insufficient or expiratory air perturbed due to atypical subglottal pressure changes (Peters & Boves, 1988). The mismatch between the glottal resistance and subglottal pressure must be overcome to initiate phonation and may influence the timing of voice onset. Since PWS present with an inadequacy to coordinate respiratory movements with laryngeal movements during the onset of phonation (Peters & Starkweather, 1990), this mismatch may be evident during their voice (speech) initiation. Inappropriate timing of respiratory and laryngeal events may thus contribute to delays in the onset of voicing in some PWS (Cross & Olson, 1987).

Possible contributors to irregularity in the initiation of vocal fold vibration (Titze, Baken & Herzel, 1993), which might be related to stuttering, are:

- The temporal schedule, on which movements are based, can be disturbed by excessive or unsteadiness in muscle contractions in the laryngeal and respiratory system (Van Lieshout, Peters, Hulstijn & Starkweather, 1989). Timing of speech movements might also be influenced by difficulty in making rapid laryngeal adjustments which control the onset of phonation (Cross & Olson, 1987). Thus, abnormally elevated muscle activation of a speech system (such as the laryngeal system) may cause a breakdown in a graded overall response (Berke, 1993) due to the interdependency between the systems (phonatory, articulatory, and respiratory systems).
- Instability in the expiratory airway stream at the glottis (Dembowski & Watson, 1991*b*). This might result in inefficient air expenditure at the glottis with most of the air expended just prior to or during the initial portion of an utterance is an important contributing factor to laryngeal-respiratory problems during speech production (McGarr & Löfqvist, 1982; Pratt & Tye-Murray, 1997).

- Coupling between the vocal folds and the vocal tract can also affect voice initiation, i.e. laryngeal impedance. If the acoustic pressures in the subglottal and supraglottal regions change dynamically, it may influence vocal fold vibration (Titze *et al.*, 1993). Peters and Boves (1988) demonstrated that even in perceptually fluent speech, PWS show atypical patterns of subglottal pressure build-up significantly more often than normally fluent speakers.
- Oscillations of vocal folds may be perturbed (Denny & Smith, 1992).

Difficulty attaining vocal fold closure and organizing prephonatory respiratory and laryngeal movements for voice initiation, reflect problems in the coordination of preparatory speech movements in PWS (Watson & Alfonso, 1987*a*). The slower onset in voicing may in turn disrupt the timing of events within and among the speech systems (Cross & Olson, 1987). Movement precision, the force and velocity of the movement as well as temporal accuracy of movements can thus influence the initiation of voice.

Thus, what seems to be an integrated effect of a relative lag in the preparation and adjustment of the vocal folds for phonation might also suggest a disruption in activating the respiratory system (Bloodstein, 1987), for example, variability in prephonatory chest wall posturing (Baken *et al.*, 1983) or a difficulty in coordinating respiration and phonation for the production of the required laryngeal response (Perkins *et al.*, 1976).

Aberrancies in the prephonatory respiratory and laryngeal behaviour for voice onset in PWS might be associated with perceptual deviancies in voice quality. The way in which vocal folds are approximated together for vibration, prior to the onset of expiration, relates to voice quality (Boone, 1983). Abruptness of voice initiations after “breath-holding” (Silverman, 1992) or hard glottal attacks are often associated with phonatory characteristics in PWS (Shipley & McAfee, 1992). A glottal attack is the result of tense adduction of the vocal folds prior to vibration followed by an abrupt release of expiratory air with the onset of voicing for a plosive produced at the glottis (Boone, 1983). Other aberrant variations in vocal fold approximation (contact) and subglottal air pressure may result in deviancies of voice pitch and abnormalities in the regulation of voice intensity (Boone, 1983), which are also associated with speech characteristics of PWS; for example: abnormally high or low pitch levels (Van Riper, 1982; Shipley & McAfee, 1992), glottal fry (Silverman, 1992), and too loud or too soft voice (Shipley & McAfee, 1992).

2.3.2.1.3 Physiologic and Acoustic Studies: Characteristics of Laryngeal-Respiratory Dynamics during Speech Initiation of Persons who Stutter (PWS) and Normally Fluent Speakers

Voice initiation time (VIT) or voice reaction time (VRT) can be defined as the time lapse between the appearance of an external stimulus (controlled by the experimenter) and a subject's initiation of glottal vibration for phonation (Adams *et al.*, 1984). Thus, VIT represents the time period between a specific non-speech event (a response indicator) and the start of voicing. Measuring speech initiation time (SIT) is similar to VIT, but subjects are required to respond to the stimulus by not only phonating, but initiating a word or longer utterance that begins with a voiced sound. The time lag between presentation of the stimulus cue and voicing onset is thought to reflect a speaker's ability to control rapid laryngeal adjustments for voicing. Shipp *et al*. (1984) posed that it is not laryngeal adjustment time that principally determines VRT (or VIT), but rather, the temporal interval necessary for activation of the respiratory system. Considering Adams' *et al.* (1984) and Shipp's *et al.* (1984) description of VRT, it appears that the temporal coordination between respiratory events and laryngeal behaviour will determine VRT.

VRT and VIT studies as summarized in Tables 2.6.1 to 2.6.3 indicate that most PWS are slower than normal speakers in their ability to initiate voicing. Regardless whether aberrant timing in muscle movements in PWS is characterized by overactive muscle contractions or by muscles that are contracting too soon before onset of speech production, both these explanations suggest that PWS might have a generalized difficulty inhibiting muscular contraction (Starkweather, 1982).

Conclusions regarding the initiation of vocal fold activity (laryngeal behaviour) in speech production are mainly drawn from voice or laryngeal reaction time (VRT or LRT) data (Watson & Alfonso, 1983; Bakker & Brutten, 1989; Peters *et al*., 1989). Following Bakker and Brutten's (1989) division of LRT into laryngeal premotor time and laryngeal adjustment, measuring laryngeal adjustment time ought to reflect the period of laryngeal onset during speech initiation. Similar to Bakker and Brutten's (1989) differentiation of laryngeal time intervals during speech initiation, is the division of laryngeal initiation time made by Peters *et al*. (1989). Peters *et al*. (1989) divided speech initiation into pre- and postlaryngeal intervals. By differentiating between these intervals, some information can be obtained regarding the onset of laryngeal activity prior to voice initiation (i.e. laryngeal onset time).

According to Peters' *et al*. (1989) definitions of the two laryngeal intervals, the period of laryngeal (vocal fold) activity onset prior to voice initiation, is represented in postlaryngeal activity. The postlaryngeal interval (*L-A*) reflects the time between the beginning of laryngeal EMG activity and the onset of the acoustic signal and forms therefore part of the actual speech production process. The postlaryngeal period is again sub-divided into (a) *interval L-G* which can be defined as the time between the beginning of laryngeal EMG activity and the initial glottal closure (G), and (b) *interval G-A* which is the period between initial adduction of the vocal folds (G) and the onset of the acoustic signal (A). The absolute difference for the *G-A* subinterval was found to be larger than that for the *L-G* subinterval, suggesting that the *G-A* period has the greatest variability in timing control (Peters *et al.*, 1989). Possible reasons for this finding are difficulty in initiating glottal movements, or incoordinating glottal movements with supra- and subglottal pressure. Since Peters *et al.* (1989) used vowels to measure this interval (*G-A*), the *G-A* interval reflects the period between the onset of vocal fold activity and initiation of vocal fold vibrations, thus *laryngeal onset time* (*LxOT).* In the current study the term *laryngeal onset time* will refer to the period between initial glottal approximation and vocal fold vibration during speech initiation.

Both, Bakker and Brutten (1989) and Peters *et al.* (1989) attempted to locate PWS's delay in initiation. These studies indicate that PWS's delay in initiating cued phonation is attributable to both the preparation and the execution of the onset of phonation. In other words, PWS have a reduced ability to quickly program a required speech response and a lessened ability to quickly posture the larynx for phonation. The most important aspect highlighted by these studies is that PWS show deviancies in timing during the initiation of phonation which involves preparatory laryngeal movements.

However, there are VRT and VIT studies that have found statistically non-significant differences between PWS and normally fluent speakers, as Cross and Olson (1987) have shown (Table 2.6.2). The reason for similar results between PWS and normally speakers in some studies (such as Cross & Olson, 1987) may be due to the level of motor complexity of the speech-like utterances used as test stimuli. A difference in the timing of voice onset when phonating an isolated vowel (such as Cross & Olson used in their study), and producing a vowel-consonant-vowel (VCV) word, is not contradictory but rather emphasize the influence of motor complexity on the period prior to actual voice initiation. Isolated vowel production does not require complex serially ordered motor movements beyond those necessary to

initiate glottal vibration and sustain expiratory airflow for a brief time. In contrast, for VCV productions the speech mechanism gestures need to initiate and sustain phonation for the initial vowel which is temporally overlaid by a set of complex, serially ordered, forward-projecting articulatory movements for production of the following consonant-vowel (CV) segments (Dembowski & Watson, 1991*b*). In addition, Starkweather *et al.* (1976) found significant differences in initiation times for CV versus VC (the VC's being faster), C(*voiceless)*V versus C(*voiced)*V (the voiced CV's faster), and meaningful versus nonsense syllables (the nonsense syllables faster).

Till *et al*. (1983) support the hypothesis that the phonatory tasks differ on the basis of motor complexity. Movement response complexity is usually considered to be directly proportional to the number of muscle groups or speech production mechanisms involved in the movement(s) (Keele, 1981). Movements are sequentially and temporally ordered when performing a specific task (such as speech production) (Harrington & Haaland, 1997). Each movement is determined, in part, by the actions following that movement (Harrington & Haaland, 1997). Therefore, reaction time will increase as the number of movements contained within a sequence increases (see Till *et al*., 1983 in Table 2.6.1*)*. Similarly, reaction time is fastest when the sequences are motorically simple (see Cross & Olson, 1987 in Table 2.6.2).

Temporal coordination of movements, postural adjustments as well as rate of movements are features of speech motor control associated with motor complexity, and are usually indicated in descriptions of stuttering behaviour (Van Riper, 1982; Shipley & McAfee, 1992; Silverman, 1992). Since these features associated with motor complexity, relate to speech motor control (Van der Merwe, 1997), it seems possible that motor complexity can affect the preparatory processes of movements (Reich *et al*., 1981) and concomitantly the initiation of voice/speech.

Another aspect that needs consideration in interpreting speech motor characteristics of PWS is stuttering severity. Research that focussed on the phonatory lag in PWS indicated that the magnitude of the deficit in executing prephonatory respiratory and/or laryngeal events might be determined by the severity of stuttering (Watson & Alfonso, 1983). Watson and Alfonso (1983) concluded that although both mild and severe PWS were delayed in initiating phonatory responses, severe PWS might experience more difficulty in laryngeal adjustments for phonation.

Finally, most VRT and VIT research findings were based on fluent utterances in PWS. These results of PWS's *fluent* speech are biased, since irregular or deviant physiological activities during perceptually fluent speech could have influenced the results. The interpretation of temporal parameters such as voice onset time (VOT) in PWS's so-called fluent speech must therefore be regarded with caution of possible subacoustic stuttering behaviour.

TABLE 2.6.1: SUMMARY OF STUDIES ON VOICE INITIATION OF PERSONS WHO STUTTER AND NORMAL SPEAKERS

PARTICIPANTS	METHOD	RESULTS
Reich, Till and Goldsmith (1981): Laryngeal and manual reaction times of stuttering and non-stuttering adults		
26 Adults were selected: • 13 Stuttering speakers: - Stuttering severity of PWS varied between moderate to severe • 13 Normal speakers The participants were matched by age.	• Reaction-time intervals for laryngeal tasks were measured by a throat transducer using an auditory stimulus as external cue. • PWS and the normal speakers were required to do the following vocal tasks: (a) Non-speech vocal task: (i) inspiratory phonation; (ii) expiratory throat clearing; (b) Speech-mode vocal task: (i) production of the isolated vowel /ʌ/; (ii) the word, "upper" (i.e. /ʌpəˠ/). • Disfluent responses, as judged by the experimenters, were rejected.	• PWS and normal speakers differed significantly only on the speech-mode laryngeal responses. PWS were slower than normal speakers on the isolated vowel production and on the production of the word "upper", i.e. /ʌpəˠ/. • Standard deviations in isolated vowel and word productions were greater for PWS, compared to non-speech vocal tasks. • Longer laryngeal reaction times (LRT) are suggested to reflect learned anticipatory fears of phonatory initiation and maladaptive prephonatory muscular sets.
Till, Reich, Dickey and Seiber (1983): Phonatory and manual reaction times of stuttering and non-stuttering children		
26 Children were selected: • 13 Stuttering speakers • 13 Normal speakers The participants were matched by age, gender and handedness.	• Phonatory reaction times were measured by a vibrational transducer, using an auditory stimulus as external cueing signal. • Reaction times were measured for 4 different laryngeal tasks, i.e.: (a) Non-speech phonatory responses, requiring (i) inspiratory phonation and (ii) expiratory throat clearing; (b) Speech-like phonatory responses, requiring (iii) abrupt initiation of the isolated vowel /ʔʌ/, and (iv) the word "upper", i.e. /ʔʌpəˠ/. • Only fluent responses were analyzed. Subjects were instructed to signal if s/he thought a phonatory response was "not smooth".	• PWS were slower and more variable than the normal fluently speakers only during phonatory initiation of throat clearing and "upper", i.e. /ʔʌpəˠ/. • Lack of a significant group difference for the isolated vowel production for PWS implies according to Till *et al*. (1983) that further development of the stuttering disorder may result in more temporal disintegration of sensorimotor events required for vowel initiation. • Linguistic complexity (thus, meaningfulness of the utterance) does not appear to explain the pattern of results adequately. Phonatory tasks presumably differed on the basis of motor complexity.
Borden, Baer and Kenney (1985): Onset of voicing in stuttered and fluent utterances		
16 Adult participants: • 8 Stuttering speakers: - Stuttering severity of PWS: Mild and severe PWS were included • 8 Normal speakers The participants were matched by age, gender and educational level.	• Electroglottographic (EGG) and acoustic analyses of the first few glottal pulses of voicing were monitored. • Two different sequences of the digits 2, 3, 4, and 5 (i.e. 3-4-2-5; 4-2-5-3) had to be repeated 5 times for 10 utterances from each subject. • Utterances were grouped as fluent/stuttered based on perceptual and physiological judgements, and analyzed as such.	• The control group's patterns of change in laryngeal impedance looked similar for all normal speakers. • EGG and acoustic waveforms of PWS when fluent indicated no obvious differences from those of normal speakers. • When PWS were disfluent, voice initiation after a block was generally characterized by a gradual instead of abrupt build-up of the EGG signal. This finding lends support from a physiological level to the therapy technique of "easy onset" of voicing for fluency. • EGG waveforms also served to help differentiate mild from severe PWS. EGG waveforms for severe PWS when disfluent differed from the normal speakers in several respects: (a) a steeper decrease in vocal fold contact, (b) a less stable or prolonged open phase, and (c) a more gradual build-up of the EGG signal. These differences disappeared upon repeated trials of the same utterance (adaptation). • EGG data for PWS revealed the existence of idiosyncratic ritualised laryngeal behaviour, sometimes including physiological tremor during voiceless blocks. The laryngeal tremor can be associated with tremor in the lower lip.

PWS: Persons who stutter ***C : Consonant*** ***V : Vowel***

TABLE 2.6.2: SUMMARY OF STUDIES ON VOICE INITIATION OF PERSONS WHO STUTTER AND NORMAL SPEAKERS *(Continued)*

PARTICIPANTS	METHOD	RESULTS
Cross and Olson (1987): Investigating the relationship between jaw kinematics and voice onset for stutterers and non-stutterers in a voice reaction time task		
20 Adult participants: • 10 Stuttering speakers: Stuttering severity of PWS varied from mild to severe • 10 Normal speakers The participants were matched by age and gender.	• Vocal responses were transduced by a microphone, and jaw movements were monitored with a strain-gauge system. • Reaction-time responses were measured using an auditory stimulus as external cue. Participants were required to phonate /ʌ/ as rapidly as possible. • Responses perceived by either subject or experimenter as disfluent were excluded from the data analysis.	• Jaw reaction time (JRT) and voice reaction time (VRT): - The difference in overall VRT between the PWS and the normal speakers was slower, but not significant (p=0.12). - Jaw reaction time of the PWS and the normal speakers were statistically non-significant. - Correlations between onset of jaw movements and onset time of vocalization for PWS and the normal speakers suggest that variation in onset time in one system was predictive of similar variation in onset time in another. • Temporal relationship between jaw movement and vocal onset: - Correlations between VRT and jaw-voice difference scores (JVD) for PWS and the normal speakers indicate, that vocal onset was temporally related to termination of jaw opening. - Six PWS exhibited timing relationships between jaw movement and vocal onset similar to those of the normal speakers. Four PWS had atypical long reaction times and exhibited a different articulatory-vocal timing pattern.
Watson and Alfonso (1987b): An analysis of prephonatory kinematic data based on acoustic laryngeal reaction time (LRT) in normal speakers, mild stutterers and severe stutterers		
6 Adult male participants: • 4 Stuttering speakers: - 2 Mild PWS - 2 Severe PWS • 2 Normal speakers	• Acoustic and kinematic data of prephonatory respiratory and phonatory responses were obtained by different instrumentation: (a) Acoustic responses were received by a microphone; (b) Vocal fold movements were monitored by transillumination instrumentation; (c) Thoracic and abdominal wall movements were monitored using *Respitrace* instrumentation. • Participants were required to phonate the isolated vowel /i/. An auditory warning signal (synthetic /i/) was used as onset cue and a phonate cue as the offset cue. The duration of the interval between onset and offset of phonation is the foreperiod. • Only fluent responses were analyzed. Perceptual and acoustic criteria were used to identify fluent utterances.	• Acoustic data indicated that LRT values vary as a function of stuttering severity and foreperiod. • Kinematic data revealed that, in general, the mild PWS demonstrated delayed initiation of respiratory events and appropriate organization of respiratory and laryngeal events while the severe PWS demonstrated delayed initiation of laryngeal events and inappropriate organization of respiratory and laryngeal events. Thus, kinematic and acoustic data both account for group differences in acoustic LRT values as a function of foreperiod.
Peters and Boves (1988): The interactions of respiration (the build up of subglottal pressure), phonation and articulation in fluent speech utterances of stutterers		
17 Adult male participants: • 10 Stuttering speakers: - 1 Mild PWS - 4 Moderate PWS - 5 Severe PWS • 7 Normal speakers The participants were matched by age and gender.	• Only perceptually fluent speech utterances were used in the analysis, based on two criteria: (a) no visible sign of struggle in facial features or body before or during speech production; (b) no audible hesitations, prolongations, or repetitions. • Speech material consisted of 80 words: 50% of these words were monosyllabic words of the VC or CVC type, mostly meaningful words. The rest were meaningful multisyllabic words having 3-4 syllables, with stress on the first syllable. • Acoustic, aerodynamic and EGG instrumentation were used. • Auditory and visual warning signals preceded the auditory response signal.	• Results for prepared versus unprepared speech production did not indicate significant differences in subglottal air pressure build-up and phonation in PWS. • Signal Analysis: - Analysis of subglottal air pressure signals: 7 types of pressure build-up were distinguished, in which 4 types were regarded as deviant. PWS evidenced deviant patterns of subglottal pressure build-up more often than did the normal speakers. Deviant patterns of subglottal pressure build-up that PWS showed include: (a) monotonically increasing pressure with phonation; (b) non-monotonically increasing pressure before start of phonation; (c) pressure increases to a level too high for comfortable phonation; and (d) extremely slow build up of pressure overshoot. - EGG records of voice onset were classified as abrupt or gentle. PWS evidenced abrupt voice onsets significantly more often as opposed to the normal speakers. Acoustic measures of abruptness of voice onsets, first syllable duration, and average syllable duration were also obtained, but no significant difference existed between the PWS and the normal speakers.

PWS: Persons who stutter ***C : Consonant*** ***V : Vowel***

TABLE 2.6.3: SUMMARY OF STUDIES ON VOICE INITIATION OF PERSONS WHO STUTTER AND NORMAL SPEAKERS *(Continued)*

PARTICIPANTS	METHOD	RESULTS
Bakker and Brutten (1989): Compared the laryngeal premotor, adjustment, and reaction times of stutterers and normally fluent speakers		
24 Stuttering speakers: • 23 male PWS • 1 female PWS 24 Normal speakers: • 23 male normal speakers • 2 female normal speakers The participants were matched by mean age.	• External cueing was done by presenting 3 different stimuli: visual, auditory and somatosensory. • Laryngeal reaction times (LRT) (including laryngeal premotor times and laryngeal adjustment times) were determined from EGG recording during the production of /a/. • Only fluent responses were analyzed. The recording of fluent responses was based on the PWS's indication that stuttering occurred.	• The results revealed that the LRT of PWS were significantly longer that those of the normal speakers. • The reaction times (RT) for PWS were significantly longer during both the laryngeal premotor time period and laryngeal adjustment time. - The *laryngeal premotor time* is the time between the onset of the cue-stimulus and the onset of low frequency EGG changes that precede voice initiation. - The *laryngeal adjustment time period* is the time during which prephonatory laryngeal adjustments for voice initiation are made, which is obtained by subtracting laryngeal premotor time from the total LRT.
Peters, Hulstijn and Starkweather (1989): Acoustic and physiological reaction times of stutterers and normally fluent speakers during speech production		
20 Adult male stuttering speakers: • 5 Very mild PWS • 9 Moderate PWS • 6 Severe PWS 20 Normal speakers The participants were matched by age.	• Each participant produced utterances of 3 different lengths: (a) one syllable words, (b) polysyllabic words and (c) sentences. These utterances were produced in two conditions of time pressure (i.e. high and low) and two conditions of preparation (i.e. delayed and immediate responding) using an auditory stimulus as external cue. • Simultaneous recordings in the laryngeal, articulatory and acoustic domains were made. To determine initiation of laryngeal and articulatory muscle activity, electromyographic (EMG) measurements were taken. Vocal fold resistance was recorded using an EGG. The speech signal was recorded using a microphone. • Only utterances judged to have been spoken fluently were analyzed. Fluent utterances were defined as those productions where no visual or audible moments of stuttering were present.	• The results suggest that the reaction times (RT) of PWS and the normal speakers are both increased by longer utterances, and that the effect is proportionally greater for the PWS. • The requirement to respond with minimal preparation produced longer RT in both groups, particularly for longer utterances, but this finding was stronger for PWS than for the normal speakers, especially in the prelaryngeal subintervals (thus, the time between the onset of the stimuli and the initiation of laryngeal EMG activity). • The analysis of subintervals indicated that the additional time taken by PWS in responding was located in the earlier parts of the response, particularly in longer utterances.
Dembowski and Watson (1991b): Preparation time and response complexity effects on stutterers' and normally fluent speakers' acoustic LRT		
18 Adult male participants divided in three groups: • 12 Stuttering speakers: - 6 Mild PWS - 6 Severe PWS • 6 Normal speakers The participants were matched by age.	• Visual stimuli with a warning cue and phonatory cue were presented during two conditions: (a) randomly varied intertrial intervals (ITIs) determined by a computer, and (b) intervals determined by the subject. • Two types of responses were elicited. Firstly, the isolated sustained vowel /a/, which was produced with a neutral upper vocal tract and minimised interaction between laryngeal movements and supralaryngeal articulatory configurations. Secondly, an evenly stressed VCV disyllable in which the flanking vowels were always /a/ and the medial consonant was a voiceless stop (i.e. /apa/). • Only perceptually fluent responses were acoustically analyzed.	• The three groups differed significantly from one another in most conditions, but the magnitude of the difference between mild and severe PWS was notably greater than the difference between mild PWS and the normal speakers. This suggests that aberrant laryngeal behaviour tends to vary with the severity of stuttering. • LRT changes as a function of stimulus condition showed that, in general, the normal speakers were best able to use a preparation-facilitating stimulus condition to reduce LRT, and severe PWS least able to do so. LRT changes as a function of response complexity showed that only the normal speakers produced statistically significant within-group differences.

PWS: Persons who stutter ***C:: Consonant*** ***V : Vowel***

2.3.2.2 Laryngeal-Articulatory/Supralaryngeal Coordination during Speech Initiation

2.3.2.2.1 Normal Laryngeal-Articulatory Dynamics during Speech Initiation

Accurate voicing requires precise timing of supralaryngeal activity (oral articulation) and laryngeal activity (vocal fold vibration for voice onset). The time interval between the release of the consonant burst to the onset of vocal fold vibration for the production of the vowel that follows the consonant, i.e. voice onset time (Lisker & Abramson, 1964 in Borden & Harris, 1984), reflects the coordination between the articulatory and phonatory speech systems. A voiceless speech sound is produced in the absence of vocal fold *vibration.* Each voiceless stop (in English and Afrikaans) has a voiced counterpart, e.g. /p/ and /b/ (Borden & Harris, 1984; De Villiers & Ponelis, 1987). These two sounds differ in terms of laryngeal (vocal fold) activity involved. For /b/ the vocal folds are vibrating when the lips part, while for /p/ the folds come together sometime after the occlusion in the supraglottal area is broken. The voiced-voiceless distinction in initial stops can be viewed as the contrast in timing between the opening of the articulatory occlusion and the start of voicing (Borden & Harris, 1984).

The temporal relationship between the glottal and supraglottal events is measured as VOT and is usually determined in milliseconds. Of significance are the limits in the accuracy with which the speech mechanism can synchronize the gesture at the glottis with the articulatory closure or the release (Rothenberg, 1968). Thus, there are specific boundaries within which VOT has to be initiated in order to produce the correct result (e.g. /b/ or /p/). With voiced sounds, VOT can be initiated 180 ms before the release of air (thus, -180 ms) and 25 ms after this release (thus, +25 ms). In voiceless sounds, voicing needs to be initiated between 40 ms and 120 ms after the release of air (+40 ms to +120 ms) (Zlatin, 1974; Cooper, 1977). VOT can be seen as a within syllable time interval, the boundaries which are defined by articulatory and phonatory events; it is measured after an utterance has started (Adams *et al.*, 1984). The complexity of laryngeal and supralaryngeal coordination during speech initiation is thus reflected by VOT.

From the above discussion it follows that laryngeal behaviour has an important function in the production of consonants. The voicing and devoicing of stops require a change in the conformation of the vocal folds that must be precisely timed with the supraglottal articulation

(Borden, Harris & Raphael, 1994). During the preparatory movements before speech initiation (prephonatory phase), a defined temporal relationship exists between voluntary presetting of the phonatory musculature and the continuous reflex adjustments of laryngeal muscle tone (Wyke & Kirchner, 1976). In relation to the articulation of voiced and voiceless phonemes – uttering a voiced phoneme elicits adduction of the vocal folds and inhibition of many abductor fibers, whereas opposite cord movement is evident with voiceless phonemes (Wyke & Kirchner, 1976). This vocal fold action is thought to occur at 50 ms to 100 ms prior to any changes in subglottic pressure (Wyke & Kirchner, 1976).

2.3.2.2.2 Abnormal Laryngeal-Articulatory Characteristics during Speech Initiation of Persons who Stutter

The inability of PWS to initiate voice as quickly as normally fluent speakers should influence their onset of speech production (see Tables 2.6.1 - 2.6.3). Timing is an important feature of speech production. According to Starkweather (1982) this integral relationship between timing and speech production can be seen in: (a) the changes in articulatory rate that occur at clause and word boundaries; (b) the duration of vowels which is an integral part of syllable stress; and (c) the changes in rate of speech with the speaker's autonomic arousal. Each of these timing aspects is associated with stuttering (Starkweather, 1982; Silverman, 1992). "But timing is particularly important as a signal of the voicing feature. In voiced plosive consonants, the plosive burst and the onset of voicing are simultaneous or approximately so, but in voiceless plosives the onset of voicing is delayed by about 50 ms or more. It is an obvious place to look for difference between PWS and normally fluent speakers, either from a timing or a vocalization viewpoint" (Starkweather, 1982:24). Unlike vocal reaction time that measures the time interval before voice onset, voice onset time (VOT) measures a specific time period which involves onset of voicing during speech initiation.

The timing of articulatory and phonatory movements, as measured in the various intends in speech production processes just before onset of speech, seems to be more variable in PWS than normal speakers (Zimmermann, Smith & Hanley, 1981; Janssen, Wieneke & Vaane, 1983; Watson & Alfonso, 1983; Janssen & Wieneke, 1987). The interaction between articulatory and laryngeal systems may therefore be a factor in PWS's apparent delays in vocal onset (Zimmermann, 1980*a*; 1980*b*). In other words, difficulty in initiating or timing articulatory events with laryngeal activity may contribute to delays in PWS's voice onset ability (Cross & Olson, 1987).

2.3.2.2.3 Acoustic studies: Characteristics of Laryngeal-Articulatory Coordination during Speech Initiation of Persons who Stutter and Normally Fluent Speakers

Acoustic studies investigating VOT in PWS illustrate an obvious trend (see 2.3.2.2.3). In most of the studies mentioned in Tables 2.7.1 to 2.7.2, stuttering subjects had longer (slower) VOT scores than normal speakers. This slowness in the stuttering subjects' VOT cuts across productions of isolated CV syllables to longer linguistic units in reading material (Hillman & Gilbert, 1977). The possibility exists that the relative slowness in VOT of PWS is an inherent feature of their speech-timing control (Adams, 1987).

Delays in voice onset may be associated with longer onset time of articulatory and/or laryngeal movements, asynchrony in articulator-voice timing, and/or difficulty in making adjustments in both the articulatory and laryngeal mechanisms (Cross & Olson, 1987; Dembowski & Watson, 1991*b*). Thus, if maintaining timing between articulatory onset and voicing is important, then variation in the onset time of either system, or both, could result in a reciprocal change in the initiation of voice (or speech)**.** Delaying initiation of articulatory activity, for example, would necessitate delaying onset of phonation to maintain the temporal-spatial relationship needed for the production of a specific sound or syllable, while maintaining a speech rate appropriate for normal speech perception. Also, difficulty making rapid laryngeal adjustments might affect initiation of articulatory movements adversely.

Some of the studies on PWS's voice onset time (see Tables 2.7.1 – 2.7.2), reveal limitations in their interpretations of slower voice onsets. Firstly, using acoustic onset of voice as the criterion measure does not provide specific information about laryngeal behaviour prior to voicing. The factors which contribute to the slowness of voice onset in PWS are difficult to specify from only acoustic data. Probable "causes" of longer voice onset time in PWS deriving from such data are merely speculative (Healey & Gutkin, 1984). In this regard, there is some evidence that abnormal functioning in PWS's laryngeal system occurs during the period prior to actual speech production (Freeman & Ushijima, 1978; Cross & Olson, 1987; Dembowski & Watson, 1991*b*), which will therefore not be acoustically measurable. For example, Freeman and Ushijima (1978) found that PWS co-contracted the abductor and adductor laryngeal musculature prior to phonation of the first word in an utterance.

A second aspect concerning VOT studies is that many of these investigations focussed mainly on aberrant laryngeal behaviour to explain slower voice onset time in PWS. Prosek, Montgomery, Walden and Schwartz (1979) did not find evidence of abnormally high levels of laryngeal tension, but suggested that the slow voicing onset in the stuttering subjects was due to failure to temporally coordinate the onset of the airstream and/or release of the prephonatory laryngeal posture. Thus, respiratory influences might also contribute to delayed onsets of speech production. In addition, longer VOT may reflect the excess articulatory tension which a PWS may experience during the build-up of intra-oral pressure (before the release of the articulatory closure) when uttering a specific speech sound (Healey & Gutkin, 1984). A blockage of the airflow with a concomitant increase in intra-oral pressure can contribute to a slower onset of voicing. Starkweather (1982) posed that PWS's failure to release articulatory closure and to initiate phonation simultaneously during fluent productions of voiced stop sounds suggest that PWS's vocal and articulatory functioning differ from that of normal speakers. It can therefore be reasoned that a more comprehensive idea of the interaction among systems during speech initiation and their contribution to aberrant behaviours influencing the initiation of speech would be provided by investigations that focus on respiratory, laryngeal and articulatory coordinations.

Aside from these limitations, sampling differences exist among VOT studies that may also influence comparing and interpreting results of the onset of voicing in PWS. These sampling differences include: (a) the fluency criteria used by researchers, (b) defining the levels of stuttering severity and (c) the effect of the phonetic context of the utterance on stuttering.

Many studies only inferred on PWS's so-called perceptually fluent utterances, without confirming by means of physiological measures that no abnormal speech behaviour was present (Agnello *et al.*, 1974; Hillman & Gilbert, 1977; Metz, Conture & Caruso, 1979). These studies also only utilized acoustic measurements to analyze the speech sample. If subtle physiological aberrancies were present, they would have been overlooked or might not have been detected by the acoustic signal. Thus, different acoustic studies seem to have used different fluency criteria. Differences in fluency criteria between these studies may partially account for dissimilar VOT results for PWS's "fluent" speech.

Few researchers have classified their subjects according to degree of stuttering severity (see Borden *et al.*, 1985 and Borden *et al.*, 1987 in Table 2.7.2). The criteria used to differentiate

fluent speech from disfluent speech will also determine stuttering severity. Thus, some researchers may base their levels of stuttering severity only on perceptual observations, while others may use physiological as well as perceptual observations. As seen in studies by Borden *et al.* (1985; 1987) mild PWS and severe PWS differed significantly in terms of EGG measures and VOT.

Limited studies on stuttering have considered the effect that the nature of the phonetic context has on the motor speech system during speech production (Peters & Boves, 1988; Peters *et al.*, 1989; Dembowski & Watson, 1991*b*). Certain contextual factors of speech motor control have been indicated to place different demands on the speech production process. Contextual factors of speech motor control (Van der Merwe, 1997) will guide the researcher to select a speech sample that is representative of the aspect of speech production to be studied.

Despite the above-mentioned limitations in previous studies, VOT can provide significant information about the temporal relations between supraglottal and glottal activities during speech production for PWS. Since stuttering is associated a general time delay between coordinative movements, then PWS's ability to regulate rapid supraglottal and glottal activities during speech production would have a high probability for indicating differences from that of normally fluent speakers. In view of "the relative brevity, rapidity and constancy of change of supraglottal and glottal behaviours, it would probably not take a great deal of perturbation in terms of rate, range and force adjustments within and between these components of the speech production system to create disruption, albeit subtle, that could lead to a perceptible disruption in speech fluency" (Conture, Colton & Gleason, 1988:649). The presence of such disruption in temporal coordination between supraglottal and glottal activities in PWS may be indicated by VOT.

TABLE 2.7.1: STUDIES COMPARING THE VOICE ONSET TIMES OF PERSONS WHO STUTTER (PWS) AND NORMAL SPEAKERS

PARTICIPANTS	METHOD	RESULTS
Agnello, Wingate and Wendell (1974): VOT of children and adult stutterers		
24 Stuttering subjects: • 12 Stuttering children • 12 Stuttering adults Both age groups were matched with normal speakers.	• Nonsense syllables with voiceless (i.e. /p/) and voiced (i.e. /b/) consonants in different syllable positions (initial, final, medial) - /pa/, /ba/, /ap/, /ab/, /apa/ and /aba/ were used. • Acoustically fluent tokens were analyzed.	• VOT for children was longer than for adults. • VOT for stuttering children was longer than for the non-stuttering children. • VOT for adult PWS was longer than for the adult normal speakers. • It was not certain that the acoustically fluent tokens were free from muscular abnormality.
Hillman and Gilbert (1977): VOT for voiceless stop consonants in the fluent continuous speech of stutterers		
20 Adult participants: • 10 PWS • 10 Normal speakers	• A spectrographic analysis of voiceless stop consonants (i.e. /p, t, k/) in CV syllables during oral reading. • Only perceptually fluent utterances were analyzed.	• VOT values of PWS were longer than for the normal speakers. • For the PWS and the normal speakers, the average VOT increased as the place of articulation moved back in the oral cavity, and for both groups the differences between the consonants were the same. The average VOT increased from /p/, followed by /t/ and then /k/. • The study did not ensure that only fluent utterances were measured.
Metz, Conture and Caruso (1979): VOT, frication and aspiration during stutterers' fluent speech		
10 Adult participants: • 5 PWS • 5 Normal speakers	• A spectrographic analysis of 18 different sound clusters in monosyllabic words, all of which were composed of stop consonants or stop consonant clusters joined with various vowels. • Only fluent productions were examined.	• VOT data for PWS were longer on only six of the 18 clusters (i.e. *p, br, pr, dr, tw, b*). • The aspiration duration for /tw/ was significantly slower in PWS, but there were no other significant differences for aspiration and frication between the group of PWS and the group of normal speakers. • No difference between voiced and voiceless transitions was found for the subjects. Laryngeal adjustments were made for all types of transitions, voiced-to-voiced (*v-v*), voiced-to-voiceless (*v-vl*) and voiceless-to-voiced (*vl-v*).
Zimmermann (1980b): Articulatory dynamics of fluent utterances of stutterers and normal speakers		
13 Adult participants: • 6 PWS • 7 Normal speakers The participants were not matched for age and gender.	• Lateral view cinefluorographic films were made during productions of three *CVC* words (i.e. *mom, pop, bob*). Movements of lower lip and jaw were monitored. The following measurements were made: (a) time from beginning to end of a movement (*transition time*); (b) time between end of downward movement into vowel and beginning of upward movement out of vowel (*steady state*); and (c) time between beginning of downward movement and onset of voicing (VOT). • Only perceptually and physiologically fluent utterances were analyzed.	• PWS were showed longer transition times, steady states and VOT for both voiced and voiceless plosives compared to the normal speakers. • Slower movements observed in PWS were present in the oral articulators (jaw and lip transition times), as well as in the laryngeal movements participating in VOT. • The mean differences between PWS and the normal speakers were smaller for laryngeal movements than for oral movements. • Differences between the PWS and the normal speakers were possibly a result of uncontrolled differences between the two groups pertaining to age and gender differences, or the presence of undetected abnormal muscle activity in PWS.
Metz, Samar, and Sacco (1983): An acoustic analysis of stutterers' fluent speech before and after therapy		
14 Young-adult stuttering subjects: • 12 Male PWS • 2 Female PWS	• An acoustic analysis was made of a reading passage with 16 stop consonant-vowel-stop consonant (*CVC*) target words located in the passage; as well as a list of 24 monosyllabic (*CV*) English words (including 12 word-initial voiced *CV*s and 12 word-initial voiceless *CV*s). Each target word was embedded in a carrier phrase. • Only fluent utterances were analyzed.	• Acoustic variables were analyzed, including voiced stop consonant VOTs and voiceless stop consonant VOTs. • Both voiceless and voiced VOT duration significantly increased following therapy. • The standard deviations for both voiceless and voiced consonants for PWS increased following therapy. • The data support the notion that the perceptually fluent speech of PWS is not fully "normal".

PWS: Persons who stutter ***C : Consonant*** ***V : Vowel***

TABLE 2.7.2: STUDIES COMPARING THE VOICE ONSET TIMES OF PERSONS WHO STUTTER (PWS) AND NORMAL SPEAKERS *(Continued)*

PARTICIPANTS	METHOD	RESULTS
Healey and Gutkin (1984): Analysis of stutterers' voice onset times and fundamental frequency contours during fluency		
20 Age-matched male adults: • 10 PWS • 10 Normal speakers	• Oscillographic and spectrographic analyses of VOT and fundamental frequency (F0) at vowel onset, average vowel F0, and speech and range of F0 change were obtained from fluent productions of 18 monosyllabic utterances. • Nine voiceless (*vl*) stop CV utterances were used and nine voiced (*v*) stop CV utterances. • All test syllables were placed within a carrier phrase.	• PWS had longer mean VOTs for stop *C(vl)* compared to the normal speakers. PWS's mean values for F0 at vowel onset, average vowel F0, range of F0 change, and speed of F0 change were larger than the mean scores for the normal speakers. The PWS's results, except for range of F0 change, were not statistically different from that of the normal speakers. • PWS had longer mean VOTs for voiced stop consonants compared to the normal speakers, which were indicated to be statistically significant. • There was a significant phoneme main effect for all subjects. VOT measures for voiced stops increased in duration as the point of articulation progressed from bilabial to alveolar to velar placements.
Borden, Baer, and Kenney (1985): Onset of voicing in stuttered and fluent utterances		
16 Participants: • 8 Adult stuttering speakers: - Mild PWS - Severe PWS • 8 Normal speakers The participants were matched by gender, age and educational level.	• Acoustic and EGG analyses of the first few glottal pulses of voicing were monitored. VOT was measured for the utterance /tu/ when performing an adaptation task. • 2 Different sequences of the digits 2, 3, 4, and 5 (3-4-2-5 and 4-2-5-3) had to be repeated 5 times for 10 utterances from each PWS and the normal speakers. • Utterances were grouped as fluent/stuttered based on perceptual and physiological judgements, and analyzed as such.	• Fluent utterances of the mild PWS were within normal limits of VOT. • The VOT for the severe PWS varied depending on the place in the utterance where the block occurred. If the block occurred elsewhere in the series of four digits, VOT on /tu/ was within normal limits, "but if the moment of stuttering occurred at the junctive of the voiceless /t/ and the voiced /u/, then VOT was artificially shortened (as when the subject voiced the stop) or it was markedly long (when voicing became difficult to initiate)" (Borden *et al.,* 1985:371). • VOT data did not suggest an overall deficit in VOT among PWS unless they were stuttering.
Adams (1987): Voice onsets and segment duration of beginning stutterers		
10 Children: • 5 PWS • 5 Normal speakers The participants were matched by age and gender.	• Initial stop consonants (e.g. /b/, /p/, /k/, /t/) for measurement of VOT. Target words were measured in isolation, in elicited sentences and conversation. • Target words used for spectrographic analysis had to be articulated correctly, perceptually fluent, grammatically correct, and effected with appropriate prosody (stress).	• The mean VOTs for PWS were slower, and their mean initial consonant and vowel durations were all longer. The mean VOTs for voiceless plosives for PWS were more prominent than for the voiced plosive /b/, when compared to the normal speakers. • The SD values of PWS were larger compared to the SD values of the normal speakers. • Compared to the normal speakers, PWS were not only slower in VOT and longer segment durations, but also more variable in their control of these temporal parameters elements of speech.
Borden, Kim, and Spiegler (1987): Acoustic analyses of stop consonant-vowel relationships during fluent and stuttered utterances		
14 Participants were divided into 3 groups of stuttering speakers and normal speakers: • 4 Mild PWS • 5 Severe PWS • 5 Normal speakers	• Acoustic measures were made on *stop-gap*, *VOT*, and *vowel durations* of the utterance /tu/ in the context "4-2-5". (*Stop-gap durations* for /t/ were measured from last glottal pulse of previous voiced utterance "four" to burst of /t/; *VOT* was measured from onset of burst for /t/ release to onset of voicing for /u/; *Vowel duration* for /u/ was measured from first glottal pulse to last). • Relationships between consonant and vowel durations were studied by producing scatter plots of all tokens within each group, using stop-gap and VOT for consonant time. • Both fluent and stuttered utterances were analyzed.	• Comparisons of VOT across groups revealed no significant differences among fluent utterances. • A significant difference was found only between the VOT for the stuttered utterances of the severe PWS and that for the normal speakers. The percentages of time taken for VOT within the whole utterance differed less across groups than did absolute duration values. • Ratios between stop-gap duration and VOT increased with severity. However, when fluent most mild PWS were within normal limits of gap/VOT, as were the fluent utterances of severe PWS. • For all participants mean vowel durations were the longest, VOTs the shortest and stop-gap durations for /t/ closure were in between. Mild PWS did not differ significantly from the normal speakers, but severe PWS differed significantly from the normal speakers in both vowel and stop-gap durations even when perceived to be fluent.

PWS: Persons who stutter ***C: Consonant*** ***V: Vowel*** ***SD: Standard deviation***

2.3.2.3 Conclusive remarks on Stuttering as a Breakdown in Laryngeal-Respiratory-Articulatory Coordination during Speech Initiation

From the discussion on laryngeal dynamics during voice initiation, it is evident that voice initiation during speech production requires the vocalization gesture (i.e. inhalation, vocal fold approximation, and simultaneous prolonged exhalation) to be repeatedly and precisely initiated to enable onset of voicing in coordination with articulatory (e.g. jaw, lip, lingual) movements and respiratory dynamics (Ludlow, 1993). The initiation of voice is thus a function of not only the laryngeal system, but also respiration and articulatory movements and their integration (Adams *et al*., 1984; Peters & Boves, 1988). In other words, intersystem synchronization between the speech production processes is essential for the fluent initiation of speech.

Intersystem discoordination of the speech production processes suggests that slowness in onset of voicing during speech execution is the result of aberrant activities of the different speech processes or a combination of phonation, respiration and articulation. Deficiency in intersystemic coordination has been observed in the speech behaviour of PWS (as indicated in Tables 2.6.1 – 2.6.3 and Tables 2.7.1 – 2.7.2). The time delay between coordinative movements might be a reason why PWS exhibit difficulty in moving smoothly and sequentially from one musculature position to the next during speech production (Conture *et al*., 1988). In studies of the acoustic and physiological aspects of fluency and stuttering reviewed in this section, fluent speech seems to the result of the correct timing between airflow and vocal fold activity, and the prompt smooth initiation and maintenance of air flow and vocal fold vibration. The synchronized integration of subglottic pressure, glottal resistance and supraglottal pressure for fluent speech is thus dependent on spatiotemporal coordination of speech motor activities. A disruption in the intersystemic coordination prevents the accurate timing, initiation and maintenance of airflow and/or laryngeal activity, and subsequently, vocalization.

Acoustic and physiological studies investigating laryngeal dynamics in stuttering (such as summarized in Tables 2.6.1 – 2.6.3 and Tables 2.7.1 – 2.7.2) have led to questions about timing and duration in the speech of PWS. The main laryngeal characteristics of stuttering indicated in these studies were a delay in the initiation of voicing, a mistiming and excessive contraction of laryngeal and supraglottal muscles (especially the disruption of reciprocity of

abductors and adductors). Even in perceptually fluent speech, it has been found that PWS have difficulty with timing, initiation and coordination of phonatory, respiratory and articulatory movements (Zimmermann, 1980*b*; Till *et al.*, 1983; Watson & Alfonso, 1987*b*; Bakker & Brutten, 1989). These studies suggest that PWS may have an underlying dysfunction in speech motor control (Borden *et al.*, 1985; Peters *et al.*, 1989; Dembowski & Watson, 1991*b*).

The breakdown in speech motor control may be due to a delay in the organization and transmission of normal neural commands to the specific muscles of the speech production system (Adams, 1981). In addition to slow integration and forwarding of efferent neural commands, Adams (1981) also raised the possibility that the speech muscles of PWS may receive inappropriate neural commands from the speech motor control system. In coherence with Adams' (1981) view, Kehoe (1997) and Kent (2000) have described stuttering as a breakdown in the presetting of muscle commands. A breakdown in motor programming in stuttering is thus indicated (Van der Merwe, 1997). Muscles of the speech mechanisms in PWS are supposedly activated in ways that inhibit rapid onset of voicing. The delay in voicing involves abnormal increases of muscle tone in articulatory and laryngeal structures (Adams *et al.*, 1984) or disrupted coordination between agonist-antagonist laryngeal musculature (Adams *et al*., 1984) which results in a breakdown of reciprocal muscle activity. Thus, long onset times and inappropriate timing of coordinated movements during initiation of voice/speech in stuttering may be indicative of atypical timing control and difficulty in preparation of movements before initiation.

The extent of slower VIT and VOT in PWS appears to be related to stuttering severity (Borden, 1983; Watson & Alfonso, 1987*a*; 1987*b*). This suggests that mild and severe PWS may be distinguished on the basis of their ability to execute preparatory events during the period before voice initiation as well as initiation events during speech production. Severity criteria that reflect characteristics of speech breakdown such as timing latencies may therefore be reliable predictors of aberrant movement patterns among PWS (Cross & Olson, 1987). Further investigation is warranted to establish if the magnitude of the deficit in preparation and initiation of speech production is determined by severity of this disorder.

Research has clearly established that abnormal vocal fold activity is part of the stuttering characteristics for most persons who stutter (Perkins *et al.*, 1976; Freeman, 1979; Van Riper,

1982; Adams *et al.*, 1984; Peters & Boves, 1988; Peters *et al.*, 1989; Dembowski & Watson, 1991*b*). Queries as to whether abnormal laryngeal activity results in stuttering or whether stuttering causes abnormal laryngeal behaviour have lead to a vastly growing interest into the speech motor control of PWS (Watson, Freeman, Chapman, Miller, Finitzo, Pool & Devous, 1991; Williams & Brutten, 1994; Denny & Smith 1997; Freeman, 1999). Consequently, increasing emphasis in stuttering research has been toward vocal fold behaviour in PWS as a symptom resulting from a failure in control of speech motor activities. Results from some of this research were unfortunately inconsequential due to flaws in methodological considerations, including differences in rating stuttering severity of the PWS, inconsistencies in fluency criteria and the nature of the speech material. However, acoustic and physiologic studies based on laryngeal behaviour and speech motor control in stuttering did reveal that the integration of voice with articulation is a critical element of fluent speech. Thus, "anything that prevents smooth integration of voice with articulation will lead to disfluent speech and possibly stuttering" (Freeman, 1999:2).

The above discussion on laryngeal behaviour in PWS has emphasized the presence of abnormal activities during speech initiation. This behaviour does not exist in isolation, but seems to involve respiratory-phonatory-articulatory coordination. When studying the timing and coordination of PWS's laryngeal dynamics consideration should be given to the complexity of the speech production process. This would enable the researcher to render important information regarding aspects of speech motor control in stuttering. An understanding of stuttering behaviour is developed when stuttering characteristics are clearly differentiated from non-stuttering characteristics. In an attempt to explain stuttering as a breakdown in speech motor control, a theoretical framework that includes the characteristics of normal speech motor control is therefore imperative.

2.4. A THEORETICAL FRAMEWORK FOR THE CHARACTERIZATION OF NORMAL AND PATHOLOGICAL SPEECH SENSORIMOTOR CONTROL

The need to work from a theoretical framework based on the normal process of speech production for the understanding of the nature of stuttering has long been proclaimed by researchers in the field of stuttering (Neilson & Neilson, 1987; Caruso, Gracco & Abbs, 1987; Starkweather, 1987; Wingate, 1987; Dalton & Hardcastle, 1989; McClean, 1990; Kent, 2000;

Max, Guenther, Gracco, Ghosh & Wallace, 2004). McClean (1990) has emphasized that a narrowly defined hypothesis on the neural mechanisms of speech motor control is needed to explain the symptoms of PWS's speech. Involvement of the neural systems and mechanisms underlying motor control by the central nervous system (CNS) in PWS might lead to a better understanding of the breakdown in their speech production. A recently published model of normal speech production that explains both normal and disrupted processes in the speech production process, is Van der Merwe's (1997) theoretical framework for the characterization of pathological speech sensorimotor control.

2.4.1 Van der Merwe's Four-Level Framework for the Characterization of Pathological Speech Sensorimotor Control

Van der Merwe (1997) proposed a framework for the sensorimotor control of speech in which four levels of processing are identified, namely linguistic-planning, motor planning, motor programming and motor execution. Van der Merwe's (1997) model differs from traditional models of speech production. Traditionally the speech production process was depicted as consisting of three stages, namely, linguistic encoding (semantic, syntactic and phonological), articulatory programming (also known as motor planning-programming) and execution (Darley, Aronson & Brown, 1975). The significant contribution of Van der Merwe's (1997) model is the well-defined level of motor programming, thereby differentiating motor planning from motor programming, as well as motor programming from motor execution. Defining the distinctive levels or phases of speech *motor* control has important implications for the characterization of motor speech disorders and the interpretation of their symptoms.

The idea that speech motor control consists of different levels or phases is compatible with evidence showing that some key functions of motor representation seem to be allocated to different neural areas (Harrington & Haaland, 1997). Van der Merwe (1997) has ascribed the control of each stage to a coalition of specific neural structures (Table 2.9**).** The components of this framework will be discussed with emphasis on motor programming. Motor programming relates to the prominent characteristics of stuttering, namely, difficulty in temporal coordination of speech muscles and problems in repeated initiation (Van der Merwe, 1997).

2.4.1.1 Contextual factors

Van der Merwe (1997) mentioned specific variables that determine the complexity of control required by each motor level. Some utterances may require more complex control strategies than others. For example, voluntary speech production involves a higher degree of motor control than automatic speech. It follows that a breakdown in speech motor control may manifest more readily during voluntary speech production than automized speech. Thus, by qualifying the contextual factors of the speech material or sample when studying the speech motor behaviour of a specific motor speech disorder (e.g. stuttering), it is possible to gain insight into the speakers' control of complex speech motor activities.

Contextual factors that influence the speech motor control system are outlined by Van der Merwe (1997) as voluntary *versus* involuntary (or automatic) speech, sound/phonological structure, motor complexity of the utterance, length of the utterance, familiar *versus* unfamiliar utterances, and rate of speech. Conditions that increase or decrease stuttering have been listed in the literature (Andrews *et al.*, 1982; Van Riper, 1982; Young, 1985; Starkweather, 1987; Van Lieshout *et al.*, 1989; Silverman, 1992; Kehoe, 1998). These conditions place different demands on speech motor control, which affect the frequency of stuttering and confirm Van der Merwe's (1997) theory of context-sensitivity of speech production. The conditions that influence the frequency of stuttering are summarized in Table 2.8.

Variation in contextual factors will manifest in the spatial and temporal aspects of speech movements and will therefore affect the research results (Van der Merwe, 1997). Identifying these contextual factors gives rise to new interpretations of theories that associated stuttering with a breakdown in cognitive-linguistic processing (Hamre, 1984; Watson & Freeman, 1997), as well as proposed explanations for fluency evoking conditions (Wingate, 1970; Tornick & Bloodstein, 1976; Perkins *et al.*, 1979; Andrews *et al.*, 1982).

TABLE 2.8: A SUMMARY OF CONTEXTUAL FACTORS THAT INFLUENCE STUTTERING

CONTEXTUAL FACTORS THAT INFLUENCE STUTTERING
Voluntary versus involuntary speech
• The production of a meaningful utterance requires a heightened sense of awareness and indicates that such utterance is more voluntary. Thus, the fact that content or meaningful words rather than function words influence stuttering (Kehoe, 1998), may be indicative of the difficulty PWS experience in the control of voluntary speech. • Kehoe (1998) has stated that after practising and repetition, the successive movements become easier to produce within a shorter time and require less feedback. Consequently this might result in a decrease in attention demands (Kehoe, 1997*a*), suggesting that speech becomes more automatic.
Sound or phonological structure
• PWS experience more difficulty with words than with syllables, and more disruptions are evident in syllables than in isolated sounds (Van Riper, 1982). • Phonetic transitions have been suggested to influence stuttering. Voiced to voiced transitions show less stuttering behaviour than unvoiced to voiced transitions (Adams & Reis, 1974). Voiceless-voiceless and voiced-voiceless shifts produce more disfluencies than unvoiced to voiced transitions (Manning & Koufal in Van Riper, 1982).
Motor complexity of the utterance
• The motor complexity of the initial sound or syllable has an effect on the occurrence of stuttering (Starkweather, 1987). PWS experience more difficulty to produce initial consonants than initial vowels (Silverman, 1992; Kehoe, 1998). The possible explanation why consonants are more difficult to control than vowels, is the assumption that stuttering seems to increase with the number of muscle components involved (MacKay & MacDonald, 1984). • Uys (1970) as well as Vaane and Janssen (1978 in Bloodstein, 1995) indicated that words beginning with vowels in Afrikaans and Dutch are stuttered on more than consonants, because initial vowels in these languages are produced with hard glottal attacks. Thus, these vowels have similar features as the consonants in Afrikaans and Dutch.
Length of the utterance
• Longer words are more stuttered on than shorter words (Kehoe, 1998). Length of the utterance in terms of motor processing implies the number of movements needed for that particular utterance. • As the length of the utterance/sentence increases, the speed of the first word also increases (*NCS*, 2002). Thus, the speed of word-per-time-unit increases, which places higher demands on speech coordination. Individuals who stutter appear to have an easier time producing single word utterances *versus* multiword phrases or sentences (Caruso, 2000). Practising a single novel movement facilitates motor learning for complex tasks (Schmidt, 1988).
Familiarity of the utterance
• Less frequently used words have more moments of stuttering (Van Riper, 1982; Kehoe, 1998), which can be related to the influence of motor learning as indicated above. Thus, the more frequently the word is practised, the easier it is to retrieve the "motor program" for that word (Kehoe, 1997*a*).
Speech rate
• Manipulating speech rate influences stuttering (Young, 1985; Caruso, 2000). PWS have been found to stutter less if they speak slowly (Andrews *et al.,* 1982; Van Lieshout *et al.,* 1989; Caruso, 2000). If the required rate of speech is faster than normal (in the absence of an external temporal controller, such as a metronome) moments of stuttering increase (Young, 1985). A slow rate allows more time for the speaker to plan, program and execute speech movements; thus, facilitates the coordination of respiration, phonation and successive articulatory movements (Caruso, 2000).

2.4.1.2 Intention in Verbal Communication

The first "event" in the speech production process, though not recognized as a formal stage, is the intention of the speaker to communicate verbally (Van der Merwe, 1997). The intention to initiate predetermined behaviour is regulated by frontal-limbic formations of the forebrain (Mogeson, Jones & Yim, 1980). Functional connections exist between the limbic system and the basal ganglia (via the nucleus accumbens) (Gray, 1995) and the supplementary motor area (SMA) (Van der Merwe, 1997). Both the SMA and basal ganglia are indicated in motor programming (Gray, 1995; Van der Merwe, 1997). Thus, the possibility exists that the fronto-limbic system is involved in the initial intention to communicate, as well as in motor programming. The intention to communicate is however distinct from the motor control of the actual speech act. Speech movements are therefore not initiated and the specifications of the temporal parameters are not involved at this stage.

In addition to the basal ganglia and SMA, Jurgens and Zwirner (1996) also suggested the existence of a functional link between the periaqueductal grey (PAG) at midbrain level and certain limbic structures (i.e. anterior cingulate cortex and dorsomedial hypthalamus). Results of the role of the PAG in limbic vocal fold control on a *primate* indicated that the limbic system is responsible for non-verbal emotional vocal utterances. "The PAG represents a crucial relay station of the limbic vocal control pathway" (Jurgens & Zwirner, 1996:2921). Although preliminary conclusions cannot be made, the role of the PAG in limbic vocal fold control might have implications for disorders in vocal fold control (such as stuttering).

The fundamental function of the limbic system is to regulate biological drives (Duffy, 1995), but it also provides the emotional motivation to act (Ayres, 1980). Thus, the intention or motivation to communicate relates to expression of emotion (including fear, anxiety, and stress). Fear anxiety and stress are the most common of the covert reactions associated with stuttering (Van Riper, 1982; Miller & Watson, 1992). These emotions relate to the expectation of unpleasantness. In this regard, Van Riper (1982:147) commented that "each stutterer develops his own particular pattern of fear-invested cues based on his perceptions, and these fears can generalize to other cues, following the laws of classical conditioning". To establish an understanding of emotional impact on motor behaviour, it is necessary to explain the possible influence of the limbic system on motor behaviour. In the limbic system's relation to motor behaviour, a differentiation should be made between the voluntary motor

responses following a conscious intention to speak and the role of subconscious, reflex-like inhibitory reactions to conditioned stimuli (Gray, 1995; LeDoux, 1996).

The limbic system and the associated "Papez circuit"[*], as well as areas of the temporal and frontal neocortex and the ascending noradrenegic and serotonergic pathways that innervate these forebrain regions form part of the "behavioural inhibition system" (BIS)[*] (Gray, 1995). The conditioned stimuli for activity in the BIS are related to punishment, omission or termination of reward, as well as novel stimuli (Gray, 1995). "The behaviour elicited by these stimuli consists in *behavioural inhibition* (interruption of any ongoing behaviour*); an increment in the level of arousal*, such that the next behaviour to occur is carried out with extra vigour and/or speed; and *an increment in attention*, such that more information is taken in, especially concerning novel features of the environment" (Gray, 1995:661). According to Gray (1995), any one of the inputs (such as anxiety or stress) to the BIS elicits all the above-mentioned outputs. Activation of the anxiety system (or BIS) depends on the individual's ability to compare actual input with expected input. If a match occurs between expected and actual input, the BIS is not activated and general behaviour is not modified (Gray, 1995). In the case of a mismatch, the BIS is activated and takes control of behaviour. Anticipatory stuttering behaviour or underlying fear associated with covert reactions to stuttering (Van Riper, 1982) might therefore activate the BIS. The BIS provides a possible explanation for the effects of disruption to the monitoring of motor programs due to negative emotional reactions (Gray, 1995).

Certain functions of the BIS structures suggest that this system might play a prominent role in stuttering:

- The amygdala appears to mediate the emotional significance of events and has been found the key structure in classical conditioning (LeDoux, 1996). According to Dodge (2003) the amygdala can be regarded as the brain's "early response system" with a primary role in the mediation of conditioned responses associated with survival and fear, as well as preconditioning the autonomic nervous system to allow automatic decisions and action by the cortical sensorimotor system. These responses are subconscious and reflex-like.

[*]The *Papez loop* includes the amygdala, the hippocampus, the cingulate cortex, several nuclei of the thalamus, the temporal neocortex, periquaeductal gray (PAG), reticulo-pontis caudis, the septum and the mammillary bodies at the base of the hypothalamus (LeDoux, 1996).
[**]The "*behavioural inhibition system*" (BIS) is part of the *comparator model* of anxiety and stress by Gray (1982; 1995). The model is accepted by theorists in the field of neuropsychology and sensory integration (Fisher, Murray & Bundy, 1991).

- The loop of amygdala-temporal neocortex-dorsomedian nucleus seems to subserve speech initiation and production (Cappa & Vallar, 1992).

The limbic system, as part of the BIS system, is therefore important for both inhibiting certain motor behaviour and driving others. Wood *et al.* (1980) suggested that the limbic system is associated with stuttering. Careful consideration must thus be given to the role of the limbic system when hypothesizing about neuromotor control in stuttering.

The four phases of speech motor control, i.e. linguistic-symbolic planning, motor planning, motor programming and motor execution will be the focus in the discussion to follow. Van der Merwe's (1997) viewpoint is reflected throughout this discussion. Additional research supporting her perspective on speech motor control will be indicated.

2.4.1.3 Linguistic-Symbolic Planning

Linguistic-symbolic planning is the first formal stage in the speech production process as postulated by Van der Merwe (1997). This stage entails the syntactic, lexical, morphological and phonological planning of the intended utterance. The selection and sequencing of the phonemes take place, governed by the phonotactic rules of the language. Linguistic-symbolic processing is non-motor in nature. This implies that the linguistic and motor planning of speech are performed at two distinct levels of the speech production process. With regard to the neural structures responsible for linguistic-symbolic planning, there are indications of involvement of the temporal-parietal area, especially Wernicke's area, as well as Broca's area (Wise, Chollet, Hadar, Friston, Hoffner & Frackowiak, 1991; Van der Merwe, 1997).

Circumlocutions and/or interjection overuse due to word-finding problems, and revisions and repetitions due to difficulty in language formulating are characteristics of a neurogenic speech deficit at the level of linguistic-symbolic planning (Dalton & Hardcastle, 1989). Symptoms such as these may result in dysfluent speech. For instance, Perkins (1983) suggested a voluntary dysfluency, as in prolongation of the isolated vowel /ə/ for example, is a deliberate strategy invoked by the speaker while attempting to resolve a high-level linguistic query, such as lexical retrieval. These features form part of the speech pattern found in certain types of aphasia, due to an underlying linguistic-planning problem.

According to Kleinow and Smith (2000) language formulation processes affect speech motor stability in PWS. Linguistic complexity is thought to be a factor that might contribute to the disruption of speech motor stability characteristic of stuttering (Kleinow & Smith, 2000). In other words, "the speech motor systems of PWS may be susceptible to the *linguistic demands* required to produce a more complex utterance" (Kleinow & Smith, 2000:548). Whether an inability to process the linguistic-symbolic demands is characteristic of a subtype of stuttering, needs further investigation. Stuttering is generally regarded as a motor speech disorder, suggesting that the nature of this disorder is associated with a breakdown in neuromotor control (Caruso, 2000). Also, the extent to which the motor complexity of the linguistic unit plays a role in the breakdown in PWS's speech motor stability has not been clearly indicated by those researchers supporting the notion that language formulation is related to stuttering (Hamre, 1984; Postma, Kolk & Povel, 1990; Watson *et al.*, 1991).

2.4.1.4 Motor Planning

In order for phonemes to be actualized on the articulatory level, the phonemic representation of the utterance has to be converted to a code that can be interpreted by the motor system. The code can be defined as the invariant motor plan consisting of specified spatial and temporal goals for each phoneme in the planned unit (Van der Merwe, 1997). These different motor goals for each phoneme are *articulator-specific*, not muscle-specific. The core motor plans are stored in the sensorimotor memory (Van der Merwe, 1997). This motor plan is regarded as the generator of predicted action (Gray, 1982; Jeannerod, 1997). In other words, goal-directed actions (including speech movements) result from the generation of predictions (which is the role of the core motor plan).

