J Rehabil Med 2011; 43: 398–403

ORIGINAL REPORT

IS ACTIVITY LOSS PREDICTIVE FOR DEVELOPMENT OF UPPER LIMB OEDEMA AFTER STROKE?

Nick Gebruers, PT[1,2], Steven Truijen, PhD, MSc[1,5], Sebastiaan Engelborghs, PhD, MD[1,2,3,4] and Peter Paul De Deyn, PhD, MD[1,2,3]

From the [1]Department of Health Care Sciences, Artesis University College of Antwerp, Merksem, [2]Department of Neurology and Memory Clinic, Middelheim General Hospital (ZNA), [3]Laboratory of Neurochemistry and Behaviour, Institute Born-Bunge, [4]Department of Nursing Sciences and [5]Department of Medicine, Faculty of Medicine, University of Antwerp, Antwerp, Belgium

Objective: **To investigate the disuse hypothesis as an explanation for upper limb oedema in patients with stroke.**
Design: **Longitudinal observational study.**
Patients: **Patients with acute hemiparetic stroke were recruited from 2006 to 2009 (*n* = 139).**
Methods: **Patients wore 2 uni-axial accelerometers, 1 on each wrist, for 2 periods of 48 h with a 1-week interval. Activity performed by the patients with acute stroke was measured by determining total activity, measured as a total sum of raw counts, and calculating the ratio variable. The National Institute of Health Stroke Scale (NIHSS), Fügl-Meyer Assessment and modified Rankin Scale were used. During a 3-month follow-up, patients underwent 3 assessments of upper limb oedema.**
Results: **The incidence of upper limb oedema range for the objective evaluation was 7.7–14.7%, while the incidence for the subjective evaluation ranged from 11.5% to 18.1%. No significant differences were found between patients with and without oedema concerning the activity variables; therefore no prognostic value could be determined.**
Conclusion: **No difference in upper limb activity was found between patients with and without oedema after stroke. It is doubtful that loss of activity of the paretic limb is solely responsible for the development of upper limb oedema after stroke.**

Key words: stroke; oedema; ambulatory monitoring.

J Rehabil Med 2011; 43: 398–403

Correspondence address: P. P. De Deyn, Laboratory of Neurochemistry and Behaviour, Institute Born-Bunge, University of Antwerp, Universiteitsplein 1, BE-2610 Antwerp, Belgium. E-mail: peter.dedeyn@ua.ac.be

Submitted July 6, 2010; accepted December 14, 2010

INTRODUCTION

Oedema of the upper limb in patients with stroke is common, but is often unrecognized. Oedema of the hand and/or arm is experienced negatively by patients and medical staff. Oedema may cause additional problems that will slow down the rehabilitation process. Heaviness, stiffness and reduced active motion are described as symptoms provoked by oedema (1–3). Oedema after stroke may be either an isolated problem or a symptom of a complex regional pain syndrome (CRPS) (1).

Early studies have found diverging incidences of oedema after stroke. In 1957 Exton-Smith & Crocket DJ (4) found an incidence of 16% among 130 patients with hemiplegia. Unfortunately, the criteria used to diagnose oedema in this study were not mentioned. In contrast, Tepperman et al. (5) found oedema in 82.8% of 85 included patients with stroke. In this study oedema was defined as the visible swelling of wrist and hand in combination with a negative bone scan. More recent studies have found incidences of 33% in a population of patients with stroke admitted to a rehabilitation centre. These studies (2, 6) used more standardized diagnostic methods. Oedema was present when the volume difference between the hands was greater than two standard deviations based on population data provided by Vasiliauskas et al. (3). Since these population data are available only for hand volumes, an additional study was performed by Gebruers et al. (7) to determine the volume difference between the arms. In this study prediction formulas were presented based on the arm volumes of 250 healthy subjects (7).

