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Interventions for motor apraxia following stroke (Review)

West C, Bowen A, Hesketh A, Vail A

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[Intervention Review]

Interventions for motor apraxia following stroke

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ABSTRACT

Background

Apraxia is a cognitive disorder that can occur after stroke. It prevents a person from carrying out a learned movement. Various interventions are used to treat apraxia but evidence of their benefit has been lacking.

Objectives

To determine which therapeutic interventions targeted at motor apraxia reduce disability.

Search methods

We searched the Cochrane Stroke Group Trials Register (last searched November 2006). In addition, we searched the following databases: the Cochrane Central Register of Controlled Trials (CENTRAL) (*The Cochrane Library* Issue 3, 2006), MEDLINE (1966 to November 2007), EMBASE (1980 to November 2006), CINAHL (1982 to November 2006), PsycINFO (1974 to November 2006), the Research Index of the Occupational Therapy Journal (searched November 2006), REHABDATA (1956 to November 2006), the National Research Register (searched November 2006) and Current Controlled Trials Register (searched November 2006). We reviewed the reference lists of all articles that we identified as relevant. We made efforts to find both published and unpublished trials by writing to key authors and journals.

Selection criteria

Randomised controlled trials of therapeutic intervention for motor apraxia in stroke.

Data collection and analysis

One review author searched the titles, abstracts and keywords. Four review authors extracted data and analysed trial quality. We contacted investigators for further details of trials if necessary.

Main results

Three trials including a total of 132 participants were included in the review. There was evidence of a small and short-lived therapeutic effect in the two studies that reported change in activities of daily living (102 participants) but this was not considered clinically significant and did not persist at the longer-term follow up.

Authors' conclusions

There is insufficient evidence to support or refute the effectiveness of specific therapeutic interventions for motor apraxia after stroke. Further research of higher quality is required. As we did not review whether patients with apraxia benefit from rehabilitation input in general, they should continue to receive general stroke rehabilitation services.

Interventions for motor apraxia following stroke (Review)

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PLAIN LANGUAGE SUMMARY

Interventions for motor apraxia following stroke

People with motor apraxia after stroke often have difficulty carrying out everyday activities such as making a hot drink. Some people cannot select the right object at the right time or have difficulty using objects (such as a spoon) correctly. Apraxia is not due to muscle weakness or sensory loss. Instead it seems to be a loss or disturbance of the conceptual ability to organise actions to achieve a goal. This review of three studies, including 132 participants, suggests that further high quality research is required before specific treatment techniques can be accepted or rejected. Patients with apraxia should continue to receive general stroke rehabilitation services but better quality research is needed to identify optimal apraxia treatments.

BACKGROUND

Cochrane

The World Health Organization has defined stroke as 'a syndrome of rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than vascular in origin' (WHO 1978). Stroke is the largest disabling condition in England and Wales with 100,000 first strokes occurring each year (Blais 1994). Stroke can affect people's physical, sensory and cognitive abilities (Wade 1985). The Stroke Association estimates that in the UK 300,000 of the 60 million population are living with disabilities caused by a stroke (Westcott 2000).

Apraxia is a neuropsychological deficit that can affect stroke patients. It refers to 'disorders of the execution of learned movement which cannot be accounted for by either weakness, inco-ordination, or sensory loss, or by incomprehension of or inattention to command' (Geschwind 1975). In this review we shall confine the discussion of apraxia to that affecting the limbs. Apraxia of speech is dealt with in a separate Cochrane review (West 2005).

Motor apraxia is difficult to diagnose. The available tests are inconsistent and appear to test for different aspects of apraxia (Butler 2002). The taxonomy of motor apraxia has been disputed, but many clinicians and researchers now support the classical idea that there are two forms: ideomotor and ideational (Liepmann 1920). Others have described motor apraxia in functional terms, for example dressing apraxia and the apraxia of gait. These classifications have been disputed as they describe the affected functional task rather than the underlying condition (Geschwind 1985). Ideomotor apraxia can affect the patient by hindering their ability to select, sequence and use objects (Heilman 1985) and it is thought to affect people more in test situations than in normal activities of daily living (ADL). Patients with ideational apraxia are unable to perform a skilled activity because they have lost the conceptual ability to organise the actions required to achieve their goal (Jackson 1999). For example, they may attempt to put clothes on the wrong part of their body. There does not, however, appear to be a clear consensus on the definitions of ideomotor and ideational apraxia (Tate 1995).

The reported prevalence of motor apraxia after stroke is inconsistent. There is evidence to suggest that apraxia affects both left and right-brain damaged patients, with it being more prevalent in the left (Rothi 1997). Both the anterior and posterior lesions in the left hemisphere are known to produce apraxic symptoms, as this is the dominant hemisphere for the storage and execution of learned movements (Kareken 1998). Original studies showed that 50% of patients with right-sided hemiplegia suffered from motor apraxia (Liepmann 1905). This has been confirmed by another study (De Renzi 1980).