During the production of speech, the core motor plans of the sequence of phonemes (sounds) are recalled from the sensorimotor memory and adjustments of the temporal and spatial parameters of movements for each phoneme are made according to the context of the utterance. These adjustments via internal feedback include adaptation of spatial specifications to the phonetic context and rate of production, as well as temporal specifications to segmental durations, speech rate, coarticulation potential and interarticulatory synchronization. The central monitoring of the efference copy ensures that the adaptation of the core motor plan takes place within the limits of equivalence (Van der Merwe, 1997). The central monitoring of the efference copy can be defined as a knowledge of the expected effects of movements

(Brooks, 1986; Keller, 1987), or a neural representation of the acoustic configuration to be reached (Brooks, 1986). Internal feedback is therefore a continuous process during adaptation of the core motor plan, and may be supported by peripheral sensory feedback. The temporally ordered structure-specific motor plan subroutines are then systematically fed forward to the motor programming system (Van der Merwe, 1997). Thus, motor planning is a goal-directed phase where the motor goals for each speech sound are identified and arranged to occur concurrently and sequentially. The neural areas associated with motor planning are Broca's area, the supplementary motor area (SMA) and areas 5 and 7 in the parietal lobes (Van der Merwe, 2005) (see Table 2.9).

As emphasized by the above discussion, specifications of the temporal and spatial parameters of speech movements are crucial for obtaining the on-target acoustic output. A disruption of the motor planning phase may result in speech symptoms such as slow struggling speech with distortion and even sound substitutions (Van der Merwe, 1997). A motor speech disorder associated with an underlying motor planning difficulty, is verbal apraxia (Van der Merwe, 1997).

2.4.1.5 Motor Programming

The following level of speech sensorimotor control is the motor programming level where motor plans are converted into motor programs. The stored motor plan of predicted actions triggers specific motor programs. Whereas motor planning refers to the planning of the temporal and spatial goals for speech production, motor programming refers to the selection and sequencing of motor programs for the movements of the individual muscles of these articulators (including the vocal folds) (Van der Merwe, 1997). Thus, motor planning predicts *what* action needs to be taken for a specific speech utterance (the strategy), while motor programming defines *how* this action will be taken (tactics).

At the programming level of the motor process, sets of muscle commands (or motor programs) are structured and sequenced before the actual movement begins (Marsden, 1984; Van der Merwe, 1997). For speech motor programming, it entails the selection and sequencing of motor programs for the movement of the muscles of articulatory and phonatory structures (Van der Merwe, 1997). In order to accomplish synchronized movements, the process of selecting the appropriate muscles is followed by the inhibition or activation of

these muscles in the correct temporal sequence and then finally, the required amount of excitation for each muscle is applied (DeLong, 1971). The timing and magnitude of muscle contraction in agonists, antagonists, synergists and postural fixators must be specified before movement onset (Ayres, 1980; Marsden, 1984). Programming of sets of muscle commands prior to movement execution is thus crucial for the successful initiation of successive muscle movements.

During motor programming, certain movement parameters are specified including muscle tone, movement velocity, force and range as well as mechanical stiffness of the joints (Van der Merwe, 2005). Motor programs need to adapt to different external constraints and the different contextual demands in which the action takes place. The specifics of motor commands are made prior to movement. These prespecifications of programs can be modified or updated by sensory feedback. The motor programming process relies on sensory information to adjust the timing and magnitude of the motor program patterns underlying the muscle movements (Gracco, 1987; Van der Merwe, 1997). As a result of this constant need of adjusting motor programs, the same action is never copied several times in precisely the same way (Kent, 1998). Thus, motor programs may be regarded as dynamic structures that can be reassembled *de novo* for each action (Kent, 1998).

Repeated initiation and feed forward of co-occurring and successive motor programs, and integration with breathing patterns for speech conclude the motor programming process (Van der Merwe, 1997). Gordon and Meyer (1987) mention that the preparatory time for movements is apparently fastest when successive sequences are structurally similar. This might explain the relation between the amount of preparation time needed for movement initiation and the complexity of the movement (Watson & Alfonso, 1982; Peters & Hulstijn, 1987; Phillips *et al*., 1989).

Motor programming is thought to be involved in aspects such as the *initiation* of complex, successive muscle movements, the *continuous monitoring and adjusting* motor activities by using tactile-kinesthetic and auditory feedback to *direct* complex movements (Gracco, 1987; Jeannerod, 1997; Van der Merwe, 1997). A disruption in the motor programming of speech is expected to present with certain speech symptoms such as: (a) Problems to regulate the rate of the speech sound or the utterance, resulting in a too fast or too slow speaking rate or an irregular speech rate; (b) Postural fixations of speech structures resulting in abnormally long

pauses; (c) A breakdown in synchronizing articulatory (including the vocal folds) movements with respiration which can result in arhythmic breathing; (d) Problems in initiating speech movements, resulting in repetitions of initial sounds or syllables.

Effective motor programming is important for *fluent* speech production. Smith's (1989) description of fluent speech production is parallel to the process of motor programming according to Van der Merwe's (1997) model. Smith (1989:252) states that "the normal flow of sound production is the acoustic consequence of movement, which in turn is the result of an organized neural input to the motoneuron pools that innervate the muscles involved in speech. This synaptic input needs to be organized, both temporally and spatially, so that the appropriate forces are developed in the right muscle at the right time". If a breakdown occurs in the spatiotemporal organization of synaptic, it may result in inappropriate innervation of the muscles. The acoustic consequence may be disfluent speech production.

Speech symptoms associated with a breakdown in motor programming seem to be associated with stuttering behaviour such as mentioned by Van Riper (1982) and Silverman (1992). These similarities in speech symptoms support previous views by Van Riper (1982), Hulstijn (1987) and Starkweather (1987) that stuttering might be caused by ineffective motor programming. Starkweather*(1987), in fact, proposed a model of speech fluency that includes the dimensions of continuity, rate, effort, duration of segments and coarticulation. According to his model, stuttering will result from a breakdown in these dimensions. When relating Starkweather's (1987) model to Van der Merwe's (1997) theoretical framework, the possibility exists that stuttering involves an underlying breakdown in the motor programming process.

Neural areas that are associated with the control of motor programming include the basal ganglia, the lateral cerebellum, SMA, and the fronto-limbic system (septo-hippocampal system and cingulate cortex) (Wu *et al.*, 1995; Fox *et al.*, 1996; Van der Merwe, 2005) (see Table 2.9). The neural structures involved in motor programming perform complementary functions (Van der Merwe, 1997), but the precise role of each needs further studying (see Tables 2.10.1 - 2.10.3).

* Starkweather (1987) excludes language fluency from his model, which is not to say that the influence of linguistic factors on stuttering is also excluded, but rather emphasizes that stuttering is explicitly a motor speech disorder with motor programming features.

2.4.1.6 Motor Execution

Motor execution refers to the actual realisation of speech on an articulatory, phonatory and respiratory level (Van der Merwe, 1997). At this stage the spatio-temporal parameters of speech that have been specified during motor planning and programming are finally converted into muscle movements (Van der Merwe, 1997). During the execution of movement closed-loop tactile-kinaesthetic feedback is supposedly available for control (Van der Merwe, 1997). Acoustic feedback is also implemented (Van Riper, 1982; Van der Merwe, 1997). Although acoustic feedback is available, it is not necessarily constantly utilized during speech production (Van der Merwe, 1997).

Constraints involving the transmission of neural impulses are related to biological factors, such as the diameter of the axon and composition of the myelin sheath (Dalton & Hardcastle, 1989). Also, the time taken for a neural impulse to reach a muscle is related to the length of the nerve fibre and the number of synapses traversed before reaching the muscle (Lenneberg, 1967 in Murdoch, 1990). "The length of the motor pathway for a specific cranial nerve has interesting implications for the temporal coordination of muscular systems, such as the tongue and the larynx, where overall lengths of the motor nerves supplying these organs are different" (Dalton & Hardcastle, 1989:15). Other constraints on the motor execution level arise from the mechanics of muscular contraction (Dalton & Hardcastle, 1989). For example, the degree of force developed by a muscle and the time taken to actualize this force in contraction will depend on certain factors including mechanical properties of the muscle including its mass, elasticity and inertia, the number of motor units, and the mechanical properties of an imposed load (Dalton & Hardcastle, 1989). Neural structures associated with the motor execution level are the cerebellum, basal ganglia, motor cortex and the motor units in the muscles (Wu *et al.*, 1995; Fox *et al*., 1996; Van der Merwe, 2005) (see Table 2.9).

Speech symptoms due to a dysfunction in motor execution are related to the biomechanical dysfunctions of the speech production mechanisms - respiration, phonation and articulation. A motor execution dysfunction will be characterized by speech symptoms such as a reduction of precision of articulation due to abnormal muscle tones (Van der Merwe, 1997). A lack of intonational variations can be accompanied by weakness of the laryngeal or respiratory muscles (Yorkston, Beukelman & Bell, 1988). Prosody of speech can thus be disturbed by the reduced range of movement in these muscles. Speech characteristics of lower motor

neuron dysarthria are associated with motor execution problems (Van der Merwe, 1997).

2.4.1.7 The implications of Van der Merwe's Model for Stuttering

The four-level framework for the characterization of pathological speech sensorimotor control proposed by Van der Merwe (1997) enables researchers to ascribe speech symptoms of a motor speech disorder to a problem in motor planning, motor programming or motor execution. If stuttering is therefore considered a motor programming disorder, its speech motor characteristics (including laryngeal behaviour) should correlate with the speech motor characteristics of disorders associated with motor programming, such as hyperkinetic dysarthria.

2.4.2 Stuttering within the Context of a Theoretical Framework of Speech Sensorimotor Control

2.4.2.1 The Neural Structures involved in Stuttering

Describing the primary characteristics of stuttering within a model of speech sensorimotor control necessitates an understanding of the underlying neural sources contributing to this disorder's distinctive motor behaviours (Denny & Smith, 1992). Numerous investigators who have inquired on the physiological correlates of stuttering have reported that stuttered speech involved a breakdown in phonation, respiration and/or articulation (Perkins, *et al.*, 1976; Van Riper, 1982; Silverman, 1992; Van Lieshout *et al.*, 1996; Denny & Smith, 1997; Caruso, 2000; Perkins, 2001). Speech symptoms of stuttering relating to phonatory, respiratory and articulatory difficulties have been studied to make inferences about the possible breakdown in speech motor control underlying stuttering (Denny & Smith, 1997; Caruso, 2000). Further investigation, though, into the neuromotor areas involved in stuttering is necessary to contemplate the nature of these phonatory, respiratory and articulatory difficulties in stuttering within the context of speech motor control. It is therefore essential to establish the location, extent, severity and sense (i.e. *whether neural activity decreases or increases*) of abnormalities in brain function underlying stuttering for constructing a hypothesis of a breakdown in speech motor control in stuttering (Ingham, Fox, Ingham, Zamarippa, Martin, Jerabek & Cotton, 1996).

According to Van der Merwe's model (1997), comparisons between neural structures associated with stuttering and those structures involved in planning, programming and execution in normal speech processing mostly indicate a dysfunction at the level of motor programming in PWS. Table 2.9 presents a (preliminary) comparison between the coalition of neural structures involved in speech sensorimotor control (Van der Merwe, 1997) and the neural substrates associated with stuttering. The neural areas involved in motor programming comprise the basal ganglia and the lateral cerebellum*, that perform complementary functions, as well as the supplementary motor area (SMA), the motor cortex and the fronto-limbic system (Table 2.9). Though, exploring stuttering as a dysfunction in motor programming does not exclude the possible involvement of the other levels of motor processing. Table 2.9 indicates that neural areas involved in stuttering are not merely limited to the level of motor programming.

* The influences of the basal ganglia and cerebellum are considered to be the most important in the so-called extrapyramidal system (Murdoch, 1990).

TABLE 2.9: A COMPARISON BETWEEN THE COALITION OF NEURAL STRUCTURES INVOLVED IN SPEECH SENSORIMOTOR CONTROL AND THE NEURAL STRUCTURES ASSOCIATED WITH STUTTERING

BASED ON THE THEORETICAL FRAMEWORK OF SPEECH SENSORIMOTOR CONTROL BY VAN DER MERWE (1997)		NEURAL STRUCTURES ASSOCIATED WITH STUTTERING MATCHING THE STRUCTURES INVOLVED IN THE FOUR LEVEL MODEL OF SPEECH MOTOR PROCESSING
FOUR LEVELS OF SPEECH SENSORI-MOTOR PROCESSING	NEURAL STRUCTURES INDICATED IN THE LEVELS OF SPEECH SENSORIMOTOR PROCESS	
Intention to Verbal Communication	- Voluntary Fronto-Limbic System	- Limbic System *(Wood et al., 1980)*
Level 1: Linguistic-Symbolic Planning	- Temporal-Parietal Area - Broca's Area and Adjacent Areas	- Temporal Parietal Areas *(Kalinoskwi, 1997)* - Temporal Lobe *(Finitzo et al., 1991; Pool et al., 1991)*
Level 2: Motor Planning	- Broca's Area - Wernicke's Area - Prefrontal Cortex - Premotor Cortex: Area 6 - Supplementary Motor Area (SMA) - Somatosensory Association Area (including Areas 5 and 7, Parietal Area)	- Sensorimotor Cortex (left hemisphere): Areas Anterior to Central Gyri are overactive opposed to the relative underactive Post Central Gyrus *(Braun, Varga, Stager, Schulz, Maisog, Carson & Ludlow, 1997)* - Wernicke's Area *(Wu et al., 1995)* - Inferior Frontal Gyrus (including Broca's Area) *(Pool et al., 1991)* - Premotor Cortex*: Superior *(Ingham et al., 1994; Fox, et al., 2000)* - SMA *(Wu et al., 1995; Fox et al., 2000)*
Level 3: Motor Programming	- SMA - Basal Ganglia - Lateral Cerebellum - Fronto-Limbic System - Motor Cortex	- SMA *(Ingham et al., 1994; Wu et al., 1995; Fox et al., 2000)* - Basal Ganglia: Nucleus Caudatus and Putamen and Corpus Striatum (*Wu et al., 1995*); Substantia Nigra *(Wu et al., 1995)* - Cerebellum *(Lastovka, 1995; Wu et al., 1995; Fox et al., 1996; 2000)* - Limbic System - Anterior Cingulate Cortex (ACC) *(Wood et al., 1980; Finitzo et al., 1991)* - Motor Cortex *(Fox et al., 1996)*
Level 4: Execution	- SMA - Cerebellum - Basal Ganglia - Motor Cortex - Thalamus - Brainstem - Motor Units	- SMA *(Ingham et al., 1994; Wu et al., 1995; Fox et al., 2000)* - Basal Ganglia (including Corpus Striatum) *(Wu et al., 1995)* - Cerebellum *(Lastovka, 1995; Wu et al., 1995; Fox et al., 1996; 2000)* - Primary Motor Area (especially M1) *(Fox et al., 2000)* - Thalamus *(Pool & Finitzo, 1989 in Watson & Freeman, 1997)*

Lebrun (1997) as well as Abwender, Trinidad, Jones, Como, Hymes and Kurlan (1998) reasoned that stuttering is related to deregulation of the subcortical components involved in speech motor control. This view was based on the similarities between the involuntary movements in stuttering and other types of unintentional verbal behaviours associated with an

* According to Fox *et al*. (2000) activation in the inferior pre-motor area (the brain's speech program unit) was essentially the same in PWS and normal speakers. This eliminates this area, responsible for the creation of speech plans, as a culprit in stuttering.

underlying subcortical dysfunction, such as Tourette's syndrome (Abwender *et al.*, 1998). Since most neurological disorders associated with a dysfunction of subcortical motor nuclei (so-called extrapyramidal diseases) commonly display involuntary movements (Yorkston *et al*. 1988; Murdoch, 1990; Abwender *et al*., 1998), there is a possibility that abnormal muscle contractions which often accompany disfluencies in PWS are equally unintentional and are brought on directly by subcortical nuclei (Lastovka, 1995). According to Lastovka (1995), subclinical tremor may be present in the muscles of PWS even when they are not speaking or at rest. Following Lastovka's (1995) view, certain struggle behaviour in PWS such as eye blinking, grimacing, head nodding or turning, body rocking and foot stamping can be regarded as primary symptoms of stuttering. These physical concomitants (excluding behaviours that are deliberate reactions to stuttering) might have the same involuntary origin as the verbal disfluencies (Lastovka, 1995). The fact that "stutterers exhibit more than just disruptions in speech prior to and during instances of stuttering" (Conture, 1990:7), strengthens the view of subcortical involvement in stuttering.

Stuttering speakers' highly emotional reactions to speech can be interpreted within the model of speech motor control. When supposing that stuttering is a motor programming disorder, according to Van der Merwe's model (1997), it is crucial to acknowledge the fact that subcortical motor nuclei are closely linked (both anatomically and functionally) to the cerebral structures subserving emotional life (known as the limbic system) (Murdoch, 1990). Therefore, excitation of the limbic system may possibly lead to activation of the subcortical motor nuclei (see 2.4.1.2.). When the brain area (presumably the cortex) that regulates subcortical motor nuclei, is unable to maintain its control in the presence of the heightened subcortical excitation, the result may be partial disinhibition of the subcortical nuclei (Lebrun, 1998). If emotional stress increases, then the cortex may be unable to curb the temporary excitation of the subcortical nuclei by the limbic system. This implies that emotions can indirectly influence the level of activation in the subcortical motor nuclei (in particular the basal ganglia). The excitation of these nuclei causes or facilitates insufficient regulation of their functions.

Subcortical motor nuclei are primarily involved in the more routinized, less conscious aspects of voluntary actions, such as the articulatory movements in speech production (Lebrun, 1998). Lebrun (1998) posited that by having a stuttering speaker either speak in a paced, syllable-timed way or imitating a fluent speaker's speech, presumably causes a certain "*de-*

automatization of articulation" (Lebrun, 1998:122). These altered speaking conditions induced fluency presumably because they activated an increase in cortical control of the speech act and subcortical participation was subsequently inhibited via the cortex's regulation (Lebrun, 1998). Thus, the possibility of the subcortical motor nuclei becoming deregulated was reduced and speech motor control improved as was reflected by fluent speech production in PWS.

The above discussion illustrated that the neurophysiology of sensorimotor control for speech production involves not only specific cortical areas but is subserved by a mosaic of neuronal structures comprising cortical regions, subcortical structures and their respective connections (including the interactive synaptic behaviour between these neural structures). Atypical neurophysiology in speakers who stutter can therefore not be attributed to only a focal brain part. Rather, the neural structures associated with stuttering indicate anomalous activity pertaining to the complementary functioning of certain structures due to neural synaptic links (Table 2.9). In other words, multiple neural structures contribute to neurophysiological changes in the "fluency generating system"* (Watson & Freeman, 1997) of PWS. Stager (1990) suggested that neural conduction abnormalities between the neural structures differ for the individual stuttering speakers, which implies that PWS could be divided into subgroups based on the specific neural structures involved.

Motor speech features of stuttering can be related to the inadequate functioning of the neural structures involved in this disorder. A review of the complementary functions of the neural structures associated with stuttering in relation to its symptoms, may prove to be significant to the understanding of stuttering as a motor speech disorder such as presented in Tables 2.10.1, 2.10.2 and 2.10.3. If the different neural structures involved in stuttering show evidence of a breakdown in functioning, these dysfunctions may give an indication to the symptoms of stuttering. For instance, characteristics of a dysfunction of the SMA seem to correlate with stuttering symptoms (Fox, Ingham, Ingham, Hirsch, Downs, Martin, Jerabek, Glass & Lancaster, 1996). In this respect, Fox *et al.* (1996) showed that overactivation of the SMA, as seen in PWS could result in involuntary interruptions or perseverations due to a breakdown in the initiation and precise timing of speech movements. However, preliminary brain imaging research on neural functioning in PWS is depicted by controversial findings (Viswanath, Rosenfield & Nudelman, 1992; Fox, Lancaster & Ingham, 1993). Controversial findings

* The fluency generating system refers to sensorimotor control in normally fluent speakers.

pertaining to levels of neural activity in the left or right hemispheres of PWS may be due to the fact that speech processing for different speech tasks employs different neural circuits, e.g. silent reading *versus* oral reading. Oral reading shows increased activity in the motor cortex and temporal cortex, as opposed to silent reading (De Nil, Kroll, Kapur & Houle, 2000). Thus, interpretations of brain imaging findings on stuttering must be regarded with caution.

Brain imaging results do support the possibility that subgroups of PWS exist, depending on the involvement of neural structures (Fox *et al.*, 2000; Max, Guenther, Gracco, Ghosh & Wallace, 2004). Based on these neural structures, stuttering may involve aspects of linguistic-symbolic planning, motor planning and execution (Table 2.9). It is proposed that the severity of stuttering might relate to the amount of neural structures showing anomalous functioning. Another possibility is that if PWS employ other neural structures in a specific speech processing phase, such as motor programming, it may suggest the use of "compensatory" neural circuits to circumvent or reduce the inadequacy of the programming of speech tasks (De Nil *et al.*, 2000; Max *et al.*, 2004). For example, De Nil *et al.* (2000) suggested that the activation of Broca's area during *silent reading* in PWS, could indicate memory retrieval of the efference copy and silent rehearsal of the articulation pattern of words. Furthermore, the discussion surrounding atypical lateralization of language processes in PWS has been associated with possible compensatory processes of neural systems. For example, Braun *et al.* (1997) speculated that activation of left hemispheric regions may be related to the production of stuttered speech, whereas, activation of right hemispheric regions may represent compensatory processes associated with reducing stuttering symptoms.

Observations of altered brain behaviour in PWS during stuttered and fluent speech acts, strengthen the possibility that stuttering may involve a breakdown in the preparation of motor sequences for speech production (Braun *et al.*, 1997; Wu *et al.*, 1995; De Nil *et al.*, 2000; Fox *et al.*, 2000). Wu *et al.* (1995) demonstrated that brain activity in PWS differs from normally fluent speakers in two respects: (a) *Non-reversible trait characteristics* in brain activity are present during stuttering and induced fluency. An example of a trait characteristic in PWS seems to be a left caudate hypometabolism (Wu *et al.*, 1995); (b) *State dependent characteristics* mean that brain activity alters upon the occurrence of stuttering. For instance, hyperexcitation in the substantia nigra and the limbic system is evident in PWS during induced fluency (Wu *et al.*, 1995). Though, these preliminary findings on atypical brain activity cannot yet indicate whether stuttering results form an underlying breakdown in the

coordination between controllers of involuntary, autonomic movements and controllers of voluntary, goal-directed movements (Denny & Smith, 1997) (see 2.3.1.3.2.), or whether stuttering results from the involvement of specific neural structures (Tables 2.10.1 - 2.10.3) which cause (irreversible or variable) deregulation of complementary functions with other neural substrates via direct or indirect neural connections.

In summary, findings of functional neuroimaging (especially positron emission tomography) studies have shown some degree of anomalous brain function in PWS. These abnormalities in brain functions are multifocal and include cortical as well as subcortical regions (Tables 2.10.1 - 2.10.3). Though no evidence of prominent, focal neural lesions in (adult developmental) PWS have been reported. Anomalous brain functioning in PWS *during rest* seems to be consistent with deficits in their speech motor and language processing (Watson & Freeman, 1997). This suggests an atypical functional trait in PWS's brain activity. For PWS, neural structures showing anomalous activity in most brain imaging studies, are: the ACC (part of the limbic system), the cerebellum, basal ganglia, primary motor area, SMA and the temporal cortex (Table 2.9). Together these neural structures form a group of characteristics associated with motor programming, such as, setting the specifications for precise, sequential motor behaviour, initiation and temporal synchronization of speech (Van der Merwe, 1997). It seems reasonable therefore to assume that stuttering behaviour portrays a breakdown in motor programming.

TABLE 2.10.1: THE SPEECH MOTOR FUNCTIONS AND POSSIBLE MOTOR SPEECH DYSFUNCTIONS OF THE NEURAL SUBSTRATES INDICATED IN MOTOR PROGRAMMING AND STUTTERING

NEURAL SUBSTRATE	LEVEL OF ACTIVITY IN STUTTERING	SUGGESTED FUNCTIONS OF THE NEURAL SUBSTRATE	POSSIBLE MOTOR SYMPTOMS (INCLUDING SPEECH SYMPTOMS) AS A RESULT OF A DYSFUNCTION OF THE NEURAL SUBSTRATE
Supplementary Motor Area (SMA)	• Increased activity in left hemisphere (Wu *et al.*, 1995).	• The SMA in association with the basal ganglia, is involved in the preparation of complex movements in response to conscious intention (Tanji, 1996; Van der Merwe's review, 1997). The SMA triggers the release (initiation) of motor programs (Goldberg, 1985, Fox *et al.,* 1996) and is essential for precise timing of complex movements such as speech (Tanji & Shima, 1994; Fox *et al.,* 1996). It specifically plays a role in the generation of sequences that fit into a precise motor plan (Jeannerod, 1997). • SMA activation decreases when a sequential motor task becomes more automatic throughout acquisition (Seitz & Roland, 1992). • According to Halsband and Passingham (1982), the SMA plays a critical role in the appropriate selection of motor behaviour without sensory guidance.	• Dysfunction of the SMA manifests as irregular, discoordinated movements or an inability to terminate a motor response and initiate the next, thus motor perseverations (Jordaan & Jordaan, 1989). Stuttering could possibly result from an interruption of speech motor programs which the SMA must repeatedly restart (Fox *et al.,* 1996). • Increased activity in the left SMA results in hesitations, distorions and repetitions in speech • Lesions limited to the left SMA result in difficulties in reproducing rhythmic movements from memory, although these sequences can be repeated under auditory pacing (Halsband *et al.,* 1993).
Basal Ganglia (including the Substantia Nigra)	• The basal ganglia are less active in the left hemisphere, especially with reference to the caudate nucleus and corpus striatum (Wu *et al.*, 1995). • Increases in stuttering are related to blood flow increases in the putamen together with the ventral thalamus and inferior anterior cingulate in the left hemisphere during a sentence construction task (Braun *et al.,* 1997). • Indications of an atypical dopamine metabolism (Riley, Maguire & Wu, 2001). Hyperpolarization of dopamine in the left hemisphere (Rastatter & Harr, 1988).	• The basal ganglia function as a program generator for precise, sequential motor behaviour, including speech (Murdoch, 1990; Gray, 1995). The structures are associated with initiation, temporal synchronization, timing and automized production of speech (Van der Merwe, 1997). The basal ganglia also seem to play an important role in regulating muscle tone and postural adjustments during skilled movements (Phillips *et al.,* 1989). • The basal ganglia are involved in matching the required movement with limitations set by the (physical) environment, for instance speaking with restricted mandibular movement (Brown in Duffy, 1995). • Specific functions of the *corpus striatum* is "*the setting and maintaining central preparatory states related to the internal; generation of individual behavioural acts"* (Schultz & Romo, 1992:363). • The *substantia nigra* is responsible for supplying the neurotransmitter substance dopamine (DA) (Murdoch, 1990). Accumbens and striatal DA are involved in the initiation of movement (Gray, 1995; Salamone, 1992). It is also associated with the control of rate and range of movements (Marsden, 1982 in Murdoch, 1990). Accumbens DA is thought to be associated with procedural learning in sensorimotor integration (Salamone, 1992). Dopamine plays a role in the control of emotional processes (Wasowicz, 2001).	• Lesions of the caudate nucleus and putamen, as components of the so-called extrapyramidal system, have been associated with *tremor* (Murdoch, 1990). • Articulatory inaccuracy (Square & Martin, 1994). • Prosodic excess or insufficiency (Square & Martin, 1994; Duffy, 1995). • Excessive, involuntary movements, which causes variability in the locus, speed, constancy, regularity and predictability of movements (Duffy, 1995). • According to Duffy (1995), a deficiency of DA supply to the basal ganglia will result in heightened muscle tone together with an increase in resistance to passive movements. This condition is known as rigidity. In rigidity, movements are stiff and slow which may cause difficulty in the initiation or termination of movements (Duffy, 1995). • Lesions of the basal ganglia are assosciated with abnormalities in the ability to estimate temporal intervals (Meck, 1996). The speed of the internal timing system for motor control seems to be affected (Meck, 1996).
Superior Premotor Area (SPM)	• Increased activity in the left hemisphere (Fox *et al.,* 1996).	• The *"motor programs"* for speech are created in the SPM (Fox *et al.,* 1996). • The SPM is thought to be involved in movement preparation (Fox *et al.,* 1996). • Considering that the premotor area has links with the basal ganglia and cerebellum, and reciprocal connections with the primary motor cortex (Duffy, 1995), then increased activity in the SPM may also affect the activity levels of these structures. The opposite could also be true, that increased activity in the primary motor cortex, basal ganglia or the cerebellum could influence the functioning of the SPM.	• Based on the function of the SPM area, it is likely that increased activity levels in the SPM would affect the preparation of movement.

TABLE 2.10.2: THE SPEECH MOTOR FUNCTIONS AND POSSIBLE MOTOR SPEECH DYSFUNCTIONS OF THE NEURAL SUBSTRATES INDICATED IN MOTOR PROGRAMMING AND STUTTERING *(Continued)*

NEURAL SUBSTRATE	LEVEL OF ACTIVITY IN STUTTERING	SUGGESTED FUNCTIONS OF THE NEURAL SUBSTRATE	POSSIBLE MOTOR SYMPTOMS (INCLUDING SPEECH SYMPTOMS) AS A RESULT OF A DYSFUNCTION OF THE NEURAL SUBSTRATE
Cerebellum	• Increased activity in the left hemisphere (Fox *et al*., 2000). • Reduced activity in the right hemisphere of the cerebellum (Wu *et al.,* 1995).	• The cerebellum refines the smoothness of the contraction of synergist and antagonist muscles (coordination) (Gentil, 1990) by specifying the range, length, force and rate of programmed movement (Houk & Gibson, 1987 in Van der Merwe, 1997). • Precise temporal relationships between muscles for rapid movements are also established by the cerebellum (Murdoch, 1990). • According to Guenther and Perkell (2004) auditory 'targets' / 'goals' are compared to incoming auditory information from the primary auditory cortex by the posterior superior temporal gyrus. The difference between the target and actual auditory signal represents and 'error' signal. The cerebellum transforms the auditory 'error' signal into a motor velocity signal that can act to zero the difference between the target and actual auditory signal (Guenther & Perkell, 2004). Thus, the cerebellum is partially involved in initiating muscle movements; it continually monitors and adjusts motor activities, by using tactile-kinaesthetic and auditory feedback to direct complex movements (Murdoch, 1990). In other words, it performs a regulatory role (Phillips *et al*., 1989) and is suggested to help facilitate motor learning (Murdoch, 1990). • Involuntary or postural movements controlled by the basal ganglia system are also adjusted by the cerebellum in a manner similar to voluntary movements (such as speech). Even reflex actions mediated through the spinal cord or brainstem, are influenced by the cerebellum. The difference between the coordination of voluntary and involuntary movements is the different pathways that are used (Phillips *et al.,* 1989). • The cerebellum plays an important role in the regulation of respiration (Liotti, Brannan & Fox, 2001). This underscores Prescott's (1970 in Ayres, 1980) comment that the cerebellum modulates functions subserved by the limbic system. (Also see Limbic System, Table 2.10.3).	• Dysfunction of the cerebellum has been associated with intention tremor, discoordinated and disorganized muscular activity, disorders of muscle tone and posture (Murdoch, 1990). • Speech symptoms are characterized by a breakdown in the articulatory and phonatory-prosodic aspects of speech (Murdoch, 1990). Related articulatory characteristics are imprecise consonants, vowel distortions, errors of direction and range of articulatory movements (Kent, Netsell & Abbs, 1979; Hirose, 1986 in Murdoch, 1990). Irregular articulatory movements are associated with a disturbance in the initiation of muscle activity in repetitive movements (Hirose, 1986 in Murdoch, 1990). • The prosodic features can be either insufficient or excessive (Yorkston *et al*., 1988; Brown, Darley, Aronson, 1970 in Murdoch, 1990). Insufficient prosody involves the loss of stress, rhythm, and inflection (monopitched/monoloudness) of speech utterances (Brown *et al.* 1970 in Murdoch, 1990). Prosodic excess is associated with variations of excessive and equal stress; prolonged sounds, prolonged intervals and slow rate.
Primary Motor Mouth Area	• The primary mouth area is equally activated in both hemispheres (Braun *et al*., 1997; Fox *et al.,* 1996). • Increased activity in the frontal motor cortex of the right hemisphere was observed during silent reading (De Nil *et al*., 2000).	• The *primary motor area (M1)* is directly responsible for controlling the speech muscles. It controls the contractions of individual muscles for voluntary motor activities (Murdoch, 1990), preprogrammed at subcortical levels through afferent (sensory) connections (Square, Roy & Martin, 1997). This activity is stronger on the left hemisphere in normally fluent speakers, suggesting left brain control of speech (Fox *et al*., 1996).	• Speech symptoms from a dysfunction in the primary motor mouth area (i.e. a lack of dominance or reversed dominance) may manifest as a deregulation of oral movements (Square & Martin, 1994).

TABLE 2.10.3: THE SPEECH MOTOR FUNCTIONS AND POSSIBLE MOTOR SPEECH DYSFUNCTIONS OF THE NEURAL STRUCTURES INDICATED IN MOTOR PROGRAMMING AND STUTTERING *(Continued)*

NEURAL SUBSTRATE	LEVEL OF ACTIVITY IN STUTTERING	SUGGESTED FUNCTIONS OF THE NEURAL SUBSTRATE	POSSIBLE MOTOR SYMPTOMS (INCLUDING SPEECH) AS A RESULT OF A DYSFUNCTION OF THE NEURAL SUBSTRATE
Limbic System, especially the Anterior Cingulate Cortex (ACC)	• Increases in stuttering are related to blood flow increases in the inferior anterior cingulate (as well as certain subcortical nuclei, namely the putamen and ventral thalamus) in the left hemisphere during a sentence construction task (Braun *et al.,* 1997). De Nil *et al*. (2000) also found increased activity in the left hemisphere, which included the limbic system, during silent reading.	• Subcortical motor nuclei are closely linked (anatomically and functionally) to the limbic system. The limbic system includes the orbital frontal region, the cingulate gyrus, and the medial portions of the temporal lobe (Duffy, 1995). Together these structures play an important role in subserving emotional activity. Stimulation of these structures of the limbic system may influence the subcortical motor nuclei due to nerve substrates or networking (Zraick & LaPointe, 1997). • The limbic areas of the cortex do not only influence the emotional aspects of experience, but are also involved in the regulation of memory and learning and the modulation of motivation (Murdoch, 1990). The limbic system is also thought to operate in association with autonomic functions (Murdoch, 1990). • The anterior cingulate cortex (ACC) forms the vocal fold control pathway to the PAG in the midbrain associated with non-verbal emotional vocal utterances (Jurgens & Zwirner, 1996). • Besides the PAG, the ACC also has neural connections with the amygdala, the autonomic brainstem nuclei and with motor systems, including the primary motor cortex and striatum (Dum & Strick, 1993; Devinsky, Morrell & Vogt, 1995). The ACC forms part of an adaptive brain circuit, which includes the amygdala, involved in automatic respiratory control and conscious perception of air hunger (Brannan, 2001). Liotti *et al.* (2001) confirmed this association between ACC and respiratory control by indicating that the ACC has a key role in mediating conscious awareness of breathlessness (Liotti *et al.,* 2001). • The ACC is part of an attention system that selects information for action (Dum & Strick, 1993; Devinsky *et al*., 1995) and motor initiation (Wood *et al.,* 1980; Goldberg, 1985). The activity in the ACC increases when motor tasks are new and effortful, as opposed to familiar overpractised motor tasks (Paus, Petrodes, Evens & Meyer, 1993). If the expected output does not match the actual output (see 2.4.1.2.), the cingulate cortex brings the current motor program to a halt (Gray, 1995). • The ACC, areas of the thalamus and the cerebellum are activated milliseconds before the onset of speech (Logan, 1999). This suggests that these areas are major contributors to speech initiation (Logan, 1999).	• According to Zraick and LaPointe (1997), when neural structures underlying the limbic system are unable to regulate the systems' function in the presence of enhanced subcortical excitation, the result may be partial disinhibition of the subcortical nuclei. The temporary excitation of the subcortical motor nuclei may result in disturbances of muscle tone and involuntary movements (Zraick & LaPointe, 1997). • Duffy (1995) stated that a dysfunction would probably affect the prosodic-emotional character of speech. Speech may be characterized by insufficient, exaggerated, or distorted prosody (Duffy, 1995). Thus, the speaker may have difficulty conveying vocally his/her emotional state.

2.4.2.2 A Parallel between Stuttering and Motor Programming Disorders

2.4.2.2.1 Speech Symptoms associated with Motor Programming Disorders

A motor speech disorder involving the motor programming level, as depicted by Van der Merwe (1997), would imply a defect in the programming of muscle tone, rate, direction and range of movements required for speech production. An impairment of repeated initiation and a breakdown in the transmission of successive motor programs necessary for speech production can also be present (Van der Merwe, 1997). The extent of involvement of different neural structures at this level will determine the nature of the programming defects (Van der Merwe, 1997). The most prominent speech symptoms (auditory observable effects) resulting from these motor programming defects, would probably be imprecise sound production (sound distortion), defects in speech rate and/or problems in the initiation of movement (Van der Merwe, 1997). The speech symptoms associated with an underlying motor programming disorder indicate some similarities with those of stuttering, especially with regard to the initiation of voice for speech production (see Table 2.11). Table 2.11 summarizes the speech symptoms characteristic of a breakdown in motor programming that seems to be present in stuttering.

TABLE 2.11: INDICATIONS OF SPEECH SYMPTOMS IN STUTTERING THAT ARE CHARACTERISTIC OF MOTOR PROGRAMMING DEFICITS

		SPEECH CHARACTERISTICS ASSOCIATED WITH MOTOR PROGRAMMING DISORDERS	DIFFERENCES AND SIMILARITIES BETWEEN STUTTERING SYMPTOMS AND THE SYMPTOMS ASSOCIATED WITH A MOTOR PROGRAMMING DISORDER
SPEECH SYMPTOMS ASSOCIATED WITH A MOTOR PROGRAMMING DYSFUNCTION *(according to Van der Merwe, 1997)*	**Sound Distortions**	Sound distortions are related to imprecise movements of articulators due to slower or delayed movements, abnormal variations of fundamental frequency or delayed segmental durations (Kent & Netsell, 1975; Kent *et al*., 1979).	Though, sound distortions have not been reported as primary characteristics of stuttering, the speech motor features (Kent & Netsell, 1975; Kent *et al*., 1979) associated with sound distortions have been reported to occur in stuttering: (a) slower movements of articulators or laryngeal structures (Hutchinson & Watkin, 1976; Van Riper, 1982; Archibald & De Nil, 1999) may result in sound distortions such as devoicing of voiced sounds (Shipley & McAfee, 1992); (b) abnormal perturbations in the fundamental frequency of PWS reflect difficulty in maintaining a fixed (stable) laryngeal posture during vowel production (Newman, Harris & Hilton, 1989); and (c) delayed segmental durations (Kent & Adams, 1989). When PWS are forced to accelerate their speech, the accuracy of their speech movements may decline and possibly result in a less precise manner of articulation, which can be perceived as sound distortions, such as reported by Peters *et al.* (1989) during high time stress conditions.
	Defects in Speech Rate	Defects in speech rate resulting from motor programming disorders are not regarded as compensatory changes in the speaking rate (Van der Merwe, 1997). Thus, changes in speech rate (a slower, faster or more variable rate) may be a general, constant defect in motor activities, which may affect any motor action.	For PWS, slower rates in motor activities do not exclusively involve speech, but manual tasks as well (Webster, 1990). A slower speech rate is associated with increased stiffness in joints due to higher levels of muscular tension (Van Lieshout, Peters, Starkweather & Hulstijn, 1993; Archibald & De Nil, 1999). Archibald and De Nil (1999) posed that due to the speed-accuracy trade-off, it is also possible that PWS's slower speech movements suggest that they adopt a more careful approach in order to compensate for perceived difficulty with task completion. Thus, the PWS were consciously and purposefully speaking at a reduced rate. Speaking rate may vary according to difficulty of the task and is not primary to the breakdown in PWS's speech. Stuttering is thus characterized by a general slowness in motor activities that may be reflected in speech rate.
	Problems in the Initiation of Movements	A breakdown in the preparation of movement can have an influence on the actual initiation of the movement (Phillips *et al.,* 1989). A possible explanation for this poses that the individual tends to rely less upon the advance preparation of movement and more upon feedback guidance (Phillips *et al.,* 1989). A breakdown in the integration of peripheral feedback and specification of movements by the efference copy (which is processed by the motor programming level) may thus lead to problems in initiation of movements (Gracco & Abbs, 1989).	Since most moments of stuttering occur on *initial* gestures (Van Riper, 1982), excessive tension in some PWS's speech musculature is noted *prior to* execution (Bloodstein, 1995), and aberrant articulator positioning *precedes* speech initiation in PWS (Zimmermann, 1980a), suggest that the period before speech movements may be a time in which aberrant inputs are likely to occur in PWS. Longer (slower) initiation times associated with stuttering (Prosek *et al.,* 1979; Reich *et al.,* 1981; Hayden, Adams & Jordahl, 1982) may be the result of a breakdown in preparation of movement (Phillips *et al.,* 1989). Defects in the preparatory process of movement also have an effect on postural adjustments (Phillips *et al.,* 1989). A suggestion has been made that some of the abnormalities of muscular postural adjustments to the larynx, such as seen in PWS, may be the result of a breakdown between the coordination of vocalization and defensive respiratory reflexes during preparation of voluntary speech production (Parry, 1994). However, this proposal by Parry (1994) has not been empirically validated.

2.4.2.2.2 A Comparison between Stuttering and a Motor Programming Disorder with Basal Ganglia Involvement

Stuttering presents with specific speech motor symptoms that relate to motor speech disorders of neurologic origin. To develop an understanding of stuttering as a motor speech disorder, it is imperative to gain knowledge of the neural basis of speech motor control (Fox *et al.*, 2000; Max *et al.*, 2004). Speech motor symptoms due to basal ganglia involvement (such as *hyperkinetic dysarthria*) are associated with a breakdown in motor programming (Van der Merwe, 1997). Recent neurophysiological studies showed similarities between stuttering and motor disorders involving the basal ganglia (and associative structures), such as Tourette's syndrome (Abwender *et al.*, 1998) and hyperpolarization of dopamine (Rastatter & Harr, 1988).

Table 2.12 shows that both hyperkinetic dysarthria and stuttering evidence problems in the precise timing and sequencing of movements pertaining to aspects of rate of movement, initiation and regulation of muscle tone, coordination of movements, and involuntary movements. Similarities in acoustic characteristics of phonation, respiration, articulation and prosody are the result of the spatio-temporal aspects involved in these disorders. Reflecting on the parallels between stuttering and motor speech disorders with basal ganglia involvement with regard to physiological and acoustic features (see Table 2.12) contributes to the possibility of motor programming involvement in stuttering. Furthermore, a possible relationship between these physiological (motor speech) symptoms and stuttering severity has been indicated in several studies (Borden, 1983; Watson & Alfonso, 1987*b*; Archibald & De Nil, 1999).

TABLE 2.12: PARALLELS BETWEEN A MOTOR SPEECH DISORDER WITH BASAL GANGLIA INVOLVEMENT (SUCH AS HYPERKINETIC DYSARTHRIA) AND STUTTERING

HYPERKINETIC DYSARTHRIA *(Darley, Aronson & Brown, 1975; Kent & Netsell, 1975; Kent et al., 1979; Phillips et al., 1989; Murdoch, 1990)*	**STUTTERING**
PHYSIOLOGICAL BEHAVIOURS	
▪ Involuntary movements such as tremor, as well as abnormal involuntary contractions of the limbs, trunk, neck, face and muscles of the speech mechanism; ▪ A disturbance of rhythm and rate of motor activities that is characterized by abrupt, intermittent, frequent, compulsive movements involving a relatively small part of the body. ▪ A problem with coordinating more than one movement at a time, which is indicative of a problem in the sequencing of movements; ▪ A generally high degree in variability of motor control; ▪ A problem in the initiation of movements.	▪ Involuntary movements such as tremor-like speech muscle activity (*Van Riper, 1982; Denny & Smith, 1992),* excessive muscular activity *(Freeman & Ushijima, 1978)*, abnormal associated movements of the limbs, head, face, neck and trunk *(Lebrun, 1997)*; ▪ The disturbance of rate of motor activities is qualified as a generally slower motor response rate *(Williams & Bishop, 1992);* ▪ A specific impairment in multiple movement coordination *(Caruso, Abbs & Gracco, 1988)* and the regulation of timing *(Kent & Adams, 1989)*. Conditions that disrupt coordination in stuttering are associated with rate of repetitive movements, precision of motor and perceptual timing and force control *(Keele, Ivry & Pokorny, 1987)*; ▪ PWS appear to have an underlying deficiency in the precise sequencing and timing of muscle movements of speech *(Borden, 1983; De Nil, Kroll & Houle, 2001)* independent of the phonological system (*MacKay & MacDonald, 1984*). Thus, PWS are less precise in temporal patterning of a sequence of movements and rhythm of movements *(Van Riper, 1982; Borden, 1983)*; ▪ Inconsistent with preparation and initiation of speech and manual responses *(Webster, 1990; Williams & Bishop, 1992)*. This is reflected in general increased latency of movement onset. ▪ Problems in maintaining postural adjustments are indicated by vocal tract instability *(Robb, Blomgren & Chen, 1998).*
ACOUSTIC CHARACTERISTICS OF SPEECH PRODUCTION PROCESSES	
Laryngeal-Phonatory Characteristics: Intermittently harsh voice; Strained-strangled voice quality; Distorted vowels; Irregular pitch fluctuations; Vocal arrests; Monopitch; Excess loudness variations; Monoloudness.	*Laryngeal-Phonatory Characteristics: (Borden et al., 1985; Shipley & McAfee, 1992; Bakker & Brutten, 1989)* Delays in phonatory onset; Hard glottal attacks; Pitch breaks; Abnormal variations in pitch; Vocal stoppages; Too loud/soft voice; Alternating loudness.
Respiratory Characteristics: Sudden forced inspiration or expiration; Transient breathiness.	*Respiratory Characteristics: (Bloodstein, 1995)* Abnormal variations in loudness; Audible inhalation and/or exhalation; Arrhythmic breathing; Prolonged expirations or inspirations.
Articulation: Imprecise consonants (associated with reduced precision in articulatory movements); Vowel distortion; An irregular breakdown in articulation.	*Articulation: (Hutchinson & Watkin, 1976; Borden et al., 1985*; *Van Lieshout et al., 1993)* Easy or hard articulatory onset; Irregular articulatory breakdown*;* Stiffness in articulatory movements is related to higher levels of muscular tension*;* Lip tremors.
Prosody: Defects in speech rate; Inappropriate silences; Short phrases; Prolonged intervals; Prolonged phonemes; Excessive, equal or reduced stress on commonly unstressed words; Stuttering-like repetitions.	*Prosody: (Van Riper, 1982; Shipley & McAfee, 1992)* Silent pauses within words or phrases; Prolonged sound productions; Excessive or atypical stressing; Slower speaking rate; Repetitions

2.4.3. Conclusive remarks: Similarities between Stuttering and a Motor Programming Disorder

The above discussion has focused on (a) neural structures and their complementary functions involved in stuttering; (b) the similarities between characteristics of stuttering and those of speech disorders with basal ganglia involvement; and (c) the view that the most pronounced symptoms of a motor programming disorder (including sound distortion, speech rate, and problems in initiation of movement) appear to be present in stuttering. The factual evidence reviewed in this discussion supports the possibility that stuttering is associated with a breakdown in motor programming (Van der Merwe, 1997).

Viewing stuttering as a motor speech disorder with aberrant laryngeal behaviour, suggests that the nature of stuttering is associated with a disruption in motor features that influence laryngeal behaviour. In other words, a disruption in the timing or sequencing of coordinated muscle movements results in the abnormal laryngeal behaviour. A breakdown in motor programming is thus implied (Van der Merwe, 1997). Conversely, if stuttering is regarded as a motor speech disorder due to inadequate control of the larynx, then a defect in motor execution of speech is indicated (according to Van der Merwe's (1997) model). Since the exact nature of laryngeal (vocal fold) dynamics in the PWS has elicited different arguments (such as mentioned above), a better understanding of laryngeal behaviour associated with stuttering is called for.

2.5 SUMMARY OF CHAPTER 2

In Chapter 2 vocal motor control skills of PWS received more emphasis due to existing speculations regarding the relationship between laryngeal behaviour and stuttering. Conceptions regarding the relationship between laryngeal behaviour and stuttering were discussed. Viewing aberrant laryngeal behaviour as an isolated feature of stuttering was rejected. Emphasis was given to laryngeal (dys)function within a breakdown of intersystem coordination between respiratory, phonatory and articulatory movements as the prominent characteristic in stuttering . In this regard, characteristic laryngeal behaviour during speech initiation of PWS and normal speakers were discussed in conjunction with reviews of relevant studies performed in this regard. These studies revealed a need for further research into the laryngeal behaviour during speech initiation in stuttering. The purpose of such research will be to provide an accurate description of the aberrancies in voice initiation that seems to

be characteristic of PWS's speech production. Studying laryngeal onset time and voice onset time by means of acoustic and physiologic (such as electroglottographic) measurements, can therefore be useful to gain knowledge and further our understanding regarding the temporal and coordinative characteristics underlying PWS's voice initiation. According to most studies voice initiation is associated with abnormalities in the timing of laryngeal initiation, synchronization of laryngeal movements and sequencing of laryngeal, articulatory and respiratory movements in PWS's speech. These findings suggest that the nature of stuttering involves an underlying breakdown in speech motor control. The primary symptoms of stuttering might thus be better understood in the context of speech motor control. Stuttering was defined as a possible disorder of motor programming according to Van der Merwe's (1997) *Theoretical framework for the characterization of pathological speech sensorimotor control.* This viewpoint was substantiated by previous theories depicting stuttering as a motor programming disorder, comparisons of neural structures involved in stuttering and motor programming, as well as similarities between stuttering and other motor programming disorders. A possible relation between stuttering and characteristics of a dysfunction in motor programming may direct current research in understanding prominent motor speech symptoms of stuttering, such as difficulty in initiating voicing.

CHAPTER 3: RESEARCH METHOD

3.1 INTRODUCTION

The review of the literature as presented in Chapters 1 and 2 focused on laryngeal behaviour in stuttering. According to existing literature, deviancies in the timing and sequencing of speech motor activities (especially during onset of speech) are inherent to stuttering behaviour. Few studies, however, have undertaken a systematic quantitative analysis of vocal fold behaviour during speech initiation of persons who stutter (PWS). Consequently, there is a need for more research into the timing of laryngeal movements in PWS by means of complementary instrumental measurements, including both physiological and acoustic measurements. Results obtained from such measurements will lead to an accurate description of vocal fold characteristics of PWS's speech. Thus, these results will contribute to a description of the nature of the underlying motor speech dysfunction in stuttering.

The purpose of Chapter 3 is to present the research method of this study which focused on a description of the nature of vocal fold behaviour during speech initiation of PWS. This chapter outlines and describes the research aims, research design, subject selection criteria, instrumentation, material, data collection procedure and finally the data analysis and processing used in this study.

3.2 RESEARCH AIMS

3.2.1 Main aim

The main aim of this study was to determine the nature of vocal fold behaviour during voiced and voiceless speech sound initiation in a group of stuttering subjects, as compared to that of a group of normally fluent speakers.

3.2.2 Sub-aims

The sub-aims focused on different parameters of vocal fold activity during the initiation of voiced and voiceless speech utterances in persons who stutter and normally fluent speakers.

3.2.2.1 Sub-aim 1

To examine and compare the nature of physiological vocal fold activity during speech initiation of voiced and voiceless speech sounds in persons who stutter and a group of normally fluent speakers.

- **Sub-aim 1.1:Nature of prephonatory vocal fold activity during speech initiation**
  - To identify and classify types of prephonatory vocal fold activity.
  - To determine the frequency of occurrence of different types of prephonatory vocal fold activity.
  - To examine the effect of phonetic context on prephonatory vocal fold activity.

- **Sub-aim 1.2: Laryngeal onset time (L_xOT)**
  - To determine laryngeal onset time.
  - To examine the effect of phonetic context on laryngeal onset time.

3.2.2.2 Sub-aim 2

To examine and compare the nature of acoustically detected vocal fold activity during speech initiation in persons who stutter and a group of normally fluent speakers.

- **Sub-aim 2.1: Nature of voice onset energy during speech initiation**
  - To identify and classify different types of voice onset energy.
  - To determine the frequency of occurrence of different types of voice onset.
  - To examine the effect of phonetic context on voice onset.

- **Sub-aim 2.2: Voice onset time (VOT)**
  - To determine VOT of initial voiced and voiceless plosives.
  - To examine the effect of phonetic context ($C_{(v)}$VC and $C_{(vl)}$VC) on VOT.

3.3 RESEARCH DESIGN

The nature of this research project was both qualitative and quantitative. In the present study,

qualitative data were collected by perceptual analysis of the electroglottographic (EGG) signal as well as the acoustic speech signal. Quantitative data were obtained by measuring durational values from the EGG and acoustic speech signal.

The purpose of the study was to provide a detailed description of vocal fold behaviour during speech initiation for individuals who stutter. A multi-case study design, which included a control group and multiple case studies, was used. Multiple case studies were used to obtain in-depth information on vocal fold activity during the initiation of voiced and voiceless speech utterances in PWS. Employing a descriptive and exploratory strategy may in turn provide insights into new directions for investigation in this field (Leedy, 1997).

Since individuals who stutter tend to display idiosyncratic speech motor behaviour (Van Riper, 1982, Silverman, 1992), it makes the generalization of the unique vocal fold behaviour of each subject difficult. Thus, the aim of the research was rather to describe the uniqueness of PWS's vocal fold behaviour during speech initiation.

Three phonetic contexts were used which differed in initial voicing demands (i.e. a *vowel onset* in a vowel(V)-consonant(C) context, a *voiced consonant onset* in a $C_{(v)}VC$ context and a *voiceless consonant onset* in a $C_{(vl)}VC$ context). The effect of different phonetic contexts on laryngeal (vocal fold) behaviour during speech initiation in each of the stuttering subjects, was observed and interpreted in comparison with vocal fold behaviour of the control group. The observations included specific temporal parameters, such as the duration of vocal fold approximation until the onset of vibratory movements and the temporal sequencing of vocal fold activity and articulatory movements. The vocal fold behaviour (including the temporal parameters) evident in the subjects was thus the dependent variable in the study. Figure 3.1 is a schematic presentation of the research method.

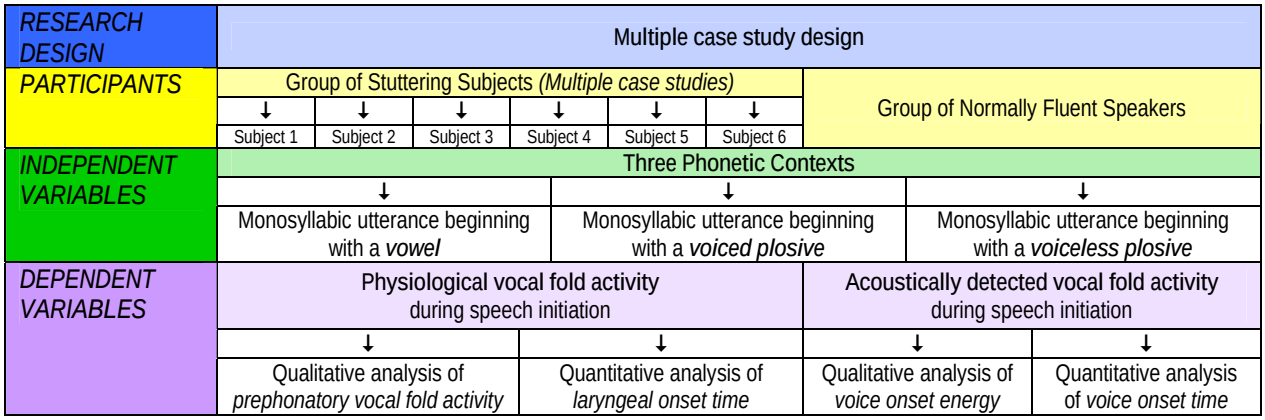


FIGURE 3.1: A SCHEMATIC PRESENTATION OF THE RESEARCH METHOD

3.4 PARTICIPANTS

Normally fluent speakers and individuals who stutter were included in the study. The normally fluent speakers as a group will be referred to as the control group, whereas the individuals who stutter will be referred to as the subjects (S1, S2, S3, S4, S5 and S6). The individual subjects also formed part of the subject group.

3.4.1 Selection criteria for participants

The criteria for selection of the individuals who stutter (subjects) and normally fluent speakers (controls) are discussed below. The criteria were adapted from Borden *et al.* (1985) to assure a degree of consistency among the selection criteria of the current study and those used in other studies. This will render more reliable comparisons between the results of the current study and other similar studies. The criteria for all participants (subjects and controls) to be included in the study were as follows:

3.4.1.1 General criteria for subjects and controls

All of the individuals participating in the current study had to comply with the following criteria:

- **Hearing**

All participants had to exhibit normal peripheral hearing with a pure tone threshold of ≤15dBHL. A hearing loss can influence speech production (Fitch, 1990).

- **Vision**

Each participant's visual ability must be perceived as normal or non-limiting. The testing material is presented in a reading format.

- **Language**

All participants had to be first language Afrikaans speakers. Since the testing material is language-based, the inclusion of first language Afrikaans speakers enables the use of the same material across all individuals. Also, language proficiency of the participants may influence their familiarity with the productions. The occurrence of repetitions and/or blockages is reduced when the utterance is familiar to the speaker, as opposed to unfamiliar productions

(Van Riper, 1982). For controlling this contextual factor (familiarity of the utterance, see 2.4.1.1), the subjects and controls were required to be competent in speaking Afrikaans.

- **Reading competence**

The individuals' reading competence had to enable them to read assigned material orally.

- **Age**

The ages of the participants ranged between 20 and 45 years, which is the period after puberty and before age-related changes can interfere with speech production (Melcon, Horr & Hixon, 1989). The age range was considered important since speech-timing control seems to be affected by maturation (Kent, 1976). Variability in speech-timing control due to immaturity of the neurological system is hereby controlled. Speech development must have reached maturity to ensure that the samples were representative of adult speech. A norm for normal adult speech production could be accurately established which will be useful to identify abnormalities in speech production as evident in PWS.

- **Gender**

The thyroid cartilage is more prominent in males than in females (Borden, Harris & Raphael, 1994), which enables better placement of the electrodes of the electroglottograph and thus, results in more reliable recordings. Also, based on availability of subjects, the greatest percentage of PWS are male (Kehoe, 1998). Consequently, only male participants were selected.

- **Weight**

For the purpose of eliminating all factors which may influence the electroglottographic recordings, all participants had to be within normal weight limits. The amount of fat tissue around the neck interferes with the current flow between the electrodes of the electroglottograph, and reduces sensitivity of registering vocal fold contact (Behrman, Agresti, Blumstein & Lee, 1998).

- **History of speech-language pathology or related dysfunctions**

Each participant had to exhibit normal orofacial structures as subjectively determined by a qualified speech-language therapist. Individuals were excluded from the study if they, according to self-reports, had a previous or current history of speech or voice problems (*other than stuttering*), laryngeal injury or surgery, anterior neck trauma, a diagnosed neurological

condition, or a chronic respiratory infection (Goozée, Murdoch, Theodoros & Thompson, 1998). These pathological conditions may interfere with the vocal fold activity reflected on the electroglottographic signal (L_x signal). Individuals acting as controls had to adhere to all these expectations and present with normal speech. Individuals that were to be included as subjects of this study, also had to comply with these criteria - except for the presence of stuttering.

3.4.1.2 Criteria for inclusion in the control group

Individuals with normal speech and language abilities comprised the *control group*, which served as a *comparison group* in the present study. The individuals included in the control group were age and gender matched with the stuttering subjects.

3.4.1.3 Subject selection criteria

The individuals included as a subject of this study had to comply with specific criteria which are used in the diagnosis of stuttering. For an individual to be included in this study, the following criteria were used:

- Subjects had to be formally diagnosed with stuttering by a qualified speech-language therapist with clinical experience in the assessment and diagnosis of fluency disorders. Speech characteristics had to include the following:
  - The subject had to exhibit *repetitions* of sounds, syllables or monosyllabic words (Peters & Guitar, 1991).
  - The subject had to exhibit *prolongations* of voiced or voiceless sounds (Van Riper, 1982). Prolongations can also include stopped movement of articulators (Conture, 1990).
  - The subject had to exhibit *blocks* which could have occurred at any level of the speech mechanism – respiratory, laryngeal or articulatory. Blocks involve the inappropriate stop of airflow or voicing, and articulatory movement with associated tenseness (Peters & Guitar, 1991; Silverman, 1992).

- The onset of stuttering behaviour had to be from childhood and not be associated with an acquired condition. According to the developmental levels of stuttering

proposed by Peters and Guitar (1991), which is based on (a) core behaviour, (b) secondary behaviour and (c) feelings and attitudes, PWS above the age of 14 years are fit into the *advanced stuttering level*. Since the criteria for age range was above 14 years (as previously mentioned) and stuttering onset had to be developmental of origin, all the subjects that were included qualified for the advanced stuttering level.

- Since the subjects' level of stuttering severity may range from very mild to severe, the stuttering severity had to be determined for each subject. The *Stuttering Severity Index* (*SSI*) (Riley, 1972) was used for this purpose.

- Individuals who stutter that were included into the subject group had to indicate whether they received previous stuttering therapy or were enrolled in any therapy programme during the time of the data collection. Since the subjects might have been enrolled in varying amounts and kinds of therapy, the possible influence of therapy on the subjects' speech production could not be controlled. Knowledge of whether or not a subject was enrolled in a therapy programme may be useful when interpreting the subjects' results on vocal fold behaviour during speech initiation.

3.4.1.4 Criteria for classification of subjects

The *Stuttering Severity Index (SSI)* (Riley, 1972) was performed to determine the severity of stuttering for each subject. The following material and instrumentation were used for recording and scoring of the data for subject selection. Instrumentation used in the administration of the *SSI* (Riley, 1972) is illustrated in Figure 3.2.

3.4.1.4.1 Material for classification of subjects

The severity classification was based on measures of a 100-word sample of the subject's conversational speech and oral reading of a phonetically balanced passage, *"Na die Wildtuin"* (Pienaar & Hooper, 1941) (Appendix A).

- **Videotape recording for selection and classification of subjects**

A National Panasonic MS 20 video camera and BASF E-200-video cassettes were used for the video-recordings of the stuttering subjects during speech tasks (including oral reading and conversational speech). For the visual-auditory analysis of the *SSI* (Riley, 1972), a Sony

Betamax SL-F60 PAL video machine and Telefunken Pal Color 1510 monitor were used.

- **Audiotape recording for selection and classification of subjects**

The subjects' speech samples were recorded on a TDK D 60 minute magnetic cassette using a Nakamichi 550 Versatile Cassette System.

3.4.1.4.2. Procedure for classification of subjects

Classification of subjects according to stuttering severity has been used in other instrumental studies such as Borden *et al.* (1985) as well as Peters and Boves (1988). The severity of stuttering may be associated with results of timing measurements of vocal fold behaviour in the different subjects (Archibald & De Nil, 1999).

- **Data-collection procedure for classification of subjects**

Both videotape recordings and audiotape recordings were used to perform the *SSI* (Riley, 1972) as recommended by Peters and Guitar (1991). The video camera was positioned 4 meters from the subject on a tripod stand and was focused on the speaker's head and upper body extremities. The recordings for subject selection and for data collection for the study were completed in the same session. Both recordings were done in a sound-attenuated room. The data for the *SSI* were recorded after the data collection for the study was performed, to avoid the effect of the former procedure on the main data. Since these recordings allowed for repetitive analysis of observed behaviour, the scoring was more accurate as opposed to on-line ratings (Peters & Guitar, 1991).

- **Data-analysis procedure for classification of subjects**

100-word samples of the subjects' conversational speech and oral reading were used in the combined ratings of three relevant aspects of speech behaviour: (a) frequency of stuttering events, (b) duration of stuttering events; and (c) physical concomitants (Riley, 1972). The severity rating was according to the *SSI* scoring protocol (Riley, 1972) done by a speech-language therapist with 5 years of clinical experience. Each subject was classified as presenting with a very mild, a mild, a moderate, a severe or a very severe stuttering disorder by the *SSI* (Riley, 1972).

