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Using Time-Out to Treat Advanced Stuttering

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<u>Contents</u>	<u>Page</u>
Abstract .....	iii
List of Tables and Figures .....	iv
Introduction .....	1
Constitutional Factors in Stuttering .....	2
Environmental Influences on Stuttering .....	5
Treatment of Stuttering .....	6
Operant Conditioning to Treat Advanced Stuttering .....	10
Time-Out .....	12
Research Questions .....	22
Method .....	22
Results .....	27
Time-Out Treatment Effectiveness .....	27
Speech Rate .....	30
Factors Affecting Treatment Outcome .....	32
Nature of the Stuttering .....	39
Discussion .....	40
Appendix A – Time-out studies investigated by Prins & Hubbard (1988) .....	46
Appendix B – Informed consent form .....	48
Appendix C – Instructions given to study participants .....	49
Appendix D – Individual outcome data – treatment group .....	50
Individual outcome data – control group... ..	51
Appendix E – Correlations between treatment outcome and predictors prior to standardisation ... ..	52
Appendix F - Standard Multiple Regression Analysis of Treatment Outcome (difference scores) using all Predictor Variables Excluding Stuttering Severity .....	53
Appendix G – Percent acute stuttering behaviours for time-out and control participants over each condition ..	54
References .....	55

### Abstract

This study involved trialing an operant conditioning procedure known as *time-out*, as a treatment for adolescents and adults who stutter. Time-out requires individuals to pause briefly after stuttering and to resume talking after a pause in this case, of five seconds. A randomised control group design was used to evaluate the effectiveness of time-out treatment. Sixty participants were randomly assigned to either a time-out treatment or control group. The results demonstrated that individuals who stutter are highly responsive to time-out treatment. The impact of the severity of the stutter, a person's age, previous treatment, and the nature of the stutter on treatment outcome, were also investigated. Baseline severity was a strong predictor of treatment outcome, and to a lesser degree, previous treatment and speech rate were found to share some influence over treatment success. In addition, there was an unexpected change in the stuttering topography over the experiment conditions.

<u>List of Tables and Figures</u>	<u>Page</u>
Table 1 – Baseline variable means, standard deviations and range for groups. ....	24
Table 2 - Percent syllables stuttered during baseline, treatment and post-treatment for time-out and control groups .....	28
Table 3 - Speech rate (syllables per minute) taken during baseline, treatment and post-treatment for time-out and control groups .....	31
Table 4 - Correlations between treatment outcome and predictors . ....	34
Table 5 - Standard multiple regression analysis of treatment outcome (difference scores) using all predictor variables .....	36
Table 6 - Standard multiple regression analysis of treatment outcome (percentage change) using all predictor variables .. ....	38
Figure 1 - Mean percent acute stuttering behaviours for time-out and control participants for each condition . ....	40

## Using Time-Out to Treat Advanced Stuttering

### Introduction

Stuttering can be a debilitating disorder, adversely affecting most aspects of an individual's life. It can cause significant frustration and impact on the emotional well being of an individual. The young child who stutters may become disheartened with the persistent breakdowns in communication and refrain from interacting with others. An older child may avoid asking questions in class and fear ridicule from other children. The embarrassment and shame often felt by adolescents may severely affect their development of self-esteem, identity and personal relationships, and perhaps even restrict their future employment prospects. As an adult, the person who stutters may avoid occupations requiring a high level of verbal skills and interaction, which in turn, may lead to feelings of apprehension and failure (Bloodstein, 1995; Guitar, 1998).

At any given time, approximately 1% of the population stutters (Bloodstein, 1995). According to the Australian Bureau of Statistics, the population of Australia is in the vicinity of 19,400,000; therefore, there exist approximately 194,000 individuals across Australia who have a stuttering disability (Australian Bureau of Demographic Statistics, June 2001).

A well established fact about stuttering is that it tends to run in families. Research in the area of stuttering and genetics suggests that individuals who stutter have an inherited predisposition to stutter (Guitar, 1998). It is generally accepted that the onset of stuttering is most likely to occur between the ages of 2 and 5 years (Andrews, et al., 1983), and typically begins as rapid, irregular repetitions of parts of words (Ambrose & Yairi, 1999;

Guitar, 1998). By contrast, normal disfluencies are effortless and relaxed single syllable, whole word repetitions, interjections and revisions (Guitar, 1998). Advanced stuttering is described as effortful speech, including prolongation of sounds, blocks, and signs of struggle (Ambrose & Yairi, 1999). These disruptions in fluency occur despite speakers knowing exactly what they wanted to say (World Health Organisation, 1977). Secondary behaviours such as eye blinking, head jerking, mouth distortions and hand movements may also accompany the stutter (Silverman, 1992). This progression from acute to more advanced stuttering may occur quickly or over many years (Yairi, Ambrose, & Niermann, 1993).

Researchers over the years have provided many theories to help explain the aetiology of stuttering (for a comprehensive account see Bloodstein, 1995). There are essentially two schools of thought – one suggesting that children who begin to stutter possess a *constitutional* predisposition, and another claiming that stuttering is a *learned response* resulting from an interaction between a child and his or her environment. A brief elaboration of these views follow, beginning with constitutional factors.

#### Constitutional Factors in Stuttering

It is generally agreed that stuttering is inherited, with up to two thirds of stuttering clients reporting that they have a relative affected with the disorder (Ambrose, Yairi & Cox, 1993). Much of the research investigating genetics and stuttering has involved family studies where interviews are conducted with family members of stuttering clients (Andrews & Harris, 1964); twin studies (Howie, 1981), comparing the incidence of

stuttering in twins, or adoption studies (Bloodstein 1995). These studies indicate that people who stutter have a genetic predisposition to stutter.

In order to understand these constitutional factors, studies have been conducted to compare individuals who stutter and those who do not, in terms of their cognitive ability (e.g., Yairi, Ambrose, Paden, & Throneburd, 1996), sensory-motor coordination (e.g., Neilson & Neilson, 1987), speech motor control (e.g., Adams, Freeman & Conture, 1984; Kent, 1983; Van Riper, 1982), speech and language development (Nippold, Schwarz, & Jescheniak, 1991; Nippold, 1990), and recently through brain imaging (Uffen, 2000). A number of theoretical perspectives have emerged from this research (Bloodstein, 1995).

The notion that stuttering is the product of a breakdown within the speech motor control system has received strong support from many researchers (e.g., Kolk & Postma, 1997; Packman, Onslow, Richard & Van Doorn, 1996; Perkins, Kent and Curlee, 1991; Van Riper, 1982; Wingate, 1988, and Zimmermann, 1980). Description of the various hypotheses is beyond the scope, and not the focus of this study. However, the models of Perkins et al. (1991) and Packman (1996) will be briefly described by way of example.

Perkins, Kent, and Curlee (1991) believed that fluent speech required synchronic integration of linguistic and paralinguistic (prosody, emotion and speaker intent) components of speech. These components were thought to be processed by different neural systems, and could be compromised by genetic constraints, brain injury, or competition for processing capacity. A disruption within the linguistic or paralinguistic components, or during the assembly of the components' information to form the speech motor plan, gave rise to disfluency. These disfluencies were said to transform into stuttering behaviours if the speaker felt pressured to begin, continue, or to accelerate an



utterance when loss of control was experienced. These researchers suggested that the pressure may be internal or external to the speaker; for example, excitement may cause increased internal pressure, while a situation requiring a quick answer may be a form of external pressure.

The fundamental principles of the Variability Model developed by Packman et al. (1996) were derived from Zimmermann's (1980) Articulatory Dynamics Model and Wingate's (1988) Modified Vocalisation Hypothesis. People who stutter were hypothesized by Zimmermann to possess unstable speech systems prone to breakdown through extreme variability within the system. It was believed that the articulatory structures operated within certain ranges of variability, in terms of their positioning, timing, and speed, and that stuttering occurred when these ranges were exceeded, thus breaking down the coordination of the articulatory system. Prosody, specifically the production of syllabic stress contrasts, was offered by Packman et al. (1996) as a source of this variability. Wingate had previously suggested that prosody was the key to stuttering. This conclusion was drawn from observations that stutter-free speech was obtained by manipulating the normal prosodic patterns of speech, for example singing, whispering, using delayed auditory feedback (which altered the speech quality by reducing speech rate and prolonging vowels), changing pitch, choral reading or speaking to a beat.

Packman et al. (1996) proposed that syllables with increased stress typically carried a higher information load, thus requiring precise control and coordination between the respiration, phonation and articulation systems. Through a study which involved acoustic analysis of samples of stutter-free speech pre and post treatment, these researchers observed that as well as an increase in fluency following treatment, speakers

were producing speech comprising less variable vowel durations (Packman et al., 1996). Thus, it was hypothesized that variation in syllable length disrupted the stability of the speech motor system resulting in stuttering, and that if a person who stuttered was to speak with more consistent vowel duration, such as is in the case when speaking with a beat or delayed auditory feedback, or by using prolonged speech, fluency would result due to the increased stabilising effects.

Further, these researchers used this hypothesis to help explain the onset of stuttering. Packman et al. (1996) had observed that all children began talking by placing equal weight and timing on syllables, and as the speech developed, the pattern became more adult-like, where different emphasis is placed on different syllables within an utterance. Thus these researchers concluded that the onset of stuttering was a result of the inability of a child's speech motor system to cope with the demands of prosodic development, specifically the production of linguistic stress contrasts.