Unlike oedema after CRPS, isolated oedema of the upper limb after stroke has received little attention in the scientific literature. The aetiology of oedema in stroke patients remains unclear. In a review article, Geurts et al. (1) stated that oedema in stroke patients is not lymphoedema. The main evidence for this conclusion came from a scintigraphic study of the lymphatic system of patients with hemiplegic stroke. In 83% of the patients, lymph flow was increased in the oedematous hemiplegic arm (8), which is in contrast to the lymph flow seen in lymphoedematous limbs (9 –11). The reason for the development of oedema after stroke is therefore more likely to be a changed microcirculation with an imbalance in the filtration and reabsorption mechanism (8). Other authors stated that oedema is fluid accumulation only at the beginning and that it will progress into lymphoedema if the oedema persists (4, 12, 13). Several theories exist about the development of oedema after stroke. Vascular changes are described and could be responsible for altered filtration and reabsorption processes at the level of the microcirculation (14, 15). Another hypothesis states that oedema is a result of increased disuse due to the hemiplegia.

The disuse of the upper limb would be responsible for fluid accumulation because the muscle pump action is decreased (12, 16, 17). In this study we tested the disuse hypothesis; examining whether the diminished activity presented by patients with hemiparesis or hemiplegia after stroke is in fact responsible for development of upper limb oedema? And, if so, whether the amount of activity of the upper limb, as measured by accelerometers, can be used as a prognostic factor for patients at risk for the development of oedema following stroke? We hypothesize that the decreased activity of the upper limbs in patients with stroke contributes to the development of upper limb oedema after stroke. This study is part of the Middelheim Interdisciplinary Stroke Study (MISS). The MISS protocol was started as a research project targeting clinical, biochemical, structural neuroimaging, neuropsychological and electrophysiological characteristics of patients after stroke.

PATIENTS AND METHODS

Patients

Patients were recruited from the neurology ward of the general hospital ZNA Middelheim Antwerp from January 2006 until September 2009. Patients were eligible for inclusion in this prospective study if they met the following criteria: diagnosis of acute (< 7 days after stroke onset) ischaemic or haemorrhagic stroke (clinical examination in combination with brain computed tomography (CT) and/or magnetic resonance imaging (MRI) scan) accompanied by a motor hemi-syndrome with at least involvement of the upper extremity. A score > 0 on the National Institute of Health Stroke Scale (NIHSS) motor item of the upper extremity was used to define the presence of motor hemi-syndrome. Patients with a previous stroke with incomplete motor function recovery were excluded. Patients with upper limb oedema prior to stroke onset were also excluded. For all patients written informed consent, by proxy if applicable, was obtained. The following demographic and clinical data were recorded in the case record forms (CRF): medication use, medical and surgical history, age, gender, height, weight, body mass index, self-reported handedness, Edinburgh Handedness Inventory score, length of stay (LoS), stroke scale scores, and type and cause of stroke determined by the Trial of ORG 10172 in Acute Stroke (TOAST) (18). The study protocol was approved by the local Institutional Review Board, CME ZNA Middelheim (approval no. 2591) (Fig. 1).

Stroke characteristics

First, it was determined whether the patients had had an ischaemic or haemorrhagic stroke. Secondly, the affected hemisphere was noted. As mentioned above, the TOAST criteria were applied to describe the cause of ischaemic stroke. The TOAST criteria allow classification of stroke aetiology into 5 major subgroups; namely, large-artery atherosclerosis, cardio-embolism, small-artery occlusion (lacunar stroke), other determined aetiology and undetermined aetiology (18, 19). To score the TOAST criteria, evidence from MRI, electrocardiography (ECG), duplex ultrasonography of the carotid and vertebral arteries and clinical information was imported to a computerized algorithm for the aetiological classification of ischaemic stroke (http://ccs.martinos.org/ccs_form.shtml) (20).

Accelerometer recordings

Accelerometer recordings were performed for 48 h at inclusion (t0) and with 1 week interval (t1), in the acute phase of stroke. The AMI Octagonal Motion Logger (Ambulatory Monitoring, New York, USA) was used. This motion logger (Fig. 2) is a uni-axial accelerometer constructed as a wrist-watch. The device has a 2 MB memory, and a 2–3 Hz low-pass filter. Its sensitivity is 0.01 g. The motion logger records information from a movement-sensitive sensor and is able to provide information about 3 different modes of measurement (21–23). All data for the current study were analysed using the Proportional Integrated Mode (PIM), which presents activity as the area under the curve (24). The patients wore two motion loggers, one on each wrist. This methodology has been used by other authors (25, 26) and has the advantage that two different activity variables can be calculated; namely, the activity of the paretic arm (sum of all activity measured by the motion logger of the impaired arm) and the ratio. The ratio is calculated as the activity of the paretic arm divided by the activity of the non-paretic arm. Further details are described elsewhere (23).

