Published in final edited form as:
J Speech Lang Hear Res. 2008 June ; 51(3): 669–687. doi:10.1044/1092-4388(2008/048).

Late childhood stuttering

Peter Howell[1], **Stephen Davis**[1], and **Roberta Williams**[2]

[1]Department of Psychology and Centre for Human Communications, University College London

[2]Department of Language and Communication Science, City University, London.

Abstract

Purpose—A longitudinal study was conducted on 76 children which examined risk factors that led children who stutter at around age eight to persist in the disorder when they reached age twelve.

Method—All children were verified as stuttering at the first assessment on the basis of a clinical referral and a further clinical assessment. When they reached twelve, they were classified as persistent or recovered on the basis of parent, child and researcher assessments. A range of measures was taken at the two age-points for determining risk factors for persistence/recovery.

Results—More males than females were affected. There was no evidence for persistence and recovery to run in families. At first referral all speakers who stuttered had high stuttering severity ratings and high proportions of dysfluencies in their speech (particularly those involving repetitions of whole words). At age 12 plus, the severity ratings of the recovered speakers and dysfluency counts dropped. The persistent speakers continued to have high severity ratings and produced more part-word dysfluencies. Temperament, measured at first assessment, differed between all stutterers and fluent controls and, when persistent and recovered speaker groups were examined separately, recovered speakers were less adaptable than persistent speakers; persistent speakers had more intense moods than controls; recovered speakers were less adaptable than controls. Detection of backward masking stimuli at 12 plus did not differ between all the children who stuttered and controls but when speakers who stuttered were differentiated by recovery group, persistent speakers had poorer backward masking thresholds than recovered speakers. Performance in motor tasks controlled by the cerebellum was assessed at 12 plus. There were indications of poor cerebellar control in speakers who persisted compared with the recovered speakers.

Conclusion—The tendency for more males than females to stutter was confirmed. Different patterns in speech were observed: Severity ratings of the recovered speakers dropped by age 12 plus. The severity ratings for the persistent speakers remained high at 12 plus and dysfluency types tended to change from whole to part words over time. Persistent and recovered speakers differed on temperamental performance at initial assessment, and performed differently on sensory and motor tasks at age 12 plus.

Introduction

Extensive information on early childhood stuttering has been available since the publication of Yairi and Ambrose (2005). Much less is known about children in the age range 8-12 plus. Selected aspects of the literature are reviewed to identify potential risk factors for persistent stuttering. The epidemiology and symptomatology for all children in a longitudinal sample

Address Correspondence to: Peter Howell, Department of Psychology, University College London, Gower Street, London WC1E 6BT, England. Telephone Number: +44 (0)20 7679 7566 Fax number +44 (0)20 7436 4276 Email address: p.howell@ucl.ac.uk.

Europe PMC Funders Author Manuscripts

of children who stutter in this age range are then reported. All the children were confirmed as stuttering at the start of the study and were designated as persistent or recovered at the end of the study. Several etiological factors that put children at risk of persistent stuttering were identified in the review. These were assessed on subsets of the sample to see which should be included in a comprehensive package for determining prognostic outcome about stuttering for children in this age range.

Epidemiology

Incidence and point prevalence—Andrews and Harris (1964) studied stuttering in 1,142 families in Newcastle-on-Tyne with children born between May and June 1947. The study (usually referred to as the 1,000 Family Survey) ended when the children were aged 15. Point prevalence of stuttering was about 1%. Incidence up to age 15 was about 5%.

Two other large-scale studies on incidence and prevalence, both drew similar conclusions. Mannson (2000) examined 1,040 children (98% of all births) born on a Danish island over a two-year period. The author and a team of four clinicians assessed speech, hearing and language in face-to-face interviews. Incidence of stuttering was estimated at 4.9% of the children after their third birthday and 5.09% after two follow-ups several years later.

Dworzynski, Remington, Rijksdijk, Howell and Plomin (2007) analyzed stuttering data from the Twins Early Development Study, TEDS (Trouton, Spinath & Plomin, 2002). TEDS is a longitudinal study of all twins born between 1994 and 1996 in the United Kingdom. Twenty five thousand eight hundred and thirty twins were surveyed with regards to stuttering at ages two, three, four and seven years. The point prevalence of stuttering was between 1 and 3% for the different ages. Incidence across the test ages was 7%.

Sex ratio—Andrews and Harris (1964) showed that more boys than girls stuttered (a ratio of 2.4:1 overall) and that the ratio increased as the children got older (indicating that girls recovered from stuttering at an earlier age than boys). The predominance of males over females has been confirmed by Yairi and Ambrose (2005) who also reported that the sex ratio increased slightly with age. The latter was also found by Dworzynski et al. (2007) who found that for each girl who stuttered at ages two and three, there were approximately 1.6 boys, whereas at ages four and seven there were approximately 1.8 boys for every girl who stuttered (same value for the two ages).

Recovery rate—Andrews and Harris's (1964) 1,000-Family Survey showed a 5% point prevalence and a 1% annual incidence rate. Thus 80% (four out of five) of the respondents recovered from stuttering and only 20% persisted. For the Yairi and Ambrose (2005) study, recovery rates were between 65 and 80% three to five years after onset. Mannson (2000) reported that recovery rate was 71.6% after two years and 85% after five to six years. Two smaller scale studies pointed to similar values for these age ranges. Ryan (2001) followed up 22 two, to three year-old children for two years and reported a 68% recovery rate. Rommel, Hage, Kalehne and Johannsen (2000) reported follow-up results for 65 five-year-old children. Three years into their study, the recovery rate was 71%.

Investigations which do not start from stuttering onset cannot produce an absolute estimate of recovery rate (Yairi & Ambrose, 2005). They are useful if it is not practicable to follow up children from around two years to age 12 and upwards. One study has examined an older group. Fritzell (1976) followed children from age seven to nine through to teenage. He reported a recovery rate of 47% between seven years and teenage. The lower recovery rate was as would be expected given that most recovery happened when the children were young.

Europe PMC Funders Author Manuscripts

Age of onset—Andrews and Harris (1964) reported that one modal onset age was three and the other was five and early onset around these ages has been confirmed by Yairi and Ambrose (2005). Consistent with this, Dworzynski et al. (2007) showed an increase in reported cases at ages three to four. The results of the 1,000 Family Survey showed, in addition, that onset can occur up to at least age 11, with a high proportion who started stuttering at age 10. Overall, 25% of stuttering onsets up to age 15 were in the age range covered by the study to be reported later. Such cases are missed in studies that cease at, or below, age eight.

Age of recovery—Andrews and Harris (1964) showed that recovery can happen at any age into teenage. Andrews, Craig, Feyer, Hoddinott, Howie and Neilson (1983) conducted a meta analysis of the recovery rates reported in several studies. They estimated that 75% of those stuttering at age four years, 50% of those stuttering at age six years, and 25% of those stuttering at age 10 years recovered by the time they reached 16 years of age. If the problem continued into teenage, the chance of recovery decreased. This was supported by the 1,000-Family Survey where no child who was stuttering after 12 years of age recovered by age 16 years.

Length of time stuttered and length of time recovered group followed up post recovery—Yairi and Ambrose (2005) reported that the highest rate of recovery was three years after onset. They emphasized that it is necessary to follow up recovered children to ensure true recovery had occurred rather than a temporary remission. They continued to see their recovered children for an average of 40 months.

Research questions—Sex ratio, recovery rate, age of stuttering onset, age of recovery and length of time stuttered need to be determined separately for: a) genders; b) persistent and recovered speakers where appropriate for children in the age range eight to 12 years.

Symptomatology and assessment

The classic list of signs of stuttering used by Johnson and associates (1959) was: 1) Interjections (which includes filled pauses); 2) Word repetitions; 3) Phrase repetitions; 4) Part-word repetitions; 5) Prolongations; 6) Broken words; 7) Incomplete phrases (abandonments); 8) Revisions. The World Health Organization's (1992) International Statistical Classification of Diseases and Related Health Problems (ICD-10) included the first six of these signs in their definition of stuttering, which characterized the problem as "disorders in the rhythm of speech, in which the individual knows precisely what he wishes to say, but at the time is unable to say it because of an involuntary, repetitive prolongation or cessation of a sound." The ICD-10 definition would not include incomplete phrases and revisions as signs because these may result when the speaker does not know what to say.

Accepting that revisions and incomplete phrases can be put to one side, there is still no agreement about whether all the remaining signs should be included in speech assessments that establish whether stuttering is present. Yairi and Ambrose's (2005) team differentiated stuttering-like dysfluencies (SLD) from other dysfluencies (OD). SLD are part-word repetitions, single-syllable word repetitions and disrhythmic phonation (Ambrose & Yairi, 1999). OD comprised interjections, multiple-syllable words and phrase repetitions, revisions or abandoned utterances. They reported that the incidence of SLD was high near onset of the disorder. This group also found that some children showed characteristics that were previously thought to emerge as children got older (e.g. prolongations) and, as such, were regarded as perpetuating signs (Yairi, Ambrose, Paden & Throneburg, 1996). Wingate (2001) disputed whether the events included in the SLD class were appropriate to index

Europe PMC Funders Author Manuscripts

stuttering. In particular, he argued that whole word and phrase repetitions (signs 2 and 3) should not be included. Yairi, Watkins, Ambrose and Padden (2001) defended their use.

Howell (2004) divided dysfluencies into those involving parts of words and those involving whole words or pauses, which were called stallings. This division was based partly on clinical observations. For instance, as mentioned, prolongations are usually considered a feature of stuttering in older speakers for whom the disorder has persisted (Conture, 1990). Howell argued that stuttering in young speakers involved mainly stallings. These and the remaining dysfluencies all reduced in incidence in speakers who recovered.