#### Environmental Influences on Stuttering

The most influential account of stuttering as learned behaviour is Wendell Johnson's "Diagnosogenic Theory" (1942). According to this theory, parents create their child's stuttering problem by drawing attention to normal disfluencies and hesitations, and in turn, this attention causes a build up of anxiety in the child as they anticipate being interrupted (Onslow, 1996). Further environmental disruptions such as the interruption from others while speaking, parents displaying signs of impatience or a lack of attention, and speaking in competition with siblings, are said to exacerbate the problem (Prins, 1983). This Diagnosogenic Theory has had, and continues to have, a powerful influence

on the way researchers and clinicians regard stuttering in terms of its origin, the factors that determine its continuation, and in the way it is treated.

### Treatment of Stuttering

In Australia, the most widely used treatment approach for early stuttering is based on operant conditioning principles (The Lidcombe Program). This program was developed by Onslow and colleagues in the mid-1980s and directly targets stuttering behaviours, rather than the causes of stuttering. Parents are instructed to provide verbal contingencies following their child's stuttered and fluent utterances, initially during set times and then as the stuttering decreases, throughout the day within the child's everyday activities (Lincoln & Harrison, 1999). Considerable research into the program has been conducted, and has revealed that low levels of stuttering are achieved and maintained for many years following inclusion in the program (Rousseau, Packman, Onslow, Dredge & Harrison, 2002).

Conversely, in other countries such as the United States, stuttering management for young children is not usually managed directly. Attanasio, Onslow and Menzies (1996) have suggested that that these opposing treatment practices are a result of Australian clinicians being influenced by behavioural models set up by Ingham and Andrews for the treatment of adults, while American clinicians have been influenced by academics who stuttered. Thus in America, management tends to follow Johnson's diagnosogenic theory which advocates that the development of an intractable stutter is caused by drawing attention to the behaviour. Treatment typically involves firstly ignoring the stutter, and secondly by identifying the stuttering child's environmental demands and pressures in order to eliminate them, thus allowing fluent speech to occur

(Prins, 1983). Information posted on the National Institute on Deafness and Communication Disorders website (2002) includes recommendations to parents to provide a relaxed home environment, to refrain from criticizing their child's speech or punishing their child for any disfluencies, to listen attentively to their child, to speak slowly, and to reduce any speech competition. At no time is the stuttering behaviour targeted or punished, as this is thought to reinforce avoidance strategies, a fear of stuttering and other maladaptive reactions (Siegel, 1998).

Current approaches with adults and adolescents include a number of cognitive behavioural treatments, psychotherapy, or both. Management is more complex because stuttering behaviour is regarded as a well-established motoric response to the individual's continued communicative failures, their belief system, fears, or inner conflicts (Siegel, 1998). Thus psychotherapy treatment aims to help an individual deal with these problems and to adjust to the disorder. Videotaping may be used to help people identify episodes of stuttering, both core and secondary behaviours, their attitudes and feelings toward their stuttering (Conture, 1990; Prins, 1997; Van Riper, 1982), and any avoidance strategies (Bloodstein, 1975). Individuals are encouraged to reduce the intensity of their anxiety and emotional responses to stuttering by speaking openly about the disorder, to seek out feared situations, and to stutter openly in order to gain a sense of empowerment and conscious control over the behaviour (Martin, 1993).

Cognitive behaviour therapy, by contrast, deals directly with the stuttering behaviour (Onslow & O'Brian, 1998). For example, Van Riper (1982) suggests that speakers pause after a stuttered word and then repeat it (cancellations), or pull out during the stutter and then ease into the word (pull-outs). Other clients may be taught to reduce

the duration of the stutter (Conture, 1990), or to use “smooth and loose” speech techniques to “override” old motor responses (Prins, 1997). Similarly, in Australia, the most predominant method of treating advanced stuttering is based on a programme that teaches individuals to speak with gentle onsets and drawn out vowel durations (Onslow & O’Brian, 1998). This prolonged speech technique emerged from the work by Goldiamond and colleagues in the 1950s (Siegel, 1998). It was discovered that by delaying speakers’ speech signal slightly, the rhythm of their speech altered and they became stutter-free. The stutter-free speech was referred to as “prolonged speech” because speakers who used the delayed auditory feedback (DAF) device were observed to extend their vocalisations and vowel duration (Onslow & O’Brian, 1998). The programme specifically comprises three phases: 1. instatement of stutter-free speech, 2. generalisation of this speech beyond the clinic, and 3. maintenance. Instatement involves teaching clients various strategies to help eliminate stuttering. For example they are taught techniques such as soft contacts, where increased airflow helps to soften all “hard” sounds, for example plosives or affricates; gentle onsets, which is a skill used to glide into each new utterance, and continuous vocalisation. Speech rate is also targeted, where clients begin to practise speaking at a rate of 90 syllables per minute (the normal speech rate is: 162-230 syllables per minute, Andrews & Ingham, 1971). When used correctly, these techniques effectively induce stutter-free speech.

Unfortunately, these strategies also result in an unnatural sounding speech quality, and it is this speech quality that has been offered as an explanation for the reported high incidence of relapse encountered in the persons’ social environment (Onslow & Packman, 1997; Perkins, 1992). For example, 72% of clients in a study by Andrews and Craig

(1988), reported that they were stuttering at a “personally unacceptable level” following treatment and a time of improvement. Forty percent of participants in a study by Craig and Hancock (1995) were also unable to maintain fluency following a prolonged speech programme. They believed their relapse was due to feeling embarrassed with the speech quality and 79% felt that the pressure to increase their speech rate within real life situations, had been the cause of their relapse.

Instatement and generalisation of this novel speech pattern, are either carried out intensely within a group over a two-week period, or individually over many weeks or months. The treatment generally relies on a high degree of expertise from the clinician. It also is time consuming, requiring up to 100 clinical hours, is mentally taxing for the participant as well as the clinician, and requires high levels of motivation and self monitoring by the client (Onslow, 1996).

The reported high level of attrition and the programme’s high number of clinic hours and intensive nature have caused the closure of many intensive group-based prolonged speech treatment programmes in Australia, including the University of New South Wales’ Programme. This facility housed the most comprehensively researched treatment programme for advanced stuttering, and served as a training centre and knowledge base for clinicians (Harrison, Onslow, Andrews, Packman, & Webber, 1998).

Given these limitations of the more complex cognitive behavioural therapy approach, simple operant conditioning methodology presents as an attractive alternative to the management of adolescents and adults who stutter.

### Operant Conditioning to Treat Advanced Stuttering

Treatment of stuttering using behavioural methodology emerged following Goldiamond's discovery that stuttering could be reduced by environmental consequences (Siegel, 1998). Specifically, Goldiamond demonstrated that stuttering frequency could be reduced through the use of the response-contingent consequences of noise, shock, and delayed auditory feedback (Martin, 1993). While DAF was seen to shape speech in order to induce fluency, the response contingent noise and shock directly targeted the stutter, resulting in stutter-free speech (Flanagan, Goldiamond, & Azrin, 1958). Following this discovery, there was considerable research conducted investigating response contingent stimuli and stuttering. Stuttering was observed to be operant-like in its nature as it was found to respond to environmental stimuli, by either increasing or decreasing, depending on the occurrence of the type of reinforcing stimulus (Ingham, 1984). In addition, published case studies showed that in contrast to the prolonged speech programs, simple operant conditioning methodology was simpler to administer, more cost effective, less labour intensive and did not result in clients using an unnatural speech pattern (Hewat, O'Brian, Onslow & Packman, 2001).

Operant conditioning, first described by Skinner in 1938 (cited in Martin & Pear, 1996), is a form of learning whereby a reinforcing stimulus (reward or punishment) is presented contingent on a target behaviour, which in turn either increases the desired behaviour or reduces it, depending on whether a reward is sought or punishment avoided (Lloyd, Mayes, Manstead, Meudell, & Wagner, 1984). Stuttering is regarded as one such behaviour that can be modified by its consequences. Ingham (1984), a leading researcher

in this field, qualified that it does not mean that operant conditioning has created the stuttering behaviour, but that it can reduce the severity.

There have been many reviews of studies investigating the ameliorative effects of operant conditioning on stuttering (Bloodstein, 1995; Costello & Hurst, 1981; Costello & Ingham, 1984; Ingham, 1982; Prins, 1988). In 1988, Prins and Hubbard conducted an analysis of all published experimental studies in which stuttering was subjected to response contingency paradigms. Sixty eight studies in total were examined. Over 70% demonstrated that stuttering was modified by either the presentation or withdrawal of a reinforcing stimulus administered closely following the occurrence of the stuttering behaviour. Authors cited in the review altered the operant conditioning components in different ways in order to investigate what effects these manipulations had on both stuttered and stutter-free speech. For example, the effect on stuttering by the presentation of a variety of positive stimuli was investigated. Examples of positive stimuli were money, tokens and examiners saying the word “right” contingent on stutter-free speech. Other studies involved experimenting with aversive stimuli presentation, such as inflicting electric shocks or a loud noise on the individual when they stuttered. In other cases, neutral reinforcers were presented, such as a light, an audible click, or the verbal statement “tree”. Withdrawal of stimuli, either positive or aversive, was also investigated. Prins and Hubbard's (1988) analysis revealed that over 60% of these studies showed a positive outcome. However, they had expected the results to be more consistent than they were, given the initial reports of the dramatic influence that operant conditioning had on stuttering. By contrast, one group of studies examined showed overwhelming predictability and consistency. These studies had investigated the effects on stuttering by



response contingent time-out from speaking, and well over 90% of these experiments obtained significant results (Appendix A).