3.4.2 Description of participants

Six adult male normally fluent speakers who met the inclusion criteria were selected to act as normal speaking controls in the study. The subject group consisted of six adults male stuttering speakers. The individuals who stutter that were included as subjects into the study, were referred by the Speech Therapy Department, One Military Hospital and the Communication Pathology Department, University of Pretoria. These subjects were professionally diagnosed as developmental stuttering speakers. The subject's participation in the study was based on informed consent. According to the *SSI* (Riley, 1972), stuttering severity of the individuals in the subject group varied from very mild (1 subject), mild (2 subjects), moderate (1 subject) to severe (2 subjects). The age range of the control group was 21 years to 32 years (mean age = 27 years) and the age range of the subject group was 22 years to 42 years (mean age = 28 years). Only one subject was not a first language Afrikaans speaker, but a bilingual English-Afrikaans speaker. None of the subjects reported a history of neurologic injury or disease, laryngeal pathology, speech (except for stuttering) or voice problems, respiratory disease, or hearing problems. Descriptive participant data are presented in Table 3.1.

TABLE 3.1: DESCRIPTIVE DATA FOR THE INDIVIDUALS IN THE CONTROL GROUP AND SUBJECT GROUP

		DESCRIPTION OF PARTICIPANTS									
									CLASSIFICATION OF SUBJECTS		
		Gender	Age (in years)	Language	Hearing	Vision	Weight	Previous Stuttering Treatment	Developmental Level of Stuttering	Stuttering Severity Index (SSI): Score	Stuttering Severity Index (SSI): Classification
CONTROL GROUP (N = 6)	Control 1	Male	21	Afrikaans	WNL	WNL	WNL	Not applicable	Not applicable	Not applicable	Not applicable
	Control 2	Male	21	Afrikaans	WNL	WNL	WNL				
	Control 3	Male	25	Afrikaans	WNL	WNL	WNL				
	Control 4	Male	32	Afrikaans	WNL	WNL	WNL				
	Control 5	Male	32	Afrikaans	WNL	WNL	WNL				
	Control 6	Male	31	Afrikaans	WNL	WNL	WNL				
SUBJECT GROUP (N = 6)	Subject 1	Male	30	Afrikaans	WNL	WNL	WNL	Yes	Advanced	23	Moderate
	Subject 2	Male	24	Afrikaans	WNL	WNL	WNL	Yes	Advanced	35	Severe
	Subject 3	Male	42	Afrikaans	WNL	WNL	WNL	Yes	Advanced	36	Severe
	Subject 4	Male	24	Afrikaans	WNL	WNL	WNL	Yes	Advanced	21	Mild
	Subject 5	Male	22	Bilingual	WNL	WNL	WNL	Yes	Advanced	21	Mild
	Subject 6	Male	23	Afrikaans	WNL	WNL	WNL	Yes	Advanced	13	Very Mild

Key: ***SSI:*** *Stuttering Severity Index (Riley, 1972)* ***WNL:*** *Within Normal Limits*

3.5 ETHICAL ISSUES

Ethical aspects should be considered in all research that involves participants (Leedy, 1997). Ethical aspects such as voluntary participation, informed consent and anonymity of participants received attention in this study. The ethical issues of the study had to be reviewed by the Ethics Committee of the University of Pretoria. The letter from the Ethics Committee is included in Appendix B(*i*).

All participants were given the right to freely choose to subject them to the scrutiny inherent in this research project. A letter of consent was compiled which described what participation in the project would involve and what demands might be made on the participant. The letter also explained the procedure for data recording and assessment. Furthermore, it was stated that the identities of the participants would be kept anonymous, and that a subject could withdraw from the study at any time. The information in the letter was conveyed verbally to each potential subject. Each potential subject had to give verbal consent to allow participation in the study. Each of the participants also had to give permission that the recorded data could be used for research purposes. Informed consent was given before the recording commenced. The letter of consent is included in Appendix B(*ii*).

3.6 INSTRUMENTATION

The instrumentation used in the study is diagrammed in Figure 3.2. Different instruments were used in the different research steps and will be discussed separately.

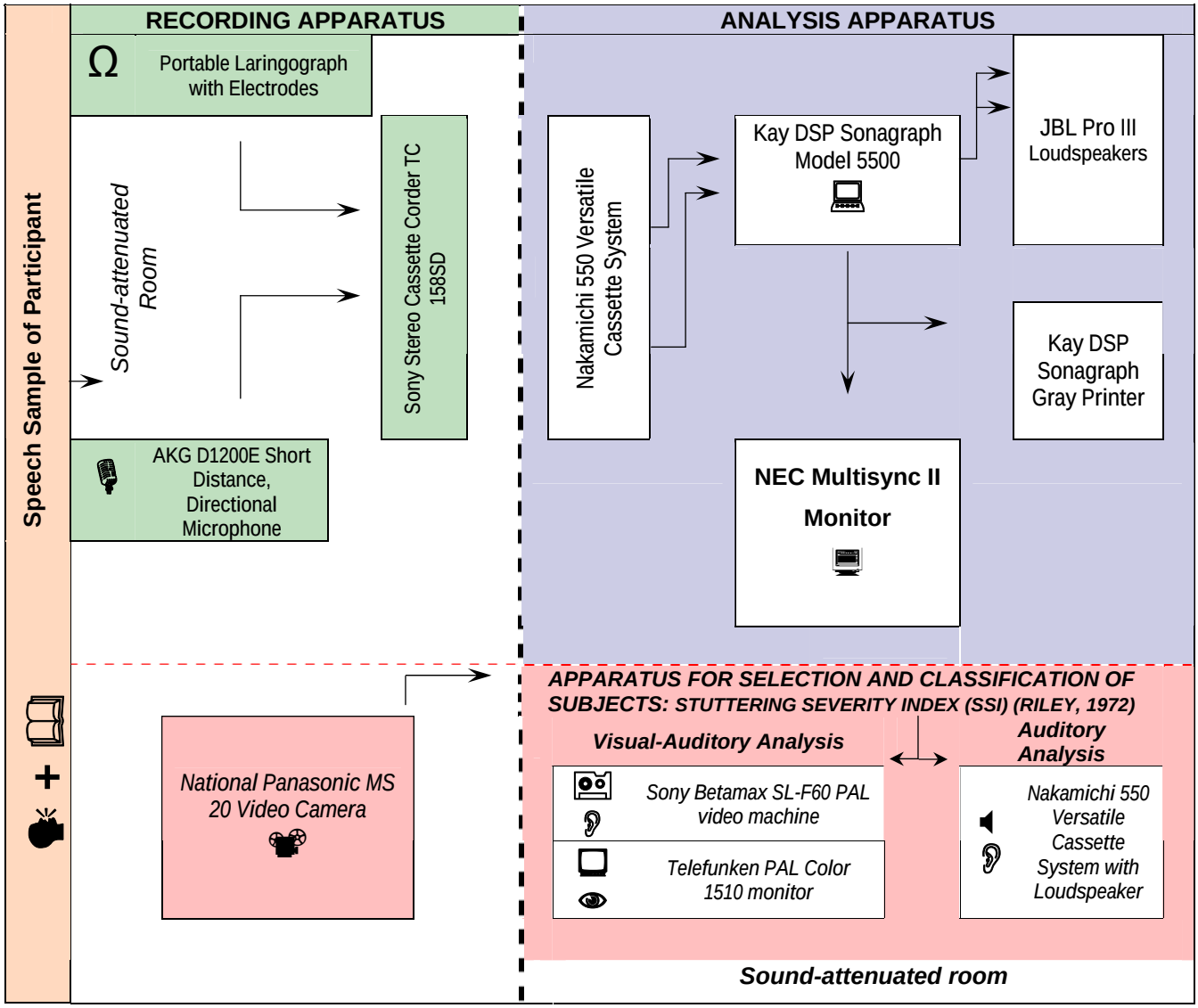


FIGURE 3.2: A SCHEMATIC REPRESENTATION OF THE INSTRUMENTAL SET-UP FOR SUBJECT SELECTION, DATA COLLECTION AND PROCESSING

3.6.1 Instrumentation for data collection and analysis

- **Electroglottograph (EGG) and electrodes**

The electroglottograph senses changes in the electrical resistance of the neck in the region of the larynx. These changes in the electrical resistance are referred to as the L_x signal and provide information on patterns of vocal fold contact or vocal fold movement.

The L_x signal is picked up by a pair of gold-plated electrodes placed on a participant's skin, one at each side of the neck at the level of the thyroid cartilage and recorded by a Portable Electro-Laryngograph (manufactured by Laryngograph Ltd.). The electrodes were held in place with an elastic band fixed around the individual's neck.

- **Electro-Voice Microphone Model 631B**

A second signal, i.e. the acoustic pressure wave, was recorded simultaneously with the L_x signal, with the aid of a microphone. The speech signal obtained by the microphone, is the acoustic product of glottal as well as articulatory activity. The short-distance, directional microphone was used to record the acoustic signal.

- **Recording of signals**

A Sony Stereo Cassette Corder TC-158 SD with a Sher-O-Dyne microphone was used for the recording of the speech samples. The material was recorded on a TDK D 60 minute magnetic cassette in a sound-attenuated room. The two signals (electroglottographic and acoustic) were recorded via two different input channels of the Kay DSP Sonagraph.

- **Kay DSP Sonagraph Model 5500**

A digital signal analyzer was used for data collection and processing. The L_x signal and acoustic speech signal were monitored during the recordings by means of a dual waveform display on the Kay DSP Sonagraph 5500.

- **Computer monitor**

A visual presentation of the speech signal and L_x signal was displayed on the NEC Multisync II screen to monitor the quality of the recordings.

- **Other instrumentation for signal analysis**

A Nakamichi 550 Versatile Cassette System was used to record the speech signal to relay it to the Kay DSP Sonagraph Model 5500 where the recorded signals were captured and digitally processed and analyzed. The speech signal was monitored with two JBL Pro III loudspeakers and displayed on a NEC Multisync II display screen. A dual display of the two recorded signals on the Kay DSP Sonagraph with the time cursors and a time axis, were used to obtain all required measurements of the electroglottographic and acoustic signals. The Kay DSP Sonagraph Gray Scale Printer Model 5510 provided hard copies of the signal displays and completed the instrumental set-up.

3.7 MATERIAL

The material for this study was selected to enable an investigation into the characteristics of vocal fold behaviour in PWS. The following aspects were taken into consideration for the selection of the speech material:

- The speech material was selected for the purpose of examining voicing behaviour during speech initiation in PWS. The choice of specific speech sounds was motivated by the need for a more comprehensive study of speech initiation, specifically pertaining to vocal fold behaviour in stuttering.
- Repetitions of the speech material were necessary to establish whether certain vocal fold behaviour was characteristic of a specific subject or only incidental. Since the adaptation feature in stuttering may induce fluency and consequently may influence the subjects' way of speech production, these repeated trials did not follow successively but in random order.
- Descriptions of contextual conditions that may affect fluency (such as motor complexity of sound structure) should be included in the selection criteria of the material (Watson & Alfonso, 1987*a*; Alfonso, 1990).

3.7.1 Syllable structure

3.7.1.1 Selection of syllable structure

The speech material consisted of three groups of one-syllabic units that differed in voicing features of the initial sound (i.e. voiced or voiceless). The selection of these syllables was based on specific syllable structures, i.e.: *vowel-consonant* (VC) and *consonant-vowel-consonant* (CVC). Table 3.2 contains the characteristics of the three phonetic contexts according to voicing characteristics. A description of the preparatory vocal fold activity for speech initiation of voiced and voiceless sounds based on the physiology of normal speech production is summarized in Appendix C. Using syllables makes it possible to control variables such as the sound structure, the phonetic characteristics of the sounds and prosodic features (e.g. equal stress on the speech segment). Also, these syllables simplified the reading task, which in turn limited pronunciation difficulties and reduced the possibility of artifact dysfluencies.

TABLE 3.2: VOICING CHARACTERISTICS OF THE PHONETIC CONTEXTS

FEATURES OF PHONETIC CONTEXTS	PHONETIC CONTEXTS		
	Group 1: *Vowel-consonant (VC) context*	**Group 2:** *Consonant (voiced)-vowel-consonant ($C_{(v)}$VC) context*	**Group 3:** *Consonant (voiceless)-vowel-consonant ($C_{(vl)}$VC) context*
Phonetic Complexity	Voicing without articulation	Voicing with articulation	Articulation without voicing
Vocal Fold Adjustment in Plosive Production*	Unidirectional glottal closure: *Voiced-voiceless*	No glottal gesture: *Voiced-voiced*	Unidirectional glottal closure: *Voiceless-voiced*
Syllable Position	Initial	Initial	Initial
Target Sound	Vowel (*i.e. /ɑ/, /ə/, /ɪ/, /ɔ/*)	Voiced consonant: *Voiced bilabial plosive /b/*	Voiceless consonant: *Voiceless bilabial plosive /p/*

*(*Rothenberg, 1968; 1983)*

The following aspects were consistent across the three phonetic context groups, to control the influence of independent variables:

- The final position of each syllable was represented by the voiceless stop consonant, /t/.
- The three phonetic context groups were represented by four syllables, respectively. These four syllables were determined by four vowels, i.e. /ɑ/, /ə/, /ɪ/ and /ɔ/. The vowels were varied, while the consonant-sound structure remained invariant. Table 3.3 contains the syllables and their respective phonetic characteristics and sound structure groups.

TABLE 3.3: CONSTRUCTION OF THE MATERIAL ACCORDING TO THE SYLLABLE STRUCTURES

SYLLABLE STRUCTURES											
GROUP 1: VC-CONTEXT				**GROUP 2: $C_{(v)}$VC-CONTEXT**				**GROUP 3: $C_{(vl)}$VC-CONTEXT**			
Voicing for Vowel initiation in VC syllables	Listed Syllables			Voicing with Articulation for $C_{(v)}$ initiation in $C_{(v)}$VC syllables	Listed Syllables			Articulation without Voicing for $C_{(vl)}$ initiation in $C_{(vl)}$VC syllables	Listed Syllables		
	Phonetic Spelling of Syllables	*Syllables in Afrikaans*	*(Translated meaning in English)*		*Phonetic Spelling of Syllables*	*Syllables in Afrikaans*	*(Translated meaning in English)*		*Phonetic Spelling of Syllables*	*Syllables in Afrikaans*	*(Translated meaning in English)*
V_1C_1	/ɑt/	At	*a first name, "Adrian"*	$C_2V_1C_1$	/bɑt/	bad	*"bath"*	$C_3V_1C_1$	/pɑt/	pad	*"road"*
V_2C_1	/ət/	it	*non-sense word*	$C_2V_2C_1$	/bət/	bed	*"bed"*	$C_3V_2C_1$	/pət/	pet	*"cap"*
V_3C_1	/ɪt/	iet	*"aught"*	$C_2V_3C_1$	/bɪt/	bied	*"to offer help"*	$C_3V_3C_1$	/pɪt/	Piet	*a first name, "Peter"*
V_4C_1	/ɔt/	ot	*"pig"*	$C_2V_4C_1$	/bɔt/	bot	*"bud"*	$C_3V_4C_1$	/pɔt/	pot	*"pot"*
Key: V_1 = /ɑ/			C_1 = /t/ V_2 = /ə/				C_2 = /b/ V_3 = /ɪ/				C_3 = /p/ V_4 = /ɔ/

3.7.1.2 Familiarity of syllable structure

Eleven of the 12 syllables were words recognized in the Afrikaans language (Odendal, Schoonees, Swanepoel, Du Toit & Booysen, 1994), with only one nonsense syllable (i.e. /ət/). By listing these syllables in a reading format, the speech material was isolated from a linguistic context. An advantage of eliciting monosyllabic responses is that PWS are seldom dysfluent* on such phonetically and linguistically simple utterances.

3.7.2 Speech sounds in syllable structures

3.7.2.1 The selection of speech sounds in the syllable structures

The consonants and vowels were selected according to certain criteria, which restricted the number of speech sounds included in the study. The criteria for the selection of consonants and vowels were as follows:

- **Consonants**

The selection of consonants was based on the voicing characteristics of the different sounds. The ability of PWS to contrast voicing during speech initiation were studied by using the voiced /b/ and voiceless /p/ plosives in the selected syllables. The selected consonants (i.e. /b/ and /p/) matched each other in all phonetic features, except in the voiced-voiceless distinction that forms the distinctive feature between /b/ and /p/. Voiceless stops in Afrikaans (such as /p/ and /t/) are produced with mild and easy muscle tension (De Villiers & Ponelis, 1987). However, in languages such as English, Sotho and Zulu voiceless stops are produced with greater muscle force and aspiration (such as /p^h/ and /t^h/) (De Villiers & Ponelis, 1987). Table 3.4 shows the classification of the consonants that were selected for this study.

TABLE 3.4: CLASSIFICATION OF THE SELECTED CONSONANTS

CLASSIFICATION OF CONSONANTS	DISTINCTIVE FEATURES OF THE SELECTED CONSONANTS	
	/p/ + vowel	/b/ + vowel
Place of Articulation	Labial	Labial
Manner of Articulation	Plosive	Plosive
Voicing feature	Voiceless	Voiced

* *Dysfluent* speech refers to a general breakdown in speech fluency, including word-finding problems. *Disfluencies* refer specifically to stuttering behaviour (Wingate, 1986).

- **Vowels**

Only short vowels were included to simplify the compilation of speech material and the analysis of the speech utterances. The four vowels, /ɑ/, /ə/, /ɪ/ and /ɔ/, differ in vertical tongue positioning as well as horizontal tongue positioning (De Villiers & Ponelis, 1987). High, middle and low vowels, as well as front, central and back vowels (De Villiers & Ponelis, 1987) formed part of the syllable structure. The classification of the vowels used in this study with regard to tongue positioning is represented in Table 3.5.

TABLE 3.5: SELECTION OF FOUR VOWELS: VERTICAL AND HORIZONTAL TONGUE POSITIONING

VERTICAL TONGUE POSITIONING	HORIZONTAL TONGUE POSITIONING		
	Front	Central	Back
High	/ɪ/	-	-
Middle	-	/ə/	/ɔ/
Low	-	/ɑ/	-

A particular vowel can be described in terms of its formants or resonance pattern. In other words, vowels are formed by a glottal source and modified by vocal tract characteristics, which include vocal fold and articulatory modifications. The fundamental frequency (f_0) of a vowel is controlled by the laryngeal mechanism (Borden & Harris, 1984). Whether a vowel is perceived as a high or low sound, depends on changes to the vocal fold length, mass and tension, as well as subsequent subglottal pressure changes (Borden & Harris, 1984). For the vowels used in this study, /ɪ/ and /ə/ have higher fundamental frequencies than /ɑ/ and /ɔ/. The latter two vowels, /ɑ/ and /ɔ/, are considered to be low frequency vowels (Borden & Harris, 1984). Kent (1993; 1998) and Maddieson (1997) indicated that in comparison with high vowels, low vowels are produced with greater loudness and longer duration. The significance of these features pertaining to vocal fold physiology in stuttering is unclear, but may be of interest later in this study and thus worth mentioning. Also, vowels with rounded (such as /ɔ/) or unrounded (such as /ɪ/) lip positions require more active articulatory coordination than the vowels requiring a neutral vocal tract (such as /ɑ/ and /ə/) (Meyer, 1993; Kent, 1998). The distinctive vocal fold adjustments and articulatory features involved in the production of the different vowels might be of importance in the interpretation of the results.

3.7.2.2 Position of speech sounds analyzed

In order to study speech initiation in PWS, the target sound in the elicited utterance was

placed in the initial position. Difficulty with the initiation of speech production is characteristic of stuttering (Lebrun & Van Borsel, 1990; Bloodstein, 1995). In general, PWS find initial consonants more difficult to produce than initial vowels (Van Riper, 1982; Bloodstein, 1995). Consonants involve a greater extent of articulation tension than vowels and are more important for speech intelligibility than vowels (Bloodstein, 1995). Certain languages such as Afrikaans and Dutch are characterized by a hard attack in the articulation of initial vowels (Uys, 1970; Vaane & Janssen, 1978 in Bloodstein, 1995). Persons who stutter might experience difficulty in production of the initial vowels of these languages, because the manner of articulations of the initial vowels seems to be similar to that of the initial consonants (Bloodstein, 1995).

3.7.3 Format of the material

Speech samples were obtained from the participants using a reading task in the format of lists of syllables. The test stimuli included twelve monosyllabic utterances. Five repetitions of the test stimuli (12 monosyllabic utterances) were recorded. However, the repetitive nature of the task allows for practice and could reduce intrasubject response variability. In order to control any such artificially induced diminution in variability in the subjects, the 12 syllables were randomly ordered into *five* different lists. Thus, the participants repeated 12 syllables in five trials, which resulted in sixty data samples per participant (subject or control). Each list ended with a syllable not included in the data-analysis. These syllables functioned as markers during the data-analysis. All of the readings were recorded in an acoustically shielded room on high-quality audiocassette recording equipment for later analysis on the electroglottograph and spectrograph. A graphic presentation of the material is provided in Appendix D.

3.8 OTHER METHODOLOGICAL CONSIDERATIONS

3.8.1 Considerations for data inclusion

Perceptual data were not considered in the present study, because the main purpose was to explore vocal fold activity in PWS during speech initiation. Speech utterances were analyzed irrespective of their perceptual status. Thus, perceptual disfluent items were included in the analysis of the data. Perceptual data were only used to establish severity of stuttering for the subjects (see 3.4.1.4). Since abnormal subacoustic stuttering behaviour may or may not co-occur with perceptually dysfluent speech (Dembowski & Watson, 1991*a*) the validity of the

study would be in question when conducting research on perceptually fluent speech production of PWS.

3.8.2 Considerations for data exclusion

The recordings of the speech samples were checked by the researcher to ascertain that valid productions of speech initiation were included. Analyzed samples had to involve clear data signals. Utterances displaying error signals or signals unacceptable for electroglottographic and spectrographic analyses were rejected from data analysis. Recordings were excluded from data analysis if they contained reading errors, or displayed distorted/unclear acoustic and/or L_x signals on the NEC Multisync monitor. Signal artifacts may be due to low frequency noise or subharmonic energy in the vocal tract (Behrman *et al.,* 1998). Speech-induced artifacts can affect waveform configuration and are generally attributed to velopharyngeal repositioning, slight velopharyngeal openings (e.g. such as in swallowing) or small movements of other neck structures relative to the electrodes (Colton & Conture, 1990). Signal artifacts may also result when the speaker has excessive muscular, glandular and fatty tissue around the larynx structure or when optimal electrode positioning was interrupted during the recording (Rothenberg & Mahshie, 1988).

From the five repetitions of the 12 syllables, only three responses were analyzed *(*see 3.6.3.). Thus, the highest possible number of utterances used in data analysis was 36 (three repeated trials of the 12 productions). If immeasurable waveforms occurred in any production of reading list 1, reading list 2 or reading list 3, it was discarded and a sample from the fourth or fifth list was included for data analysis. After the discarding of utterances with movement artifacts, the participants each had a total of 36 utterances (*3 x* 12 productions) that were considered in the data analysis.

3.9 DATA COLLECTION PROCEDURE

The portable electro-laryngograph was used simultaneously with the Kay DSP Sonagraph to collect data for the analysis of physiologic and acoustic vocal fold behaviour during speech initiation in subject and control speakers. Data of physiologic vocal fold activity was recorded by using two surface EGG electrodes, while data for the acoustic vocal fold analysis was obtained by a microphone.

3.9.1 Setting for data recording

The data recordings were administered in a single session lasting approximately 15 minutes. The recordings were conducted in an acoustically shielded room in the *Laboratory for Instrumental Communication Research* at the Department of Communication Pathology, University of Pretoria. Each participant (subject or control) was individually recorded by the researcher. In order to avoid distortion on the recordings of the electroglottograph, it was placed 1 meter from the other recording instruments (Kay Elemetrics Corporation, 1989; Colton & Conture, 1990). For the recording, the participant was seated with the EGG electrodes placed on both sides of the thyroid cartilage, approximately 1 cm from the thyroid angle (Kay Elemetrics Corporation, 1989). Adequate electrode placement was verified by observing the L_x signal during phonation of /ɑ:/ on the energy wave display of the Sonagraph. The L_x signal was constantly monitored with the EGG instrument. Optimal or close-to-optimal electrode placement was established, when the L_x signal showed minimal low frequency noise (Behrman *et al.,* 1998). The acoustic signal (via the microphone) and the L_x signal (via the electrodes) were stereo recorded simultaneously. The specified material was read into a microphone. The microphone was placed approximately 30 cm from the participant during recordings. The participant's distance from the microphone as well as the input attenuation were kept constant throughout all the recordings. The VU-meter was monitored to adjust the input level when necessary. Hereby optimal quality of the signals was possible and distortion of the signals was prevented (Kay Elemetrics Corporation, 1989).

3.9.2 Instructions for data recording

Each participant was instructed to hold his head in a stable position to minimize movement artifacts during the EGG recording. The researcher first demonstrated the task selected to collect data of vocal fold activity. In the recording task, each participant was required to read the five lists of 13* syllables in a typical speaking manner with an approximate pause of 1 *sec* between each utterance. No opportunity was provided to practice the test stimuli. The researcher did indicate to the participants that one nonsense syllable was listed and its pronunciation was reviewed.

* At the end of each list of 12 words, an additional word followed to mark the end of each list (Also see *3.7.3. Format of material*).

3.10 DATA ANALYSIS PROCEDURE

Vocal fold activity during speech initiation was examined and measurements of physiological and acoustic activity were performed. Representative portions of the electroglottographic (L_x) and the acoustic (or speech) signals were analyzed simultaneously. The two signals acquired were captured onto the digital memory of the Sonagraph. The data of both these signals would provide more information on the vocal fold characteristics during speech initiation associated with stuttering. Figure 3.3 is an example of the two signals, the L_x signal and the acoustic signal, that were studied in PWS and normal speakers.

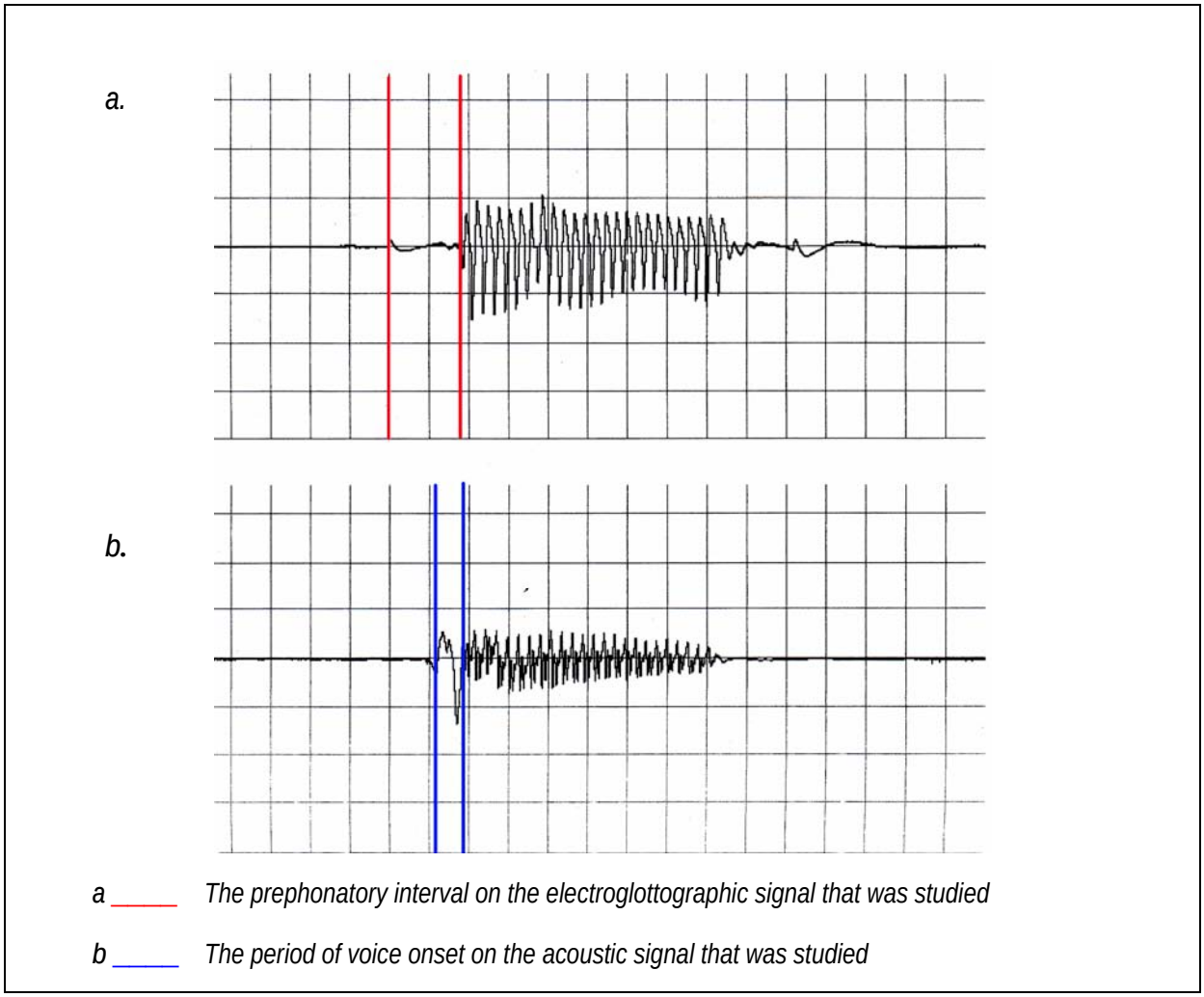


FIGURE 3.3: AN EXAMPLE OF THE DUAL WAVEFORM DISPLAY OF THE ELECTROGLOTTOGRAPHIC SIGNAL *(a)* AND ACOUSTIC SIGNAL *(b)*

The L_x signal was displayed as an energy wave over time on channel 2 and the acoustic signal was spectrographically displayed and analyzed on channel 1 of the Sonagraph. The setting used for analyzing the L_x signal (setting #08, according to the Kay DSP Sonagraph Model

5500 manual) was adapted for appropriate memory span. Measurements were made with the use of time cursors. Adjustments to the time axis were made when a more detailed examination of the L_x or the acoustic signal was needed. The combination display settings for signal analysis are provided in Appendix E. The relevant numerical and descriptive data for each participant were recorded on specific recording forms as presented in Appendix F(*i-iii*). The two displays were synchronised by matching the point of onset of periodic energy as it appears on the two displays.

3.10.1 *Data analysis for sub-aim 1*: Analysis of physiological vocal fold activity during speech initiation

The qualitative and quantitative properties of prephonatory vocal fold behaviour were analyzed by means of the L_x signal. The L_x signal reflects the time variations of the area of contact between the vocal folds (Childers & Krishnamurthy, 1985) and was used for analysing vocal fold activity. The L_x signal gives an indication of vocal fold approximation during speech initiation. Glottal closure *or* vocal fold approximation in preparation for phonation usually results in an isolated pulse in the L_x signal preceding the onset of quasi-periodic oscillation (Peters & Boves, 1988). The L_x signal provides information regarding the nature of vocal fold approximation and movement.

Analysis of the L_x signal started after a naught line of 0.5 *sec* was displayed following the preceding utterance. The first rise in amplitude of the L_x signal is an indication of the onset of the glottal pulse (thus, the onset of vocal fold approximation/adduction). For studying the prephonatory vocal fold activity, the L_x signal interval between the first indication of vocal fold approximation and the onset of vocal fold vibration for voice initiation was analyzed. The onset of phonation was identified by the appearance of regular oscillations in accordance with the onset of acoustic activity on the spectrogram. The following aspects of the prephonatory interval were studied as displayed on the L_x signal: (a) the nature of physiological vocal fold activity as a variable function of time, and (b) the duration of the period from vocal fold approximation to vocal fold vibration for speech initiation.

3.10.1.1 *Data analysis for sub-aim 1.1:* Analysis of the nature of prephonatory vocal fold activity during speech initiation

Interpretational analysis was used to identify and describe the characteristics of prephonatory

vocal fold activity in the individual controls and subjects. Vocal fold activity during the prephonatory period as displayed by the L_x signal, was visually examined. The descriptions of electroglottographic activity obtained from the L_x signals were compiled into a list.

3.10.1.1.1 Data analysis for the identification of prephonatory vocal fold activity characteristics during speech initiation

The identification of prephonatory vocal fold (activity) characteristics during speech initiation for the controls and the subjects was based on the following factors:

- All prephonatory vocal fold characteristics were considered to be dimensional and therefore, *occurring in a greater or lesser magnitude*. The occurrence of a specific vocal fold characteristic was noted irrespective of its magnitude.
- The different prephonatory vocal fold characteristics were clearly defined with consideration to the phonetic context in which it occurred (Tables 4.1.1 and 4.1.2). These descriptions formed the basis for classifying types of prephonatory vocal fold characteristics.
- A categorization process was used to classify prephonatory vocal fold (activity) characteristics into different types (Figure 4.1). For example, the L_x signal data was evaluated and matched with a description of a given type of prephonatory vocal fold activity. If no match could be found between the prephonatory vocal fold characteristics for a specific utterance (displayed by the L_x signal) and the indicated types of prephonatory vocal fold activity, it was added and classified as a specific type of prephonatory vocal fold activity.

With regard to the categorization process, specific parameters had to be determined to describe different prephonatory vocal fold activity characteristics observed in each participant. Certain parameters were thus selected to facilitate unambiguous judgments. The L_x recordings of the prephonatory period were evaluated in terms of visually detectable variance in the time and/or amplitude of the L_x waveform. The following judgment parameters were used:

- *Variability in amplitude of the L_x waveform:* The amplitude is representative of variance in approximation between the vocal folds. Studying the amplitude of the L_x signal during the prephonatory period focused on describing the waveform shape.

The waveform shape was described in terms of the presence of any variance in amplitude (indicating vocal fold approximation) and periodicity/regularity of amplitude variance during the prephonatory period. Variance in the amplitude was determined from the first amplitude envelope (or onset of a glottal pulse) following a zero-level of the waveform in which no L_x activity (thus, no change in amplitude) was observed, until the onset of regular vertical striations indicating the onset of voicing. L_x signal irregularities would be regarded as visually detectable variations in the shape of the L_x waveform, in other words, energy deviations from the naught line on the display.

- *Variability in time of the L_x waveform:* Comments were made on the continuity and periodicity of electroglottographic activity during the prephonatory period for normally fluent speakers and PWS. Timely repetitions of vocal fold activity across the different utterances suggested regular and periodic L_x activity. Visually detectable variations in the continuity of the prephonatory vocal fold activity or in the periodicity of the amplitude variations (viewed as distorted spacing between the amplitudes) indicated irregular L_x activity.

A recording sheet was prepared from the listed prephonatory vocal fold characteristics (Appendix F(*i-iii*)).

3.10.1.1.2 Data analysis for the classification of prephonatory vocal fold activity characteristics during speech initiation

Following the identification of the L_x signal properties for the initiation of VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances during prephonatory period, the different vocal fold activity characteristics across the controls and subjects were classified. Prephonatory vocal fold activity characteristics were categorized into specific types based on similarities in the amplitude and time of L_x waveforms (obtained from the EGG recording). These groups or types of prephonatory vocal fold activity were then labelled and clearly defined (see 4.2.1.1). The categorization or subtyping of prephonatory vocal fold activity was guided by prior knowledge of vocal fold physiology in speech production (Borden & Harris, 1984; Titze, 1990) and previous research on signal typing of PWS's vocal fold behaviour during speech initiation (Peters & Boves, 1988).

3.10.1.1.3 Data analysis for determining the frequency of occurrence of different types of prephonatory vocal fold activity

Frequency analysis of prephonatory vocal fold characteristics was carried out for both the control group (N=6) and the subject group (N=6). The frequency analysis was performed according to the classification of the prephonatory vocal fold activity characteristics as indicated in the current study (3.10.1.1.2). A recording sheet was used on which the presence or absence of the prephonatory vocal fold characteristics was rated as 1 or 0, respectively (Appendix F(*i-iii*)). The occurrence of different types of prephonatory vocal fold activity were rated on separate forms for the twelve VC, $C_{(v)}VC$ and $C_{(vl)}VC$ utterances for each participant.

- **L_x analysis to determine the frequency of occurrence of different types of prephonatory vocal fold activity for the control group**

In normal speech initiation, specific vocal fold behaviour or adjustments are implicated for vowel onset, voiced consonant onset and voiceless consonant onset of the different utterances. The voicing characteristic of the initial sound for the different utterances seem to influence the L_x signal and is therefore important for describing and interpreting L_x signal types. To establish which type(s) of prephonatory vocal fold activity showed the highest frequency of occurrence in the control group, the results for VC, $C_{(v)}VC$ and $C_{(vl)}VC$ utterances were analyzed separately. A table was compiled, comparing the prephonatory vocal fold characteristics of the control group for the three phonetic contexts with *normal conditions of speech initiation in the presence of different voicing requirements* and *previous findings on prephonatory vocal fold activity in normally fluent speakers* (Appendix C).

- **L_x analysis to determine the frequency of occurrence of different types of prephonatory vocal fold activity for the subjects**

To establish which types of prephonatory vocal fold activity were characteristic to the subjects' utterances during speech initiation, the results for VC, $C_{(v)}VC$ and $C_{(vl)}VC$ utterances were analyzed separately for each subject. For each subject, the 12 utterances of the three phonetic contexts were categorized according to definitions of the types of prephonatory vocal fold activity, as established in the initial part of the data analysis (3.10.1.1.1 and 3.10.1.1.2).

3.10.1.1.4 Data analysis for determining the effect of phonetic context on prephonatory vocal fold activity

To determine the effect of the initial sound on prephonatory vocal fold characteristics, L_x signals for the prephonatory period were analyzed separately for the VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances for the normal speakers and stuttering subjects. Thus, the instances in which a specific type of vocal fold behaviour occurred were recorded for each phonetic context on separate recording sheets for later processing (Appendix F(*i-iii*)) .

3.10.1.2 *Data analysis for sub-aim 1.2:* Analysis of Laryngeal Onset Time (L_xOT)

Quantitative analysis procedures were used to examine the period between vocal fold approximation and the onset of vocal fold vibration during the initiation of a vowel (in a VC context), a voiced plosive (in a CVC context) and a voiceless plosive (in a CVC context) for the normal speakers and the PWS.

3.10.1.2.1 Measurement of laryngeal onset time (L_xOT)

Laryngeal onset time (L_xOT) interval was measured from the first visible glottal activity to the onset of vocal fold vibration for speech initiation as displayed on the L_x signal. This interval was measured in seconds and subsequently converted to milliseconds (*ms*). The L_xOT value was recorded as 0 ms if no energy change in the L_x signal occurred, thus no vocal fold approximation was observed prior to voice initiation. L_xOT measurement of 0 ms indicates that the moment of vocal fold approximation coincided with the start of vocal fold vibration for the onset of voicing. L_xOT was obtained from the L_x signal for the initial sound productions of the twelve VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances. Figure 3.4 is a visual presentation of *laryngeal onset time*.

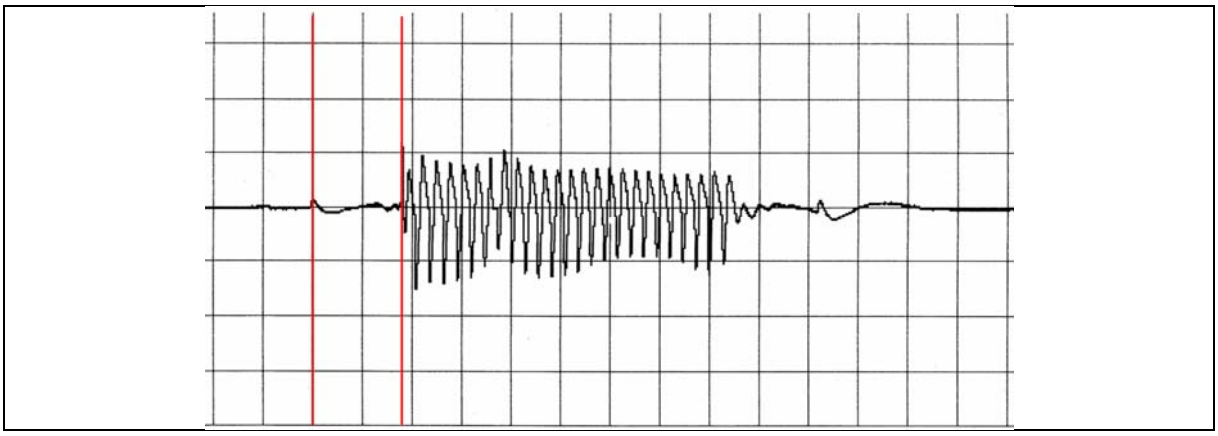


FIGURE 3.4: AN EXAMPLE OF LARYNGEAL ONSET TIME DISPLAYED ON THE ELECTROGLOTTOGRAPH

3.10.1.2.2 Data analysis for determining the effect of phonetic context on L_xOT

The effects of voicing demands on L_xOT for the control group and subjects were investigated by recording the L_xOT values for the VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances on separate recording sheets for later processing (Appendix F(*i-iii*)).

3.10.2 *Data analysis for sub-aim 2:* Analysis of acoustically detected vocal fold activity during speech initiation

The acoustic result of vocal fold movements during speech initiation was displayed on the acoustic signal. The acoustic signal provides information on the collaborative effect of coordination between vocal fold activity and articulatory movements. The acoustic signal in conjunction with data of the L_x signal allow for inferences about respiratory, phonatory and articulatory functions of speech production (Dembowski & Watson, 1991*a*).

Analysis of the period of voice onset was based on the acoustic signal and consisted of a qualitative analysis of the nature of voice onset and a quantitative analysis, i.e. voice onset time (VOT).

3.10.2.1 *Data analysis for sub-aim 2.1:* Analysis of the nature of voice onset energy

3.10.2.1.1 Data analysis for the identification and classification of voice onset energy

As opposed to prephonatory vocal fold activity, previous research has been done on the nature of voice onset whereby classifications of voice onset energy were established, namely gentle

(gradual) or abrupt voice onsets (Borden *et al*., 1985; Dembowski & Watson, 1991*a*). Voice onset energy of each VC, $C_{(v)}$VC and $C_{(vl)}$VC production was qualitatively analyzed as either, gentle or abrupt. The classification of these utterances was based on the appearance of the acoustic waveform envelope during the first 10 glottal pulses as suggested by Dembowski and Watson (1991*a*). Adjustments to the memory settings of the sonagraph were made to view the first 10 pulses in relation to the total wave pattern and the amplitude envelopes were classified as reflecting either gradual (easy) or abrupt (hard) voice onset.

- **Analysis of gradual voice onset**

An amplitude envelope was categorized as easy if it showed a gradual rise across 10 consecutive glottal pulses. These amplitudes are thus higher than the preceding pulses. A gradual voice onset is often accompanied by waveform aperiodicity from aspiration noise (Dembowski & Watson, 1991*a*). Figure 3.5 illustrates the appearance of gradual voice onset in the acoustic waveform.

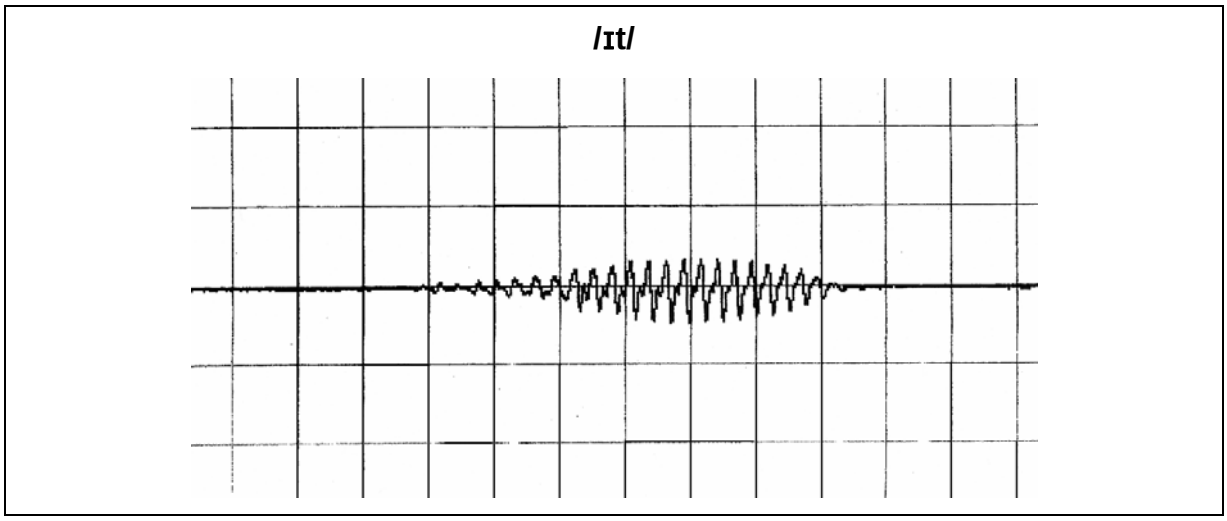


FIGURE 3.5: AN ILLUSTRATION OF A GRADUAL VOICE ONSET

- **Analysis of abrupt voice onset**

A hard voice onset showed an abrupt or prominent rise in the amplitude envelope. Abrupt voice onset can be described as the time it takes for the amplitude of the acoustic signal to increase from approximately 10% to 90% of its maximum value (Borden *et al*., 1985). An example of abrupt voice onset is illustrated by Figure 3.6.

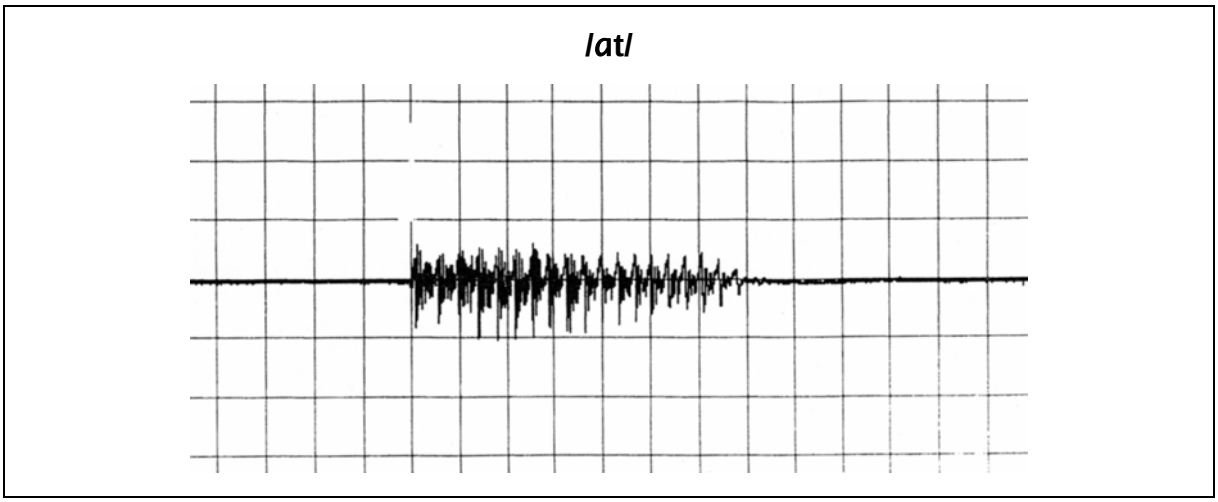


FIGURE 3.6: AN ILLUSTRATION OF AN ABRUPT VOICE ONSET

3.10.2.1.2 Data analysis to determine the frequency of occurrence of types of voice onset energy

Frequency analysis of the types of voice onset energy was done for all the participants. The frequency analysis was performed according to the classification of voice onset energy by Dembowski and Watson (1991*a*). A recording sheet was used on which the presence or absence of the types of voice onset energy (*gradual voice onset* and *abrupt voice onset*) was rated as 1 or 0, alternatively, for normally fluent speakers and stuttering subjects (Appendix F(*i-iii*)). The types of voice onset energy were marked on separate forms for the twelve VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances for each participant and were used for data processing.

3.10.2.1.3 Data analysis to determine the effect of phonetic context on voice onset energy

To determine the effect of the initial sound of VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances on voice onset energy, the instances in which a specific type of voice onset energy (gradual or abrupt) occurred were recorded on separate recording sheets for each phonetic context for later processing. These recording sheets are included in Appendix F(*i-iii*).

3.10.2.2 *Data analysis of sub-aim 2.2:* Analysis of Voice Onset Time (VOT)

3.10.2.2.1 Measurement of VOT for initial voiced and voiceless plosives

For measuring voice onset time (VOT), a combination display of the acoustic signal (sound

pressure wave), as well as a spectrogram were used. During the measurement of VOT, the two graphic displays were compared to increase the reliability of this analysis procedure. Participants' VOT intervals on wide-band spectrograms were isolated by pencilling a thin vertical line to mark the onset of the consonant bursts (acoustic energy resulting from the release of the closure in the vocal tract) on the spectrogram and another line to mark the initiation of regularly appearing vertical striations for the first glottal vibrations (Borden & Harris, 1984). The interval between the two lines, representing VOT in seconds, was converted to milliseconds (*ms*). This VOT measurement guideline was followed for both the initial voiceless /p/ and voiced /b/ stop consonants. Instances of prevoicing, which are associated with voiced stop consonants (Borden & Harris, 1984), were included in the VOT measurement as negative values as demonstrated in Figure 3.7. The speech signal was used because of the difficulty of establishing prevoicing from a spectrogram. Thus, VOT was given a positive value if the articulatory release of the stop-plosive preceded voice onset and a negative value if the release followed voice onset. Figure 3.8 illustrates a waveform with a positive VOT. If no visually observable burst was evident, the VOT value was taken as 0 *ms*.

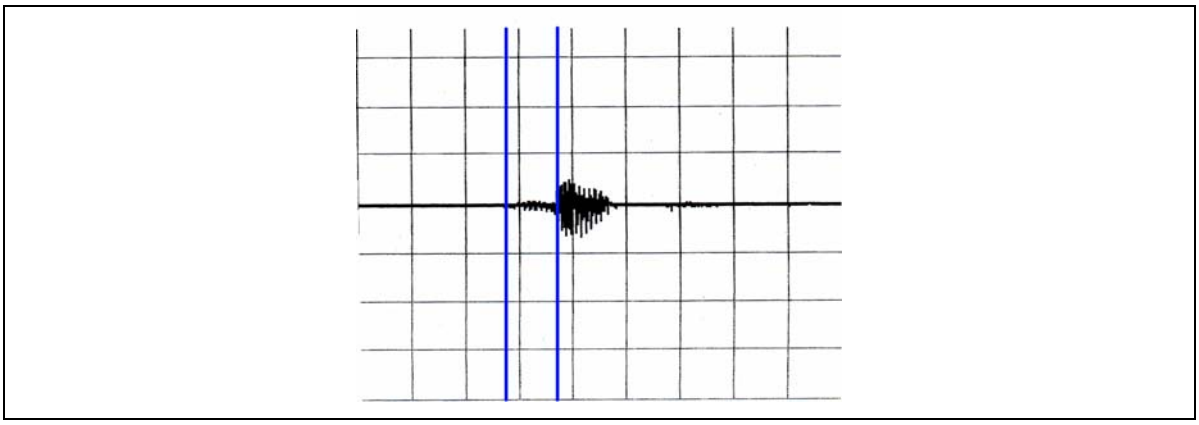


FIGURE 3.7: AN EXAMPLE OF NEGATIVE VOICE ONSET

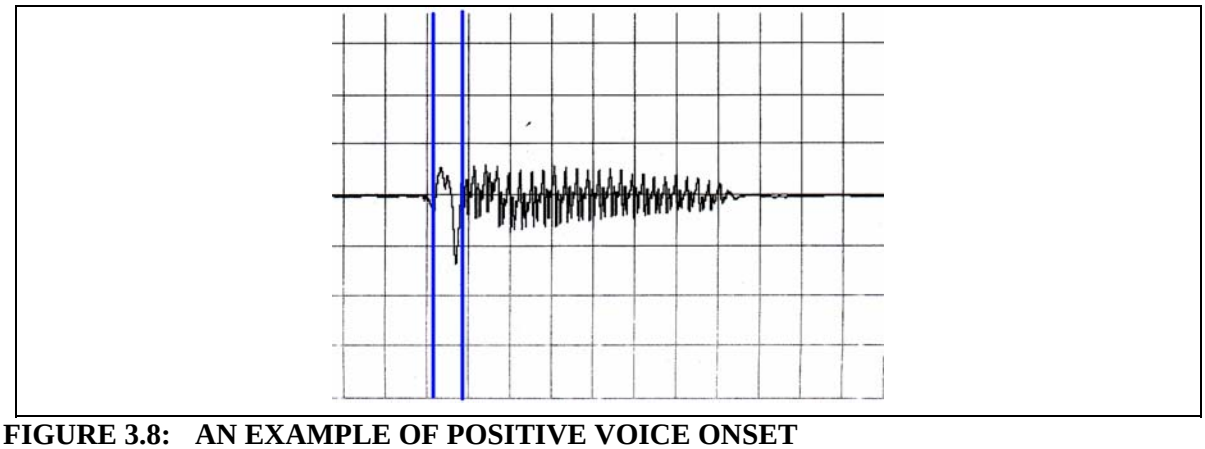


FIGURE 3.8: AN EXAMPLE OF POSITIVE VOICE ONSET

3.10.2.2.2 Data analysis to determine the effect of phonetic context ($C_{(v)}VC$ and $C_{(vl)}VC$) on VOT

The effect of voicing demands for initial /b/ and /p/ utterances on VOT for the controls and subjects were investigated by recording the VOT values for the $C_{(v)}VC$ and $C_{(vl)}VC$ utterances on separate recording sheets for later processing (Appendix F(*i-iii*)).

3.11 RELIABILITY

A ten percent sample of the data was reanalyzed by the researcher to determine intraobserver reliability. The first ten percent of recorded utterances for each signal classification (L_x signal and acoustic signal) and temporal parameter of each participant was used for reanalysis.

Two factors which could have influenced the validity of the intraobserver judgements of the nature of the prephonatory vocal fold activity, are: (a) the classification of the L_x waveform is a pilot study, and (b) the absence of a standard rating index and normative data to evaluate the prephonatory vocal fold behaviour (Titze, 1990). In an attempt to address these concerns, each of the different characteristics was clearly defined by means of a description and an illustration (Tables 4.1.1 and 4.1.2).

For the temporal parameters (L_xOT and VOT), if the difference between the original measure and the reliability measure was not more than 3 ms (Seddoh, Robin, Sim, Hageman, Moon & Folkins, 1996), or if these two measures did not differ by more than two increments (as determined by moving the cursor either to the left or the right of the original point of measurement) the two measurements were accepted as being in agreement. An experienced researcher with expertise in the field of instrumental analysis of speech samples was consulted for a second opinion when the present researcher was uncertain about a specific measurement point. In instances where the second researcher was consulted, the measurement was recorded only after consensus had been reached.

3.12 DATA PROCESSING PROCEDURES

The data processing involved a descriptive approach using measures of central tendency – the mean or average scores (Guy, Edgley, Arafat & Allen, 1987). Measures of variability – ranges and error values, were also used. The results were presented by descriptive tables and

figures of absolute values, means, ranges, and error values. Because the group size (controls and subjects) and sample size were small, statistical analysis could not be applied to the data. Caution should be taken not to interpreted significant differences between the subject group (individuals who stutter) and control group (normally fluent speakers) and different phonetic contexts (i.e. vowel onset *versus* voiced consonant onset *versus* voiceless consonant onset) as demonstrating a causal relation between groups/contexts and measurement results.

The data of the individual subjects are reflected in tables and figures, which were used to summarize the results in Chapter 4. The results for the subject group were calculated from the data of the six subjects. For the purpose of this study, the data from each member in the control group were used to calculate the mean scores for the control group.

3.12.1 *Data processing for sub-aim 1:* Processing of the physiological vocal fold activity during speech initiation

Data-processing for sub-aim one focussed on: (a) examining and comparing differences in the nature of physiological vocal fold activity during speech initiation of voiced and voiceless speech sounds in a group of normally fluent speakers and persons who stutter (3.12.1.1); (b) determining differences in onset time of vocal fold vibration for speech initiation in PWS and normal speakers, and investigating the effect of phonetic contexts on the timing of phonatory onset in the two groups (3.12.1.2).

3.12.1.1 *Data processing for sub-aim 1.1:* Processing of the nature of prephonatory vocal fold activity during speech initiation

3.12.1.1.1 Data processing for the identification and classification of prephonatory vocal fold activity

The categorization process for the identification and classification of prephonatory vocal fold activity characteristics (as displayed by the L_x signal) during speech initiation was based on the evaluation of the L_x signals of the 12 utterances for the controls. This method of categorizing different prephonatory vocal fold activity characteristics into subtypes consisted of three phases which included: (a) the identification of the presence or absence of prephonatory vocal fold approximation; (b) the identification of additional vocal fold activity in the presence of prephonatory vocal fold approximation; and (c) a description of the energy

amplitude and time to identify different characteristics of the additional prephonatory vocal fold activity following the prephonatory vocal fold approximation.

From the data of the control group, types of prephonatory vocal fold activity for the VC, $C_{(v)}VC$ and $C_{(vl)}VC$ contexts were identified. Prephonatory vocal fold activity characteristics of the different utterances showed certain similarities which were labelled as a specific type of prephonatory vocal fold activity. L_x waveform data of the subjects that failed to match any of the types of prephonatory vocal fold activity indicated for the controls, were added as a different type of prephonatory vocal fold activity. Based on the prephonatory vocal fold characteristics as indicated by the L_x waveforms of the controls and subjects, a diagram for the categorization of L_x waveforms of prephonatory vocal fold activity in normal speakers and stuttering speakers was compiled. This diagram is presented in Figure 4.1. L_x signals for VC, $C_{(v)}VC$ and $C_{(vl)}VC$ utterances in the controls and subjects were subtyped, labelled and defined based on the criteria outlined in the diagram. The different types of prephonatory vocal fold activity are listed and defined in Table 4.1.

3.12.1.1.2 Data processing for the determination of frequency of occurrence of prephonatory vocal fold activity

- **Determination of frequency of occurrence of prephonatory vocal fold behaviour for the control group**

Baken (1996) emphasized that voice signal analysis should relate back to the vocal fold physiology in some meaningful way. When processing the results of prephonatory vocal fold activity consideration must therefore be given to the *type of behaviour*, the *phonetic context* in which it occurred and the *physiologic requirements* for the onset of the initial vowel, voiced consonant or voiceless consonant utterance.

To establish the type of prephonatory vocal fold activity had the highest incidence for the VC, $C_{(v)}VC$ and $C_{(vl)}VC$ utterances in the group of normally fluent speakers, the total frequency of occurrence of each type was determined separately for the three contexts. Tables 4.2.1 to 4.2.3 depict the frequency of occurrence of the different *types of prephonatory vocal fold activity* for the six control speakers, which contributed to the total frequency of occurrence of each *type of prephonatory vocal fold activity* for the control group. The maximum number of utterances for each *type of prephonatory vocal fold activity* per phonetic context for the control group was 72 (12 utterances *x* 6 control speakers) and the minimum number, 0.

The average frequency of occurrence per *type of prephonatory vocal fold activity* was determined by dividing the total number of occurrences of a specific prephonatory vocal fold activity by the number of control speakers (N = 6). Figure 4.2 illustrates the percentage of average frequency of occurrence for the types of prephonatory vocal fold activity in the three phonetic contexts, i.e. VC, $C_{(v)}$VC and $C_{(vl)}$VC.

- **Comparison of the frequency of occurrence of prephonatory vocal fold activity between the control group and subject group**

From the data, the percentage of occurrence of each type of prephonatory vocal fold activity was determined for the three phonetic contexts in the control group and subject group (Figure 4.3), as well as the individual subjects (Figures 4.4.1 - 4.4.3). Table 4.3 presents a summary of the data of prephonatory vocal fold activity for the normal speakers and PWS. For the individual subjects, the average frequency of occurrence was calculated by totalling the number of occurrences of a particular type of prephonatory vocal fold activity divided by the number of utterances separately for each phonetic context (N = 12).

3.12.1.1.3 Data processing for the determination of the effect of phonetic context on prephonatory vocal fold activity

Furthermore, the types of prephonatory vocal fold activity were arranged in descending order for each context from highest incidence to lowest incidence. The percentages of these mean scores were also calculated by dividing the values with 12 (maximum value), multiplied by 100. This arrangement is summarized in Table 4.4.

3.12.1.2 *Data processing for sub-aim 1.2:* Processing of laryngeal onset time (L_xOT)

3.12.1.2.1 Data processing for the comparison of L_xOT between the control group and the subject group

Analyses of the absolute L_xOT values of the 12 VC, $C_{(v)}$VC and $C_{(vl)}$VC productions were made for the controls and subjects. The absolute L_xOT values were used to calculate the *mean L_xOT* and *the range of L_xOT.* Table 4.5, Table 4.6 and Table 4.7 contain the results of L_xOT values for the different phonetic contexts. (Appendix G(*i-iii*) presents the L_xOT results of the 12 VC, $C_{(v)}$VC and $C_{(vl)}$VC productions for the individual normal speakers).

The following calculations were done:

- The *mean L_xOT* was separately calculated for the three phonetic contexts in each participant. The sum of the absolute L_xOT values of the 12 productions of each context was calculated and divided by the number of productions (N = 12) per context. The result was noted as *mean L_xOT.*
- For the calculation of the *range of the L_xOT* for the three contexts in each participant, the minimum and maximum L_xOT values from the 12 utterances were used. The difference between these values can be regarded as the *range of L_xOT* and was determined for VC, $C_{(v)}$VC and $C_{(vl)}$VC productions.

3.12.1.2.2 Data processing for the determination of the effect of phonetic context on L_xOT

A table was compiled that indicates the difference in the *mean* and *range of L_xOT* between the individuals who stutter and control group for the three phonetic contexts, namely utterances beginning with a vowel, a voiced plosive, and a voiceless plosive. The data have been summarized in Table 4.8. The L_xOT values for the control group were taken as the norm in the absence of a standard L_xOT value. For each participant, the mean and range of L_xOT values for each context were deducted from the values of the control group. A positive value indicated that the duration of the subject was longer/larger in range than that of the control group, whereas a negative value indicated that the duration of the subject was shorter/smaller in range than that of the control group. As illustrated by Figure 4.5, bar graphs were constructed of the differences in mean L_xOT and range of L_xOT for the VC, $C_{(v)}$VC and $C_{(vl)}$VC contexts between the control group and each subject.

3.12.2 *Data processing for sub-aim 2:* Processing of the acoustically detected vocal fold activity during speech initiation

Data-processing for sub-aim two focused on: (a) examining and comparing differences in the nature of voice onset energy during speech initiation of voiced and voiceless speech sounds in a group of normally fluent speakers and persons who stutter (PWS) (3.12.2.1); (b) determining differences in voice onset time (VOT) of initial voiced and voiceless plosives during speech initiation in the group of normal speakers and PWS, and investigating the effect of phonetic contexts on VOT in the two groups (3.12.2.2).

3.12.2.1 *Data processing for sub-aim 2.1:* Processing of the nature of voice onset energy during speech initiation

3.12.2.1.1 Data processing for the identification and classification of the types of voice onset energy during speech initiation

According to the definitions by Dembowski and Watson (1991*a*) for gradual and abrupt voice onset energy, the voice onset energy characteristics in the acoustic signals of controls and subjects were identified and then classified. Acoustics signals for VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances in the controls and subjects were distinguished as either gradual or abrupt as indicated in Table 4.10.

3.12.2.1.2 Data processing for the determination of the frequency of occurrence of types of voice onset energy

To establish the nature of voice onset for the different phonetic contexts, the frequency of occurrence for abrupt and gradual voice onsets were determined, respectively. The total utterances from the 12 productions that showed the specific activity (i.e. sudden onset or gradual onset) were calculated for the three phonetic context groups in each participant. Percentages of abrupt and gradual voice onsets for each context were also calculated from these totals. Table 4.10 presents a summary of the nature of voice onset for the three different contexts for the control group and the subjects (N = 6). The difference in nature of voice onset during voiced and voiceless speech initiation between the subject group and control group was illustrated by comparing the results for the two groups as seen in Figures 4.6.1 and 4.6.2. This comparison enabled a prediction on normal voice onset energy for VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances, as represented by the control group.

3.12.2.1.3 Data processing for the determination of the effect of sound initiation in different phonetic contexts on the nature of voice onset energy

To determine the effect of sound initiation in different phonetic contexts on the nature of voice onset energy of each subject compared with the control group, the frequency of occurrence of the types of voice onset for each of the 12 utterances in the three phonetic contexts were ranked from highest to lowest score for the subjects and the control group. The type of voice onset energy to be considered as normal, was determined by the group of

normally fluent speakers. The ranking of the phonetic contexts according to the scores obtained for that specific type of voice onset energy was also determined by the control group's results. If, for instance, Subject 1 has a score of *four* for the VC context, *seven* for the $C_{(v)}$VC context and *two* for the $C_{(vl)}$VC context and these results were obtained for the type of voice onset in which the control group showed the highest scores, then the $C_{(v)}$VC context will be ranked as the highest, followed by the VC context and then the $C_{(vl)}$VC context. Depending on the frequency of occurrence for the control group, the $C_{(vl)}$VC context will be considered to deviate the most from the result of the control group. These results are illustrated by Figures 4.7.1 and 4.7.2.