Howell (2004) considered how such a progressive change in the types of dysfluencies could occur in persistent, but not in recovered, speakers and also suggested what CNS structures were implicated. A brief resume is given as this perspective (called EXPLAN theory) determined how symptomatology was examined and governed choice of some of the factors investigated in the study reported later. According to Howell (2004), fluency problems resulted from poor time coordination between linguistic and motor planning processes that either led to stallings or part-word dysfluencies. According to this view, speech is only dysfluent if linguistically difficult material is being produced and the speech motor output is set to too high a rate. When this situation arises, speakers need to adjust speech rate before they reach the difficult word. Stalling by repeating prior words is the way this is done in early development. If no such adjustment occurs, the problem continues. Howell (2002) proposed that the cerebellum is responsible for the required rate changes. Speakers who persist in producing speech like this will become less sensitive to the indications that speech breakdown is likely to occur because of CNS adaptation (Howell, 2002). Speakers then advance to the difficult word before they are ready, so the form of the fluency problem changes to occurrence on parts of words and this form is difficult to reverse.

According to this account, persistent speakers may respond in a different way from fluent speakers and those who have recovered (persistent speakers diverge from fluent speakers). On the other hand, the recovered speakers change from being like the persistent speakers to being like fluent speakers subsequently (recovered speakers converge on fluent speakers).

Research questions—Data are reported in the later study on: a) changes in stuttering severity for both persistent and recovered speakers that occur over development, and b) changes in dysfluency types over ages for these groups. After recovery, speakers should have lower severity than persistent speakers, but it is not known whether dysfluency types change at different rates for the two groups.

Etiological factors

Genetics—Different incidence rates between monozygotic (share all genetic material), and dizygotic, twins have been used in several studies to partial out the genetic factor from environmental sources (Andrews, Morris-Yates, Howie & Martin, 1991; Dworzynski et al., 2007; Felsenfeld, Kirk, Zhu, Statham, Neale & Martin, 2000; Ooki, 2005). The studies showed 70-85% of variance in liability to stuttering could be attributed to additive genetic effects with the remaining 30-15% attributable to the individual's unique environment.

Ambrose et al. (1993) examined whether persistent and recovered forms of stuttering were transmitted genetically. They found that individuals with a family history of persistent stuttering also tended to persist, whereas individuals with a family history of recovered stuttering also tended to recover.

Temperament—In the past few years there has been some interest in whether language problems are associated with temperament (based on an individual's physiology). The

Europe PMC Funders Author Manuscripts

Behavioral Style Questionnaire has been used with 3-7 year-old children (McDevitt & Carey, 1978) and the Middle Childhood Temperament Questionnaire (MCTQ) by Hegvik, McDevitt and Carey (1995) has been used with 8-12 year-old children. The MCTQ measures temperament along nine dimensions: activity level (amount of physical motion in the child's behavior), rhythmicity (regularity of physiological functions), approach-withdrawal (the nature of the initial response when in a new situation), adaptability (ease with which the child changes his or her routines), intensity of reactions (reaction to disappointment or failure), quality of mood (amount of positive or negative emotion), attention-span/persistence (time for which the child pursues a particular activity), distractibility (how easily diverted from ongoing behavior by extraneous stimuli), and threshold of responsiveness (amount of stimulation necessary to evoke a discernible response from the child). Comparison across three studies that compared fluent and stuttering children showed that only the adaptability dimension was significant, and in a corresponding direction (Anderson, Pellowski, Conture & Kelly, 2003; Embrechts, Ebben, Franke & van de Poel, 2000; Howell, Davis, Patel, Cuniffe, Downing-Wilson, Au-Yeung & Williams, 2004). An area which has not been investigated to date is whether temperament dimensions that are based on supposed physiological substrates, differed between persistent and recovered individuals. This was examined in the study reported below.

Sensory—A number of reports have implicated the auditory system in stuttering. The main line of support was that fluency control improved in participants who stuttered if the sound of the voice was altered before the participant heard it. Noise maskers (Cherry & Sayers, 1956; Dewar, Dewar, Austin & Brash, 1979), a frequency shifted version of the voice (Howell, El-Yaniv & Powell, 1987), or a delayed version of the voice (Ryan, 1974) all improved control in participants who stuttered.

Attempts have been made to pinpoint the type of auditory processing that is associated with stuttering. Central auditory processing may differ between speakers who stutter and controls. One measure of central auditory processing is backward masking which has been found to be associated with specific language impairment (SLI) (Wright, Lombardino, King, Puranik, Leonard & Merzenich, 1997). Wright et al. (1997) reported that SLI children had higher backward masking thresholds, but similar simultaneous masking thresholds, compared with control children. Backward masking deficits with SLI children have proved difficult to replicate, possibly because performance is more variable in disordered populations (Hill, Hogben & Bishop, 2005). Another possibility is that performance deficits are more acute in certain subgroups within a disorder than in others. This seems to be the situation with persistent and recovered speakers who stutter. Differences between controls and groups containing children who subsequently persisted and recovered from their stutter sometimes led to differences (Howell, Rosen, Hannigan & Rustin, 2000) and sometimes did not (Howell & Williams, 2004). When comparison was made between confirmed persistent and recovered speakers, a statistically significant difference in backward masking performance was found (Howell, Davis & Williams, 2006). To date, no comparison has been made of backward masking performance of persistent and recovered speakers with controls. Such analyses are reported below.

Motor—Performance needs to be assessed in motor tasks that depend on the cerebellum if this structure is implicated in stuttering (Howell, 2004). Cerebellar functioning can be assessed using the battery of ten tests used by Dow and Moruzzi (1958). The battery is divided into three components – tasks involving balance/posture, complex movements and hypotonia. No reports have been made of cerebellar performance using the Dow-Moruzzi battery on people who stutter (either when stuttering first starts or when it perpetuates). However, people with dyslexia have been reported to have deficits on virtually every task in a slightly modified version of the Dow-Moruzzi battery (Fawcett, Nicolson & Dean, 1996).

Europe PMC Funders Author Manuscripts

In the below study, the Dow-Moruzzi battery was administered to children who stutter because cerebellar timing mechanisms have been pinpointed as being involved in persistent stuttering (Howell, 2004).

Research questions—Family history of stuttering was examined to see if evidence could be provided about whether persistence and recovery runs in families. Temperament, sensory and motor performance data were obtained at around 12 plus. All this information was examined to see whether it distinguished: a) all children who stutter from controls, and b) persistent from recovered speakers.

Assessment of a group of children who stutter aged between eight and 12 years plus: A longitudinal study of children who stutter aged between eight and 12 years was conducted. Epidemiology and symptomatology were documented for all participants. A range of etiological factors was examined which, the literature suggested, could lead to persistence of the disorder at age 12 and beyond. The latter were examined on random selections of the participants to confirm whether or not they were risk factors for persistent stuttering. The eight to 12 years age range was selected as there is less information about children at these ages than younger children (Andrews & Harris, 1964; Yairi & Ambrose, 2005) even though onset and recovery have been reported to occur for speakers in this age range (Andrews & Harris, 1964).

Methods

Participants

The longitudinal study was conducted in collaboration with stuttering clinics in the United Kingdom. The children were first seen as secondary referrals to the specialist clinic from other speech language pathologists working in general practices. The children were aged at least eight and they were followed up to 12 years plus, at which age each child was designated as persistent or recovered. All children were confirmed as stuttering at the earlier age. For a speaker to be considered to have persisted, he or she had to be confirmed as still stuttering at 12 years plus. Conversely, for a speaker to be considered to have recovered, he or she had to be confirmed as no longer stuttering at 12 years plus. (Further details of these designations are given below.) Twelve years plus was chosen because recovery was rare beyond this age (Andrews & Harris, 1964).

All referrals (321 in total) to the stuttering clinics were assessed, initially, by one of several trained pathologists located in London. The pathologists were part of a team who delivered group therapy for treatment of stuttering. Samples of spontaneous monologue, dialogue and read speech were obtained at every visit, although only the spontaneous and read materials were used here. Age, gender and the other background details were collected at the first visit (detailed later).

Selection criteria for this study were that a child had to be in the stipulated age range, have been confirmed to have been stuttering before they reached 12 years of age, to have been retested when they were aged 12 plus after a minimum of 12 months since they had received treatment, to have received a course of group therapy and to speak English as their first language. Children who lived far from Central London or received individual therapy were excluded. Seventy-six children met the criteria.

Clinical exposure—The 76 children lived mainly in the south-east of England. They all attended either a one- or two-week clinic with at least one of their parents. Parents were taught to identify the behaviors they were using that were helpful or detrimental to their child's fluency and is an indirect approach that changed the communication environment

Europe PMC Funders Author Manuscripts

rather than attempting to work on the child's speech directly. During treatment, the parents and children were given instructions, training and exercises to deal with the problem, including using slow rates of speech in family interactions, use of different communication styles (looking at the child during conversation etc.), spending more time talking directly with the child (in situations where there was no television or other distractions), and how to cope with bullying, teasing etc. In all cases, treatment was reported to be restricted to that given at the clinic. The children were monitored at specified periods after treatment for 12 months. Only the initial (pre-treatment) speech samples and samples taken 12 months or more after treatment were employed, as this has been suggested as the maximum time needed to determine whether a child has responded to treatment (Finn, 1998). Thus, the children in this study had a consistent amount of treatment, none of the children involved were still affected by treatment protocols and none of the persistent cases had responded to treatment.

