The effectiveness of EMG biofeedback in the treatment of arm function after stroke

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Summary The study was designed to evaluate the effect of electromyographic (EMG) biofeedback on the recovery of arm function after stroke. Patients who had impaired arm function and were between 2 and 8 weeks after stroke were randomly allocated to receive either treatment incorporating EMG biofeedback or a control treatment in addition to their routine physiotherapy. The two groups of 20 patients were compared before and after 6 weeks of treatment and at follow-up 6 weeks later. There were no significant differences between the groups before treatment or at follow-up, but at the end of treatment those who received EMG biofeedback scored significantly higher on tests of arm function. Patients with severe impairment were shown to benefit most from EMG biofeedback but there was no difference in response to treatment according to patient's age or side of stroke. Men had higher arm function scores than women before and at the end of treatment, but not at follow-up.

Introduction

Electromyographic (EMG) biofeedback is a procedure used in the rehabilitation of stroke patients. Although relatively well established in other countries, it has only recently been introduced in the UK and has been little evaluated. In this technique patients are given information about physiological processes of which they are not aware, such as muscle activity. This information is used as a basis for learning voluntary control of the physiological response. Reviews of published studies on EMG biofeedback have reported that results of EMG biofeedback seem promising but many studies had unsatisfactory research designs, small samples, and few used random allocation to treatment groups.[1–4] Many of the controlled trials involved chronic stroke patients. Burnside *et al.* demonstrated a significant difference between EMG biofeedback and simple exercise in improving dorsiflexion or 'foot drop' in stroke patients.[5] The patients studied were all relatively late after the onset of their stroke, between 6 months and 12 years. However, physiotherapy is rarely available at this stage and so the results are not of direct relevance to clinical practice.

Studies of EMG biofeedback earlier in the rehabilitation process have generally failed to show significant differences between EMG biofeedback and conventional physiotherapy. Prevo *et al.* compared nine patients assigned to receive EMG biofeedback with nine who were assigned to conventional physical therapy.[6] Although differences were demonstrated between the groups in specific muscle activity, there were no differences in function in the arm or hand. However, as de Weerdt and Harrison point out, the treatment strategy used was unrepresentative of clinical practice.[4] Basmajian *et al.* have compared an integrated behavioural approach incorporating EMG biofeedback with a traditional physical therapy programme based on Bobath therapeutic exercises in stroke patients.[7,8] Overall there was worthwhile clinical and statistical improvement in tests of arm function but there was no statistically significant superiority of one therapy over the other. Similar results were obtained by John comparing outcome of EMG biofeedback integrated with physiotherapy with physiotherapy alone in a small sample of patients using a cross-over design.[9]

The present study is designed to evaluate the effect of EMG biofeedback on the recovery of arm function after acute stroke. Although this has disadvantages in that early after a stroke recovery of function is maximal and therefore detecting any additional benefit of a new treatment is more difficult, it is more appropriate for clinical practice since most patients only receive physiotherapy in the early stages of recovery. EMG biofeedback was not considered to be an appropriate treatment on its own but a technique to be incorporated into routine physiotherapy practice. It was therefore evaluated in this context. The placebo effect of new equipment was likely to be significant and therefore all patients were exposed to the EMG equipment but in only the experimental treatment group was the full potential of the biofeedback facility used.

Method

Design

The design was a single, blind, randomized, controlled trial.

Procedure

All stroke patients admitted to hospital in the Nottingham Health District and receiving routine physiotherapy were considered for inclusion. The senior physiotherapists at each hospital were contacted regularly for advice on patients who were medically stable and fit to cope with regular treatment. Patients were included if they:

1. were between 2 and 8 weeks after stroke;
2. had some arm function, i.e. at least a flicker of activity around the shoulder girdle;
3. had not already spontaneously recovered or did not have near normal movement;
4. were living in the Nottingham area; and
5. did not have global aphasia or dementia, as they needed to understand the instructions and concentrate in order to gain full benefit from the biofeedback facilities.

Assessment

A physiotherapist recorded demographic details including age, sex, marital status, housing, date of onset, date of admission, number of previous strokes, and any associated medical conditions interfering with arm function. A

standardized neurological examination based on the assessment by Bickerstaff[10] was also carried out. This covered general mobility, speech, visual field loss, cranial nerve involvement, reflexes, involuntary movements, associated reactions, and tests for co-ordination of the arm. The physical examination included active and passive movements of the arm, resistance to passive stretch, and any complications of pain, oedema, subluxation of the shoulder joint, and contractures throughout the arm and hand. A full sensory assessment was performed on both arms. This included kinaesthetic and tactile modalities of sensation and tests for inattention.