### *Time-Out*

The first study to examine the effect of response contingent time-out on stuttering was conducted by Haroldson, Martin, and Starr (1968). Four adults who stuttered were treated individually and instructed to cease talking when a red light was illuminated, and to resume speaking once the light was switched off. An examiner activated the light following each moment of stuttering. The results showed that there was at least an 88% reduction in stuttering for each participant. Subsequent replications of this study described similar results or even stronger positive trends (Costello, 1975; James, 1983; James, Ricciardelli, Rogers, & Hunter, 1989; Onslow & Packman, 1997; Martin & Haroldson, 1982).

In 1972, Martin and colleagues conducted another experiment which trialed time-out with pre-school children with equally impressive results (Martin, Kuhl & Haroldson, 1972). The experiment involved conversations between a puppet and the children, with the puppet disappearing for 10 seconds each time a child stuttered. Stuttering was shown to markedly decrease, with the effect still present at a one year follow-up. Onslow and colleagues at the Australian Stuttering Research Centre have continued research in this area and have subsequently developed a programme using response contingent time-out to treat childhood stuttering. This programme is used extensively throughout Australia and is described in various publications (e.g. Harrison & Onslow, 1999; Onslow, 1996).

Within the operant conditioning framework, the time-out procedure is regarded as a form of punishment. Punishment is defined as “an aversive stimulus consequence that has

the effect of decreasing the strength of an emitted response” (Dworetzky, 1985).

Punishment can be delivered by either presenting an aversive stimulus contingent on the undesired behaviour, or by withdrawing a positive one, again contingent upon the target behaviour (Martin & Pear, 1996). Time-out is regarded as both an aversive stimulus and a form of withdrawal from a positive reinforcer (Costello-Ingham, 1993). The initial interruption from speaking is seen as a withdrawal from the positively rewarding act of speaking, and the subsequent imposed period of silence is regarded as the aversive stimulus.

As well as proving to be the most effective response contingent procedure in reducing stuttering, time-out has also achieved better results when compared with the traditionally cited fluency inducing conditions, such as DAF and syllable-time speech (Martin & Haroldson, 1979). In their laboratory study, Martin and Haroldson compared five different procedures known to ameliorate stuttering, namely, response contingent time-out, speaking with the beat of a metronome, delayed auditory feedback, response contingent white noise and the presentation of the word “wrong” at the moment of stuttering. Time-out from speaking produced the greatest decrease in stuttering (an average of 75% reduction) across the largest number of participants (100% of participants). Conversely, only 60% of participants responded to “wrong” (on average stuttering reductions were less than 50%). White noise as a stimulus also achieved about a 50% reduction for about 80% of participants; speaking with a metronome reduced stuttering by up to 75% for 95% of participants, and DAF by only 54% for 85% of participants. It was noted that the metronome and DAF conditions altered individuals’ prosody, pitch and loudness, while no change was observed for the response contingent

conditions. The potency of time-out was further demonstrated in a study by Martin and Haroldson (1977). This investigation involved showing 20 individuals, who stuttered, a video of a person with a severe stutter successfully responding to contingent time-out. All 20 of the participants experienced a significant drop in stuttering just by observing the video.

However, despite time-out appearing to be the most promising behavioural methodology in the treatment of stuttering (Costello, 1975; Ingham, 1993; Bloodstein 1995), the mechanism that accounts for its success, both in terms of the procedure and the theoretical underpinnings, remains unknown. For example, is the effectiveness of time-out influenced by such factors as the correct identification of each stuttering behaviour, how close the interruption is to the stutter, whether the experimenter or the participant delivers the interruption, which stuttering behaviour is targeted, or if the interruption is actually perceived as a penalty?

Accurate identification of stuttering episodes does not emerge as a crucial factor in the successful reduction of stuttering (Adams & Popelka, 1971; James, 1976, 1981a, 1983; James et al., 1989, and Martin & Haroldson, 1982). James et al. (1989) found that even when only 33% of stuttering moments were interrupted, treatment resulted in significant reductions. In an earlier study by James (1983), results were found to be comparable between participants who self-administered time-out (SATO) with low rates of accurate identification of their own stuttered episodes (in the vicinity of 46%) and the accuracy of the experimenter administered time-out (EATO) of 80%. Likewise, Martin and Haroldson (1982) agreed 96% of the time with one of their participants who established a 32% reduction in stuttering; and only 45% of another participant who

achieved a 93% reduction. Despite the reported inaccuracies, self administered time-out appears to have durable effects. Preliminary data from a study by Hewat, O'Brian, Onslow, and Packman (2001) indicated that the clinical benefits from self-imposed time-out for a single participant were maintained at a six month follow-up. James (1981a) also found that a participant in his investigation had maintained treatment gains after 12 months.

Targeting just one type of stuttering behaviour when delivering time-out, does not appear to affect the overall reduction of stuttering (Costello & Hurst, 1981). In Costello and Hurst's study, stuttering behaviours not targeted for time-out still decreased significantly. Specifically, one participant's repetitions were targeted and their blocks and inhalation interjections decreased, another participant's jaw tremors were targeted and reduced along with their repetitions, and for the third participant, repetitions, prolongations and blocks decreased when only prolongations were the target.

The aversiveness of the imposed period of silence has been another avenue of enquiry for researchers attempting to understand the operation of the procedure (Adams, 1980; Biggs & Sheehan, 1969; Costello, 1975; James & Ingham, 1974; James et al., 1989; Wingate, 1976; Yonovitz & Shepard, 1977). Yonovitz and Shepard (1977) investigated the effects of time-out on participants' heart rate and galvanic skin response (which are known to reflect emotional arousal). It was found that participants tended to be relaxed during the period of silence, many reporting that it gave them time to think. Likewise, six out of eight participants in a study by Adams and Popelka (1971) interpreted the pause as a chance to relax, and attributed the relaxed state to the improved fluency, while the remaining two participants felt time-out served as a cue to strive for more fluent speech.

Similarly, increasing the duration of imposed silence was found not to affect results. James (1976) administered 1, 5, 10 and 20-second time-out durations, the premise being the greater the punisher the greater effect, and found that these differing lengths had little influence on the effectiveness of time-out. It was concluded “that the punishing effects of the time-out from speaking procedure were predominantly due to its involving contingent interruption of speaking and that the actual duration of the period of silence was comparatively unimportant” (James, 1976, p 206). This notion of contingency was investigated further by James (1981b) who found that contingent time-out led to a significant reduction in stuttering frequency while non-contingent time-out did not. In 1991, Gow, Ingham, and Prins suggested that perhaps it was not the contingency factor, but just the predictability of time-out that achieved the required outcome. However, this theory was not validated, as once again, it was found that only clients who had received contingent time-out responded to the approach, while neither regular nor irregular time-out delivery produced comparable results.

Thus it appears that the initial component of time-out, namely contingent interruption of speech, is the critical factor in stuttering reduction (Costello-Ingham, 1993). Following investigation of the literature, Costello noted that a variety of stimuli, such as response contingent loud noise, burst of DAF, verbal reprimands or an electric shock, could convincingly reduce stuttering behaviours as long as the stimulus was arranged to occur immediately following the moment of stuttering (Costello 1975). Biggs and Sheehan (1969) suggested that the contingent interruption was best explained in terms of a “distraction” effect rather than a punisher. By contrast, Cooper, Cady, and Robbins (1970) and Wingate (1976) maintained that the stimulus simply served to draw an

individual's attention to the stutter. Then once alerted, the individual created alternative internal consequences for the act through self regulation, thereby exercising control over the behaviour resulting in increased fluency (Prins, 1993).

The reason why response contingent time-out is the most effective operant conditioning procedure stimulus when compared to other stimuli, such as loud noise, audible clicks, electric shock, etc., remains unresolved. It could be attributed to the intrinsic nature of the stimuli. Each stimulus provides a signal that there has been a stutter, but then all stimuli except time-out distract or prevent the individual making the necessary adjustments to their speech to enhance fluency. By contrast, the period of silence associated with time-out creates the opportunity for the adjustments and in effect even small amounts of time are still sufficient. The participants in James' (1983) study were given the freedom to determine the time-out duration and interestingly all chose times less than two seconds in duration.

Therefore, in light of the preceding research, it can be concluded that in a clinical setting, contingent interruption of stuttering will typically result in increased fluency, with time-out proving to be the most consistent and predictable stimulus in terms of treatment outcome. In addition, the effects of this interruption are not altered by how accurately the time-out is delivered, as seen in the studies by Adams and Popelka (1971), James (1976, 1981a, 1983), James et al. (1989), and Martin and Haroldson (1982). These studies showed significant reductions in stuttering even when only about a third of the stutters were accurately identified. Likewise, this procedure is effective regardless of whether an individual self-administers the time-out or whether it is delivered by a clinician. It also remains effective whether the interruption is perceived by the individual as a distraction,

signal or penalty. Lastly, time-out reduces all stuttering types regardless of the behaviour targeted.

Despite strong evidence demonstrating that time-out is the most potent operant conditioning procedure regardless of the accuracy of administration, how it is perceived, or which stuttering behaviour is targeted, the fact remains that individuals exposed to time-out respond with varying degrees of success. Therefore, investigating factors associated with the participants may help to determine which variables account for the disparity between individuals' responsiveness to the treatment. Siegel (1998) suggested a number of possibilities. One explanation offered was that time-out may be more effective for those individuals who are motivated to suppress the behaviour, compared with those who are not as concerned by their stutter.

