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Changes in oro-facial function and hand-grip strength during a 2-year observation period after stroke

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Abstract

Objectives Recovery from stroke-related oro-facial impairment has rarely been investigated. In this longitudinal study chewing efficiency, maximum lip and bite force as well as masseter muscle thickness were evaluated and compared with hand-grip force.

Materials and methods Thirty-one hospitalized stroke patients and 24 controls were recruited for this cohort study with 2-year follow-up. Chewing efficiency was evaluated with a color mixing ability test, lip forces with a traction dynamometer, bite force with a digital gauge, masseter muscle thickness using ultrasound measurements and grip strength with a hand dynamometer.

Results During the 2-year observation period, patients were evaluated four times. A total of 21 patients dropped out of the study. Stroke patients showed significantly impaired chewing efficiency and lower lip forces than controls with no significant improvement over time. Bite forces were not

different between ipsi- and contralesional sides, in contrast to contralesional hand-grip strength which was significantly impaired and did not improve during the observation period. On the first examination with a median of 40 days after stroke, masseter thickness was reduced contralesional, but did not continue to show significant side-differences during follow-up.

Conclusions Stroke affects the upper limb and the masseter muscles differently on a functional and morphological level. Further research is needed to evaluate the predictive value of oro-facial parameters on functional outcome after stroke.

Clinical relevance Impaired chewing efficiency and reduced lip force are quantifiable symptoms in stroke patients which seem not to improve in absence of oro-facial rehabilitation procedures.

Keywords Stroke recovery · Chewing efficiency · Hand-grip strength · Bite force · Lip force · Masseter muscle thickness

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Introduction

Infarction or hemorrhage in the region of the cortical masticatory area or the motoneuron pools in the brain stem [1] might affect oral sensation, and impair strength as well as coordination of the muscles involved in oro-facial function. Hence, many stroke survivors show symptoms like chewing difficulties, hoarding of food or the inability to form or position a bolus in preparation for swallowing [2]. In hospitalized stroke patients, Oral Health Related Quality of Life proved to be significantly reduced, especially in the domains “functional limitation” and “physical pain” and was significantly related to their poor chewing function [3], but reports on its quantitative assessment are scarce [2, 4, 5].

The cortical forearm representation area is closely located to the cortical masticatory area; thus, a lesion resulting from middle cerebral artery (MCA) stroke is likely to affect both hand and oro-facial function. However, anatomical- and transcranial magnetic stimulation (TMS) studies have revealed cortico-trigeminal projections from the motor cortex to both ipsi- and contralateral masseter motoneuron pools with contralateral dominance [6, 7], whereas the hand is mainly guided by the contralateral hemisphere [8]. Thus, input pathways from the stomatognathic apparatus to the cerebral cortex and output pathways from the cerebral cortex to the muscles of the head and neck show cross dominance but the dominance may be less prominent compared with the spinal nerves that innervate the extremities [9]. Consequently, maximum bite forces in stroke patients are not reduced when compared to healthy controls [5, 10], but hand-grip strength (HGS) is permanently impaired in the majority of stroke patients [11]. Although there seems to be an early onset of atrophy of the contralesional masseter muscle [12] there are no longitudinal observations to compare it to upper limb atrophy [13].

Tooth loss and the related loss of masticatory function may lead to a decreased intake of calories, protein, vitamins, and iron often resulting in mal- or undernutrition [14]. Thus impaired chewing and swallowing might contribute to the frequently observed weight loss [15] which seems to be linked to unfavorable functional outcome [16], or even mortality [17].

The aim of this study was to evaluate over 2 years longitudinal changes in chewing efficiency, maximum restraining lip force (MLF), maximum voluntary bite force (MBF), masseter muscle thickness (MMT), and HGS in stroke patients.

Material and methods

The study protocol was approved by the University Hospitals of Geneva's ethics committee (N.A.C. CER 06-002/06-018). Written informed consent was obtained from each subject.

Subjects

Between November 2006 and December 2007, 31 patients recovering from stroke were recruited consecutively from the Division of Neurorehabilitation of the Department of Clinical Neurosciences of the University Hospitals of Geneva (Switzerland) by a senior consultant neurologist. Inclusion criteria comprised understanding of the patient information, consent to participate, a hemi-syndrome with facial palsy and the capability to perform various clinical tests. Previous stroke did not elude participation. The

National Institute of Health Stroke Scale (NIHSS) [18] was used to evaluate the neurological deficit.

A total of 24 healthy controls similar to the patient group in regard to age, gender and dental state were recruited from staff and patients of the Geneva Dental School.

Study design

The current study was designed as a cohort study (group definitions: stroke group, control group). Stroke patients were assessed as soon as possible after stroke [T1], after 6 [T2], 12 [T3] and 24 [T4] months. Controls were assessed at baseline [T1], after 12 [T3] and 24 [T4] months.

At T1, a cross-sectional (a priori) analysis comprised all study participants. For the longitudinal analysis only study participants who attended at least three out of four examinations were taken into consideration.

Some of the a priori analysis have been reported previously, but are included for completeness [5, 12].