Criteria for early, late and very late groups—Three target age ranges were designated; 8-9, 10-11 and 12 plus. Children for whom speech samples and initial assessments were available at 8-9 and 12 plus (whether or not they had a recording at 10-11) were grouped together and are referred to as the early group. There were also children first recorded at 10-11 and also recorded at 12 plus. These are referred to as the late group. Finally, there was a very late group who were first seen when they were aged 12 plus. To be included in this study, they also had to provide another speech sample at least 12 months after treatment. Information provided by respondents for the early, late and very late groups (Ns = 32, 22 and 22 respectively) was analyzed separately.

Assessments made for classifying participants as persistent or recovered—Four assessments were made at 12 plus, which were used to determine whether the child was still stuttering: 1) Participant's assessment; 2) Parent's assessment; 3) Researchery's assessment; 4) a measure of stuttering severity. All these assessments are described in full below. The child, parent and researcher assessments were not done at the initial assessment session (as they were not applicable to pre-treatment because they included questions intended to determine whether the child had recovered or not).

Initial screening (before onset of treatment)—The initial diagnosis was made by a trained pathologist. The criteria for stuttering were that: a) a child had to have been referred to a clinic that specialized in childhood stuttering, and b) the specialist clinic had to confirm that diagnosis and admit the child to group (rather than individual) treatment. Criterion b) kept treatment and follow-up constant for all participants.

Archive recordings obtained at intake—Monologue, dialogue and reading speech samples each of a minimum of 200 syllables were taken when the child first attended the specialist clinic. The samples were recorded on a Sony DAT recorder using a Sennheiser K6 microphone. A note was made of distracting sounds, facial grimaces, head movements and any other body movements. These were used to assess the frequency and duration of stuttering and any associated physical concomitants. All this information was scored according to stuttering severity instrument, SSI-3 (Riley, 1994) using the specified guidelines. SSI-3 scores were obtained by a trained researcher who had about ten years' experience with stuttering. SSI-3 scores were not used for diagnosis at intake.

Child's assessment (minimum 12 months post treatment)—Assessments were based on Boberg and Kully's (1994) questionnaire, which these authors used for assessing the impact of their therapy program on stuttering. The original questionnaire assessed 15 attributes, some of which were specific to their treatment. Seven were directly applicable for

Europe PMC Funders Author Manuscripts

the current assessment (2, 6, 9, 10, 11, 12, 14), and three others (3, 4 and 5) were combined into one further attribute. All eight resulting attributes were assessed by giving a statement to which participants chose a response that matched their view. Table 1 presents the questions, the endpoint tags for the scale and the corresponding question numbers from Boberg and Kully (1994). These were scored in the same way as with the parents (see below).

Parent's assessment (minimum 12 months post treatment)—Parents' views about the fluency of their son or daughter were assessed using the same eight attributes as those used with the child. The statements given in Table 1 were changed from the first to the third person (i.e. to refer to the child). The speech performance questionnaires were completed by the parents and children at the time of the researcher's assessment. The responses to all eight attributes were summed and a threshold (score of 21) used to indicate recovery (score of less than 21) or persistence (score of 21 or greater).

Researcher's assessment (minimum 12 months post treatment)—The researcher who made the initial SSI-3 assessments visited the child's home and recorded an interview that lasted approximately 90 minutes. The researcher gave a rating which was designed to reflect what pathologists do when assessing a client's response to treatment. During his visit, the researcher talked with a parent and the child about the speech problem and experience in clinic. He also sought their views about communication style and self-confidence in a range of typical environments. These included home, social gatherings with adults and children in and out of school. Performance and experience in school were assessed in terms of inter-personal relationships with staff and other children (including bullying). General health issues were also examined, including frequent absence from school and childhood illnesses. Factors taken into consideration were speech fluency, social skills, and whether the child had a positive self image. Each of these three factors was scored on a scale of 0 – 3; for example a score of 0 on social skills indicated outgoing and inquisitive, whilst a score of 3 showed the child was very shy, withdrawn and unresponsive. The scores for the three factors were summed to give one score between 0 (0 on all three scales) and 9 (3 on all three scales); high scores indicated that the disorder was persistent.

Stuttering severity instrument (minimum 12 months post treatment)—During the 90-minute visit referred to, a second 20-minute recording for SSI-3 was made using the DAT recorder and Sennheiser K6 microphone. This was scored in the same way as at the initial assessment (Riley, 1994). This SSI-3 score was used to separate persistent and recovered participants. Participants who persisted had to score 24 or above. The same cutoff was used to designate participants as recovered (scores lower than 24). Subsequent analysis showed (reported below) showed that at intake, most stutterers could be separated from controls using an SSI-3 criterion of 16 (all stutterers had scores above this but few of the controls did). Hypothetically, a person who stuttered could score 16 at intake, increase to a score of 23 at 12 plus and still be considered to have recovered. To preempt this, the additional constraint was added that for individuals to be designated as recovered, SSI-3 scores had to drop by at least two points relative to the original assessment. The 24-point criterion equates to approximately 3-4% stuttered syllables in the speaking and reading tasks, an average dysfluency length of 0.5 – 1 seconds and physical concomitants rated as “not noticeable unless looking for it” or “barely noticeable to the casual observer”.

Combined criteria for designating participants as recovered or persistent—To be designated as persistent, the parent, child and researcher all had to designate the child as still stuttering and SSI-3 score at the time of the second assessment had to be 24 or greater. To be designated as recovered, the parent, child and researcher had to designate the child as

Europe PMC Funders Author Manuscripts

not stuttering, SSI-3 score at the time of the second assessment had to be less than 24 and to show a drop between assessments of at least two points.

Control children—Age-matched control children were obtained from schools in the same geographical area as the children who stutter. Two age groups were recorded and assessed by SSI-3. The first group had a similar age distribution as the early and late stuttering groups at intake. There were 35 speakers (21 male, 14 female). The age range was 8 years to 11 years 9 months and the *mean* was 9 years 11 months (*standard deviation* 13 months). The second group had ages that corresponded to those at the second assessment of all the stuttering groups (12 plus). There were 19 children in this group (11 male and 8 female) and age ranged from 12 years 2 months to15 years 8 months and the *mean* was 14 years one month (*standard deviation* 14 months). All the control children reported no history of speech or hearing difficulty and this was confirmed by the child's teacher. The groups were similar to the children who stuttered in terms of spread of occupational classifications (professional, managerial, skilled non-manual, skilled manual, partly skilled and unskilled). Statistics on educational attainment of the schools were compared with those that the children who stuttered attended and there were no differences (assessment of educational level of individual children at the time of the recordings was precluded because of teaching schedules). The control children were used to provide SSI-3 estimates for a fluent population and included in a pool to supply age and gender matched fluent controls for the etiological studies.

Dysfluency assessments—The children who stuttered were divided into those who persisted and those who recovered. The speech was transcribed using conventions described in detail in Howell and Huckvale (2004). Counts were made of Johnson's dysfluency categories 2-3 (whole word and phrase repetitions, referred to collectively as stallings) and 4-6 (prolongations, part-word repetitions and broken words, referred to collectively as part-word dysfluencies). Each of these dysfluency types was tallied separately for persistent and recovered speakers at intake and at 12 plus. A second judge reassessed eight randomly selected samples from intake and 12 plus from the children who stutter. Inter-judge fluency data were obtained on all words and gave a kappa coefficient of .92 which is much higher than chance (Fleiss, 1971).

Family history of stuttering—Family history data were obtained for participants using questionnaires adapted from Janssen, Kloth, Kraaimaat and Brutten (1996). The data were used to examine whether persistent and recovered forms were transmitted to offspring (Ambrose et al., 1993).

Temperament—The MCTQ, described earlier, was administered (Hegvik et al., 1982) for assessing temperament in 8-12-year-old children.

Auditory assessment—Auditory assessments were made of children who stuttered (at intake and at 12 plus) and of fluent controls matched to the age of the children at 12 plus. Standard air-conducted pure tone audiograms were obtained, which indicated that hearing was within normal limits for all the children whose data are reported below (children who stutter and fluent controls). Further tests were conducted to estimate absolute threshold, simultaneous masking and backward masking performance with a broadband masking stimulus. The latter test condition was an indication of central hearing difficulties and the others were standard assessments. Thresholds for detecting a stimulus which appeared in one of three intervals were estimated in each of these conditions. In all cases, the stimulus consisted of a brief probe tone which was present in one interval (selected at random). The stimulus alone was present in this interval in absolute threshold trials. In the two masking

Europe PMC Funders Author Manuscripts

conditions (simultaneous and backward masking), the tone was masked with a concurrent broadband noise masker or with the same broadband masker that appeared after the tone in the backward masking condition. The intervals were signaled when one of three faces displayed on a computer screen changed from a neutral to an open-mouthed expression (these appeared in left-to-right sequence). The participant indicated which of the three intervals had contained the tone by clicking on the corresponding face graphic. Feedback was given by an appropriate change in the selected graphic (smile or frown). Thresholds were determined using a Levitt (1971) tracking procedure and estimated to within 2 dB. (Further details of the procedure are given in Howell & Williams, 2004, and Howell et al., 2006, which used the same procedures.)

Cerebellar assessment (Dow-Moruzzi motor battery)—The modified form of the Dow and Moruzzi (1958) battery, as described by Fawcett et al. (1996), was used. A description of the three components of the battery (balance, posture, complex movement) follows.

Balance—An electronic force platform, SwayWeigh (Raymar Healthcare Products), was used to measure wobble, defined by the variation in weight-distribution over time. Participants removed their shoes and stood upright on the active plate of the SwayWeigh while it was calibrated for their weight. Following calibration each participant took his/her shoes off and stood with the right foot on the active plate and the left foot on the fixed one (left and right feet were approximately 10 cm apart). The experimenter blindfolded them. They looked straight ahead and stood as still as possible. They were allowed time to become accustomed to the blindfold; this was determined by the experimenter based on when the child was stable with body weight equally distributed between left and right legs. They continued balancing while variation in weight distribution was recorded via a link to a Dell PC onto a Picolog data-recording program for 30 seconds. The dependent variable was the variation in weight distribution over the 30 seconds of the measurement epoch. Balance measurements were obtained: 1) with arms by the side; 2) with arms outstretched with the palms of the hands facing down.