Scales

Two scales (NIHSS and Fugl-Meyer Assessment (FMA)) were scored at the start of the two 48-h actigraphy recording periods and at every oedema assessment. The Edinburgh Handedness Inventory (EHI) was scored at inclusion. At the end of the 3-month follow-up period an additional clinical rating scale was scored, namely the modified Rankin Scale (mRS) to define the degree of disability and dependence for daily activities. Motivation to use these scales is described elsewhere (23).

Evaluation of oedema

Oedema assessment was performed at the end of the second actigraphic measuring period (t1), at 1 month (t2) and at 3 months follow-ups (t3) (Fig. 1). Evaluation of oedema was performed by a clinical investigation in combination with a volumetric measurement based on water displacement. The clinical investigation consists of an anamnesis combined with a visual inspection and palpation, further referred to as the subjective assessment. Clear signs of oedema, such as swelling of the dorsum of the hand and or arm, were assessed and registered in the CRF.

The volumetric measurement, further referred to as the objective assessment, based on water displacement, has been described in detail previously (7). The volume of the paretic arm derived from the water displacement was compared with the volume of the non-paretic arm by means of the prediction formulas described by Gebruers et al. (7) in 2007. Oedema was present when the volume of the paretic arm was higher than the upper bound calculated by the prediction formulas.

If a patient was suspected to have oedema due to the presence of a CRPS a bone scan was performed to differentiate between disuse oedema and oedema as a symptom of CRPS. CRPS was suspected when disproportionate pain, temperature changes, decreased range of motion, skin

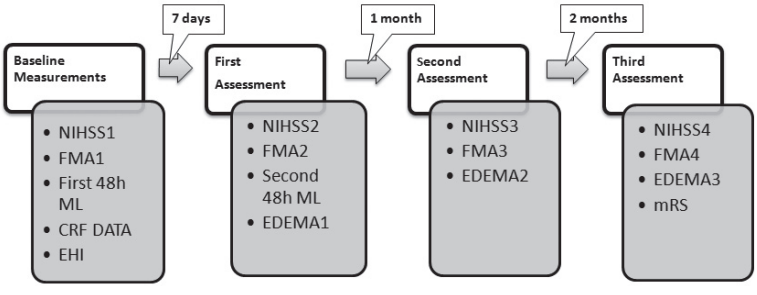

Fig. 1. Timing of the assessed items. NIHSS: National Institute of Health Stroke Scale; FMA: Fugl-Meyer Assessment (arm section); ML: motion loggers; CRF: clinical research form; EHI: Edinburgh Handedness Inventory; EDEMA: subjective as well as objective assessment; mRS: modified Rankin Scale.

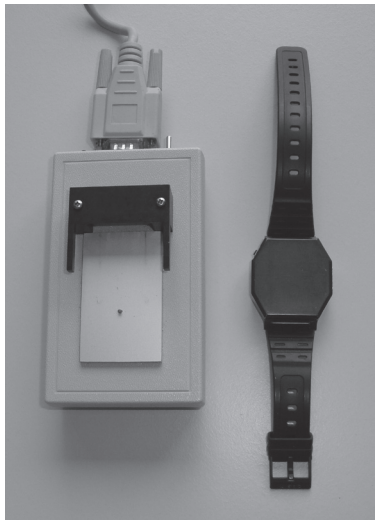

Fig. 2. Octagonic basic motion logger and interface (Ambulatory Monitoring Inc., New York, USA).

changes were present and no other plausible cause could be detected. Patients with CRPS were excluded from the sample, as discussed later.

