

Rehabilitation of hemiparesis after stroke with a mirror

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With an ageing population in an industrialised world, the global burden of stroke is staggering (millions of strokes a year). Hemiparesis is one of the most common and disabling sequelae of stroke. In patients with immobile phantom limbs after amputation, vivid kinaesthetic sensations of movement can be evoked when the patient watches movement of the non-amputated hand or arm in a vertical parasagittal mirror.¹ Encouraged by a good initial result,² we have now done a larger trial of mirror therapy on patients with hemiparesis following stroke.

All patients were at least 6 months post-stroke proven by computed tomography or magnetic resonance imaging (mean 4.8 years post-stroke, SD 8.2 years, range 6 months to 26.25 years), to preclude effects from spontaneous recovery. The patients gave written informed consent. The patients were randomly assigned to spend the first 4 weeks using a mirror or transparent plastic, and then crossed over to the other treatment for the next 4 weeks. We used mirrors sized 18×24 inches (45 cm×60 cm) made of plastic with a mirror coating, and transparent plastic sheets of the same size. Patients were put on a practice schedule of 15 min, twice a day, 6 days a week, moving both hands or arms symmetrically (moving the affected arm as best they could) while watching the good arm in the mirror, or the paretic arm through the clear plastic (figure). A "bootstrapping" approach was employed in designing regimens for patients, typically moving proximal to distal, working from movements patients could perform to those they could not. The patients were videotaped at 0, 2, 4, 6, and 8 weeks making all of the cardinal movements of the upper limb. Subjective comments were obtained from the patients, and the patients' progress was assessed from the videotape by two senior neurologists from our team who were unaware of which treatment the patients used first, or the patients' subjective comments, assessing the change from baseline in patients' movement ability in terms of range of motion, speed, and accuracy using a -3 to +3 scale with 0 representing no change.

Subjectively, all the patients liked using a mirror more than the clear plastic and felt that the mirror was more helpful than the plastic. One patient said that while "all my



Demonstration of the technique

other methods of therapy exercise my muscles, the mirror is the only one which exercises my brain and nerves". Another said he liked using the mirror and thought it was helpful because while using the mirror "it looks like my bad arm is moving normally," even though it was not. Another patient characterised working with the mirror as "a blessing." Both graders found that substantially more patients improved on mirror than on control (7-1 and 4-1, table).

The mirror provides patients with "proper" visual input—the mirror reflection of the moving good arm looks like the affected arm moving correctly—and perhaps "substitutes" for the often decreased or absent proprioceptive input. Use of the mirror may also help recruit the premotor cortex to help with motor rehabilitation.³ The premotor cortex has a number of features suggesting it might possibly be a link from the visual image in the mirror to motor rehabilitation following stroke: non-trivial contributions to the descending corticospinal tracts; more bilateral control of movement than the motor cortex itself; and intimate connection between premotor areas and visual input.⁴ On a number of neurological and psychological levels, mirror therapy may help to reverse elements of learned disuse⁵ of the affected limb.

We are encouraged that mirror therapy may be beneficial

Patient number	Age, sex, handedness	Side of CVA, severity, time since CVA	Rehabilitation focus	First four weeks			Second four weeks		
				Treatment	G1	G2	Treatment	G1	G2
1	59, M, R	RCVA, mild, 2.1 years	Hand, wrist, elbow	C	+1	+0.5	M	+1	+1
2	55, M, R	RCVA, severe, 1 year	Shoulder, elbow	C	0	0	M	+1	+0.5
3	53, F, R	RCVA, extremely severe, 1.5 years	Shoulder	M	+0.5	0	C	0	0
4	55, M, R	RCVA, extremely severe, 6 months	Shoulder	M	+1	0	C	0	0
5	54, F, L	RAVM, severe, 26.25 years (AVM resection and CVA during pregnancy)	Shoulder, elbow	C	0	0	M	+1	0
6	60, F, R	RAVM, severe, 4.75 years	Shoulder, elbow	C	0	0	M	+1	0
7	53, F, R	RCVA, moderate-severe, 2.25 years	Shoulder, elbow	M	+0.5	0	C	0	0
8	73, M, R	RCVA, severe, 4.5 years	Shoulder, wrist	C	0	0	M	+1	+0.5
9	62, M, R	LCVA, moderate, 8 months	Shoulder, elbow, wrist	M	+1	+0.5	C	+1	+0.5

All patients had severely decreased to absent proprioception in the affected limb.

CVA=cerebrovascular accident (R=right; L=left); AVM=arteriovenous malformation; G=grader; M=mirror; C=control (transparent plastic).

Patient characteristics

for at least some patients with hemiparesis following stroke, giving impetus to do larger trials of mirror therapy.