3.12.2.2 *Data processing of sub-aim 2.2:* Processing of voice onset time (VOT)

3.12.2.2.1 Comparison of VOT for initial voiced and voiceless plosives between the control group and subjects

To establish whether voice onset time differs between the subjects and the control group, the average VOT value and VOT error value of $C_{(v)}$VC and $C_{(vl)}$VC productions were calculated for all participants.

- The *absolute VOT value* of initial voiced and voiceless plosives in 12 CVC utterances, respectively, were recorded for each subject and control. The absolute VOT values for the subjects are summarized in Table 4.11 and Table 4.12. (VOT data of the initial voiced and voiceless plosives for individuals in the control group are available in Appendix H(*i-ii*)). The *average VOT* was separately calculated for the voiced and voiceless CVC contexts in each participant. The sum of the absolute VOT values of the 12 productions of each context was calculated and divided by the number of productions (N = 12) per context. The result was noted as the *average VOT* for $C_{(v)}$VC or $C_{(vl)}$VC productions for a participant. The *average VOT* values of the six participants of the subject group and control group were used to determine the *average VOT* for the specific group**.**

- To estimate the influence of the variation of voicing with articulation, *the average VOT error value* was determined for each subject. The error value can be described as the variation between the normal limits of VOT and the measured VOT value. The normal limits for a voiced, bilabial plosive range between *-180 ms to +15 ms*

(Zlatin, 1974). The average VOT error value for the twelve $C_{(vl)}VC$ utterances were determined by calculating the values in *ms* above 150 *ms* and below 25 *ms*, divided by 12. The same calculation was done for $C_{(v)}VC$ utterances, where the sum of milliseconds above +15 or below -180 were divided by 12. For determining the VOT error value for an *aspirated* voiceless bilabial plosive, VOT values between *+25 ms* (Lisker & Abramson, 1964 in Borden & Harris, 1984) and *+150 ms* are usually taken as the normal limits (Blumstein, Cooper, Goodglass, Statlender & Gottlieb, 1980) for first language (L1) speakers of English. For Germanic languages such as Dutch, the lower VOT for an *unaspirated* voiceless bilabial plosive can be accepted as *+10 ms* (Lisker & Abramson, 1964 in Cho & Ladefoged, 1999). Afrikaans shows similarities with Dutch with regards to speech sound production (Wissing & Roux, 1995). Due to a lack of available normative data for VOT in Afrikaans, the values indicated for voiceless /p/ production in Dutch were used. For the current study the VOT limits for /p/ productions ranged between *+10 ms* and *+150 ms*. An average VOT error value for the $C_{(v)}VC$ and $C_{(vl)}VC$ contexts was determined separately, for all the subjects from the VOT error values of the 12 utterances in each context group. These results are indicated in Tables 4.11 and 4.12.

3.12.2.2.2 Data processing for the determination of the effect of phonetic context ($C_{(v)}VC$ and $C_{(vl)}VC$) on VOT

The minimum and maximum VOT values for the voiced plosive and voiceless plosive CVC contexts were used.

- Two bar diagrams were created in order to illustrate the magnitude of difference between the minimum and maximum VOT (in ms) for /b/ and /p/ productions for each subject. The minimum and maximum VOT range values for /b/ and /p/ productions of the control group were used as the norm. Figures 4.8.1 and 4.8.2 show if the subjects functioned within the VOT range of the control group for /b/ and /p/ productions, respectively. The magnitude of the VOT range for voiced/voiceless productions indicates the repeatability of VOT for the productions for the specific subject. Thus, if the VOT values for the 12 productions are comparable, the difference between minimum and maximum VOT values (VOT range) for that particular phonetic context (i.e. C(v)VC or C(vl)VC) will be small. Greater VOT range values may reflect variability in VOT for utterances.

- Table 4.13 consists of a summary of the minimum and maximum VOT values, the VOT range, the error value for VOT range for the subjects and the mean VOT for prevocalic /b/ and /p/ productions for the control group and the six individual subjects. The error value for VOT range (in ms) for the subjects was determined by the difference between the VOT range for the particular subject and the control group. Values greater (>x) than the VOT range for the control group were regarded as deviant. If a subject showed a range less than the VOT range for the control group for initial /b/ or /p/ productions, the error value for VOT range for that subject was indicated as 0 ms. The difference was shown on the table as <x.

- Figures 4.8.1 and 4.8.2, together with the VOT results in Table 4.13, will show if the subjects had smaller minimum VOT values, greater maximum VOT values or a larger VOT range than the control group. Smaller minimum VOT values will suggest a shorter time lag in VOT, while greater maximum VOT values will point to slower or delayed VOT when compared to the control group. A large VOT range suggests variability or less consistency in the timing of voice onset.

3.13 SUMMARY OF CHAPTER 3

As introduction to this chapter, the theoretical motivation underlying this research was presented briefly. The research method of this study was then discussed against this background and entailed a description and discussion of the sub-aims, research design, subject selection criteria and procedures, instrumentation and speech material used. Finally, the data collection procedure, data analysis and processing procedures for each sub-aim were discussed in detail.

CHAPTER 4: RESULTS

4.1 INTRODUCTION

In this chapter, the results of the study will be presented. The results are organized according to the sub-aims of this study. The influence of the initiation of voiced and voiceless utterances on specific parameters of vocal fold behaviour in the speech of individuals who stutter will be described.

4.2 *RESULTS FOR SUB-AIM 1:* PHYSIOLOGICAL VOCAL FOLD ACTIVITY DURING SPEECH INITIATION

The results of physiological vocal fold activity during speech initiation included data on the nature of prephonatory vocal fold activity (4.2.1) and laryngeal onset time (4.2.2).

4.2.1 *Results for sub-aim 1.1:* The nature of prephonatory vocal fold activity during speech initiation

The results presented in this section refers to the identification and classification of prephonatory vocal fold activity (4.2.1.1); the nature of prephonatory vocal fold activity in normal speakers (4.2.1.2); frequency of occurrence of different types of prephonatory vocal fold activity (4.2.1.3); effect of speech sound initiation in different phonetic contexts on prephonatory vocal fold activity (4.2.1.4).

4.2.1.1 Identification and classification of prephonatory vocal fold activity during speech initiation

In order to classify prephonatory vocal fold activity characteristics during speech initiation in a group of normal speakers and a group of stuttering speakers, different types of prephonatory vocal fold activity were identified. The information regarding the process used to categorize the prephonatory vocal fold activity characteristics of the normal speakers and individuals who stutter, is summarized in Figure 4.1 (also see 3.10.1.1.1). Five types of prephonatory vocal fold activity were identified during the initiation of VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances. Type 1

represents absence of a detectable vocal fold approximation, Type 2 indicates vocal fold approximation without additional prephonatory vocal fold activity, Type 3 represents vocal fold approximation with laryngeal preadjustments, Type 4 depicts vocal fold approximation with irregular prephonatory vocal fold activity, and Type 5 illustrates vocal fold approximation with prevoicing. The five types of prephonatory vocal fold activity were based on the different vocal fold activity characteristics (as reflected on the L*x* waveform) of all the participants. These types or categories of vocal fold events observed during the prephonatory period in the controls and subjects are described and illustrated in Tables 4.1.1 and 4.1.2. The effect of speech sound initiation in different phonetic contexts (i.e. VC, $C_{(v)}$VC and $C_{(vl)}$VC) on the prephonatory vocal fold activity has been addressed separately.

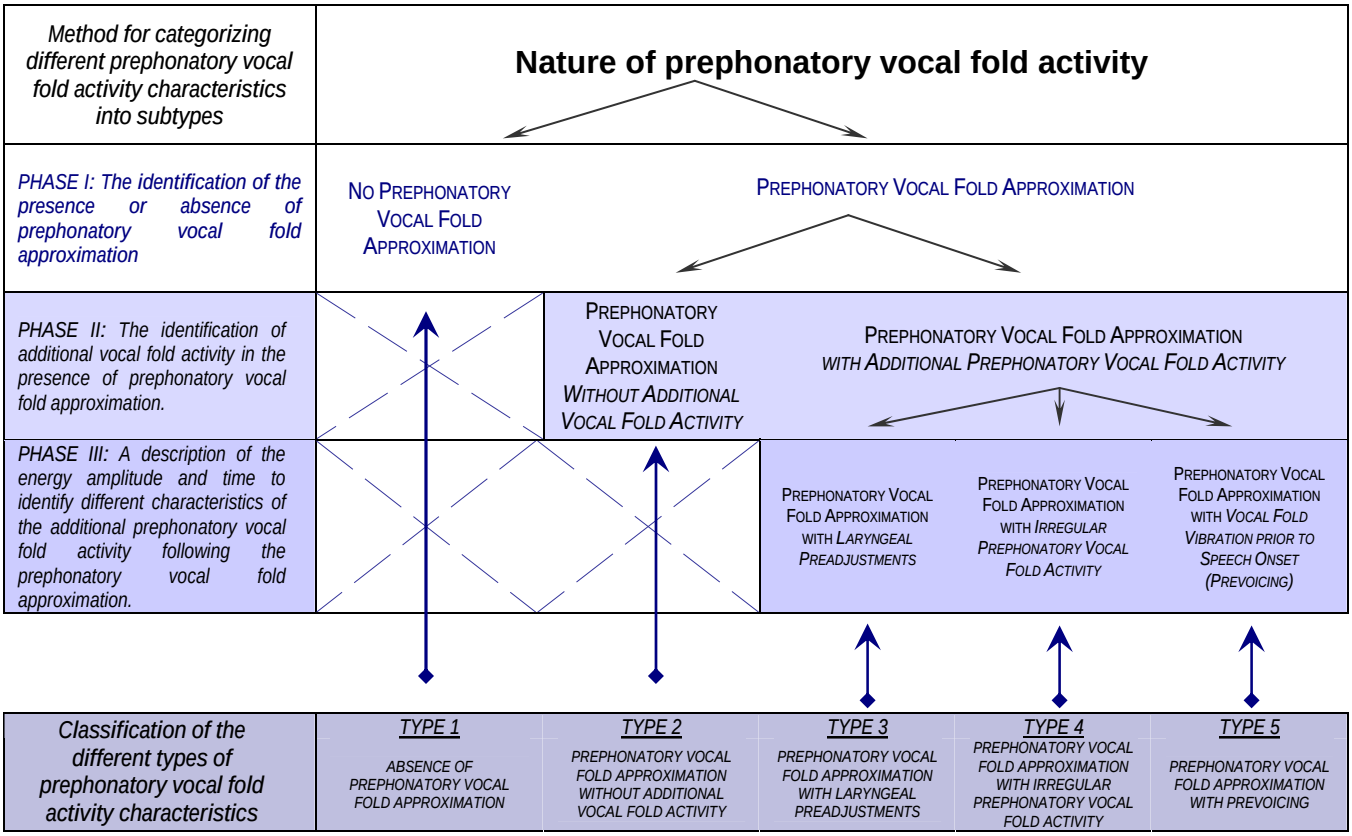


FIGURE 4.1: A FLOW CHART OF THE PROCESS FOR THE IDENTIFICATION AND CLASSIFICATION OF DIFFERENT PREPHONATORY VOCAL FOLD ACTIVITY CHARACTERISTICS

TABLE 4.1.1: A SUMMARY OF THE TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY DURING SPEECH INITIATION IN A GROUP OF NORMALLY FLUENT SPEAKERS AND PERSONS WHO STUTTER (PWS): TYPE 1 AND TYPE 2

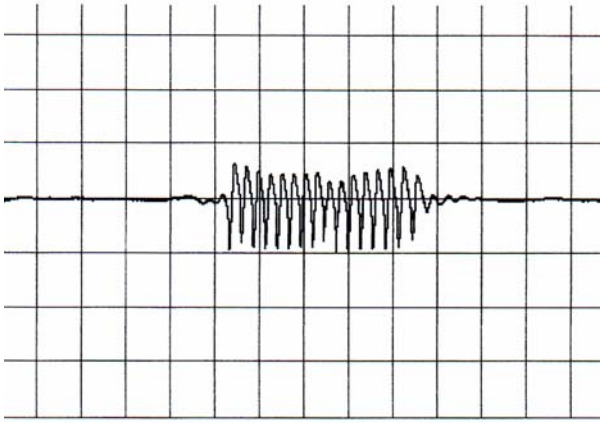
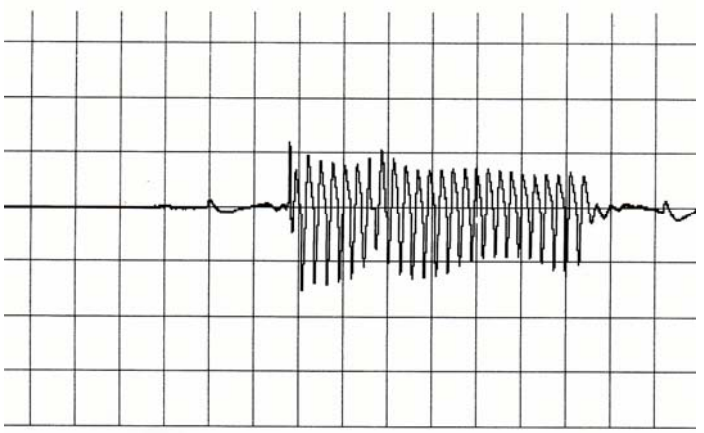
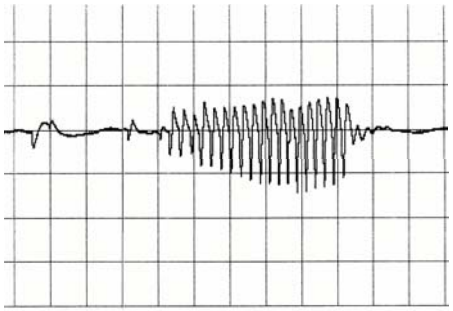
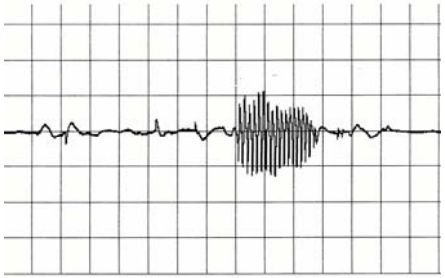
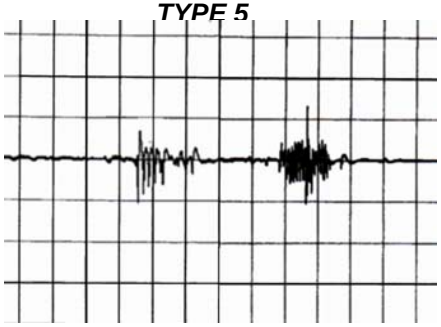
TYPE OF PREPHONATORY VOCAL FOLD ACTIVITY	DESCRIPTION OF TYPE OF PREPHONATORY VOCAL FOLD ACTIVITY	ILLUSTRATION OF TYPE OF PREPHONATORY VOCAL FOLD ACTIVITY AS DISPLAYED ON THE L*x* SIGNAL
TYPE 1: ABSENCE OF PREPHONATORY VOCAL FOLD APPROXIMATION	Type 1 was present if no change in amplitude occurred in the L*x* signal before the start of vocal fold vibrations for the utterance. When phonation is initiated from an open glottis position in utterances beginning with a voiceless consonant, it is regarded as normal speech behaviour (Peters & Boves, 1988). The acoustic burst for the voiceless consonant (/p/) precedes voice onset for the vowel. If vocal fold approximation is absent for voiced plosives, it may indicate that the voiced characteristic of the sound was substituted with voiceless production (/p/ instead of /b/). The absence of vocal fold approximation would be regarded as inappropriate vocal fold positioning in preparation of initial vowel productions. When no prevoicing vocal fold approximation is evident, it can be an indication of deliberate and extremely gentle voice onset (Peters & Boves, 1988). The vocal folds may be adducted or approximated very gently and therefore the change in the energy amplitude over time pulse in the L*x* signal may not be detectable	***TYPE 1*** 
TYPE 2: VOCAL FOLD APPROXIMATION WITHOUT ADDITIONAL PREPHONATORY VOCAL FOLD ACTIVITY	Normal phonation onset appears to be associated with prephonatory arytenoid posturing (adduction) (Hess, Verdolini, Bierhals, Mansmann & Gross, 1998). The initial closure gesture of the glottis in preparation for phonation can be seen as a single amplitude peak in the L*x* signal preceding voice onset. No additional L*x* wave activity is present (Peters & Boves, 1988). This event coincides with the moment that the subglottal pressure first starts to increase (Peters & Boves, 1988). For the purpose of the study, prephonatory vocal fold adduction is perceived as the first pulse of the L*x* signal, after a baseline was established. According to Borden *et al.* (1985) the presence of a single L*x* signal pulse does not necessarily indicate complete glottal closure. This type of L*x* waveform is seen in both VC and $C_{(v)}$VC productions, it can also be present in $C_{(vl)}$VC productions. Although voiceless sound production does not require vocal fold adduction, the vocal folds might approximate in preparation to the following vowel.	***TYPE 2*** 

TABLE 4.1.2: A SUMMARY OF THE TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY DURING SPEECH INITIATION IN A GROUP OF NORMALLY FLUENT SPEAKERS AND PERSONS WHO STUTTER (PWS): TYPE 3, TYPE 4 AND TYPE 5

TYPE OF PREPHONATORY VOCAL FOLD ACTIVITY	DESCRIPTION OF THE TYPE OF PREPHONATORY VOCAL FOLD ACTIVITY	ILLUSTRATION OF TYPE OF PREPHONATORY VOCAL FOLD ACTIVITY AS DISPLAYED ON THE L*x* SIGNAL
TYPE 3: VOCAL FOLD APPROXIMATION WITH ASSOCIATED PREPHONATORY VOCAL FOLD ACTIVITY	Some individuals showed short, aborted vocal fold approximations without phonation before voicing such as reported by Hess *et al.* (1998). Type 3 refers to instances where the first pulse indicating glottal closure was followed by a second pulse (or intermediate slower changes to vocal fold contact). The second pulse reflects a change of impedance preceding voice onset (momentary amplitude modulations), which may suggest laryngeal (vocal fold) adjustments to establish better vocal fold contact (Borden *et al.*, 1985). This indicates vocal fold movement and not vocal fold vibration (Hess *et al.*, 1998). The amplitude of the first curve or pulse will therefore be expected to be higher than for the second curve, although amplitude of the L*x* signal may not be a valid index of the strength of vocal fold approximation (Peters & Boves, 1988). Hess *et al.* (1998) associated distinct modulations in amplitude (e.g. amplitude-doubling) with laryngeal adjustments prior to phonation. These events occurred in normal speaker's speech production activity, though at infrequent intervals.	***TYPE 3*** 
TYPE 4: VOCAL FOLD APPROXIMATION WITH IRREGULAR PREPHONATORY VOCAL FOLD ACTIVITY	If vocal fold approximation was followed by aperiodic vocal fold activity before voice initiation, it was considered as irregular prephonatory vocal fold activity. Irregular vocal fold behaviour prior to phonation can be continuous or intermittent aperiodic activity in the L*x* signal. This type of L*x* waveform is not an artefact; it could be associated with a failure to control laryngeal muscle activity prior to phonation (Freeman & Ushijima, 1978; Hess *et al.*, 1998). The waveform displaying irregular activity can be described as having a jagged shape. The frequency domain is substantially featureless. Continuous irregular prephonatory vocal fold activity suggests that the individual maintained the behaviour. Intermittent irregular vocal fold behaviour reflects multiple aborted adductions.	***TYPE 4*** 
TYPE 5: VOCAL FOLD APPROXIMATION WITH VOCAL FOLD VIBRATION (PREVOICING)	If the L*x* signal indicated vocal fold adduction associated with vibratory activity preceding speech initiation, it was recorded as Type 5. Vocal fold vibration was observed as repetitive periodic pulses or tremulous vibratory cycles in the L*x* signal in the absence of detectable acoustic activity. Vocal fold vibration could be related to vocal fold tremor such as observed by Borden *et al.* (1985). The presence of glottal vibration could also be associated with instances where voicing precedes articulation, such as in CVC syllables starting with a voiced /b/. This prevoicing speech behaviour would be reflected as negative voice onset time.	***TYPE 5*** 

4.2.1.2 Description of the nature of prephonatory vocal fold activity in a group of normal speakers

According to the data in Tables 4.2.1 to 4.2.3, the type of prephonatory vocal fold activity that occurred most frequently across the 12 VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances for the control group (NS 1-6) was Type 2, *prephonatory vocal fold approximation without additional vocal fold activity*. For Type 2, the control group obtained an average of 7 for the VC context. The averages for the $C_{(vl)}$VC and $C_{(v)}$VC contexts were respectively, 5.7 and 5.2 (Tables 4.2.2 and 4.2.3).

A summary of the percentages of the average frequency of occurrence of Types 1 to 5 prephonatory vocal fold activity during the initiation of VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances in the control group are displayed in Figure 4.2. Figure 4.2 illustrates that Type 2 occurred most frequently during speech initiation for the control group, but it also shows that the other types of prephonatory vocal fold activity were present to a greater or lesser extent for the different phonetic contexts. Type 3, *prephonatory vocal fold approximation with laryngeal preadjustments,* was seen in more than 25% of VC productions for the control group. Type 5, *prephonatory vocal fold approximation with prevoicing,* was observed in more than 25% of the $C_{(v)}$VC productions for this group. If Type 5 co-existed with negative voice onset time (VOT), it can be regarded as normal speech activity. Type 1, *an absence of vocal fold approximation,* was evident in a small percentage (<25%) for the $C_{(vl)}$VC context in the control group. Type 4, *irregular prephonatory vocal fold activity,* occurred in less than 25% of the utterances across all three phonetic contexts. Compared to the VC context, more instances of Type 4 were noted in the $C_{(v)}$VC and $C_{(vl)}$VC contexts. The effect of motor complexity on normal speech production of these utterances needs to be considered when discussing this finding.

TABLE 4.2.1: THE FREQUENCY OF OCCURRENCE OF SPECIFIC TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY FOR 12 VC UTTERANCES IN THE SIX NORMAL SPEAKERS (NS)

VC Context	Normal Speakers (Control Group)s	Specific Types of Prephonatory Vocal Fold Activity				
		TYPE 1	TYPE 2	TYPE 3	TYPE 4	TYPE 5
		No prephonatory vocal fold approximation	Prephonatory vocal fold approximation without additional vocal fold activity	Prephonatory vocal fold approximation with laryngeal preadjustments	Prephonatory vocal fold approximation with irregular vocal fold activity	Prephonatory vocal fold approximation with prevoicing
Frequency of occurrence of Specific Prephonatory Vocal Fold Activity in 12 VC productions in Control Speakers (NS=6)	NS 1	0	2	10	0	0
	NS 2	0	8	4	0	0
	NS 3	0	12	0	0	0
	NS 4	0	5	1	6	0
	NS 5	0	5	6	1	0
	NS 6	0	10	0	2	0
Total Number of Utterances per Type of Prephonatory Vocal Fold Activity for Control Group (N=72)		**0**	**42**	**21**	**9**	**0**
Average Frequency of Occurrence of Types of Prephonatory Vocal Fold Activity for Control Group (N=12)		**0**	**7**	**3.5**	**1.5**	**0**

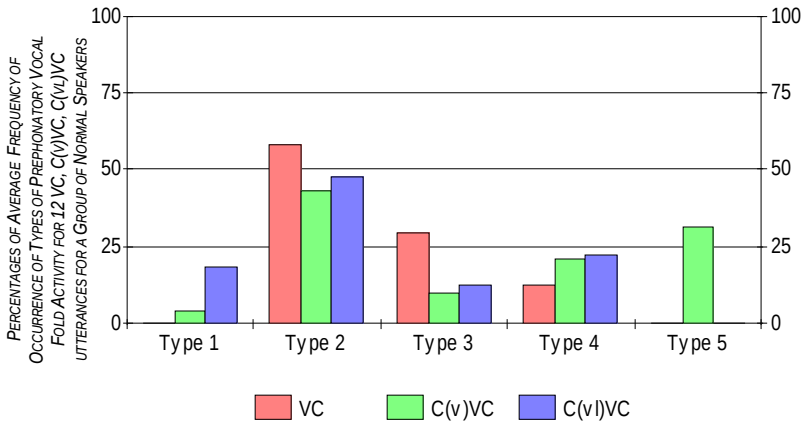
KEY: Type of prephonatory vocal fold activity with highest total number of utterances or frequency of occurrence within specific context

TABLE 4.2.2: THE FREQUENCY OF OCCURRENCE OF SPECIFIC TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY FOR 12 $C_{(v)}$VC UTTERANCES IN THE SIX NORMAL SPEAKERS (NS)

$C_{(v)}$VC Context	Normal Speakers (Control Group)s	Specific Types of Prephonatory Vocal Fold Activity				
		TYPE 1	TYPE 2	TYPE 3	TYPE 4	TYPE 5
		No prephonatory vocal fold approximation	Prephonatory vocal fold approximation without additional vocal fold activity	Prephonatory vocal fold approximation with laryngeal preadjustments	Prephonatory vocal fold approximation with irregular vocal fold activity	Prephonatory vocal fold approximation with prevoicing
Frequency of occurrence of Specific Prephonatory Vocal Fold Activity in 12 $C_{(v)}$VC productions in Control Speakers (NS=6)	NS 1	0	8	4	0	0
	NS 2	3	3	0	0	6
	NS 3	0	10	2	0	0
	NS 4	0	0	0	9	7
	NS 5	0	0	1	4	10
	NS 6	0	10	0	2	0
Total Number of Utterances per Type of Prephonatory Vocal Fold Activity for Control Group (N=72)		**3**	**31**	**7**	**15**	**23**
Average Frequency of Occurrence of Types of Prephonatory Vocal Fold Activity for Control Group (N=12)		**0.5**	**5.2**	**1.2**	**2.5**	**3.8**

TABLE 4.2.3: THE FREQUENCY OF OCCURRENCE OF SPECIFIC TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY FOR 12 $C_{(vl)}$VC UTTERANCES IN THE SIX NORMAL SPEAKERS (NS)

$C_{(vl)}$VC Context	Normal Speakers (Control Group)s	Specific Types of Prephonatory Vocal Fold Activity				
		TYPE 1	TYPE 2	TYPE 3	TYPE 4	TYPE 5
		No prephonatory vocal fold approximation	Prephonatory vocal fold approximation without additional vocal fold activity	Prephonatory vocal fold approximation with laryngeal preadjustments	Prephonatory vocal fold approximation with irregular vocal fold activity	Prephonatory vocal fold approximation with prevoicing
Frequency of occurrence of Specific Prephonatory Vocal Fold Activity in 12 $C_{(vl)}$VC productions in Control Speakers (NS=6)	NS 1	4	6	2	0	0
	NS 2	8	4	0	0	0
	NS 3	1	9	2	0	0
	NS 4	0	3	0	9	0
	NS 5	0	0	5	7	0
	NS 6	0	12	0	0	0
Total Number of Utterances per Type of Prephonatory Vocal Fold Activity for Control Group (N=72)		**13**	**34**	**9**	**16**	**0**
Average Frequency of Occurrence of Types of Prephonatory Vocal Fold Activity for Control Group (N=12)		**2.2**	**5.7**	**1.5**	**2.7**	**0**



SPECIFIC TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY DURING THE INITIATION OF VC, C(V)VC AND C(VL)VC UTTERANCES IN A GROUP OF NORMAL SPEAKERS

FIGURE 4.2: A BAR CHART DISPLAYING THE PERCENTAGES OF THE AVERAGE FREQUENCY OF OCCURRENCE OF SPECIFIC PREPHONATORY VOCAL FOLD CHARACTERISTICS DURING THE INITIATION OF VOICED AND VOICELESS UTTERANCES IN A GROUP OF NORMAL SPEAKERS

4.2.1.3 Frequency of occurrence of different types of prephonatory vocal fold activity in persons who stutter (PWS) compared to normal speakers

Table 4.3 indicates the numeric data of the frequency of occurrence from the 12 VC, $C_{(vl)}$VC and $C_{(vl)}$VC utterances of different types of prephonatory vocal fold activity in the control group (NS) and subject group (S). The following description of the different types of vocal fold activity which manifested during the prephonatory period in the subjects and controls will be based on Table 4.3 and Figure 4.3. Figure 4.3 illustrates the difference in the mean frequency of occurrence for Type 1, Type 2, Type 3, Type 4 and Type 5 in the VC, $C_{(v)}$VC and $C_{(vl)}$VC contexts respectively between the control group and subject group. Figures 4.4.1 to 4.4.3, in addition, depicts the frequency of occurrence of the five types of prephonatory vocal fold activity across the phonetic contexts for the six individual subjects. Figure 4.3 and Figures 4.4.1 to 4.4.3 are based on the data in Table 4.3 of the types of prephonatory vocal fold activity for VC, $C_{(v)}$VC and $C_{(vl)}$VC productions in the subject group and control group.

TABLE 4.3: NATURE OF PREPHONATORY VOCAL FOLD ACTIVITY IN PERSONS WHO STUTTER: A SUMMARY OF THE FREQUENCY OF OCCURRENCE OF DIFFERENT TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY IN SUBJECTS (N=6) ACROSS 12 PRODUCTIONS FOR THREE PHONETIC CONTEXTS. THE FREQUENCY VALUES ARE GIVEN AS THE NUMBER OF RESPONSES AS WELL AS THE PERCENTAGE OF RESPONSES FOR A SPECIFIC TYPE OF VOCAL FOLD ACTIVITY. MEAN FREQUENCY SCORES OF THE DIFFERENT TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY IN VC, $C_{(v)}VC$ AND $C_{(vl)}VC$ CONTEXTS FOR THE GROUP OF SUBJECTS AND CONTROLS ARE SUMMARIZED.

	SUBJECTS (STUTTERING SEVERITY FOR EACH SUBJECT IS INDICATED)	DIFFERENT TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY														
		TYPE 1 ABSENCE OF VISIBLE VOCAL FOLD APPROXIMATION			***TYPE 2*** VOCAL FOLD APPROXIMATION WITHOUT ADDITIONAL PREPHONATORY VOCAL FOLD ACTIVITY			***TYPE 3*** VOCAL FOLD APPROXIMATION WITH LARYNGEAL PREADJUSTMENTS			***TYPE 4*** VOCAL FOLD APPROXIMATION WITH IRREGULAR PREPHONATORY VOCAL FOLD ACTIVITY			***TYPE 5*** VOCAL FOLD APPROXIMATION WITH VOCAL FOLD VIBRATION (PREVOICING)		
PHONETIC CONTEXTS		VC	$C_{(v)}VC$	$C_{(vl)}VC$	VC	$C_{(v)}VC$	$C_{(vl)}VC$	VC	$C_{(v)}VC$	$C_{(vl)}VC$	VC	$C_{(v)}VC$	$C_{(vl)}VC$	VC	$C_{(v)}VC$	$C_{(vl)}VC$
FREQUENCY OF OCCURRENCE (I.E. NUMBER OF RESPONSES AND PERCENTAGE OF RESPONSES) OF SPECIFIC PREPHONATORY VOCAL FOLD ACTIVITY IN 12 SPEECH PRODUCTIONS FOR DIFFERENT PHONETIC CONTEXTS IN SIX SUBJECTS	S1 III	0	0	0	3	0	0	3	0	0	5	12	12	1	0	1
		0%	*0%*	*0%*	*25%*	*0%*	*0%*	*25%*	*0%*	*0%*	*42%*	*100%*	*100%*	*8%*	*0%*	*8%*
	S2 IV	0	0	0	0	0	1	7	1	4	5	11	7	0	7	0
		0%	*0%*	*0%*	*0%*	*0%*	*8%*	*58%*	*8%*	*33%*	*42%*	*92%*	*58%*	*0%*	*58%*	*0%*
	S3 IV	0	0	0	1	0	0	0	1	1	11	12	11	2	0	0
		0%	*0%*	*0%*	*8%*	*0%*	*0%*	*0%*	*8%*	*8%*	*92%*	*100%*	*92%*	*17%*	*0%*	*0%*
	S4 II	0	0	0	7	7	5	2	2	3	3	3	4	0	1	1
		0%	*0%*	*0%*	*58%*	*58%*	*42%*	*17%*	*17%*	*25%*	*25%*	*25%*	*33%*	*0%*	*8%*	*8%*
	S5 II	0	0	2	5	7	4	3	1	2	4	3	4	0	1	0
		0%	*0%*	*17%*	*42%*	*58%*	*33%*	*25%*	*8%*	*17%*	*33%*	*25%*	*33%*	*0%*	*8%*	*0%*
	S6 I	0	0	0	0	0	4	3	3	1	9	12	7	0	1	0
		0%	*0%*	*0%*	*0%*	*0%*	*33%*	*25%*	*25%*	*8%*	*75%*	*100%*	*58%*	*0%*	*8%*	*0%*
MEAN FREQUENCIES OF DIFFERENT PREPHONATORY VOCAL FOLD ACTIVITY FROM 12 PRODUCTIONS FOR SUBJECTS AND CONTROLS	*MEAN AVERAGE FOR SUBJECTS*	***0***	***0***	***0.3***	***2.7***	***2.3***	***2.3***	***3***	***1.3***	***1.8***	***6.2***	***8.8***	***7.5***	***0.5***	***1.7***	***0.3***
	MEAN AVERAGE FOR CONTROLS	***0***	***0.5***	***2.2***	***7***	***5.2***	***5.7***	***3.5***	***1.2***	***1.5***	***1.5***	***2.5***	***2.7***	***0***	***3.8***	***0***
KEY		*The highest percentage of responses across types of prephonatory vocal fold activity for the VC, $C_{(v)}VC$ and $C_{(vl)}VC$ contexts in S and NS.*														
		****STUTTERING SEVERITY:*** *I – Very Mild* *II - Mild* *III – Moderate* *IV – Severe*														

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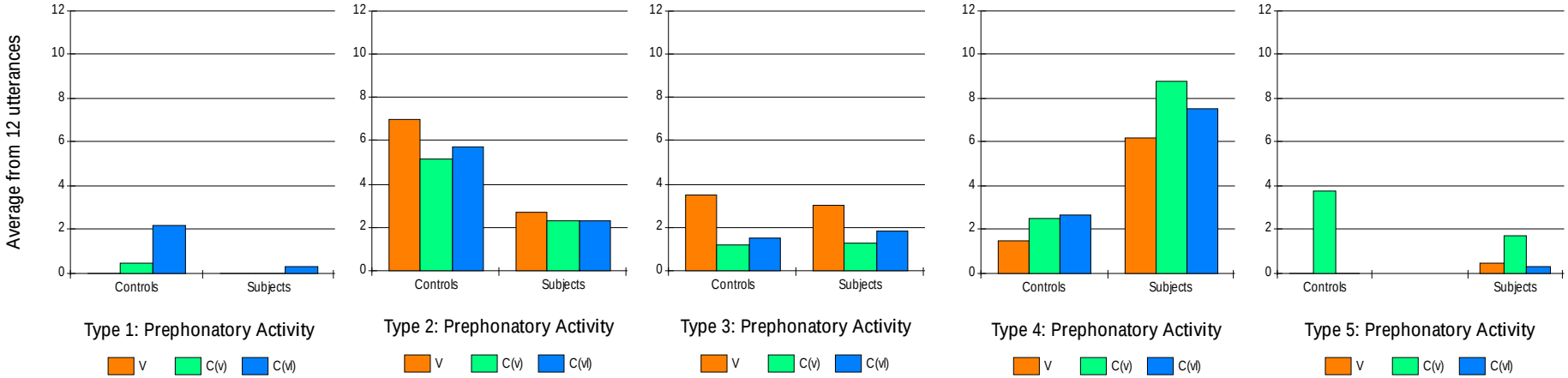

FIGURE 4.3: THE AVERAGE OCCURRENCE OF DIFFERENT TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY DURING INITIATION OF 12 VC, $C_{(v)}$VC AND $C_{(vl)}$VC UTTERANCES: A COMPARISON BETWEEN THE CONTROL GROUP AND THE SUBJECT GROUP (based on Table 4.3)

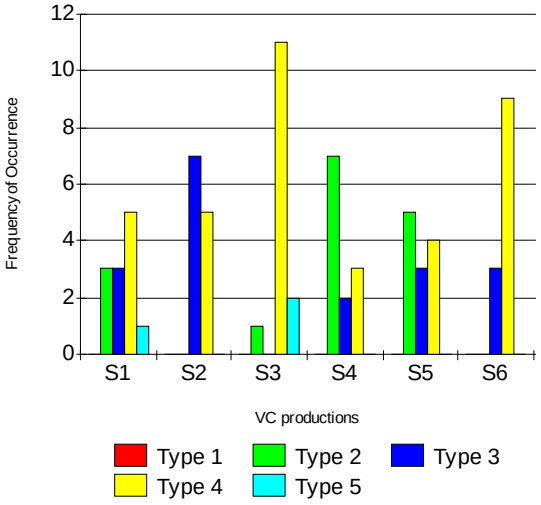

FIGURE 4.4.1: A BAR CHART OF THE FREQUENCY OF OCCURRENCE OF DIFFERENT TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY DURING THE INITIATION OF 12 *VC* UTTERANCES FOR THE SIX PERSONS WHO STUTTER (based on Table 4.3)

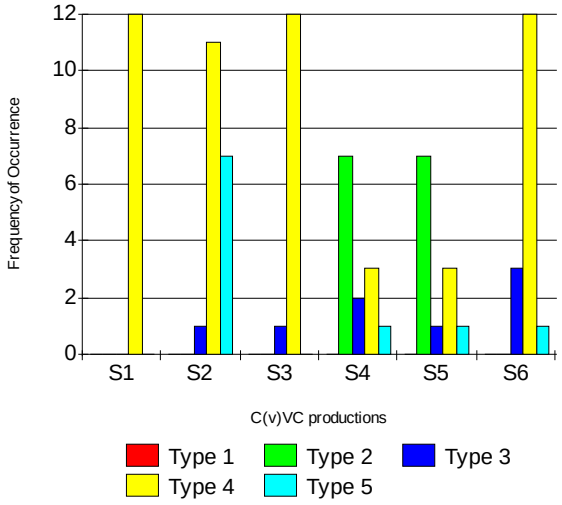

FIGURE 4.4.2: A BAR CHART OF THE FREQUENCY OF OCCURRENCE OF DIFFERENT TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY DURING THE INITIATION OF 12 $C_{(v)}$VC UTTERANCES FOR THE SIX PERSONS WHO STUTTER (based on Table 4.3)

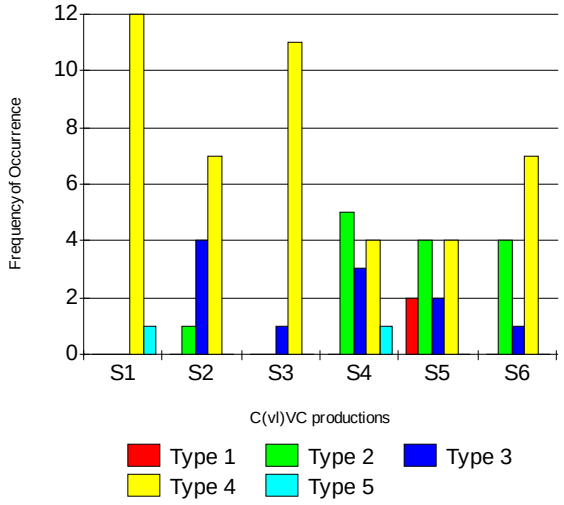

FIGURE 4.4.3: A BAR CHART OF THE FREQUENCY OF OCCURRENCE OF DIFFERENT TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY DURING THE INITIATION OF 12 *$C_{(vl)}$VC* UTTERANCES FOR THE SIX PERSONS WHO STUTTER (based on Table 4.3)

- **Type 1: Absence of prephonatory vocal fold approximation during speech initiation**

Control group: Table 4.3 shows that the control group had some incidences of *the absence of prephonatory vocal fold activity prior to actual speech initiation* (Type 1) for the $C_{(v)}VC$ (average frequency = 0.5) and $C_{(vl)}VC$ (average frequency = 2.2) contexts, but none for the VC context. For the controls, Type 1 was seen in more $C_{(vl)}VC$ productions than the voiced contexts (Figure 4.3). The control group also had more $C_{(vl)}VC$ utterances with Type 1 (average frequency = 2.2) compared to the subject group (average frequency = 0.3) (Table 4.3).

Subject group: According to the data in Table 4.3, the subject group displayed *absence of prephonatory vocal fold activity prior to actual speech initiation* (Type 1) for only the $C_{(vl)}VC$ context. The subject group's low average frequency of 0.3 is attributable to three $C_{(vl)}VC$ utterances with Type 1 in Subject 5 (Table 4.3).

- **Type 2: Prephonatory vocal fold approximation without additional vocal fold activity during speech initiation**

Control group: The control group predominantly exhibited *vocal fold approximation without additional prephonatory vocal fold activity* (Type 2) across the VC, $C_{(v)}VC$ and $C_{(vl)}VC$ contexts (Table 4.3.). For Type 2, the controls had the highest average frequency of seven for VC utterances, followed by the $C_{(vl)}VC$ (average frequency = 5.7) and $C_{(v)}VC$ contexts (average frequency = 5.2) (Table 4.3).

Subject group: Table 4.3 indicates that the subject group obtained low average frequencies of 2.7 for the VC context and 2.3 for both the $C_{(v)}VC$ and $C_{(vl)}VC$ context. Figure 4.3 illustrates that the results of Type 2 for the subject group are markedly lower than the average frequency of occurrence of Type 2 for the control group across the three contexts.

According to the data in Figures 4.4.1 to 4.4.3, Subjects 4 and 5 (in contrast with the other four subjects) showed higher average frequencies for Type 2 across all the contexts. These two mild stuttering speakers (S4 and S5) obtained results for Type 2 that are comparable to those of the control group (Table 4.3).

Subjects 1, 2, 3 and 6 had the lowest frequency of occurrence for Type 2 across the three phonetic contexts (Figures 4.4.1 - 4.4.3). Three of the mentioned subjects (S1, S2 and S3) were

rated as moderate-severe stuttering speakers (Table 3.1). As presented in Figures 4.4.1, 4.4.2 and 4.4.3, the very mild stuttering speaker, Subject 6, evidenced similar prephonatory vocal fold behaviour to Subject 2, a severe stuttering speaker, for all the contexts.

- **Type 3: Prephonatory vocal fold approximation with laryngeal preadjustments during speech initiation**

Control group: Table 4.3 shows that the control group had mean frequencies of occurrence below four for *vocal fold approximation with laryngeal adjustments during speech initiation* (Type 3) across the VC (average frequency = 3.7), $C_{(v)}$VC (average frequency = 1.2) and $C_{(vl)}$VC (average frequency = 1.7) contexts. Thus, only VC utterances had a slight inclination toward Type 3.

Subject group: For the subject group, Type 3 occurred less frequently than Types 2 and 4 for the VC, $C_{(v)}$VC and $C_{(vl)}$VC contexts (Table 4.3) . Similarities were noted in the mean scores of Type 3 for the subject group and control group across all three contexts. For the VC utterances the subjects obtained a mean score of 3 and the controls a score of 3.5; the $C_{(v)}$VC context showed mean scores of 1.3 for subjects compared to 1.2 for controls, and for the $C_{(vl)}$VC context, subjects and controls had mean scores of 1.8 and 1.5, respectively (Table 4.3). As displayed by Figure 4.3, the group of stuttering speakers presented with a higher incidence of Type 3 in the VC context than in the CVC contexts.

For three subjects (S4, S5 and S6) the occurrence of Type 3 showed a similar distribution across the phonetic contexts than observed in the control group (Table 4.3). Subject 2 had the highest number of utterances displaying Type 3 for VC (i.e. seven) and $C_{(vl)}$VC (i.e. four) utterances (Table 4.3). In contrast to the other subjects, Subject 1 and Subject 3 exhibited a small number of utterances with Type 3. Subject 1 had three utterances and Subject 3, only two utterances out of the possible 36 utterances that were characterized by Type 3 (Table 4.3).

- **Type 4: Prephonatory vocal fold approximation with irregular vocal fold activity during speech initiation**

Control group: The control group displayed limited *irregular vocal fold activity in the prephonatory period* (Type 4) (Table 4.3). In this group, less than an average of three utterances across the $C_{(vl)}$VC (average frequency = 2.7), $C_{(v)}$VC (average frequency = 2.5) and VC (average frequency = 1.5) contexts were characterized by Type 4 (Table 4.3).

Subject group: In contrast to the control group, the subject group presented with mean scores above six for Type 4 in each context (Table 4.3). According to Table 4.3, the highest occurrence of Type 4 was evidenced in the $C_{(v)}$VC context (average frequency = 8.8). The subject group also obtained an average frequency of occurrence of 7.5 for the $C_{(vl)}$VC context and 6.2 for the VC context (Table 4.3). In comparison with the other types of prephonatory vocal fold activity, a tendency toward Type 4 across all three phonetic contexts was illustrated for the group of stuttering speakers (Figure 4.3).

The utterances of four subjects (S1, S2, S3 and S6) were predominantly depicted by Type 4, with the $C_{(v)}$VC context showing the most instances of Type 4 (Figure 4.4.2). Thus, Subjects 1, 2, 3 and 6 evidently experienced irregular vocal fold activity before actual speech initiation which might be reflected in laryngeal onset time (L_xOT) measurements (see 4.2.2.).

The mild stuttering speakers (S4 and S5) showed a lower incidence of Type 4 compared to other types of prephonatory vocal fold activity across the three contexts (Table 4.3). These two subjects' results corresponded with the control group's result for Type 4 (Table 4.3). However, both Subject 4 and Subject 5 displayed a higher frequency of occurrence of VC (i.e. three in S4; five in S5), $C_{(v)}$VC (i.e. three in both S4 and S5) and $C_{(vl)}$VC (i.e. four in both S4 and S5) utterances with Type 4 compared to the group of normal speakers (Table 4.3).

- **Type 5: Prephonatory vocal fold approximation with prevoicing during speech initiation**

Control group: According to Table 4.3, *vocal fold approximation with vocal fold vibration prior to actual speech production* (Type 5) was only noted in the $C_{(v)}$VC context for the control group. An average of 3.8 of the control group's $C_{(v)}$VC utterances (thus, more than 25%) displayed Type 5 (Table 4.3). *Prevoicing activity* (Type 5) in $C_{(v)}$VC production might possibly be an indication of negative voice onset and will be compared to the values of voice onset time (VOT) (see 4.3.2).

Subject group: Figure 4.3 reflects that for the subject group, Type 5 was detected in each phonetic context. However, the frequency of occurrence of Type 5 was insignificant across the contexts. Mean scores of below two were obtained for the $C_{(v)}$VC (mean score = 1.7), VC (means score = 0.5), and $C_{(vl)}$VC (mean score = 0.3) productions (Table 4.3). In comparison with the control group (average frequency = 3.8) a lower frequency of occurrence of prevoicing were observed for $C_{(v)}$VC productions in the subject group (average frequency = 1.7) (Table 4.3). If

prevoicing relates to negative VOT, it might indicate that fewer $C_{(v)}VC$ productions in the persons who stuttered were characterized by negative VOT. The individual incidences of Type 5 in Subjects 1, 3 and 4 contributed to the small mean scores of the VC (mean score = 0.5) and $C_{(vl)}VC$ (mean score = 0.3) contexts in the subject group (Table 4.3).

4.2.1.4 Effect of speech sound initiation in different phonetic contexts on prephonatory vocal fold activity

Table 4.4 displays the types of prephonatory vocal fold activity that occurred most frequently during VC, $C_{(v)}VC$ and $C_{(vl)}VC$ productions in both the control group and subject group. Different voicing demands seems to have different effects on prephonatory vocal fold activity which might vary from subject to subject (Table 4.3). For this reason, the individual subjects' responses during the initiation of voiced and voiceless speech utterances are presented in Figures 4.4.1 to 4.4.3.

TABLE 4.4.: THE ARRANGEMENT *(IN DESCENDING ORDER)* OF THE TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY IN TERMS OF FREQUENCY OF OCCURRENCE FOR THE VC, $C_{(v)}VC$ AND $C_{(vl)}VC$ CONTEXTS IN THE CONTROL GROUP AND THE SUBJECT GROUP

	TYPES OF PREPHONATORY VOCAL FOLD ACTIVITY ARRANGED IN DESCENDING ORDER FROM HIGHEST TO LOWEST MEAN FREQUENCY OF OCCURRENCE FOR THE VC, $C_{(v)}VC$ AND $C_{(vl)}VC$ CONTEXTS											
CONTROL/SUBJECT GROUP	CONTROL GROUP						SUBJECT GROUP					
PHONETIC CONTEXT	VC		$C_{(v)}VC$		$C_{(vl)}VC$		VC		$C_{(v)}VC$		$C_{(vl)}VC$	
	Type	Mean Score	Type	Mean Score	Type	Mean Score	Type	Mean Score	Type	Mean Score	Type	Mean Score
MEAN FREQUENCY OF OCCURRENCE — High mean scores	2	7 (58%)	2	5.2 (43%)	2	5.7 (48%)	4	6.2 (52%)	4	8.8 (73%)	4	7.5 (63%)
	3	3.5 (29%)	5	3.8 (32%)	4	2.7 (23%)	3	3 (25%)	2	2.3 (19%)	2	2.3 (19%)
	4	1.5 (13%)	4	2.5 (20%)	1	2.2 (18%)	2	2.7 (23%)	5	1.7 (14%)	3	1.8 (15%)
	1, 5	0	3	1.2 (1%)	3	1.5 (13%)	5	0.5 (0.04%)	3	1.3 (11%)	1, 5	0.3 (0.03%)
Low mean scores			1	0.5 (0.04%)	5	0	1	0	5	0		

Percentages of mean scores are indicated in brackets

- **VC Context: Vowel onset**

Control group: According to the data in Table 4.4, the control group reflected the highest frequency of occurrence of seven out of 12 utterances for *vocal fold approximation without additional prephonatory activity* (Type 2). While Type 2 was predominantly observed in the VC context (58% of the utterances), the control group also displayed Type 3 in 29% of the productions.

Subject group: The effect of the VC context on the nature of prephonatory vocal fold activity in the subject group, shows that productions with vowel onset were mainly characterized by *irregular prephonatory vocal fold activity* (Type 4). Type 4 was noted for 52% of the VC utterances, and secondly, with a smaller percentage of 25% was Type 3 (Table 4.4).

For three subjects (S3, S6 and S1 to a lesser extent), the VC productions were mainly characterized by *irregular prephonatory vocal fold activity* during speech initiation (Type 4) (Figure 4.4.1). In Subjects 2 and 6, the same types of prephonatory vocal fold activity were evidenced, i.e. Type 3 and Type 4 for VC productions (Figure 4.4.1). However, Subject 2 showed a higher percentage of Type 3 (58%), and Subject 6 of Type 4 (58%) (Table 4.3). Subject 4 and Subject 5 displayed a similar distribution of types of prephonatory vocal fold activity for VC productions with the highest occurrence of Type 2 (Type 2 > Type 4 > Type 3) (Figure 4.4.1). These two subjects were rated as mild PWS and their results for the VC context are in close comparison with the control group (Table 4.3).

- **$C_{(v)}$VC Context: Voiced consonant onset**

Control group: For the controls, $C_{(v)}$VC utterances showed the highest frequency of occurrence of Type 2 (average frequency = 5.2) (Table 4.4). Also, *prevoicing vocal fold activity* (Type 5) was observed in more than 25% of the controls' utterances (average frequency = 3.8) (Table 4.4).

Subject group: The effect of $C_{(v)}$VC productions on prephonatory vocal fold activity in the subject group was depicted by irregular activity (Type 4) for 73% of the utterances (Table 4.4).

Four subjects (S1, S2, S3 and S6) displayed mainly $C_{(v)}$VC productions with irregular vocal fold activity (Type 4) (Figure 4.4.2). For three of these subjects (S1, S3 and S6) all 12 $C_{(v)}$VC utterances were depicted by Type 4 (Table 4.3). Although Subject 2 evidenced a high incidence of Type 4 (92%), he also showed *prevoicing vocal fold activity* (Type 5) in 58% of the $C_{(v)}$VC

productions (Table 4.3). This percentage is notably higher than indicated for the control group (32%) (Table 4.3).

Subjects 2 and 6, both revealed prephonatory vocal fold activity of Types 3, 4 and 5 for $C_{(v)}$VC utterances, though, the frequency of occurrence of these types differed for these two subjects (Figure 4.4.2). Subject 6 had more utterances with Type 3 (Type 4 > Type 3 > Type 5) than Subject 2, who had a higher incidence of Type 5 (Type 4 > Type 5 > Type 3) (Figure 4.4.2).

Subject 4 and Subject 5 showed the same distribution of Types 2, 3, 4 and 5 (Figure 4.4.2). For these two mild PWS, Type 2 was predominantly observed in the $C_{(v)}$VC context, followed by Type 4 (Figure 4.4.2). Their results of Type 2 (average frequency: 7) were similar to the control group's average frequency of 5.2 for $C_{(v)}$VC productions (Table 4.3).

- **$C_{(vl)}$VC Context: Voiceless consonant onset**

Control group: For the control group, 48% of the $C_{(vl)}$VC productions displayed Type 2, followed by only 23% productions with Type 4 (Table 4.4).

Subject group: As Table 4.4 indicates, the subject group evidenced Type 4 in 63% of the $C_{(vl)}$VC productions. Only small percentages of Type 2 (19%) and Type 3 (15%) were obtained for the $C_{(vl)}$VC context in this group (Table 4.4).

Subject 1 and Subject 3 evidenced *irregular prephonatory vocal fold activity* (Type 4) in 100% and 92% of the $C_{(vl)}$VC utterances, respectively (Table 4.3). Interesting to note is that these two subjects' results for the $C_{(vl)}$VC context are comparable to their results for the $C_{(v)}$VC context (Figure 4.4.3). Subject 2 and Subject 6 also showed a large percentage of $C_{(vl)}$VC utterances displaying Type 4, i.e. 58%. Fewer utterances evidenced Type 2 and Type 3 in these two subjects (Figure 4.4.3). Although judged as a mild PWS, Subject 6 had similar results for $C_{(vl)}$VC utterances to Subject 2, a severe PWS.

$C_{(vl)}$VC utterances for the mild PWS (S4 and S5) displayed similar frequencies of occurrence in Types 2, 3 and 4 (Figure 4.4.3). For Subject 4, Type 2 and Type 4 occurred in 42% and 33% of the utterances, respectively, and for Subject 5, 33% in both types (Table 4.3).

4.2.1.5 Summary of results on the nature of prephonatory vocal fold activity during speech initiation

In summary, differences in nature of prephonatory vocal fold activity for VC, $C_{(v)}$VC and $C_{(vl)}$VC productions were mainly displayed by Type 2 and Type 4 for the control group and subject group. The effect of different voicing demands on the nature of prephonatory vocal fold activity indicates that CVC utterances are predominantly associated with irregular prephonatory vocal fold activity (Type 2) for the subject group. Furthermore, $C_{(v)}$VC productions (i.e. /b/) had a greater effect on the nature of prephonatory vocal fold activity than $C_{(vl)}$VC (i.e. /p/) for the subject group. Analysis of the results for laryngeal onset time (L_xOT) will reveal whether or not phonetic contexts depicted by irregular prephonatory vocal fold behaviour are related to delayed L_xOT (see 4.2.2.).

Finally, a possible relation between the severity of speakers' stuttering and the occurrence of irregular prephonatory vocal fold activity (Type 4) was observed. The subjects classified as mild, moderate or severe differed in the frequency of occurrence of Type 4, with exception of the very mild PWS (S6*).* Differences in the distribution of Type 2 and Type 4 between the control group and subject group were noted across all three phonetic contexts (Table 4.4). The small difference between the controls and subjects in the average frequencies for Types 1, 3 and 5 across the phonetic contexts was insignificant (Figure 4.3). Apparent similarities between the group of controls and subjects must not obscure the importance of individual instances in which prephonatory vocal fold activity in subjects differed from that of the control group.

4.2.2 *Results for sub-aim 1.2:* Laryngeal onset time (L_xOT)

Laryngeal onset time (L_xOT) represents the prephonatory period in which laryngeal adjustments (e.g. vocal fold approximation or closure) are made prior to the start of phonation. These laryngeal adjustments are synchronized with subglottal pressure changes for the initiation of phonation (Peters & Boves, 1988). The L_xOT value can be regarded as the approximate time a speaker needs to initiate vocal fold vibration following vocal fold approximation.

The next description of the L_xOT results will include tendencies noted in the control group and the subject group pertaining to *mean* L_xOT and *range* of L_xOT across the three phonetic contexts.

The influence of different voicing demands (i.e. vowel onset in VC productions, voiced consonant onset in $C_{(v)}VC$ productions, and voiceless consonant onset in $C_{(vl)}VC$ productions) on *laryngeal onset time* during speech initiation of the PWS will also be presented.

4.2.2.1 Laryngeal onset time (L_xOT) for speech sound initiation in different phonetic contexts

Table 4.5, Table 4.6 and Table 4.7 summarize the L_xOT values for, respectively, the VC, $C_{(v)}VC$ and $C_{(vl)}VC$ contexts of the individual subjects (S1, S2, S3, S4, S5 and S6). The mean L_xOT and range of L_xOT of the subject group (S) and control group (NS) for the different contexts are also presented in Tables 4.5, 4.6 and 4.7. Figure 4.5 illustrates the difference in mean L_xOT and range of L_xOT between the control group and the subject group for the three contexts. The L_xOT range is the difference between the maximum and minimum L_xOT values for a given context. The range of L_xOT reflects on the consistency or variability of the L_xOT values across the number of utterances (N = 12) for a specific voicing context. On a physiological level, the variance in L_xOT might give an indication of the stability of vocal fold control in coordination with subglottal pressure changes during the prephonatory (or prevoicing) period.

TABLE 4.5: THE ANALYSIS OF L_xOT VALUES (in ms) OF THE 12 VC PRODUCTIONS: A PRESENTATION OF THE L_xOT VALUES, MEAN AND RANGE OF L_xOT FOR THE SIX SUBJECTS (S1 – S6). COMPARISONS OF THE MEAN AND RANGE L_xOT VALUES FOR THE VC CONTEXT BETWEEN THE SUBJECT GROUP AND THE CONTROL GROUP ARE INCLUDED.

LARYNGEAL ONSET TIME (L_xOT) IN ms OF VC PRODUCTIONS													
12 VC PRODUCTIONS					SUBJECTS (N = 6) (STUTTERING SEVERITY IS INDICATED AS: I VERY MILD; II MILD; III MODERATE; IV SEVERE)						MEAN L_xOT (IN ms) FOR V1C, V2C, V3C, V4C CONTEXTS	SUBJECT GROUP	CONTROL GROUP
					S1 III	S2 IV	S3 IV	S4 II	S5 II	S6 I			
PHONETIC CONTEXT	VOWEL-CONSONANT (VC)	V1C	/ɑt/	1	248.4	742.2	951.5	139.1	157.8	157.8	/ɑt/	312.58	176.85
			/ɑt/	2	485.9	176.6	645.3	125	245.3	373.4			
			/ɑt/	3	298.4	170.3	376.6	151.6	107.8	73.44			
		V2C	/ət/	4	148.4	596.9	485.9	337.5	417.2	251.6	/ət/	259.81	210.18
			/ət/	5	143.7	89.06	314.17	295.3	132.8	171.9			
			/ət/	6	273.4	296.9	250	243.7	142.2	85.94			
		V3C	/ɪt/	7	101.6	154.7	264.1	182.8	278.1	201.6	/ɪt/	185.23	191
			/ɪt/	8	148.4	340.6	228.1	114.1	104.7	125			
			/ɪt/	9	181.2	157.8	273.4	123.4	223.4	131.2			
		V4C	/ɔt/	10	123.4	260.9	407.8	157.8	123.4	85.94	/ɔt/	207.1	180.89
			/ɔt/	11	198.4	143.7	431.2	109.4	50.0	215.6			
			/ɔt/	12	418.7	125	328.1	131.2	278.1	139.1			
MEAN L_xOT IN ms FOR 12 VC PRODUCTIONS					230.83	271.22	413.01	175.91	188.4	167.71	MEAN L_xOT IN ms FOR VC CONTEXT	241.18	189.73
RANGE OF L_xOT FOR VC PRODUCTIONS (MAXIMUM – MINIMUM L_xOT IN ms)					*(485.9-101.6)* 384.3	*(742.2-89.06)* 653.14	*(951.5-228.1)* 723.4	*(337.5-109.4)* 228.1	*(417.2-50)* 367.2	*(373.4-73.44)* 229.96	AVERAGE L_xOT RANGE FOR VC CONTEXT	442.68	256.36

* *Maximum L_xOT in ms* *Minimum L_xOT in ms*

TABLE 4.6: THE ANALYSIS OF L_xOT VALUES (in ms) OF THE 12 $C_{(v)}VC$ PRODUCTIONS: A PRESENTATION OF THE L_xOT VALUES, MEAN AND RANGE OF L_xOT FOR THE SIX SUBJECTS (S1 – S6). COMPARISONS OF THE MEAN AND RANGE L_xOT VALUES FOR THE $C_{(v)}VC$ CONTEXT BETWEEN THE SUBJECT GROUP AND THE CONTROL GROUP ARE INCLUDED.