Another possibility offered by James, et al. (1989) and Siegel (1998), was that perhaps some individuals were better than others in using the period of silence to access previously-learned strategies to manage stuttering. James et al. (1989) speculated that individuals who responded well to time-out may be making "more effective use of their fluency skills they possessed than would ordinarily be attained in the absence of such contingencies" (p 604). Further, James suggested that those individuals who did not respond to time-out as well as others may benefit from help accessing fluency. To test this hypothesis, response contingent time-out was delivered to a group of 20 individuals; then, depending on their initial responsiveness to the treatment, they were separated into either a low response (LR) or high response (HR) group. The LR group were taught strategies to enhance their fluency, and then re-exposed to time-out. Results showed that these once low responders were now responding as well as the original high responders. Four months

later all 20 participants were again exposed to time-out contingencies and this time all responded equally.

In light of this research, it may be hypothesized that individuals, who have been taught fluency-inducing skills prior to exposure to time-out, should respond far more favourably to the treatment than those who have not. Siegel (1998) proposed to investigate this hypothesis by conducting a study that set up distraction tasks during the time-out period of silence. If the hypothesis is correct, the participants' shift in focus will prevent them from rehearsing previously learned strategies for dealing with stuttering, thus they will not respond to the time-out.

Interestingly, James et al. (1989) also found that the high responders in the study tended to be individuals with more severe stutters. It was hypothesised that the LR, who were people with milder stutters, were using all "fluency reserves" (p. 604) to achieve less stuttering, perhaps due to their response to social penalties in their natural environment, and therefore could not improve much more in the contingent environment without the aid of additional fluency strategies. By contrast, individuals with severe stutters (HR) might not be exposed to penalties for stuttering in their natural environment, thus not making maximum use of their existing fluency skills until exposed to time-out in the clinical setting.

It is not known whether the effects of severity reported by James et al., were the direct result of the stutter frequency alone, or if the nature of the stutter also played a part. Measures reported in James et al (1989) paper were expressed in terms of syllables stuttered only. Likewise, the seventeen time-out papers investigated by Prins and Hubbard (1988) did not consistently report thorough descriptions of participants' stuttering



behaviours or frequency counts. Instead they mentioned either criteria for inclusion (e.g. >4% syllables stuttered), or reported a severity category such as mild-moderate, and made limited description of the nature of the stuttering behaviour (see Appendix A). Therefore, it is difficult to determine what degree of influence the nature of the stutter, the frequency of the stuttering behaviours, or both, have on the treatment outcome.

By contrast, in the paediatric population, Onslow and colleagues have conducted several studies demonstrating the power of time-out in the elimination of stuttering in children, and have described the severity both in terms of stuttered syllables and, stuttering topography (Harrison & Onslow, 1999; Jones, Harrison, Onslow & Packman, 2000; Lincoln & Onslow, 1997; Onslow, Packman, Stocker, van Doorn, & Siegel, 1997). For example, in a study involving three school aged children, Onslow et al. (1997) found that only two of the children responded to the behavioural effects of time-out, and that the child who did not respond had initially presented with more effortful speech characterised by frequent sound prolongations, blocks and grimacing. In addition, in their clinical experience, Onslow and colleagues have found that children with more advanced stuttering behaviours often needed reinforcement for stutter-free speech until they began to display more acute types of stutters rather than blocks, and then responded to the response contingent time-out programme (Onslow, 1996). Consequently, these researchers have suggested that time-out may be more effective with younger adults, who present with acute types of stutters.

In light of this research, it would be expected that if a person presenting with a high proportion of acute types of stuttering behaviour, was exposed to response contingent time-out, they would experience a significant reduction in stuttering. Conversely,

someone producing mainly advanced stuttering behaviours would not be expected to respond as well to the same treatment. In addition, in accordance with findings by James et al. (1989), a better response would be expected from individuals experiencing more severe stuttering, than those with mild stuttering, as would those who had received previous therapy comprising fluency-enhancing techniques.

Therefore, this study was carried out to demonstrate the effectiveness of response contingent time-out as a treatment for stuttering in adolescents and adults, and to determine if factors such as stuttering severity, topography, age, and treatment history influenced treatment outcome. In addition, it was conducted to possibly extend an interest in using time-out as a powerful alternative treatment to the existing prolonged speech programmes, and to stimulate more research in its operation and success in order to understand the factors influencing its effectiveness.

A randomised control group design with baseline, treatment and post-treatment phases was used to investigate the effectiveness of time-out in its ability to establish fluency. A multiple regression analysis was then conducted to identify what factors were associated with the treatment outcome. Independent variables were stuttering frequency, speech rate, type of stutter, age, duration of previous treatment, and whether treatment was received during the year prior to this experiment. Treatment outcome was the dependent variable.

Specific research questions were:

1. Does time-out treatment reduce stuttering in adolescents and adults?
2. Do the outcomes of time-out treatment vary in relation to the type and frequency of stuttering?
3. Do outcomes vary in relation to amount and recency of previous treatment?

## Method

### Participants

Sixty people with a stuttering disorder volunteered to participate in the study. They were recruited from Speech Pathology clinics in the community and advertisements through publications of the Western Australian Speak Easy Association. Principal selection criteria included being over 13;0 years of age and presenting with any occurrence of an audible sound or syllable repetition, a silent or audible prolongation, or audible activity signifying struggle (Wingate, 1964).

Exclusion criteria for this study were developmental disability, neurological impairment or uncorrected vision or hearing. Each participant was informed in writing that they were under no obligation to remain in the study and free to leave at any time (see Appendix B). Participants were randomly assigned to either an experimental *time-out* or *control* group.

Table 1 displays stuttering levels (percent syllables stuttered), speech rate (syllables per minute), stuttering topography, age range, and treatment history (previous treatment in months and treatment in the last year) for each group. At baseline as a group

participants presented with moderate levels of stuttering, but ranged from mild (~1%SS) to very severe (~28%SS). Stuttering behaviours were defined as being either *acute* stuttering, comprising urgent repetitions of monosyllabic words, part-words and sounds; or *advanced* stuttering, including all silent and audible prolongations and blocking behaviours (drawn from Guitar, 1998, and Ambrose & Yairi, 1999). The groups did not differ significantly in severity, speech rate, stuttering topography, age or treatment history. Participants predominantly exhibited advanced stuttering behaviours; however, the ratio of acute to advanced stuttering behaviours did differ amongst individuals.

Comparisons between the two groups were conducted using the Mann Whitney U Test. This non-parametric test was used because data were not normally distributed based upon tests for skewness (Tabachnick & Fidell, 2001), and homogeneity of variance, investigated using the Fmax procedure (Keppel, 1991), was violated. Assumptions of independence and random selection were met. Comparison test results indicated that the two groups did indeed present with similar baseline stuttering frequency ( $U = 401.5$ ,  $p > .05$ ) and topography ( $U = 362.5$ ,  $p > .05$ ), speech rate ( $U = 330.5$ ,  $p > .05$ ), age range ( $U = 418.0$ ,  $p > .05$ ), and treatment history (previous treatment:  $U = 362.0$ ,  $p > .05$ , treatment in the last year:  $\chi^2 = 3.59$ ,  $p > .05$ ).

Table 1  
*Baseline Variable Means, Standard Deviations and Range for Groups*

Variables	Time-Out Participants (n = 30)	Control Participants (n = 30)
<b>Percent Syllables Stuttered</b>		
<i>M</i>	5.82	4.85
<i>SD</i>	6.40	4.96
Range	1.23 – 28.27	0.67 – 23.3
<b>Syllables Per Minute</b>		
<i>M</i>	211	236
<i>SD</i>	57	51
Range	70 – 296	107 – 317
<b>Percent Acute Stuttering Behaviours</b>		
<i>M</i>	19.58	31.95
<i>SD</i>	15.95	29.48
Range	0 – 66.7	0 - 100
<b>Age (years)</b>		
<i>M</i>	31.9	32.9
<i>SD</i>	12.9	12.05
Range	16 - 61	17 – 61
<b>Previous treatment (months)</b>		
<i>M</i>	26.2	35.63
<i>SD</i>	27.6	35.34
Range	0-120	1 - 120
<b>Treatment in last year</b>		
Yes	14	7
No	16	23

### Equipment

Participants' speech samples were recorded using a Sony audio tape recorder, Model Number TCM 5000 EV, with a Sony electret condenser lapel microphone. The time-out apparatus consisted of a red light on the top of a square black platform (18 cm x 18 cm x 7 cm), with the control switch attached by a 2 metre cord. The control switch was operated by the experimenter who pressed it for five seconds, contingent on each stutter. A Dalfar Speechmate SDC-01 button-press counting device (Dalfar Pty Ltd, Sydney Australia) was used to measure percent syllables stuttered and syllables per minute for each speech sample.

### Procedure

Experimental sessions were held in participants' homes. Rooms were quiet with no distractions. The participant and experimenter sat opposite each other at a table. The time-out apparatus was placed in the middle of the table for the time-out participants. The duration of each session lasted approximately two hours, regardless of whether the participant was receiving treatment or acting as a control.

An instruction sheet was given to the participant prior to the experiment, outlining the procedure (see Appendix C). During the baseline phase, the experimenter took a series of speech samples on-line, comprising 500 syllables. A stable baseline was considered achieved once three consecutive on-line samples measured readings within 2%SS of each other. Individuals spoke spontaneously on topics of their own choice or on those suggested by the experimenter.

The treatment phase comprised two 20-minute periods. During this phase, time-out individuals were instructed to cease talking whenever the red light was illuminated and to re-commence conversation once it was switched off. The light remained on for five seconds and was contingent on each stuttering episode as identified by the experimenter. During the time-out period, all social reinforcers in forms of eye contact, smiles, nods and conversation comments were ceased. Normal disfluencies, described by Guitar (1998) as relaxed single syllable whole word repetitions, interjections and revisions, were not interrupted. The control participants also participated in the 2 x 20 minute talking periods, but were not exposed to the time-out condition; instead they were encouraged to continue speaking. Post measurements were taken immediately following the “treatment” period where 3 x 500 syllable samples were collected from each participant.