Posture—Posture tasks equivalent to the two balance tasks were performed. The tasks were the same except that during a 30-second trial period the participant was pushed between the second and fourth lumbar vertebrae and on the upper arm from the right and left side by the experimenter. Pressure was applied with the palm of the hand to each contact point for one second and then released. The experimenter exerted approximately 2kg of pressure (previously calibrated by practicing pushing at 2kg on kitchen scales). Analysis for reliability of the researcher's pushing pressure showed that this was accurate to +/− 3%. The dependent variable was the variation in weight distribution over the 30 seconds of each measurement epoch.

Complex movement—The complex movement tasks used were: past pointing, finger-to-finger pointing, and finger-to-thumb opposition. Although it is conceivable that these tasks involve memory as well as motor movement, Ramus, Pidgeon and Frith (2003) have shown (using factor analysis of four cerebellar tasks) that most variance was accounted for by a motor factor.

For the past-pointing task, a bullseye target with ten concentric rings with radii increasing in 10mm steps was fastened onto a wall at eye level and at arm's length from the participant. A marker pen was held in the dominant hand. The participant had five practice attempts and was then blindfolded and made ten test attempts to hit the bullseye with the marker pen. The marker pen provided a permanent record of performance. The attempts were scored ten for

Europe PMC Funders Author Manuscripts

the bullseye and the score decreased by one for each ring. Attempts that fell outside the target received no points. The ten attempts for each participant were added together to give a score of between 0 and 100 for each participant. The dependent variable for this task was the cumulative score over ten attempts.

In the finger-to-finger task the participant placed the index finger of the non-dominant hand through a 10mm-radius hole at the center of a bullseye target. The target had four concentric rings that increased in radius by 10mm. Participants brought the hands together in front of their body and attempted to touch their index fingers together as quickly as possible. Participants had two practice attempts and were then blindfolded and made five experimental attempts. Attempts scored five when fingers touched, and scored one less for each concentric ring. Attempts that fell outside the target received no points. The dependent variable for this task was the cumulative score over five attempts (giving a score of between 0 and 25 for each participant).

The finger-to-thumb task required the index finger and thumb of one hand to be placed on the thumb and finger, respectively, of the other hand. The top thumb and finger were kept together and one hand was turned clockwise and the other counterclockwise until the finger and thumb touched again and then the direction of the moving thumb and finger was reversed. Participants practiced the sequence until they completed five movements fluently. They then performed the successive opposition ten times, as fast as possible. The time taken to do this was recorded by stopwatch. The dependent variable for this task was the total time taken to perform ten movements.

Reliability of the estimate was assessed relative to six independent raters who timed these actions from video recordings of seven participants. The times recorded by the independent raters and the experimenter varied by +/− 0.25 sec (approximately 4%) which was not significant by a paired sample *t*-test ($t(6) = -2.28$, *ns*). Inter-rater reliability was 1.00 to two decimal points (significant at $p<.001$). The experimenter timed the videos on two separate occasions a week apart. These were used for estimating intra-rater reliability. The times varied by +/− 0.11 sec (approximately 2%). A paired sample *t*-test revealed no significant difference between the two times ($t(6) = 1.40$, *ns*). Intra-rater reliability was 1.00 (to two decimal points again).

Results

Epidemiology

Sex ratio—A higher number of males (64) than females (12) stuttered (a ratio of 5.33 males to each female) and the difference was significant by sign test ($p<.00001$).

Recovery rate—Recovery rates were around 50% overall, for both genders and for each age group (section a of Table 2). The differences in recovery rates between early, late and very late age groups were not significant. Most of the epidemiological findings were not significant (exceptions are indicated in the text). However, it was notable that recovery rate was about 12% higher for the late and very late groups than for the early group.

Age of onset—Sixty-two out of 76 participants (81.6%) indicated the age at which their stuttering started (the remainder were asked, but could not remember). Section b of Table 2 shows that age of onset was about four and a half years overall, and this was about the same for both genders, for persistent and recovered speakers and for each age of referral group (the differences were not significant statistically).

Age of recovery—All 41 recovered participants reported the age at which they had recovered. It was notable that the range was large. As seen in section c of Table 2, once again, there was little difference between the two genders. There was an insignificant trend across the three age groups for age of recovery to happen later, the later the age of referral.

Length of time stuttered and length of time recovered group followed up post recovery—It was possible to determine the length of time the recovered participant stuttered for 32 participants (78% of the recovered group); the age at which their stuttering started was not supplied for the remainder. Speakers who recovered stuttered for 8 years 11 months on average. No analysis was made across genders as only two females provided data. The differences in length of time participants stuttered did not differ significantly between early, late and very late age groups (all results are given in section d of Table 2).

All 41 children who recovered were followed up for a minimum of two years up to a maximum of eleven years. The average follow-up time was 5 years 10 months. Females were followed up for significantly longer than males ($t(30) = 2.024$, $p < .05$). Statistically, all age groups were followed up for the same amount of time. Statistics are summarized in section e of Table 2.

To summarize, only gender had effects on eventual stuttering outcome.

Symptomatology (SSI-3 scores and dysfluency counts)

A summary of the SSI-3 scores is given in Figure 1 as box and whisker plots. The plot at the left gives the intake scores separately for the children subsequently identified as persistent and recovered, and for the intake controls. The plot at the right of Figure 1 gives the comparisons between persistent and recovered speakers at age 12 plus and their age-matched controls.

At the first recording every participant who stuttered (persistent and recovered) scored 16 or higher on SSI-3. An SSI-3 score of 16 is rated as mild and reflects about 1-3% stuttered syllables in the speaking and reading tasks, an average dysfluency length of less than one second, and physical concomitants rated as noticeable when looking for them. Of the 35 intake-controls, two scored 16 and six greater than 16 (out of 35). The 16-point criterion divided the two fluency groups approximately. The participants in the stuttering groups were divided at intake into persistent or recovered using the information obtained subsequently. Statistical comparisons were made by independent *t* test for the three pairs of groups (shown in the first column of Table 3). The intake scores of those speakers who went on to persist differed from those of the controls and from those speakers who went on to recover. The persistent speakers also differed from the recovered ones.

Comparisons were made next on the 12-plus SSI-3 scores for the three pairs of groups using age-matched control speakers (19 in total). The results are shown in the second column of Table 3 and revealed a different pattern from those at intake. At 12 plus, the control speakers had significantly lower scores than the persistent speakers, but now the controls did not differ from the recovered speakers. Also, the scores of the persistent speakers were significantly higher than those of the recovered speakers. The different pattern of results was not because a different control group was used, because when the younger intake control group was used for the present comparisons (third column in Table 3) the same pattern of results was obtained as just reported. Thus, the control speakers had significantly lower scores than the persistent speakers but not the recovered speakers. The persistent speakers differed significantly from the recovered speakers. This showed the SSI-3 scores of the recovered speakers decreased and became indistinguishable from either group of controls. As groups were divided at 12 plus into persistent/recovered using an SSI-3 criterion, differences between these groups would have been expected. It would not follow, however that the scores of the recovered speakers would be the same as the controls.

Counts of stalling and part-word dysfluency types were made on the two-minute spontaneous speech samples of each speaker. Figure 2 shows the means and standard errors at initial and final assessment (labeled on the abscissa). Data from persistent speakers are joined by dotted lines and those of recovered speakers by solid lines. Stallings and part-word dysfluencies are given separate symbols and can be identified from the inset panel.

Statistical analysis confirmed the impression in Figure 2, that there was no significant difference between: 1) stallings; and 2) part-word dysfluencies for persistent and recovered speakers at intake. At the later attendance, for the persistent speakers, part-word dysfluencies increased whilst stallings decreased. The persistent speakers showed a cross-over interaction pattern in which part word dysfluencies started low and increased over time whereas stallings showed the opposite pattern. An ANOVA on these speakers with factors session (intake/12 plus) and dysfluency type (stalling/part-word) confirmed this, as the interaction term was significant ($F(1,136) = 31.70$, $p < .001$). There was also a significant effect of session which showed dysfluency rate decreased for this group ($F(1,136) = 14.33$, $p < .001$).

The corresponding data for the recovered participants showed a decrease for both types of dysfluency over time. A similar ANOVA to that conducted on the persistent speakers confirmed that the interaction was not significant, but there was a significant effect of session again, showing dysfluency rate decreased over time for this group ($F(1,160) = 4.77$, $p < .05$).

SSI-3 scores showed that the recovered group shifted away from the persistent speakers and towards the fluent controls. At 12 plus, the SSI-3 scores of recovered speakers were not distinguishable from those of controls. Persistent speakers were different from controls insofar as they maintained high SSI-3 scores. The persistent speakers also showed an increased number of part-word dysfluencies over time whilst the recovered speakers showed a reduction in both stallings and part-word dysfluencies over time to the level of those of fluent speakers.

Etiological factors

Genetics—Forty-one of the 76 children who stuttered provided family history data (33 male and 8 female). Eighteen were persistent (12 males, six females) and 23 were recovered (21 males, two females). Twenty-three were from the early group, ten were from the late group and eight were from the very late group. Family history data were obtained for 42 control probands matched in age to the stuttering probands (20 male and 22 female).