Apraxia is thought to have an adverse influence on ADL independence (Goldenberg 1998; Sundet 1988). Research into the different therapeutic interventions available to treat apraxia is limited. Types of interventions include:

 strategy training in daily living activities: this technique teaches internal (for example, the patient is taught to verbalise and implement the task steps at the same time) or external (for example, when aids are used to overcome a functional barrier) compensatory strategies that enable a functional task to be completed. These strategies will not have been used prior to the stroke (Van Heugten 1998);

- sensory stimulation: stimulations including deep pressure, sharp and soft touch are applied to the patients' limbs (Butler 1994);
- proprioceptive stimulation: the patient leans on and puts weight through their upper and lower limbs;
- cueing, verbal or physical prompts: given to enable each stage of the task to be completed;
- chaining (forward or backward): the task is broken down into its component parts. Using backward chaining the task is completed with facilitation from the therapist apart from the final component, which the patient carries out unaided. If successful next time further steps are introduced. Forward chaining is the reverse of backward chaining;
- normal movement approaches: the therapist facilitates the body through normal movement patterns.

Rehabilitation can occur at any phase post stroke. There is a conceptual distinction between the effects a disease may have at different levels (WHO 2001): impairment, activity (disability) and participation (handicap). Therapists' provision of aids and environmental adaptations aim to help the person adapt to their impairment rather than change the underlying impairment itself. Some rehabilitation approaches may be aimed at the level of impairment.

The task of this review is to systematically consider the evidence from randomised controlled trials on the effectiveness of therapeutic interventions aimed specifically at altering motor apraxia following stroke.

OBJECTIVES

The main questions we wish to address are as follows.

(1) In stroke patients with motor apraxia who are undergoing rehabilitation, do therapy interventions targeted at motor apraxia achieve a sustained reduction in disability compared with no or placebo intervention six months after treatment?

(2) In this population, is one specific targeted intervention (compared with another specific targeted intervention) more likely to achieve a sustained reduction in disability?

METHODS

Criteria for considering studies for this review

Types of studies

We included randomised controlled trials of interventions for stroke patients with motor apraxia. We would have excluded from analysis second and subsequent phases of cross-over trials, as the design would not be appropriate in this context.

Types of participants

The review was confined to data from reports of studies on adult patients with motor apraxia (irrespective of the definition of apraxia used by the authors of the study) following a stroke. We excluded trials that included participants whose deficits were the result of head trauma, brain tumour, or other brain damage unless a subgroup of stroke patients could be identified for whom there were separate results, or more than 75% of patients in the sample



are stroke patients. All types of apraxia (that is ideomotor and ideational) were considered for inclusion except apraxia of speech and oral apraxia. Apraxia of speech has been covered in a separate Cochrane review (West 2005).

Types of interventions

We included trials in which a comparison was made between an 'active' treatment group that received one of the various motor apraxia interventions and a control group that received either an alternative motor apraxia intervention, placebo or none. Possible treatment interventions included: tactile and proprioceptive stimulation, strategy training in daily living activities, cueing, chaining, (forward or backward) and normal movement approaches. We excluded trials including only drug therapies. We recorded duration and quantity of intervention.

Types of outcome measures

The primary outcome was the average level of independence in activities of daily living, as defined by the original authors, at six months after therapy. Recognised measures, for example the Barthel Index (Mahoney 1965), the Assessment of Motor and Process Skills (Fisher 1994) and the Functional Independence Measure (Keith 1987) were included.

Secondary outcomes included:

- (1) independence in ADL at the scheduled end of the intervention (ordinal);
- (2) independence in ADL at 12 months (ordinal);
- (3) death (binary);
- (4) quality of life measures (ordinal);
- (5) ability to gesture/pantomime/use objects (ordinal);
- (6) effects on family and carer, e.g. Carer Strain Index, measures of carer's mood (ordinal);
- (7) carer and family perceptions of outcome (ordinal);
- (8) economic resources (continuous);
- (9) apraxic patient's mood (ordinal);

(10) adverse events, e.g. fatigue, falls, accident rates (binary).

Search methods for identification of studies

See: 'Specialized register' section in Cochrane Stroke Group

(1) We searched the Cochrane Stroke Group Trials Register, which was last searched by the Review Group Co-ordinator in November 2006. In addition, we searched the Cochrane Central Register of Controlled Trials (CENTRAL) (*The Cochrane Library* Issue 3, 2006), MEDLINE (1966 to November 2006), EMBASE (1980 to November 2006), CINAHL (1982 to November 2006), PsycINFO (1974 to November 2006), the Research Index of the Occupational Therapy Journal (searched November 2006), REHABDATA (1956 to November 2006), the National Research Register (searched November 2006) and Current Controlled Trials Register (searched November 2006) (Appendix 1).

(2) We had planned to handsearch a number of relevant journals. However, after checking the Master List of journals being searched by The Cochrane Collaboration to avoid duplication of effort (http://www.cochrane.us/masterlist.asp), we found that the selected journals had already been handsearched. The resulting trials would therefore be found from our search of the Cochrane Central Register of Controlled Trials.

(3) We searched the reference lists of all relevant references.

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(4) In order to identify further published and unpublished trials we contacted authors of published apraxia articles and wrote to appropriate journals (*Clinical Rehabilitation, British Journal of Occupational Therapy, Physiotherapy Frontline* and *The Psychologist*).

Data collection and analysis

Selection of trials

One review author (CW) searched titles, abstracts and keywords of both published and unpublished papers to assess their eligibility for inclusion using a systematic approach. Only papers that obviously did not meet the eligibility criteria were discarded. Articles that may have met the inclusion criteria were obtained in full and screened by CW. All review authors read the remaining studies and formed a consensus on the final inclusion and data extraction.