Clinical management of oedema

The clinical management of oedema on our neurology ward aims at the prevention of development of upper limb oedema. Patients therefore underwent an interdisciplinary oedema preventive treatment during their stay on the acute neurology ward. Our interdisciplinary approach to prevent oedema development consisted of a combination of clinical interventions and is the standard therapy for all stroke patients admitted to our neurology ward. All staff members were responsible for correct positioning of the paretic/hemiplegic arm. Whereas positioning is traditionally applied to prevent contractures/spasticity (27, 28), in this case it was combined with elevation to diminish the negative effects of gravity. Nursing staff and physiotherapists active on the ward had followed a Bobath course to better understand the neuro-developmental treatment and to act appropriately. All blood samplings, blood pressure measurements and/or monitoring were performed on the non-paretic arm. The aim was to achieve early mobilization of the arm and the patient. Patients were instructed to promote self-mobilization of the paretic arm and were asked to repeat the exercises explained to them by the physical therapists. Occupational therapists stimulated the patients to be responsible for their own activities of daily living as far as possible. A similar preventive approach, although in a rehabilitation centre, was used in the study of Boomkamp-Koppen et al. (6).

Statistical analyses

Descriptive statistics were used for the demographic variables of the sample. Correlations between the actigraphic data (PIM) and stroke scales were calculated using a Spearman's correlation test, as all NIHSS and FMA items are scored as an ordinal rank order. Incidence scores for oedema were then calculated, with exclusion of the CRPS patients (n=6). The variables used to determine the differences between the patients with and without oedema were tested for equal variances and normality by the Levene's and Kolgomorov-Smirnov test, respectively. Since no normal distribution and no equal variance were present, differences among oedema and non-oedema patients were tested by means of a Mann–Whitney U test.

Statistical analyses were performed on all available data of the patients who completed the assessments for any given time-point. All statistical analyses were performed by SPSS 12.0 (SPSS Inc., Chicago, USA) and a probability level of 0.05 was used for statistical significance.

Table I. *Demographic descriptive results of the participating patients at baseline (t0) and for the patients with and without oedema after 1 week (t1)*

Variable	Total t0 n=130	Oedema t1 n=15	No oedema t1 n=88
Age, years, mean (SD)	74.6 (11.5)	75 (10.9)	73.2 (10.8)
Body length (m), mean (SD)	1.69 (0.09)	1.7 (0.1)	1.69 (0.09)
Body mass (kg), mean (SD)	75.9 (14.9)	83.8 (16.4)	75.6 (14.8)
BMI, mean (SD)	26.4 (4.1)	28.8 (4.4)	26 (4)
LoS (days), mean (SD)	16.6 (10.5)	23 (16.3)	16 (9.3)
FMA (/66)			
Median (IQR)	23.5 (48)	4 (25)	31.5 (52)
NIHSS (/42), median (IQR)	8 (8)	13 (6)	7 (6)
Gender, % (n)			
Male	60 (78)	73 (11)	65 (57)
Female	40 (52)	27 (4)	35 (31)
Side of paresis, % (n)			
Left	52 (67)	66 (10)	49 (43)
Right	48 (63)	34 (5)	51 (45)
Type of stroke, % (n)			
Ischaemic	91 (119)	93 (14)	91 (80)
Haemorrhagic	9 (11)	7 (1)	9 (8)
Handedness, % (n)			
Right-handed	92 (120)	100 (15)	91 (80)
Left-handed	6 (7)		7 (6)
Ambidextrous	2 (3)		2 (2)

SD: standard deviation; BMI: body mass index; LoS: length of stay; IQR: interquartile range; FMA: Fugl-Meyer Assessment (arm section); NIHSS: National Institute of Health Stroke Scale score.

RESULTS

A total of 139 patients with acute stroke were recruited for this prospective study. Three patients died during the baseline period. Another 6 patients were removed from the analyses because they were diagnosed as having CRPS. These patients were excluded because their oedema could be attributed to

Table II. *Spearman rho correlations between actigraphic variables and stroke scales*

	NIHSS t0	NIHSS t1	NIHSS t2	NIHSS t3	FMA t0	FMA t1	FMA t2	FMA t3	mRS t3
Ratio t0	–0.25**	–0.37**	–0.28**	–0.36**	0.28**	0.47**	0.36**	0.50**	–0.13
Ratio t1	–0.43**	–0.49**	–0.49**	–0.46**	0.61*	0.71**	0.66**	0.63**	–0.49**
Activity paretic arm t0	–0.61**	–0.59**	–0.52**	–0.53**	0.73**	0.70**	0.58**	0.50**	–0.61**
Activity paretic arm t1	–0.56**	–0.61**	–0.56**	–0.55**	0.68**	0.71**	0.59**	0.53**	–0.59**

*p<0.05; **p<0.01; the mRS was scored only at the end of the 3-month follow-up.
t0: at baseline; t1: after 1 week; t2: at 1 month follow-up; t3: at 3 month follow-up; NIHSS: National Institute of Health Stroke Scale score; FMA: Fugl-Meyer Assessment (arm section); mRS: modified Rankin Scale.