Patients were also assessed by an independent assessor (F.M.N.) on three occasions: at the beginning of the study, at 6 weeks and 12 weeks later. She did not know to which group patients had been allocated. She administered two outcome measures for the trial. These were the Action Research Armtest (ARA)[11] to demonstrate any change in overall functional recovery, and the arm recovery section of the Brunnstrom–Fugl Meyer test (BFM)[12] to indicate changes in recovery of ability.

The ARA consists of four subscales: grasp, grip, pinch, and gross movement. Each subscale measures different and specific aspects of upper limb function. These have good inter-rater and test–retest reliabilities and correlate with empirical assessments of activities of daily living.[11] The BFM is an objective and standardized test of motor performance and includes a section on the arm. This is based on the observation that there are different stages in the recovery of the arm. The shoulder, elbow and forearm were tested in five progressive stages of recovery. The wrist and hand were tested separately as they are considered to recover independently of the rest of the arm. Although this test takes longer to administer it provides more detail on how and where the recovery occurs.

The patients were stratified into one of four groups, based on their initial score on the BFM test. Previous studies using the arm section of the BFM test indicated that these divisions used would create four equal-sized groups.[13] The four groups were as follows.

Group I:	initial score 0–11
Group II:	initial score 12–22
Group III:	initial score 23–32
Group IV:	initial score 33 and over

The stratification was to prevent any bias occurring from differences in the initial level of the arm recovery as this affects the eventual outcome.[14] The patients were stratified and then randomly allocated within each treatment group. This was to ensure a balance of allocation for each group. The random numbers were put into sealed and numbered envelopes for each stratification group. Even numbers indicated the control treatment group and odd numbers were used to indicate the experimental treatment group.

Treatment

The equipment used was the Biodata EMG-120 biofeedback system. For both treatment groups the equipment was positioned near the patient to allow for the placebo effect of new equipment. The machine was plugged into the mains and the patients were connected to the machine by the electrodes.

For the control treatment group there was no skin or electrode preparation. The siting of the electrodes was for convenience either over the upper fibres of the trapezius, pectoral or deltoid muscles depending on the area of treatment for that session. The system was switched on, but the auditory feedback was switched off and the main voltmeter for the visual feedback not visible to the patient. The external voltmeter was not used.

For the experimental treatment group the equipment was used to its fullest capacity. Silver–silver chloride Beckmann-style electrodes were used with a saline conducting gel. Taking into account that the apparatus was more sophisticated than would normally be available in a physiotherapy department it was decided that application of the technique would be maintained within the following limits.

Filters	– generally set at 100/1000 band width. – notch filter only used if necessary.
Time constant	– set at 0.1 s.
Auditory feedback	– set on click mode.
Visual feedback	– the portable voltmeter was placed within the patient's field of vision and moved as needed.
Meter ranges	– set as required to the level of muscle activity (sensitivity level on portable EMG machines).

The three main objectives of treatment for both groups were to normalize muscle tone, work for normal active movement and then aim for functional goals. The strategies of treatment for the arm and its progression were as described in Crow.[15] The experimental treatment group, whilst working within the same strategies as the control treatment group, also used the strategies for incorporating EMG biofeedback within the treatment session.

During each treatment session the patient was assessed for the main problem relating to arm recovery. For the patients in the experimental treatment group the information was then used to pick a target muscle for the rest of the treatment session. Siting of the electrodes was based on the muscle activity at that time and not a predetermined position for each muscle, as this would vary from day to day with changing conditions. If the goals for a session were achieved early in the session or if it was realized during the treatment sessions that there was a more appropriate target muscle, then the electrodes were moved accordingly. Every effort was made to standardize motivation and enthusiasm shown to each treatment group about the treatment that they received and the same two physiotherapists treated the patients in both groups so as to avoid possible treatment bias.

Patients were also seen by the physiotherapist for their ward throughout the treatment period. The patient was made aware that both physiotherapists were working towards the same goal in a similar manner so as not to confuse the patient.

Records were kept on a daily basis of the main results of the treatment session: whether the treatment was before or after the routine physiotherapy and the frequency of the routine physiotherapy. Aids and equipment available for use during the treatment sessions included a low plinth or chair, table or high plinth, pillows, wedge-shaped cushion, towels, a walking stick and a wheelchair. Small hand equipment (e.g. quoits, cones, balloons, water-filled cylindrical bottle, a knife, fork and spoon) were also available.