LARYNGEAL ONSET TIME (L_xOT) IN ms OF $C_{(v)}VC$ PRODUCTIONS													
12 $C_{(v)}VC$ PRODUCTIONS					STUTTERING SUBJECTS (N = 6) (STUTTERING SEVERITY IS INDICATED AS: I VERY MILD; II MILD; III MODERATE; IV SEVERE)						MEAN L_xOT(IN ms) FOR $C_{(v)}V_1C$, $C_{(v)}V_2C$, $C_{(v)}V_3C$, $C_{(v)}V_4C$ CONTEXTS	SUBJECT GROUP	CONTROL GROUP
					S1 III	S2 IV	S3 IV	S4 II	S5 II	S6 I			
PHONETIC CONTEXT	CONSONANT(VOICED)-VOWEL-CONSONANT ($C_{(v)}VC$)	$C_{(v)}V1C$	/bɑt/	1	309.4	156.3	220.3	150	79.69	112.5	/bɑt/	226.91	159.68
			/bɑt/	2	526.6	67.19	212.5	46.88	200	385.9			
			/bɑt/	3	395.3	168.7	376.6	25	479.7	171.9			
		$C_{(v)}V2C$	/bət/	4	440.6	448.4	867.2	134.4	26.56	276.6	/bət/	292.02	167.2
			/bət/	5	789.1	253.1	164.1	139.1	34.37	279.7			
			/bət/	6	775	40.62	140.6	196.9	142.2	107.8			
		$C_{(v)}V3C$	/bɪt/	7	410.9	176.6	423.4	282.8	59.37	242.2	/bɪt/	267.1	190.04
			/bɪt/	8	626.6	289.1	221.9	132.8	382.8	212.5			
			/bɪt/	9	490.6	314.1	153.1	132.8	195.3	60.94			
		$C_{(v)}V4C$	/bɔt/	10	564.1	329.7	192.2	48.44	46.88	223.4	/bɔt/	277.6	150.6
			/bɔt/	11	678.1	195.3	306.2	267.2	134.4	479.7			
			/bɔt/	12	618.7	232.8	320.3	145.3	51.56	162.5			
MEAN L_xOT IN ms FOR 12 $C_{(v)}VC$ PRODUCTIONS					552.08	222.66	299.87	141.8	152.74	226.3	MEAN L_xOT (IN ms) FOR $C_{(v)}VC$ CONTEXT	265.91	166.88
RANGE OF L_xOT FOR $C_{(v)}VC$ PRODUCTIONS (MAXIMUM – MINIMUM L_xOT IN ms)					*(789.1-309.4)* 479.7	*(448.4-40.62)* 407.78	*(867.2-140.6)* 726.6	*(282.8-25)* 257.8	*(479.7-26.56)* 453.14	*(479.7-60.94)* 418.76	AVERAGE L_xOT RANGE FOR $C_{(v)}VC$ CONTEXT	457.3	242.38

* *Maximum L_xOT in ms* *Minimum L_xOT in ms*

TABLE 4.7: **THE ANALYSIS OF L_xOT VALUES (in ms) OF THE 12 $C_{(vl)}VC$ PRODUCTIONS: A PRESENTATION OF THE L_xOT VALUES, MEAN AND RANGE OF L_xOT FOR THE SIX SUBJECTS (S1 – S6). COMPARISONS OF THE MEAN AND RANGE L_xOT VALUES FOR THE $C_{(vl)}VC$ CONTEXT BETWEEN THE SUBJECT GROUP AND THE CONTROL GROUP ARE INCLUDED.**

LARYNGEAL ONSET TIME (L_xOT) IN ms OF $C_{(vl)}VC$ PRODUCTIONS													
12 $C_{(vl)}VC$ PRODUCTIONS				STUTTERING SUBJECTS (N = 6) (*STUTTERING SEVERITY IS INDICATED AS: I VERY MILD; II MILD; III MODERATE; IV SEVERE*)						MEAN L_xOT, IN ms, FOR $C_{(vL)}V1C$, $C_{(vL)}V2C$, $C_{(vL)}V3C$, $C_{(vL)}V4C$ CONTEXTS	SUBJECT GROUP	CONTROL GROUP
				S1 III	S2 IV	S3 IV	S4 II	S5 II	S6 I			
PHONETIC CONTEXT	CONSONANT(VOICELESS)-VOWEL-CONSONANT	$C_{(vl)}V1C$	/pɑt/ 1	453.1	28.12	853.1	79.69	279.7	182.8	/pɑt/	240.45	134.14
			/pɑt/ 2	589.1	45.31	262.5	26.56	14.06	260.9			
			/pɑt/ 3	500	76.56	484.4	112.5	48.44	31.25			
		$C_{(vl)}V2C$	/pət/ 4	225	20.31	309.4	140.6	181.2	673.4	/pət/	188.45	123.35
			/pət/ 5	353.1	200	287.5	54.69	0	50			
			/pət/ 6	375	56.25	346.9	56.25	12.5	50			
		$C_{(vl)}V3C$	/pɪt/ 7	851.6	42.19	370.3	379.7	346.9	51.56	/pɪt/	263.01	127.68
			/pɪt/ 8	278.1	85.94	310.7	221.9	385.9	40.62			
			/pɪt/ 9	609.4	54.69	642.2	34.37	0	28.12			
		$C_{(vl)}V4C$	/pɔt/ 10	831.2	48.44	443.7	32.81	353.1	239.1	/pɔt/	250.66	112.07
			/pɔt/ 11	567.2	135.4	359.4	34.37	128.1	48.44			
			/pɔt/ 12	504.7	78.13	321.9	103.1	240.6	42.19			
MEAN L_xOT IN ms FOR 12 $C_{(vL)}VC$ PRODUCTIONS				511.46	72.61	416	106.38	165.87	141.53	MEAN L_xOT (IN ms) FOR $C_{(vL)}VC$ CONTEXT	235.64	124.31
RANGE OF L_xOT FOR $C_{(vL)}VC$ PRODUCTIONS (MAXIMUM – MINIMUM L_xOT IN ms)				*(851.6-225)* 626.6	*(200-20.31)* 179.69	*(853.1-262.5)* 590.6	*(379.7-26.56)* 353.14	*(385.9-0)* 385.9	*(673.4-28.12)* 645.28	AVERAGE L_xOT RANGE FOR $C_{(vL)}VC$ CONTEXT	463.54	196.30

* *Maximum L_xOT in ms* *Minimum L_xOT in ms*

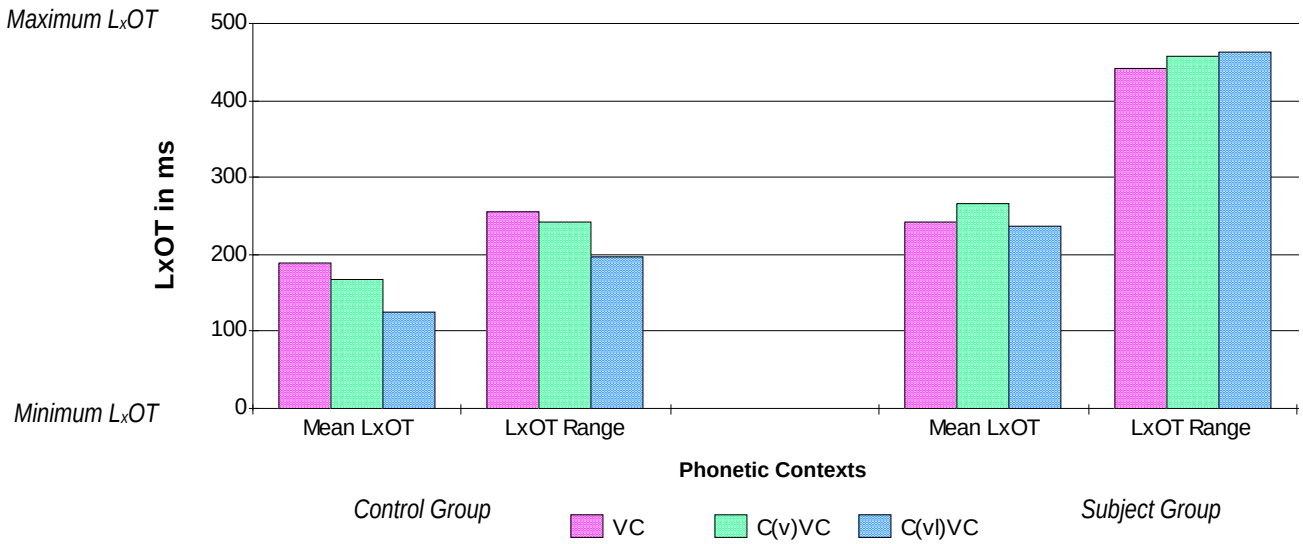

FIGURE 4.5: **A BAR CHART OF THE COMPARISONS OF THE MEAN L_xOT (in ms) AND RANGE OF L_xOT (in ms) FOR THE VC, $C_{(v)}VC$ AND $C_{(vl)}VC$ CONTEXTS BETWEEN THE CONTROL GROUP AND THE SUBJECT GROUP**

- **L_xOT of controls and subjects for initiation of speech sounds in VC context**

Table 4.5 shows the *mean L_xOT* and *range of L_xOT* for the VC context of the control group and subject group, including the individual subjects. The data information (i.e. L_xOT values) for the description of the L_xOT results in the VC context is based on Table 4.5, except where indicated differently.

- **Mean L_xOT of controls and subjects for vowel onset**

Between-group comparison: The control group obtained a mean L_xOT of 189.73 ms for VC productions. The subject group showed a notably greater mean value of 241.18 ms compared to the control group. For the subject group, the mean L_xOT value obtained for the initial vowel productions of /at/ differed greatly from the mean L_xOT values of the other types of VC productions (i.e. /ət/, /ɪt/, /ɔt/). This result is attributed to four of the six subjects (S1, S2, S3 and S6) who displayed maximum L_xOT values for /at/ productions ranging between 373.4 ms and 951.5 ms. The two mild PWS (S4 and S5) exhibited maximum L_xOT values for /ət/ productions. Both the initial vowels in /at/ and /ət/ are produced with a central position of the articulators (see 3.6.2). These VC utterances could have been preceded by the voiceless glottal plosive, /ʔ/ (De Villiers & Ponelis, 1987). In such cases the complexity of the phonetic context would have changed to a $C_{(vl)}$VC context, i.e. /ʔat/ or /ʔət/.

Subject group (within-group comparison): For VC production, three subjects rated as moderate to severe PWS (S1, S2 and S3) presented with high L_xOT values, i.e. 230.83 ms, 271.22 ms and 413.01 ms, respectively. The other subjects (S4, S5 and S6) had mean L_xOT values which ranged between 167.71 ms and 188.4 ms. Their stuttering severity ranged from very mild to mild. The results of these three subjects (S4, S5 and S6) for mean L_xOT were comparable to that of the control group (187.73 ms).

- **Range of L_xOT of controls and subjects for vowel onset**

Between-group comparison: The subject group's L_xOT range was 442.68 ms, whereas the control group had a smaller range of 256.36 ms for VC productions. The greater L_xOT range for the subjects is due to the magnitude of the difference between the minimum and maximum L_xOT.

Subject group (within-group comparison): For VC productions, two of the six subjects (S2 and S3) obtained values for range of L_xOT (635.14 ms and 723.4 ms, respectively) that exceeded the range L_xOT of both the control group (256.36 ms) and the subject group (442.68 ms). These two individuals (S2 and S3) were regarded as moderate to severe PWS. The L_xOT range for Subject 4 and Subject 6 (a mild and very mild PWS, respectively) corresponded with that of the control group (256.36 ms) with values of 228.1 ms and 229.96 ms. Subject 1 (a moderate PWS) and Subject 5 (a mild PWS) obtained similar results for L_xOT range, i.e. 384.3 ms and 367.2 ms, respectively. The possible relation between stuttering severity (based on perceptual ratings) and the degree of L_xOT range for VC utterances did not show consistency within the results for the individual subjects.

- **L_xOT of controls and subjects for initiation of speech sounds in $C_{(v)}$VC context**

Table 4.6 shows the *mean L_xOT* and *range of L_xOT* for the $C_{(v)}$VC context of the control group and subject group, including the individual subjects. The data information (i.e. L_xOT values) for the description of the L_xOT results in the $C_{(v)}$VC context is based on Table 4.6, except where indicated differently.

- **Mean L_xOT of controls and subjects for voiced consonant onset**

Between-group comparison: The mean L_xOT of $C_{(v)}$VC productions for the subject group was 265.91 ms. The subject group showed a longer mean L_xOT compared to the control group's mean score of 166.88 ms. Thus, the subjects required a longer period to initiate voicing after laryngeal approximation was accomplished.

Subject group (within-group comparison): The two mild PWS (S4 and S5) obtained mean L_xOT values of 141.8 ms and 152.74 ms, respectively, for the $C_{(v)}$VC context. These results were similar to the mean L_xOT of the control group (166.88 ms). Four of the six subjects (S1, S2, S3 and S6) had mean L_xOT values varying from 222.66 ms to 552.08 ms. Subject 1 (a moderate PWS) displayed the largest mean L_xOT value of 552.08 ms for $C_{(v)}$VC productions. These results show that most subjects had a longer mean L_xOT than the control group. Stuttering severity might have been associated with longer L_xOT in the $C_{(v)}$VC context, though consistency in this regard was not seen amongst all six subjects (e.g. Subject 6 in Table 4.5).

- **Range of L_xOT of controls and subjects for voiced consonant onset**

Between-group comparison: For the $C_{(v)}VC$ context, the range of L_xOT for the subject group of 457.3 ms was markedly greater compared to the control group (242.38 ms). This result is indicative of the high variance of L_xOT values during the initiation of voiced consonant onsets for the subjects. The large range between minimum and maximum L_xOT values for the subject group reflects the inconsistency and instability detected in the subjects' control of speech production processes during speech initiation.

Subject group (within-group comparison): The range of L_xOT of the individual subjects varied between 257.8 ms and 726.6 ms for $C_{(v)}VC$ productions. Great differences were thus displayed in L_xOT range within the subject group. Five of the six subjects (S1, S2, S3, S5 and S6) obtained a L_xOT range greater than the control group (242.38 ms) with values varying from 407.78 ms to 726.6 ms. The subject group, excluding Subject 4, showed a tendency toward more variability in L_xOT values for $C_{(v)}VC$ utterances.

- **L_xOT of controls and subjects for initiation of speech sounds in $C_{(vl)}VC$ context**

Table 4.7 shows the *mean L_xOT* and *range of L_xOT* for the $C_{(vl)}VC$ context of the control group and subject group, including the individual subjects. The data information (i.e. L_xOT values) for the description of the L_xOT results in the $C_{(vl)}VC$ context is based on Table 4.7, except where indicated differently.

- **Mean L_xOT of controls and subjects for voiceless consonant onset**

Between-group comparison: As for the VC and $C_{(v)}VC$ contexts, the $C_{(vl)}VC$ context for the subject group displayed a larger mean L_xOT value (235.64 ms) than the control group (124.31ms). Additionally, for the subject group the mean L_xOT value for /pət/ (188.45 ms) was smaller as opposed to the mean scores for the other three $C_{(vl)}VC$ utterance types, i.e. /pɑt/ (240.45 ms), /pɪt/ (263.01 ms) and /pɔt/ (250.66 ms). The individual subjects' mean L_xOT values showed great differences within the group, with values varying from 72.6 ms to 511.46ms.

Subject group (within-group comparison): Three of the six subjects (S4, S5 and S6) obtained mean L_xOT values for $C_{(vl)}$VC productions ranging between 106.38 ms and 165.38 ms. These three subjects' *mean scores* were similar to that of the control group (124.31 ms). Subjects 4, 5 and 6 were rated as very mild or mild PWS. Two subjects (S1 and S3) indicated as moderate-severe PWS, obtained high mean L_xOT values of 511.46 ms and 416 ms for the $C_{(vl)}$VC context when compared to the other four subjects. A tendency toward longer mean L_xOT for moderate-severe PWS is reflected in the $C_{(vl)}$VC context. This coincides with results for the VC and $C_{(v)}$VC contexts.

- **Range of L_xOT of controls and subjects for voiceless consonant onset**

Between-group comparison: The control group's value for *range* of L_xOT (196.30 ms) for $C_{(vl)}$VC productions was similar to the *mean* L_xOT (124.31 ms). Table 4.7 reflects the similarities in L_xOT values between the different $C_{(vl)}$VC productions, which varied between 112.07 ms and 134.14 ms for the controls. The L_xOT range indicates small differences between minimum and maximum L_xOT values amongst the individual controls (see Appendix G(*iii*)). Thus, the control group showed a tendency toward maintaining constant values for L_xOT across the 12 $C_{(vl)}$VC productions (see Appendix G(*iii*)).

In comparison to the control group, the range of L_xOT (463.54 ms) for initial /p/ productions indicated distinctive differences between the minimum and maximum L_xOT values for the subject group. Thus, the subjects' results demonstrate variability in laryngeal onset time of the $C_{(vl)}$VC productions.

Subject group: From the six subjects, five (S1, S3, S4, S5 and S6) showed high values for range of L_xOT for $C_{(vl)}$VC productions and varied from 353.14 ms to 645.28 ms. These subjects' stuttering severity ranged between very mild and severe. Subjects 1, 3 and 6 obtained the longest L_xOT. Subject 2 showed the smallest difference between the minimum and maximum L_xOT values (20.31 ms and 200 ms) and was rated a severe stuttering subject. Thus, the magnitude of L_xOT range for the $C_{(vl)}$VC context, as well as the $C_{(v)}$VC context did not show consistency with the subjects' level of severity.

The maximum and minimum L_xOT value for three subjects (S2, S3 and S5) were within the same $C_{(vl)}VC$ utterance group. In other words, the prevocalic voiceless /p/ utterances - from which the L_xOT range was calculated - were followed by the *same* vowel. For example, the L_xOT range for Subject 2 was calculated from the smallest L_xOT value seen in /pət/4 and highest L_xOT score in /pət/5. However, this phenomenon did not occur consistently for all the individual subjects.

4.2.2.2 Effect of phonetic context on L_xOT in normal speakers and persons who stutter

Table 4.8 shows the arrangement of the three phonetic contexts according to the greatest value for mean L_xOT and range of L_xOT range for the control group and individual subjects (S1 - S6). The phonetic context with the highest L_xOT values is indicated, as well as the magnitude of the difference between the values for the VC, $C_{(v)}VC$ and $C_{(vl)}VC$ contexts in the control group and six subjects.

TABLE 4.8: ARRANGEMENT OF THE PHONETIC CONTEXTS (INCLUDING VC, $C_{(v)}VC$ AND $C_{(vl)}VC$ CONTEXTS) ACCORDING TO HIGHEST VALUE FOR MEAN L_xOT AND RANGE OF L_xOT FOR THE CONTROL GROUP (NS) AND THE SUBJECTS (S1-S6)

	ARRANGEMENT OF THREE PHONETIC CONTEXTS FROM HIGHEST TO SMALLEST VALUE FOR MEAN L_xOT (M) AND L_xOT RANGE (R) FOR THE CONTROL GROUP (NS) AND SIX SUBJECTS (S1 - S6)													
	ARRANGEMENT OF PHONETIC CONTEXTS FOR MEAN L_xOT (VALUES FOR MEAN L_xOT IN ms ARE INDICATED)							ARRANGEMENT OF PHONETIC CONTEXTS FOR RANGE OF L_xOT (VALUES FOR RANGE OF L_xOT IN ms ARE INDICATED)						
	NS	S1	S2	S3	S4	S5	S6	NS	S1	S2	S3	S4	S5	S6
Mean L_xOT / L_xOT Range	**M**	**M**	**M**	**M**	**M**	**M**	**M**	**R**	**R**	**R**	**R**	**R**	**R**	**R**
Highest L_xOT	VC 189.73	$C_{(v)}VC$ 552.08	VC 271.22	$C_{(vl)}VC$ 416	VC 175.91	VC 188.4	$C_{(v)}VC$ 226.3	VC 256.36	$C_{(vl)}VC$ 626.6	VC 653.14	$C_{(v)}VC$ 726.6	$C_{(vl)}VC$ 353.14	$C_{(v)}VC$ 453.14	$C_{(vl)}VC$ 645.28
↓	$C_{(v)}VC$ 166.88	$C_{(vl)}VC$ 511.46	$C_{(v)}VC$ 222.66	VC 413.01	$C_{(v)}VC$ 141.8	$C_{(vl)}VC$ 165.87	VC 167.71	$C_{(v)}VC$ 242.38	$C_{(v)}VC$ 479.7	$C_{(v)}VC$ 407.78	VC 723.4	$C_{(v)}VC$ 257.8	$C_{(vl)}VC$ 385.9	$C_{(v)}VC$ 418.76
Lowest L_xOT	$C_{(vl)}VC$ 124.31	VC 230.83	$C_{(vl)}VC$ 72.61	$C_{(v)}VC$ 299.87	$C_{(vl)}VC$ 106.38	$C_{(v)}VC$ 152.74	$C_{(vl)}VC$ 141.53	$C_{(vl)}VC$ 196.3	VC 384.3	$C_{(vl)}VC$ 179.69	$C_{(vl)}VC$ 590.6	VC 228.1	VC 367.2	VC 229.96

Control group: For the control group, the effect of different voicing requirements during initiation of voiced and voiceless speech sounds on *mean L_xOT* indicates that the VC context had

the longest L_xOT, followed by the $C_{(v)}$VC context and the $C_{(vl)}$VC context (Table 4.8). A smaller *mean L_xOT* value of 124.31 ms was obtained for the $C_{(vl)}$VC context compared to the VC and $C_{(v)}$VC contexts with values of 189.73 ms and 166.88 ms, respectively (Table 4.8). The magnitude of the difference of laryngeal onset time between the VC and $C_{(v)}$VC contexts was less than in the case of the $C_{(v)}$VC and $C_{(vl)}$VC context (Table 4.8). This indicates that the initiation of voiced speech utterances had a larger influence on L_xOT, than voiceless speech utterances.

The different contexts showed a similar effect on the *range of L_xOT* in controls than for the *mean L_xOT* values, with the greatest value for VC productions (256.36 ms), followed by $C_{(v)}$VC productions (242.38 ms) and $C_{(vl)}$VC utterances (196.30 ms) (Table 4.8). *Range of L_xOT* across the different contexts also suggests that the VC and $C_{(v)}$VC contexts were depicted by the more variable L_xOT values than the $C_{(vl)}$VC context. The difference between the *L_xOT range* for the VC context and $C_{(v)}$VC context were small, compared to the voiceless consonant onset context (Table 4.8). There was a marked similarity between the values of *mean L_xOT* and *range of L_xOT* for each context of the control group (Figure 4.5).

Subject group: The *mean L_xOT* and *L_xOT range* differed across the three phonetic contexts for the subjects. Certain phonetic contexts may therefore have a greater effect on L_xOT than other for a specific individual who stutters. Thus, the individual subjects within the subject group could have displayed unique results in terms of L_xOT. To establish whether the six subjects exhibited a trend toward longer L_xOT or more variable L_xOT for a specific phonetic context, the values for *mean L_xOT* and *range of L_xOT* in the VC, $C_{(v)}$VC and $C_{(vl)}$VC contexts were arranged from the highest (or greatest) to the lowest (or smallest) value as seen in Table 4.8. The effect of different phonetic contexts on L_xOT for each subject is presented in Tables 4.9.1 and 4.9.2.

TABLE 4.9.1: A SUMMARY OF THE L_xOT RESULTS IN THE SIX INDIVIDUAL SUBJECTS: THE EFFECT OF DIFFERENT PHONETIC CONTEXTS (VC, $C_{(v)}$VC AND $C_{(vl)}$VC) ON L_xOT IN INDIVIDUAL SUBJECT WITH MODERATE TO SEVERE STUTTERING INVOLVEMENT

THE EFFECT OF DIFFERENT PHONETIC CONTEXTS ON L_xOT IN THE INDIVIDUAL SUBJECTS WITH MODERATE TO SEVERE STUTTERING INVOLVEMENT
SUBJECT 1: A SUBJECT WITH *MODERATE* STUTTERING INVOLVEMENT
▪ The longer mean L_xOT values were obtained for the $C_{(v)}$VC and $C_{(vl)}$VC contexts compared to the VC context (Table 4.8). Thus, Subject 1 showed high L_xOT values for utterances with initial voiced and voiceless consonants. This finding suggests a general delay in the initiation of vocal fold activity for these CVC productions. ▪ The range of L_xOT values for all three phonetic contexts for Subject 1 exceeded the control group's results of L_xOT range (Table 4.8). Highly variable L_xOT values were noted for utterances across the three contexts for this subject. The $C_{(vl)}$VC (626.6 ms) and $C_{(v)}$VC (479.7 ms) contexts for Subject 1 indicated marked differences between the L_xOT range values in relation to VC utterances (384.3 ms) (Table 4.8). ▪ Interesting to note is that Subject 1 evidenced a higher mean L_xOT value (552.08 ms) than for the range of L_xOT (479.7 ms) for the $C_{(v)}$VC context (Table 4.8). Subject 1 was the only subject with this difference in values of mean L_xOT and range of L_xOT (i.e. mean L_xOT > range L_xOT). High L_xOT values were obtained across all 12 $C_{(v)}$VC productions (especially for /bət/5, /bət/6, /bɪt/8, /bɔt/11 and /bɔt/12) (Table 4.6). Subject 1 maintained long laryngeal onsets prior to vocal fold vibration during the $C_{(v)}$VC utterances.
SUBJECT 2: A SUBJECT WITH *SEVERE* STUTTERING INVOLVEMENT
▪ Although Subject 2 shows the same arrangement of the phonetic contexts for mean L_xOT as the control group (i.e. VC > $C_{(v)}$VC > $C_{(vl)}$VC), distinctly higher mean values were obtained for this subject (Table 4.8). This was evident especially for nine of the 12 $C_{(v)}$VC productions in Subject 2 that showed longer L_xOT than the mean L_xOT of the control group (166.88 ms) (Table 4.6). ▪ Subject 2 had the greatest mean L_xOT and range of L_xOT for VC and $C_{(v)}$VC productions, which differed greatly from the smaller mean and range scores for $C_{(vl)}$VC productions (Table 4.8). For the VC context and $C_{(v)}$VC context similar mean L_xOT values were obtained (i.e. 271.22 ms and 222.66 ms, respectively) (Table 4.8). The voicing component in the VC and $C_{(v)}$VC productions could have contributed to the delayed and highly variable L_xOT.
SUBJECT 3: A SUBJECT WITH *SEVERE* STUTTERING INVOLVEMENT
▪ For Subject 3, the VC, $C_{(v)}$VC and $C_{(vl)}$VC contexts showed longer mean L_xOT compared to the control group (Table 4.8). The greatest differences were noted for the VC (413.01 ms) and $C_{(vl)}$VC (416 ms) contexts where all productions displayed higher L_xOT values than the mean L_xOT values of the control group (189.73 ms and 124.31 ms, respectively) (Tables 4.5 and 4.7). ▪ The range of L_xOT for Subject 3 differed greatly from the other subjects' results across the three contexts (Table 4.8). The VC and $C_{(v)}$VC showed particularly high values for range of L_xOT for Subject 3 (723.4 ms and 726.6 ms, respectively) (Table 4.8). The VC context had L_xOT values ranging between a minimum of 228.1 ms and a maximum of 951.5 ms (Table 4.5) and for the $C_{(v)}$VC context, the L_xOT values varied between a minimum of 140.6 ms and a maximum of 867.2 ms (Table 4.6). The maximum L_xOT value for the $C_{(v)}$VC context was a single incident across the 12 productions that showed such a delayed L_xOT (Table 4.6). Thus, L_xOT for especially the voiced contexts were depicted by highly variable values.

TABLE 4.9.2*:* A SUMMARY OF THE L_xOT RESULTS IN THE SIX INDIVIDUAL SUBJECTS: THE EFFECT OF DIFFERENT PHONETIC CONTEXTS (VC, $C_{(v)}$VC AND $C_{(vl)}$VC) ON L_xOT IN INDIVIDUAL SUBJECT WITH VERY MILD TO MILD STUTTERING INVOLVEMENT

THE EFFECT OF DIFFERENT PHONETIC CONTEXTS ON L_xOT IN THE INDIVIDUAL SUBJECTS WITH VERY MILD TO MILD STUTTERING INVOLVEMENT
SUBJECT 4: A SUBJECT WITH *MILD* STUTTERING INVOLVEMENT
▪ For Subject 4, the arrangement of the three phonetic contexts from the greatest to smallest mean L_xOT values corresponds with that of the control group, i.e. VC > $C_{(v)}$VC > $C_{(vl)}$VC (Table 4.8). The mean L_xOT values for these contexts were also similar to the control group's results (Table 4.8). Interesting to note is that Subject 4 *(as well as the control group)* had longer L_xOT for /ət/ productions compared to the other VC productions (i.e. /ɑt/, /ɪt/ and /ɔt/) (Table 4.5). ▪ Only for $C_{(vl)}$VC utterances did the range of L_xOT for Subject 4 show a great difference when compared to the control group (i.e. 353.14 ms *versus* 196.30 ms) (Table 4.8). The magnitude of the difference between the maximum and minimum L_xOT values was attributed to a single $C_{(vl)}$VC utterance (i.e. /pɪt/7), with a L_xOT value of 379.7 ms (Table 4.7). Ten of the 12 $C_{(vl)}$VC utterances had L_xOT values *smaller* than the control group's mean L_xOT (124.31 ms) (Table 4.7). The values for range of L_xOT reflect that Subject 4 was able to maintain consistent or regular laryngeal onsets for most of the $C_{(vl)}$VC productions, as well as VC and $C_{(v)}$VC productions.
SUBJECT 5: A SUBJECT WITH *MILD* STUTTERING INVOLVEMENT
▪ Subject 5 displayed a longer mean L_xOT for $C_{(vl)}$VC productions than for $C_{(v)}$VC productions (i.e. VC > $C_{(vl)}$VC > $C_{(v)}$VC) (Table 4.8). The mean L_xOT values for Subject 5 were similar to that of Subject 4 across the three phonetic contexts (Table 4.8). ▪ Although Subject 5 obtained mean L_xOT values for the VC and $C_{(v)}$VC contexts comparable to the control group, less consistency was noted for range of L_xOT (Table 4.8). In this respect, the greatest variability for L_xOT values was seen for the $C_{(v)}$VC context (453.14 ms), followed by the $C_{(vl)}$VC (385.9 ms) and VC (367.2 ms) contexts (Table 4.8). For the $C_{(v)}$VC context, the higher range of L_xOT was due to the difference between the maximum L_xOT value of 479.7 ms (/bɑt/3) and a minimum value of 26.56 ms (/bət/4) (Table 4.6). Two of the 12 $C_{(vl)}$VC productions had L_xOT values of 0 ms, indicating the absence of an apparent vocal approximation (Table 4.7). The absence of vocal fold closure in voiceless consonant production can be regarded as normal speech behaviour (Borden & Harris, 1984), but it influenced the L_xOT range value for the $C_{(vl)}$VC context.
SUBJECT 6: A SUBJECT WITH *VERY MILD* STUTTERING INVOLVEMENT
▪ For Subject 6, the $C_{(v)}$VC context displayed the greatest mean L_xOT value (226.3 ms) (Table 4.8). Eight of the 12 $C_{(v)}$VC utterances had greater L_xOT values than the control group's mean (166.88 ms) (Table 4.6), which contributed to the higher mean L_xOT for the $C_{(v)}$VC context. Subject 6 had mean L_xOT values for the VC (167.71 ms) and $C_{(vl)}$VC (141.53 ms) contexts that were similar to the values of the control group (i.e. 189.73 ms and 124.31 ms, respectively) (Table 4.8). The difference between the mean L_xOT value for VC and $C_{(vl)}$VC contexts for Subject 6 was small. ▪ The range of L_xOT for Subject 6 differed greatly from that of control group across all contexts (i.e. $C_{(vl)}$VC > $C_{(v)}$VC > VC) (Table 4.8). The greater values for range of L_xOT for the $C_{(vl)}$VC and $C_{(v)}$VC contexts are a result of isolated incidents of long laryngeal onsets (Tables 4.6 and 4.7). Eight of the 12 $C_{(vl)}$VC productions of Subject 6 had L_xOT values *below* the control group's mean of 124.31 ms (Table 4.7). Thus, Subject 6 generally maintained L_xOT comparable to the control group for $C_{(vl)}$VC productions. A single $C_{(vl)}$VC utterance (/pət/4) showed a markedly long L_xOT of 673.4 ms (Table 4.7). For the $C_{(v)}$VC context, one utterance (/bɪt/9) had a small L_xOT value of 60.94 ms which contributed to the great difference between the maximum and minimum L_xOT values, hence the high range L_xOT value (Table 4.6). Only two utterances, /bɔt/11 (479.7 ms) and /bɑt/2 (385.9 ms), showed high L_xOT values (Table 4.6).

4.2.2.3 Summary of results on laryngeal onset time (L_xOT)

The *mean L_xOT* for the subject group were higher than for the control group across the three phonetic contexts (Figure 4.5). The mean L_xOT for the $C_{(vl)}$VC context showed the greatest difference between the subjects and controls. The difference between the subject group's and control group's mean L_xOT values for VC productions (241.18 ms; 189.73 ms) was not as revealing as for $C_{(v)}$VC productions (265.91 ms; 166.88 ms) and $C_{(vl)}$VC productions (235.64 ms; 124.31 ms) (Tables 4.5, 4.6 and 4.7). The result for the $C_{(v)}$VC context in the subject group might be of importance should the VC context be expected to have a longer L_xOT than the $C_{(v)}$VC context as in the case of the group of normally fluent speakers (Figure 4.5).

As opposed to the controls, the subject group displayed greater values for *L_xOT range* across the three contexts. These values indicate that the subjects experienced great variance between minimum and maximum L_xOT. The values of L_xOT range differed between 442.68 ms and 463.53 ms across the VC, $C_{(v)}$VC and $C_{(vl)}$VC productions for the subject group (Table 4.8). As for the mean L_xOT values, the differences between the range values for the three contexts were small.

4.2.3 Summary of results regarding physiological vocal fold activity during speech initiation

A marked difference is noted between the subject group and control group for laryngeal onset time. Compared to L_xOT of the control group, the subject group showed longer durations for laryngeal onset and more variable L_xOT values across VC, $C_{(v)}$VC and $C_{(vl)}$VC productions. These findings reflect on differences between the subjects and controls concerning the prephonatory period of speech initiation. The high incidence of atypical prephonatory vocal fold activity apparent across all three phonetic contexts, supports this characteristic in speech of the subjects (Table 4.3). Also, the phonetic context that revealed the most variability in L_xOT for the subjects, was the $C_{(vl)}$VC context. The motor complexity of a specific utterance seems to have an effect on prephonatory vocal fold activity. The arrangement of the three phonetic contexts from high to low L_xOT values (*mean L_xOT* and *range of L_xOT*) for the control group is not a reflection

of the individual subjects' results. Only two subjects (S2 and S4) showed the same arrangement of contexts for mean L_xOT than the control group. This indicates that the subject group presented with idiosyncratic prephonatory vocal fold behaviour for different phonetic contexts.

4.3 RESULTS FOR ACOUSTICALLY DETECTED VOCAL FOLD ACTIVITY DURING SPEECH INITIATION

4.3.1 *Results for sub-aim 2.1:* The nature of voice onset energy

The results of the nature of voice onset for individual subjects, as well as for the subject group and control group are summarized in Table 4.10. The data for gentle and abrupt voice onsets were subjected to comparisons between the control group and subject group for different productions with initial voiced and voiceless sounds as illustrated in Figures 4.6.1 and 4.6.2. The effect of the different phonetic contexts on the nature of voice onset for the group of normal speakers and individuals who stutter is demonstrated by Figures 4.7.1 and 4.7.2.

TABLE 4.10: THE NATURE OF VOICE ONSET: A NUMERIC SUMMARY OF THE OCCURRENCE OF GRADUAL AND ABRUPT VOICE ONSET IN THE INDIVIDUAL SUBJECTS (S1 – S6) ACROSS 12 PRODUCTIONS IN DIFFERENT PHONETIC CONTEXTS. THE AVERAGE FREQUENCIES OF OCCURRENCE OF GRADUAL AND ABRUPT VOICE ONSET FOR VC, $C_{(v)}$VC AND $C_{(vl)}$VC PRODUCTIONS IN THE CONTROL GROUP (NS) AND SUBJECT GROUP (S) ARE INCLUDED.

	SUBJECTS *STUTTERING SEVERITY: I: VERY MILD; II: MILD; III: MODERATE; IV: SEVERE*	NATURE OF VOICE ONSET FOR THE VC, $C_{(v)}$VC AND $C_{(vl)}$VC CONTEXTS					
		GRADUAL VOICE ONSET			ABRUPT VOICE ONSET		
		VC	$C_{(v)}$VC	$C_{(vl)}$VC	VC	$C_{(v)}$VC	$C_{(vl)}$VC
Occurrence of Gradual or Abrupt Voice Onset in 12 productions across different Phonetic Contexts in Subjects (N = 6). Percentages are indicated.	**S1 III**	5 *42%*	12 *100%*	7 *58%*	7 *58%*	0 *0%*	5 *42%*
	S2 IV	6 *50%*	8 *67%*	10 *83%*	6 *50%*	4 *33%*	2 *17%*
	S3 IV	3 *25%*	10 *83%*	3 *25%*	9 *75%*	2 *17%*	9 *75%*
	S4 II	5 *42%*	11 *92%*	12 *100%*	7 *58%*	1 *8%*	0 *0%*
	S5 II	7 *58%*	6 *50%*	8 *67%*	5 *42%*	6 *50%*	4 *33%*
	S6 I	4 *33%*	7 *58%*	9 *75%*	8 *67%*	5 *42%*	3 *25%*
AVERAGE FREQUENCY OF OCCURRENCE OF GRADUAL OR ABRUPT VOICE ONSET FROM 12 PRODUCTIONS FOR S AND NS	AVERAGE FREQUENCY OF OCCURRENCE FOR S	5 *42%*	9 *75%*	8.17 *68%*	7 *58%*	3 *25%*	3.83 *32%*
	AVERAGE FREQUENCY OF OCCURRENCE FOR NS	9.83 *82%*	9.5 *79%*	6.67 *56%*	2.17 *18%*	2.5 *21%*	5.33 *44%*

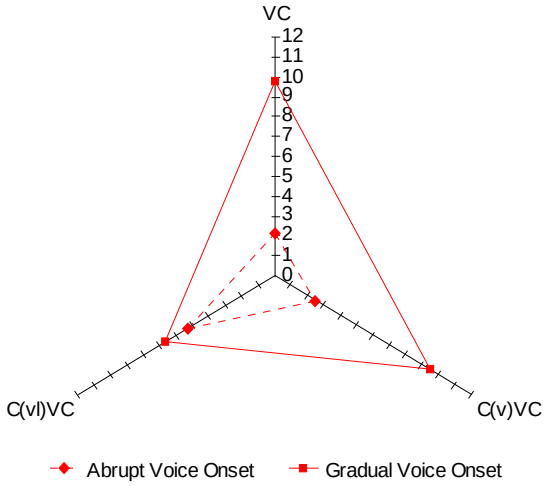

FIGURE 4.6.1: THE FREQUENCY OF OCCURRENCE OF GRADUAL AND ABRUPT VOICE ONSETS ACROSS 12 VC, $C_{(v)}$VC AND $C_{(vl)}$VC PRODUCTIONS IN THE CONTROL GROUP

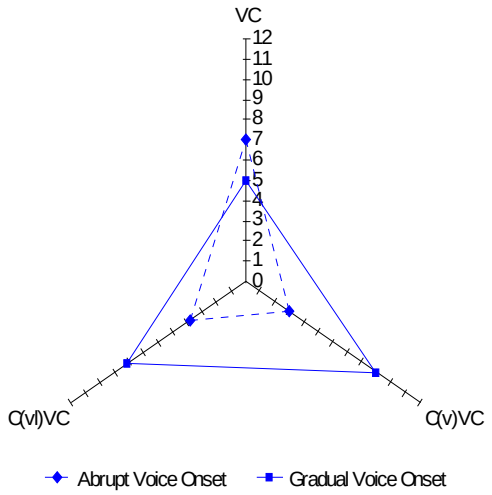

FIGURE 4.6.2: THE FREQUENCY OF OCCURRENCE OF GRADUAL AND ABRUPT VOICE ONSETS ACROSS 12 VC, $C_{(v)}$VC AND $C_{(vl)}$VC PRODUCTIONS IN THE SUBJECT GROUP

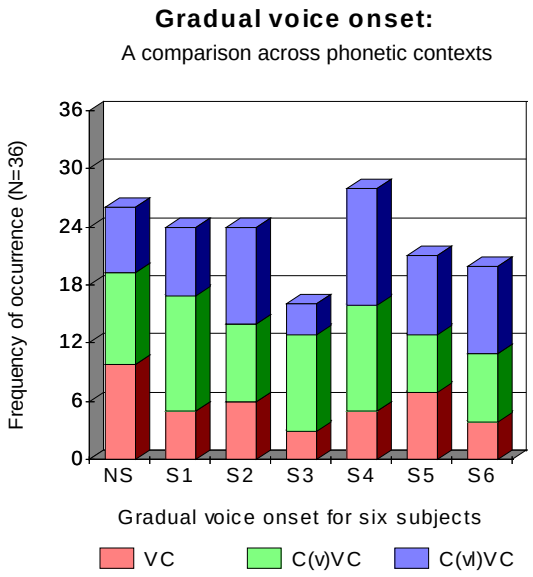

FIGURE 4.7.1: A STACKED BAR CHART OF A COMPARISON OF THE FREQUENCY OF OCCURRENCE OF GRADUAL VOICE ONSETS IN 12 VC, $C_{(v)}$VC AND $C_{(vl)}$VC PRODUCTIONS BETWEEN THE SIX SUBJECTS (S1 - S6) AND THE CONTROL GROUP (NS)

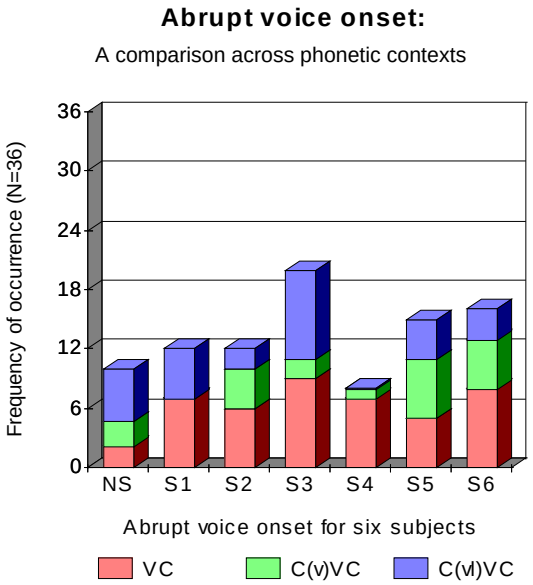

FIGURE 4.7.2: A STACKED BAR CHART OF A COMPARISON OF THE FREQUENCY OF OCCURRENCE OF ABRUPT VOICE ONSETS IN 12 VC, $C_{(v)}$VC AND $C_{(vl)}$VC PRODUCTIONS BETWEEN THE SIX SUBJECTS (S1 - S6) AND THE CONTROL GROUP (NS)

4.3.1.1 Identification and classification of types of voice onset

The identification and classification of types of voice onset were based on the criteria set by previous research by Borden *et al.* (1985) and Dembowski and Watson (1991*a*). Voice onset energy of each production was qualitatively analyzed as either, gentle or abrupt (see 3.10.2.1.1). The nature of voice onset for VC, $C_{(v)}$VC and $C_{(vl)}$VC productions was classified into these two types of voice onset, i.e. gentle/gradual or abrupt/sudden voice onsets (Borden *et al*., 1985; Dembowski & Watson, 1991*a*).

4.3.1.2 Frequency of occurrence of gradual and abrupt voice onset

Control group: In the onset of voicing for normal speech production, a gradual rise in amplitude of the voice signal is usually expected (Borden *et al*., 1985). According to Table 4.10, the control group displayed a higher average frequency of occurrence for gradual voice onset for VC (average score = 9.83; 82%), $C_{(v)}$VC (average score = 9.5; 79%) and $C_{(vl)}$VC (average score = 6.67; 56%) contexts, as opposed to the occurrence of abrupt voice onset. The control group exhibited mostly gradual voice onsets for the voicing contexts (i.e. VC and $C_{(v)}$VC) (Figure 4.6.1). For the $C_{(vl)}$VC productions, 56% showed a gradual voice onset and 44%, an abrupt voice onset (Table 4.10). Thus, the $C_{(vl)}$VC utterances presented with a small difference between gradual and abrupt voice onset (Figure 4.6.1).

Subject group: For the subject group, 75% (average score = 9) $C_{(v)}$VC productions and 68% (average score = 8.17) $C_{(vl)}$VC productions displayed gradual voice onsets (Table 4.10). The subject group maintained a *higher frequency of occurrence* for gradual voice onset than for sudden onsets during the initiation of voiced and voiceless consonants. The VC context showed abrupt voice onsets for 58% (average score = 7) and gradual onsets for 42% (average score = 5) of the productions (Table 4.10). Vowel productions had thus a slightly higher incidence of abrupt voice onset, as opposed to gentle onset (Figure 4.6.2).

A distinctive difference in nature of voice onset between the subject group and the control group was noted for VC productions. The subjects exhibited abruptness in voice onset for 58% (average score = 7) of the VC productions in comparison to the 18% (average score = 2.17) in controls

(Table 4.10). Both the subjects and controls displayed higher percentages for gradual voice onsets in $C_{(v)}VC$ and $C_{(vl)}VC$ utterances. For the $C_{(v)}VC$ context, similar percentages of occurrence for gradual and abrupt voice onset were obtained for the subject group (75% and 25%, respectively) and the control group (79% and 21%, respectively) (Table 4.10). In view of the great differences of L_xOT values for $C_{(v)}VC$ and $C_{(vl)}VC$ productions between the subjects and controls (Table 4.6 and Table 4.7), the existence of gradual voice onset for initial /b/ and /p/ productions for the subjects may be regarded as a compensatory approach to voice onset for fluent speech.

4.3.1.3 Effect of sound initiation in different phonetic contexts on the nature of voice onset energy

- **VC Context: Vowel onset**

For the control group, 82% (average score = 9.83) of the VC utterances was depicted by gradual voice onset (Table 4.10). Contrary to the control group's results, four of the six subjects (S1, S3, S4 and S6) obtained a higher percentage of occurrence for abrupt voice onset for the VC context, ranging between 58% and 95% (Table 4.10). Subject 3 (75%; average score = 9) and Subject 6 (67%; average score = 8) mainly showed abruptness during voice onset of VC productions (Table 4.10). The stuttering severity of these two subjects varied between severe and very mild, thus no apparent relation existed between tendency to abruptness in voice onset in VC productions and stuttering severity.

- **$C_{(v)}VC$ Context: Voiced consonant onset**

As for the VC context, the control group mainly exhibited gradual voice onsets for $C_{(v)}VC$ utterances, i.e. 79% (average score = 9.5) (Table 4.10). Most subjects (S1, S2, S3, S4 and S6) in the subject group also showed gradual voice onsets for the $C_{(v)}VC$ productions which ranged between 58% and 100% utterances (Table 4.10). Although these subjects obtained a high incidence of gradual voice onset for $C_{(v)}VC$ productions, the underlying reason for this occurrence presumably differs from that of the control group.

- **$C_{(vl)}VC$ Context: Voiceless consonant onset**

The control group did not show a meaningful difference between the frequency of occurrence of gentle voice onset (56%; average score = 6.67) and abrupt voice onset (44%; average score = 5.33) for the $C_{(vl)}VC$ context (Table 4.10). In the subject group, five of the six subjects (S1, S2, S4, S5

and S6) evidenced mainly gradual voice onset in $C_{(vl)}VC$ productions with percentage of occurrence ranging between 58% and 100% (Table 4.10). Subject 3 was the only subject who evidenced markedly more instances of abrupt voice onset during $C_{(vl)}VC$ productions, i.e. 75% (average score = 9) (Table 4.10).

4.3.1.4 Summary of results on the nature of voice onset energy

According to the results of the abrupt versus gradual onset of voicing for the *total productions* in individual subjects, most subjects (with the exception of S3) were inclined to produce gradual voice onsets during speech initiation (Figures 4.7.1 and 4.7.2). The great difference between the nature of voice onset for vowel initiation and for (voiced or voiceless) consonant initiation may be a reflection of the influence that motor complexity of the phonetic context has on the onset of vocal fold vibration. It is important to keep in mind that this finding does not imply that gradual voice onset in the subjects is an indication of a so-called normal onset of voicing. For instance, Subject 2 and Subject 6 had abnormally short VOT for /p/ productions (Table 4.12), though the $C_{(vl)}VC$ context showed a higher percentage of gradual voice onset as opposed to abrupt onset for both these subjects (Table 4.10). A gentle voice onset could be a compensatory technique that an individual who stutters employs during speech initiation.

As shown in Figure 4.7.2, all subjects displayed different frequencies of occurrence pertaining to gradual versus abrupt voice onsets across the three contexts. No apparent (or consistent) similarity was found between the nature of voice onset (gentle or abrupt) and stuttering severity. For instance, only two of the three subjects ranging between moderate to severe PWS (S1 and S2) displayed some similarities in nature of voice onset (Figures 4.7.1 and 4.7.2). The three subjects rated as very mild to mild PWS (S4, S5 and S6) also had similar results for only two subjects, i.e. Subject 5 and Subject 6 (Figures 4.7.1 and 4.7.2). The individuals who stutter seemed to have differed with relation to the initiation of voiced or voiceless speech utterances. The results of the nature of voice onset in different phonetic contexts for the individual subjects could be related to individual findings of the nature of prephonatory vocal fold activity (see 4.2.1.), as well as the VOT results (see 4.3.2.).

4.3.2 *Results for sub-aim 2.2:* Voice onset time (VOT)

Precisely timed movements of specific articulators in coordination with laryngeal-phonatory and respiratory movements determine the acoustic characteristics of a particular sound. Plosive production involves a change of glottal adjustment, which is timed relatively consistently in relation to the period of articulatory closure and followed by the sudden release of the closure (Borden & Harris, 1984). The timing of the coordination between the beginning of vocal fold vibration and the release of the articulatory occlusion is depicted by *voice onset time* (VOT) (Borden & Harris, 1984). The temporal margins between the glottal and supraglottal events differ for voiced and voiceless sound equivalents (such as /b/ and /p/) (Borden & Harris, 1984). VOT can therefore be used to establish voiced-voiceless distinction between /b/ and /p/ productions. VOT may be affected by the coarticulatory gestures of the following sound. Thus, the effect of the adjacent vowel on laryngeal-oral interarticulatory coordination of the initial voiced/voiceless plosive consonant must be considered when interpreting VOT results (Hoole, 1997).

In the following description of the results, possible variations in the timing during voice onset for /b/ productions in a $C_{(v)}VC$ context and /p/ productions in a $C_{(vl)}VC$ context would be indicated by VOT. The subjects' *mean scores* and *error values* related to VOT for /b/ productions and /p/ productions are shown in Table 4.11 and Table 4.12, respectively. The mean *scores* and *error values* for VOT for the control group and the subject are also indicated in Table 4.11 and Table 4.12. Table 4.13 is a summary of the VOT values for initial /b/ and /p/ productions that includes the *minimum and maximum VOT values*, the *VOT range* for the control group and the six subjects, as well as the *error values for the VOT range* for the subjects (S1 - S6). Furthermore, the VOT range for prevocalic /b/ and /p/ productions for the control group and six subjects are illustrated in Figures 4.8.1 and 4.8.2, respectively.

TABLE 4.11: THE ANALYSIS OF VOICE ONSET TIME (VOT) VALUES (in ms) OF THE 12 $C_{(v)}VC$ PRODUCTIONS: A PRESENTATION OF THE ABSOLUTE VOT VALUES, MEAN AND ERROR VALUES OF VOT FOR THE SIX INDIVIDUAL SUBJECTS (S1 – S6). COMPARISONS OF THE MEAN AND ERROR VOT VALUES FOR THE $C_{(v)}VC$ CONTEXT BETWEEN THE SUBJECT GROUP AND THE CONTROL GROUP ARE INCLUDED.

VOICE ONSET TIME (VOT) IN ms OF /b/ PRODUCTIONS															
12 $C_{(v)}VC$ PRODUCTIONS					ABSOLUTE VOT AND VOT ERROR VALUES (IN *ms*) FOR THE SIX SUBJECTS							MEAN VOT VALUES (IN ms) FOR THE FOUR DIFFERENT $C_{(v)}VC$ PRODUCTIONS IN THE SUBJECT GROUP AND THE CONTROL GROUP		
					ABSOLUTE VOT VALUE (AV)	SUBJECTS (N = 6) (STUTTERING SEVERITY IS INDICATED AS: I VERY MILD; II MILD; III MODERATE; IV SEVERE)						xAV	SUBJECT GROUP	CONTROL GROUP
					VOT ERROR VALUE (EV)	S1 III	S2 IV	S3 IV	S4 II	S5 II	S6 I	*xEV*		
PHONETIC CONTEXT	VOICED CONSONANT-VOWEL-CONSONANT ($C_{(v)}VC$)													
		$C_{(v)}V1C$	/bɑt/	1	AV	-167.7	+9.38	+10.94	+12.5	-45.31	-62.5	xAV for /bɑt/	-34.49	-51
					EV	*0*	*0*	*0*	*0*	*0*	*0*			
			/bɑt/	2	AV	-123.4	+12.5	+12.5	-121.9	+18.75	+10.94			
					EV	*0*	*0*	*0*	*0*	*3.75*	*0*	*xEV for /bɑt/:*	*0.76*	*0.68*
			/bɑt/	3	AV	-104.7	+12.5	+7.81	-100	-28.12	+25			
					EV	*0*	*0*	*0*	*0*	*0*	*10*			
		$C_{(v)}V2C$	/bət/	4	AV	-142.2	+10.94	+9.38	+17.19	-57.81	+20.31	xAV for /bə/	-22.79	-54.07
					EV	*0*	*0*	*0*	*2.19*	*0*	*5.31*			
			/bət/	5	AV	-175	+10.94	0	+12.5	-78.13	+17.19			
					EV	*0*	*0*	*0*	*0*	*0*	*2.19*	*xEV for /bə/:*	*1.09*	*1.06*
			/bət/	6	AV	-98.44	+10.94	+12.5	-14.84	+25	+9.38			
					EV	*0*	*0*	*0*	*0*	*10*	*0*			
		$C_{(v)}V3C$	/bɪt/	7	AV	-131.2	+10.94	+18.75	+20.31	+28.12	+18.75	xAV for /bɪ/	-5.72	-40.7
					EV	*0*	*0*	*3.75*	*5.31*	*13.12*	*3.75*			
			/bɪt/	8	AV	-131.2	+15.63	+15.63	-21.88	+17.19	+29.69			
					EV	*0*	*0.63*	*0.63*	*0*	*2.19*	*14.69*	*xEV for /bɪ/:*	*2.87*	*2.32*
			/bɪt/	9	AV	-14.06	+10.94	+15.63	+12.5	+21.88	-40.62			
					EV	*0*	*0*	*0.63*	*0*	*6.88*	*0*			
		$C_{(v)}V4C$	/bɔt/	10	AV	-84.37	+10.94	+14.06	-54.69	+18.75	+21.88	xAV for /bɔ/	-18.22	-27.89
					EV	*0*	*0*	*0*	*0*	*3.75*	*6.88*			
			/bɔt/	11	AV	-148.4	+10.94	+15.63	+17.19	+32.81	+23.44			
					EV	*0*	*0*	*0.63*	*2.19*	*17.81*	*8.44*	*xEV for /bɔ/:*	*2.36*	*1.06*
			/bɔt/	12	AV	-115.6	+10.94	+15.63	+17.19	-81.25	-53.12			
					EV	*0*	*0*	*0.63*	*2.19*	*0*	*0*			
		MEAN VOT FOR ABSOLUTE VALUES (xAV) OF THE 12 $C_{(v)}VC$ PRODUCTIONS				-119.69	+11.46	+12.37	-16.99	-10.68	+1.7	xAV for $C_{(v)}VC$ context:	-20.31	-43.41
		AVERAGE OF VOT ERROR VALUES (xEV) OF THE 12 $C_{(v)}VC$ PRODUCTIONS				*0*	*0.5*	*0.5*	*0.99*	*4.79*	*4.27*	*xEV for $C_{(v)}VC$ context:*	*1.77*	*1.28*

TABLE 4.12: THE ANALYSIS OF VOICE ONSET TIME (VOT) VALUES (in ms) OF THE 12 $C_{(vl)}$VC PRODUCTIONS: A PRESENTATION OF THE ABSOLUTE VOT VALUES, MEAN AND ERROR VALUES OF VOT FOR THE SIX INDIVIDUAL SUBJECTS (S1 – S6). COMPARISONS OF THE MEAN AND ERROR VOT VALUES FOR THE $C_{(vl)}$VC CONTEXT BETWEEN THE SUBJECT GROUP AND THE CONTROL GROUP ARE INCLUDED.

VOICE ONSET TIME (VOT) IN ms OF /p/ PRODUCTIONS														
12 $C_{(vl)}$VC PRODUCTIONS					ABSOLUTE VOT AND VOT ERROR VALUES (IN *ms*) FOR THE SIX SUBJECTS							MEAN VOT VALUES (IN ms) FOR THE FOUR DIFFERENT $C_{(vl)}$VC PRODUCTIONS IN THE SUBJECT GROUP AND THE CONTROL GROUP		
					ABSOLUTE VOT VALUE (AV)	SUBJECTS (N = 6) (STUTTERING SEVERITY IS INDICATED AS: I VERY MILD; II MILD; III MODERATE; IV SEVERE)						xAV	SUBJECT GROUP	CONTROL GROUP
					VOT ERROR VALUE (EV)	S1 III	S2 IV	S3 IV	S4 II	S5 II	S6 I	xEV		
PHONETIC CONTEXT	VOICELESS CONSONANT-VOWEL-CONSONANT ($C_{(vl)}$VC)	$C_{(vl)}$V1C	/pɑt/	1	**AV**	+12.5	+10.94	+7.81	+18.75	+18.75	+10.94	xAV for /pɑt/	+24.74	+15.37
					EV	*0*	*0*	*2.19*	*0*	*0*	*0*			
			/pɑt/	2	**AV**	+125	+10.94	+12.5	+25	+18.75	+15.63			
					EV	*0*	*0*	*0*	*0*	*0*	*0*	*xEV for /pɑt/:*	*0.16*	*0.14*
			/pɑt/	3	**AV**	+73.44	+20.31	+14.06	+18.75	+21.88	+9.38			
					EV	*0*	*0*	*0*	*0*	*0*	*0.62*			
		$C_{(vl)}$V2C	/pət/	4	**AV**	+15.63	+12.5	+10.94	+20.31	+21.88	+10.94	xAV for /pə/	+16.5	+18.82
					EV	*0*	*0*	*0*	*0*	*0*	*0*			
			/pət/	5	**AV**	+18.75	+14.06	+14.06	+17.19	+21.88	+15.63			
					EV	*0*	*0*	*0*	*0*	*0*	*0*	*xEV for /pə/:*	*0.03*	*0.07*
			/pət/	6	**AV**	+21.88	+10.94	+12.5	+25	+23.44	+9.38			
					EV	*0*	*0*	*0*	*0*	*0*	*0.62*			
		$C_{(vl)}$V3C	/pɪt/	7	**AV**	+14.06	+18.75	+9.38	+18.75	+31.25	+10.94	xAV for /pɪ/	+17.02	+18.72
					EV	*0*	*0*	*0.62*	*0*	*0*	*0*			
			/pɪt/	8	**AV**	+20.31	+12.5	+15.63	+18.75	+25	+15.63			
					EV	*0*	*0*	*0*	*0*	*0*	*0*	*xEV for /pɪ/:*	*0.03*	*0.16*
			/pɪt/	9	**AV**	+21.88	+12.5	+15.63	+17.19	+17.19	+10.94			
					EV	*0*	*0*	*0*	*0*	*0*	*0*			
		$C_{(vl)}$V4C	/pɔt/	10	**AV**	+9.38	+9.38	+23.44	+15.63	+9.38	+25	xAV for /pɔ/	+19.53	+21.27
					EV	*0.62*	*0.62*	*0*	*0*	*0.62*	*0*			
			/pɔt/	11	**AV**	+21.88	+9.38	+29.69	+20.31	+25	+17.19			
					EV	*0*	*0.62*	*0*	*0*	*0*	*0*	*xEV for /pɔ/:*	*0.17*	*0.10*
			/pɔt/	12	**AV**	+26.56	+20.31	+15.63	+26.56	+37.5	+9.38			
					EV	*0*	*0*	*0*	*0*	*0*	*0.62*			
		MEAN VOT IN ms FOR ABSOLUTE VALUES (xAV) FOR 12 $C_{(vL)}$VC PRODUCTIONS				+31.77	+13.54	+15.11	+20.18	+22.66	+13.42	*xAV for $C_{(vl)}$VC context:*	+19.45	+18.54
		AVERAGE OF VOT ERROR VALUES (xEV) IN ms FOR 12 $C_{(vl)}$VC PRODUCTIONS				*0.05*	*0.1*	*0.23*	*0*	*0.05*	*0.16*	*xEV for $C_{(vl)}$VC context:*	*0.1*	*0.12*

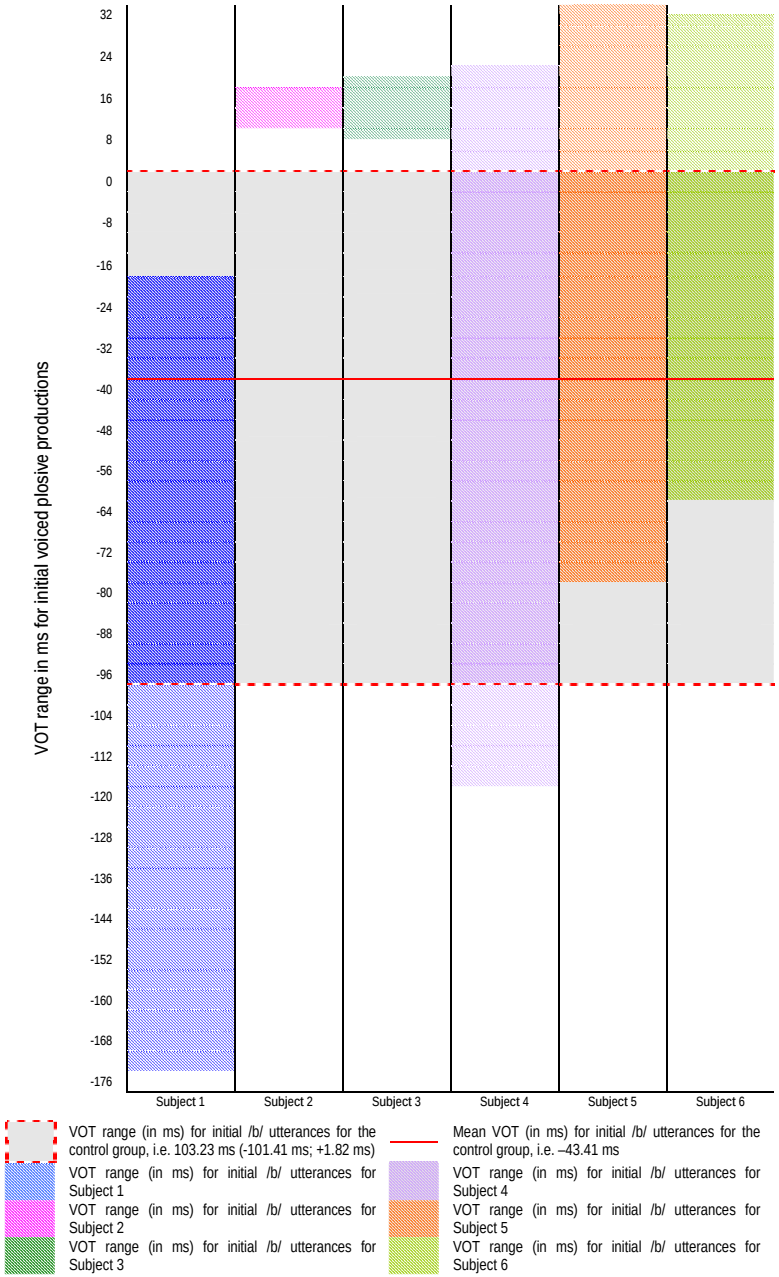

FIGURE 4.8.1: AN ILLUSTRATION OF THE VOT RANGE FOR INITIAL /b/ UTTERANCES IN THE SIX SUBJECTS COMPARED TO THE MEAN VOT AND THE VOT RANGE FOR INITIAL /b/ UTTERANCES IN THE CONTROL GROUP

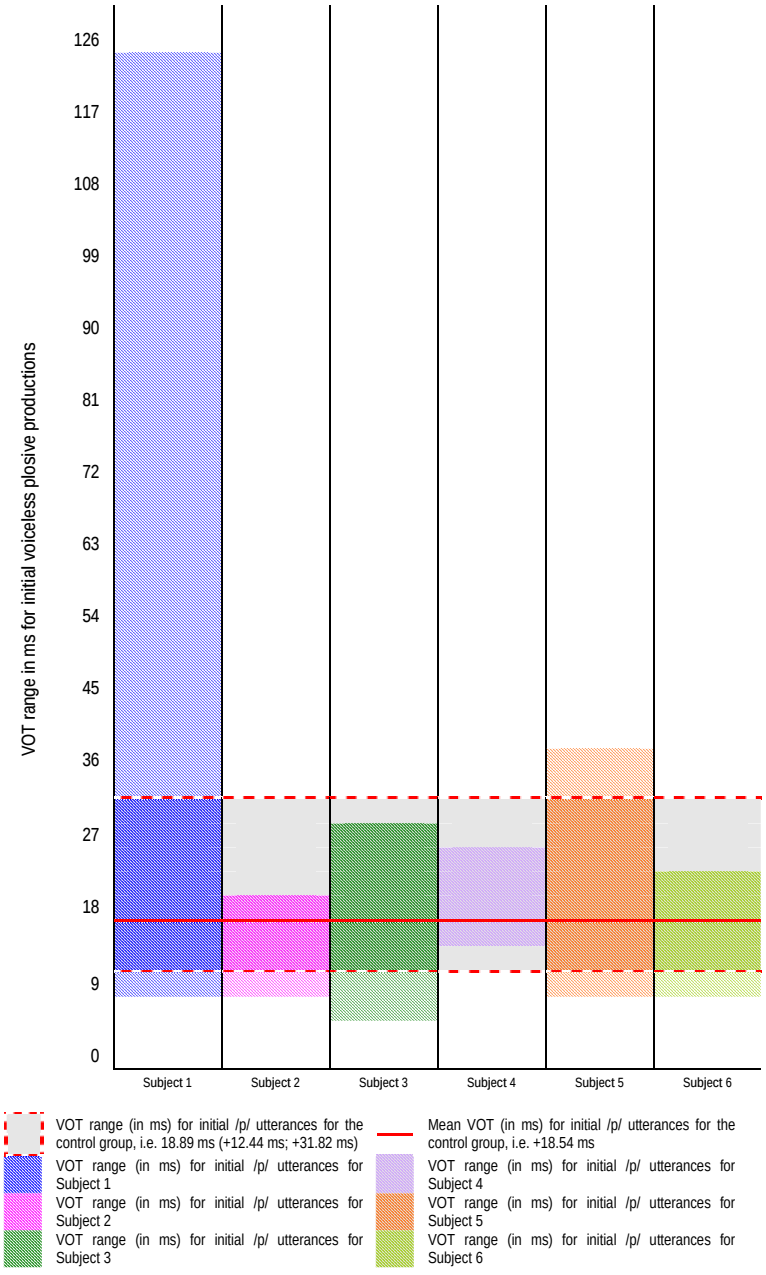

FIGURE 4.8.2: AN ILLUSTRATION OF THE VOT RANGE FOR INITIAL /p/ UTTERANCES IN THE SIX SUBJECTS COMPARED TO THE MEAN VOT AND THE VOT RANGE FOR INITIAL /p/ UTTERANCES IN THE CONTROL GROUP

TABLE 4.13: A SUMMARY OF VOICE ONSET TIME (VOT) VALUES (IN ms) FOR INITIAL VOICED AND VOICELESS PLOSIVE PRODUCTIONS IN THE SIX INDIVIDUAL SUBJECTS. COMPARISONS OF THE MAXIMUM AND MINIMUM VOT, VOT RANGE AND MEAN VOT BETWEEN THE SUBJECT GROUP AND THE CONTROL GROUP ARE INCLUDED (based on Table 4.11 and Table 4.12)

Summary of VOT values for initial voiced and voiceless plosives													
VOT values (in ms)	**VOT (in ms) for initial /b/ in $C_{(v)}$VC context**							**VOT (in ms) for initial /p/ in $C_{(vl)}$VC context**					
	Control Group	**Subject Group (N = 6)**						**Control Group**	**Subject Group (N = 6)**				
		Subject 1	Subject 2	Subject 3	Subject 4	Subject 5	Subject 6		Subject 1	Subject 2	Subject 3	Subject 4	Subject 5	Subject 6
Minimum VOT (in ms)	-101.41	-175	+9.38	+7.81	-121.9	-81.25	-62.5	+12.44	+9.38	+9.38	+7.81	+15.63	+9.38	+9.38
Maximum VOT (in ms)	+1.82	-14.06	+15.63	+18.75	+20.31	+32.81	+29.69	+31.33	+125	+20.31	+29.69	+26.56	+37.5	+25
VOT range (in ms) *(Maximum VOT - Minimum VOT)*	103.23	160.94	6.25	10.94	142.21	114.06	92.19	18.89	115.62	10.93	21.88	10.93	28.12	15.62
Error value for VOT range for subjects *(VOT range Sn – VOT range for NS)*	VOT range for S*n* - 103.23	*(160.94-103.23)* >57.71	*(6.25-103.23)* 0 *(<96.98)*	*(10.94-103.23)* 0 *(<92.29)*	*(142.21-103.23)* >38.98	*(114.06-103.23)* >10.83	*(92.19-103.23)* 0 *(<11.04)*	VOT range for S*n* - 18.89	*(115.62-18.89)* >96.73	*(10.93-18.89)* 0 *(<7.96)*	*(21.88-18.89)* >2.99	*(10.93-18.89)* 0 *(<7.96)*	*(28.12-18.89)* >9.23	*(15.62-18.89)* 0 *(<3.27)*
Mean VOT (in ms) of 12 productions	-43.41	-119.69	+11.46	+12.37	-16.99	-10.68	+1.7	+18.54	+31.77	+13.54	+15.11	+20.18	+22.66	+13.42

** The error value for VOT range for a subject is indicated as 0 when that subject showed a range that was less than the VOT range for the control group for initial /b/ or /p/ productions. If the error value was 0, the magnitude of the difference in VOT range between the subject and control group was indicated as <x.*

4.3.2.1 Voice onset time (VOT) of the initial voiced plosive /b/ in $C_{(v)}VC$ productions

Table 4.11 shows the *mean VOT* and *VOT error values* for the $C_{(v)}VC$ context of the control group and subject group, including the individual subjects. The data in Table 4.11 are used for the description of the VOT results of initial /b/ productions, unless otherwise indicated.