CRPS. Therefore, the results of 130 patients are presented. Descriptive results for the total sample at baseline, as well as demographic variables for the oedema and non-oedema patients at t1 for the objective evaluation are shown in Table I.

Correlations between the activity variables and the scales used to understand whether significant correlations were present were calculated. Table II gives an overview of the calculated correlations.

The incidence of oedema based on both evaluation protocols was then calculated. The results of the oedema evaluation during follow-up are presented in Table III.

As a final step the differences between patients with and without oedema were tested, based on the objective evaluation, to find significant differences among oedema and non-oedema patients. Since the activity variables were not normally distributed and both scales were scored on an ordinal scale, a Mann–Whitney *U* test was used. The raw values and the outcome of Mann–Whitney *U* test are presented Table IV.

Table III. *Incidence of arm oedema and arm volumes of the oedema patients at different time-points.*

	t1	t2	t3
Incidence objective oedema (water displacement), *n* (%)	15/103 (14.6%)	10/86 (11.6%)	6/78 (7.7%)
Incidence subjective oedema (visual inspection and palpation), *n* (%)	12/107 (11.2%)	14/99 (14.1%)	12/92 (13.0%)
Agreement between objective and subjective measurement (kappa)	0.77 $p<0.001$	0.48 $p<0.001$	0.48 $p<0.001$
Arm volume (ml) of the affected arm, median (IQR)	2365 (557)	2092 (535)	2370 (762)
Arm volume (ml) of the unaffected arm, median (IQR)	2033 (564)	1873 (690)	2253 (888)

First, the incidence of oedema was calculated at every assessment. The agreement between the objective and subjective evaluation was calculated by means of a kappa score. Secondly, the arm volumes of the oedema patients based on the objective evaluation were calculated for every time-point. t1: after 1 week; t2: at 1 month follow-up; t3: at 3 month follow-up; IQR: interquartile range.

DISCUSSION

The aim of this study was to investigate the disuse hypothesis to explain upper limb oedema development in patients with stroke. Disuse and the accompanying dependency due to a hemiparetic arm might be the reason why patients with stroke experience upper limb oedema (12, 16, 17).

There were significant correlations between activity variables and the scales used, which suggest a relationship between severity of stroke, paresis and activity. However, when patients with and without oedema were compared, no significant differences were found for the activity variables. Only at t1 did we find a trend ($p=0.053$) for the activity of the hemiparetic arm between patients with and without oedema. This trend was no longer existent at t2 or t3. We compared the patients with and without oedema based on the objective evaluation. Moderate to good agreement (kappa) was found between the subjective and objective evaluations, which is comparable to the results found by Post et al. (2). However, the volumetric evaluation uses a standardized protocol and has proven reliability (7, 29,