Results

Subjects

There were 42 patients who entered the trial, of whom 39 completed the full 12 weeks. Three patients died during the trial period and two died during the treatment period, and have therefore been excluded from the data

analysis. The third patient died during the 6-week follow-up period and had completed two of the three assessments and has therefore been included in the data analysis. All analyses are from the 40 patients who completed the treatment period of the trial. The patients were treated by two physiotherapists—physiotherapist A (J.L.C.) treated eight patients in the treatment group and 12 controls, physiotherapist B (W.D.W.) treated 12 patients in the treatment group and eight controls. The patient details are shown in Table 1.

The stratification of the 20 patients in the experimental treatment group was: Group I = 9, Group II = 1, Group III = 5, Group IV = 5. The 20 patients in the control treatment group divided into the stratification groups as follows: Group I = 12, Group II = 2, Group III = 3, Group IV = 3.

Comparison between treatment groups

Before treatment, there was no statistically significant difference between the treatment groups on either the ARA test ($p = 0.20$) or the BFM test ($p = 0.06$) (Mann-Whitney U-test one-tailed). After 6 weeks of treatment, the difference between the groups was statistically significant for both outcome measures (ARA $p = 0.05$, BFM $p = 0.02$). Following a further 6 weeks without the additional treatment there were no statistically significant differences between the groups on either test (ARA $p = 0.16$, BFM $p = 0.09$). Results are shown in Table 2 and displayed graphically in Figure 1.

It can be seen from Figure 1 that at the first assessment point the two treatment groups started at different levels on both outcome measures, despite the randomization. There was no statistically significant difference between the groups at that point. To substantiate the significant difference between groups after the treatment period, the changes in scores were analysed. The difference scores were compared and found to be statistically significant for the ARA test ($p = 0.04$), but not statistically significant for the BFM test ($p = 0.09$). Results are shown in Table 3.

Comparison of stratified subgroups

As the number of patients in the stratification subgroup I was approximately half of the total for both treatment groups and the numbers in subgroups II, III and IV were too small for statistical analysis, two subgroups were created—those with lower initial scores (stratification group = I) and those with higher initial scores (stratification = II, III and IV).

The results of each assessment were compared. In the severe group (stratification I) there were no significant differences between the treatment groups before treatment but there was a statistically significant difference at the end of the treatment period on both measures. At follow-up there was a significant difference between the groups on the ARA test but not the BFM. In the moderate group (stratification II, III and IV) there were no statistically significant differences between the treatment groups. Results are shown in Table 4.

Table 1 Details of patients

		Group		
		Experimental treatment	*Control treatment*	*Comparison*
Age	Mean	67·4	68·05	$t = 0.21$
	SD	10.45	9·53	NS
	Range	43–80	53–89	
Sex	Male	14	11	$\chi^2 = 0.42$
	Female	6	9	NS
Side of stroke	Left	12	14	$\chi^2 = 0.11$
treated	Right	8	6	NS

NS = not significant $p > 0.05$.

Table 2 Comparison of arm function scores between treatment groups

Assessment	*Occasion*		*Experimental treatment*	*Control treatment*	*U*	*p*	
ARA	1	Mean	9·8	6·1			
		SD	18·8	12·4	172·0	0.20	NS
		Median	0·5	0			
		Range	0–57	0–38			
ARA	2	Mean	21·3	12·5			
		SD	22·8	21·7	143·5	0·05	*
		Median	12·5	0			
		Range	0–57	0–57			
ARA	3	Mean	20·5	15·9			
		SD	21·9	22·9	157.0	0.16	NS
		Median	15·0	0			
		Range	0–57	0–57			
BFM	1	Mean	22·4	16·0			
		SD	16·7	14·8	143·0	0·06	NS
		Median	19·5	8·0			
		Range	5–58	4–54			
BFM	2	Mean	34·5	22·9			
		SD	22·0	21·1	124·5	0·02	*
		Median	37·0	13·5			
		Range	6–66	4–65			
BFM	3	Mean	35·2	27·0			
		SD	20·6	21·2	142·0	0·09	NS
		Median	31·0	17·0			
		Range	7–66	4–66			

ARA = Action Research Arm test.
BFM = Brunnstrom–Fugl Meyer test.
U = Mann-Whitney U-test.
*$p < 0.05$ (one-tailed).
NS $p > 0.05$ (one-tailed).