The Ambrose et al. (1997) data showed that individuals with a family history of persistent stuttering also tended to persist, whereas individuals with a family history of recovered stuttering also tended to recover. This claim was checked on the current data with older probands than those used by Ambrose et al., and age-matched controls were also available for analysis. There was a similar ratio of persistent to recovered relatives in each of the three proband groups. There was no association between persistent versus recovered probands and persistence/recovery of relatives ($\chi^2 = 0.07$, $df = 1$, *ns*). When the control probands were included as a third stuttering-type group, there was still no significant association between the stutter type of the proband (persistent/recovered/not stuttering) and persistence/recovery of relatives ($\chi^2 = 0.09$, $df = 2$, *ns*). To summarize, no evidence was found for this group that persistent and recovered forms of stuttering run in families.

Europe PMC Funders Author Manuscripts

Europe PMC Funders Author Manuscripts

Temperament—MCTQ data were available for 15 children who stuttered (12 male, three female, *mean* age 11 years 8 months, *standard deviation* 16) and from 14 age-matched fluent controls (eight male, six female, *mean* age 10 years 10 months, *standard deviation* 25). Eight of the children who stuttered were subsequently classified as persistent and seven had recovered.

When controls were compared to children who stuttered (persistent and recovered in one group) there were significant differences between groups on approach/withdrawal and threshold. Examination of the means indicated that the children who stuttered were significantly more withdrawn than controls and had a significantly higher threshold when reacting to changes in light, temperature and sound. This and the rest of the statistics on temperament that were significant are given in Table 4.

When the persistent group was compared to fluent controls, the significant differences reported earlier remained (and these were in the same direction as those found for the analysis before stuttering groups were differentiated). Additionally, it was found that the persistent group was significantly different from controls on intensity of mood. Examination of the means indicated that the persistent group expressed significantly less emotion than fluent controls.

When the recovered group was compared with the controls, a significant difference in the same direction to that obtained in the earlier analysis on all speakers occurred on the approach/withdrawal factor. It was also found that the recovered group was significantly less adaptable than controls.

Comparison between the persistent and recovered groups also showed a significant difference on the adaptability dimension. The recovered group was again less adaptable than the persistent group.

The main features of the temperament data as they pertain to predicting persistence/recovery are that speakers who recovered had less adaptable temperaments than controls and persistent speakers. Speakers who persisted did not have less adaptable temperaments than controls but they did have less intense mood swings than this group.

Sensory—Standard pure tone audiometry showed that hearing was within normal limits for all participants. More detailed psychoacoustic assessments were made on 48 children who stuttered (40 boys, eight girls) and 36 controls (21 boys and 15 girls). They all provided threshold in absolute, simultaneous masker and broadband backward masker conditions. They were all aged 12 plus when the hearing assessments were made.

The difference between age-matched controls and all stuttering children was only significant for the absolute threshold condition ($t(82)=-2.16$, $p < .05$). The difference in thresholds was small, at 1.6dB, and the estimates were unexpected in that children who stuttered had slightly lower thresholds than the control speakers. On this basis, it may be concluded that there were only minor differences in hearing ability when stuttering groups were not distinguished.

When the children who stuttered were separated into the recovery groups, the difference between persistent and recovered participants was significant for the backward masking condition ($t(28) = 2.58$, $p < .025$). In this condition, the persistent group had higher thresholds (by about 10 dB) than the recovered group although there was overlap in performance between the two groups. No differences between controls and either persistent or recovered speakers were significant. In summary, by 12 plus, the persistent group showed about 10 dB poorer thresholds with a broadband backward-masker compared to controls and recovered speakers.

Motor—Thirty-eight controls and 41 of the children who stuttered (15 who persisted and 26 who recovered) performed the Dow-Moruzzi battery of tests. Data were not available for all tests in the battery due to occasional equipment malfunction (4.3% of the total). The tests were only performed at 12 plus and controls were age-matched.

Analyses were conducted which compared: 1) controls against all the children who stuttered; 2) persistent versus controls; 3) recovered versus controls; and 4) persistent versus recovered. Tests where there were significant differences between groups are summarized in Table 5.

When controls were compared with all the children who stuttered there were differences in past pointing and balance time with arms by the side. In both cases controls performed significantly better than children who stutter.

When persistent and control speakers were compared, there were significant differences on the past pointing task, with controls performing significantly better than persistent speakers. There were also differences on balance time when arms were by the side and balance time with arms outstretched. In both the latter cases, persistent speakers showed significantly more variability than controls.

When recovered and control speakers were compared, there was a significant difference between performance on the past pointing task with controls performing significantly better than the recovered speakers. There were no significant differences on any of the balance tasks.

Finally, when persistent and recovered groups were compared there were differences for balance with arms by the side and postural stability with arms by the side. In both cases recovered speakers showed significantly more variability than persistent speakers.

In terms of persistent/recovered differences, cerebellar motor deficits occurred in children who persisted in their stutter in balance with arms by the side and the body-posture task with arms by the side.

Discussion

The principal goal was to identify risk factors for persistent stuttering in the areas of epidemiology, symptoms and etiology. Several factors were identified which suggested how differential diagnosis of stuttering from fluent speech could be made. Details are given here of on-going work that uses these and other factors to predict persistence and recovery of stuttering employing structural equation modeling (SEM).

One of the main findings of this study was that there was remarkably little difference between children who persisted or recovered from stuttering in the age range 8-12 plus in epidemiology. There was a chance of around 50% of persistence or recovery and this did not depend on gender nor on age of attendance at clinic. Recovery rate was lower in this study than in others with younger children (Andrews & Harris, 1964; Yairi & Ambrose, 2005), which is consistent with the view that most recovery happened when the children were young. It is also of note that, at 53.9%, the estimate of recovery rate was close to the 47% reported by Fritzell (1976) for children with similar ages to those in the current study. Age of onset was roughly the same for both genders and did not depend on age of attendance at clinic. Also, age of onset of four years seven months was comparable with those reported in other studies, including those with younger children (Andrews & Harris, 1964; Yairi & Ambrose, 2005). Age of recovery was the same for gender and age of referral groups, although there was a hint that later attendance at clinic was associated with lower recovery rates. The reason for late attendance at clinic for the children in the current study was not clear, given that onset age was comparable to that reported by Yairi and Ambrose (2005). The children who recovered had stuttered for nearly nine years and they had been followed up, on average, for nearly six years to ensure that there was no relapse (the time the children were followed up was longer than the 40-month period reported by Yairi and Ambrose, 2005). In fact, the only epidemiological factor that was significant was that more boys were affected than girls (5.33:1). This has been reported elsewhere and is considered as a major risk factor in other research. The sex ratio in the current study was higher than in Andrews and Harris (1964). This was expected, as females recover at a younger age than males (Andrews & Harris, 1964) so an older sample should consist predominantly of males.

A score of 16 points on SSI-3 at intake separated persistent and recovered children from 77% of the age-matched fluent controls. At the final assessment, the SSI-3 scores of the recovered group had reduced and they were not statistically distinguishable from their age-matched controls. At this later age, the SSI-3 scores of the persistent group were significantly higher than those for both the recovered and the age-matched control groups. Using persistent/recovered designations determined from the information obtained at later attendance, showed that the recovered speakers' SSI-3 scores were about seven points lower than those of the persistent speakers at intake. Though there was overlap in SSI-3 scores for the two groups of children, the SSI-3 scores at intake could be used in conjunction with other measures for assessing risk of persistent stuttering. SSI-3 scores have been used for contrasting the fluency of speakers who stutter and control speakers by our own and other research groups (Arnold, Conture & Ohde, 2005; Davis, Shisca & Howell, in press; Howell, 2007; Howell et al., 2006). In addition to the symptom changes that were apparent in SSI-3 scores, there was also a change in counts of different dysfluency types for the persistent speakers. Part-word dysfluencies increased relative to stallings for the persistent group of speakers. Both types of dysfluency reduced in speakers who recovered, and the counts of the two types were not distinguishable from age-matched fluent controls at the earlier or later age. Therefore, the increase in counts of part-word repetitions across sessions is another risk factor for persistent stuttering, as noted elsewhere (Conture, 1990; Howell, 2007).

Of the etiological factors examined as potentials for risk, persistence and recovery showed no tendency to run in families. The reason why the current study failed to find fewer persistent relatives in the recovered than the persistent proband group could be due to Ambrose et al.'s (1997) sample being younger than the current one, so recovery of the speakers was not fully resolved in their study. That is, a shift of some speakers from their recovered group who had high numbers of recovered relatives to the persistent group who had few recovered relatives would reduce the reported differences between proband groups. Family history (genetics) was not a risk factor for persistence for speakers in this age range although there are indications from other literature (Yairi & Ambrose, 2005) that it would be useful, particularly when working with younger children.

In all but one case where etiological factors for persistence/recovery were identified, the persistent group performed differently from the recovered and the control groups. The exception was for the temperamental dimension of adaptability where recovered speakers were less adaptable than control speakers or persistent speakers (tests made from age 10 years and upwards). This temperament dimension was the only one that was significant and in the same direction in three previous studies (Anderson et al., 2003; Embrechts et al., 2000; Howell et al., 2004). This was the only temperamental variable that was specific to recovery. In addition, approach and threshold distinguished all the speakers who stuttered from controls. Thus these temperamental variables were not sensitive to persistence/ recovery although they were for stuttering in general. They should be included in a model intended to distinguish the gross fluency groups.

At the time when persistence and recovery were established, backward masking thresholds were about 10dB worse for persistent speakers than for recovered speakers, as also found by Howell et al. (2006). Signal intensity needed to be about 10 dB higher to be detected by persistent participants compared to recovered ones. Backward masking threshold is an additional risk factor that should be used for predicting risk of persistence. It does not appear useful for distinguishing children who stutter in general from fluent speakers, as the scores for the control speakers were not statistically different from the scores of either of the groups of children who stuttered, as reported elsewhere (Howell & Williams, 2004).