Table IV. *Differences between patients with and without oedema.*

Variable	No oedema t1 ($n=88$)	Obj oedema t1 ($n=15$)	p	No oedema t2 ($n=76$)	Obj oedema t2 ($n=10$)	p	No oedema t3 ($n=72$)	Obj oedema t3 ($n=6$)	MWU
Activity of HP arm t0 Counts/h, median (IQR)	30,078 (37,072)	31,687 (16,033)	NS	36,187 (36,102)	34,010 (25,678)	NS	37,146 (36,789)	19,722 (53,560)	NS
Activity of non-HP arm t1 Counts/h, median (IQR)	36,826 (57,600)	23,269 (13,684)	NS	38,169 (52,880)	55,868 (47,576)	NS	42,953 (52,221)	21,683 (69,991)	NS
Activity of HP arm t0 Counts/h, median (IQR)	73,591 (41,913)	64,036 (57,332)	NS	80,294 (40,715)	55,868 (47,576)	NS	80,747 (39,240)	66,751 (43,508)	NS
Activity of non-HP arm t1 Counts/h, median (IQR)	82,880 (50,707)	67,120 (42,397)	NS	83,459 (49,770)	63,487 (45,438)	NS	84,342 (50,306)	79,706 (60,390)	NS
Ratio t0, median (IQR)	0.55 (0.53)	0.39 (0.37)	NS	0.47 (0.52)	0.39 (0.55)	NS	0.52 (0.51)	0.42 (0.45)	NS
Ratio t1, median (IQR)	0.59 (0.61)	0.43 (0.36)	NS	0.59 (0.63)	0.57 (0.53)	NS	0.62 (0.59)	0.45 (0.60)	NS
NIHSS t0, median (IQR)	7 (6)	13 (6)	*	6.5 (7)	12 (9)	NS	6 (6)	12.5 (9)	*
NIHSS t1, median (IQR)	4.5 (6)	8 (5)	*	4 (7)	10.5 (10)	*	3.5 (6)	8.5 (10)	NS
FMA t0, median (IQR)	31.5 (52)	4 (25)	*	32.5 (50)	10 (27)	NS	36 (50)	10 (37)	NS
FMA t1, median (IQR)	46 (50)	22 (40)	*	55.5 (47)	13.5 (38)	*	56 (43)	13.5 (47)	NS
LoS, median (IQR)	14 (12)	22 (23)	NS	13 (9)	26 (14)	*	14.5 (10)	24 (48)	NS

*$p<0.05$ with Mann–Whitney *U* test.

No significant differences between patients with and without oedema were found for the activity variables at any time-point. t1: after 1 week; t2: at 1 month follow-up; t3: at 3 month follow-up; NS: not significant; HP: hemiparetic; IQR: interquartile range.

30). Therefore, we used the objective evaluation to divide the sample into patients with and without oedema.

Based on the results of the Mann–Whitney *U* test, which did not identify differences in activity among patients with and without oedema, we concluded that the amount of activity presented in the acute phase of stroke does not contribute to post-stroke oedema development. This implies that the disuse hypothesis is not solely explanatory for upper limb oedema after stroke.

The results presented here also have some clinical implications. In the literature several attempts to treat upper limb oedema in patients with stroke have been discussed. Both Dirette et al. (16) and Giudice (31) described a protocol based on continuous passive motion to treat oedema in patients with stroke. Although both studies found a decrease in the oedema volume, these results are clinically not significant since volume reduction was limited to 2.7% (31). Another approach to treat hand oedema after stroke is the use of neuromuscular stimulation. Again, statistical significant differences were found between pre- and post-stimulation oedema volumes (12), but these results were also clinically inadequate. The hand volumes decreased by 2.64% and the arm volumes decreased by 1.97% after 30 min of neuromuscular stimulation (12). Although the above-mentioned studies have methodological limitations, it could be that the attempts to treat oedema were relatively unsuccessful because oedema development is not entirely explained by the loss of upper limb activity, as is demonstrated in the present study.

Since no successful treatment for upper limb oedema after stroke is available to date, the focus should be on its prevention. We have described the standard therapy to prevent oedema in our neurology ward. The incidence of oedema found in the present study is lower than that found in recent literature. We found the incidence of upper limb oedema after stroke ranged from 7.7% to 14.6%, while in the literature the incidence ranges from 28% to 33% (2, 6, 32). However, comparison between studies investigating the development of oedema after stroke is difficult because of methodological differences.

This study has some limitations, as follows: first, although a large number of patients were included in the study, the number of patients with oedema was low. It is therefore probable that no statistical significance was found, due to the low number of oedema patients. Secondly, in addition to the 3 patients who died during baseline measurements, another 25 (18%) patients died during follow-up. This percentage is within the normal expectancy since other studies found percentages of 28% (33) and 21% (34). As presented by the changing sample sizes we were confronted with other dropouts. The main reason was our inability to trace some patients after discharge despite the fact that contacts were registered in the clinical research form. It is probable that these patients stayed with relatives or friends during rehabilitation. An additional reason for missing data, as is clearly seen by the changing sample sizes in Table III, was that some patients withdrew their consent for the volumetric evaluation.