Tape recordings of participants’ speech behaviour during each phase were evaluated by the author. The quantity of syllables differed between individuals during the treatment period. Therefore, in order to ensure consistency, samples of 2,500 syllables were taken from each 20 minute phase. A percentage of syllables stuttered (%SS) and the number of syllables stuttered and spoken fluently were obtained. Rate of speech (SPM), calculated by counting the number of syllables uttered per minute, was also measured in order to monitor any change associated with the reduction in stuttering (Bloodstein, 1950, cited in James et al., 1989). Once these calculations were determined, the taped speech was again heard in order to tally the frequency of the different types of stuttering behaviours.

### *Reliability*

A survey of clinicians' rating reliability, conducted by Ingham in 1990, found that different clinics markedly disagree in stuttering counts. Therefore, all measurements in this study were performed by the primary investigator. Intra-rater reliability was achieved by the investigator analysing each speech sample at least twice – once to obtain preliminary measures of %SS and SPM, and a second time to retrieve a count of the different types of stutters. The stutter frequency differed between the two collections by more than five stutters in four speech samples only. Therefore, the stutter frequency was re-counted for each of these samples, and stutters judged to be borderline were not counted. As a result, intra-rater agreement was met.

To check inter-rater reliability, 10 percent of the speech samples were independently assessed by a Speech Pathologist with more than 20 years clinical experience in the management of stuttering disorders. Both the investigator and the second rater were originally trained to identify stuttering behaviours using Wingate's (1976) definition. A Pearson-Product-Moment correlation of the corresponding measurements was carried out. Interjudge reliability for percent syllables stuttered was .87 and ratings of syllables per minute speech rate was .97, indicating adequate inter-rater reliability.

## Results

### Time-Out Treatment Effectiveness

Table 2 presents the frequency of stuttering (%SS) means, medians, standard deviations and range for time-out and control participants over the three conditions. These



data show a drop in stuttering levels during the treatment condition by the time-out individuals, while there was an increase during the same phase for the controls. Eighty seven percent of the time-out participants showed a decrease in stuttering to levels of under 2%SS, and 70% to levels of less than 1%SS (see Appendix D for individual outcome data). During the post treatment condition, when the treatment was withdrawn, the time-out group's mean %SS increased, but did not return to baseline level overall. During the same phase, the control group's mean %SS continued to increase.

Table 2

*Percent Syllables Stuttered During Baseline, Treatment and Post-Treatment for Time-Out and Control Groups*

	Time-Out (n = 30)	Control (n = 30)
Baseline		
<i>M</i>	5.82	4.85
median	3.68	3.017
<i>SD</i>	6.40	4.96
range	1.23 – 28.27	0.67 – 23.3
Treatment		
<i>M</i>	1.16	5.86
median	0.68	3.83
<i>SD</i>	1.39	5.21
range	0.2 – 6.8	0.5 – 19.5
Post-Treatment		
<i>M</i>	3.89	6.37
median	2.33	4.50
<i>SD</i>	5.63	5.08
range	0.5 – 25.63	0.53 – 20.73

Non-parametric analyses were used to test the significance of these changes because the data were severely skewed and there was significant violation of the homogeneity of variance assumption. A positively skewed distribution arose because there were more mild to moderate cases of stuttering than the more severe cases. According to Onslow (2001), this pattern is typical with both adults and children who stutter. Violation of homogeneity of variance is due primarily to the time-out condition, where time-out treatment reduced participants' stuttering levels close to 0%SS. The smaller variance for the time-out participants compared to the control group (see the standard deviations in Table 2) reflects a floor effect that is present for the time-out individuals, but not controls. Non-parametric assumptions were met, however, as group data had a similar shaped distribution, were randomly sampled, and independent. To control for the cumulative Type 1 error rate from conducting multiple planned comparisons using non-parametric tests, a Bonferroni adjusted rejection probability level of .007 was used (Keppel, 1991). This adjustment was based on maintaining a family-wise Type 1 error rate of .05 when undertaking seven planned comparisons.

The Mann-Whitney Test was applied to the data initially for comparisons between groups. As stated in the method section, both groups presented with similar levels of stuttering at baseline. However, during the treatment phase, tests showed a significant difference between the two groups,  $U = 85.5, p < .007$ . There was also a significant difference between the groups during the post-treatment condition,  $U = 234.5, p < .007$ .

Tests were then conducted to investigate the changes in stuttering levels over the three conditions separately for each group. A Wilcoxon Signed-Rank Test yielded a

significant decrease in the percent syllables stuttered for the time-out participants during the treatment phase compared with baseline,  $Z = 4.78, p < .007$ , and a significant difference remained between baseline and post treatment conditions,  $Z = 2.83, p < .007$ . In contrast, the control participants showed a significant increase in the amount of stuttering from baseline to treatment,  $Z = 2.95, p < .007$ , and an even greater difference between baseline and post-treatment measures,  $Z = 3.99, p < .007$ .

### *Speech Rate*

Table 3 displays the speech rate means for both groups during each of the three conditions. Unlike %SS, assumptions for ANOVA were upheld. Consequently, the means for each condition were compared using two-way analysis of variance with group (time-out vs. control) as a between participants factor and condition (baseline vs. treatment vs. post treatment) as a repeated measures factor. The data show that time-out participants increased their speech rate during the treatment phase, while the mean rate for the control group decreased. The main effect of group was not significant,  $F < 1$ . There was a main effect of condition,  $F(2, 116) = 11.31, p < .05$ . Examination of the marginal means for the effect of conditions (i.e., collapsing across group) showed that treatment condition had higher rate than baseline condition, post treatment condition was in between. However, condition interacted significantly with group,  $F(2, 116) = 17.72, p < .05$ . Simple main effect analysis was carried out to look at the effect of condition separately for each group. Condition was significant for time-out participants,  $F(2, 58) = 16.89, p < .05$ , and controls,  $F(2, 58) = 3.22, p < .05$ . Mean rate for the time-out participants during treatment was higher than baseline,  $F(1, 58) = 4.09, p < .05$ , as was post treatment,  $F(1, 58) = 13.75, p < .05$ . For the controls, speech rate at post-treatment was significantly lower,

$F(1, 58) = 6.39, p < .05$ . The difference between rate during treatment and baseline was not significant,  $F(1, 58) = 1.19, p > .05$ .

The results show, therefore, that in conjunction with a reduction in percent syllables stuttered, the time-out group increased their speech rate. By contrast, the control group increased their stuttering severity across conditions, and reduced their speech rate.

Table 3

*Speech Rate (Syllables per Minute) Taken During Baseline, Treatment and Post-Treatment for Time-Out and Control Groups*

Conditions	Time-Out (n = 30)	Control (n = 30)
Baseline		
<i>M</i>	211	236
<i>SD</i>	57	51
range	70 - 296	107 - 317
Treatment		
<i>M</i>	247	233
<i>SD</i>	34	55
range	163 - 300	104 - 329
Post-Treatment		
<i>M</i>	234	229
<i>SD</i>	51	54
range	77 - 300	102 - 325

### Factors Affecting Treatment Outcome

In order to investigate what factors might predict individuals' responsiveness to the treatment, treatment outcome was measured in two ways. First, as a difference score, calculated by subtracting the stuttering frequency count (%SS) during treatment from the frequency count at baseline. As noted above, a floor effect was evident where most time-out participants clustered together at a low level of stuttering frequency in the treatment condition producing a much smaller variance compared to other conditions. One consequence of this floor effect is that the difference score is generally larger for more severe participants (they have more room to improve) compared to less severe participants. The correlation between baseline severity and treatment outcome measured in terms of difference scores is very high ( $r = .96, p < .001$ ). This may be interpreted as an artefact of the floor effect; it does highlight the effectiveness of the time-out procedure because most time-out participants were brought to a similar low level of stuttering frequency, regardless of initial severity.

A second calculation of the treatment outcome, in terms of percentage change, was conducted to control to some extent for initial severity level (Block & Cornthwaite, 1999). This was obtained by dividing the individual %SS scores during treatment by the corresponding baseline %SS measurements, subtracting the result from 1, and then multiplying by 100.

Two separate regression analyses were undertaken, one for each outcome measure. A standard multiple regression procedure was used (Tabachnick & Fidell, 2001) where all

variables are entered into the analysis at the same time and tests of individual predictors are of the unique, as opposed to shared, variance, explained by each predictor. The predictor variables included stuttering severity scores at baseline (%SS), speaking rate (SPM), the nature of the stutter at baseline (% acute stutters), age of individuals in years, amount of previous treatment in months, and whether the time-out participant had received other treatment within the last year.

Multiple regression assumptions of multicollinearity and singularity, normality, linearity, homoscedasticity and independence of residuals (determined by examining the residual scatterplots) were tested using procedures recommended by Tabachnick and Fidell (2001). Data were also screened for univariate and multivariate outliers (detected by Mahalanobis distance). The distribution for both the treatment outcome (difference scores) and the stuttering severity was normalised after a log (base 10) transformation. One univariate outlier (individual presenting with 100% acute stuttering) was removed. A square root transformation of the previous treatment scores was also undertaken to normalise the distribution. Although each transformation affects the interpretation of those variables, it was felt that because of the relatively small number of participants the transformations were preferable to optimise the regression analysis in order to explore potential factors associated with treatment outcome. Transformation had minimal effect on the size and significance of the correlations.