In normal production of the prevocalic /b/, the normal limits for voice onset time (VOT) range between -180 ms and +15 ms (Zlatin, 1974). The negative VOT indicates that vocal fold vibration was started before the release of the lip closure, as opposed to the positive value where the articulatory release precedes vocal fold vibration. When VOT for a /b/ production exceeds the dynamic constraints of +15 ms (Zlatin, 1974), the production may be acoustically perceived as a voiceless /p/. The reason for this possibility is that the lower limit of VOT for the voiceless /p/, i.e. +10 ms (Lisker & Abramson, 1964 in Borden & Harris, 1984) overlaps with the VOT range for /b/ productions. If the occurrence of voice is at the same time as the articulatory release or before the release (thus, for VOT values of 0 ms and below) the voiced production has an unaspirated quality (Shibles, 1995). In Afrikaans, voiced plosives are normally produced without aspiration (De Villiers & Ponelis, 1987; Wissing & Roux, 1995). According to Cho and Ladefoged (1999), VOT values of +30 ms and below are representative of unaspirated plosives.

Between-group comparison: For /b/ productions, the mean VOT for the control group was -43.02 ms and -20.31 ms for the subject group. Both the subject group and control group had negative mean VOT values within the range of normal VOT for prevocalic /b/ productions, i.e. -180 ms to +15 ms (Zlatin, 1974). No apparent difference in mean VOT between the two groups was found. Based on the mean VOT, no delay in the onset of voicing for /b/ productions was evident for the subject group. The control group had an average VOT range that varied between a minimum VOT value of -101.41 ms and a maximum VOT value of +1.82 ms (Table 4.13). The control group showed a tendency toward negative voice onsets for /b/ productions (see Appendix H(*i*)). This finding is consistent with research by Wissing and Roux (1995) regarding negative VOT for voiced plosives in Afrikaans.

Both the control and subject group showed greater negative VOT values for /bɑt/ and /bət/ utterances, compared to VOT values for /bɪt/ and /bɔt/. For instance, the subject group exhibited

VOT values of -34.49 ms and -22.79 ms for /bɑt/ and /bət/, respectively, compared to -5.72 ms and -18.22 ms for /bɪt/ and /bɔt/. This phenomenon was more prominent for the control group. The subject group had higher VOT error values for /bɪt/ and /bɔt/ utterances (i.e. 2.87 ms and 2.36 ms) as opposed to error values for /bɑt/ and /bət/ utterances (i.e. 0.76 ms and 1.09 ms). The greater error values for /bɪt/ and /bɔt/ utterances suggest that the subject group experienced difficulty with interarticulatory synchronization during voice onset for these utterances. Differences in VOT between the four prevocalic /b/ productions (/bɑt/, /bət/, /bɪt/ and /bɔt/) may relate to the phonetic properties of the adjacent vowel that influenced VOT for /b/ productions as seen in both groups.

VOT error values showed a small difference between the subject group (1.77 ms) and the control group (1.28 ms). Thus, both groups seemed to be able to maintain relative consistency in the timing of voice onset for the prevocalic /b/ productions. These results reflect that the subjects, in general, were able to temporally coordinate vocal fold adjustments and articulatory movements for voice onset for voiced /b/ productions.

The similar results of the subject group and control group for VOT of /b/ productions should not overlook the possibility that the average VOT values for /b/ productions obscured the VOT results for the individual subjects. The average VOT values for the subject group may have masked indications of abnormalities in the absolute VOT of the individual subjects. Comparisons of the values for VOT range and mean VOT for the individual subjects are therefore necessary.

Subject group (within-group comparison): The six subjects (S1 to S6) showed mean VOT values for initial /b/ utterances within the range of -180 ms and +15 ms, which reflected normal VOT for voiced plosive productions (Zlatin, 1974). Three of the six subjects obtained negative mean VOT values (i.e. S1 = -119.69 ms; S4 = -16.99 ms; S5 = -10.68 ms). For Subject 1, vocal fold vibration was initiated prior to articulatory release of lip closure for the twelve /b/ productions. Whereas Subject 1 showed a tendency to prevoicing of /b/ productions, Subjects 4 and 5 showed infrequent incidents of negative VOT. The VOT range values for /b/ productions for Subjects 1, 4 and 5 were greater than the control group's average VOT range of 103.23 ms (i.e. 160.94 ms; 142.21 ms; 114.06 ms, respectively) (Table 4.13). These results for the three

subjects (S1, S4 and S5) suggest that their /b/ productions were characterized by moments of inconsistencies in the timing of intergestural laryngeal-phonatory and articulatory movements during voice onset.

Subjects 2, 3 and 6 showed positive mean VOT values for prevocalic /b/ productions (i.e. +11.46 ms, +12.37 ms and +1.7 ms, respectively) (Table 4.13). The VOT results for these three subjects indicated that the onset of voicing follows after the articulatory release. The VOT range values for /b/ productions in Subjects 2, 3 and 6 were markedly smaller, compared to the value of 103.23 ms for the control group (6.25 ms, 10.94 ms and 92.19 ms, respectively) (Table 4.13). Error values for average VOT range of 0 ms were obtained for these subjects, which may suggest consistency or stability in speech motor activities during voice onset of /b/ productions. Normal speech activity during VOT for /b/ productions is not necessarily implied hereby. Limited variability in the timing of laryngeal-articulatory speech activities for Subjects 2, 3 and 6 can be associated with a restricted range of coarticulatory movements.

As illustrated by Figure 4.8.1, the maximum VOT value for five of the six subjects (S2 - S6) exceeded the upper limit for normal VOT of voiced /b/ productions, i.e. +15 ms (Zlatin, 1974). Thus, the majority of the subjects had a tendency toward greater VOT values than +15 ms which indicate a delay or slowness in VOT for /b/ productions. This finding suggests that irregularity or slowness in voice onset timing for prevocalic /b/ productions was evident in all subjects except Subject 1. For these five subjects at least 50% of the 12 $C_{(v)}VC$ utterances was depicted by VOT values greater than +10 ms (i.e. S2 = 11 utterances; S3 = 9 utterances; S4 = 7 utterances; S5 = 6 utterances; S6 = 8 utterances). If +10 ms is considered to be the minimum limit for VOT of voiceless /p/ productions (Lisker & Abramson, 1964 in Cho & Ladefoged, 1999), then these voiced /b/ productions for Subjects 2, 3, 4, 5 and 6 could have been produced as voiceless /p/ utterances. On the other hand, the subjects could have substituted the voiced plosive /b/ with a voiced fricative /β/ by a narrowed or approximated articulatory closure.

Table 4.13 indicates that Subjects 2 and 3 had markedly small VOT range values for /b/ productions (i.e. 6.25 ms and 10.94 ms, receptively). For instance, the absolute VOT values for Subject 2 ranged between +9.38 ms and +15.63 ms, with identical VOT values (+10.94 ms) for eight of the twelve /b/ utterances. Subjects 2 and 3 also showed similar mean VOT values (S2 = +11.46 ms; S3 = +12.37 ms) and small error values (S2 = 0.5 ms; S3 = 0.5 ms) (Table 4.13).

Thus, Subjects 2 and 3 were inclined to longer VOT for /b/ productions and they showed consistent timing for voice onset across the 12 utterances. The VOT results for /b/ productions in these two subjects reflect marked consistency (or constrain) in the timing of articulatory release for /b/ and the onset of vocal fold vibration.

The subjects (S5 and S6) with the highest maximum VOT values (i.e. +32.81 ms; +29.69 ms) for /b/ productions also showed the greatest error values for VOT (i.e. 4.79 ms; 4.27 ms) (Table 4.13). These subjects had absolute values greater than +15 ms for most /b/ utterances (seven of the twelve /b/ productions for each subject). This apparent delay in voice onset is possibly associated with a mistiming in the coordination between articulatory and vocal fold movements. A delay in VOT for /b/ utterances may have been acoustically perceivable as breathy or aspirated /b/ productions or as voiceless /p/ productions. Slightly aspirated voiced plosive production occurs when vocal fold vibration begins after the release of the articulatory occlusion (Shibles, 1995). Subject 5 was a bilingual (English-Afrikaans) speaker, which partly explains the occurrence of voiced aspiration, since plosive production in English is characterized by aspirated production (Cho & Ladefoged, 1999). Aspiration may also be used as a compensatory gesture to avoid a breakdown in speech initiation.

No clear indication of a relation between stuttering severity and VOT for /b/ productions was seen in the results. Some evidence was seen in the subjects with severe stuttering (S2 and S3) who showed a tendency toward positive VOT and restricted VOT range values, as opposed to the subjects with mild stuttering (S4 and S5) with negative mean VOT values and greater VOT ranges, but further in-depth investigation is needed.

4.3.2.2 Summary of results on VOT of the initial voiced plosive /b/ in $C_{(v)}VC$ productions

The VOT results for prevocalic voiced /b/ productions in the subject group reflect the following: (a) A comparison of the mean VOT values indicates no marked difference between the control group and the subject group; (b) The subject group showed no apparent delay in mean VOT for voiced /b/ productions. However, the individual subjects (excluding Subject 1) indicated slowness in VOT for /b/ productions compared to the control group; (c) Consistency in VOT for the subject group was indicated by the small error value; (d) Individual differences in VOT

results for prevocalic /b/ productions were evident in two subjects (S1 and S6) when compared to the results of the control group; (e) Subjects with the same level of stuttering severity revealed similar trends in VOT results for prevocalic /b/ productions. For instance, speakers with severe stuttering, i.e. Subjects 2 and 3, had similar values for mean VOT and VOT range. This phenomenon in VOT was also seen in the two mild stuttering speakers. The moderate to severe stuttering speakers (S1, S2 and S3) generally maintained consistent VOT values across the voiced /b/ productions, whereas results for the very mild to mild stuttering speakers (S4, S5 and S6) indicated highly inconsistent VOT values (scattered between negative and positive values). These indications that stuttering severity may be associated with VOT results warrant further investigation.

4.3.2.3 Voice onset time (VOT) of the initial voiceless plosive /p/ in $C_{(vl)}VC$ productions

Table 4.12 shows the mean VOT and VOT error values for the voiceless /p/ productions in the $C_{(vl)}VC$ context of the control group and subject group, including the individual subjects. The data in Table 4.12 are used for the description of the VOT results of initial /p/ productions, unless stated differently.

For perception of voiceless initial stops in English the difference in timing between the opening of the articulatory occlusion and the start of voicing ranges between +35 ms and +150 ms in normal speech production (Blumstein *et al.,* 1980). Lisker and Abramson (1964 in Cho & Ladefoged, 1999) determined the minimum VOT limit for perceiving unaspirated voiceless bilabial plosives in Dutch as *+10 ms.* Afrikaans and Dutch show similarities in phonetic features (specifically pertaining to laryngeal dimensions for plosive productions) (Wissing & Roux, 1995; Iverson & Salmons, 2003). Since limited normative data exists for VOT in Afrikaans, the VOT limits of voiceless plosives for Dutch were used for voiceless plosives in Afrikaans. For the purpose of this study, *values between +10 ms and +150 ms were regarded as normal VOT for prevocalic /p/ productions.* When a plosive is produced *without* aspiration as in Afrikaans, it implies a faster articulatory release and results in a shorter voice onset time in contrast to the slower release of aspirated /p/ productions. Furthermore, a prevocalic voiceless plosive /p/ may be influenced by the voicing property of the adjacent vowel due to the effect of coarticulation. In such a case VOT values for the productions of voiced /b/ and voiceless /p/ can overlap and the

voiceless plosive can be characterized by voiced production. Thus, a prevocalic /p/ production with a VOT value of less than +15 ms could be perceived as a voiced /b/.

Between-group comparison: The *mean VOT* for the subject group of +19.45 ms was in close relation to that of the control group, i.e. +18.54 ms. Both, the controls and subjects had *mean VOT* values *between* +10 ms and +150 ms, which is the normal VOT range for unaspirated voiceless plosive productions (Lisker & Abramson, 1964 in Cho & Ladefoged, 1999). The subject group obtained a small VOT error value of 0.1 ms for /p/ productions, which was comparable to the 0.12 ms of the control group. Based on VOT results, no obvious differences were found between the control group and subject group. This finding implies that the subject group displayed no apparent deviancy in the timing voice onset for voiceless plosive productions.

The only aspect on which these two groups did differ, was for mean VOT for /p*a*t/ productions. For these prevocalic /p/ utterances, the subject group obtained an average VOT value of +24.74ms compared to the +15.37 ms of the control group. Thus, the controls showed a shorter lag in VOT for /p*a*t/ productions. The voicing effect of the vowel /*a*/ following the initial voiceless plosive in /p*a*t/ could have influenced the voice onset time (and voiceless quality) of the prevocalic /p/ and therefore resulted in the small mean VOT value as seen for the control group.

Subjects (within-group comparison): Analysis of the VOT data indicates that the subjects showed mean values for prevocalic /p/ productions that ranged between +13.42 ms and +31.77 ms. All subjects (S1 - S6) obtained mean VOT values above +10 ms which is regarded as the minimum limit for voiceless /p/ productions (Lisker & Abramson, 1964 in Cho & Ladefoged, 1999). According to the mean VOT values for voiceless plosives, the six subjects performed within the range of normal VOT.

Subjects 2, 3 and 6 obtained similar mean VOT values for /p/ productions, i.e. +13.54 ms, +15.11 ms and +13.42 ms, respectively. The small mean VOT for these three subjects are attributed to the fact that above 50% of the 12 /p/ productions had VOT values of below +15 ms (S2 = 9 utterances; S3 = 7 utterances; S6 = 7 utterances). Since +15 ms is the maximum limit for

voiced plosive productions (Zlatin, 1974), these prevocalic /p/ productions for Subjects 2, 3 and 6 seem to show VOT features of voiced /b/ productions. Thus, the shorter lag in VOT for /p/ productions might indicate that most /p/ productions of 50% of the subjects adopted the acoustic properties of voiced /b/ utterances. No definite comparison was seen between severe stuttering and abnormalities in VOT for /p/ productions as Subject 2 and Subject 3 were rated with severe stuttering, whereas Subject 6 was considered to be a very mild stuttering speaker.

Subjects 1, 4 and 5 obtained mean VOT values above +15 ms, which ranged between +20.18 ms and +31.77 ms. For these three subjects a small number of /p/ productions had VOT values below +15 ms (S1 = 4 utterances; S4 = 1 utterances; S5 = 1 utterance). The influence of the neighbouring vowel on the VOT of the initial /p/ also needs to be considered in the interpretation of these results. Important to note is that the great *mean VOT* for /p/ productions in Subject 1 is due to two /p/ utterances that had markedly longer voice onset times, i.e. +125 ms (/pat/$_2$) and +73.44 ms (/pat/$_3$) (Table 4.12). These two utterances could have been produced with an aspirated or breathy quality. Ten of the twelve /p/ utterances had VOT values varying between +9.38 ms and +26.56 ms (Table 4.12). Subject 1 seemed to have maintained consistency in VOT for most /p/ productions. Thus, the great *VOT range* of 115.62 ms for Subject 1 is attributed to two instances of delayed VOT (i.e. /pat/$_2$, /pat/$_3$). Despite the fact that Subjects 1, 4 and 5 obtained similar mean VOT values, they differed in stuttering severity. Subjects 4 and 5 were rated with mild stuttering, whereas Subject 1 was considered to be a severe stuttering speaker. The results for Subjects 1, 4 and 5, as for Subjects 2, 3 and 6, did not show an apparent relation between stuttering severity and VOT for /p/ productions.

Five subjects (S1, S2, S3, S5 and S6) obtained minimum VOT values for /p/ productions that range between +7.81 ms and +9.38 ms; thus, less than +10 ms (Table 4.13). This means that the lower limit of VOT range for /p/ utterances for these five subjects overlapped with that of the /b/ productions. These subjects also showed minimum VOT values for /p/ productions below the minimum value of the control group (+12.44 ms) (Table 4.13). Two of these subjects (S1 and S5) obtained maximum VOT values above the +31.33 ms of the control group (i.e. +125 ms and +37.5 ms, respectively) (Table 4.13). Since Subjects 1 and 5 had higher maximum VOT values and lower minimum values for /p/ productions compared to the control group, it suggests that these subjects experienced great variability in VOT for /p/ utterances. The magnitude of the

VOT range (*maximum VOT – minimum VOT*) for /p/ productions was the greatest for Subject 1, i.e. 115.62 ms (Table 4.13). Subject 5 had a smaller range value of 28.12 ms as seen in Table 4.13. The error value for VOT range for /p/ productions was 96.73 ms for Subject 1 and 9.23 ms for Subject 5 (Table 4.13). This shows that Subjects 1 and 5 displayed more variance in VOT range compared to the control group and the other subjects.

4.3.2.4 Summary of results on VOT of the initial voiceless plosive /p/ in $C_{(vl)}VC$ productions

The VOT for prevocalic /p/ utterances in the subject group indicate the following: (a) The mean VOT values for prevocalic /p/ utterances in the subject group and the control group were both within the normal range of +10 ms to +150 ms; (b) No delay in VOT for prevocalic /p/ productions was evident for the subject group. All subjects (excluding S1) obtained mean VOT values for /p/ productions that are comparable to that of the control group; (c) There was a voicing effect induced by the vowel following the initial voiceless plosive; (d) VOT range for most subjects (except S1 and S5) were in close relation to the range of the control group; (e) No apparent relation was found between stuttering severity and VOT for /p/ productions.

4.3.2.5 Effect of initial voiced and voiceless plosives on VOT in normal speakers and persons who stutter (PWS)

Figures 4.8.1 and 4.8.2 illustrate the effect of prevocalic voiced/voiceless plosive productions on VOT for the subjects. These figures show the magnitude of difference between minimum and maximum VOT values for /b/ and /p/ productions for the six subjects (S1 - S6) in comparison to that of the control group. The results summarized in Table 4.13 were used to indicate differences in VOT between /b/ and /p/ productions in the subjects as well as the control group.

Control group: The control group showed a clear distinction in voiced-voiceless plosive productions with relation to VOT range values. The limits for VOT range for voiced /b/ varied from -101.41 ms to +1.82 ms and for voiceless /p/ productions, the limits varied from +12.44 ms to +31.33 ms (Table 4.13). In other words, the maximum VOT value for /b/ (+1.82 ms) did not overlap with the minimum VOT value for /p/ (+12.44 ms) in this group. A tendency toward

negative VOT (or prevoicing) for /b/ productions with a mean VOT of -43.41 ms was seen for the controls (Figure 4.8.1).

The control group also had a greater VOT range for /b/ productions (103.23 ms) as opposed to /p/ productions (18.89 ms) (Table 4.13). Thus, the /p/ productions showed a higher incidence of repeatability in VOT compared to /b/ productions. For the control group, the smaller VOT range for /p/ productions may relate to consistency in timing of articulatory and laryngeal activities for voiceless-voiced transitions.

Subject group: In this study, the VOT range values for /b/ and /p/ productions of the subjects were compared to the norm set by the control group. The control group had a definite trend toward prevoicing of /b/ productions, but only three of the six subjects showed negative mean VOT values. Based on the VOT range for /b/ productions most subjects (S2, S3, S4, S5 and S6) had maximum VOT values greater than the control group (Figure 4.8.1). The VOT maximum values for /b/ productions for the subjects (except S1) overlapped with their minimum values for /p/ productions. Minimum VOT values for /p/ productions for most subjects (S1, S2 S3, S5 and S6) were smaller compared to the value of the control group (Figure 4.8.2). These results reflect that the subjects showed inconsistencies in VOT for utterances requiring voiced-voiced transitions in prevocalic /b/ productions as well as voiceless-voiced transitions in prevocalic /p/ productions. The VOT limits for /b/ and /p/ in these subjects overlapped which imply indistinctive voiced-voiceless differentiation in stuttering speakers. This finding may relate to abnormalities in the timing between voice onset and articulatory release for voiced and voiceless plosive productions in stuttering speakers.

Furthermore, two of the six subjects (S2 and S3) had minimum VOT values for /p/ productions identical to the minimum values of their /b/ productions (S2 = +9.38 ms; S3 = +7.81 ms) (Table 4.13). For both these subjects the mean VOT for voiced /b/ productions were similar to that of the voiceless /p/ productions. For instance, the mean VOT for /b/ productions for Subject 2 was +11.49 ms, which is close to the VOT value of +13.54 ms for /p/ productions; for Subject 3 a mean VOT of +12.37 ms for /b/ utterances and +15.11 ms for /p/ productions were obtained (Table 4.13). While Subjects 2 and 3 did not show an overlap in VOT range values for /b/ productions with that of the control group (Figure 4.8.1), their VOT range values for /p/ productions overlapped with that of the control group (Table 4.13). This finding may suggest

that Subjects 2 and 3 had difficulty in the temporal differentiation between voiced-voiced versus voiceless-voiced transitions as indicated by the VOT range. Thus, managing voiced-voiceless contrast during perceptually fluent speech production could have been a problem for these two subjects.

Notably small VOT range values were seen in two of the six subjects for /b/ productions (S2 = 6.25 ms; S3 = 10.94 ms) and for /p/ productions (S2 = 10.93 ms; S4 = 10.93 ms) (Table 4.13). Limited variance between minimum and maximum VOT values for these subjects is implied by this data. The small range values suggest that the absolute VOT values for /b/ and /p/ productions for these specific individuals were highly consistent.

As in the case of the control group, three of the six subjects had greater VOT range values for /b/ productions than /p/ productions (S4 = /b/, 142.21 ms > /p/, 10.93 ms; S5 = /b/, 114.06 ms > /p/, 28.12 ms; S6 =/b/, 92.19 ms > /p/, 15.62 ms) (Table 4.13). Only two subjects showed greater VOT range values for /p/ productions as opposed to /b/ productions (S2 = /b/, 6.25 ms < /p/, 10.93 ms; S3 = /b/, 10.94 ms < /p/, 21.88 ms) (Table 4.13). A single subject (S1) maintained great differences between maximum and minimum VOT values for /b/ and /p/ productions, with a higher range value for /b/ productions (S1 = /b/, 160.94 ms > /p/, 115.62 ms). This subject possibly had instances of difficulty in controlling VOT for /b/ and /p/ plosive utterances. Interesting to note is that Subjects 4, 5 and 6 were rated with very mild to mild stuttering and Subjects 2 and 3, as moderate to severe stuttering speakers. Indications that inconsistencies in VOT range for voiced/voiceless plosive productions in the subjects might be associated with stuttering severity are present.

Despite similarities in VOT values among subjects, it seems as if the complexity of coordination between vocal fold activity and articulatory movements for /b/ and /p/ utterances had different effects on the VOT range for each subject (Figures 4.8.1 and 4.8.2). In other words, the magnitude of VOT range for /b/ and /p/ productions differed for the individual subjects. For instance, Subject 1 obtained great VOT range values for both /b/ and /p/ productions, while Subject 2 had very restricted VOT range values for both /b/ and /p/ productions as shown in Figures 4.8.1 and 4.8.2. Further investigation is needed to indicate if the magnitude of difference in VOT range for a specific production can be related to a failure in speech motor control in persons who stutter.

4.3.2.6 Summary of results on voice onset time (VOT)

The results for the subjects regarding the voice onset time differed greatly from that of laryngeal onset time for initial /b/ and /p/ productions. The temporal aspects for prevocalic voiced /b/ and voiceless /p/ utterances in the subjects showed less variability in voice onset than the period prior to voice onset (L_xOT). The subject group functioned within normal limits for voice onset for initial /b/ productions. For initial /p/ productions, the subject group was inclined to a shorter lag in VOT, but still evidenced a mean VOT for voiceless stops within the limit for voiced/voiceless differentiation. However, the *mean* VOT results for the subject group obscured the VOT results for the *individual* subjects. The VOT results for the majority of the individual subjects indicated difficulty with the timing of voice onset for initial /b/ and /p/ productions. These subjects' VOT for voiced plosive productions overlapped with the lower VOT limit for voiceless plosive productions (+10 ms) and *vice versa*. These results indicated that prevocalic voiceless /p/ plosives were voiced and voiced /b/ plosives, devoiced (or misvoiced). As differences in VOT were observed between the mild and the moderate stuttering speakers, a possible relation between level of stuttering severity and deviancies in VOT is suggested. Individual differences were noted in VOT for plosive productions for some subjects (i.e. S1 and S6) which did not consistently relate to stuttering severity. From the VOT results it can be concluded that irregularities in the timing of voice onset for /b/ and /p/ productions were evident in the subjects. These problems in VOT may be associated with deviancies in temporal coordination of prephonatory behaviour as reflected by laryngeal onset time (L_xOT).

4.3.3 Summary of results regarding acoustically detected vocal fold activity during speech initiation

In general, the qualitative nature of the voice onset (abrupt/gradual) for the subject group was not different from that of the group of normal speakers for VC, $C_{(v)}$VC and $C_{(vl)}$VC productions. PWS evidenced more abruptness during actual voicing in the VC context, than the $C_{(v)}$VC or the $C_{(vl)}$VC context. Inconclusive results were also found for VOT of voiced and voiceless plosive productions for the subject group versus control group. Results on acoustically detected vocal fold activity during speech onset did not show an explicit difference between the group of stuttering speakers and the group of normal speakers. For the individual subjects, slowness or

inconsistency in VOT for /b/ and /p/ were found. VOT range for initial voiced /b/ and voiceless /p/ plosives and VOT limits for voiced-voiceless distinction showed distinct differences in individual subjects compared to the normal speakers.

4.4 CONCLUSIVE REMARKS ON THE RESULTS REGARDING PHYSIOLOGICAL AND ACOUSTICALLY DETECTED VOCAL FOLD ACTIVITY DURING SPEECH INITIATION

The results of this study showed that the nature of prephonatory vocal fold activity across the VC, $C_{(v)}$VC and $C_{(vl)}$VC contexts for the subject group was predominantly depicted by atypical behaviour. A comparison between the laryngeal onset time (L_xOT) results of the subjects and control speakers showed marked differences across the phonetic contexts. The slowness and variability in L_xOT confirmed the abnormal prephonatory vocal fold activity seen in the subject group. Whereas significant differences were found between the two groups' average L_xOT for the $C_{(v)}$VC and $C_{(vl)}$VC contexts, the VOT data for these phonetic contexts revealed that both groups functioned within normal limits for VOT. However, differences pertaining to VOT range for prevocalic /b/ and /p/ productions were seen for the individual subjects. Disruptions in speech behaviour were more pronounced for L_xOT than for VOT in the subjects. This finding suggests the period prior to actual voice onset to be most affected by stuttering. The nature of voice onset in VC, $C_{(v)}$VC or $C_{(vl)}$VC contexts did not reveal differences between the control group and subject group. The difference in nature of prephonatory vocal fold activity versus the nature of voice onset in stuttering speakers confirms the notion that the period in which preparatory movements for speech initiation occur is distinctively different to that of normal speakers.

The results also highlighted certain aspects that need consideration in relation to deviant speech motor behaviour during voice initiation, such as the duration of preparatory movements, the frequency of occurrence of atypical/deviant speech behaviour, and the variability of the speech behaviour studied, as well as the motor complexity required for the utterance type.

4.5 SUMMARY OF CHAPTER 4

The results of this study are presented in Chapter 4 according to the formulated sub-aims in Chapter 3. These results entail a description of vocal fold behaviour during speech initiation in different phonetic contexts (VC, $C_{(v)}$VC and $C_{(vl)}$VC) in persons who stutter (PWS) as well as in normally fluent speakers. Chapter 4 serves as an introduction to Chapter 5 where the results are interpreted and discussed with reference to relevant research on vocal fold dynamics and speech motor control in stuttering. Interpretations of the results supported by/in association with previous research on stuttering will be based on theoretical explanations for normal and atypical speech production.

CHAPTER 5: DISCUSSION

5.1 INTRODUCTION

Chapter 5 includes an interpretation and discussion of the results with reference to related research. Furthermore, the interpretation of these results will be based on a theoretical framework for the characterization of pathological speech sensorimotor control (Van der Merwe, 1997). The chapter features physiological and acoustically detectable vocal fold activity during speech initiation of the persons who stutter (PWS). The discussion will highlight tendencies in vocal fold activity during speech initiation in PWS with possible theoretical explanations thereof. Although the results only reflect on perceptually fluent speech production, the existence of subtle motor aberrancies has not been excluded. Rather, the study aims to emphasize the presence of subtle discoordinations in the speech of PWS not perceptible by listeners.

5.2 *DISCUSSION OF SUB-AIM 1:* PHYSIOLOGICAL VOCAL FOLD ACTIVITY DURING SPEECH INITIATION

Normal speech production is mainly dependent on the coordination of respiratory, phonatory and articulatory activities, as well as the harmonic integration (maintenance) of subglottal pressure, glottal resistance and supraglottal pressure changes (Adams, 1974). A disturbance in this coordination *might* manifest as an acoustically detectable disfluency depending on the nature, intensity, duration and timing of the breakdown. Findings of abnormal coordination of laryngeal muscle activity during perceptually fluent speech (Freeman & Ushijima, 1978; Shapiro, 1980) support the current viewpoint of physiological stuttering behaviour. A general agreement exists that PWS's perceptually fluent speech cannot be described as "normal" (Love & Jeffress, 1971; Adams & Runyan, 1981; Zimmermann *et al.*, 1981; Klich & May, 1982; Metz *et al.*, 1983). Based on the view that stuttering has a physiological basis, no distinction will be made between perceptually fluent and disfluent productions for the PWS in the current study.

5.2.1 *Discussion of sub-aim 1.1*: The nature of prephonatory vocal fold activity during speech initiation

The discussion of the nature of prephonatory vocal fold activity during speech initiation includes the description of the nature of prephonatory vocal fold activity in normal speakers and persons who stutter (PWS) (5.2.1.1); and prephonatory vocal fold activity in stuttering within the framework of speech sensorimotor control (5.2.1.2).

5.2.1.1 Description of the nature of prephonatory vocal fold activity in normal speakers and persons who stutter (PWS)

The description of the nature of prephonatory vocal fold activity in normal speakers and persons who stutter includes a discussion of the frequency of occurrence of different types of prephonatory vocal fold activity in normal speakers and persons who stutter (5.2.1.1.1); and the effect of sound initiation in different phonetic contexts on prephonatory vocal fold activity in normal speakers and persons who stutter (5.2.1.1.2).

5.2.1.1.1 Frequency of occurrence of different types of prephonatory vocal fold activity in normal speakers and persons who stutter (PWS)

From the five different types of prephonatory vocal fold activity that were identified, the control group showed a tendency toward Type 2 (see Table 4.3). Thus, the group of normal speakers showed consistency in the occurrence of vocal fold approximation for VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances, which is regarded as normal speech behaviour. *Additional prephonatory vocal fold activity* (Type 3) was seen in a small number of utterances as illustrated in Figure 4.2. Additional laryngeal behaviour during speech initiation was therefore a rare occurrence in the normal speakers. The results for prephonatory vocal fold activity of the control group suggest that the normal speakers presented with temporally coordinated vocal fold behaviour prior to speech initiation.

In the subject group, prephonatory vocal fold activity across the phonetic contexts was mostly characterized by Type 4. Thus, a large proportion of the L_x waveforms from the group of PWS

displayed irregular (or aperiodic) vocal fold activity. Abnormal vocal fold activity in PWS has been reported by Shapiro (1980), Freeman and Ushijima (1978), Van Riper (1982), Peters and Boves (1988), and Peters *et al*. (1989).

- **Type 1: Absence of prephonatory vocal fold approximation during speech initiation**

Control group: For the group of normally fluent speakers, prephonatory vocal fold activity was *present* for most utterances across the three phonetic contexts (VC, $C_{(v)}$VC and $C_{(vl)}$VC). A small frequency of occurrence of Type 1 was obtained for the voiceless utterances. The control group consistently showed vocal fold activity prior to speech initiation for VC utterances – hence the mean score of 0 for Type 1 (see Table 4.3).

Type 1 implies that phonation was preceded by an open glottis condition, which means an absence of vocal fold adduction during the prephonatory speech phase. This vocal fold behaviour could be regarded as normal in utterances beginning with a voiceless consonant (Peters & Boves, 1988). For VC and $C_{(v)}$VC utterances, phonation is initiated *at the moment* when the subglottal pressure has reached its maximum level. Only for $C_{(vl)}$VC productions, phonation can start *directly after the moment* where the subglottal pressure has reached its maximum level (Peters & Boves, 1988). Such productions are associated with aspiration of the consonant which is characteristic of English. For normal speech initiation, voiceless consonants involve articulatory movements without vocal fold vibration. Thus, vocal fold adduction is not a prerequisite for the production of initial /p/ utterances.

Type 1 might reflect the *respiratory breath holding strategy*. This prephonatory strategy is thought to involve the positioning of the vocal folds in an abducted posture and fixation of the chest wall before phonatory onset (Cavallo & Baken, 1985). From a phonetic viewpoint, any laryngeal adjustment can be described as abducted if it does not facilitate the generation of acoustic energy at normal volume velocities of the breath stream (Rothenberg, 1968). However, this explanation does not exclude the possibility that the vocal folds could have been fixated in an abducted posture.

Subject group: The absence of prephonatory vocal fold approximation for the subjects was seen in only a small number of utterances. The low frequency of occurrence of Type 1 for the subject group and the group of fluent speakers shows that this type of vocal fold activity during speech initiation is uncommon. The subjects' results might indicate that the vocal folds were fixated in an abducted position during the initiation of the utterances (Conture *et al.*, 1977). The absence of prephonatory laryngeal approximation in the subjects might be associated with a moment of subacoustic disfluency. Another possibility is that the subjects used a compensatory strategy to avoid stuttering. In this regard Peters and Boves (1988) suggested that the vocal folds might be approximated very gently during a deliberate and extremely gentle voice onset. As a result of this gentle vocal fold approximation, the L_x waveform may not indicate a laryngeal approximation. This condition can be associated with a very slow subglottal pressure build-up (Peters & Boves, 1988). Phonation starts long before subglottal pressure reached its maximum level (Peters & Boves, 1988), and consequently the intersystem synchronization between the phonatory system and respiratory system is simplified.

One subject (S5) showed apparent similarities with the normal speakers' pre-speech activities. This subject showed mild stuttering involvement and was the only bilingual (English-Afrikaans) speaker in the subject group. Two factors need to be considered in the interpretation of the nature of prephonatory vocal fold activity of this subject: (a) the characteristics of English plosives concerning the manner of production, namely breathy or aspirated, and (b) the effect of stuttering severity on the nature of prephonatory vocal fold activity.

- **Type 2: Prephonatory vocal fold approximation without additional vocal fold activity during speech initiation**

Control group: Prephonatory vocal fold approximation without additional vocal fold activity during speech initiation (Type 2) is considered to be representative of normal vocal fold activity for speech production (Peters & Boves, 1988). The control group showed a tendency toward Type 2 (see Table 4.3). Thus, the prephonatory period for the controls mostly reflected normal vocal fold behaviour across the three phonetic contexts. In the case of Type 2, phonation is initiated with glottal closure or with approximated vocal folds for VC and $C_{(v)}$VC productions in preparation to voicing. In the $C_{(vl)}$VC context, the vocal folds might be approximated to prepare for the phonetic properties of the vowel following the initial voiceless consonant.

In preparation for the aerodynamic events of phonation – the chestwall is displaced ± 100 ms before phonatory onset to generate alveolar pressure (Baken, Cavallo & Weissman, 1979). During this prephonatory period, subglottal air pressure is elevated in the presence of approximated or adducted vocal folds. The initial adduction of the vocal folds in preparation for phonation coincides with the moment where the subglottal pressure starts to rise (Peters & Boves, 1988). This prephonatory strategy is referred to as the *laryngeal breath holding strategy* (Shipp *et al*., 1984). Phonation starts shortly before the subglottal pressure has reached its maximum level (Peters & Boves, 1988) and thus involves precise timing between the phonatory and respiratory systems.

Laryngeal postural adjustments prior to phonation as depicted by Type 2 may also reflect a hard glottal onset. Normal phonation onset is associated with little prephonatory arytenoid posturing, whereas hard glottal stop onset involves distinct and forceful prephonatory posturing (Hess, Verdolini, Bierhals, Mansmann & Gross, 1998). In the current study no distinction was made between laryngeal approximation for normal voice onsets and glottal closure for hard glottal attack onsets, since such differentiation needs to be based on data regarding interarytenoid contact pressure in conjunction with a description of the rise in amplitude of the L_x signal.

Subject group: For the subject group, only two of the six individuals (S4 and S5) showed prephonatory vocal fold behaviour comparable to that of the normal speakers. Both these individuals were perceptually judged as having mild stuttering involvement. Thus, based on this qualitative analysis of nature of prephonatory vocal fold activity, normal L_x wave patterns of prephonatory vocal fold activity were observed for the mild PWS (see Figures 4.2.1 - 4.2.3). If vocal fold activity prior to voice initiation showed less atypical behaviour in mild PWS, then the severity of stuttering might be related to the occurrence of deviant vocal fold activity in preparation to actual speech production.

The majority of the subjects deviated from the control group's tendency to Type 2. Since Type 2 is regarded as normal prephonatory vocal fold activity, it suggests that most subjects were inclined to atypical vocal fold behaviour. For these subjects, a general difficulty was indicated in

the control of vocal fold movements in coordination with changes in subglottal pressure prior to voice initiation (see Type 4).

- **Type 3: Prephonatory vocal fold approximation with laryngeal preadjustments during speech initiation**

Control group: Additional prephonatory vocal fold activity was seldom present in the normal speakers across the three phonetic contexts. Although vocal fold approximation with associated vocal fold activity during the prephonatory speech phase in normal speakers is not considered to be abnormal, its occurrence is uncommon (Hess *et al.*, 1998). Hess *et al*. (1998) described these incidents as subtle arytenoid adjustments that were interpreted as laryngeal adjustments associated with subvocal rehearsals. The prephonatory vocal fold activity for the control group was seldom associated with intermittent incidents of (secondary) prephonatory vocal fold activity following the laryngeal approximation.

Subject group: The subject group obtained similar results for Type 3 than the controls. Secondary deflections of the amplitude peak on the L_x signal could be related to the frequent repositioning of the vocal folds in the prephonatory period (Conture *et al.*, 1977). Similarly, Zimmermann (1980*b*) reported repeated adjustments in articulatory structures of PWS's fluent utterances which could lead to an increase in muscle activity. Based on Zimmermann's (1980*b*) comment, if associated vocal fold activity occurs frequently during the prephonatory phase, it could heighten vocal fold tension and consequently result in possible fixation of the vocal folds or irregular vocal fold activity prior to the onset of phonation.

- **Type 4: Prephonatory vocal fold approximation with irregular vocal fold activity during speech initiation**

Control group: For the group of normally fluent speakers, Type 4 occurred in only a small number of utterances which suggests stability in the vocal fold control for the period before speech initiation.

Subject group: A high frequency of irregular prephonatory vocal fold activity (Type 4) was observed for the subjects. Irregularity in vocal fold behaviour indicates great variability in vocal motor control during the prephonatory period for the subjects. This result confirms previous

findings that the preparatory period before voice initiation is the time interval in the speech of PWS which is characterized by disruptions of vocal fold activity (Zimmermann, 1980*b*; Van Riper, 1982; Peters *et al.*, 1989). Thus, the subjects might have experienced abrupt disruptions during the initiation of prephonatory vocal fold activities or problems in coordinating vocal fold movements with articulatory activities (Peters *et al.*, 1989). The occurrence of random and unpredictable irregular prephonatory vocal fold activity for the PWS might be explained by the finding by Riley and Ingham (2000) that PWS are slower in their ability to initiate phonation and present with highly variable vocal fold activity. The relation between the delay in phonatory onset and irregular vocal fold activity in PWS is therefore suggested. Irregularities in prephonatory vocal fold activity can cause a further breakdown in the sequencing of successive speech movements (Prosek *et al.*, 1979). The current finding suggests instability in the regulation of temporally sequenced speech movements prior to phonation in PWS.

The occurrence of Type 4 in CVC utterances, especially with an initial voiceless plosive, could also be explained by whispered speech onset. According to Rothenberg (1983), during whispered speech onset a glottal adjustment is present in which only some portion of the glottis is constricted to produce turbulence. No vibrations of the vocal folds occur. The onset of vocal fold vibration only occurs with the initiation of the following vowel. The PWS might have used whispered speech onset to enhance fluency.

The results for Type 4 suggest that initial sound productions that involve an articulatory as well as a voicing component (i.e. $C_{(v)}$VC or $C_{(vl)}$VC) are more difficult to initiate smoothly in PWS than utterances with only an initial voicing component (i.e. VC productions). Thus, the subjects' results for Type 4 for the three phonetic contexts seem to relate to the complexity of respiratory-phonatory-articulatory coordination of the VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances.

- **Type 5: Prephonatory vocal fold approximation with prevoicing during speech initiation**

Control group: A small number of $C_{(v)}$VC utterances presented with prevoicing for the onset of the initial voiced plosive. Within normal speech production, Type 5 can be considered to be characteristic of instances where vocal fold vibration precedes the release of the articulatory burst in a $C_{(v)}$ production. Anticipatory voicing or prevoicing is evident for these utterances. Since the

voicing feature of the initial sound in the $C_{(v)}VC$ utterance occurs before articulatory onset, it is measured as negative voice onset time (Borden & Harris, 1984; Hoole, 1997).

Subject group: A small frequency of occurrence of Type 5 was noted across the three phonetic contexts for the subject group. Fewer utterances were characterized by prevoicing in the subject group as opposed to the control group. This result may suggest that the subjects employed a slow, gentle voice onset for $C_{(v)}VC$ productions. The presence of vocal fold vibration for VC or $C_{(vl)}VC$ utterances during the prephonatory period is regarded as atypical vocal fold behaviour. The presence of vocal fold vibration before speech initiation might be indicative of vocal fold tremor such as reported by Borden *et al*. (1985). Only three of the six subjects showed instances of atypical prevoicing for VC productions (S1 and S3) and $C_{(vl)}VC$ productions (S3 and S4) (see Table 4.3).

5.2.1.1.2 Effect of sound initiation in different phonetic contexts on prephonatory vocal fold activity in normal speakers and persons who stutter

The influence of contextual factors in motor control needs to be considered when a relation between prephonatory vocal fold activity and the phonetic contexts is suggested. Van der Merwe (1997) identified certain conditions or contextual factors that influence speech motor control such as speech rate, familiarity of utterance, length of utterance, the sound structure and motor complexity of the utterance (see Table 2.8). In the current study, the factor that might have influenced the participants' speech behaviour for VC, $C_{(v)}VC$ and $C_{(vl)}VC$ productions is motor complexity. The degree of motor complexity differs amongst the phonetic contexts. The influence of this contextual condition on prephonatory vocal fold activity for the three phonetic contexts will be discussed.

Control group: For the control group, VC utterances showed the highest score of normal prephonatory vocal fold activity, followed by $C_{(vl)}VC$ productions (see Table 4.4). Normally for *VC production*, vocal fold adduction or approximation is needed for the initiation of voicing for the initial vowel of the utterance. This explains the occurrence of Types 2 and 3 in the period prior to actual speech production for the control group. The finding that VC utterances showed the least instances of atypical prephonatory vocal fold activity for the group of normal speakers,

is associated with the limited speech motor requirements for its production. In the *$C_{(vl)}$VC context, vocal fold approximation may be present* in preparation to the following voiced sound. Type 2 with Type 1 and Type 3 are regarded as non-deviant types of prephonatory vocal fold behaviour for $C_{(vl)}$VC productions. Thus, the group of normally fluent speakers mainly presented with normal vocal fold behaviour prior to the initiation of voiceless consonant productions. The results of the normal speakers reflected that the VC and $C_{(vl)}$VC productions were regulated by a stable speech motor system.

The *$C_{(v)}$*VC context for the control group was *generally* characterized by types of prephonatory vocal fold activity considered to be typical of normal speech production (Types 2 and 5). However, more instances of deviant prephonatory vocal fold activity (Types 1 and 4) for the $C_{(v)}$VC context were noted as opposed to the other two contexts. In normal production of voiced plosives, the period before voice initiation is associated with vocal fold approximation/adduction (with/without additional laryngeal adjustments, or with/without vocal fold vibration). The absence of vocal fold approximation prior to speech onset (Type 1) for only a small number of $C_{(v)}$VC productions might indicate that occasionally vocal fold adduction coincided with vocal fold vibration for the voiced plosive. The occurrence of Type 1 in $C_{(v)}$VC contexts could be related to an abrupt manner of voice initiation of the voiced plosive. Another possibility is that the voiced consonant, /b/, might have been substituted for the voiceless counterpart, /p/, which does not require vocal fold adduction. Also, during a very gentle voice onset, the L_x signal might not register whether the vocal folds are in an adducted or abducted position. The phonetic context with the most coarticulatory demands for speech production, i.e. $C_{(v)}$VC context, displayed more instances of variable vocal fold behaviour in the control group. Thus, motor complexity of the phonetic context appeared to have an effect on the prephonatory glottal activity of the group of normal speakers, but to a lesser extent than in the group of PWS.

Subject group: The phonetic context with the most instances of atypical prephonatory vocal fold activity for the group of PWS, was $C_{(v)}$VC utterances followed by $C_{(vl)}$VC utterances and then VC utterances (see Table 4.4). This observation suggests that when a PWS produces an utterance with a great degree of motor complexity, abnormal fluctuations in the laryngeal biomechanism are present in the period before speech onset. This apparent breakdown in motor control of the laryngeal activities in the PWS is considered to be underlying to the variability in prephonatory

vocal fold activities (Watson & Alfonso, 1987*b*; Dembowski & Watson, 1991*b*). For CVC productions with /p/ as initial sound, like in /b/-VC productions, the subjects showed a tendency toward irregular vocal fold behaviour (Type 4) during the prephonatory phase. Thus, the subjects frequently experienced difficulty in the motor control of prephonatory vocal fold activities in those utterances requiring a voiced or voiceless consonant onset. These results imply that motor complexity of the speech utterance has a prominent effect on prephonatory vocal fold activity for the group of PWS. The occurrence of atypical prephonatory vocal fold characteristics seems to be an index of motor complexity of the utterance. In other words, greater complexity of interarticulatory synchronization seems to result in more instances of abnormalities in prephonatory vocal fold activity of the subject group.

For the VC context, the subjects obtained irregular prephonatory vocal fold activity (Type 4) for 52% of the productions and 48% (Type 2 and Type 3) was prephonatory activity associated with normal speech production (see Table 4.4). The higher incidence of normal prephonatory vocal fold activity in VC utterances in comparison to CVC productions can be explained by the difference in motor complexity in VC *versus* CVC utterances. VC productions require less coarticulatory interaction as opposed to voiced/voiceless CVC productions. Motor complexity for initial consonant utterances involves more structures (i.e. muscles, ligaments and cartilages) to coordinate compared to initial vowels (Kent, 1998). Initial vowel production does not involve oral constriction/occlusion over glottal timing such as in the case of plosive production. Less variability in prephonatory vocal fold activity is associated with an increase of movement accuracy and motor control (Meyer, 1993). For the subjects, a decrease in variability seems to be related to reduced coarticulatory requirements. In Afrikaans, however, the initial vowel production is sometimes preceded by a glottal stop, i.e. /ʔ/. In these cases the sound structure will change to the $C_{(vl)}$VC context with increased demands on motor coordination.

Although individual differences were detected among the PWS, it appears as if the occurrence of deviant prephonatory vocal fold activity is dependent on the motor complexity of the utterance. The PWS showed more irregularities in prephonatory vocal fold behaviour for $C_{(v)}$VC and $C_{(vl)}$VC utterances than for VC utterances. Considering that the demands for coordination of the speech motor activities (including respiratory, phonatory and articulatory activities) vary according to the phonetic context, it could explain the number of irregularities in the initiation of

sounds with both dynamic phonatory and articulatory involvement as opposed to sound initiation with only phonatory involvement. According to Peters *et al*. (1989) and Dembowski and Watson (1991*b*), preparation deficits along with high motor initiation variability in PWS are associated with the motoric complexity of the specific speech tasks. However, the group size was too small to generalize the findings, but the results within the group of PWS clearly indicate that the frequency of speech motor deviancies increases as motor complexity of the speech task increases.

Individual variations in tendencies toward types of prephonatory vocal fold activity for CVC productions were indicated. Two PWS rated with moderate-severe stuttering (S1 and S3) showed predominantly atypical vocal fold activity in $C_{(v)}VC$ and $C_{(vl)}VC$ utterances (Type 4 and Type 5). These subjects seem to have been greatly affected by motor complexity of the utterance. For one subject rated with severe stuttering (S3), the prephonatory period was mainly characterized by deviancies in vocal fold activity across all three phonetic contexts. Abnormal prephonatory vocal fold activity seems to be constantly present during speech initiation in this subject. Watson and Alfonso (1987*b*:445) ascribed prephonatory abnormalities in severe PWS to "infrequent and delayed initiation of poorly organized respiratory and laryngeal events".

Subject 6 obtained high scores for atypical vocal fold behaviour for the two voicing contexts, i.e. $C_{(v)}VC$ and VC. This subject might therefore experience specific problems with voicing activities. In $C_{(vl)}VC$ utterances, most of the prephonatory vocal fold activity for Subject 6 were also characterized by aberrant vocal fold behaviour. Although perceptually judged as a mild PWS, the results of Subject 6 corresponded with that of Subject 2, a severe PWS, as opposed to those of the mild PWS (S4 and S5) as would have been expected. The mild PWS (S4 and S5) showed low scores for atypical prephonatory vocal fold behaviour. The difference in distribution of types of prephonatory glottal activity between the mild PWS (S4 and S5) and moderate-severe PWS (S1, S2 and S3) was notably greater than the difference between the mild PWS and the group of normal speakers. According to Watson and Alfonso (1987*b*), mild PWS executed organized respiratory and laryngeal prephonatory activities and showed improvements in the initiation and timing of prephonatory events when provided with a longer preparatory period, which were not observed for severe PWS. The current results seem to point toward a relation between stuttering severity and atypical prephonatory vocal fold activity, but lack evidence to substantiate Watson and Alfonso's (1987*b*) findings.

In summary, the presence of atypical vocal fold characteristics has been found in normal as well as stuttering speakers' speech production. However, the frequency of occurrence differed greatly between the normal and the stuttering speakers. In the normal speakers, only a few utterances were characterized by abnormalities, indicating that this type of activity seldom occurs in normal speech production. Atypical prephonatory vocal fold activity during speech initiation seems to be common to stuttering behaviour. The severity of stuttering as rated perceptually does not seem to be a reliable predictor of the presence of aberrancies in prespeech vocal fold behaviour as seen from the results of Subject 6 and Subject 2. However, four out of the six subjects (S1, S3, S4 and S5) did show a possible relation between stuttering severity and the occurrence of aberrant vocal fold behaviour. The perceptually fluent speech of PWS has often been characterized by a breakdown in laryngeal motor control (physiological blocks), too subtle to be identified as disfluencies (Adams *et al.*, 1984). Thus, the incidence of abnormalities in prephonatory speech motor activities might be a more reliable predictor of stuttering severity, as opposed to perceptual ratings of behaviour.

5.2.1.2 Prephonatory vocal fold activity in persons who stutter (PWS) within the framework of speech sensorimotor control

Aberrant vocal fold behaviour in the time prior to voice/speech initiation has been reported extensively in stuttering research (Freeman & Ushijima, 1978, Shapiro, 1980; Reich *et al.*, 1981; Hayden *et al.*, 1982; Van Riper, 1982, Conture *et al.*, 1985; Peters & Boves, 1988; Peters *et al.*, 1989; Webster, 1990). Most of these studies suggest that abnormalities in the vocal fold activity of PWS might result from a breakdown in the synchronization between laryngeal biomechanics, subglottal pressure, and activity of the respiratory muscles during the prephonatory phase of speech production. Research on vocal fold activity of PWS confirms the presence of irregularities during their speech initiation, e.g.: (a) Excessive laryngeal muscle tension and muscular activity (Freeman & Ushijima, 1978; Van Riper, 1982); (b) Deviant vocal fold positioning (i.e. the vocal folds are fixed in an adductor or abductor position) (Conture *et al.*, 1985); (c) Inconsistencies in movement preparation for speech production (Webster, 1990); (d) Slowness in movement initiation (Reich *et al.*, 1981; Hayden *et al.*, 1982). If there is a slowness or delay in phonation in the presence of a faster respiratory rate, it can result in asynchronization

between phonation and respiration (Davis *et al.*, 1993); (e) A disruption in the smooth and continuous subglottal air pressure build-up patterns before starting phonation (Peters & Boves, 1988). For example, if respiratory defensive reflexes are stimulated or triggered prior to phonation, it might result in a disruption to the sequence of the initiation of voice or speech and to the pattern of intercostals, diaphragm and laryngeal muscle activity (Davis *et al*., 1993). These disruptions in speech motor activities might be related to timing problems (Peters *et al.*, 1989). The effect of this disruptiveness on speech production is thought to be dependent on both, degree and duration of muscle activity (Shapiro, 1980).

Bakker and Brutten (1989) and Peters *et al.* (1989) suggest that atypical vocal fold characteristics evidenced during the prephonatory period in PWS may be caused by a deficit in motor programming. The prephonatory vocal fold activities in PWS seem to relate to speech motor symptoms ascribed to a deficit of the motor programming level as defined in the framework by Van der Merwe (1997) (see 2.4.1.5, Chapter 2). According to Van der Merwe (1997), deviant speech motor behaviour associated with a motor programming disorder may be associated with abnormalities in muscle tone, a disruption in the repeated initiation of movement and a disturbance in tone, rate, velocity and range of movement.

5.2.2 *Discussion of sub-aim 1.2*: Laryngeal Onset Time (L_xOT)

The discussion of laryngeal onset time (L_xOT) includes a discussion of L_xOT for sound initiation in different phonetic contexts in normal speakers and persons who stutter (5.2.2.1); and a possible explanation of L_xOT in persons who stutter within a framework of speech sensorimotor control (5.2.2.2).

5.2.2.1 Laryngeal onset time (L_xOT) for sound initiation in different phonetic contexts

The discussion of laryngeal onset time (L_xOT) for sound initiation in different phonetic contexts includes a discussion of the differences in mean L_xOT (5.2.2.1.1) and L_xOT range (5.2.2.1.2) between normal speakers and PWS.

5.2.2.1.1 Differences in *mean L_xOT* between normal speakers and persons who stutter

Marked differences in L_xOT were noted between the control group and the subject group across VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances. Laryngeal onset time in the subject group was generally longer than for the control group. The group of normal speakers showed longer L_xOT for VC productions than for $C_{(v)}$VC and $C_{(vl)}$VC productions. $C_{(vl)}$VC productions displayed shorter lags in L_xOT than $C_{(v)}$VC productions for this group. Compared to the normal speakers, the PWS showed individual differences with regard to the effect of phonetic context on L_xOT.

Control group: According to the results, normal speech production seems to require longer L_xOT for VC productions than for CVC productions. In a monosyllabic production, the vowel is regarded as the nucleus (central part) of the syllable and is consequently produced with more effort or stress (Abercrombie, 1967; De Villiers & Ponelis, 1987). Stressed vowel production in a VC context is accomplished by extra force of respiratory movement to increase subglottal pressure in the presence of increased vocal fold tension (Abercrombie, 1967). Furthermore, productions of the central vowels (/*a*/, /ə/) are usually prolonged when in the initial syllable position, as opposed to /ɪ/ and /ɔ/ (Kent, 1998). Subsequently, more subglottal pressure is needed to sustain vocal fold vibration for prolonging a voiced (vowel) sound, as well as higher strain is placed on the vocal folds for continued periodic vibrations (Kent, 1998). These prephonatory adjustments in respiratory and laryngeal-phonatory activities must be synchronized to enable and to sustain vocal fold vibration during speech initiation. The L_xOT results for VC productions in the group of normal speakers suggest that vowel stress possibly resulted in longer preparation time prior to normal speech initiation.

Since vocal fold vibration is a required for the production of the initial voiced sound in $C_{(v)}$VC utterances, preparatory adjustments of the vocal folds will precede the utterance. During the initiation of $C_{(vl)}$VC productions, vocal fold vibration is not needed for the initial voiceless sound. For normal speakers, the presence of prephonatory vocal fold activities in $C_{(vl)}$VC productions can be related to anticipatory laryngeal coarticulation for production of the vowel following the initial voiceless consonant. This implies that L_xOT in $C_{(vl)}$VC productions reflects on the transition period from the initial voiceless consonant to the subsequent vowel. $C_{(vl)}$VC productions is therefore expected to have shorter L_xOT, as opposed to longer L_xOT in utterances

which require vocal fold vibration for the initiation of the first sound (i.e. VC and $C_{(v)}$VC productions). Thus, it appears as if the voicing feature of the phonetic contexts affected the L_xOT for the group normal speakers. The apparent relationship between mean L_xOT and the voicing feature of a given phonetic context in the normal speakers is consistent with findings by Peters and Boves (1988). The L_xOT results for the control group seem to indicate normal speech behaviour.

Subject group: In general, the group of PWS showed longer L_xOT than the group of normal speakers. Thus, compared to the normal speakers, PWS showed a longer duration between prephonatory vocal fold approximation and the onset of vocal fold vibration for speech initiation. A general slowness or delay in the initiation of vocal fold vibrations for phonatory onset might account for the L_xOT result of PWS. The finding is supported by previous studies indicating longer voice initiation time and laryngeal reaction time in PWS's fluent and disfluent speech productions (Reich *et al.*, 1981; Till *et al.*, 1983; Cross & Olson, 1987; Watson & Alfonso, 1987*b*; Bakker & Brutten, 1989; Peters *et al.*, 1989; Dembowski & Watson, 1991*b*).

Longer L_xOT could be indicative of a conscious compensatory strategy employed by PWS to ensure fluent speech production. The longer L_xOT is in this case not due to a *breakdown* in coordination, but it can be associated with a technique for stuttering management. Coarticulation and most likely coordination vary with speaking rate (Wieneke, Janssen & Belderbos, 1987). Coarticulatory interaction can be reduced at slow speaking rates. A slow rate allows greater preparation time for movements and reduces kinematic interaction and coarticulation (Wieneke *et al.*, 1987; Caruso, 2000). A slow speaking rate can be associated with a slower initiation time. In this regard, the slowness in laryngeal onset is a manner to reduce the coarticulatory movements between the vocal folds and subglottal pressure per time unit in PWS's attempt to maintain fluency.

Another explanation for longer L_xOT in PWS, could point to difficulty in the temporal-sequential arrangements of complex speech movements in preparation to speech production. A breakdown in the preparatory speech motor activities might affect the timing and sequencing of the successive movements (Prosek *et al.*, 1979). In view of the current L_xOT results, the stuttering speakers possibly required a longer preparation period to synchronize vocal fold adjustments with

an increase in subglottal pressure necessary for phonatory onset. A delay in the initiation of voicing can occur if the glottal resistance is too high for the lower subglottal pressures or the build-up of subglottal pressures is inadequate to set the vocal folds into vibration (Peters & Boves, 1988). Another possibility is that the glottal resistance might be high as well as the subglottal pressure but that these actions are not synchronized. The result of this asynchrony is a forceful release of the high pressure which is followed by sudden contraction of the vocal folds (hard glottal attack). The sudden glottal closure is due to the Bernoulli effect to equalize intra-oral air pressure and subglottal pressure (Borden & Harris, 1984). Such breakdown in aerodynamic coordination could explain the longer L_xOT as observed in the stuttering speakers. The possibility that increased latencies of movement onset in PWS are associated with physiological disruptions in preparatory movements prior to speech and less precision (or slowness) in the temporal sequencing of movements has also been suggested by previous research (e.g. Zimmermann, 1980*b*; Adams *et al*., 1984; Healey & Gutkin, 1984; Starkweather, 1987; Bakker & Brutten, 1989; Peters *et al*., 1989; Van Lieshout *et al.*, 1993).

A disruption in the temporal regulation and sequencing of preparatory laryngeal-respiratory activities may affect subsequent speech motor activities. Thus, discoordinations in laryngeal-phonatory-respiratory behaviour during speech onset may manifest as hard glottal attacks, arrhythmic vocal fold vibrations, irregular abduction of the vocal folds, or disrupted voicing during continuous rapid glottal activity (Freeman & Ushijima, 1978; Adams *et al*., 1984; Borden *et al.*, 1985: Peters *et al.*, 1989). These disruptions (depending on the degree of involvement) in speech behaviour may be perceptible as disfluent speech (Adams *et al.*, 1984). Van Lieshout *et al*. (1993) predict that PWS with longer initiation times have problems in maintaining fluency and will show disfluencies if their motor control strategy fails.

The individuals who stutter showed individual differences relating to the effect of phonetic context on L_xOT. Compared to the mean L_xOT for *VC productions* in the normal speakers, the moderate-severe PWS showed longer onset times, while the results of very mild-mild PWS were similar to that of the normal speakers. For the moderate-severe PWS this could indicate that the subglottal pressure might have increased gradually and vocal fold vibration started well after the pressure had reached its maximum level (Peters & Boves, 1988). Consequently, voice initiation is delayed. Following the subglottal pressure build-up, a sudden release of the adducted vocal

folds might have resulted in a glottal burst or attack. Hard glottal attacks in the presence of VC productions may occur in Afrikaans when the initial vowel is produced with greater force due to the presence of the preceding voiceless glottal stop, i.e. /ʔ/ (De Villiers & Ponelis, 1987). This increase of demands on the timing and coordination of prephonatory activities may result in a delay or breakdown in laryngeal onset prior to speech initiation in PWS.

Compared to the normal speakers, some individuals who stutter required a longer period to initiate voicing for *$C_{(v)}VC$ utterances* after laryngeal approximation was achieved. The effect of motor complexity involved in preparatory movements for the initiation of $C_{(v)}VC$ productions may related to this finding. For these PWS, the complexity of the preparatory movements involving laryngeal, respiratory, articulatory coordination to initiate a prevocalic voiced plosive may require a longer prephonatory period than for VC productions. Also, irregularities in complex, successive speech movements during the prephonatory period of these productions might have resulted in a breakdown in movement control, indicated as long L_xOT.

The majority of the stuttering speakers maintained mean L_xOT values similar to that of the normal speakers for $C_{(vl)}VC$ productions. For two individual subjects (S1 and S3), the mean L_xOT of $C_{(vl)}VC$ utterances differed greatly from the normal speakers' results (see Table 4.8) The apparent delay in L_xOT for these subjects may suggest that subglottal pressure has built up to a maximum level which is too high for smooth phonatory onset of the subsequent vowel. Failure in the control of subglottal pressure is indicated hereby. According to Watson and Alfonso (1987*b*) and Peters and Boves (1988) this can be indicative of inadequate coordination between respiratory and articulatory gestures. For most stuttering speakers, the mean L_xOT for $C_{(vl)}VC$ utterances was similar to the group of normal speakers. This finding suggests that a decrease in laryngeal-phonatory requirements prior to speech onset results in enhanced temporal control. Thus, it is possible that a reduction in the complexity of speech motor coordination may promote temporal control.