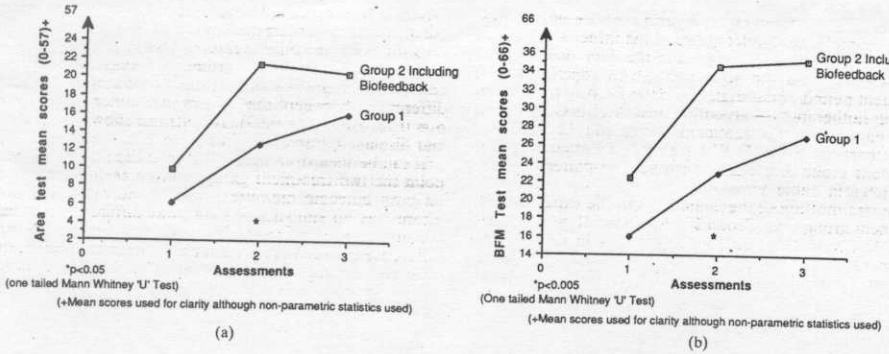

Figure 1 Comparison of treatment groups on (a) the ARA and (b) the BFM test.

Table 3 Comparison between treatment groups on change in arm function

Assessment	*Period*		*Experimental treatment*	*Control treatment*	*U*	*p*	
ARA	Treatment (Test 2–Test 1)	Mean	11·40	6·40			
		SD	16·12	12·54	138·5	0·04	*
		Median	6·50	0			
		Range	0–55·0	−4·0–44·0			
	Follow-up (Test 3–Test 2)	Mean	1·00	3·35			
		SD	8·19	8·07	184·0	0·42	NS
		Median	0	0			
		Range	−21·0–20·0	0–33·0			
BFM	Treatment (Test 2–Test 1)	Mean	12·10	6·90			
		SD	12·53	10·67	149·5	0·09	NS
		Median	10·00	3·00			
		Range	0–38·0	−2·0–42·0			
	Follow-up (Test 3–Test 2)	Mean	2·16	4·10			
		SD	5·93	5·12	157·0	0·15	NS
		Median	2·00	3·00			
		Range	−9·0–14·0	0–18·0			

ARA = Action Research Arm test.
BFM = Brunnstrom–Fugl Meyer test.
U = Mann-Whitney U-test.
p = probability (one-tailed).
* $p < 0{\cdot}05$.
NS $p > 0{\cdot}05$.

Effect of patient characteristics on outcome

The demographic details of age, sex and side of stroke treated were examined to identify patients who benefitted most from the EMG biofeedback treatment. The patients were divided into two age groups. The younger group was 65 years and under, whilst the older group was 66 years and over.

The results for the three demographic variables considered are shown in Table 5. There were no significant differences between age groups or side of stroke. However, statistically significant differences were found between the sexes. On the first two assessments men performed to a level significantly higher than the women on the BFM test.

Discussion

The results of the study indicated that EMG biofeedback facilitated the recovery of arm function during the period in which the treatment was given but the beneficial effects did not persist once treatment ended. One possible explanation is that the patients in the experimental treatment group reached a plateau in their recovery and were in a phase of consolidation of the learning process during the follow-up period. Bach-y-Rita described the recovery process in stages, which included plateaus or periods when no specific improvement occurs.[16] These periods allow patients time to consolidate the information they have just acquired before they move on to the next recovery stage. This is equivalent to the fixation phase of skill acquisition described by Fitts[17] and Gentile.[18] It is possible that, during the short follow-up period, patients in the experimental treatment group were consolidating the information they had just learnt.

In contrast the control treatment group improved at a slower rate and did not reach a plateau in their recovery until a later time. It may be that they had not reached the fixation phase of learning during the treatment period. Further evaluations with several follow-up assessments over a longer time would help clarify this possibility.

The lack of persistence of the difference between the two treatment groups may also be due to the short treatment period. Patients received 18 sessions treatment over a 6-week period. For the transfer of training to be fully effective, the training has to be of sufficient duration for skills acquired in the clinical setting to generalize to

Table 4 Comparison between treatment groups for stratified subgroups (one-tailed) ($n = 40$)