Quality assessment

We described the methodological quality of the included studies for the following aspects:

- concealment of allocation (whether adequate, inadequate, or unclear);
- type of design (e.g. parallel, factorial, cross-over);
- blinding to allocation (of therapist, patient and outcome assessment);
- definition of terms (e.g. of stroke, apraxia, outcome, and intervention);
- intention-to-treat analysis (whether undertaken, possible from report, impossible or unclear);
- completeness of follow up (proportion of randomised patients in analysis).

Data extraction

In addition to outcome data the following were documented by CW and one other review author: (1) settings (e.g. hospital, community, nursing home); (2) type of intervention; (3) length of rehabilitation; (4) profession(s) involved; (5) co-interventions implemented; (6) length of disease; (7) level of severity; (8) presence of other symptoms that may affect the level of disability (e.g. hemiplegia, unilateral spatial neglect); and (9) tools the authors used to identify motor apraxia. We requested information that was unclear or missing from the reports from the corresponding author.

Data analysis

Our primary analysis pooled all therapeutic studies of active intervention versus no or placebo treatment to address objective (1) above. To address objective (2), we also analysed subgroups of studies categorised according to therapeutic approach, as outlined under 'Types of interventions'. This included a comparison of each approach versus no or placebo treatment, and will include direct comparisons of different approaches if any are identified in future updates of this review.

We have treated activities of daily living (ADL) and other ordinal scales for the secondary outcomes as continuous outcomes unless and until accepted meta-analytic techniques for ordinal outcome data become available. We abstracted, calculated or requested means and standard deviations. For all binary outcomes, we

incorporated deaths in the worse outcome category. For practical reasons, we excluded deaths from outcomes that were treated as continuous. Death rates between the two groups were low and similar because studies only included patients who were well enough to undergo rehabilitation for motor apraxia. Any imbalance in death rates between the groups in future updates will be discussed, including descriptive consideration of whether analyses of raw data from individual trials could alter conclusions.

Our intention was to extract mean (SD) for the primary outcome, and this was possible for included studies. If this is not the case in future updates, we will extract and compare binary data for the primary outcome as an additional secondary analysis.

We combined results for continuous outcomes using weighted mean difference by a fixed-effect model. However, it is anticipated that future studies may use different scales to measure the same underlying constructs. If this is the case, we will use the standardised mean difference and results translated back into one of the original scales for reporting purposes. We combined results for binary outcomes using the Peto-modified odds ratio (OR), and translated these to risk differences across the observed range of control group rates for reporting purposes. We noted and discussed statistical heterogeneity.

We carried out sensitivity analyses on the primary outcome. These included use of a random-effects analysis, omission of studies that do not describe an adequate method of allocation concealment, and imputing values for missing data if appropriate.

RESULTS

Description of studies

There were no excluded studies as no studies that appeared to meet the eligibility criteria were found not to on closer examination. Data from 132 participants in three studies were included (Donkervoort 2001; Edmans 2000; Smania 2000). Smania 2000 reported data for 13 patients but we have only included data for the first 10 patients who were appropriately randomised. Edmans 2000 provided segregated data on the nine patients with apraxia included in her published report.

The participants all had lesions in the left hemisphere. Apraxia was defined in Donkervoort 2001using the De Renzi test (De Renzi 1980), in Smania 2000 using the Van Heugten test (Van Heugten 1999), and in Edmans 2000 using the test by Kertesz and Ferro (Kertesz 1984). The mean ages of groups were between 63 and 70 years. The sex (male/female) of the experimental groups was 64/49 (Donkervoort 2001), 8/2 (Smania 2000) and 3/6 (Edmans 2000). The study participants came from the Netherlands (Donkervoort 2001), Italy (Smania 2000), and England (Edmans 2000) and were from either a rehabilitation unit (Donkervoort 2001; Edmans 2000; Smania 2000) or nursing home (Donkervoort 2001). The time since stroke was a mean of about 100 days (Donkervoort 2001), and ranged from two to 36 months (Smania 2000) and from 22 to 76 days (Edmans 2000). In the Donkervoort study (Donkervoort 2001) 56 (19%) participants had recurrent stroke, but none had a history of apraxia prior to their current stroke. There was no previous history of cerebrovascular attacks in the stroke patients participating in the Smania study (Smania 2000), and status was not reported in the Edmans study (Edmans 2000). All studies excluded people with marked psychiatric problems.

The comparisons in the studies differed. Donkervoort 2001 used strategy training (integrated into usual occupational therapy) compared to usual occupational therapy. Smania 2000 compared gesture training for apraxia with conventional treatment for aphasia (Smania 2000). Edmans 2000 compared two specific methods for apraxia in addition to standard occupational therapy: transfer of training and functional approach. As the latter is more standard practice, we have chosen arbitrarily to treat this as the control group. Donkervoort 2001 reported that the experimental group had on average 25 occupational therapy sessions lasting in total 15 hours whilst the control group had 27 occupational therapy sessions with a total of 19 hours, during an eight week period. Patients in Smania 2000 received training sessions of approximately 50 minutes duration three times a week. The gesture training stopped once all training sections were completed, or a maximum of 35 treatment sessions (approximately 11 weeks). In Edmans 2000, participants received training for 2.5 hours per week for six weeks.

In Donkervoort 2001, the assessment of apraxia was made by a trained researcher following clinical screening by the medical team. The intervention was delivered by occupational therapists and assessment made by a blinded research assistant. The professions involved in assessment of eligibility, intervention and outcome assessment are not clear in Smania 2000. In Edmans 2000, a psychologist assessed apraxia at the outset, occupational therapists delivered the interventions, and outcomes were assessed both by nurses and an independent, blinded occupational therapist.