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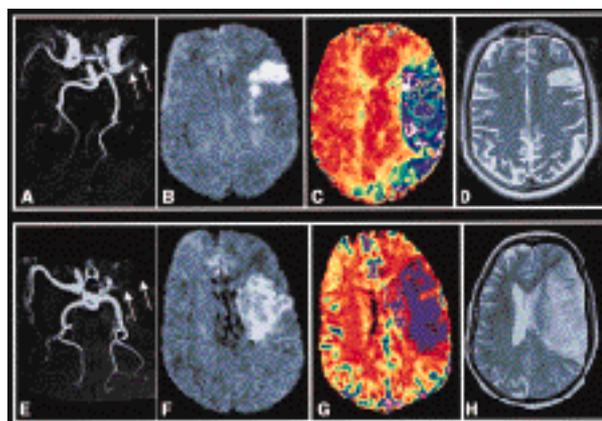
Early recanalisation in acute ischaemic stroke saves tissue at risk defined by MRI

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Acute ischaemic stroke can now be treated by early thrombolysis,^{1,2} but questions remain which patients are suitable for this therapy. Although clinical examination and computed tomography (CT) combined reveal all major contraindications for thrombolysis, they fall short of identifying the target population for early recanalisation; this subgroup of patients may be characterised by having a sizable amount of—potentially salvageable—brain tissue at risk.³ The new rapid magnetic resonance imaging (MRI) techniques of diffusion-weighted imaging (DWI) and perfusion-weighted imaging (PWI) have been shown both experimentally and in patient studies to reliably detect and define ischaemic regions of the brain.⁴ For clinical practice it is presently assumed that DWI shows the area of irreversibly injured tissue, whereas PWI shows the area of reduced cerebral perfusion.⁵ The mismatch between these areas, with the PWI abnormality being larger than the DWI abnormality, may represent the tissue at risk. The purpose of our study was to find support for this assumption by correlating early MRI findings with morphological and clinical outcome depending on early recanalisation.

In 35 patients (16 men, 19 women) aged 29–83 (mean 62) years, MRI was done within 90–690 (mean 314) min after stroke onset and at days two and five; the protocol (stroke MRI) consisted of fast DWI, PWI, T2-weighted images (T2WI), and MR angiography (MRA). Clinical data were assessed with four neurological scores (NIH stroke scale=NIHSS; Scandinavian Stroke Scale=SSS; Barthel Index=BI; modified Rankin Scale=RS) initially and at days 2, 5, 30, and 90. Nonparametrical tests were used for statistical evaluation.

21/35 patients were identified by the initial MRI to have a considerable mismatch between the abnormal area on DWI



A–D: 46-year-old patient 2 h 30 min after onset of left hemispheric symptoms

MRA (A) shows left middle cerebral artery (MCA) occlusion. DWI (B) shows a small area of ischaemically injured tissue in the left frontal lobe, whereas PWI (C) shows a disturbed perfusion in most of MCA territory. After early recanalisation T2-weighted image on day five (D) shows only a small left frontal infarct. Patient had an NIHSS of 1 at day 90.

E–H: 46-year-old patient 2 h after onset of left hemispheric symptoms

MRA (E) shows left MCA occlusion. DWI (F) shows medium-sized area of injury in left basal ganglia, whereas PWI (G) again shows a disturbed perfusion of most of MCA territory. Early thrombolysis failed in MCA recanalisation. T2WI (H) on day 5 shows an infarct that involves nearly the entire left MCA territory. Patients had an NIHSS of 7 at day 90.

and the abnormal area on PWI (PWI/DWI >1.2). In all of these patients MRA at day 1 showed occlusion of a main cerebral artery (intracranial internal carotid artery or middle cerebral artery). 14/35 patients did not have a PWI/DWI mismatch, in these patients MRA did not show a major vessel occlusion. 11/21 patients of the mismatch group received alteplase, five of them received the drug after 3 h from symptom onset. MRA at day 2 showed recanalisation in 8/21 patients with a mismatch, 6 of whom had received alteplase intravenously within 5 h after symptom onset and two had had spontaneous recanalisation; in the other 13 patients the vessel remained occluded. Of the 6 patients who received alteplase and had early recanalisation, three were treated within the first 3 h of stroke onset and three were treated between 3 h and 5 h.

Clinical follow-up examination showed for the recanalisation group a significantly better outcome on day 30 (NIHSS, $p=0.0016$; SSS, $p=0.003$; BI, $p=0.0013$; RS, $p=0.0011$), and on day 90 (NIHSS, $p=0.017$; SSS, $p=0.011$; BI, $p=0.005$; RS, $p=0.006$); there was also significantly greater improvement from day 1 to day 30 (NIHSS, $p=0.0074$). The significance regarding improvement disappeared, though, at day 90 (NIHSS, $p=0.49$), because there was one late death in this group due to cardiopulmonary embolism. Follow-up MRI showed significantly smaller infarcts in the recanalisation group than in the non-recanalisation group at day 2 as evident on DWI ($p=0.0016$) and at day 5 as evident on T2WI ($p=0.0005$). Furthermore, the increase of infarct size from day 1 to day 2 on DWI ($p=0.0055$) and from day 1 to day 5 on T2WI ($p=0.0011$) was significantly smaller in the recanalisation group than in the non-recanalisation group (table).

	NIHSS 1 NS	NIHSS 30*	NIHSS 90*	BI 90*	BI 90*	RS 30*	RS 90*	DWI 1 NS (cm ²)	T2WI 5* (cm ²)	T2WI 5- DWI 1* (CM ²)
Recanalisation (n=8)	11	3	6	80	83	1.5	1.2	30	36	6
Non-recanalisation (n=13)	14	20	18	20	35	4.7	4.1	49	192	143

NS=not significant. * $p<0.05$.

Mean stroke-scale scores and lesion volumes (on day one, five, 30 or 90) for the recanalisation and non-recanalisation group