			Stratification Group I				*Stratification Groups II to IV*			
			Experimental treatment (n = 9)	*Control treatment (n = 12)*	*U*	*p*	*Experimental treatment (n = 11)*	*Control treatment (n = 8)*	*U*	*p*
ARA	1	Mean	0·00	0·08			17·91	15·13		
		SD	0·00	0·29	49·5	0·19	22·73	16·09	43·5	0·48
		Median	0	0			5·00	7·50		
		Range	0	0–1			0–57	0–38		
	2	Mean	3·22	0·00			36·00	31·25		
		SD	5·43	0·00	36·0	0·02*	20·80	24·80	39·0	0·34
		Median	0	0			44·00	29·50		
		Range	0–13	0			0–57	0–57		
	3	Mean	4·89	0			34·70	39·63		
		SD	9·97	0·00	36·0	0·02*	20·26	18·68	33·0	0·26
		Median	0	0·00			42·50	43·00		
		Range	0–28	0			0–57	10–57		
BFM	1	Mean	7·67	6·42			34·55	30·50		
		SD	1·94	1·73	32·5	0·06	12·99	13·88	38·5	0·32
		Median	8·00	6·00			32·00	30·50		
		Range	5–10	4–9			16–58	13–54		
	2	Mean	16·11	8·17			49·64	45·13		
		SD	12·58	3·93	24·0	0·02*	15·51	15·62	34·5	0·22
		Median	10·00	6·00			58·00	45·50		
		Range	6–44	4–16			17–66	17–65		
	3	Mean	19·89	11·67			49·10	50·13		
		SD	13·86	6·29	33·5	0·07	15·26	12·12	39·5	0·48
		Median	11·00	11·00			52·50	53·0		
		Range	7–48	4–25			21–66	29–66		

ARA = Action Research Arm test.
BFM = Brunnstrom–Fugl Meyer test.
U = Mann-Whitney *U*-test.
*significant at $p < 0·05$ (one-tailed).

Table 5 Comparison of patients according to demographic variables

Assessment	*Occasion*	*Young versus old (n=23) (n=17) p*	*Male versus female (n=25) (n=15) p*	*Right versus left (n=14) (n=26) p*
ARA	1	0·47	0·09	0·68
	2	0·77	0·12	0·48
	3	0·80	0·13	0·53
BFM	1	0·74	0.02*	0·75
	2	0·60	0·03*	0·57
	3	0·48	0·13	0·48

ARA = Action Research Arm test.
BFM = Brunnstrom–Fugl Meyer test.
U = Mann-Whitney *U*-test (two-tailed).
*significant $p < 0·05$.

motor and functional activities in other environments. Extending the treatment period may increase the effectiveness of the procedure. In previous studies that have shown beneficial effects of EMG biofeedback[5,6] the treatment periods have been comparable to ours.

There were minor discrepancies between the results on the ARA and BFM tests. This would suggest that the experimental treatment had greater effect on functional activities than on motor ability. It may be that the role of the EMG biofeedback was to facilitate the transfer of skills from the trained activity to non-trained tasks.

Investigation of the effects of sex, age and side of stroke in the response to treatment indicated that these were not important determinants of recovery. However, severity of initial motor function did seem important. Patients who had low scores on the BFM test initially showed more benefit from the EMG biofeedback. These severely impaired patients require most information to even attempt a movement; therefore, by supplying additional accurate information about the movement the patient is able to learn the movement.

These results therefore indicate that EMG biofeedback has more potential as a component of physiotherapy than some previous studies. This may partly be a result of considering a larger sample of patients. The studies reported in the introduction all consisted of smaller patients groups and this may account for the lack of difference between groups studied by Basmajian *et al.*[7,8] and John[9] with similar patients to those in the present study.

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Book review

Detested sport that owes its pleasure to another's pain

Soft Tissue Injuries in Sport. Sylvia Lachmann. Blackwell Scientific Publications, Oxford, 1988, 144 pp., 80 illus., £16.95. ISBN 0–632–01964–6

This book is written by a consultant in rehabilitation at Addenbrookes Hospital, Cambridge, and is designed for medical staff regularly involved with sports injuries.

Part I offers an understanding of the mechanisms involved in soft-tissue injury, repair and the factors that influence them. Descriptions are clear and backed up by line drawings. There is also a useful chapter which explains some of the methods and 'magic rays' so beloved by physiotherapists and so bewildering to so many doctors!

Part II deals with the diagnosis and management of specific soft-tissue injuries, region by region. Concise descriptions of the injuries are supplemented by clear line drawings. The latter remind the reader of the appropriate anatomy, long banished to the recesses of an overtaxed medical mind. Some of the diagnostic methods used, however, are unclear and more specific advice about rehabilitative exercises and training for individual injuries would have been welcomed. The inclusion of local anaesthetic/steroid techniques would also have been useful (perhaps in the form of an appendix).

The book offers a clear guide to the mechanics of soft-tissue repair. The descriptions of various injuries provide a concise guide to those wishing an introduction to sports injuries (e.g. GPs, medical students and physiotherapists). However, the oversimplification of some of the diagnostic and management methods may limit the usefulness of the book to those regularly involved with the management of sports injuries.

KEVIN GRUFFYDD-JONES