The outcomes used in the studies were different. Donkervoort 2001 reported as primary outcome the Van Heugten (Van Heugten 1999) measure of ADL at end of intervention and at five months after initial assessment, but also reported Barthel among secondary outcomes. Smania 2000 reported a number of impairment outcomes at the end of intervention, but nothing regarding activities of daily living. Edmans 2000 reported a number of outcomes including the Barthel measured both by nurses and occupational therapists at the end of intervention. We have used the occupational therapist assessments in the analyses.

Risk of bias in included studies

All included studies claimed to be randomised controlled trials using two-group parallel designs. Standard, though different, assessments of apraxia and outcomes were used. Due to the nature of the interventions it would not have been possible to blind therapists or patients.

Donkervoort 2001 randomised participants using sequentially numbered, non-transparent, sealed envelopes prepared from random number tables. Allocation was stratified by institution type, time since stroke and apraxia score, and a Zelen correction (Zelen 1974) was used to ensure balance. The outcome assessments were carried out by a blinded research assistant. Patients were not specifically informed which intervention they were receiving, although clearly the interventions would not have appeared similar. Stroke was defined using the WHO criteria (WHO 1989). The trialists referred to an article in which the intervention was defined in sufficient detail to replicate (Van Heugten 1998). Of 113 randomised patients, 108 (96%) underwent baseline assessment, 97 (86%) were assessed at the end of intervention, and 86 (76%) at the final assessment. Reasons for withdrawal at each stage



were reported and balanced between the groups. Analyses were by intention to treat for those patients with outcome data.

Smania 2000 used simple randomisation on the first 10 subjects without mention of concealment. After noticing an imbalance the following three subjects were assigned to the control group and their data have been excluded from our analyses. There was no mention of blinding of outcome assessment, which is a potential source of avoidable bias. Stroke was defined by computerised tomography (CT) scan and clinical evidence of left-sided, unilateral vascular lesions. The intervention was defined in sufficient detail to replicate. There were complete follow-up data for the 10 included patients.

Edmans 2000 described a randomisation scheme using preprepared envelopes from random number tables. Edmans informed the review authors that allocations were stored in sealed, opaque, numbered envelopes, only opened at the time of recruitment in the presence of a witness. The outcome assessments were carried out independently by a blinded nurse and occupational therapist. The post-treatment assessor was blinded to allocation. No definition of stroke was given. Intervention details were not provided in the study or a later paper. Some randomised patients were not assessed for apraxia due to language impairment. Complete follow-up data were made available to this review for the nine patients assessed to have apraxia.

Effects of interventions

The graphs of continuous outcomes are set so that values to the right favour the experimental group. For binary outcomes, lower odds in the experimental group are always shown to the left. For adverse outcomes (such as death) this means that values to the left favour the experimental group.

Our protocol specified comparison of the average levels of independence in activities of daily living. Presented below are comparisons of the average changes from baseline in these levels. These change score analyses have been chosen because they usually provide more precise estimates of the same treatment effects in the randomised trial setting.

Comparison 1.1: Change in Barthel at six months after end of therapy

Only Donkervoort 2001 reported on the primary outcome described in this review's protocol. Using the Barthel ADL Index, the study did not find evidence of a lasting difference in functional performance six months post stroke: mean difference (MD) 0.17, 95% confidence interval (CI) -1.41 to 1.75, P = 0.83, in favour of the experimental group.

Comparison 1.2: Change in Barthel at end of therapy

Donkervoort 2001 and Edmans 2000 both reported the Barthel at end of intervention, and reported very similar group differences. The overall MD was 1.28, 95% CI 0.19 to 2.38, P = 0.02, in favour of the experimental group.

Comparison 1.3: Change in Barthel at 12 months after end of therapy

No trials reported data for this outcome.

Comparison 1.4: Death

There were no deaths in the studies of Edmans 2000 or Smania 2000, but seven in the study by Donkervoort 2001: odds ratio (OR) 0.41, 95% CI 0.09 to 1.9, P = 0.25, in favour of the experimental group but providing no evidence of differential death rates.

Comparison 1.5: Quality of life measures

No trials reported data for this outcome.

Comparison 1.6: Ability to gesture, pantomime, use real objects

Only Smania 2000 reported on this outcome, using both ability to gesture and to use real objects: MD for gesture training 8.4, 95% CI -15.8 to 32.6 points on a 0 to 72 scale, P = 0.50 in favour of the experimental group. MD for using real objects 1.2, 95% CI -3.2 to 5.6 points on a 0 to 14 scale, P = 0.59, in favour of the experimental group but again providing no evidence of differential ability.

Comparison 1.7: Effects on family and carer

No trials reported data for this outcome.

Comparison 1.8: Carer and family perceptions

No trials reported data for this outcome.

Comparison 1.9: Economic resources

No trials reported data for this outcome.

Comparison 1.10: Apraxic patient's mood

No trials reported data for this outcome.

Comparison 1.11: Adverse events

No trials reported data for this outcome.

DISCUSSION

Only Donkervoort 2001 reported on the primary outcome for this review. Using the Barthel Index the study did not find evidence of a lasting difference in functional performance six months post stroke. This review does however suggest that therapeutic intervention produces a small but statistically significant improvement on the Barthel immediately after intervention as both Donkervoort 2001and Edmans 2000 found in favour of the experimental group. These results whilst encouraging have limited application for clinical practice due to the small effect and the fact that it did not persist at follow up. No studies compared one intervention with any other. Only Smania 2000 reported on test performance, for example the ability to gesture and the use of objects. Neither was statistically significant. Death rates were low and similar for all the studies. This was expected as only patients that were well enough to undergo rehabilitation would have been included. No studies reported on quality of life measures, effects on family and carer, their perceptions of outcome, economic resources, mood or adverse events. If future research is carried out it would be appropriate for these to be used as secondary outcome measures.