Assessments of cerebellar performance were also made at the times when persistence and recovery were established. Variability when balancing with arms by the side was the only factor that differed between persistent and recovered children (recovered children were poorer), and between persistent and controls (persistent children were poorer). Poor performance on balance when arms were held by the side appears to be a risk factor for persistent stuttering. Deficits on the Dow-Moruzzi battery that distinguished between the three fluency groups potentially implicate different cerebellar control in stuttering, as predicted by EXPLAN theory. Past pointing looks like a candidate for differentiating controls from all children who stutter, but not for persistent/recovered groups. Apart from these findings on cerebellar tasks, there were only two other significant effects (postural stability with arms by the side for persistent versus recovered speakers, and balance time with arms outstretched for persistent versus control speakers).

Future work and limitations

The primary goal of the current research was to assess demographics, symptoms and etiological variables as risk factors for persistent stuttering. Demographics and symptomatology were examined on all participants. Etiological factors were examined only for subsamples of the participants. This allowed a wide range of potential risk factors to be explored. More remains to be done with the current data. In particular, examination of the linguistic properties of the language sample is ongoing.

The approach of trawling a wide range of factors in sub-samples allowed those that are associated with a high risk of persistence to be identified. Others that do not appear to be important for this purpose do not need to be collected further. The current results suggest most epidemiological factors are not relevant for risk of persistence, nor are many of the temperament dimensions and standard measures of auditory function. There are other factors that the literature suggests may also be relevant for predicting persistence, such as state anxiety, which has been reported to differ between persistent and recovered speakers at 12 plus (Davis et al., in press). Anxiety could only be a risk factor for persistence if it was present at an early stage. To establish whether this is the case or not, suitable tests that can be employed for measuring children's anxiety would need to be developed; parental report tests have been used by others (Yairi & Ambrose, 2005). Speakers who go on to persist in their stuttering would have to show higher anxiety levels than recovered speakers at the earlier age.

After the set of risk factors has been defined and data obtained, their success at predicting persistence and recovery can be assessed retrospectively (on available data) and prospectively (on newly-collected data). One appropriate technique is SEM (Levine, Petrides, Davis, Jackson & Howell, 2005) which allows models to be set up in terms of how variables relate to each other in the form of a path diagram. The pattern of correlations

Europe PMC Funders Author Manuscripts

between variables can then be inspected to see whether the model is a reasonable fit to the data. SEM distinguishes observed variables (actually measured) from latent variables (hypothetical constructs). If EXPLAN were set up as an SEM model for predicting persistence versus recovery, the latent variables would be: 1) linguistic planning; and 2) motor programming and execution. Language factors that are measured should correlate together and with the planning latent variable. Similarly, motor factors should correlate together and with the execution latent variable. According to EXPLAN, fluency problems arise from poor coordination between the two processes inherent in the latent variables. Thus observed variables that affect both latent variables (correlate with planning and execution) should occur. One method for locating variables that affect both latent variable would be to insert a phrase in a syntactically easy or complex frame (Kleinow & Smith, 2000) and assess variability in how the utterance is spoken at two speech rates (Smith & Kleinow, 2000). The condition where linguistic and motor processing demands are high should correlate with planning and execution variables. The condition where linguistic demand is high but motor demand is low should correlate with the planning variable alone. The condition where linguistic demand is low but motor demand is high should correlate with the execution variable.

Specification of an SEM model also focuses attention on what observable output variables are appropriate for determining the state of stuttering as persistent or recovered). Some authors advocate speech measures as paramount (Wingate, 2001), whereas clinicians often emphasize the importance of a person's self esteem in speaking situations. Some fundamental questions about outcome measures can be addressed using SEM techniques: Can you have cases where self esteem is improved but speech performance is not and vice versa? Does persistent stuttering affect one of these variables (speech performance) and recovered forms affect the other (self esteem)?

Apart from modeling persistence and recovery, these data are also valuable for: 1) sub-typing different forms of stuttering; and 2) comparison with other groups of individuals reported to be stuttering. An example of the former would be to divide the sample into stutterers and clutterers and see whether they show the same pattern of epidemiology, symptomatology and etiology to answer questions about the relationship between these forms of dysfluent speech. Examples of the latter would be comparison with individuals whose primary home language is not English to determine whether they are truly stuttering.

The main limitation in these data is that many of the tests cannot be conducted at early and late ages. This was mentioned in connection with anxiety, where there are no tests suitable for young children. Similar limitations apply to sensory and motor tasks. Availability of tests that can be carried out at a range of ages would allow research questions raised by this study to be examined. For example, they could be used to address why recovered speakers acquired a problem in past pointing (a complex movement task). Hypothetically, it is possible that maintaining fluency focuses attention on speech control. This may be at the expense of other complex movement tasks like past pointing. This possibility is worth further investigation as, if it is correct, it would indicate that achieving fluency can affect movement complexity tasks.

The current study has also been limited to assessment of persistence and recovery. All individuals had treatment to keep this factor constant. Thus, there were no individuals who had no treatment. Therefore, the impact of treatment versus lack of it on recovery rates cannot be determined.

Europe PMC Funders Author Manuscripts

Clinical implications

Clinicians may monitor the following six factors (identified in the current study) that put a speaker at risk of persisting in stuttering: 1) gender; 2) SSI-3 scores (those of persistent stutterers were higher than those who recovered); 3) part-word repetition rate and how it changes over time (this increased in persistent speakers); 4) persistent speakers had more adaptable temperaments than recovered speakers at intake; 5) the persistent speakers had worse backward masking thresholds than the recovered speakers at 12 plus; 6) balance with arms by the side was more variable in recovered than persistent speakers at 12 plus. Bear in mind that factors 4-6 were identified from randomly selected subsets of the overall sample rather than the entire sample.

At the first recording all children scored 16 or higher on the SSI-3, and 72 out of the 76 (95%) scored 21 or higher, which would be classified as moderate stuttering using SSI-3. The demographics section showed that the children were, generally speaking, well past the age where they reported stuttering had started and that chance of persistence did not depend on age group. It is surprising that these children attended late at clinic given that they clearly had a problem, as revealed by the SSI-3 scores at their first attendance and the earlier they attended the less time they stuttered. The late attendance may be, as Yairi and Ambrose (2005) observed, due to the oft-repeated cliché: leave stuttering alone and it will go away. Progressive speech, motor, sensory and temperamental changes occur. The main lesson for the pathologist is, then, how to get the message over not to delay treatment.

It seems important to attempt a comparison of the present results for older children who stutter, with those of Yairi and Ambrose (2005) for young, preschool-age children. To begin, the children in current study all received treatment, as did 89.5% of the participants in Yairi and Ambrose. In both studies, children who persisted did not respond to treatment. For obvious reasons, it is not known whether the persistent children in the present study would have recovered if they had received treatment earlier. In addition, it is not known whether some of the persistent children in Yairi and Ambrose's study showed unassisted recovery at later ages. The most cautious assumption, given the different possible pathways that persistence and recovery may take in older and younger children, is that the factors that put a child at risk for persistent stuttering apply at all ages.

It is also worth considering how both age and developmental changes across the children in both studies, along with the risk factors for persistence and recovery may have influenced the outcome of the treatment received. One possibility assumes that divergence between recovered and persistent children occurs at older ages (i.e. the present sample), but does not differ in form from that reported by Yairi and Ambrose for younger children. In the present study, children who recovered across the range of ages exhibited similar proportions of stalling and advancing dysfluency types. In this sense, they did not change their pattern of stuttering. Conversely, children who persisted changed their patterns of stuttering over time, as did the younger children in Yairi and Ambrose's study.

The observation that changes in stuttering type and proportion over time characterize persistence may suggest that protracted and perhaps habituated production of stuttering leads to adaptations in the central nervous system, and that such adaptations reduce the chance of recovery, either naturally or as the result of therapy (e.g., Howell et al., 2000). Such adaptations may arise from the cerebellum which has traditionally been linked to sensory-motor integration (Stein & Glickstein, 1992). Adaptations in other areas of the brain, such as the arcuate fasciculus (Watkins, Smith, Davis & Howell, in press), might also potentially be involved. It may be the case that adaptive modifications in central nervous system functioning occurring up to and beyond age 12 yield more intransigent stuttering behavior that requires different treatments from those that are effective for younger ages. Reports in the clinical literature suggest that this is the case.

A second possibility is that a positive family history of persistent stuttering is associated with a genetic predisposition for no, or limited, response to treatment. Presently, this speculation is in need of empirical support. It is possible that children in families with a history of persistence may exhibit temperament, speech and central nervous system behaviors and adaptations that interact through family history, and that therapy specifically designed for such intransigent forms is required.

Acknowledgments

This work was supported by grant 072639 from the Wellcome Trust to Peter Howell.

References

Ambrose NG, Cox NJ, Yairi E. The genetic basis of persistence and recovery in stuttering. Journal of Speech, Language and Hearing Research. 1997; 40:567–580.

Ambrose NG, Yairi E. Normative disfluency data for early childhood stuttering. Journal of Speech, Language and Hearing Research. 1999; 42:895–909.

Ambrose NG, Yairi E, Cox N. Genetic aspects of early childhood stuttering. Journal of Speech and Hearing Research. 1993; 36:701–706. [PubMed: 8377483]

Anderson JD, Pellowski MW, Conture EG, Kelly EM. Temperamental characteristics of young children who stutter. Journal of Speech, Language and Hearing Research. 2003; 46:1221–123.