5.2.2.1.2 Differences in *L_xOT range* between normal speakers and persons who stutter

Temporal specifications of speech movements are important for the coordination of the multiple articulators (including the vocal folds) (Gracco, 1988). If a timing measure (such as L_xOT)

shows variability, then intersystemic coordination is likely to be affected. Since a decrease in variability is associated with an increase of movement accuracy and motor control (Meyer, 1993), it implies that highly variable movements manifest due to a lack in movement control. A lack in control of speech motor activities could be related to difficulty in the sequencing of complex movements and precise timing of these sequenced movements, such as seen in stuttering (Perkins *et al.*, 1976; Prosek *et al.*, 1979; Van Riper, 1982; Bloodstein, 1987). Thus, any breakdown in the regulation of the respiratory system in supplying subglottal airflow, or a failure to coordinate expiratory actions and the concomitant adjustments of the phonatory system (and articulatory system) during prephonation could be the result of instabilities in the speech motor control of PWS.

Control group: The small differences are noted between the *mean L_xOT* and *range of L_xOT* for the phonetic contexts in the group of normal speakers. This finding suggests that the normal speakers were able to regulate vocal fold activities and/or subglottal pressure changes during the prephonatory period (speech motor activities) with consistency. Also, similar values of L_xOT range between the different contexts for the group of normal speakers are noted. This observation provides further support to the notion that fluent speakers' speech motor activities generally reflects predictability and consistency in speech motor control (Kent & Adams, 1989).

Subject group: Compared to the results of the normal speakers, the group of PWS obtained high values for L_xOT range across the phonetic contexts. This great difference between the maximum and minimum L_xOT values in PWS reflects variability in absolute L_xOT values. Studies on voice initiation confirm the existence of variability in the timing of laryngeal-phonatory activities during the prespeech period in PWS (Reich *et al.*, 1981; Till *et al.*, 1983; Watson & Alfonso, 1987*b*; Peters *et al*., 1989; Dembowski & Watson, 1991*b;* Williams & Bishop, 1992). Variability (or deviancies) in the timing of prephonatory laryngeal movement suggests inappropriate control of speech motor activities in PWS (Peters *et al*., 1989).

The following explanation may account for the highly variable L_xOT as seen in the stuttering speakers: During the prephonatory period, movements in preparation to vocal fold vibration are temporally organized (regulated) and coordinated as determined by the specific phonetic context of the utterance (Dembowski & Watson, 1991*b*). A higher demand is placed on motor speech

control for the regulation and temporal coordination of complex movements. If a speaker presents with a defect in motor speech control (such as stuttering), then greater demands on the motor control system may result in longer processing time with a greater possibility of variability and inconsistency in speech coordination (Till *et al.*, 1983). An underlying difficulty in the regulation of laryngeal-phonatory activities in coordination with respiration and articulation during the prespeech period may result in variable speech behaviour (Perkins *et al.*, 1976; Watson & Alfonso, 1987*a*). PWS showed irregular prephonatory vocal fold activity for VC, $C_{(v)}$VC and $C_{(vl)}$VC productions (see Table 4.2) that seems to be associated with the variability in L_xOT. This finding supports previous views (Van Riper, 1982; Peters & Hulstijn, 1987; Peters & Boves, 1988; Denny & Smith, 1997; Archibald & De Nil, 1999) that a breakdown in intersystemic coordination during speech initiation is characteristic to stuttering.

The majority of the stuttering speakers had markedly higher L_xOT range values for *VC productions* compared to the group of normal speakers. For VC productions, most of the stuttering speakers might have experienced instabilities in aerodynamic coordination of the laryngeal-phonatory and respiratory systems (Van Lieshout *et al.*, 1993). If the initial vowel in the VC context was preceded by the voiceless glottal plosive, /ʔ/ (De Villiers & Ponelis, 1987), then the higher degree in motor complexity of the utterance could have influenced the smooth regulation of coarticulatory movements in PWS. This perspective is further strengthened by the magnitude of atypical prephonatory glottal activity in VC productions for PWS (see Table 4.2).

For the $C_{(v)}$VC and $C_{(vl)}$VC utterances, five of the six individuals who stutter had higher L_xOT range values than the normal speakers. Greater variability in the preparatory vocal fold activities for actual speech production in the majority of stuttering speakers is hereby indicated. Inconsistency in L_xOT for these utterances can be attributed to the stuttering speakers' tendency toward irregular or variable prephonatory vocal fold activity (see Table 4.2). These stuttering speakers could have experienced great difficulty in maintaining stability in vocal fold activities and/or subglottal pressure changes during the prephonatory period (Peters & Boves, 1988).

Problems in the timing of laryngeal-phonatory adjustments in coordination with respiration and articulation during prephonation in PWS are associated with a breakdown in speech motor control. Such timing deviancy in the coordination of speech production processes needs to be

considered within the framework of motor programming (Abbs & Connor, 1989; Aravind Kumar & Savithri, 1998). The following discussion on the speech behaviour in stuttering will therefore be founded on motor programming as depicted by Van der Merwe (1997).

5.2.2.2 Laryngeal onset time (L_xOT) in persons who stutter within a framework of speech sensorimotor control

Since motor programming involves spatiotemporal specifications for muscle movements (Van der Merwe, 1997), deficits in timing or synchronized movement sequence in stuttering speakers could be related to a disorder at the level of motor programming. The L_xOT results in the stuttering speakers indicated delayed laryngeal-phonatory movement onset and a possible breakdown in coordination of respiratory, laryngeal-phonatory and articulatory processes. These behaviours can be associated with the slowness in PWS's speech movements and involuntary interruptions of speech activities observed by Van Borsel, Van Lierde, Van Cauwenberge, Guldemont and Van Orshoven (1998)[*]. PWS are thought to be slower and much less consistent in preparing and initiating vocal responses (Williams & Bishop, 1992). PWS seem to be less skilled to activate/inhibit selected motor programs in the appropriate temporal relationship and to adjust these motor programs according to the movement requirements (including force, rate, velocity of movement, range of movement and mechanical stiffness of the joints) of a specific task. Deviancies in L_xOT for individual stuttering speakers could be viewed as the inability to regulate or adjust movements according to the motor demands placed by the specific utterance.

A general slowness in the preparatory vocal fold adjustments prior to speech onset in PWS, suggest an underlying problem in the timing and coordination of speech movements (Watson & Alfonso, 1987*a*). Possible reasons for this vocal fold behaviour in stuttering might relate to neural control of automatic processes *versus* voluntary processes such as proposed by Denny and Smith (1997) and Kehoe (1997*a*). Denny and Smith (1997) suggest that asynchronization might occur between the automatic processing of the respiratory activities and the processing of the articulatory-phonatory activities, or the laryngeal mechanism might receive competing messages from the two control mechanisms (i.e. automatic processing and cognitive control). The larynx is

[*] According to Van Borsel *et al*. (1998) these behaviours might be related to overactivity of the supplementary motor region. PWS show extensive activity in the supplementary motor region (see Chapter 2) which is greatly reduced when fluency control is practiced (Van Borsel *et al*., 1998).

involved in automatic responses (including a closing valve for the airway passage and a stabilization point during strenuous activities) which are controlled by lower brain structures. The larynx is also concerned with the regulation of subglottal pressure for speech production and vocal fold vibration to produce voice which is controlled by higher neural centers (Titze, 1993; Denny & Smith, 1997; *NCS*, 2002). The latter function of the larynx requires precise timing and graded tensing. In PWS, a failure in the neuromotor control of speech motor activities may result in automatic responses interfering with the graded, precise movements of speech production. Another possibility may be that the higher control centres exert ineffective strategies to regulate high-speed movements and/or apply excessive inhibitory control when the rate of interarticulatory synchronization increases. This inefficiency in neuromotor control might account for PWS's failure to control particular skilled speech movements such as prephonatory vocal fold adjustments during speech initiation.

Increased movement variability in PWS is associated with a decrease in stability and coordination of the activity (Adams, 1981; Archibald & De Nil, 1999). In order to regain or facilitate stability and coordination of successive respiratory, laryngeal-phonatory and articulatory movements, a slower movement onset may be applied by PWS. Slower movement onset or initiation permits more time for the speaker to program and/or execute speech movements (Kent, 1984, Abbs & Connor, 1989). According to Van Lieshout *et al.* (1993), longer onset times do not implicate a *disorder* of timing in PWS. Individuals who stutter might employ a speed-insensitive strategy for voluntary control of speech motor movements which could result in a general slowness of specific movements (Van Lieshout *et al.*, 1993).

5.2.3 Summary: Physiological vocal fold activity during speech initiation in persons who stutter

An investigation into the physiological vocal fold activity, as determined by glottalgraphic assessment, shows distinct differences between the group of PWS and the group of normal speakers. The stuttering speakers presented with a greater tendency toward irregular prephonatory vocal fold activity as well as longer and more deviant L_xOT than normal fluent speakers. Based on existing literature, indications of slowness and irregularities in vocal fold activity prior to speech initiation have a neurophysiological basis. Characteristics of

prephonatory vocal fold activities during speech initiation in PWS seem to be related to speech motor characteristics of motor programming disorders. Atypical vocal fold activities and variability in L_xOT increased with an increase in motor demands for speech production. This finding suggests that increased motor complexity of vocal fold adjustments may result in a breakdown in speech motor control. Furthermore, it is clearly indicated that atypical physiological events that intermittently influence the processes of respiration, phonation and/or articulation are not limited to moments of overt stuttering.

Despite the tendency to abnormal prephonatory vocal fold activity in the group of PWS, aspects such as the frequency of occurrence, nature and severity of abnormal prephonatory speech motor activity differed from individual to individual. Individual differences underscore the fact that stuttering speakers present with idiosyncratic speech behaviour during fluent and disfluent speech production. Some indications of a relationship between stuttering severity and L_xOT were evident, but not enough evidence was obtained to substantiate the notion.

5.3 *DISCUSSION OF SUB-AIM 2:* ACOUSTICALLY DETECTED VOCAL FOLD ACTIVITY DURING SPEECH INITIATION

5.3.1 *Discussion of sub-aim 2.1:* The nature of voice onset

A discussion of the nature of voice onset energy includes a discussion of the differences of the nature of voice onset energy in normal speakers and persons who stutter (PWS) (5.3.1.1); and a possible explanation of the nature of voice onset in PWS within a framework of speech sensorimotor control (5.3.1.2).

5.3.1.1 Description of the nature of voice onset in normal speakers and persons who stutter (PWS)

The description of the nature of voice onset energy in normal speakers and persons who stutter (PWS) includes a discussion of the frequency of occurrence of gradual or abrupt voice onset in normal speakers and PWS (5.3.1.1.1); and the effect of sound initiation in different phonetic contexts on the nature of voice onset energy in normal speakers and PWS (5.3.1.1.2).

5.3.1.1.1 Frequency of occurrence of gradual or abrupt voice onset in normal speakers and persons who stutter

Control group: The voice onset of speech utterances in the control group was mainly characterized by a gradual rise in amplitude of the voice signal. While the VC and $C_{(v)}$VC contexts showed a high incidence of gradual voice onset, the $C_{(vl)}$VC productions for this group showed a small difference between gradual and abrupt voice onset. A gradual voice onset is usually expected for normal speech production (Peters & Boves, 1988). However, in a study by Borden *et al.* (1985) the normal speakers showed abrupt voice onset for initial voiceless consonant productions. The abruptness in prevocalic /p/ productions might be attributed to the influence of the neighbouring vowel, especially under conditions of increased speaking rate, shorter utterance duration or changes in tone of voice (increased stress). The reason for this abrupt onset was probably due to a sudden release of the adducted vocal folds following the articulatory burst for the initial /p/ (Borden *et al.*, 1985). Thus, the initial /p/ could have been influenced by the voicing features of the subsequent vowel[*]. Evidence of abrupt voice onset in $C_{(vl)}$VC utterances may indicate that the group of normal speakers were able to coordinate complex speech motor activities with rapidness and accuracy. In this case, abruptness in voice onset implies efficient processing of complex movement variables. This is supported by the observation that $C_{(vl)}$VC utterances showed regular prephonatory laryngeal behaviour prior to voice onset in the presence of a shortened L_xOT. It is important to keep in mind that the manner of voice onset cannot be isolated from the preceding laryngeal-phonatory activities before voice onset. In other words, the onset of voicing is temporally synchronized with the foregoing period of laryngeal preadjustments.

Subject group: The nature of voice onset for $C_{(v)}$VC and $C_{(vl)}$VC productions in the subject group resembled that of the control group. This finding, if viewed in isolation, is non-conclusive, but it might be of great significance when interpreted with the results of the prephonatory vocal fold behaviour. For the control group, gradual voice onsets were preceded by well-organized laryngeal-phonatory activities, contrary to the stuttering subjects who showed gradual voice onsets that were preceded by irregular prephonatory vocal fold behaviour. The high incidence of

[*] The abrupt voice onset in prevocalic /p/ productions may be related to the high VOT error score for $C_{(vl)}$VC productions in the normal speakers (see Table 4.12).

abnormal prephonatory vocal fold behaviour in CVC productions suggests that these productions were not preceded by smooth, regulated preparatory speech movements (see 5.2.1). Gradual voice onsets for the $C_{(v)}$VC and $C_{(vl)}$VC contexts in most PWS were probably due to a compensatory strategy they used to control disfluency (Borden *et al.*, 1985; Peters & Boves, 1988). The possibility that the PWS used a gradual (easy) onset of voicing to inhibit or prevent disruptiveness during actual speech production, is further supported by prolonged L_xOT in $C_{(v)}$VC and $C_{(vl)}$VC productions (see Table 4.6 and Table 4.7). A delay in laryngeal-phonatory initiation will enable the PWS to process fewer muscle movements per time unit, and thereby simplify temporal coordination to establish movement accuracy. Furthermore, Dembowski and Watson (1991*a*) stated that gradual or easy voice onset is associated with aspirated sound production - thus, the release of the articulatory occlusion is followed by a delay in onset of vocal fold vibration. This statement implies that gradual voice onset may be related to longer VOT in the $C_{(v)}$VC and $C_{(vl)}$VC contexts for the stuttering speakers.

The stuttering speakers could also have applied gentle voice onset as a fluency-evoking technique in the absence of prolonged laryngeal initiation time as seen in different phonetic contexts for the two mild PWS and one very mild PWS (S4, S5 and S6). Since the level of subglottal pressure required for voice onset is related to the intensity of voice onset, it follows then that less pressure is needed for softer vocalizations (except in cases of forced whispered speech) (Stathopoulus, 1986). Less vocal fold tension is needed for voice onset when subglottal pressure is reduced. This proposes that a gentle or gradual voice onset may be associated with a reduced intensity level and a lower level of subglottal pressure. A possible link might therefore exist between the manner of voice onset and vocal intensity level. Gentle voice onset in comparison to abrupt onset, seems to involve less complex speech production activities; thus, the complexity of respiratory, phonatory and articulatory coordination required for voice onset are reduced and simplified for initiation**.**

The magnitude of difference between gradual and abrupt voice onsets was smaller for the $C_{(vl)}$VC productions due to a single PWS (S6) who displayed more instances of abruptness during voice onset. This individual who stutters approached the onset of voicing for $C_{(vl)}$VC utterances in the same way as the group of normal speakers and exhibited similar values for mean L_xOT than the normal speakers. However, the presence of irregular prephonatory vocal fold activity and the

great variability in absolute durations of L_xOT of this stuttering speaker (S6) seem to indicate that the general timing of prespeech activities is not representative of the increased levels of muscle activity associated with stuttering. The prediction is that obvious temporal disruptions in certain individuals who stutter "may emerge from a problem that has more to do with improper levels of activity than improper timing" (Borden *et al.*, 1985:371).

Both abrupt voice onset and gradual onset of voicing can be associated with speech characteristics of PWS (Peters & Boves, 1988; Webster, Morgan & Cannon, 1987; Dembowski & Watson, 1991*a*). Compared to the results of the control group, the subject group showed a slightly higher incidence of abrupt voice onset for initial vowel productions. The presence of hard glottal attacks for VC productions in the stuttering speakers might explain this finding (see 5.3.1.1.2). The two individuals who exhibited the most instances of abrupt voice onset for VC productions differed in relation to level of stuttering severity, i.e. a severe PWS and a very mild PWS. This finding indicates that perceptual ratings of stuttering severity are not reliable in predicting abnormalities in PWS's speech motor activities.

5.3.1.1.2 Effect of sound initiation in different phonetic contexts on the nature of voice onset in normal speakers and persons who stutter

- **VC Context: Vowel onset**

Most of the stuttering speakers showed a tendency toward abrupt voice onset for VC productions, compared to the gradual onsets of the group of normal speakers. The abruptness in voice onset might be indicative of a mistiming in the coordination of laryngeal movements and changes in subglottal air pressure prior to the onset of voicing (Webster *et al.*, 1987). Another explanation for an abrupt onset is a problem in the aerodynamic interface of the laryngeal-phonatory and respiratory systems (Van Lieshout *et al.*, 1993), which can result in a hard glottal attack. A hard glottal attack is usually produced with more intensity and force, and is associated with great interarytenoid and intraglottal contact pressures (Hess *et al.*, 1998). Borden and Harris (1984) describe it as a manner of voice initiation in which the vocal folds are tightly adducted prior to vibration and the sudden opening creates an airburst of a plosive. In Afrikaans, a hard glottal attack is normal speech behaviour for the glottal plosive /ʔ/ to precede the onset of the VC syllable (De Villiers & Ponelis, 1987). Considering this normal phenomenon in vowel

production of Afrikaans, together with the fact that vowel production in PWS has been related to restricted spatial and temporal movements in the presence of high levels of laryngeal and articulatory muscular tensions (Klich & May, 1982; Prosek, Montgomery, Walden & Hawkins, 1987), it is likely that PWS will experience abruptness during voice onset. For VC productions, stuttering speakers experience thus additional vocal fold contact pressures that are exerted for the glottal plosive in the presence of the already-tensed vocal folds. An increased level of subglottal air pressure is needed to overcome this excessive tension and to initiate vocal fold vibration.

In addition, the contextual influences of the initial vowel in VC utterances might have imposed further strain on the weak speech motor control system of the PWS. For example, initial vowels in VC contexts are usually stressed, which require a higher level of subglottal air pressure and concomitantly increased vocal fold tension (Abercrombie, 1967). Also, the duration of the preceding vowel is shorter before a voiceless stop (Guenther & Perkell, 2004), which involves fast movements with greater coarticulatory overlap. Greater stress with shorter durations for speech sound productions set higher demands on the speech motor control system for accurate timing and synchronization of speech activities. The possibility exists that these specific physiological demands are placed on a speech motor control system with an underlying incompetence or vulnerability in temporal coordination. The subjects might have experienced difficulty in managing these speech motor demands effectively during VC productions.

Gradual voice onsets for the initiation of VC utterances in the stuttering speakers can be related to fluency enhancement strategies via increased vowel durations. Gradual onsets might be associated with increased vowel durations (Riley & Ingham, 2000). Increased vowel durations allow more time for speech motor preparation (Riley & Ingham, 2000). In such a case, stuttering would be reduced as a by-product of longer vowel duration. Since each of the PWS received previous speech therapy (see 3.1.4.3., Chapter 3), it is possible that gradual voice onsets could have been employed as a compensatory strategy to induce fluency.

- **$C_{(v)}$VC Context: Voiced consonant onset**

Gradual voice onsets were characteristic of the initiation of $C_{(v)}$VC utterances in both the normal speakers and the stuttering speakers. For the stuttering speakers, though, prephonatory vocal fold irregularities were noted with a prolonged preparatory time prior to voice onset indicated by the

L_xOT results. This implies that the stuttering speakers have the ability to control or inhibit a breakdown in speech motor coordination prior to speech onset, if the duration of movement initiation is prolonged. Delayed voice onset may be a motor compensatory strategy to control fluency as proposed by Borden *et al*. (1985) and Peters and Boves (1988).

- **$C_{(vl)}$VC Context: Voiceless consonant onset**

The group of normal speakers showed relatively the same incidence of gentle and abrupt voice onsets for the $C_{(vl)}$VC context. Contrary to the normal speakers' results, the stuttering speakers showed a distinct tendency toward gradual voice onset in $C_{(vl)}$VC. Gradual voice onsets seem to indicate the PWS's ability to control a possible breakdown in speech production for a particular phonetic context. For instance, four PWS showed gradual voice onsets for $C_{(vl)}$VC productions, but only two of the four PWS (i.e. S2 and S4) also had gradual voice onsets for $C_{(v)}$VC productions. Thus, the speech motor requirements of the $C_{(v)}$VC and $C_{(vl)}$VC utterances affected the individual stuttering speakers differently. $C_{(v)}$VC utterances involve a voiced-voiced transition from the initial sound to the adjacent vowel which implies sustained phonation, while initiation of $C_{(vl)}$VC utterances involve a voiceless-voiced transition. Smooth, effortless voiceless-voiced transitions are attained when the vocal folds are positioned in the initial stages of speech initiation to prepare for onset of vocal fold vibration of the following vowel. For PWS, fluent transition during voice onset may be adversely affected when there is a breakdown in the preadjustments of the laryngeal-phonatory system due to an increased vocal fold tension, difficulty in pretuning of the vocal folds, or a breakdown in regulating subglottal air pressure. Wieneke *et al.* (1987) and Caruso (2000) claimed that fluent voice initiation is achieved by efficient coarticulatory speech movements during the preparatory period for $C_{(vl)}$VC productions (as well as $C_{(v)}$VC productions). The results for the stuttering speakers could therefore indicate that stuttering is characterized by reduced efficiency in the control of coarticulatory movements.

5.3.1.2 The nature of voice onset in persons who stutter (PWS) within a framework of speech sensorimotor control

In stuttering preparatory laryngeal adjustments might influence the manner of voice onset in two different ways. Firstly, the PWS's approach to voice onset might change in an attempt to control initial instabilities in speech motor activities and may present with a delay in laryngeal onset

time. On the other hand, the prephonatory irregularities might cause a breakdown in subsequent motor coordinations. Discoordinations may also be related to a general failure in the temporal coordination of speech motor activities that present with consistent disruptions in speech production. Although attracted to the idea to ascribe gradual voice onset to the first condition and abrupt voice onset to the latter, it would be regarded as superficial and biased. Manner of voice onset, in isolation, is not a reliable indicator of the underlying difficulties PWS experienced during voice onset. In an attempt to understand manner of voice onset, the important aspect is the behaviour sequence underlying abrupt/gradual voice onset in PWS.

Movement characteristics related to a disorder in motor programming, are associated with problems in movement onset, precise timing of coordinated movements and preparation of complex movements (Kent & Adams, 1989; Van der Merwe, 1997). The difficulties underlying gradual/abrupt voice onset in PWS seem to correlate with these symptoms associated with a disorder in motor programming. In accordance to Van der Merwe's (1997) view of motor programming – it is proposed that PWS present with a deficiency in making the appropriate adjustments to motor programs pertaining to movement rate, muscle tension and positioning prior to movement onset of a specific utterance.

Gradual voice onsets in the presence of preceding vocal fold irregularities reflect PWS's ability to control voice onset and is usually associated with a delay in movement onset. The abnormalities of motor coordination associated with abrupt voice onset in PWS are presumably related to a problem in the levels of coordinated activity of the many muscles cooperating for a specific function, such as those that set the position and tension of the vocal folds (Borden *et al.*, 1985). A small interruption in the interaction between muscle tension, posture and rate of movement could affect the coordination of speech motor activities involved in voice onset and might result in a fluency breakdown when time pressure is exerted.

5.3.2 *Discussion of sub-aim 2.2*: Voice onset time (VOT)

5.3.2.1 Voice onset time (VOT) of prevocalic voiced /b/ in $C_{(v)}VC$ productions

Control group: For voice onset of initial /b/ productions, the control group functioned within the normal range of VOT (i.e. -180 ms and +15 ms) as indicated by Zlatin (1974). VOT for /b/ utterances was characterized by prevoicing, which is a typical feature of voiced plosive productions for Afrikaans (Wissing & Roux, 1995). This result indicates that the control group was efficient in timing and coordinating the onset of vocal fold vibration and the release of the articulatory closure for /b/ productions. For voiced plosive productions in Afrikaans, vocal fold vibration may precede the release of the articulatory occlusion, or it may have a short interval between articulatory release and the onset of voicing. VOT for voiced /b/ productions of the control group therefore reflected normal speech behaviour.

Subject group: VOT results for /b/ productions indicated that the subject group performed similarly to the control group. VOT error values for /b/ utterances indicated similar results for the subject group (1.77 ms) and control group (1.28 ms). Thus, no apparent difference was found between the subject group and the control group for VOT of voiced plosive productions. Denny and Smith (2000) also showed that during perceptually fluent speech, VOT of voiced plosives in PWS corresponded with that of normal speakers. PWS seemed to be able to maintain relative consistency in the timing of voice onset for the prevocalic /b/ productions. In general, the PWS achieved temporal coordination of vocal fold adjustments and articulatory movements for voice onset of voiced /b/ productions within normal limits.

The subject group had greater VOT error values for /bɪt/ and /bɔt/ utterances compared to /bɑt/ and /bət/ utterances. These differences in VOT error values for specific /b/ productions suggest that the phonetic properties of the adjacent vowel might have influenced the VOT. This phenomenon was also seen in the control group, but to a lesser extent. Scobbie, Gibbon, Hardcastle and Fletcher (2000, in Bates *et al*., 2002) reported that VOT of the target consonant is influenced by the adjacent vowel. In other words, VOT seems to reflect the anticipatory coarticulation effect of the adjacent vowel on the initial consonant (Bates, Watson & Scobbie, 2002). Thus, differences in VOT error values for /bɑt/ and /bət/ productions compared to /bɪt/

and /bɔt/ productions might relate to the level of complexity of intergestural coordination. Since the vowels /ɑ/ and /ə/ require neutral positioning of the intra-oral structures, no active articulatory adjustments are needed after release of lip closure for /b/. Thus, the CV transition from /b/ to a central vowel (i.e. /ɑ/ or /ə/) mainly required the continuance of voicing which implies glottal adjustments in /bɑt/ and /bət/ productions. The greater articulatory demands of the high front vowel, /ɪ/ and the rounded back vowel, /ɔ/ could have resulted in more extreme articulatory configurations for the /b/ productions in the subjects. The increase in motor complexity for these utterances might have influenced VOT for the subject group. Greater VOT error values for $C_{(v)}VC$ utterances in the subject group may therefore indicate instances of mistiming or slowness in the coordination of articulatory gestures (including vocal fold gestures) for voice onset. Difficulties in temporal coordination for the subject group may relate to deviancies in antagonistic gestures of the articulatory system (including vocal folds) during speech initiation that result in less articulatory compatibility between the sound productions in the utterance (Van Riper, 1982). Slowness in VOT for the subjects can also be a motor strategy in the timing and coordination of articulatory gestures during speech initiation to maintain fluency, or it may result from innate differences in their speech motor planning/programming (Van Riper, 1982).

The individual subjects showed specific differences in VOT for /b/ productions from the control group. The VOT results for /b/ productions indicated that the three of six subjects were slower (less negative VOT values) than the normal speakers in the onset of voicing. Slower onset times can be associated with slower movements of the speech mechanism (Archibald & De Nil, 1999). Slower timing might result in movements having a higher degree of precision than those made at a faster rate (Archibald & De Nil, 1999). Subjects might have adopted a more careful task approach for /b/ productions in order to compensate for anticipated difficulty with the initiation of the speech task. This statement implies that movement speed decreased for the subjects as voicing complexity increased. The subjects probably used slower timing to maintain fluent speech production (Archibald & De Nil, 1999). This notion of a compensatory strategy for fluency is supported by the small VOT error values for /b/ productions in most subjects, which suggests consistency and repeatability in VOT.

The results further suggest that the timing for voice onset for most subjects differed from those of the controls with regard to the upper limit of VOT for /b/ productions. These subjects showed a

tendency towards greater positive VOT values for /b/ productions as opposed to the normal speakers. For instance, great positive VOT values (greater than +10 ms) were seen in more than 50% of the /b/ utterances for five of the six subjects. Thus, the majority of the subjects maintained slow voice onsets for voiced /b/ productions. This finding is consistent with research on VOT in stuttering and nonstuttering adults that shows stuttering adults take longer to initiate voicing than do nonstuttering adults, especially with regard to voiced productions (Metz *et al.*, 1979; Healey & Gutkin, 1984; Shenker & Finn, 1985). The greater VOT values for /b/ productions could indicate a slower release of the articulatory gesture before onset of vocal fold vibration. According to Stevens, Keyser and Kawasaki (1986), the more extended the articulatory contact area, the longer the VOT. This means that the release of the bilabial closure for /b/ may be slightly prolonged if there is lip frication involved. Alternatively, when lip contact pressure is too high due to increased levels of muscle tension, voice onset may be delayed for that utterance. Higher levels of muscular tension in the articulatory movements are characteristic to PWS (Van Lieshout *et al.*, 1993). Lower velocities and displacements and longer durations in the movements of PWS are associated with a breakdown in speech motor control (Zimmermann, 1980*a*).

The VOT for /b/ productions for the subjects overlapped with the lower limit of VOT for /p/ productions. The timing overlap may result in sound distortion of the /b/ production. A sound distortion does not imply purposely substituting one sound for another as compensatory measures during speech initiation, rather, it refers to a mistiming in sound production resulting in adjustments in the properties (such as voiced/voiceless) of that particular sound. Slower initiation of voicing may also be due to slightly aspirated voiced plosive productions. Slightly aspirated voiced production occurs when vocal fold vibration begins after the release of the articulatory occlusion (Shibles, 1995). Aspiration is not characteristic to voiced plosive production in Afrikaans, though its occurrence in English production is a normal feature (Wissing & Roux, 1995). The subjects might have used slight aspiration as a compensatory gesture for easy, gradual voice onset in order to avoid a breakdown in speech initiation.

Despite their perceived fluency during /b/ productions, speech initiation in most subjects seemed to contain irregularities and brief time lags related to difficulties in the coordination, timing and overall control of speech motor activities. In cases where the PWS showed indications of

difficulty in quickly initiating voicing, irrespective if they occurred occasionally or frequently, heightened muscular tension might have been a product of a failure to produce rapid, accurate speech movements. Depending on the frequency of occurrence, the long-lag in VOT of the PWS could have been either momentary incidences of a lack of timing control, or regular incidences characteristic to their speech behaviour. However, in the current study the small time lags and asynchronies in VOT did not result in an audible (overt) breakdown in speech production. The subacoustic nature of the aberrancies in the stuttering speakers' speech behaviour might have been attributed to the simplicity of the speech task. Subtle articulatory mistimings in the coordination of speech movements in perceptually fluent speech have been reported for PWS (Archibald & De Nil, 1999). More complex speech tasks, such as faster, longer or unfamiliar utterance could have resulted in audible disfluent speech.

In comparison to the group of normally fluent speakers, 50% of the subjects showed highly consistent VOT for /b/ productions. A small difference between the maximum and minimum VOT values for these voiced plosive productions existed. The limited VOT range could suggest stiffness in speech movements which can result in restricted velocity in movement (Archibald & De Nil, 1999). Reduced movement velocity may be related to higher levels of muscular tension. Previous studies on stuttering have indicated excessive muscular tension in the larynx and the presence of disruption of reciprocity in laryngeal abductor and adductor muscles in PWS (Freeman, 1979, Freeman & Ushijima, 1978; Shapiro, 1980; Van Lieshout *et al*., 1993). Higher levels of activity in articulatory movements have also been reported during perceptually fluent utterances (Adams *et al.,* 1984). Research showed abnormal orbicularis oris activity, abnormal activity of the cricothyroid muscle and abnormal activity of the posterior cricoarytenoid muscle prior to speech initiation (Shapiro, 1980).

Marked idiosyncratic features in VOT for voiced /b/ productions were noted for a single stuttering speaker, namely, Subject 1. This subject consistently showed negative VOT for /b/ productions. A tendency to initiate vocal fold vibration prior to articulatory release during voice onset is indicated hereby, which might be associated with prephonatory vocal fold abnormalities prior to voice onset. Irregular vocal fold activity were evident in the period before the onset of voicing in the $C_{(v)}VC$ context for this subject. In view of this finding, frequent occurrence of negative VOT for /b/ productions might suggest the presence of heightened glottal tenseness or

an inability to control vocal fold activity in coordination with articulation. Alternatively, prevoicing could have been employed to reduce the complexity of interarticulatory coordination in order to establish fluency. Prevoicing in this regard may have been used as a compensatory strategy to control fluent speech production.

A possible relation between stuttering severity and VOT for /b/ was implied by the results. The subjects with severe stuttering involvement seemed to have differed from the mild stuttering speakers with regard to VOT range for /b/ productions. An explanation for the small VOT range for /b/ productions suggests that speakers with severe stuttering experienced stiffness of their phonatory-articulatory movements. This possibility was also mentioned by Archibald and De Nil (1999). These researchers suggested that speakers with very mild and moderate-severe stuttering may differ in the stiffness of their articulatory movements. Further investigation is needed to verify whether such relationship exists.

5.3.2.2 Voice onset time (VOT) of prevocalic voiceless /p/ in $C_{(vl)}VC$ productions

Control group: VOT of /p/ productions for the control group was within normal limits ranging between +10 ms and +150 ms (Lisker & Abramson, 1964 in Cho & Ladefoged, 1999). This group showed a tendency toward shorter timing in voice onset, with an overlap of the upper limit of VOT for /b/ productions. The voicing feature of the vowel is anticipated during the production of the preceding voiceless consonant. The duration of the adjacent vowel seemed to have influenced the coarticulatory gesture for /p/ productions. According to Oosthuizen (1989) and Kent (1993) a shorter lag in VOT due to the influence of the adjacent sound reflects normal speech behaviour. The voicing of consonants preceding a vowel as for English has been documented (Kent, 1993). In Afrikaans, both voiced and voiceless plosive, are produced with a short VOT (Oosthuizen, 1989).

Subject group: The subject group showed similar mean VOT for /p/ productions than the control group with no marked difference in VOT error value between the groups. This finding is consistent with VOT data on voiceless stops that reported no significant difference between the stuttering speakers and the normal speakers during perceptually fluent speech (Watson & Alfonso, 1982; Healey & Gutkin, 1984; Borden *et al.,* 1987; Denny & Smith, 2000). The

absence of between-group differences in VOT for /p/, however, does not imply normal speech production for the subject group. Peters and Hulstijn (1987) stated that the coordination and timing of laryngeal and articulatory processes are not realized in the same way in PWS as in normal speakers. Speech movements are precisely timed with some movements being separated by about 10 ms (Kent & Moll, 1972) - therefore, small differences in VOT values for individual subjects might be of significance when co-occurring with movement irregularities.

When reviewing the results of the individual subjects, three of the six PWS had smaller VOT values for /p/ productions compared to the group of normally fluent speakers. This could indicate difficulty in coordinating vocal fold behaviour with articulatory movements for voiceless-voiced transitions in $C_{(vl)}VC$ productions. Katz (1987) reported that coarticulatory abnormalities in speakers with motor speech disorders (such as stuttering) are related to the specific motor and timing requirements of the utterance. Abnormal coarticulation is most likely to occur in phonetic sequences that require the coordination of two or more independent articulators (such as the lips and vocal folds for /p/ productions) (Katz, 1987). Great contrasts in the distinctive features of the voiceless plosive /p/ and the following (voiced) vowels for $C_{(vl)}VC$ productions are present. For example, the vowel /*a*/ is formed at the back of the mouth and involves the complete opening of the vocal tract (Borden & Harris, 1984). The voiceless /p/, on the other hand, is formed in the front of the mouth and involves the closing of the vocal tract (at the lips) (Borden & Harris, 1984). Voiceless /p/ productions require virtually no vocal intensity, while voiced /*a*/ require maximum amount of vocal intensity during production. In order to limit or reduce the contrast between the voiceless-voiced transition, the voiceless /p/ might have been voiced to match the voicing feature of the adjacent vowel. This means that voicing could have assimilated progressively during the initiation of prevocalic /p/ productions for some PWS. Therefore, VOT of the initial /p/ utterance within the $C_{(vl)}VC$ context for these stuttering subjects might indicate that coarticulatory overlapping occurred to such an extent that the distinctive features of the voiceless /p/ was equalized with phonetic features of the following sound.

In addition to the short time lags of VOT, most subjects showed a greater degree of overlapping in VOT of /p/ productions with /b/ productions compared to the group of normal speakers. The overlapping in timing of voice onset might have resulted in imprecise plosive productions, in which case the voiceless /p/ could have been perceived as the voiced /b/. Perhaps this

overlapping of VOT for /p/ productions in the stuttering speakers was attributed to an inability to establish voiced-voiceless contrast, which resulted in voicing inaccuracies. Rezaei-Aghbash, Whiteside and Cudd (1999) suggest that deviancies in the timing and coordination of phonatory-articulatory movements for voice onset may be involved in faulty voicing contrasts in PWS. It is unclear whether abnormally short VOT for /p/ in stuttering speakers is an indication of a compensatory strategy used for anticipated difficulties in phonatory-articulatory coordination, or an indication of problems in controlling voiced-voiceless contrast during speech initiation; however, both these explanations seem to relate to irregularities in speech motor timing, coordination and/or control during speech initiation.

Idiosyncratic features for voice onset of /p/ productions were evident in one subject (S1) that showed a delay in VOT. The long-lag VOT for this individual who stutters was in contrast to the shortened VOT for /p/ productions in the majority of the subjects. However, long-lag VOT occurred in only a few utterances for this individual subject. These single instances of irregularities in VOT may be the result of momentary failures in preparation of voluntary speech movements (i.e. /p/ production) (Phillips *et al.*, 1989). A possibility is that the individual's lips were constricted or narrowed as a compensatory gesture for these utterances, instead of the definite lip closure as normally required for the production of the bilabial /p/. If this was the case, the /p/ could have been produced with a constriction of the lips resulting in the production of the voiceless bilabial fricative, /θ/. According to Stromsta (1986), intra-oral pressure might be minimized by lack of complete articulatory occlusion in the presence of prolonged voiceless stop plosives. Some evidence exists relating reduced levels of labial muscle activity with stuttering (Stromsta & Fibiger, 1980 in Conture, 1987; McClean *et al.* 1984).

A delay in the onset of voicing can also be associated with inefficiency of aerodynamic function relating to laryngeal and/or articulatory activities during speech production. If the difference in air pressure across the vocal folds is inadequate, there will be no dynamic airflow to set the vocal folds into vibration, or if the air in the oral cavity is at a pressure similar to that below the vocal folds, vocal fold vibration might be delayed due to the lack of dynamic airflow. Starkweather (1982) suggested that longer VOT for /p/ might be attributed to reduced subglottic pressure and weak airflow. For the production of the initial /p/, an oral occlusion would be of no value if the appropriate air pressures had not been generated beforehand as to create a basis for airflow. A

shortage of airflow would heighten the stress condition on the speaker's speech production system for acoustic energy needed for the following vowel in $C_{(vl)}VC$ utterances. Instabilities in the control of respiratory-related pressures in perceptually fluent speech of PWS have been documented (Peters & Boves, 1988; Peters *et al.*, 1993). In addition, VOT may be delayed when contact pressures of the articulatory closure (i.e. adducted vocal folds or closed lips) are too high to be released by the airstream (Freeman, 1979; Shapiro, 1980). An abrupt or sudden voice onset may correspond with the delay in voice onset for an individual stuttering speaker which suggests heightened muscle tension (Peters & Boves, 1988). In general, the long-lag voice onset for some /p/ productions in this stuttering speaker suggest deviancies in the temporal coordination of respiratory, phonatory and articulatory activities with associated variability in muscle tension in the speech production system.

Most subjects showed great consistency for absolute VOT values of /p/ productions. For instance, two stuttering speakers (S2 and S6) had abnormally short lags in VOT for /p/ productions compared to the other PWS and the normal speakers. The magnitude of their VOT range for /p/ was also relatively restricted**,** which reflects that their absolute VOT values were highly consistent. According to Archibald and De Nil (1999), the restricted range in the timing interval of movements is associated with increased muscle tension in speech movements. Abnormal tension in articulatory and laryngeal muscles in PWS's perceptually fluent utterances has been reported by various kinematic studies (Freeman, 1979; Zimmermann, 1980*b*; Shapiro, 1980; Jaencke, Bauer, Kaiser & Kalveran, 1997; Archibald & De Nil, 1999). Thus, inadequate reciprocal adjustments of the antagonistic muscles may result in a reduced range of articulatory movements. The increase in muscle tension could reflect articulatory rigidity (or stiffness) in the speech production mechanism of PWS (Van Lieshout *et al.*, 1993; Archibald & De Nil, 1999). The degree of muscle rigidity and/or restrictions in the range of movements for PWS could have been differentially involved in the upper and lower lips, tongue, jaw and vocal folds.

Another possibility is that the reduced range in the timing of the target sound, /p/, in PWS is a compensatory strategy to inhibit excessive involuntary movements. Thus, limited variation in articulatory-phonatory coordinative activities may result from the PWS's efforts to control the muscle tension of those speech structures involved. In such a case, restricted articulatory and/or laryngeal movements suggest that PWS have a reduced ability to adapt to movement variables.

Starkweather (1987) suggested that the breakdown in coordination of the articulatory and laryngeal systems becomes more apparent as their movement complexity increases; for instance, speech initiation with voiceless-voiced transitions in $C_{(vl)}$VC context as opposed to voiced-voiced transitions in $C_{(v)}$VC context. Consistency in speech movements might have been maintained to control speech fluency. This could be a compensatory gesture by the stuttering speakers to stabilize the speech production system (Robb, Blomgren & Chen, 1988; Jaencke *et al.*, 1997; Archibald & De Nil, 1999). In other words, those PWS showing restricted VOT ranges seem to have maintained stability in speech motor control by reducing the range of articulatory and/or phonatory movements. Reduced VOT ranges for /p/ in PWS can therefore be attributed to a learned motor speech control strategy to compensate for movement complexity, or to a primary difference in motor speech control reflected in heightened muscle tension and rigidity in articulatory and laryngeal movements. Limitations in the range of speech movements, whether intentional or involuntary, are associated with difficulty in the initiation of movements.

Interesting to note is that the stuttering speakers with restricted VOT ranges for /p/, showed a higher percentage of gradual voice onset as opposed to abrupt onset. Gradual voice onset is associated with light articulatory contact of the upper and lower lips, in other words, reduced tension in the lip muscles (Borden *et al.*, 1985; Dembowski & Watson, 1991*a*). In view of the fact that the PWS received previous therapy, gradual voice onsets with highly consistent VOT could indicate that a compensatory strategy was used to control voice onset for $C_{(vl)}$VC productions.

The apparent consistency of VOT for /p/ productions as seen in individual subjects was in great contrast to the variability found in their timing of prephonatory vocal fold activity (see 5.2.2.1). Less variability in the temporal coordination between articulatory and laryngeal gestures might be indicative of better speech motor control during voice onset, than prior to voice onset. .Thus, the VOT results for /p/ utterances of the current study support Metz *et al.*'s (1983) notion that the perceptually fluent speech of PWS is not fully "normal". Thus, although these utterances were perceptually fluent, they did indicate subtle abnormalities compared to normal fluent speech production. These PWS seem to have experienced problems with effective speech motor control during the initiation of speech.

5.3.2.3 Effect of voiced and voiceless plosives on voice onset time (VOT) in normal speakers and persons who stutter (PWS)

Control group: The control group showed VOT range values for /b/ and /p/ productions that were within the normal limits as accepted by previous studies (Zlatin, 1974; Lisker & Abramson, 1964 in Cho & Ladefoged, 1999). The control group also showed a tendency toward negative VOT for /b/ productions. Available VOT research on voiced-voiceless contrast for Afrikaans is limited (Meyer, 1993). Since many of the Germanic languages such as Dutch show similar voice onset features to Afrikaans (Wissing & Roux, 1995), VOT research on these Germanic languages (especially Dutch) has been used to explain voice onset features in Afrikaans. Oosthuizen (1989) as well as Wissing and Roux (1995) stated that both voiced plosive consonants and voiceless plosive consonants in Afrikaans are within the short VOT range and overlapping in VOT values exist in normal speech production. In Germanic languages such as Dutch voiced stops are produced with prevoicing and voiceless stops are produced with a short-lag VOT without aspiration (Jansen, 2004; Lisker & Abramson, 1964 in Cho & Ladefoged, 1999). Based on findings by Wissing and Roux (1995), the tendency of the control group toward negative VOT for prevocalic /b/ productions can be regarded typical speech behaviour in Afrikaans. The VOT results for the control group seemed to have reflected normal speech behaviour for /b/ and /p/ productions during speech initiation.

Important to note was the clear difference in VOT range values between /b/ productions and /p/ productions. In other words, the maximum VOT for /b/ productions did not overlap with the minimum VOT for /p/ productions. A voiced-voiceless distinction between /b/ and /p/ productions was indicated by these results. This finding suggests that the normal speakers showed a voiced-voiceless contrast within a given phonetic context (CVC). However, Docherty (1992) and Shibles (1995) stated that VOT is not a useful measure to indicate voiced-voiceless contrast. Indications exist that VOT depends on the dynamics of the phonetic context in which the target sound occurs (Docherty, 1992). VOT is not only the result of speech physiology (as determined by the phonetic context), but is in part determined by language specific rules (phonology) (Docherty, 1992). Voicing contrast involves more than only VOT. Shibles (1995) reasoned that consonants should be regarded as only partially voiced or partially voiceless, which

will cause difficulty in describing the degree of voicing within the dynamic speech production process.

According to Ball and Jones (1984) as well as Shibles (1995) it is not the voiced-voiceless feature for /b/ and /p/ that distinguish these two sounds from each other. For instance, /p/ may be produced from air captured within the oral cavity which, when released, makes a strong burst and results in a short-lag VOT. Little difference may be found in VOT between /b/ and /p/ when followed by the same vowel sound (Shibles, 1995). Additional features such as intervocalic/vowel duration must be used to contrast /b/ from /p/. Shriberg and Kent (2003) found that for all languages, vowels before voiced consonants have longer duration than if before voiceless consonants. Thus, /b/ versus /p/ distinctions may be regarded as prevoiced aspects of vowels (Shibles, 1995).

Existing norms for VOT for /b/ productions and /p/ productions were used as guidelines to explain VOT for the control group. The VOT results of the control group were comparable with normal speech behaviour. However, comparing the VOT results for /b/ and /p/ productions of the subjects with the normal limits for VOT presented inconclusive findings**.** Since the VOT results for /b/ and /p/ productions were obtained within a given phonetic context for the subjects (and the control group), it is likely that the phonetic context influenced the subjects' speech behaviour. For that reason, the discussion of the effect of voiced and voiceless plosives on VOT of the subjects was based on the voice onset activity of the control group**.** VOT results of the control group were therefore used as representative of normal speech behaviour in an attempt to explain the VOT in the subjects.

Subject group: Despite similarities in the VOT of /b/ and /p/ productions between the subjects and control group, it seemed that the coordination or timing of laryngeal and articulatory processes was not realized in the same way in the subjects as in the control group. The majority of the subjects showed more deviancies in voice onset of /b/ productions as opposed to /p/ productions. In both /b/ and /p/ productions a tendency was seen that involved the overlapping of VOT limits of voiceless and voiced plosives, respectively. For b/ productions, a tendency was seen in exceeding the upper limit of +15 ms. For /p/ productions, the subjects showed smaller minimum VOT values than the control group that overlapped with the upper limit for /b/

productions. Thus, the VOT results for the PWS failed to indicate voiced-voiceless contrasts. Voiced-voiceless contrast in the initial plosives /b/ and /p/ requires the precise timing and coordination of the opening of the articulatory occlusion and the start of voicing (Abramson, 1977; Forrest & Rockman, 1988). This precision in temporal coordination seemed to be lacking in the subjects.

Based on the VOT results, indications of deficiencies in timing of the release of the articulatory closure and the initiation of phonation for /b/ and /p/ productions within a given phonetic context were evident for the subjects. According to Rezaei-Aghbash *et al.* (1999), PWS may produce voiceless plosives with a very short-lag VOT as is usually found in voiced plosives and conversely, they may produce voiced plosives with a long-lag VOT which is characteristic of voiceless plosives. Inadequate timing between voice onset and articulatory release may result in sound distortions or misarticulations (Kent & Netsell, 1975; Kent *et al.,* 1979). Perhaps voicing substitutions, as seen in most subjects, were due to too quick release of the articulatory occlusion or too quick initiation of voicing. This VOT feature of PWS is more prominent during stuttered speech (dysfluencies) (Rezaei-Aghbash *et al.,* 1999). In other words, the voice onset of PWS is not necessarily slow or delayed, but can be characterized by mistimed phonatory-articulatory movements. Poor timing in coarticulation and overadaptation from one articulatory movement to another for speech production may result in the quick initiation of voicing. Starkweather (1982) stated that PWS's initial sound production might be more affected by subsequent sounds in a given phonetic context than the initial sound production of normal speakers.

In three of the six subjects, /b/ productions and /p/ productions showed error values for VOT range in comparison to the control group. Similar to findings by Healey and Gutkin (1984), these subjects had greater differences in VOT range for /b/ productions than for /p/ productions. For prevocalic /b/ productions, most subjects indicated slower and more variable VOT compared to the control group, although it was still within normal range. Jaencke (1994) and Denny and Smith (2000) also showed enhanced variation of VOT in PWS during perceptually fluent speech. For /p/ productions, most subjects had restricted VOT range values. This may imply that VOT for voiced /b/ productions reflected more variability, while /p/ utterances were produced with higher consistency. The differences in VOT for voiced and voiceless productions in a CVC

context for the individual subjects, may be attributed to the motor demands of the phonetic context on the speaker's speech motor control system.

In general, VOT of the subjects showed slowness for /b/ productions and a restricted VOT range with consistency for /p/ productions. VOT for /b/ and /p/ productions of PWS showed an overlap of time limits, which is indicative of indistinctive voiced-voiceless contrasts. The subjects showed clear evidence of mistiming in articulatory and laryngeal activities during speech initiation of /b/ and /p/ productions. The different VOT features for /b/ and /p/ productions seemed to be associated with the motor complexity of the utterance. The motor complexity of the phonetic context against the speech rate seems to determine the extent to which this disruption is acoustically perceptible (Starkweather, 1982). In addition, the VOT findings underscored findings by Winkler and Ramig (1986) that the articulatory-laryngeal functioning of PWS differs from that of the normal speakers during perceptually fluent speech.

The discussion on VOT of stuttering speakers supports Bates, Watson and Scobbie's (2002) view that VOT is not a cue for the target voicing status of the initial consonant. VOT can perhaps be seen as a progressive response to coarticulatory overlapping which is dependent on both articulatory compatibility and the mechanical constraint imposed on the articulators (such as the tongue and vocal folds) during speech production. The articulatory compatibility and mechanical constraint are determined by the articulatory configuration and articulatory distance for the specific utterance.

5.3.2.4 Voice onset time (VOT) of voiced and voiceless plosives in persons who stutter within a framework of speech sensorimotor control

The apparent similarities in VOT results between the PWS and the normal speakers are briefly discussed in order to highlight the influence of different motor demands of the speech task on speech motor control. A detailed discussion of VOT features in the PWS as possible indicators of a deficient speech sensorimotor control system follows next.

The lack of differences in VOT between the PWS and the normal speakers might be related to the articulatory demands of the speech tasks used in this study. Several studies (Reich *et al.*, 1981;

Watson & Alfonso, 1982; Till *et al.*, 1983; Winkler & Ramig, 1986) suggested that speech timing in PWS may be influenced by the motor demands and complexity of the response. Winkler and Ramig (1986) found that timing difficulties were not seen in simple speech tasks, but notable in complex tasks. Simplified speech tasks, such as CVC productions, seem to require less motor processing time and may serve to equalize disparate speech-timing abilities of PWS and normal speakers (Healey & Adams, 1981). Longer utterances, as opposed to shorter utterances, are motorically more complex and may show greater difficulty in the initiation and controlling of speech activities. Longer more complex speech tasks are related to slower or more variable VOT. For the stuttering speakers, it can be argued that if the speech tasks of the current study required higher motor demands, longer onset times associated with greater variability in motor speech timing control would have been evident.

Most PWS showed a tendency to slowness in timing during voice onset for voiced /b/ productions, while voiceless /p/ productions were associated with possible stiffness in movements. This behaviour may indicate subtle difficulties in controlling and stabilizing the temporal coordination between supraglottal and laryngeal structures (Boutsen, 1995). Below the threshold of awareness, PWS's perceptually fluent speech contains small time lags and asynchronies that may be related to difficulties in motor organization, timing and control (Zimmermann, 1980*c*). Perhaps these deficiencies in timing manifest as (episodic) problems in voiced-voiceless contrast, irregularities in the magnitude of VOT range and/or VOT deviancies particularly as the complexity of speech production increases. According to Kent (1976), variation in VOT should decrease with speech motor maturity as an indication of stability in speech production.

If accuracy in timing of movements is an indicator of speech motor stability, then timing inaccuracies reflect a breakdown or deficiency in a control system. Durational precision of speech movements depends on neuromuscular skills, but also on the durational and articulatory demands of the utterance (Crystal & House, 1988). In this regard, the duration of VOT reflects two aspects of speech rate: the speed of articulatory movement, and the extent of coarticulatory overlapping (Starkweather, 1982). The speed-accuracy trade-off in PWS's speech movements is related to the motor complexity of the utterance (Archibald & De Nil, 1999). Slower movements may have been related to enhance precision of motorically complex utterances, while less

complex movements may have resulted in faster movements (Archibald & De Nil, 1999). Long VOT, which represents slower speech movements, was seen in single /b/ productions in the PWS. Slower movements might have enabled better motor speech timing control and therefore accuracy during these productions of the PWS. Since long-lag VOT in PWS is associated with more variability (Macken & Barton, 1980), these movements were possibly depicted by subtle aberrancies despite perceptually fluent speech.

In the following discussion possible reasons for long-lag VOT of /b/ productions and highly consistent VOT of /p/ productions in PWS will be presented from a speech sensorimotor control perspective. These abnormalities in VOT of PWS may suggest an inability to regulate speech movements effectively with regard to range, duration, force and rate. This inability could result in inappropriate muscle tone and postural adjustments required for the initiation of a specific movement which may point to a breakdown in motor programming according to the framework of speech sensorimotor control proposed by Van der Merwe (1997). According to this model, interarticulatory synchronization (e.g. VOT) is related to motor planning (Van der Merwe, 1997). However, motor programming disorders, such as dysarthria associated with cerebellar disease, also show VOT errors which indicate deviancies in the accuracy and range of articulatory movements, possibly as a result of disturbances in muscle tone and postural control (Erasmus, Van der Merwe & Groenewald, 1993).

The first possible explanation for VOT errors in PWS suggests that increased variability of speech movements or a breakdown in temporal precision of speech movements can be related to reduced efficiency or instability of the neuromotorsystem in PWS (Adams, 1981; Archibald & De Nil, 1999). More specifically, this behaviour results from instability of the system that compares and integrates the feedforward and feedback information (Max, Guenther, Gracco, Ghosh & Wallace, 2004). According to Max *et al.* (2004:25): “If consequences of the prepared commands cannot be accurately predicted based on an efference copy and concurrent afferent inflow, a mismatch may arise between the predicted and actual consequences of the executed movement. As a result of such a mismatch the CNS may respond by re-attempting the movement and re-issuing the central commands until the sensory consequences are interpreted as matching the desired consequences, sustaining the already ongoing commands until the conflict is resolved or avoided by relying on moment-to-moment afferent feedback, or generating a different set of

commands." This process of repeated repairs in search for an efferent-afferent match relates to Adams' view (1981) that PWS may be slow to organize and forward normal motor commands to specific sets of muscles. Prolonged or repeated muscle contraction may be evident which is associated with greater variability of speech movements or temporal inaccuracy of speech movements. Indications of extreme variability (or impedance changes) such as wide postural excursions of either too much adduction or too much abduction to allow effective voice initiation have been reported.

In order to inhibit the occurrence of maladaptive responses to mismatches between predicted and actual movement consequences, the speech motor control system of PWS may use a motor strategy that involves longer durations (Max *et al.*, 2004). Considering there is always a time lag between a motor command and its auditory and somatosensory consequences (Kehoe 1997*a*), then PWS's preference for longer movement duration during speech movements could be attributed to the fact that the slower movements, compared to faster movements, are less affected by the delays associated with afferent input. Longer movement durations allow more time for the processing and integration of afferent inputs (Jeannerod, 1997). The speech motor control systems of PWS are thought to rely heavily on afferent feedback for the temporal coordination of complex goal-orientated motor behaviours that includes making subtle adjustments during the completion of movements (Jeannerod, 1997; Max *et al.*, 2004). Slower movements observed in stuttering speakers may represent a preferred motor strategy for complex sequential multi-articulatory movements, rather than physiological limitations in the speed of movement execution (Archibald & De Nil, 1999; Max *et al.*, 2004).

Secondly, in addition to slower integration and forwarding of movement commands, PWS may also send inappropriate commands to the periphery as suggested by Adams (1981). Speech musculature (including respiratory, laryngeal and/or articulatory muscles) could be activated or inhibited by such inappropriate commands in ways that could influence voice onset (Adams, 1981). Thus, the presetting of muscle groups for speech initiation may be affected in PWS. Presetting facilitates or inhibits the excitability of certain motor neurons, and thereby modifies the relationship among muscle groups and determines the nature of their behaviour (Max *et al.*, 2004). In this regard, a specific group of muscles may fail to function as a coordinative structure when triggered by inappropriate afferent signals (Jeannerod, 1997; Max *et al.*, 2004). Inefficient

control of the level of excitation of the muscle group may, in turn, cause inappropriate muscle activity (Jeannerod, 1997; Max *et al.*, 2004). This implies maladaptive changes in the movement properties of the coordinative structure. The motor properties are normally manipulated to promote flexibility and adaptiveness in the coordinated activities for speech production (Max *et al.*, 2004). In the case where either the triggering input or the actual tuning (i.e. presetting of the muscle groups) is aberrant, the speaker will be less efficient in adapting to the movement requirements of the intended motor goal. For example, motor commands may cause an abnormal increase in the muscle tone or the degree of contraction in the intrinsic laryngeal muscles that determine glottal width. This could lead to too much adduction or too much abduction for voice onset which may delay voice onset time. EMG studies on reciprocity in laryngeal abductor and adductor muscles in stuttering speakers by Freeman (1979), Freeman and Ushijima (1978) and Shapiro (1980) support such a possibility.

Thirdly, the sensorimotor control system of the stuttering speaker seems to have a limited capacity or inefficiency to process and integrate afferent input with motor commands (Max *et al.*, 2004). PWS's movements are less automatic and more dependent on feedback systems (Kehoe, 1997*a*,*b*; De Nil *et al.*, 2001), and/or they might have weak feedforward projections and an over-reliance on afferent feedback (Kehoe, 1997*a*,*b*; Max *et al.*, 2004). This inefficiency in sensory or motor monitoring will affect the ability of the speech motor control system to adapt to a specific contextual variable for speech production. Any changes in a contextual variable may cause instability in the functioning of the feedback controller. For instance, PWS might be very rigid in their timing of speech movements for productions of simple sound structures, to ensure accuracy. The desired movements for CVC utterances are also simpler to predict as opposed to more complex utterances. When demands on the speech sensorimotor control system for predicting movements decrease, there is less dependency on feedback control to match predicted outcomes and actual outcomes (Kehoe, 1997*a*). The sequence of speech movements for the utterance becomes a more automatic response and may increase in precision and speed. Thus, the timing for repeated productions of the same utterance is expected to be relatively consistent with greater movement accuracy. This view seems to be supported by the consistency of VOT for /p/ productions of some stuttering speakers. Consistency in VOT for PWS can be associated with heightened muscle tension or rigidity in the range of speech movements for particular utterances. Increased muscle tension, in turn, may relate to stuttering speakers' effort to predict and produce

accurate movements. Indications of abnormal stiffness in articulatory gestures of PWS (Archibald & De Nil, 1999) during voice initiation add further support. The PWS might have used a learned approach for the speech productions in order to control accuracy. If this should be the case, these PWS's ability to quickly adapt to different stress conditions might be limited or reduced. A breakdown in the speech production of PWS is expected in the presence of increased contextual demands on the speech sensorimotor control system.

5.3.3 Summary: Acoustically detected vocal fold activity during speech initiation in persons who stutter (PWS)

Acoustically detected vocal fold activity during speech initiation did not reveal apparent differences between the group of stuttering speakers and the group of normally fluent speakers. Irregularities in acoustically detected vocal fold behaviour were not as prominent as physiological pre-laryngeal activity during speech initiation. A breakdown in temporal coordination of PWS occurs during the preparation of speech production, which might be acoustically detectable during voice onset depending on the PWS's ability to compensate and the motor complexity of the utterance. Thus, acoustically detected voice onset features in PWS can be regarded as a by-product of subsequent physiological aberrancies. This is confirmed by Borden *et al.*'s (1985:371) statement "that the timing of laryngeal-supralaryngeal coordination is not the parameter at fault in PWS; rather it may be that levels of the laryngeal activity previous to voice onset are faulty".

The nature of voice onset showed that both the normally fluent speakers and the stuttering speakers managed to produce gentle voice onsets for /b/ and /p/ speech productions. This could indicate that the PWS articulated with a deliberately reduced articulatory effort level. In the gentle voice onset condition, the group of PWS could have changed the underlying physiologic processes preceding the acoustically detected voice onset behaviour. Gradual voice onsets for speech utterances of the group of normal speakers were preceded by well-organized laryngeal-phonatory activities, while irregular prephonatory vocal fold behaviour preceded the gradual voice onsets for PWS. The high incidence of abnormal prephonatory vocal fold behaviour in CVC productions suggests that these productions for PWS were not preceded by smooth, regulated preparatory speech movements.

Voice onset time of /b/ and /p/ productions for the two groups of participants, PWS and the normal speakers, were similar. The fact that group differences in VOT scores were not revealing, does not imply that these results are meaningless. In other words, "the absence of evidence for a phenomenon is not the same as evidence for the phenomenon's non-existence" (Starkweather, 1982:28). VOT ranges for voiced and voiceless plosive productions overlapped to a great extent for the PWS. These results suggest that the PWS were more deficient in the control of voiced-voiceless contrasts as opposed to the normal speakers.

Contextual factors such as complexity of the utterance, length and speech rate seemed to have influenced the VOT results for the normal speakers and the stuttering speakers. When evaluating the results qualitatively, it might be reasoned that the subject group used a different strategy in voice onset than the control group in order to accomplish the same acoustic output. If high stress conditions are inflicted, however, such as increased task complexity or speaking rate then PWS may show greater differences in VOT when compared to the group of normal speakers.

Individual stuttering speakers showed idiosyncratic behaviour with relation to voice onset that could be indicative of a breakdown in temporal coordination in speech initiation. VOT for /b/ and /p/ productions for the individual stuttering subjects might have been related to poor timing of muscle action or abnormal high muscle tension in their speech production mechanism. Both explanations suggest that individual stuttering speakers failed to control the temporal dimensions of prevocalic voiced and voiceless plosive productions during voice onset.

Abnormalities in VOT of stuttering speakers relate to an inability to regulate speech movements effectively with regard to range, duration, force and rate. Muscle tone and postural adjustments required for the repeated initiation of a specific movement might be adversely affected hereby. These speech motor characteristics can be associated with a deficiency in speech sensorimotor control; more specifically the level of motor programming is indicated.

5.4 CONCLUSIVE REMARKS ON THE RESULTS FOR PHYSIOLOGICAL AND ACOUSTICALLY DETECTED VOCAL FOLD ACTIVITY DURING SPEECH INITIATION

The present study focused on physiological speech behaviours that preceded voice onset (or prephonatory speech activities) and acoustically detected vocal fold activity during speech initiation in six stuttering speakers and six normal speakers. Current findings suggest a breakdown in PWS's prephonatory speech activities that involves a disruption of motor speech timing control of respiratory-phonatory-articulatory activities. The results emphasized that the major delay in speech initiation in PWS is located in the earliest parts of the response. The sequence of following responses during voice onset may involve stiffness in speech movements, static vocal tract positions, or disturbances in muscle tension in speech activities. The results of this study support the notion by Boutsen (1995) that there are physio-acoustic differences, specifically relating to the prephonatory activities, in the fluent speech of PWS and normal speakers.

Within a framework of motor programming, the results of this study suggest that stuttering could involve disruptive effects in the temporal synchronization of vocal fold adduction and abduction with rapid changes in respiratory and articulatory activities inherent to speech production, or deficits confined to the motor control of the musculature of specifically the larynx. Both these explanations for aberrant laryngeal behaviour in stuttering implicate a breakdown in the motor programming of complex speech motor activities that involves force, degrees of movement range, rate, and temporal accuracy during the initiation of successive movements. The possibility exists that certain PWS have a tendency towards inefficient or unstable motor control of the vocal fold musculature involved during speech initiation, and others a more generalized deficit in timing of coordinated respiratory-phonatory-articulatory activities.