The review found and included only three trials with a small number of participants (132). All the trials used different therapeutic interventions, including strategy training (Donkervoort 2001), a transfer of training approach (that is, practising one task with the aim of it generalising to related tasks) (Edmans

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2000), and gesture training (Smania 2000). Not all the therapeutic interventions suggested in the literature have been evaluated. The quantity of treatment intervention varied between 15 hours and 29 hours and duration was from six weeks to 19 weeks. The assessment tools used to diagnose apraxia were all different and we are unsure whether they actually measure the same underlying construct. The participants came from rehabilitation units (Donkervoort 2001; Edmans 2000; Smania 2000) and nursing homes (Donkervoort 2001). It is not clear whether participants from rehabilitation units in England and Italy and nursing homes in the Netherlands are comparable in terms of level of dependency. The interventions were only reported in enough detail to replicate in two of the three studies. Edmans 2000 is to report on the intervention in a future article. Without detail of the intervention a trial is of little clinical value.

Donkervoort 2001 used adequately concealed randomisation utilising sequentially-numbered, non-transparent, sealed envelopes, prepared from random number tables. Edmans 2000 used a similar process but the recruiter prepared the envelopes prior to allocation. This is a potential source of bias. It would be preferable if the recruiter were not involved in the preparation of the envelopes. Smania 2000 reported using simple randomisation on the first 10 patients, but once an imbalance was noticed a 'restricted randomisation scheme' was implemented without mention of concealment. The randomisation process is unclear. Donkervoort 2001and Edmans 2000 reported using a blinded outcome assessor whilst Smania 2000 did not mention blinding. This is a possible source of bias.

In summary, the review has not found strong evidence to support therapeutic intervention for motor apraxia in stroke patients. We have found no evidence that the impairment of motor apraxia is altered, or that intervention aimed specifically at motor apraxia alters disability. This should not be misinterpreted as evidence that rehabilitation does not work for patients with motor apraxia. The quality of the studies is acceptable for the review but there are study limitations as outlined above. The findings of this review suggest that good quality randomised controlled trials are warranted. Apraxic assessments used in future studies need to measure both the level of impairment and activity (WHO 2001). Impairment measures are useful for describing the sample and the type and severity of motor apraxia. This is needed for decisions about whether results from the samples studied can be generalised to a typical heterogeneous clinical population. It is also important for future researchers to consider evaluating their treatment in terms of the patients' opinion of outcome.

AUTHORS' CONCLUSIONS

Implications for practice

Specific therapeutic intervention for motor apraxia following stroke cannot be supported or refuted by results from randomised controlled trials.

Implications for research

There is a need for more and higher quality trials of therapeutic intervention for motor apraxia. Trials should be sufficiently large to detect functionally meaningful differences in long-term outcome. Interventions should be explicitly defined and outcome measures need to include how apraxia affects everyday life.

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internal consistency and diagnostic value. The Clinical Neuropsychologist 1999;13:182-92.

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West C, Hesketh A, Vail A, Bowen A. Interventions for apraxia of speech following stroke. Cochrane Database of Systematic Reviews 2005, Issue 4. [Art. No.: CD004298. DOI: 10.1002/14651858.CD004298]

Westcott 2000

Westcott P. Stroke - questions and answers leaflet. London: The Stroke Association, 2000.

CHARACTERISTICS OF STUDIES

Characteristics of included studies [ordered by study ID]

+ 2001

WHO	1978
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World Health Organization. Offset Series No. 43, Cerebrovascular disease: a clinical and research classification. Geneva: World Health Organization, 1978.

WHO 1989

World Health Organization. Special report: recommendations on stroke prevention, diagnosis and therapy. Stroke 1989;20:1407-31.

WHO 2001

World Health Organization. International Classification of Function. Geneva: World Health Organization, 2001.

Zelen 1974

Zelen M. The randomization and stratification of patients to clinical trials. *Journal of Chronic Diseases* 1974;27:365-75.

Methods	A randomised, single blind, controlled trial design. Patients were randomised using sealed envelopes prepared from random number tables. Patients were pre-stratified on institution type, time since stroke and apraxia score and a Zelen correction was used to prevent unequal distribution.					
Participants	Netherlands 113 left stroke Exptl n=56, cntrl=57 Mean age: exptl 68, cntrl 63 Sex (male/female): exptl 29/27, cntrl 35/22 Inclusion criteria: left hemisphere stroke, apraxia, staying on an inpatient unit (15 rehabilitation centres and 35 nursing homes) Exclusion: history of apraxia, stroke has occurred less than 4 weeks or more than 2 years ago, age younger than 25 years and older than 95 years, history of traumatic brain damage, brain tumour, psychiatric history Professional assessing apraxia at onset was a trained researcher following screening by the medical team					
Interventions	Strategy training (integrated into usual occupational therapy) compared to occupational therapy Strategy training: teaching the patient internal/external compensatory approaches to assist ADL per- formance Intervention period 8 weeks Intervention was delivered by occupational therapists The intervention was defined in enough detail in a further study (see Van Heugten 1999)					
Outcomes	Outcomes were measured at baseline, 8 weeks and 5 months Outcomes collected: ADL measures (Van Heugten measure of ADL, Barthel ADL Index, ADL judgement list filled in independently by the OT and patient) Apraxia, motor functioning (Motricity Index, functional motor test), additional tests (verbal compre- hension, memory, neglect, mental status) Assessment was made by a blinded research assistant					
Notes	Allocation by random number table Blocks of size 2 plus Zelen correction could make allocation predictable					