Pearson product-moment correlation coefficients (using transformed variables) are presented in Table 4 (refer to Appendix E for matrix containing untransformed correlations).

Table 4  
*Correlations Between Treatment Outcome and Predictors*

Variables	Stuttering Age	Percent Acute Severity	Previous Stuttering	Recent Treatment	Speech Rate	
Treatment Outcome Difference Scores <sup>a</sup>	-.09	.96***	-.16	.38*	.01	-.76***
Treatment Outcome Percent Change Scores <sup>b</sup>	.08	.38*	.16†	.31	.09	-.26
Age		-.12	.37*	.20	.03	-.05
Stuttering Severity <sup>c</sup>			-.23	.34*	-.03	-.80***
Percent Acute Stuttering <sup>d</sup>				-.20	.10	.24
Previous Treatment <sup>e</sup>					-.18	-.49**
Recent Treatment <sup>f</sup>						.00

*Note.* <sup>a</sup> Difference between treatment and baseline conditions, and log10 transformed

<sup>b</sup> Untransformed percentage change scores

<sup>c</sup> Percent syllables stuttered at baseline and log10 transformed

<sup>d</sup> Percent of acute stuttering behaviours (sound, syllable and word repetitions)

<sup>e</sup> Previous treatment duration transformed by the square root

<sup>f</sup> Whether or not participants received treatment in the last year

\*  $p < .05$ , \*\*  $p < .01$ , \*\*\*  $p < .001$ , †  $a = .052$

Analysis revealed a strong association between baseline severity and treatment outcome (calculated as difference scores). In addition, there was a positive association between treatment outcome and the amount of previous treatment received ( $r = .38$ ,

$p < .05$ ), implying that participants' responsiveness to treatment increased with the more treatment they had received in the past. Speech rate showed a significantly strong negative correlation with treatment outcome ( $r = -.76, p < .001$ ), and a negative association with baseline severity ( $r = -.80, p < .001$ ). Thus, participants who presented with a slower speech rate tended to be more severe and to respond better to time-out treatment.

Interestingly, there was a significant correlation between previous treatment received and speech rate ( $r = -.49, p < .01$ ). The more treatment a participant had received, the slower they spoke at baseline. But the participants who presented with the more severe stuttering tended to have received the most treatment ( $r = .34, p < .05$ ). Finally, a significant, but again, modest relationship was found between age and percent repetitions ( $r = .37, p < .05$ ), that is, there was a trend for the younger participants to present with a larger proportion of advanced stuttering types. No other correlations were significant.

The multiple regression analysis with difference scores as the dependent measure, revealed a strong and significant overall multiple R of .97,  $F(6, 22) = 57.21, p < .001$ . Table 5 displays the standardised and unstandardised regression coefficients for each predictor variable, the semi-partial correlations squared, multiple R,  $R^2$  and adjusted  $R^2$ , as well as the predictive variabilities. Stuttering severity was the only predictor that accounted for a significant amount of unique variance in the prediction of the treatment outcome. No other individual predictors were significant. The relative importance of the predictors is also reflected in the squared semipartial correlations, which depicts the proportion of the variance in the dependent measure uniquely predicted by each independent variable. This proportion of variance is very small for all predictors except for the baseline severity (34%). The results also indicate that a reasonably large



proportion (59%) of the predictive variance associated with baseline severity (which on its own accounts for 92% of variance of the dependent variable) is shared with at least one of the other predictors.

Table 5

*Standard Multiple Regression Analysis of Treatment Outcome (difference scores) using all Predictor Variables*

Variables	Regression Coefficients		Squared
	Unstandardized	Standardized	Semi-Partial Correlation <sup>f</sup>
Stuttering Severity <sup>a</sup>	1.18***	1.01	0.3364
SPM <sup>b</sup>	0.00058	0.08	0.0016
Age	-0.00034	-0.01	0.0001
Previous Treatment <sup>c</sup>	0.016	0.10	0.0064
Percent Acute Stuttering <sup>d</sup>	0.0023	0.08	0.0049
Recent Treatment <sup>e</sup>	0.34	0.04	0.0016
	Multiple $R = .97***$	Multiple $R^2 = .94$	
	Adjusted $R^2 = .92$		
	Unique variability = .34	Shared variability = .59	

*Note.* <sup>a</sup> Percent syllables stuttered at baseline after log10 transformation

<sup>b</sup> Syllables per minute speech rate

<sup>c</sup> Previous treatment duration transformed by the square root

<sup>d</sup> Percent of acute stuttering behaviours

<sup>e</sup> Whether or not participants received treatment in the last year

<sup>f</sup> Provides the proportion of variance in the dependent variable (difference outcome scores) that is explained uniquely by stuttering severity at baseline

\*\*\*  $p < .001$ .

A further multiple regression analysis was conducted to examine the role of other predictors after excluding baseline severity (see Appendix F for the output of this analysis). Results revealed a significant overall multiple R of .78,  $F(5, 23) = 6.94$ ,  $p < .001$ , with only speech rate (SPM) displaying a unique prediction to outcome. The fact that SPM does not provide a significant unique contribution to the prediction in the first analysis suggests that speech rate shares predictive variance with baseline severity. A subsequent multiple regression, which excluded both baseline severity and speech rate, resulted in a non-significant overall multiple R = .43,  $F(4, 24) = 1.35$ ,  $p > .05$ , confirming that no other factors were significantly related to treatment outcome.

Multiple regression analysis was also conducted using treatment outcome, calculated as a percentage shift. Pearson correlations between the predictors and the percentage change outcome measure are also presented in Table 4. Baseline severity and treatment outcome were again significantly, but more moderately, correlated ( $r = .39$ ,  $p < .001$ ). That is, the higher the severity at baseline, the better the outcome. The amount of previous treatment showed a modest association but was only marginally significant ( $p = .052$ ). No other predictors were significantly correlated with the treatment outcome.

Table 6 displays the results of the standard multiple regression analysis. The analysis revealed a moderate but non significant overall multiple R of .54,  $F(6, 22) = 1.53$ ,  $p > .05$ , with none of the factors providing any significant unique predictive value for responsiveness to treatment. However, stuttering severity at baseline did explain the largest amount of unique variance (10%), and was approaching significance ( $p = .087$ ).

Table 6

*Standard Multiple Regression Analysis of Treatment Outcome (percentage change) using all Predictor Variables*

Variables	Regression Coefficients		Squared
	Unstandardized	Standardized	Semi-Partial Correlation <sup>f</sup>
Stuttering Severity <sup>a</sup>	26.642*	0.56	0.1030
SPM <sup>b</sup>	0.084	0.29	0.0240
Age	-0.025	-0.02	0.0003
Previous Treatment <sup>c</sup>	2.265	0.34	0.0784
Percent Acute Stuttering <sup>d</sup>	0.348	0.28	0.0590
Recent Treatment <sup>e</sup>	4.442	0.13	0.0169
	Multiple $R = .54\dagger$	Multiple $R^2 = .29$	
	Adjusted $R^2 = .10$		
	Shared variability = .28		

*Note.* <sup>a</sup> Percent syllables stuttered at baseline after log10 transformation

<sup>b</sup> Syllables per minute speech rate

<sup>c</sup> Previous treatment duration transformed by the square root

<sup>d</sup> Percent of acute stuttering behaviours

<sup>e</sup> Whether or not participants received treatment in the last year

<sup>f</sup> Provides the proportion of variance in the dependent variable (percentage change scores) that is explained uniquely by stuttering severity at baseline

\* Approaching significance  $p = .087$ ;  $\dagger p > .05$

### Nature of the Stuttering

The percentage of acute types of stutters at baseline did not affect the treatment outcome, as revealed by the multiple regression analysis. However, an unexpected increase in the mean percentage of acute stuttering was observed over the experimental conditions for the time-out group. Figure 1 gives the mean percentage of acute stuttering for the groups over the three conditions. See also Appendix G for the means and standard deviations. An analysis of variance was conducted with group (time-out vs. control) as a between participants factor, and condition (baseline vs. treatment vs. post-treatment) as a repeated measures factors. An examination of the underlying assumptions of ANOVA for percentage of beginning stutters, in contrast to the percent syllables stuttered, were not violated.

Results showed a significant main effect of condition,  $F(2, 116) = 3.99, p < .05$ , and an interaction between condition and group,  $F(2, 116) = 4.73, p < .05$ , but no main effect of group,  $F < 1$ . Simple effect analysis of the interaction found there was a significant change over the conditions for time-out participants  $F(2, 58) = 5.77, p < .01$ . There was no significant difference among the condition means for controls,  $F < 1$  (see Figure 1).

Pairwise comparisons of the condition means were conducted on the time-out participants' data which revealed a significant increase in acute stuttering behaviours between baseline and treatment conditions,  $F(1, 58) = 6.46, p < .05$ , but no significant difference between treatment and post-treatment measures,  $F < 1$ .