In conclusion, this study found no difference in upper limb activity between patients with and without oedema after stroke. It is unlikely that the loss of activity of the paretic limb is solely responsible for the development of upper limb oedema after stroke.

ACKNOWLEDGEMENTS

This research was supported by: the Artesis University College of Antwerp, Belgium (grant no. G817); the Thomas Riellaerts Research Fund; the Institute Born-Bunge; the agreement between the Institute Born-Bunge and the University of Antwerp; the Interuniversity Attraction Poles (IAP) programme P6/43 of the Belgian Federal Science Policy Office, Belgium; a Methusalem Grant of the Flemish Government; Medical Research Foundation Antwerp; Neurosearch Antwerp; and the Fund for Scientific Research – Flanders (FWO–F).

REFERENCES

1. Geurts AC, Visschers BA, van Limbeek J, Ribbers GM. Systematic review of aetiology and treatment of post-stroke hand oedema and shoulder-hand syndrome. Scand J Rehabil Med 2000; 32: 4–10.
2. Post MW, Visser-Meily JM, Boomkamp-Koppen HG, Prevo AJ. Assessment of oedema in stroke patients: comparison of visual inspection by therapists and volumetric assessment. Disabil Rehabil 2003; 25: 1265–1270.
3. Vasiliauskas R, Dijkers M, Abela MB, Lundgren L. Characteristics in addition to size of the contralateral predict hand volume but are not clinically useful. J Hand Ther 1995; 4: 258–263.
4. Exton-Smith AN, Crockett DJ. Nature of oedema in paralysed limbs of hemiplegic patients. Br Med J 1957; 2: 1280–1283.
5. Tepperman PS, Greyson ND, Hilbert L, Jimenez J, Williams JI. Reflex sympathetic dystrophy in hemiplegia. Arch Phys Med Rehabil 1984; 65: 442–447.
6. Boomkamp-Koppen HG, Visser-Meily JM, Post MW, Prevo AJ. Poststroke hand swelling and oedema: prevalence and relationship with impairment and disability. Clin Rehabil 2005; 19: 552–559.
7. Gebruers N, Truijen S, Engelborghs S, De Deyn PP. Volumetric evaluation of upper extremities in 250 healthy persons. Clin Physiol Funct Imag 2007; 27: 17–22.
8. Werner GT, Gerhards W, Ingrisch H, Schutte B. Szintigraphische Untersuchungen des Lymphtransportes in gelähmten oberen Extremitäten. VASA Zeitschrift fur Gefasskrankheiten 1997; 26: 25–28.
9. Modi S, Stanton AW, Svensson WE, Peters AM, Mortimer PS, Levick JR. Human lymphatic pumping measured in healthy and lymphoedematous arms by lymphatic congestion lymphoscintigraphy. J Physiol 2007; 583: 271–285.
10. Stanton AW, Mellor RH, Cook GJ, Svensson WE, Peters AM, Levick JR, et al. Impairment of lymph drainage in subfascial compartment of forearm in breast cancer-related lymphedema. Lymphatic Res Biol 2003; 1: 121–132.
11. Szuba A, Shin WS, Strauss HW, Rockson S. The third circulation: radionuclide lymphoscintigraphy in the evaluation of lymphedema. J Nucl Med 2003; 44: 43–57.
12. Faghri PD. The effects of neuromuscular stimulation-induced muscle contraction versus elevation on hand edema in CVA patients. J Hand Ther 1997; 10: 29–34.
13. Rymal C. Comprehensive decongestive therapy for lymphedema in patients with a history of cerebral vascular accident. Clin J Oncol Nurs 2003; 7: 677–678.
14. Wang JS, Chen SY, Lan C, Wong MK, Lai JS. Neuromuscular electric stimulation enhances endothelial vascular control and hemodynamic function in paretic upper extremities of patients with stroke. Arch Phys Med Rehabil 2004; 85: 1112–1116.
15. Wang JS, Yang CF, Liaw MY, Wong MK. Suppressed cutaneous endothelial vascular control and hemodynamic changes in paretic