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Donkervoort 2001 (Continued)

Bias	Authors' judgement	Support for judgement
Allocation concealment?	Low risk	A - Adequate

Methods	Randomised, single blinded controlled trial. Used a randomisation scheme using pre-prepared en- velopes from random number tables. Edmans informed the review authors that the recruiter prepared the allocations prior to the study. Allocations were stored in sealed, opaque, numbered envelopes, only opened at the time of recruitment in the presence of a witness							
Participants	the functional approach hemisphere strokes Inclusion criteria: all ag able to give consent	Unit egic participants in trial, data from 9 apraxics were abstracted. 6 apraxics in n (mean age 70) and 3 in the transfer of training approach (mean age 69). All left es, able to complete the RPAB, functional use in one hand, patient or family I for the apraxia at the outset						
Interventions	Comparison of the transfer of training and functional treatment approaches Transfer of training: practising one perceptual task will affect the performance on other perceptual tasks, i.e. the cause of the perceptual problem is treated The functional approach: repetitive practice of specific daily living tasks. Intervention given for 2.5 hours per week for 6 weeks in additional to general OT OTs delivered the interventions							
Outcomes	post intervention Other routine assessme Outcomes were assesse	ans ADL Index and RPAB assessments were completed before and immediately ents were also collated, e.g. the apraxia test by Kertesz and Ferro ed by nurses and an independent, blinded OT published by a later article						
Notes	ria included: medically	the stroke unit were participating in an evaluation study, the selection crite- stable, transfer with 2 nurses, no discharge date, able to tolerate 30 minutes of olete 2 out of 4 specified functional tasks						
Risk of bias								
Bias	Authors' judgement	Support for judgement						
Allocation concealment?	High risk	C - Inadequate						

Smania 2000

Methods	Randomised, controlled trial First 10 patients assigned to exptl/cntrl group Following 3 used a restricted randomisation scheme placed in cntrl group; the last 3 were not included in this review
Participants	Italy Neurological Rehabilitation Unit 10 strokes accepted into the review: exptl 6, cntrl 4 Mean age: exptl 69.3 years, cntrl 63 years Sex (male/female): exptl 5/1, cntrl 3/1 Duration of stroke: exptl mean 14.7 months, cntrl mean 18 months

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Smania 2000 (Continued)	Neurologic severity (range 0 to 18): exptl mean 6.5, cntrl mean 7.5 Inclusion criteria: limb apraxia, length of illness at least 2 months, right handed, left hemisphere stroke Exclusion: history of cerebrovascular attacks or psychiatric disorders Professional assessing eligibility was not clear							
Interventions	each lasting 50 minute							
Outcomes	A battery of tests including an oral apraxia test, a constructional apraxia test and 3 limb praxic function tests. No tests regarding ADL were carried out Professional assessing outcome was not clear The intervention was clear enough to replicate							
Notes	Only the first 10 assigned have been included in the study as they were truly randomised Large difference in stroke duration between exptl and cntrl groups							
Risk of bias								
Bias	Authors' judgement	Support for judgement						
Allocation concealment?	Unclear risk	B - Unclear						

ADL: activities of daily living cntrl: control exptl: experimental OT: occupational therapy/therapist RPAB: Rivermead Perceptual Assessment Battery

DATA AND ANALYSES

Comparison 1. Experimental therapy versus standard care

Outcome or subgroup title	No. of studies	No. of partici- pants	•	
1 Change in Barthel at six months after end of therapy	1	83	Mean Difference (IV, Fixed, 95% CI)	0.17 [-1.41, 1.75]
1.1 Strategy training	1	83	Mean Difference (IV, Fixed, 95% CI)	0.17 [-1.41, 1.75]
2 Change in Barthel at end of therapy	2	102	Mean Difference (IV, Fixed, 95% CI)	1.28 [0.19, 2.38]
2.1 Strategy training	1	93	Mean Difference (IV, Fixed, 95% CI)	1.29 [0.16, 2.42]
2.2 Transfer of training	1	9	Mean Difference (IV, Fixed, 95% CI)	1.20 [-3.20, 5.60]
3 Change in Barthel at 12 months after end of therapy	0	0	Mean Difference (IV, Fixed, 95% CI)	0.0 [0.0, 0.0]

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Cochrane Database of Systematic Reviews