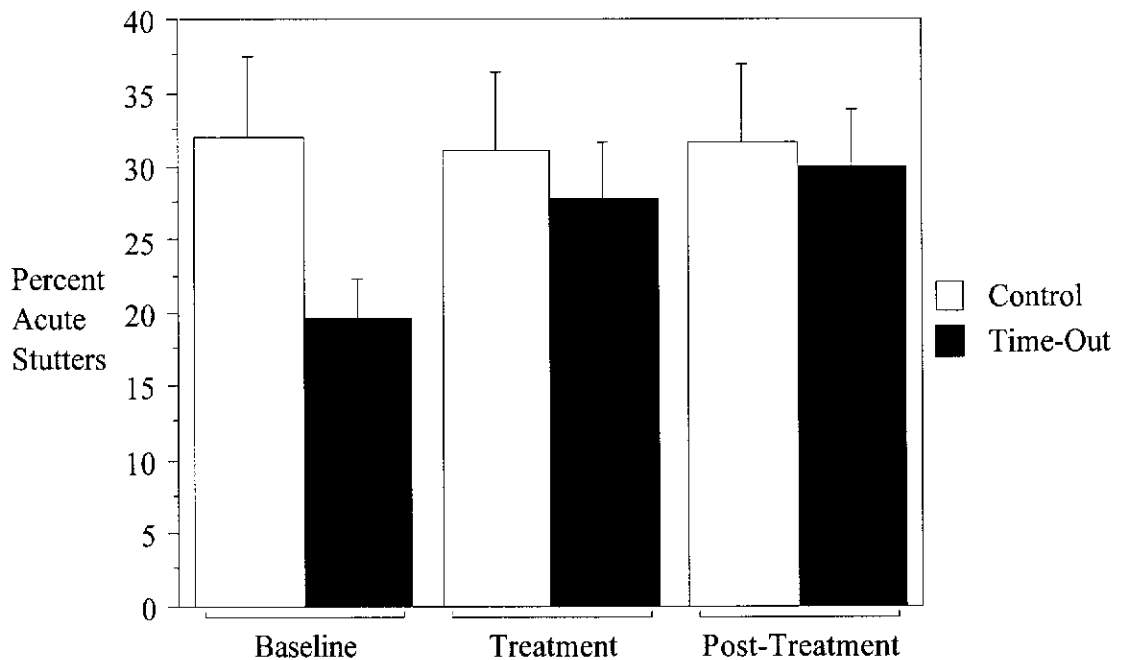


Figure 1. Mean percent acute stuttering behaviours for time-out and control participants for baseline, treatment and post-treatment conditions (error bars correspond to +1 standard error of the mean).

### Discussion

This study successfully demonstrated that time-out treatment can effectively ameliorate stuttering. There was a significant reduction in stuttering frequency from the baseline to the treatment conditions for the time-out individuals. All participants experienced an increase in fluency, and almost half achieved a reduction in stuttering of over 80 percent. By contrast, the controls, who were placed in the same environment for the same length of time but were not exposed to time-out, experienced a significant increase in stuttering levels. These results clearly demonstrate that the procedure of time-

out is a powerful clinical tool in the reduction, and in some cases elimination, of stuttering.

Eighty three percent of participants commented that the delivery of time-out alerted them to their stuttering and reminded them to attempt to be more fluent. Twenty four percent felt that the interruption to their speech was aversive. These comments were consistent with those made by individuals in similar studies (Cooper et al., 1970; Wingate, 1976; and Martin & Haroldson, 1977). Within a social cognitive learning context, it could be argued that any interruptive stimulus delivered contingent upon the stuttering behaviour will act to draw attention to it and in turn, reduce the response. Prins (1993) has suggested that once alerted to the stutter, a speaker can exercise control over the behaviour through internal self regulation.

The results of this study were successful despite the nature of the stuttering. Multiple regression analysis revealed that there was no significant relationship between the percentage of acute stuttering and the treatment outcome, that is, regardless of the topography of the stuttering, time-out treatment effectively reduced the behaviour. The shift from effortful chronicity of speech to easier repetitions of sounds, syllables and words indicated that stuttering is dynamic in its form. This finding might suggest that people with predominantly acute types of stuttering (as opposed to small percentages of acute stuttering as was the case in this study), may indeed respond even more favourably to the treatment procedures of time-out than the participants in this experiment who exhibited predominantly chronic stuttering behaviours.

It is not known whether this significant shift in the stuttering topography was a typical response to a treatment procedure that does not involve modifying the speech

pattern as is the case with fluency shaping programmes. The reason for this shift is open to speculation. Packman and Lincoln (1996) suggest that syllable repetitions, as well as syllable-timed speech, help to stabilise the speech system by reducing the variability of vowel durations across syllables. Therefore, an increase in the quantity of repetitions evident in the participants' speech over the experimental conditions, may be a sign of an increasingly more stable paralinguistic system. Acoustic analysis of participants' fluent speech pre and post treatment may provide some evidence to support this hypothesis.

The stuttering topography shift is an interesting finding because it potentially sets up for the future, more flexible options for the treatment of adults who stutter. If stuttering behaviours of adults are malleable, then perhaps using the simpler stuttering treatment methodology of response contingent time-out as opposed to a complex programme such as prolonged speech, may enable clients to respond more quickly to treatment, and exhibit more natural sounding and socially tolerable stuttering behaviours.

Yet another significant finding was the fact that participants who presented with more severe stuttering, in terms of frequency, demonstrated greater improvement than those with mild stutters. This discovery was consistent with findings of James et al. (1989). The relationship was particularly strong when the treatment outcome was considered in terms of difference scores between baseline and treatment percent syllables stuttered, although this may be due to the floor effect. That is, the more severe the stuttering the greater the room for improvement. The correlation was not as strong, although still significant, when the outcome was measured by calculating a percentage shift.

These findings support James' et al. (1989) hypothesis that people with severe stuttering do not ordinarily draw on their fluency reserves unless exposed to such contingencies. Individuals with milder stuttering may already be maximising their fluency reserves much more. This may also explain why Andrews and Craig's (1988) participants, who presented with more severe stuttering before a fluency inducing programme, were more prone to relapse once they returned to their natural speaking environment. Certainly, within the paediatric population it has been found that when exposed to response contingent stimulation treatment, children with the most severe stuttering tended to be more prone to relapse (Jones, Onslow, Harrison, & Packman, 2000; Onslow & Packman, 1997).

In this investigation, time-out proved successful despite the age of the individuals to whom it was administered. Time-out was also successful despite the history of treatment that the participants had experienced. Of interest was the fact that individuals with a longer history of failed therapy from previous treatments exhibited more severe stuttering and gained a stronger treatment effect than did those who had a relatively recent and short history of clinical experience.

Slowed speech rate has been documented as facilitating fluent speech, albeit with an unnatural sounding quality (Bloodstein, 1995). In this study, the time-out treatment achieved successful results without individuals slowing down their speech rate. In fact, data showed that speech rate actually increased. Therefore, it brought about a more natural speed of conversation exchange on the part of the treatment participants. These observed increases in speech rate occurred despite 59% of participants stating that the periods of silence gave them time to slow down. However, this finding may have been the product of



fewer stutters and how speech rate was measured rather than an actual increase in rate of articulation. That is, in this experiment, speech rate was measured by the amount of syllables produced over a minute of time. Therefore, the duration of each stutter and the quantity of stuttering will influence the overall syllables per minute speaking rate measurement. This seems likely in view of the fact that the control group showed an overall decrease in speech rate as a group. Two time-out participants made noticeable adjustments to their speech quality, choosing to use previously-learned prolonged speech rate in order to avoid stuttering. By contrast, the investigator was unable to perceive any obvious alterations to the speech quality of the other 28 individuals. Thus, time-out produced an effective reduction in stuttering with no apparent compromise in terms of speech rate or altered natural quality of speech production.

It has been of great interest to conduct this study investigating the effects of time-out on stuttering behaviour. Using a strong sample size, this treatment methodology was found to be overridingly effective for every person participating in the experiment. Reductions in stuttering occurred no matter how severe the stuttering was, how old the participant was, or how much treatment they had previously received (although there was a moderate relationship between treatment outcome and previous treatment), and all in the space of 40 minutes.

The potency of time-out has been well documented in the literature. However, it has not translated into clinical management as one would have predicted with such a strong research history. It is hoped that the present study will trigger further interest and contribute towards re-addressing this limitation.

Areas for future directions could be to determine the durability and the maintenance of the time-out procedure's effects over time. As this study only investigated the establishment of fluency over a 40-minute session, and not the long term effectiveness, little is known of the robustness of these effects, or the amount of time-out treatment sessions necessary to promote generalisation and maintenance of fluency. In addition, another avenue of future adult treatment may be to investigate the potential malleability of chronicity of stuttering in adults to more acute and easier stuttered behaviours.

## Appendix A

*Contingent Time-Out studies Investigated by Prins and Hubbard (1988)*

Authors	n	Age	Nature	Baseline severity	Sig.
Martin & Haroldson (1982)	10 controls 10 EATO 10 SATO	adult	No data given	C – 13 wpm E – 16 wpm S – 15 wpm	Yes
Costello (1975)	3	adolescent s	Repetitions Prolongations Blocks	~ 20 stutters per minute	Yes
Haroldson et al (1968)	4	adult	No data given	Unstable baselines	Yes
Martin, Berndt (1970)	1	12;0	No data given	28 stutters over 2 min	Yes
Martin & Haroldson (1979)	20	19-46 years	No data given	<i>M</i> = 12 words stuttered	Yes
James (1976)	45	15-65 yrs	No data given	>4%SS	Yes
Adams & Popelka (1971)	8	18-27 yrs	Repetitions Prolongations	<i>M</i> = 11 stutters / passage	Yes
Mowrer (1978)	6	12-38 yrs	No data given	3-26 disfluencies/ min	Yes
Martin & Haroldson (1971) Cond. II	4	adults	No data given	3.5%-10%SS	Yes
James (1983)	33	adolescent s & adults	No data given	7-35%SS	Yes
James (1981a) Exp I	1	adult	No data given	moderate	Yes
James & Ingham (1974)	14	15-30 years	Presenting with Wingate's definition	<i>M</i> = 7%SS	Yes

Costello & Hurst (1981)	3	18;0 19;0 40;0	All presented with reps, prolongations and blocks	10 stut/min 19 stut/min 18 stut/min	Yes
James (1981c)	33	16-58 years	Presenting with Wingate's definition	$M = 15\%SS$	Yes
James (1981b)	36	16-57 years	No data given	$M = 13\%SS$	Yes

## Appendix B

*A Study Investigating Treatment of Advanced Stuttering***Informed Consent Form**

The aim of this study is to investigate two different approaches to the treatment of stuttering and to investigate the effect of the type of stuttering on the treatment outcome. People who participate in this study will attend one appointment, lasting approximately 2 hours. Participants will be randomly allocated to either one of the two treatment approaches, but will not be informed of which until the end of the experiment as it may affect the results of the study. The appointment will essentially involve having a conversation with the principle investigator. There are no foreseeable risks, discomfort or benefits associated with participation in either treatment group within this study.