Andrews G, Craig A, Feyer A-M, Hoddinott S, Howie P, Neilson M. Stuttering: A review of research findings and theories circa 1982. Journal of Speech and Hearing Disorders. 1983; 48:226–246. [PubMed: 6353066]

Andrews, G.; Harris, M. The syndrome of stuttering. London: Heinemann; 1964. Clinics in Developmental medicine. No 17

Andrews G, Morris-Yates A, Howie P, Martin N. Genetic factors in stuttering confirmed. Archives of General Psychiatry. 1991; 48:1034–1035. [PubMed: 1747019]

Arnold HS, Conture EG, Ohde RN. Phonological neighborhood density in the picture naming of young children who stutter: A preliminary study. Journal of Fluency Disorders. 2005; 30:125–148. [PubMed: 15949541]

Boberg E, Kully D. Long-term results of stuttering treatment program. Journal of Speech and Hearing Research. 1994; 37:1050–1059. [PubMed: 7823551]

Cherry C, Sayers B. Experiments upon the total inhibition of stammering by external control and some clinical results. Journal of Psychosomatic Research. 1956; 1:233–246. [PubMed: 13377328]

Conture, EG. Stuttering. second edition. Englewood Cliffs, NJ: Prentice Hall; 1990.

Cox N, Kramer P, Kidd K. Segregation analysis of stuttering. Genetic Epidemiology. 1984; 1:245–253. [PubMed: 6549563]

Davis S, Shisca D, Howell P. Anxiety in speakers who persist and recover from stuttering. Journal of Communication Disorders. in press.

Dewar A, Dewar AW, Austin WTS, Brash HM. The long-term use of an automatically triggered auditory feedback masking device in the treatment of stammering. British Journal of Disorders of Communication. 1979; 14:219–229.

Dow, RS.; Moruzzi, G. The physiology and pathology of the cerebellum. Minneapolis: University of Minnesota Press; 1958.

Dworzynski K, Remington A, Rijksdijk F, Howell P, Plomin R. Genetic and environmental etiology in cases of recovered and persistent stuttering in an unselected longitudinal sample of young twins. American Journal of Speech-Language Pathology. 2007; 16 xxx-xxx.

Embrechts, M.; Ebben, H.; Franke, P.; van de Poel, C. Temperament: A comparison between children who stutter and children who do not stutter. In: Bosshardt, H-G.; Yaruss, JS.; Peters, HFM., editors. Fluency Disorders: Theory, Research, Treatment and Self-Help. Proceedings of the Third

World Congress on Fluency Disorders in Nyborg, Denmark; Nijmegen: Nijmegen University Press; 2000. p. 557-562.

Fawcett AJ, Nicolson RI, Dean P. Impaired performance of children with dyslexia on a range of cerebellar tasks. Annals of Dyslexia. 1996; 46:259–283.

Felsenfeld S, Kirk KM, Zhu G, Statham DJ, Neale MC, Martin NG. A study of the genetic and environmental etiology of stuttering in a selected twin sample. Behavior Genetics. 2000; 30:359–366. [PubMed: 11235981]

Finn, P. Recovery without treatment: A review of conceptual and methodological considerations across disciplines. In: Cordes, AK.; Ingham, RJ., editors. Treatment efficacy for stuttering: A search for empirical bases. San Diego: Singular Publishing Group; 1998.

Fleiss JL. Measuring nominal scale agreement among many raters. Psychological Bulletin. 1971; 76:378–382.

Fritzell, B. The prognosis of stuttering in schoolchildren: A 10-year longitudinal study; Proceedings of the XVI Congress of the International Society of Logopedics and Phoniatrics; Basel: Karger; 1976. p. 186-187.

Hegvik, RL.; McDevitt, SC.; Carey, WB. The Carey temperament scales. Scottsdale, AZ: Behavioral-Developmental Initiatives; 1995.

Hill PR, Hogben JH, Bishop DMV. Auditory frequency discrimination ability in children with specific language impairment: A longitudinal study. Journal of Speech, Language and Hearing Research. 2005; 48:1136–1146.

Howell, P. The EXPLAN theory of fluency control applied to the treatment of stuttering by altered feedback and operant procedures. In: Fava, E., editor. Pathology and therapy of speech disorders. Amsterdam: John Benjamins; 2002. p. 95-118.

Howell P. Assessment of some contemporary theories of stuttering that apply to spontaneous speech. Contemporary Issues in Communicative Sciences and Disorders. 2004; 39:122–139.

Howell P. Signs of developmental stuttering up to age eight and at 12 plus. Clinical Psychology Review. 2007; 27:287–306. [PubMed: 17156904]

Howell, P.; Davis, S.; Patel, H.; Cuniffe, P.; Downing-Wilson, D.; Au-Yeung, J.; Williams, R. Fluency development and temperament in fluent children and children who stutter. In: Packman, A.; Meltzer, A.; Peters, HFM., editors. Theory, Research and Therapy in Fluency Disorders; Nijmegen: Nijmegen University Press; 2004. p. 250-256.

Howell P, Davis S, Williams SM. Auditory abilities of teenage speakers who persisted, or recovered, from stuttering. Journal of Fluency Disorders. 2006; 31:257–270. [PubMed: 16920188]

Howell, P.; El-Yaniv, N.; Powell, DJ. Factors affecting fluency in stutterers when speaking under altered auditory feedback. In: Peters, H.; Hulstijn, W., editors. Speech Motor Dynamics in Stuttering. New York: Springer Press; 1987. p. 361-369.

Howell P, Huckvale M. Facilities to assist people to research into stammered speech. Stammering Research. 2004; 1:130–242. [PubMed: 18418475]

Howell P, Powell DJ. Hearing your voice through bone and air: Implications for explanations of stuttering behaviour from studies of normal speakers. Journal of Fluency Disorders. 1984; 9:247–264.

Howell P, Rosen S, Hannigan G, Rustin L. Auditory backward-masking performance by children who stutter and its relation to dysfluency rate. Perceptual and Motor Skills. 2000; 90:355–363. [PubMed: 10833723]

Howell P, Williams S. Development of auditory sensitivity in children who stutter and fluent children. Ear and Hearing. 2004; 25:1–10. [PubMed: 14770013]

Janssen P, Kloth S, Kraaimaat F, Brutten G. Genetic factors in stuttering: a replication of Ambrose, Yairi and Cox's (1993) study with adult probands. Journal of Fluency Disorders. 1996; 21:105–108.

Johnson, W.; and associates. The onset of stuttering. Minneapolis: University of Minnesota Press; 1959.

Kidd K, Kidd J, Records M. The possible causes of the sex ratio in stuttering and its implications. Journal of Fluency Disorders. 1978; 3:13–23.

Kleinow J, Smith A. Influence of length and syntactic complexity on the speech motor stability of the fluent speech of adults who stutter. Journal of Speech, Language and Hearing Research. 2000; 43:548–559.

Kully D, Boberg E. An investigation of interclinic agreement in the identification of fluent and stuttered syllables. Journal of Fluency Disorders. 1988; 13:309–318.

Levine SZ, Petrides KV, Davis S, Jackson C, Howell P. The use of structural equation modeling in stuttering research: Concepts and directions. Stammering Research. 2005; 1:344–363. [PubMed: 18677423]

Levitt H. Transformed up-down methods in psychoacoustics. Journal of the Acoustical Society of America. 1971; 49:467–477. [PubMed: 5541744]

Mannson H. Childhood stuttering: Incidence and development. Journal of Fluency Disorders. 2000; 25:47–57.

McDevitt SC, Carey WB. The measurement of temperament in 3-7 year old children. Journal of Child Psychology and Psychiatry and Allied Disciplines. 1978; 19:245–253.

Ooki S. Genetic and environmental influences on stuttering and tics in Japanese twin children. Twin Research. 2005; 8:69–75.

Ramus F, Pidgeon E, Frith U. The relationship between motor control and phonology in dyslexic children. Journal of Child Psychology and Psychiatry. 2003; 44:712–722. [PubMed: 12831115]

Riley, GD. Stuttering Severity Instrument for Children and Adults. Third Edition. Austin, TX: Pro-Ed; 1994.

Rommel, D.; Hage, A.; Kalehne, P.; Johannsen, H. Developmental, maintenance, and recovery of childhood stuttering: Prospective Longitudinal data 3 years after first contact. In: Baker, K.; Rustin, L., editors. Proceedings of the Fifth Oxford Disfluency Conference; Windsor, Berkshire: Chappell Gardner; 2000. p. 68-182.

Ryan, B. Programmed stuttering therapy for children and adults. Springfield: C. C. Thomas; 1974.

Ryan B. A longitudinal study of articulation, language, rate, and fluency of 22 preschool children who stutter. Journal of Fluency Disorders. 2001; 26:107–127.

Smith A, Kleinow J. Kinematic correlates of speaking rate changes in stuttering and normally fluent adults. Journal of Speech, Language and Hearing Research. 2000; 43:521–526.

Stein JF, Glickstein M. Role of the cerebellum in visual guidance of movement. Physiological Reviews. 1992; 72:972–1017.

Tallal P, Piercy M. Defects of non-verbal auditory perception in children with developmental aphasia. Nature. 1973; 241:468–469. [PubMed: 4705758]

Trouton A, Spinath FM, Plomin R. Twins early development study (TEDS): A multivariate, longitudinal genetic investigation of language, cognition and behavior problems in childhood. Twin Research. 2002; 5:444–448. [PubMed: 12537874]

Vishwanath N, Lee HS, Chakraborty R. Evidence for a major gene influence on persistent developmental stuttering. Human Biology. 2004; 76:401–412. [PubMed: 15481675]

Watkins K, Smith S, Davis S, Howell P. Structural and functional abnormalities of the motor system in developmental stuttering. Brain. in press.