extremities with edema in the extremities of patients with hemiplegia. Arch Phys Med Rehabil 2002; 83: 1017–1023.
16. Dirette D, Hinojosa J. Effects of continuous passive motion on the edematous hands of two persons with flaccid hemiplegia. Am J Occup Ther 1994; 48: 403–409.
17. Roper TA RS, Tallis RC. Intermittent compression for the treatment of the oedematous hand in hemiplegic stroke: a randomized controlled trial. Age Ageing 1999; 28: 9–13.
18. Adams HP Jr, Bendixen BH, Kappelle LJ, Biller J, Love BB, Gordon DL, et al. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. Stroke 1993; 24: 35–41.
19. Goldstein LB, Jones MR, Matchar DB, Edwards LJ, Hoff J, Chilukuri V, et al. Improving the reliability of stroke subgroup classification using the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) criteria. Stroke 2001; 32: 1091–1098.
20. Ay H, Benner T, Arsava EM, Furie KL, Singhal AB, Jensen MB, et al. A computerized algorithm for etiologic classification of ischemic stroke: the Causative Classification of Stroke System. Stroke 2007; 38: 2979–2984.
21. Nagels G, Engelborghs S, Vloeberghs E, Lemmens W, Pickut BA, De Deyn PP. Correlation between actigraphy and nurses' observation of activity in dementia. Int J Geriatr Psychiatr 2007; 22: 84–86.
22. Nagels G, Engelborghs S, Vloeberghs E, Van Dam D, Pickut BA, De Deyn PP. Actigraphic measurement of agitated behaviour in dementia. Int J Geriatr Psychiatr 2006; 21: 388–393.
23. Gebruers N, Truijen S, Engelborghs S, Nagels G, Brouns R, De Deyn PP. Actigraphic measurement of motor deficits in acute ischemic stroke. Cerebrovascular Dis (Basel, Switzerland) 2008; 26: 533–540.
24. Ancoli-Israel S, Cole R, Alessi C, Chambers M, Moorcroft W, Pollak CP. The role of actigraphy in the study of sleep and circadian rhythms. Sleep 2003; 26: 342–392.
25. Uswatte G, Foo WL, Olmstead H, Lopez K, Holand A, Simms LB. Ambulatory monitoring of arm movement using accelerometry: an objective measure of upper-extremity rehabilitation in persons with chronic stroke. Arch Phys Med Rehabil 2005; 86: 1498–1501.
26. Uswatte G, Giuliani C, Winstein C, Zeringue A, Hobbs L, Wolf SL. Validity of accelerometry for monitoring real-world arm activity in patients with subacute stroke: evidence from the extremity constraint-induced therapy evaluation trial. Arch Phys Med Rehabil 2006; 87: 1340–1345.
27. de Jong LD, Nieuwboer A, Aufdemkampe G. Contracture preventive positioning of the hemiplegic arm in subacute stroke patients: a pilot randomized controlled trial. Clinical Rehabil 2006; 20: 656–667.
28. Rabadi MH. Randomized clinical stroke rehabilitation trials in 2005. Neurochemical Res 2007; 32: 807–821.
29. Farrell K, Johnson A, Duncan H, Offenbacker T, Curry C. The intertester and intratester reliability of hand volumetrics. J Hand Ther 2003; 16: 292–299.
30. Taylor R, Jayasinghe UW, Koelmeyer L, Ung O, Boyages J. Reliability and validity of arm volume measurements for assessment of lymphedema. Phys Ther 2006; 86: 205–214.
31. Giudice ML. Effects of continuous passive motion and elevation on hand edema. Am J Occup Ther 1990; 44: 914–921.
32. Leibovitz A, Baumoehl Y, Roginsky Y, Glick Z, Habot B, Segal R. Edema of the paretic hand in elderly post-stroke nursing patients. Arch Gerontol Geriatr 2007; 44: 37–42.
33. Bronnum-Hansen H, Davidsen M, Thorvaldsen P. Long-term survival and causes of death after stroke. Stroke 2001; 32: 2131–2136.
34. Sweileh WM. Predictors of in-hospital mortality after acute stroke: impact of renal dysfunction. Int J Clin Pharmacol Ther 2008; 46: 637–643.