Outcome or subgroup title	No. of studies	No. of partici- pants	-	
4 Death	3	132	Peto Odds Ratio (Peto, Fixed, 95% CI)	0.41 [0.09, 1.89]
4.1 Strategy training	1	113	Peto Odds Ratio (Peto, Fixed, 95% CI)	0.41 [0.09, 1.89]
4.2 Transfer of training	1	9	Peto Odds Ratio (Peto, Fixed, 95% CI)	0.0 [0.0, 0.0]
4.3 Gesture training	1	10	Peto Odds Ratio (Peto, Fixed, 95% Cl)	0.0 [0.0, 0.0]
5 Quality of life measures	0	0	Mean Difference (IV, Fixed, 95% CI)	0.0 [0.0, 0.0]
6 Ability to gesture, pan- tomime, use real objects	1		Mean Difference (IV, Fixed, 95% CI)	Subtotals only
6.1 Gesture training	1	10	Mean Difference (IV, Fixed, 95% CI)	8.40 [-15.84, 32.64]
6.2 Using real objects	1	10	Mean Difference (IV, Fixed, 95% CI)	1.20 [-3.22, 5.62]
7 Effects on family and carer	0	0	Mean Difference (IV, Fixed, 95% CI)	0.0 [0.0, 0.0]
8 Carer and family percep- tions	0	0	Mean Difference (IV, Fixed, 95% CI)	0.0 [0.0, 0.0]
9 Economic resources	0	0	Mean Difference (IV, Fixed, 95% CI)	0.0 [0.0, 0.0]
10 Apraxic patient's mood	0	0	Mean Difference (IV, Fixed, 95% CI)	0.0 [0.0, 0.0]
11 Adverse events	0	0	Odds Ratio (M-H, Fixed, 95% CI)	0.0 [0.0, 0.0]

Analysis 1.1. Comparison 1 Experimental therapy versus standard care, Outcome 1 Change in Barthel at six months after end of therapy.

Study or subgroup	Experimental		Control		l Control		Me	an Difference	Weight	Mean Difference
	Ν	Mean(SD)	Ν	Mean(SD)	F	ixed, 95% CI		Fixed, 95% CI		
1.1.1 Strategy training										
Donkervoort 2001	43	3 (4.1)	40	2.8 (3.2)	-		100%	0.17[-1.41,1.75]		
Subtotal ***	43		40		-		100%	0.17[-1.41,1.75]		
Heterogeneity: Not applicable										
Test for overall effect: Z=0.21(P=0.83)										
Total ***	43		40		-		100%	0.17[-1.41,1.75]		
Heterogeneity: Not applicable										
Test for overall effect: Z=0.21(P=0.83)										
			Fa	vours control -4	-2	0 2	⁴ Favours exp	perimental		



Analysis 1.2. Comparison 1 Experimental therapy versus standard care, Outcome 2 Change in Barthel at end of therapy.

Exp	erimental	c	Control	Mean Difference	Weight	Mean Difference
Ν	Mean(SD)	Ν	Mean(SD)	Fixed, 95% CI		Fixed, 95% CI
45	2.4 (3)	48	1.2 (2.5)		93.85%	1.29[0.16,2.42]
45		48		•	93.85%	1.29[0.16,2.42]
)						
3	4 (3)	6	2.8 (3.5)		6.15%	1.2[-3.2,5.6]
3		6			6.15%	1.2[-3.2,5.6]
)						
48		54		•	100%	1.28[0.19,2.38]
P=0.97);	l ² =0%					
)						
), df=1 (P	=0.97), l ² =0%					
) (()	N 45 45 2) 3 3 3 (P=0.97); 2)	$45 2.4 (3) 45 2.2 (3) 45 22) 3 4 (3) 3 3 3 3 3) 48 (P=0.97); ^2=0\%$	N Mean(SD) N 45 2.4 (3) 48 45 48 48 2) 3 4 (3) 6 3 4 (3) 6 6 3 6 6 6 3) 54 (P=0.97); 1 ² =0% 54	N Mean(SD) N Mean(SD) 45 2.4 (3) 48 1.2 (2.5) 45 48 48 1.2 (2.5) 3 4 (3) 6 2.8 (3.5) 3 6 2.8 (3.5) 6 3) 6 54 (P=0.97); 1 ² =0% 2 2	N Mean(SD) N Mean(SD) Fixed, 95% CI 45 2.4 (3) 48 1.2 (2.5) \bullet 45 48 \bullet \bullet 2) 3 4 (3) 6 2.8 (3.5) 3 6 \bullet \bullet a) 48 54 \bullet (P=0.97); 1 ² =0% \bullet \bullet	N Mean(SD) N Mean(SD) Fixed, 95% CI 45 2.4 (3) 48 1.2 (2.5) 93.85% 45 48 93.85% 93.85% 2) 3 4 (3) 6 2.8 (3.5) 6 3 6 2.8 (3.5) 6 6.15% 3 6 - 100% (P=0.97); 1 ² =0% - 100%

Analysis 1.4. Comparison 1 Experimental therapy versus standard care, Outcome 4 Death.

Study or subgroup	Experimental n/N	Control n/N	Peto Odds Ratio Peto, Fixed, 95% Cl	Weight	Peto Odds Ratio Peto, Fixed, 95% Cl
1.4.1 Strategy training					
Donkervoort 2001	2/56	5/57	←	100%	0.41[0.09,1.89]
Subtotal (95% CI)	56	57		100%	0.41[0.09,1.89]
Total events: 2 (Experimental), 5 (C	ontrol)				
Heterogeneity: Not applicable					
Test for overall effect: Z=1.14(P=0.2	:5)				
1.4.2 Transfer of training					
Edmans 2000	0/3	0/6			Not estimable
Subtotal (95% CI)	3	6			Not estimable
Total events: 0 (Experimental), 0 (C	ontrol)				
Heterogeneity: Not applicable					
Test for overall effect: Not applicab	le				
1.4.3 Gesture training					
Smania 2000	0/6	0/4			Not estimable
Subtotal (95% CI)	6	4			Not estimable
Total events: 0 (Experimental), 0 (C	ontrol)				
Heterogeneity: Not applicable					
Test for overall effect: Not applicab	le				
Total (95% CI)	65	67		100%	0.41[0.09,1.89]
Total events: 2 (Experimental), 5 (C	ontrol)				
Heterogeneity: Not applicable					
Test for overall effect: Z=1.14(P=0.2	.5)				
	Favo	urs experimental	0.1 0.2 0.5 1 2 5	¹⁰ Favours treatment	