I, \_\_\_\_\_ have read the information about this study and questions I have asked have been answered to my satisfaction. I give my permission for the tasks described above to be administered. I understand that the information obtained about me is confidential and that I can withdraw from the study at any time without prejudice.

Signed \_\_\_\_\_ on the \_\_\_\_\_ day of \_\_\_\_\_ 19\_\_\_\_.  
(participant)

Signed \_\_\_\_\_ of \_\_\_\_\_  
(researcher) (Institute)  
on the \_\_\_\_\_ day of \_\_\_\_\_ 19\_\_\_\_.

## Appendix C

*Instructions given to time-out (1) and control (2) participants***Instructions (1)**

The study in which you have volunteered to participate consists of three parts. In the first part you are required to engage in conversation with me for approximately 20 minutes. During this time I will be rating at least 1500 syllables of your speech. This will enable me to assess the present severity of your stutter.

Part 2 will involve speaking for two 20-minute periods with a 10 minute break in between. A red light will be placed on the table in front of you and I will ask you to stop speaking immediately the light is activated. When the light goes off, commence speaking again. Each time the light comes on it will stay on for approximately 5 seconds.

The final part of this experiment is just a repeat of part 1 and should only take a further 20 minutes. Do you have any questions?

**Instructions (2)**

The study in which you have volunteered to participate consists of three parts. In the first part you are required to engage in conversation with me for approximately 20 minutes. During this time I will be rating at least 1500 syllables of your speech. This will enable me to assess the present severity of your stutter.

Part 2 will involve speaking for two 20-minute periods with a 10 minute break in between. I will not be rating your speech during this time.

The final part of this experiment is just a repeat of part 1 and should only take a further 20 minutes. Do you have any questions?

## Appendix D

*Individual Outcome Data – Treatment Group*

Participant	Baseline %SS	Treatment %SS	Post-Treatment %SS	Treatment Outcome <sup>a</sup>	Treatment Outcome <sup>b</sup>
3	20.83	.70	2.73	20.13	96.64
4	6.57	.45	2.83	6.12	93.15
28	4.83	0.35	2.97	4.48	92.76
30	5.67	0.45	1.20	5.22	92.06
10	7.53	0.60	2.57	6.93	92.04
21	2.30	0.20	2.10	2.10	91.30
23	5.43	0.60	3.00	4.83	88.96
6	2.63	0.30	1.13	2.33	88.61
25	2.57	0.30	0.97	2.27	88.31
15	4.47	0.55	1.33	3.92	87.69
12	1.30	0.20	.50	1.10	84.62
29	5.40	0.85	4.40	4.55	84.26
27	1.93	0.35	2.50	1.58	81.90
1	8.53	1.70	0.87	6.83	80.08
20	21.07	4.45	25.63	16.62	78.88
22	2.93	0.65	2.77	2.28	77.84
2	6.47	1.50	3.17	4.97	76.80
7	28.27	6.80	19.73	21.47	75.94
26	1.70	0.45	0.60	1.25	73.53
11	2.73	0.75	1.70	1.98	72.56
9	3.70	1.10	1.63	2.60	70.27
5	2.60	0.80	3.33	1.80	69.23
19	7.63	2.35	12.10	5.28	69.21
24	1.23	0.40	0.80	0.83	67.57
17	2.30	0.75	0.80	1.55	67.39
18	4.97	1.80	7.10	3.17	63.76
14	1.37	0.65	2.17	0.72	52.44
16	1.57	0.95	0.97	0.62	39.36
13	2.30	1.40	3.03	0.90	39.13
8	3.67	2.45	2.17	1.22	33.18

Note. <sup>a</sup> Difference scores between baseline and treatment measures

<sup>b</sup> Percent change scores between baseline and treatment measures

*Individual Outcome Data – Control Group*

Participant	Baseline %SS	Treatment %SS	Post-Treatment %SS	Treatment Outcome <sup>a</sup>	Treatment Outcome <sup>b</sup>
31	2.97	7.65	9.67	-4.68	61.18
32	3	2.3	3.27	0.7	23.33
33	8.23	10.75	10.00	-2.52	23.44
34	6.8	12.05	12.47	-5.25	43.57
35	3.03	5.7	8.07	-2.67	46.84
36	6.37	7.55	6.73	-1.18	15.63
37	0.7	2.55	2.4	-1.85	72.55
38	1.97	1.95	3.2	0.02	1.02
39	2.2	2.1	2.1	0.1	4.55
40	5.4	9.9	9.73	-4.5	45.45
41	5.07	5.7	4.57	-0.63	11.05
42	3.7	4.9	5.43	-1.2	24.49
43	6.13	7.05	8.77	-0.92	13.05
44	1.9	2.3	2.17	-0.4	17.39
45	1.43	3.15	6.6	-1.72	54.60
46	0.67	0.5	0.53	0.17	25.37
47	4.4	4.65	4.63	-0.25	5.38
48	2.03	2.2	2.83	-0.17	7.73
49	15.23	17.8	18	-2.57	14.44
50	1.1	1.1	1.37	0	0
51	23.3	19.5	20.73	3.8	16.31
52	2.03	2.55	2.1	-0.52	20.39
53	1.9	1.35	2.57	0.55	28.95
54	1.7	1.5	3.17	0.2	11.76
55	2.33	2.33	3.33	0	0
56	3.67	3.55	4.4	0.12	3.27
57	3.7	4.1	4.43	-0.4	9.76
58	13.83	17.2	15.13	-3.37	19.59
59	8.93	10	10.7	-1.07	10.7
60	1.77	1.8	1.9	-0.03	1.67

Note. <sup>a</sup> Difference scores between baseline and treatment measures

<sup>b</sup> Percent change scores between baseline and treatment measures



## Appendix E

*Correlations between Treatment Outcome and Predictors prior to Standardisation*

Variables	Age	Stuttering Severity	Percent Acute Stuttering	Previous Treatment	Recent Treatment	SPM
Treatment Outcome Difference Scores <sup>a</sup>	-.07	.99***	-.22	.38*	.11	-.80***
Treatment Outcome Percent Change Scores <sup>b</sup>	.08	.26	.16	.25	.09	-.26
Age		-.07	.37*	.25	.03	-.05
Stuttering Severity <sup>c</sup>			-.25	.36*	-.07	-.81***
Percent Acute Stuttering <sup>d</sup>				-.26	.10	.24
Previous Treatment <sup>e</sup>					-.14	-.44**
Recent Treatment <sup>f</sup>						.00

**Note.** <sup>a</sup> Difference between treatment and baseline conditions, and log10 transformed

<sup>b</sup> Untransformed percentage change scores

<sup>c</sup> Percent syllables stuttered at baseline and log10 transformed

<sup>d</sup> Proportion of acute stuttering behaviours

<sup>e</sup> Previous treatment duration transformed by the square root

<sup>f</sup> Whether or not participants received treatment in the last year

$p < .05$ , \*\*  $p < .01$ , \*\*\*  $p < .001$

## Appendix F

*Standard Multiple Regression Analysis of Treatment Outcome (difference scores) using all Predictor Variables Excluding Stuttering Severity*

Variables	Regression Coefficients		Squared
	Unstandardized	Standardized	Semi-Partial Correlation <sup>e</sup>
SPM <sup>a</sup>	0.00058***	0.08	0.4186
Age	-0.00034	-0.01	0.0243
Previous Treatment <sup>b</sup>	0.016	0.10	0.0031
Percent Acute Stuttering <sup>c</sup>	0.0023	0.08	0.0072
Recent Treatment <sup>d</sup>	0.34	0.04	0.0003
Multiple $R = .78^{***}$		Multiple $R^2 = .60$	
Adjusted $R^2 = .52$			
Unique variability = .42		Shared variability = .45	

*Note.*<sup>a</sup> Syllables per minute speech rate

<sup>b</sup> Previous treatment duration transformed by the square root

<sup>c</sup> Percent of acute stuttering behaviours

<sup>d</sup> Whether or not participants received treatment in the last year

<sup>e</sup> Provides the proportion of variance in the dependent variable (difference outcome scores) that is explained uniquely by stuttering severity at baseline

\*\*\*  $p < .001$ .

## Appendix G

*Percent Acute Stuttering Behaviours for Time-Out and Control Participants over each Condition*

Conditions	Time-Out (n = 30)	Control (n = 30)
<b>Baseline</b>		
<i>M</i>	19.58	31.95
<i>S.D.</i>	15.95	29.48
range	0 - 66.7	0 - 100
<b>Treatment</b>		
<i>M</i>	27.76	31.14
<i>S.D.</i>	21.77	29.52
range	0 - 76.97	0 - 95.35
<b>Post-Treatment</b>		
<i>M</i>	29.95	31.72
<i>S.D.</i>	21.95	27.67
range	2.73 - 87.33	2.53 - 100

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