5.5 SUMMARY OF CHAPTER 5

The discussion and interpretation of the results of this study are presented in Chapter 5 with reference to relevant research on vocal fold dynamics and speech motor control in stuttering. The discussion focused on physiological and acoustically detected vocal fold behaviour during speech

initiation in PWS and normal speakers. Speech motor behaviour, with special reference to vocal fold behaviour, were considered in terms of different phonetic contexts (VC, $C_{(v)}$VC and $C_{(vl)}$VC). Interpretations of the results supported by/in association with/accordance with previous research on stuttering were based on theoretical explanations for normal and atypical speech production. The conclusion of the study will follow in Chapter 6 and indications for future research will be given.

CHAPTER 6: CONCLUSION

6.1 INTRODUCTION

The study has focused on physiological and acoustically detectable vocal fold activity during speech initiation in persons who stutter (PWS). Results indicated abnormalities in the timing and coordination of laryngeal activities in the perceptually fluent speech production of PWS. Vocal motor activities in PWS showed distinctive aberrancies during the prephonatory phase of speech initiation. However, the laryngeal system (vocal fold activity) functions in coordination with the articulatory and respiratory systems for speech production, which implies that the breakdown in temporal coordination for PWS involves the speech motor control system (i.e. laryngeal, respiratory and articulatory processes). The inquiry into the breakdown of speech motor control during speech initiation in PWS supported previous researchers' suggestion that stuttering is related to a motor programming disorder. The emergent awareness of the relation between speech motor characteristics of PWS (such as aberrant prephonatory vocal fold activity) and neurological functioning will enable researchers to refine theories on the nature of stuttering.

Chapter 6 entails the conclusion of this study, as well as an evaluation of the research methodology. This is followed by recommendations for further research and a discussion of the theoretical and clinical implications of this study.

6.2 A REVIEW OF THE FINDINGS

In the study of *physiological vocal fold activity* during speech initiation by means of electroglottographic assessment, the results for the group of normal speakers and the group of PWS were as follows:

With regard to *the nature of prephonatory vocal fold activity*:

- Different prephonatory vocal fold characteristics were identified for VC, $C_{(v)}VC$ and $C_{(vl)}VC$ utterances in the control group and subject group. The vocal fold characteristics were classified into five groups or types:
  - Type 1: Absence of prephonatory vocal fold approximation;

- Type 2: Vocal fold approximation without additional prephonatory vocal fold activity;
- Type 3: Vocal fold approximation with associated prephonatory vocal fold activity;
- Type 4: Vocal fold approximation with irregular prephonatory vocal fold activity; and
- Type 5: Vocal fold approximation with vocal fold vibration (prevoicing).

- The control group showed a tendency towards vocal fold approximation without additional prephonatory vocal fold activity (Type 2) for speech initiation, while the subject group was more inclined to vocal fold approximation with irregular prephonatory vocal fold activity (Type 4).

- Both the control group and subject group showed instances of abnormal prephonatory vocal fold activity during speech production. Though, in the controls only a few utterances were characterized by abnormalities, indicating that this type of activity seldom occurs in normal speech production. Atypical prephonatory vocal fold activity during speech initiation was evident in most utterances for the subjects.

- Atypical prephonatory vocal fold activity was more apparent for $C_{(v)}VC$ and $C_{(vl)}VC$ utterances than VC utterances in the subjects. The effect of different phonetic contexts on prephonatory vocal fold activity suggested that atypical vocal fold behaviour in individuals who stutter becomes more distinctive as the complexity of laryngeal-articulatory-respiratory coordination increases. The motor complexity of the speech utterance seems to have a prominent effect on the nature of vocal fold behaviour in stuttering.

- In addition, the incidence of atypical prephonatory vocal fold activity seemed to be associated with stuttering severity. The subjects with mild, moderate and severe stuttering involvement (with exception of the very mild PWS), differed in the extent of atypical vocal fold behaviour they showed. However, this possible relation between stuttering severity and the frequency of abnormal speech motor activities needs further investigation.

With regard to *laryngeal onset time (L_xOT):*

- The subject group showed longer and more variable L_xOT values compared to the control group. The delay in timing of laryngeal-phonatory onset in the subjects could be related to asynchronization between the breath stream and the onset of laryngeal activities for speech production. In agreement with previous reports, this finding proposes that the individuals who stutter have preparation deficits in laryngeal-phonatory activities along with motor initiation variability during speech onset. The greater variability in L_xOT points to a less stable speech motor control mechanism.

- Also, motor complexity of the phonetic context seemed to have influenced the mean L_xOT for both the control group and the subject group. The effect of the complexity of the phonetic context was greater for the subjects. The finding was associated with the fact that higher motor demands were placed on the coordination and timing of a set of muscle groups for a specific movement goal.

In the study of *acoustically detected vocal fold activity* by means of spectrographic assessment, the following results have been found:

With regard to *the nature of voice onset:*

- The control group showed a tendency toward gradual voice onsets for VC, $C_{(v)}$VC and $C_{(vl)}$VC utterances. Normal speech production seems to be reflected by gradual voice onset following well-timed prephonatory vocal fold activity during speech initiation.

- The $C_{(v)}$VC and $C_{(vl)}$VC utterances of the subject group were characterized by gradual voice onsets, while VC productions showed a greater number of abrupt voice onsets. Gradual voice onsets in the subject group are not necessarily an indication of normal speech behaviour. This possibility is underscored by the marked prominence of vocal fold irregularities in the period prior to voice onset. The slow rise in amplitude-over-time in the acoustic waveform could indicate abnormal and adaptive activities in voice onset for the subjects. Gradual voice onset may be a compensatory strategy that the subjects use to initiate voicing when they are experiencing difficulty. This manner of voice initiation, which is associated with fluency enhancement in stuttering, was regularly used by the subjects.

- Manner of voice onset for individual subjects seemed to be influenced by idiosyncratic prephonatory vocal fold activities prior to voice onset. Manner of voice onset, in isolation, is not a reliable indicator of underlying difficulties experienced during voice onset. In combination with other measurements (such as voice onset time), it may contribute to explain subjects' competency in coordinating different articulatory structures (including vocal folds) for specific phonetic contexts during voice onset.

- In addition, no apparent (or consistent) similarity was found between the nature of voice onset (gentle/abrupt) and stuttering severity.

With regard to *voice onset time:*

- The control group and subject group showed mean VOT for prevocalic /b/ and /p/ productions within normal limits.

- The VOT range for /b/ productions was greater than for /p/ productions in the control group. This tendency was also seen in most of the PWS.

- VOT for /b/ productions showed that the normally fluent speakers had a tendency toward prevoicing. Negative VOT values for /b/ productions were also indicated in three of the six PWS.

- Compared to the results of the normally fluent speakers, most PWS showed slower VOT for /b/ productions (although within normal VOT limits). VOT for /b/ productions were mostly consistent with only single instances of abnormal timing between voicing and articulatory release in the PWS.

- For prevocalic /p/ productions, the control group as well as the subject group showed short-lag VOT. Mean VOT values for voiceless stops were similar between the control group and subject group. However, individual subjects showed a smaller (restricted) VOT range for /p/ productions when compared to the normally fluent speakers.

- VOT range values for /b/ productions and /p/ productions differed markedly for the control group. This finding may indicate that voice-voiceless contrasts were a distinctive feature in the control group's speech production. VOT range values for /b/ and /p/ productions overlapped for most subjects. Thus, VOT values for /b/ and /p/ productions did not indicate voice-voiceless contrasts for the subjects.

- For the individual subjects, VOT for /b/ productions were slower and overlapped with VOT for /p/ productions. PWS may have used compensatory strategies to reduce voice production requirements (Perkins *et al.,* 1976) during speech initiation. Compared to /b/ productions, VOT range for /p/ productions were more consistent and showed a tendency toward a restricted VOT range for the individual subjects. This difference in VOT range for /b/ and /p/ productions, suggests that the phonetic complexity of the initial phoneme may have an effect on the PWS's ability to assert control over phonatory and articulatory adjustments during speech initiation.

The data of this study indicate that the individuals who stutter presented with irregularities in vocal fold activities during speech initiation of perceptually fluent utterances. This means that the muscles of the speech production mechanism (especially the vocal folds) in PWS could be activated in ways that influence the rapid initiation of voicing. Deviancies in vocal fold activity may involve abnormal increases of muscle tone in the articulatory and laryngeal structures or disrupted coordination between agonist-antagonist laryngeal musculature which results in a breakdown of reciprocal muscle activity (Adams *et al.,* 1984). Slow laryngeal onset times and inappropriate timing of coordinated movements (such as voice-voiceless overlap of VOT and restricted VOT range) during initiation of speech in PWS may be indicative of abnormal temporal control of speech movements and difficulty in preparation of these movements before initiation.

The salient vocal fold characteristics observed during speech initiation in PWS indicate involvement at the level of motor programming of speech sensorimotor control. A breakdown in motor programming suggests a fault in the accurate specification of spatio-temporal dimensions of movement sequences. From this perspective, speech motor activities (including vocal fold activity) in stuttering may show abnormalities in muscle tone, rate, velocity, and range of movements. Difficulties in the initiation of successive movements are also associated with inefficient motor

programming. The proposal that stuttering is a motor programming disorder could guide further explanations of speech motor activities of PWS.

6.3 EVALUATION OF THE RESEARCH METHODOLOGY

Based on the research methodology followed in this study, certain adaptations are recommended for future research of this nature. The following limitations were identified:

- *Subject group:*
  - *Group size:* A smaller group size or three case studies would have resulted in a concise descriptive study. Since stuttering is characterized by idiosyncratic behaviour for individual speakers, it may be difficult to show trends in speech motor behaviour for a group of PWS. For instance, measurement averages or mean scores often mask abnormalities in individual speakers' speech behaviour (such as slowness or variability).
  - *Homogeneity in stuttering severity*: The subject group of the current study consisted of a heterogenic group of participants ranging from very mild to severe stuttering speakers. This caused difficulties in the management of the data. Stuttering severity was not investigated in the study, but there were indications that abnormal prephonatory activities might have been associated with stuttering severity. Depending on the extent of the study, PWS included in the subject group should either be rated with the same level of stuttering severity or divided into two groups with different levels of stuttering severity.
  - *Variables:* The possible variables that might influence the data need more detailed consideration. Individual stuttering speakers are differently influenced by variables such as involvement of neural substrates, contextual factors (including motor complexity of the utterance, speech rate, and length of the utterance) and stuttering severity. In the presence of insufficient speech sensorimotor control, the timing of motor commands in PWS can be easily disrupted when considering the amount of variables that needs to be processed and monitored.

- *Perceptually fluent speech:* The definitions for "perceptually fluent speech" differ among studies, as well as the procedures that have been used to identify perceptually fluent

speech. This causes difficulty in comparing current results with previous findings of so-called perceptually fluent speech. In addition, research has shown that perceptually fluent speech of PWS is not regarded as normal speech production. For this reason, a separate procedure to distinguish between stuttered speech and fluent speech for the PWS was not included in the current study. However, the material was of such nature to reduce the possibility of perceptually *dis*fluent response, i.e. one-syllabic words were used. A clear definition that includes exact criteria of "perceptually fluent speech" is necessary when studying PWS's speech production.

- *Response task*: In the current study, the response task consisted of reading lists of simple monosyllabic CVC words, which raises doubt as to whether it reflected the PWS' average speech rate. Increased speech rate could have influenced the VOT results by showing short-lag VOT. Duration of VOT is related to speech rate, specifically the rate of articulatory movement and the extent of coarticulatory overlapping (Starkweather, 1982). Speech rate is an important contaminating variable to consider in future studies.

6.4 RECOMMENDATIONS FOR FURTHER RESEARCH

The contribution of future research to a better understanding of stuttering will depend on a refined perspective of stuttering as a motor speech disorder. Based on the results of this study, a few points are emphasized that could be addressed in future prospective studies on stuttering:

- In order to obtain responses imitating a more natural speech context, the stimulus material can be presented within simple, meaningful sentences. For instance, the sentence is read to the subject, whereupon three questions can be asked requiring single word responses. These responses will consist of the target words. For example:
  - Instruction sentence:
    - "It en At staan langs die pad" *(It and At stand beside the road)*
  - Questions for eliciting monosyllabic words:
    - "Wie is saam met At?" (*Who is with At?)* – "It"
    - "Waar staan hulle?" (*Where are they?)* – "pad"

- "Is Ben saam met It?" (*Is Ben with It?)* – "At"

A comparison of results obtained by reading wordlists and those obtained by answering simple questions might be insightful. Furthermore, these sentences could follow in sequence to form a short story. At the end of the questioning, the subject could be asked to retell the story and in that way use the stimulus words within continuous speech. A comparison of results by using the stimulus material in single word productions versus continuous speech might show interesting differences.

- The subtyping of the L_x signal presented with interesting results for both normally fluent speakers and PWS. The present results are the only available data on speech initiation that differentiate L_x signal types based on prephonatory vocal fold activity. The validity of L_x signal typing as a clinical tool can be improved by assessing *intra- and interrater reliability* using experienced voice clinicians. A panel of clinicians can compile a profile of normal types of prephonatory vocal fold activity to be used as a norm when assessing speech pathologies. Other factors important for L_x signal typing for future studies are:

  - *Definitions of L_x signal types:* The definitions of L_x signal types need to be clear and descriptive to enable distinction between different types.
  - *Size of control group and subject group:* More individuals would need to be included in the control group and subject group when conducting such investigation to ensure reliability and validity of the different types.
  - *Stuttering severity of the subject group:* A differentiation must be made between levels of stuttering severity. The effect of stuttering severity on L_x signal types may reveal correlations between perceptual symptoms and physiologic characteristics of stuttering.
  - *Physiological measurement tools / Instrumentation:* L_x signal typing of vocal fold behaviour was based on electroglottographic (EGG) signal.

Signal typing of the L_x waveform helps to further our understanding on the nature of the vocal fold dynamics in different phonetic contexts for normal speech production, as well as for speech pathologies such as stuttering. The dynamics of vocal fold activity relate back to the physical requirements for voicing. The process of signal typing involves the analysis

of the signal pattern (or form) with the focus on identifying atypical vocal fold activity. L_x signal typing might be used to differentiate between stuttered speech and fluent speech in PWS. The clinical value of this non-evasive, objective measurement tool may include: providing biofeedback during therapy intervention and monitoring therapy progress and effectiveness.

- No maximum or minimum limits for L_xOT exist for different phonetic contexts (including VC, $C_{(v)}$V, and $C_{(vl)}$V) in normally fluent speakers. Thus, L_xOT does not seem to be constrained to a specific range for a particular phonetic context, as in the case of VOT. However, a possible relation between phonetic complexity with reference to varying voicing demands and the magnitude of L_xOT values was indicated. The study has shown that the prephonatory period for speech initiation varies for different phonetic contexts. Whether or not speech sounds differ on the basis of the duration of prephonatory period (or L_xOT) is unclear and needs further investigation. Normative data of L_xOT is currently lacking that will enable research on the subject of prephonatory period during speech initiation of PWS.

- Comparisons between stuttering and other vocal motor disabilities (e.g. dysphonic speakers and hyperkinetic dysarthria) based on L_x signal typing and L_xOT, might show distinctive differences in prephonatory speech motor activities for these pathologies.

- More stuttering subjects need to be included in studies focusing on specifically physiological vocal fold characteristics. This would allow the researcher to perform statistical analysis of the data and enable a greater degree of generalization of the findings.

- Research on the relation between L_x signal types and other electro-physiological/kinematic data can provide useful information on the nature of prephonatory vocal fold behaviour in PWS. For instance, the time-interval between the moment of glottal closure/approximation and phonatory onset might be a direct indicator of the rate of airflow change rather than of vocal fold activity. In this regard, electroglottographic analyses of vocal fold behaviour in combination with subglottal pressure measures such as phonation threshold pressure (PTP) might provide greater insight into the prephonatory activities of PWS. A combination of measurements, such as mentioned, may further our understanding of the neuromuscular

mechanisms underlying stuttering and may aid in characterizing individual differences in PWS. In-depth descriptions of abnormal speech motor activities in stuttering are important to identify the neural sources that produce the aberrant motor behaviour (Denny & Smith, 1992).

- Studies need to verify if VOT values "within normal limits" for PWS refer to normal speech production. PWS may use different motor strategies than normal speakers to accomplish the same audible or acoustic result. This matter may be addressed by showing how PWS function under high stress conditions such as time stress or increased contextual complexity.

- More repetitions of the utterances need to be included for analysis to determine the following:
  - The effect of motor complexity of a specific phonetic context on vocal fold activity;
  - The effect of vowel transitions on the preceding sound;
  - The effect of the repetitious nature of the speech task on the incidence of irregular activities and the timing of speech activities. Winkler and Ramig (1986) suggested that the repetitious nature of a speech task will neutralize differences that PWS might have. Thus, if a PWS repeats an utterance several times consecutively, his timing abilities or asynchronies should approximate those of the normal speakers. Conscious, repeated initiations of the same utterance allow for accuracy shaping in timing and coordination for that production. Research into the repeated initiation of the same utterance in PWS may reveal important information on fluency-shaping activities during speech initiation.

- Further investigation is warranted to clarify whether stuttering is a disorder specific to the speech production process, or if it originates from a generalized sensorimotor deficiency (i.e. a general incompetency to generate a "temporal program" for motor behaviour). Questions that need to be addressed are:
  - How do linguistic variables affect the frequency of stuttering behaviour in PWS?
  - How do timing variables affect motor speech control in PWS?
  - How do the peripheral physiological events explain the perceptually observed

events in stuttering?

- The VOT results of PWS indicated that the speech movements for voice onset during fluent speech were performed slower in some cases and more variable in others. Slower VOT might suggest that speech movements were appropriately timed relative to other movements within the articulatory, phonatory and respiratory subsystems which resulted in the desired auditory effect. A distinction between slower and mistimed movements during the perceptually fluent speech of PWS is necessary to reflect on critical aspects of the mechanisms underlying the disorder or a preferred motor strategy that is used to avoid or minimize *dis*fluencies (Max *et al.*, 2004).

- Research on stuttering has disclosed information about the dynamic interplay among complex cortical and subcortical systems involved in the planning, programming, execution, and monitoring of speech movements (see Chapter 2). The current results confirm abnormal functioning of motor speech activities in stuttering. This dysfunction in speech motor control suggests involvement of the underlying neural mechanism. A combination of brain imaging and peripheral instrumentation when investigating speech behaviour of PWS will enable further insight into the nature of stuttering.

6.5 IMPLICATIONS OF THE STUDY

Viewing stuttering as a disorder of motor programming as depicted by the framework for the characterization of pathological speech sensorimotor control by Van der Merwe (1997) has important theoretical and clinical implications. Theoretically, it provides a framework that can be used to develop testable hypotheses regarding the nature of speech motor difficulties in stuttering. Clinically, this viewpoint has several benefits relating to the development of assessment and treatment protocols.

6.5.1 Theoretical implications of the study

The theoretical implications of the current study relate to developing a model of speech sensorimotor control for stuttering by describing one of the salient characteristics of stuttering,

namely, aberrancies in glottal activity during speech initiation. The theoretical implications of this study regarding *glottal activity during speech initiation in stuttering* are as follows:

- Accurate descriptions of the salient speech motor characteristics (such as abnormal glottal activity) in PWS are needed to develop a better insight into the breakdown in speech motor control during speech initiation in stuttering. In other words, if stuttering is associated with a dysfunction in motor programming, then speech motor difficulties relating to movement specifications such as muscle tone, movement velocity, force and range of movement, and mechanical stiffness of the joints might be evident in respiratory, laryngeal-phonatory and articulatory activities of PWS.

- The nature of prephonatory vocal fold activity during speech initiation in normal speech production and stuttering can be described by identifying and classifying different vocal fold characteristics. The classification of different vocal fold characteristics contributes to a better understanding of prephonatory vocal fold activity during speech initiation of different phonetic contexts in normal speakers and individuals who stutter.

- The prephonatory period preceding voice onset seems to be characterized by variability and slowness in the timing of laryngeal-phonatory movements in PWS. However, the period of voice onset in PWS shows less distinctive differences in laryngeal-articulatory activities compared to normal speakers. This finding strongly suggests that stuttering is characterized by a breakdown in speech motor control during the prephonatory period preceding speech onset. Furthermore, deviant vocal fold activity is evident in perceptually *fluent* utterances, which may suggest that variability and/or slowness in prephonatory speech motor activities during speech initiation are inherent to stuttering.

- If stuttering is regarded as a motor programming dysfunction with a breakdown in speech motor control during speech initiation, then it needs to be established whether such lack in motor control is limited to speech, or whether it includes all intentional motor activities.

- Combining qualitative data (i.e. descriptions of the nature of physio-acoustic vocal fold activity) with quantitative measurements (i.e. L_xOT and VOT) have created a better

understanding of physiological and acoustically detectable vocal fold activity during speech initiation for normal speech production and stuttering.

- The effect of the phonetic context is clearly indicated as an important factor in timing measures (such as L_xOT and VOT) in normal speakers and individuals who stutter. In this regard, motor complexity of the utterance needs to be considered when examining the timing aspects of speech initiation.

- The current findings show that instrumental analyses are needed to accurately identify and describe the speech motor difficulties associated with stuttering behaviour. Perceptual observations are not reliable in identifying these subacoustic behaviours. However, if stuttering is viewed as a disorder in speech motor control, a specific definition (with criteria for (*dis*)fluency) needs to be formulated in order to identify and differentiate its characteristics from other speech motor disorders. Accurate descriptions of physio-acoustic vocal fold activity in stuttering could thus be valuable in making a differential diagnosis between stuttering, spasmodic dysphonia and other disorders involving deviancies in vocal fold motor control.

6.5.2 Clinical implications of the study

The study developed a better insight into the physiological and acoustically detectable glottal activity in individuals who stutter, especially pertaining to prephonatory vocal fold activity preceding speech onset. Clinical approaches to intervention (including assessment, effective therapy techniques, and termination criteria for therapy) are implicated by a better understanding into the speech motor activities of stuttering. The results of the study have several implications for the clinical setting:

- The use of physio-acoustic instruments as stuttering assessment tools is underscored by the fact that perceptual assessments are unable to accurately and reliably identify speech motor difficulties in perceptually fluent speech in PWS. However, the availability, accessibility and cost-effectiveness of such instrumental assessment procedure are queried.

- The prephonatory period preceding speech onset has shown the highest incidence of speech motor difficulties in PWS, but this period is acoustically undetectable. To address these difficulties, an instrumental approach to therapy might also be used to increase cognitive control in PWS. Biofeedback might be a useful therapy strategy during the course of intervention.

- Understanding the nature of speech motor difficulties in stuttering (such as abnormal vocal fold activity) within a framework of deficient motor programming might enable clinicians to formulate new stuttering therapy techniques. For instance, therapy could focus on the conscious monitoring of specific movement requirements, including muscle tone, rate of movement, range of movement, movement velocity, and mechanical stiffness of the joints, in order to control spatio-temporal and force dimensions of speech motor activities.

- The effectiveness of stuttering therapy has been indicated by a decrease in perceptually detectable *dis*fluencies (i.e. silent blocks/fixations, repetitions, prolongations and hesitations). As indicated by the present study, perceptually fluent speech is not necessarily stutter-free, subtle motor aberrancies may exist during speech initiation that are not perceivable by the listener. This problem concerning treatment of perceptually *dis*fluent *versus* fluent speech in stuttering leads to critical questions with regard to therapy, such as: - If stuttering involves more than only moments of perceptually *dis*fluent speech, what should the focus of stuttering therapy be? What criteria should be used to determine the effectiveness of therapy: reduction in abnormal speech motor activity or decrease in perceptual *dis*fluencies? If a PWS does not present with perceptually detectable *dis*fluencies, must that individual be terminated from therapy?

- The results for the group of PWS showed that a decrease in motor complexity of the utterance can be associated with less variable vocal fold behaviour. The control of contextual factors (such as motor complexity, rate, familiarity, and length of utterance) seems to have a fluency enhancement effect on stuttering. In other words, when these factors are regulated to reduce requirements for motor control, motor activities show increased stability and coordination. The contextual factors need to be considered when applying stuttering therapy techniques. By making gradual adjustments to contextual

factors in therapy, an individual who stutters might learn to control his/her fluent speech production with an increase in the level of difficulty of these contextual factors.

6.6 CONCLUDING REMARKS

In the last two decades, there have been few attempts at formulating a comprehensive framework that would account not only for the salient characteristics of stuttering involving laryngeal-phonatory, articulatory and respiratory activities, but also for the various phenomena known to be associated with stuttering (e.g. empirical results regarding sensorimotor performance in PWS, fluency-enhancing conditions, positive results of specific treatment programs). This study has aimed to describe one of the salient characteristics of stuttering, i.e. abnormalities on vocal fold activity during speech initiation, within a theoretical framework of speech sensorimotor control and thereby enabled progress towards a better understanding of stuttering as a motor programming dysfunction.

A review of the literature pertaining to vocal fold activity during speech initiation in stuttering indicated that PWS have a tendency towards abnormal vocal fold activity which is associated with a breakdown in the timing control of coordinative movements of phonation, respiration and articulation during speech production. Understanding the nature of vocal fold activity in stuttering within a theoretical framework of speech sensorimotor control for the characterization of pathological speech production (Van der Merwe, 1997) was underscored. Based on recent insights into the neural control of movement and modelling of the underlying mechanisms, stuttering has been associated with a breakdown in the programming process of motor speech control. Against this background, the purpose of the study was formulated to describe glottal activity during speech initiation in stuttering by means of electroglottographic and acoustic analyses.

The results of the current study have supported the view that PWS exhibit difficulty in the programming of successive and sequential speech movements during initiation. Individuals who stutter seemed to have experienced more irregularities in vocal fold activity in the preparatory period before acoustically detected voice onset. Variability and slowness in laryngeal-phonatory activity in the prephonatory period preceding voice onset in PWS (as indicated by L_xOT) appear to have influenced the acoustically detected voice onset to a greater or lesser extent. The findings suggested that PWS have more difficulty in coordinating laryngeal-phonatory movements with

increased motor complexity of the utterance. Since the PWS and normal speakers have not shown marked differences with regard to VOT, it is suspected that PWS may have regained control of their speech mechanism during the audible output of voice onset or used compensatory motor strategies to maintain fluency. However, if motor complexity of the utterance were to be increased, PWS might not be able to effectively compensate for aberrancies in speech motor control. With regard to physiological and auditory vocal fold activity and acoustically detectable vocal fold activity, it has clearly been indicated that PWS show the greatest difficulty in speech motor coordination in the period preceding actual voice onset.

Explanations have been presented for the vocal fold characteristics in individuals who stutter that relate the specific findings to deviant motor programming. PWS seem to have insufficient temporal control of complex successive movements with the emphasis on vocal fold motor control and this breakdown in temporal control are most evident during the period of speech initiation. In addition, it has been suggested that inefficient motor programming skills of PWS are induced by increased production demands.

The study has shown that more research investigating vocal fold activity during speech initiation in normal speakers and stuttering speakers is needed. To enable generalization of the results, a repetition of this study is recommended which will include a larger group size of stuttering speakers, intra- and interrater judgment of qualitative speech motor activities, and with adjustments to the stimulus material.

The importance of this type of research for a better understanding and accurate description of vocal fold activity during speech initiation in stuttering was emphasized, since difficulty in voice initiation is regarded as one of the primary characteristics of stuttering. In-depth knowledge of the speech motor symptoms of PWS would result in compiling effective assessment and treatment protocols.

To conclude, this study aimed to describe abnormal vocal fold activities during speech initiation in stuttering within a theoretical framework of speech sensorimotor control. The results presented in this study suggest that abnormalities in prephonatory vocal fold activity preceding speech onset in individuals who stutter are likely to be related to difficulties in motor programming. Further research is needed to confirm whether the underlying cause of the breakdown in vocal fold motor

control, is specifically related to speech production or whether a more generalized disability in sensorimotor control is indicated in stuttering. In the quest for a better understanding of the underlying cause of stuttering behaviour, it is important to support Conture's (2001:27) description of what contributes to scientific progress: "…the more sources of theoretical input we receive, from as many different perspectives as possible, the greater the possibility that no relevant issue will be overlooked... it is only by offering different theories and therapies in the marketplace of ideas that the truth will emerge… Disagreements, not agreements, typically foster and encourage new insights into old problems and are part of the stuff from which progress is made."

6.7 SUMMARY OF CHAPTER 6

In conclusion of the study on the laryngeal behaviour in stuttering a review of the findings was given. The research methodology was critically evaluated and improvements on the current study for future research were suggested. The theoretical and clinical implications were discussed as to indicate the significance and relevance of the study.

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APPENDIX A:

READING PASSAGE

APPENDIX A: READING PASSAGE

Na die wildtuin

By so 'n samekoms is die verbroedering gou. Na die aandete kom almal bymekaar om die smeulende laervuurtjies en dan word allerhande verhale vertel van ervarings op pad en van jagters en wild. Kort-kort klou kinders aan Pa se hand of Ma se rok vas, en begin hare ongemaklik rys, as Vuilbaard naby die laerplek sy stem laat hoor. Ja, dan is ons nie meer so manhaftig as vanoggend nie, toe ons grootgepraat het om hom aan sy stert te vang.

In die laerplek is dit darem doodveilig en weldra verdwyn een-vir-een vermoeide reisiger uit die groepie om rus in die gerieflike beddens te gaan soek. Almal wil fris en uitgerus voel, as hulle die bewoners van die wildtuin besoek. Eindelik gloei daar net hier en daar 'n kooltjie in die donker nag. Bo blink die sterre helder oor slapende reisigers. Wie weet of Vuilbaard nie naby die laer rondsnuffel nie. Na ontbyt spat almal weer in verskillende rigtings uitmekaar. Ons is gewaarsku om nie op pad uit karre te klim nie. Jy vra my wat ons alles gesien het? Nee, vrind, dis moeilik om te sê. 'n Mens doen so baie indrukke op dat jy beswaarlik alles kan onthou.

Ons het dit ook gelukkig getref om buffels, olifante en kameelperde raak te loop.

P. de Villiers-Pienaar en A.G. Hooper (1941)

'n Engels-Afrikaanse Fonetiese Leesboek (pp. 73-75)

APPENDIX B:

- **APPENDIX B*i*: LETTER FROM ETHICS COMMITTEE**
- **APPENDIX B*ii*: LETTER OF CONSENT: AFRIKAANS**

APPENDIX B*i*: LETTER FROM ETHICS COMMITTEE

Lede: Navorsingsvoorstel- en Etiekkomitee
Prof D Beyers; Dr L Davis; Prof C Delport;
Dr M de Waal; Dr JEH Grobler; Prof KL Harris;
Dr JdeC Hinch; Prof E Krüger; Prof B Louw;
Prof IA Niehaus; Prof C Potgieter; Prof D Prinsloo;
Dr E Taljard; Prof J van Eeden; Prof A Wessels

Universiteit van Pretoria
Navorsingsvoorstel-en Etiekkomitee
Fakulteit Geesteswetenskappe

17 Oktober 2003

Beste Professor van der Merwe

Projek:	***An electroglottographic and acoustic analysis of glottal activity during speech initiation in stuttering***
Navorser:	**H Bezuidenhout**
Leier:	**Prof A van der Merwe**
Departement:	**Kommunikasiepatologie**
Verwysingsnommer:	**9109137**

Baie dankie vir u skrywe van 16 Oktober 2003 en aangehegte dokumentasie.

Die aansoek is deur 'n *ad hoc* komitee op 17 Oktober 2003 oorweeg.

Die komitee neem kennis van die aansoek, maar kan nie op hierdie stadium verantwoordelikheid aanvaar vir enige etiese kwessies wat ontstaan nie. Hierdie verantwoordelikheid rus nou by die leier.

Vriendelike groete

Brenda Louw

Prof Brenda Louw
Voorsitter: Navorsingsvoorstel-en Etiekkomitee
Fakulteit Geesteswetenskappe
UNIVERSITEIT VAN PRETORIA

APPENDIX B*ii*: AN EXAMPLE OF THE LETTER OF CONSENT

Geagte Meneer

INLIGTINGSBRIEF AANGAANDE DEELNAME AAN STUDIE OOR DIE STEMBANDGEDRAG IN HAKKELAARS

Ek is tans besig met 'n magistersgraadstudie waarin ek die stembandgedrag van persone wat hakkel ondersoek gedurende spraakinisiëring. Die Hakkelkliniek, Departement Kommunikasiepatologie van die Universiteit van Pretoria, privaatpraktyke in Pretoria en Een Militêre hospitaal is genader vir moontlike individue wat as proefpersone kan optree. U is reeds telefonies gekontak en het uself bereid verklaar om aan hierdie studie deel te neem. Ek bevestig hiermee die aspekte rakende die studie wat ek reeds onder u aandag gebring het:

Die doel van hierdie navorsingprojek is om stembandgedrag tydens geselekteerde enkelwoordproduksies by persone wat hakkel te ondersoek. Die projek kan nuttige inligting oplewer rakende die stembandgedrag voor en tydens spraakproduksie by persone wat hakkel. Dit hou bepaalde implikasies in vir die spraak-taalterapeut se kennisbasis van die simptomatologie van hakkel. Hieruit kan terapieriglyne voortspruit waarby die persoon wat hakkel wel kan baat. Die studie strek u nie direk tot voordeel nie, maar hou geen risiko's vir u in nie.

Vervolgens, 'n kortlikse uiteensetting van wat die studie sal behels: Vir die studie sal u versoek word om ongeveer 15 minute van u tyd beskikbaar te stel aan die navorser. Die uitvoering van die studie behels die lees van vyf woordlyste van twaalf eenlettergrepige woorde (soos "mat"). Daar sal van u verwag word om in 'n mikrofoon te praat met twee vel-elektrodes weerskante van u strottehoof geplaas. Ten tye van die opname sal u in die UP-Spraaknavorsingslaboratorium wees met die ondersoeker en die navorser verbonde aan die laboratorium teenwoordig. Die opname word op 'n oudioband opgeneem vir latere analise deur die ondersoeker. Na afloop van die lees van die woordlyste, sal die persone wat hakkel ook gevra word om 'n kort leesstuk voor te lees wat ook op 'n oudioband opgeneem word. 'n Video-opname word ook gemaak van albei leestake by die persone wat hakkel. Die videomateriaal word gebruik vir latere analise om die graad van hakkel te bepaal volgens 'n toetsprotokol, Stuttering Severity Index.

Ek wil u verseker dat u identiteit deur die loop van die studie gerespekteer sal word. Slegs die navorser sal kennis dra van u identiteit. Alle inligting word as vertroulik hanteer. Geen persoonlike verwysing word tydens die data-insameling, data-verwerking of bespreking van die resultate gedoen nie.

Ek bevestig weereens dat u besluit om aan die studie deel te neem geskied op 'n vrywilllige basis en dat u op enige stadium mag onttrek.

'n Artikel van hierdie studie sal moontlik gepubliseer word in THE SOUTH AFRICAN JOURNAL OF COMMUNICATION DISORDERS, sou u belangstel om die studie se bevindinge te wete te kom.

Indien u verdere inligting oor die voorgenome studie benodig, kan u my gerus kontak by (011) 849-3098 of hbez@lantic.net.

U deelname sal hoog op prys gestel word.

By voorbaat dank

_____
Me Hannelie Bezuidenhout
Meestersgraad-student: Kommunikasiepatologie
Studente-nommer: 91091374

_____
Prof. A van der Merwe
Studieleier: Departement Kommunikasiepatologie

_____
Prof. B Louw
Departementshoof: Departement Kommunikasiepatologie

APPENDIX C:

PREPARATORY VOCAL FOLD MOVEMENTS FOR SPEECH INITIATION OF VOICED AND VOICELESS SPEECH SOUNDS BASED ON THE PHYSIOLOGY OF NORMAL SPEECH PRODUCTION

APPENDIX C: PREPARATORY VOCAL FOLD MOVEMENTS FOR SPEECH INITIATION OF VOICED AND VOICELESS SPEECH SOUNDS BASED ON THE PHYSIOLOGY OF NORMAL SPEECH PRODUCTION

Phonetic Context	Preparatory Vocal Fold Activity for the Initiation of Voiced and Voiceless Speech Sounds based on the Physiology of Normal Speech Production	Description of Preparatory Vocal Fold Activity for the Initiation of Voiced and Voiceless Sounds during Normal Speech Production
Vowel onset in VC context	Vocal fold approximation in preparation of voiced production.	▪ In Afrikaans the vowel sound might be preceded by voiceless glottal stop, i.e. / /, which is normally inaudible (De Villiers & Ponelis, 1987). Prephonatory vocal fold adduction is thus implied.Due to coarticulation, the vowel then takes on the properties of a consonant with the glottal closure acting as the articulatory occlusion. This results in a hard glottal attack of the vowel production (Uys, 1970). ▪ The VC context in this case changes to a $C_{(vl)}$VC context. (Important to note is the different effects of the VC productions versus the $C_{(vl)}$VC productions on motor complexity).
Voiced consonant onset in $C_{(v)}$VC context	Vocal fold approximation in preparation of voiced production.	▪ In the $C_{(v)}$VC context, the vocal folds might be approximated or adducted due to the voicing properties of the initial phoneme. ▪ During the preparatory period, vocal fold vibration might result before the release of the articulatory closure (Borden & Harris, 1984; Hoole, 1997). This is still within the parameters of normal speech production. In this case, the subglottal air pressure might have exceeded that of the intra-oral cavity and set the vocal folds vibrating before release of the supraglottal air pressure for the plosive production (Borden & Harris, 1984).
Voiceless consonant onset in $C_{(vl)}$VC context	No vocal fold vibration is required for the production of the initial voiceless plosive. Vocal folds are expected to be in an abductor position prior to voiceless sound productions. However, depending on the sound context, vocal fold approximation might be present.	▪ In the $C_{(vl)}$VC context, the vocal folds might be approximated to prepare for the phonetic properties of the vowel following the initial voiceless consonant. ▪ If a glottal closure is present in preparation to the following voiced sound, it is physiologically possible for the supraglottal structures (e.g. tongue base and pharyngeal walls) to build up sufficient pressure within the intra-oral cavity to result in a consonant burst (Rothenberg, 1968). ▪ In normal dynamic speech production the $C_{(vl)}$ might take on the voiced properties of the following sound, due to interarticulatory synchronization (Hoole, 1997).

APPENDIX D:

A GRAPHIC PRESENTATION OF THE MATERIAL

APPENDIX D: A GRAPHIC PRESENTATION OF THE MATERIAL

Lys 1	Lys 2	Lys 3	Lys 4	Lys 5
at	bad	pit	bot	piet
piet	pot	ot	iet	at
bot	it	bied	pit	bot
iet	bied	pad	bad	pad
pit	pad	bot	it	it
bid	ot	at	pot	bied
it	bid	piet	bied	pit
pot	piet	bid	ot	iet
bad	at	iet	pad	bad
ot	bot	pot	bid	ot
pad	pit	bad	at	bid
bied	iet	it	piet	pot
sit	soet	sat	sif	soek

APPENDIX E:

THE COMBINATION DISPLAY SETTINGS FOR SIGNAL ANALYSIS

APPENDIX E: THE COMBINATION DISPLAY SETTINGS FOR SIGNAL ANALYSIS

KAY ELEMETRICS CORP. MODEL 5500
SIGNAL ANALYSIS WORKSTATION

Date: 3F 19FF Rec. Off Time:12:7F:03 AM
Analysis by:

INPUT SETTINGS	Channel 1	Channel 2
Source	LEFT CONNECTORS	RIGHT CONNECTORS
Frequency Range	DC - 32 KHz.	DC - 32 KHz.
Input Shaping	FLAT	FLAT
Buffer Size	4.75 SECONDS	4.75 SECONDS

ANALYSIS SETTINGS	Lower Screen	Upper Screen
Signal Analyzed	CHANNEL 1	CHANNEL 2
Analysis Format	WAVEFORM	WAVEFORM
Transform Size	100 pts. (1200 Hz)	256 pts. (469 Hz)
Time Axis	25ms (500ms)	25ms (500ms)
Frequency Axis	FULL SCALE	FULL SCALE
Analysis Window	HAMMING	HAMMING
Averaging Set Up	NO AVERAGING	NO AVERAGING

DISPLAY SETTINGS	Lower Screen	Upper Screen
Freq. Divisions	0.000 Hz.	0.000 Hz.
Dynamic Range	42 dB	72 dB
Analysis Atten.	20 dB	0 dB

Set Up Options Set to: # 50

CURSOR READINGS:
FC1: , FC2: , ^F:
FC1: dB, FC2: dB, ^F: dB
^R1: ^R2:
^T:
PITCH TC1: Hz. TC2: Hz.
AMPLITUDE TC1: dB TC2: dB

SUBJECT MATTER

APPENDIX F:

- **APPENDIX F*i*: THE DATA RECORDING FORM: AN EXAMPLE FOR THE VC CONTEXT**
- **APPENDIX F*ii*: THE DATA RECORDING FORM: AN EXAMPLE FOR THE $C_{(v)}$VC CONTEXT**
- **APPENDIX F*iii*: THE DATA RECORDING FORM: AN EXAMPLE FOR THE $C_{(vl)}$VC CONTEXT**

APPENDIX F*i*: THE DATA RECORDING FORM: AN EXAMPLE FOR THE VC CONTEXT

CONTROL / SUBJECT:	***DATE:***

CONTEXT		***PHYSIOLOGICAL PREPHONATORY VOCAL FOLD ACTIVITY***						***ACOUSTICALLY DETECTED VOCAL FOLD ACTIVITY***		
VC		***NATURE OF PREPHONATORY VOCAL FOLD ACTIVITY****					***L_xOT*** *(in ms)*	***NATURE OF VOICE ONSET****		***VOT*** *(in ms)*
		Type 1: Absence of vocal fold approximation	***Type 2: Prephonatory vocal fold approximation without additional vocal fold activity***	***Type 3: Prephonatory vocal fold approximation with laryngeal preadjustments***	***Type 4: Prephonatory vocal fold approximation with irregular prephonatory vocal fold activity***	***Type 5: Prephonatory vocal fold approximation with prevoicing***		***Gradual***	***Abrupt***	
1	/ɑt/									
2	/ɑt/									
3	/ɑt/									
Subtotal: /ɑt/										
4	/ət/									
5	/ət/									
6	/ət/									
Subtotal: /ət/										
7	/ɪt/									
8	/ɪt/									
9	/ɪt/									
Subtotal: /ɪt/										
10	/ɔt/									
11	/ɔt/									
12	/ɔt/									
Subtotal: /ɔt/										
TOTAL (from 12 words)										
AVERAGE (out of 12 words)										

RECORDING FOR NATURE OF PREPHONATORY VOCAL FOLD ACTIVITY/NATURE OF VOICE ONSET: [0] = behaviour absent [I] = behaviour present

APPENDIX F*ii*: THE DATA RECORDING FORM: AN EXAMPLE FOR THE $C_{(v)}VC$ CONTEXT

CONTROL / SUBJECT:	***DATE:***

CONTEXT		***PHYSIOLOGICAL PREPHONATORY VOCAL FOLD ACTIVITY***						***ACOUSTICALLY DETECTED VOCAL FOLD ACTIVITY***		
$C_{(v)}VC$		***NATURE OF PREPHONATORY VOCAL FOLD ACTIVITY****					***L_xOT*** *(in ms)*	***NATURE OF VOICE ONSET****		***VOT*** *(in ms)*
		Type 1: Absence of vocal fold approximation	***Type 2: Prephonatory vocal fold approximation without additional vocal fold activity***	***Type 3: Prephonatory vocal fold approximation with laryngeal preadjustments***	***Type 4: Prephonatory vocal fold approximation with irregular prephonatory vocal fold activity***	***Type 5: Prephonatory vocal fold approximation with prevoicing***		***Gradual***	***Abrupt***	
1	/bɑt/									
2	/bɑt/									
3	/bɑt/									
Subtotal: /bɑt/										
4	/bət/									
5	/bət/									
6	/bət/									
Subtotal: /bət/										
7	/bɪt/									
8	/bɪt/									
9	/bɪt/									
Subtotal: /bɪt/										
10	/bɔt/									
11	/bɔt/									
12	/bɔt/									
Subtotal: /bɔt/										
TOTAL (from 12 words)										
AVERAGE (out of 12 words)										

RECORDING FOR NATURE OF PREPHONATORY VOCAL FOLD ACTIVITY/NATURE OF VOICE ONSET: [0] = behaviour absent [I] = behaviour present

APPENDIX F*iii*: THE DATA RECORDING FORM: AN EXAMPLE FOR THE $C_{(vl)}VC$ CONTEXT

CONTROL / SUBJECT:	***DATE:***

CONTEXT		***PHYSIOLOGICAL PREPHONATORY VOCAL FOLD ACTIVITY***						***ACOUSTICALLY DETECTED VOCAL FOLD ACTIVITY***		
		NATURE OF PREPHONATORY VOCAL FOLD ACTIVITY*						***NATURE OF VOICE ONSET****		
$C_{(vl)}VC$		***Type 1: Absence of vocal fold approximation***	***Type 2: Prephonatory vocal fold approximation without additional vocal fold activity***	***Type 3: Prephonatory vocal fold approximation with laryngeal preadjustments***	***Type 4: Prephonatory vocal fold approximation with irregular prephonatory vocal fold activity***	***Type 5: Prephonatory vocal fold approximation with prevoicing***	***L_xOT*** *(in ms)*	***Gradual***	***Abrupt***	***VOT*** *(in ms)*
1	/pɑt/									
2	/pɑt/									
3	/pɑt/									
Subtotal: /pɑt/										
4	/pət/									
5	/pət/									
6	/pət/									
Subtotal: /pət/										
7	/pɪt/									
8	/pɪt/									
9	/pɪt/									
Subtotal: /pɪt/										
10	/pɔt/									
11	/pɔt/									
12	/pɔt/									
Subtotal: /pt/										
TOTAL (from 12 words)										
AVERAGE (out of 12 words)										

RECORDING FOR NATURE OF PREPHONATORY VOCAL FOLD ACTIVITY/NATURE OF VOICE ONSET: [0] = behaviour absent [I] = behaviour present

APPENDIX G:

- **APPENDIX G*i*: THE ANALYSIS OF L_xOT (in ms) OF THE 12 VC PRODUCTIONS: A PRESENTATION OF THE L_xOT VALUES, MEAN L_xOT AND RANGE OF L_xOT FOR THE SIX CONTROL SPEAKERS (C1-C6) AS WELL AS THE MEAN L_xOT VALUE AND L_xOT RANGE FOR THE CONTROL GROUP**

- **APPENDIX G*ii*: THE ANALYSIS OF L_xOT (in ms) OF THE 12 $C_{(v)}$VC PRODUCTIONS: A PRESENTATION OF THE L_xOT VALUES, MEAN L_xOT AND RANGE OF L_xOT FOR THE SIX CONTROL SPEAKERS AS WELL AS THE MEAN L_xOT VALUE AND L_xOT RANGE FOR THE CONTROL GROUP**

- **APPENDIX G*iii*: THE ANALYSIS OF L_xOT (in ms) OF THE 12 $C_{(vl)}$VC PRODUCTIONS: A PRESENTATION OF THE L_xOT VALUES, MEAN L_xOT AND RANGE OF L_xOT FOR THE SIX CONTROL SPEAKERS (C1-C6) AS WELL AS THE MEAN L_xOT VALUE AND L_xOT RANGE FOR THE CONTROL GROUP**

APPENDIX G*i*: THE ANALYSIS OF L_xOT (in ms) OF THE 12 VC PRODUCTIONS: A PRESENTATION OF THE L_xOT VALUES, MEAN L_xOT AND RANGE OF L_xOT FOR THE SIX CONTROL SPEAKERS (C1-C6) AS WELL AS THE MEAN L_xOT VALUE AND L_xOT RANGE FOR THE CONTROL GROUP.

LARYNGEAL ONSET TIME (L_xOT) IN ms for VC PRODUCTIONS													
12 VC PRODUCTIONS					CONTROL SPEAKERS (N = 6)						MEAN L_xOT IN ms FOR V1C, V2C, V3C, V4C CONTEXTS FOR CONTROL GROUP	CONTROL GROUP
					CONTROL SPEAKER 1	CONTROL SPEAKER 2	CONTROL SPEAKER 3	CONTROL SPEAKER 4	CONTROL SPEAKER 5	CONTROL SPEAKER 6		
PHONETIC CONTEXT	VOWEL-CONSONANT (VC)	V1C	/ɑt/	1	464.1	110.9	165.6	168.7	101.6	118.7	/ɑt/	**176.85**
			/ɑt/	2	240.6	170.3	175	142.2	118.7	125		
			/ɑt/	3	217.2	145.3	159.4	295.3	148.4	116.4		
		V2C	/ət/	4	75.0	212.5	568.7	146.9	220.3	310.9	/ət/	**210.18**
			/ət/	5	295.3	187.5	154.7	148.4	150	138.3		
			/ət/	6	235.9	232.8	178.1	110.9	160.9	256.2		
		V3C	/ɪt/	7	212.5	228.1	150	101.6	148.4	139.1	/ɪt/	**191**
			/ɪt/	8	195.3	203.1	268.7	235.9	140.6	107.8		
			/ɪt/	9	492.2	157.8	143.7	159.4	257.8	96.09		
		V4C	/ɔt/	10	231.2	165.6	154.7	132.8	157.8	138.3	/ɔt/	**180.89**
			/ɔt/	11	310.9	201.6	159.4	131.2	167.2	262.5		
			/ɔt/	12	300	132.8	145.3	129.7	248.4	86.72		
MEAN L_xOT IN ms FOR 12 VC PRODUCTIONS					**272.52**	**179.03**	**201.94**	**158.58**	**168.34**	**158**	MEAN L_xOT IN ms FOR VC PRODUCTIONS	**189.73**
RANGE OF L_xOT FOR VC PRODUCTIONS (MAXIMUM – MINIMUM L_xOT IN ms)					*(492.2-75)* **417.2**	*(232.8-110.9)* **121.9**	*(568.7-143.7)* **425**	*(295.3-101.6)* **193.7**	*(257.8-101.6)* **156.2**	*(310.9-86.72)* **224.18**	AVERAGE L_xOT RANGE FOR VC PRODUCTIONS	**256.36**
Maximum L_xOT in ms							*Minimum L_xOT in ms*					

APPENDIX G*ii*: THE ANALYSIS OF L_xOT (in ms) OF THE 12 $C_{(v)}VC$ PRODUCTIONS: A PRESENTATION OF THE L_xOT VALUES, MEAN L_xOT AND RANGE OF L_xOT FOR THE SIX CONTROL SPEAKERS AS WELL AS THE MEAN L_xOT VALUE AND L_xOT RANGE FOR THE CONTROL GROUP.

LARYNGEAL ONSET TIME (L_xOT) IN ms for $C_{(v)}VC$ PRODUCTIONS													
12 $C_{(v)}VC$ PRODUCTIONS					CONTROL SPEAKERS (N = 6)						MEAN L_xOT IN ms FOR $C_{(v)}V1C$, $C_{(v)}V2C$, $C_{(v)}V3C$, $C_{(v)}V4C$ CONTEXTS FOR CONTROL GROUP	CONTROL GROUP
					CONTROL SPEAKER 1	CONTROL SPEAKER 2	CONTROL SPEAKER 3	CONTROL SPEAKER 4	CONTROL SPEAKER 5	CONTROL SPEAKER 6		
PHONETIC CONTEXT	CONSONANT (*VOICED*)-VOWEL-CONSONANT ($C_{(v)}VC$)	$C_{(v)}V1C$	/bɑt/	1	201.6	33.20	442.2	312.5	214.1	82.03	/bɑt/	**159.68**
			/bɑt/	2	243.7	0	133.6	217.2	132.8	64.84		
			/bɑt/	3	215.6	0	78.13	309.4	156.6	36.72		
		$C_{(v)}V2C$	/bət/	4	234.4	107.8	392.2	254.78	132.8	180.5	/bət/	**167.2**
			/bət/	5	214.1	143.0	41.02	256.2	72.65	102.3		
			/bət/	6	218.8	114.5	93.75	289.1	70.31	91.41		
		$C_{(v)}V3C$	/bɪt/	7	264.1	0	45.31	387.5	90.62	122.7	/bɪt/	**190.04**
			/bɪt/	8	460.9	86.33	82.81	257.8	62.50	135.9		
			/bɪt/	9	329.7	51.95	442.2	232.8	102	265.6		
		$C_{(v)}V4C$	/bɔt/	10	121.9	139.1	70.70	256.2	67.19	67.97	/bɔt/	**150.60**
			/bɔt/	11	456.2	83.05	171.9	225.8	96.48	82.81		
			/bɔt/	12	143.7	33.59	343.8	196.9	92.58	60.94		
MEAN L_xOT IN ms FOR 12 $C_{(v)}VC$ PRODUCTIONS					**258.73**	**66.04**	**194.8**	**266.35**	**107.55**	**107.81**	MEAN L_xOT IN ms FOR $C_{(v)}VC$ PRODUCTIONS	**166.88**
RANGE OF L_xOT FOR $C_{(v)}VC$ PRODUCTIONS (MAXIMUM – MINIMUM L_xOT IN ms)					*(460.9-121.9)* **339**	*(143-0)* **143.0**	*(442.2-41.02)* **401.18**	*(387.5-196.9)* **190.6**	*(214.1-62.5)* **151.6**	*(265.6-36.72)* **228.88**	AVERAGE L_xOT RANGE FOR $C_{(v)}VC$ PRODUCTIONS	**242.38**
		Maximum L_xOT in ms				*Minimum L_xOT in ms*						

APPENDIX G*iii*: THE ANALYSIS OF L_xOT (in ms) OF THE 12 $C_{(vl)}VC$ PRODUCTIONS: A PRESENTATION OF THE L_xOT VALUES, MEAN L_xOT AND RANGE OF L_xOT FOR THE SIX CONTROL SPEAKERS (C1-C6) AS WELL AS THE MEAN L_xOT VALUE AND L_xOT RANGE FOR THE CONTROL GROUP.

LARYNGEAL ONSET TIME (L_xOT) IN ms for $C_{(vl)}VC$ PRODUCTIONS													
12 $C_{(vl)}VC$ PRODUCTIONS					CONTROL SPEAKERS (N = 6)						MEAN L_xOT IN ms FOR $C_{(vl)}V1C$, $C_{(vl)}V2C$, $C_{(vl)}V3C$, $C_{(vl)}V4C$ CONTEXTS FOR CONTROL GROUP	CONTROL GROUP
					CONTROL SPEAKER 1	CONTROL SPEAKER 2	CONTROL SPEAKER 3	CONTROL SPEAKER 4	CONTROL SPEAKER 5	CONTROL SPEAKER 6		
PHONETIC CONTEXT	CONSONANT(VOICELESS)-VOWEL-CONSONANT ($C_{(vl)}VC$)	$C_{(vl)}V1C$	/pɑt/	1	256.2	0	12.89	279.7	189.1	22.66	/pɑt/	**134.14**
			/pɑt/	2	301.6	0	0	212.5	118	31.25		
			/pɑt/	3	285.9	0	334.4	128.1	184.4	57.81		
		$C_{(vl)}V2C$	/pət/	4	176.6	0	26.56	268.7	190.6	64.84	/pət/	**123.35**
			/pət/	5	0	44.92	85.94	182.8	171.9	111.7		
			/pət/	6	245.3	0	12.89	306.2	167.2	164.1		
		$C_{(vl)}V3C$	/pɪt/	7	0	51.95	26.56	204.7	143.7	139.1	/pɪt/	**127.68**
			/pɪt/	8	284.4	5.47	91.53	217.2	165.6	141.4		
			/pɪt/	9	207.8	0	96.87	217.2	129.7	175		
		$C_{(vl)}V4C$	/pɔt/	10	0	0	222.7	275.0	87.5	39.84	/pɔt/	**112.07**
			/pɔt/	11	225.0	0	133.2	362.5	128.1	53.12		
			/pɔt/	12	0	12.50	112.5	164.1	107.4	93.75		
MEAN L_xOT IN ms FOR 12 $C_{(vl)}VC$ PRODUCTIONS					**165.23**	**9.57**	**96.34**	**234.89**	**148.6**	**91.21**	MEAN L_xOT IN ms FOR $C_{(vl)}VC$ PRODUCTIONS	**124.31**
RANGE OF L_xOT FOR $C_{(vl)}VC$ PRODUCTIONS (MAXIMUM – MINIMUM L_xOT IN ms)					*(301.6-0)* **301.6**	*(51.95-0)* **51.95**	*(334.4-0)* **334.4**	*(362.5-128.1)* **234.4**	*(190.6-87.5)* **103.1**	*(175-22.66)* **152.34**	AVERAGE L_xOT RANGE FOR $C_{(vl)}VC$ PRODUCTIONS	**196.30**
Maximum L_xOT in ms						*Minimum L_xOT in ms*						

APPENDIX H:

- **APPENDIX H*i*: THE ANALYSIS OF VOICE ONSET TIME (VOT) *(in ms)* OF THE 12 $C_{(v)}$VC PRODUCTIONS: A PRESENTATION OF THE VOT VALUES, MEAN AND ERROR VALUES OF VOT FOR THE SIX CONTROL SPEAKERS AS WELL AS THE MEAN VOT VALUE AND AVERAGE VOT ERROR VALUE FOR THE CONTROL GROUP**

- **APPENDIX H*ii*: THE ANALYSIS OF VOICE ONSET TIME (VOT) *(in ms)* OF THE 12 $C_{(vl)}$VC PRODUCTIONS: A PRESENTATION OF THE VOT VALUES, MEAN AND ERROR VALUES OF VOT FOR THE SIX CONTROL SPEAKERS AS WELL AS THE MEAN VOT VALUE AND AVERAGE VOT ERROR VALUE FOR THE CONTROL GROUP**

APPENDIX H*i*: **THE ANALYSIS OF VOICE ONSET TIME (VOT) *(in ms)* OF THE 12 $C_{(v)}$VC PRODUCTIONS: A PRESENTATION OF THE VOT VALUES, MEAN AND ERROR VALUES OF VOT FOR THE SIX CONTROL SPEAKERS AS WELL AS THE MEAN VOT VALUE AND AVERAGE VOT ERROR VALUE FOR THE CONTROL GROUP**

VOICE ONSET TIME (VOT) IN ms FOR $C_{(v)}$VC PRODUCTIONS												
12 $C_{(v)}$VC PRODUCTIONS				CONTROL GROUP AVERAGE VOT (AV) IN ms / VOT ERROR VALUE (EV) IN ms	AV / EV	CONTROL SPEAKER 1	CONTROL SPEAKER 2	CONTROL SPEAKER 3	CONTROL SPEAKER 4	CONTROL SPEAKER 5	CONTROL SPEAKER 6
PHONETIC CONTEXT	VOICED CONSONANT-VOWEL-CONSONANT ($C_{(v)}$VC)	$C_{(v)}$V1C	/bɑt/ 1	-51	AV	+20.31	-76.56	-45.31	+10.94	-117.2	-49.22
					EV	*5.31*	*0*	*0*	*0*	*0*	*0*
			/bɑt/ 2		AV	+21.88	-71.87	+13.28	-157.8	-135.9	-96.87
				0.68	EV	*6.88*	*0*	*0*	*0*	*0*	*0*
			/bɑt/ 3		AV	-37.5	-23.44	-10.94	+14.06	-98.44	-77.34
					EV	*0*	*0*	*0*	*0*	*0*	*0*
		$C_{(v)}$V2C	/bət/ 4	-54.07	AV	+7.81	+12.5	+7.81	-93.75	-185.3	-44.53
					EV	*0*	*0*	*0*	*0*	*5.3*	*0*
			/bət/ 5		AV	+26.56	0	-43.75	-112.5	-160.5	-18.75
				1.06	EV	*11.56*	*0*	*0*	*0*	*0*	*0*
			/bət/ 6		AV	-29.69	+10.94	+17.19	-165.6	-156.3	-45.31
					EV	*0*	*0*	*2.19*	*0*	*0*	*0*
		$C_{(v)}$V3C	/bɪt/ 7	-40.7	AV	+18.75	+21.88	-31.25	-90.62	-175	+8.59
					EV	*3.75*	*6.88*	*0*	*0*	*0*	*0*
			/bɪt/ 8		AV	+6.25	+9.38	+15.63	-146.9	-160.9	+23.44
				2.32	EV	*0*	*0*	*0.63*	*0*	*0*	*8.44*
			/bɪt/ 9		AV	+12.5	+20.31	+21.88	-118.7	-186.6	+18.75
					EV	*0*	*5.31*	*6.88*	*0*	*6.2*	*3.75*
		$C_{(v)}$V4C	/bɔt/ 10	-27.89	AV	+7.81	0	+23.44	-39.06	-110.9	-10.94
					EV	*0*	*0*	*8.44*	*0*	*0*	*0*
			/bɔt/ 11		AV	+12.89	0	+14.06	-107.8	-107.8	-10.16
				1.06	EV	*0*	*0*	*0*	*0*	*0*	*0*
			/bɔt/ 12		AV	+17.19	0	+12.50	-125.03	-101.6	+23.44
					EV	*2.19*	*0*	*0*	*0*	*0*	*8.44*
MEAN VOT(*x*AV) IN ms FOR 12 $C_{(v)}$VC PRODUCTIONS				-43.41	xAV	7.06	-8.07	-0.46	-94.4	-141.37	-23.24
AVERAGE VOT ERROR VALUE (xEV) IN ms FOR 12 $C_{(v)}$VC PRODUCTIONS				*1.28*	*xEV*	*2.47*	*1.02*	*1.51*	*0*	*0.96*	*1.72*

APPENDIX H*ii*: THE ANALYSIS OF VOICE ONSET TIME (VOT) *(in ms)* OF THE 12 $C_{(vl)}$VC PRODUCTIONS: A PRESENTATION OF THE VOT VALUES, MEAN AND ERROR VALUES OF VOT FOR THE SIX CONTROL SPEAKERS AS WELL AS THE MEAN VOT VALUE AND AVERAGE VOT ERROR VALUE FOR THE CONTROL GROUP

VOICE ONSET TIME IN MS FOR $C_{(VL)}$VC PRODUCTIONS												
12 $C_{(vl)}$VC PRODUCTIONS				CONTROL GROUP	AV	CONTROL SPEAKER 1	CONTROL SPEAKER 2	CONTROL SPEAKER 3	CONTROL SPEAKER 4	CONTROL SPEAKER 5	CONTROL SPEAKER 6
				AVERAGE VOT (AV) IN MS							
				VOT ERROR VALUE (EV) IN MS	EV						
PHONETIC CONTEXT	VOILESS CONSONANT-VOWEL-CONSONANT ($C_{(vl)}$VC)	$C_{(vl)}$V1C	/pɑt/ 1	+15.37	AV	+10.94	+12.5	+25	+14.06	+10.94	+12.5
					EV	0	0	0	0	0	0
			/pɑt/ 2		AV	+15.63	+17.19	+31.25	+10.94	+9.38	+9.38
				0.14	EV	0	0	0	0	0.62	0.62
			/pɑt/ 3		AV	+17.19	+14.06	+31.25	+9.38	+9.38	+15.63
					EV	0	0	0	0.62	0	0
		$C_{(vl)}$V2C	/pət/ 4	+18.82	AV	+17.19	+12.11	+48.44	+20.31	+27.81	+10.6
					EV	0	0	0	0	0	0
			/pət/ 5		AV	+14.06	+20.31	+26.56	+12.5	+9.38	+22.66
				0.07	EV	0	0	0	0	0.62	0
			/pət/ 6		AV	+15.63	+15.63	+31.25	+9.38	+10.94	+14.06
					EV	0	0	0	0.62	0	0
		$C_{(vl)}$V3C	/pɪt/ 7	+18.72	AV	+12.5	+14.06	+32.81	+14.06	+22.5	+15.63
					EV	0	0	0	0	0	0
			/pɪt/ 8		AV	+21.88	+14.06	+34.37	+17.19	+7.81	+16.41
				0.16	EV	0	0	0	0	2.19	0
			/pɪt/ 9		AV	+24.61	+18.75	+37.5	+10.94	+9.38	+12.5
					EV	0	0	0	0	0.62	0
		$C_{(vl)}$V4C	/pɔt/ 10	+21.27	AV	+24.22	+23.44	+35.94	+25	+18.75	+14.06
					EV	0	0	0	0	0	0
			/pɔt/ 11		AV	+40.62	+17.19	+25	+14.06	+9.38	+12.5
				0.1	EV	0	0	0	0	0.62	0
			/pɔt/ 12		AV	+13.28	+23.44	+43.75	+23.44	+9.38	+9.38
					EV	0	0	0	0	0.62	0.62
MEAN VOT (xAV)IN MS FOR 12 $C_{(vl)}$VC PRODUCTIONS				+18.54	xAV	+18.98	+16.9	+33.59	+15.11	+12.92	+13.76
AVERAGE VOT ERROR VALUE (xEV) IN MS FOR 12 $C_{(vl)}$VC PRODUCTIONS				0.12	xEV	0	0	0	0.1	0.49	0.1