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Study or subgroup	Experimental Control n/N n/N		Peto Odds Ratio Peto, Fixed, 95% Cl							Weight	Peto Odds Ratio Peto, Fixed, 95% Cl
Test for subgroup differences:	Not applicable										
		Favours experimental	0.1	0.2	0.5	1	2	5	10	Favours treatment	

Analysis 1.6. Comparison 1 Experimental therapy versus standard care, Outcome 6 Ability to gesture, pantomime, use real objects.

Study or subgroup	Expe	erimental	c	ontrol		Mea	n Difference	Weight	Mean Difference
	N	Mean(SD)	Ν	Mean(SD)		Fiz	xed, 95% CI		Fixed, 95% CI
1.6.1 Gesture training									
Smania 2000	6	37.7 (16.2)	4	29.3 (20.9)	-			100%	8.4[-15.84,32.64]
Subtotal ***	6		4					100%	8.4[-15.84,32.64]
Heterogeneity: Not applicable									
Test for overall effect: Z=0.68(P=0.5)									
1.6.2 Using real objects									
Smania 2000	6	11.7 (2.3)	4	10.5 (4.1)				100%	1.2[-3.22,5.62]
Subtotal ***	6		4			-		100%	1.2[-3.22,5.62]
Heterogeneity: Not applicable									
Test for overall effect: Z=0.53(P=0.59)									
Test for subgroup differences: Chi ² =0	.33, df=1	. (P=0.57), I ² =0%							
			Fa	vours control	-10	-5	0 5	¹⁰ Favours exp	perimental

APPENDICES

Appendix 1. MEDLINE search strategy

The search strategy for MEDLINE is given below and this was modified for the other databases.

MEDLINE (Ovid) 1966 to November 2006

1 exp cerebrovascular disorders/

- 2 (stroke\$ or poststroke\$ or cva\$).tw.
- 3 (cerebrovascular\$ or cerebral vascular).tw.
- 4 (cerebral or cerebellar or brainstem or vertebrobasilar).tw.
- 5 (infarct\$ or isch?emi\$ or thrombo\$ or apoplexy or emboli\$).tw.
- 6 4 and 5
- 7 (cerebral or intracerebral or intracranial or parenchymal).tw.
- 8 (brain or intraventricular or brainstem or cerebellar).tw.
- 9 (infratentorial or supratentorial or subarachnoid).tw.
- 10 7 or 8 or 9
- 11 (haemorrhage or hemorrhage or haematoma or hematoma).tw.
- 12 (bleeding or aneurysm).tw.
- 13 11 or 12
- 14 10 and 13
- $15\,1\,or\,2\,or\,3\,or\,6\,or\,14$
- 16 exp apraxias/
- 17 psychomotor disorders/
- 18 psychomotor performance/
- 19 motor skills/
- 20 task performance and analysis/
- 21 cognition disorders/

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22 (aprax\$ or dysprax\$ or prax\$ or practic).tw 23 (psychomotor adj3 (disorder\$ or performance)).tw. 24 16 or 17 or 18 or 19 or 20 or 21 or 22 or 23 25 randomized controlled trial.pt. 26 randomized controlled trials/ 27 controlled clinical trial.pt. 28 controlled clinical trials/ 29 random allocation/ 30 double-blind method/ 31 single-blind method/ 32 clinical trial.pt. 33 exp clinical trials/ 34 (clin\$ adj25 trial\$).tw. 35 ((singl\$ or doubl\$ or tripl\$ or trebl\$) adj25 (blind\$ or mask\$)).tw. 36 placebos/ 37 placebo\$.tw. 38 random\$.tw. 39 research design/ 40 intervention studies/ 41 cross-over studies/ 42 alternate treatment.tw. 43 latin square.tw. 44 "comparative study"/ 45 exp evaluation studies/ 46 Follow-up studies/ 47 Prospective studies/ 48 prospective.tw. 49 counterbalance\$.tw. 50 (versus or sham).tw. 51 (controls or controlled).tw. 52 or/25-51 53 15 and 24 and 52 54 limit 53 to human

WHAT'S NEW

Date	Event	Description
26 August 2008	Amended	Converted to new review format.

CONTRIBUTIONS OF AUTHORS

Carolyn West, Audrey Bowen and Andy Vail obtained funding for the production of this review from the North West Region NHS Executive under their Research Development Fund scheme. Carolyn West wrote the protocol and review with the assistance of Andy Vail, Audrey Bowen and Anne Hesketh. Carolyn West is an occupational therapist, Audrey Bowen is a psychologist, Andy Vail is a medical statistician and Anne Hesketh is a speech and language therapist.

DECLARATIONS OF INTEREST

None known

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• No sources of support supplied

External sources

• North West NHS R&D Executive, UK.



INDEX TERMS

Medical Subject Headings (MeSH)

*Stroke Rehabilitation; Activities of Daily Living; Apraxias [etiology] [*rehabilitation]; Randomized Controlled Trials as Topic; Recovery of Function; Stroke [complications]

MeSH check words

Humans