Wingate ME. SLD is not stuttering. Journal of Speech Language and Hearing Research. 2001; 44:381–383.

World Health Organization. International Statistical Classification of Diseases and Related Health Problems, Tenth Revision (ICD-10). Geneva: World Health Organization; 1992.

Wright BA, Lombardino LJ, King WM, Puranik CS, Leonard CM, Merzenich MM. Deficits in auditory-temporal and spectral resolution in language-impaired children. Nature. 1997; 387:176–178. [PubMed: 9144287]

Yairi E, Ambrose NG. Early childhood stuttering I: Persistence and recovery rates. Journal of Speech Language and Hearing Research. 1999; 42:1097–1112.

Yairi, E.; Ambrose, NG. Early childhood stuttering. Austin, TX: PRO-ED; 2005.

Yairi E, Ambrose NG, Paden EP, Throneburg RN. Predictive factors of persistence and recovery: Pathways of childhood stuttering. Journal of Communication Disorders. 1996; 29:51–77. [PubMed: 8722529]

Yairi E, Watkins R, Ambrose N, Paden E. What is stuttering? Journal of Speech, Language and Hearing Research. 2001; 44:585–592.

Europe PMC Funders Author Manuscripts

Europe PMC Funders Author Manuscripts

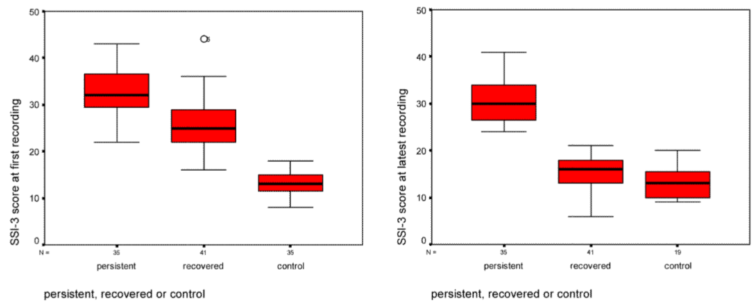

Figure 1.

Box and whiskers plots showing SSI-3 scores and variability for persistent, recovered and control speakers (labeled on the abscissa). The panel on the left presents data from initial assessment and on the right for assessment at 12 plus.

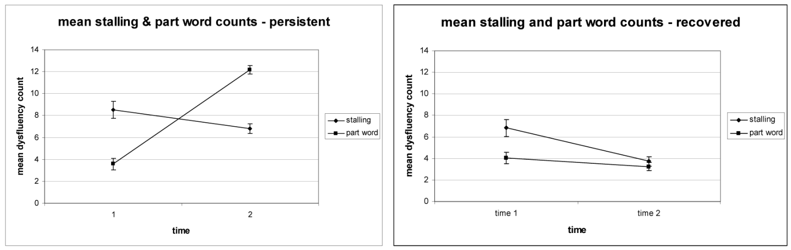

Figure 2.
Mean stalling and part word counts across test sessions (time 1 = initial assessment, time 2 = assessment at 12 plus) for persistent (left-hand panel) and recovered speakers (right-hand panel). The connected lines are for stalling or advancing dysfluency types which can be identified by the symbol (indicated in the inset).

Europe PMC Funders Author Manuscripts

Table 1

The left-hand column gives eight questions which speakers who stutter used to rate their speech. The scale endpoints are given in column two and the question numbers from Boberg and Kully's (1994) questionnaire from which the questions were derived are given in the third column. A modified version of these questions was also used by the parents to assess their child's stuttering.

Question	Scale endpoints	Boberg & Kully question Number
How would you currently rate your speech?	1=Terrific, 5=Terrible	2
How often are you able to speak fluently without thinking about your speech?	1=Always, 5=Never	6
How much are you stuttering/stammering now compared to before you first saw your therapist/pathologist?	1= Much less, 5=Much more	9
How do you feel about your speech now compared to before you first saw your therapist/pathologist?	1=Much better, 5=Much worse	10
How would you describe your consultation with your therapist/pathologist?	1=Very helpful, 5=Of no help	11
Overall, how much of a problem to you is your stuttering/stammering now, compared to before you first saw the therapist/pathologist?	1= Much less, 5=Much more	12
At this time do you consider yourself a person who stutters/stammers?	1=Definitely not, 5=Definitely yes	14
Do you think you would benefit from seeing the therapist/pathologist again?	1= Definitely not, 5=Definitely yes	3, 4 and 5

Europe PMC Funders Author Manuscripts

Table 2

Summary of epidemiological findings. Sections a-e report recovery rates, age of onset, age of recovery, length of time stuttered and length of time followed up respectively. These are broken down by gender and age of attendance group (indicated in column one). A breakdown by persistence and recovered is given for age of onset. Differences which were significant are indicated in the text.

Section a: Recovery rates overall, by gender and by age of attendance group		
	Recovered/total	**Percentage**
Recovery rate over all speakers	41/76	53.9%
Recovery rate by gender:		
Number. of males/all males	37/64	57.8%
Number. of females/all females	4/12	33.3%
Recovery rate for age of attendance:		
Number recovered in early group/total	15/32	46.9%
Number recovered in late group/total	13/22	59.1%
Number recovered in very late group/total	13/22	59.1%
Section b: Age of onset overall, by gender, by persistent/recovered and by age of attendance group		
	Mean (months)	**Standard deviation**
Age of onset over all speakers	54.8	20.9
Age of onset for genders:		
Age of onset for males	54.0	21.1
Age of onset for females	58.5	20.9
Age of onset by recovered types:		
Age of onset for persistent	57.0	24.5
Age of onset for recovered	52.7	17.0
Age of onset for age of attendance group:		
Age of onset for early	50.1	16.6
Age of onset for late	56.6	25.4
Age of onset for very late	60.7	22.5
Section c: Age of recovery overall, by gender and by age of attendance group		
	Mean (months)	**Standard deviation**
Age of recovery over all speakers	158.6	148.0
Age of recovery for genders:		
Age of recovery for males	159.9	25.2
Age of recovery for females	148.0	31.8
Age of recovery for age of attendance group:		
Age of recovery for early	148.0	28.2
Age of recovery for late	162.5	20.4
Age of recovery for very late	167.0	25.3
Section d: Length of time stuttered overall, by gender and by age of attendance group		
	Mean (months)	**Standard deviation**

Section a: Recovery rates overall, by gender and by age of attendance group		
	Recovered/total	**Percentage**
Length of time stuttered over all speakers	106.7	35.5
Length of time stuttered for genders:		
Time males stuttered	108.5	31.4
Time females stuttered	79.5	(only two cases)
Length of time stuttered for age of attendance group:		
Time early group stuttered	96.7	35.5
Time late group stuttered	112.9	31.5
Time very late group stuttered	114.0	24.3
Section e: Length of time followed up overall, by gender and by age of attendance group		
	Mean (months)	**Standard deviation**
Length of time followed up over all speakers	69.9	28.1
Length of time followed up for genders:		
Time males followed up	67.1	27.2
Time females followed up	96.0	25.9
Length of time followed up for age of attendance group:		
Time early group followed up	67.2	29.7
Time late group followed up	76.6	23.8
Time very late group followed up	66.5	31.2

Europe PMC Funders Author Manuscripts

Table 3

Summary of statistical analyses of SSI-3 scores. The groups that were compared are the labels for each row. The SSI-3 scores compared were selected by age for the stuttering groups and for the controls. Which stuttering group was selected and which control group are indicated in the label for the column.

	Stuttering groups at intake, controls at intake where appropriate	**Stuttering groups at 12 plus, controls age-matched to this group where appropriate**	**Stuttering groupsat 12 plus, controls at intake where appropriate**
persistent versus controls	$t(68) = 18.84$, $p < .001$	$t(52) = 14.57$, $p < .001$	$t(68) = 18.67$, $p < .001$
recovered versus controls	$t(74) = 12.14$, $p < .001$	$t(58) = 1.90$, *ns*	$t(74) = 1.754$, *ns*
persistent versus recovered	$t(74) = 5.46$, $p < .001$	$t(74) = 14.91$, $p < .001$	$t(74) = 14.91$, $p < .001$

Europe PMC Funders Author Manuscripts

Table 4

Temperament variables which differed significantly for comparisons between different groups. The groups that were compared are the labels for each row and the temperament variable considered is the label for the column.

	Approach/ withdrawal	**Threshold**	**Intensity of mood**	**Adaptability**
controls versus all stutterers	$t(27)=2.328$, $p<.025$	$t(27)=-2.245$, $p<.025$		
persistent versus controls	$t(20)=1.997$, $p<.05$	$t(20)=-2.745$, $p<.01$	$t(20)=2.843$, $p<.01$	
recovered versus controls	$t(19)=2.180$, $p< .025$			$t(19)=2.110$, $p<.025$
persistent versus recovered				$t(13)=-1.769$, $p<.05$

Europe PMC Funders Author Manuscripts

Table 5

Motor performance variables from the Dow-Moruzzi task which differed significantly for comparisons between different groups. The groups that were compared are the labels for each row and the Dow-Moruzzi tasks considered are given in the label for each column.

	Past pointing	**Balance Arms by side**	**Balance Arms outstretched**	**Postural stability Arms by side**
controls versus all stutterers	$t(75) = -2.95$, $p < .05$	$t(77) = 1.67$, $p < .05$		
persistent versus controls	$t(51) = -3.03$, $p < .05$)	$t(51) = -2.59$, $p < .05$	$t(51) = 1.91$, $p < .05$	
recovered versus controls	$t(60) = 2.22$, $p < .05$			
persistent versus recovered		$t(39) = -2.05$, $p < .05$		$t(37) = 2.49$, $p < .05$