Dental state

The total number of teeth, the DMFT (Decayed, Missing, Filled Teeth) index [19] and the number of occluding premolar units (OU) according to Kayser (1 molar counts as 2 OU) [20] were noted. For the stroke patients, the number of occluding units was distinguished between the ipsi- or contralesional side.

Chewing efficiency

Chewing efficiency was quantified with a previously described color-mixing ability test [21]. Therefore, a two-colored chewing gum is masticated for 20 chewing cycles on the preferred chewing side, retrieved from the oral cavity, flattened to a 1-mm-thick wafer and evaluated optoelectronically. The ratio of unmixed azure color pixels (unmixed fraction [UF]) to the total pixel number in a fixed size template is calculated and serves as a measure for chewing efficiency. UF shows characteristics of a logarithmic function (\log_{10}) on the basis of chewing cycles [5]. It will decrease for two reasons: a higher degree of color blending and a loss in mass of the specimen due to sweetener extraction; both are measures for chewing efficiency [22]. Thus, the higher the UF, the lower is the chewing efficiency.

Maximum restraining lip force

MLF was evaluated using a hard resin screen ($29 \times 17 \times 2$ mm) which was slightly curved to adapt to an average dental arch. The screen was placed in the oral vestibule and a perpendicular pulling force was applied via a steel wire

(diameter 0.9 mm, allowing for complete lip closure) connecting the plate in the midline to a traction dynamometer (Imada digital force gauge; Imada Co., Toyohashi, Japan). The participant was asked to withstand the displacement force as long as possible; three measurements were averaged for analysis.

Maximum voluntary bite force

MBF was assessed with a digital force gauge with an 8.6-mm-thick bite element (Occlusal Force-Meter GM 10®; Nagano Keiki Co. Ltd., Tokyo, Japan) [23]. The sensor was placed between the first molars of each side separately and the participant was asked to exert a maximum clenching effort but to stop when she/he started feeling uncomfortable. For the patient group, the means of three readings of the contralesional side and the ipsilesional side were calculated separately. For the controls, the mean of six recordings (3× right, 3× left) was used for analysis.

Hand-grip strength

HGS was measured using a Baseline® Hydraulic Hand Dynamometer (Baseline Evaluation Instruments, White Plains, NY, USA) which measures grip strength equivalently to a Jamar® Hand Dynamometer [24]. In the patient group, the mean value of three recordings of the contralesional and the ipsilesional sides was used for analysis. For the control group, the mean of six recordings (3× right, 3× left) was calculated.

Masseter muscle thickness

MMT in contracted muscle condition was quantified with a real time ultrasound scanner (FALCO 100, linear array transducer [6–8 MHz], PieMedical, The Netherlands) following a previously described protocol [25]. In the patient group, the mean of two readings of the contralesional and the ipsilesional sides of each were used for analysis. In the control group, the mean of four recordings (2× right, 2× left) was calculated. The error of the method in contracted muscle condition was reported to be between 0.2 and 0.4 mm [25, 26].

Statistical analysis

Continuous data are expressed as median values followed by the interquartile range in brackets; normal distribution of variables was confirmed with the Shapiro–Francia W' test. Categorical variables were compared using Fisher's exact test. Correlations were calculated using the Spearman rank correlation method. The level of significance was set at $\alpha < 0.05$; sample size calculation could not be performed due to the absence of reference data. Statistical tests were

performed using Stata Statistical Software release 11.1 (Stata Corporation, College Station, TX, USA).

A priori cross-sectional analysis at T1

The null hypothesis for the a priori cross-sectional analysis at T1 was that there was no difference in the investigated parameters between stroke and patient groups [5, 12]. Non-parametric tests (Mann–Whitney) were applied to detect differences between patients with follow-up and drop-outs and the Wilcoxon tests to compare sides of the same subjects.

Missing values and drop-outs

Because repeated measures analyses require that there are no missing values, imputation techniques might help replacing some missing data. However, because of the high drop-out rate only patients and controls who attended at least three out of four examinations (fupPG and fupCG, respectively) were included in the analysis to avoid an imputation bias [27]. However, control group (fupCG) data from T1 were carried forward to T2, as they were only seen at T1, T3 and T4. One stroke patient's T2 and another patient's T4 data were missing and imputed by carrying forward their previous value (from fupPG). Simple and multiple logistic regression models served to identify factors which could predict the dropout and provided odds ratios (OR).

Longitudinal analysis

The null hypothesis for the longitudinal analysis was that there is no difference between the stroke and control groups and no improvement over the observation period in the investigated parameters.

As no non-parametric test can simultaneously compare groups and deal with repeated measures, analysis of follow-up data comprised analysis of variance (ANOVA) with correction for repeated measures using box's conservative epsilon. The correcting factors were stroke (patient or control group) and time points as the repeated factor. The results of the Shapiro–Francia W' tests were non-significant for all investigated parameters (all $p > 0.05$); furthermore, the empirical rule of Harrel was respected [28].

Results

Brain lesions were confirmed with MRI among admission to the hospital. All 31 participants suffered from hemiparesis; among them were 17 with a combined motor and sensory deficit (Table 1). Patients were seen after a median of 40.0 (22.0) days after stroke.

Table 1 Characteristics of study participants; data presented as median (interquartile range)

	iPG	dropoutPG	fupPG	iCG	fupCG
Number of participants	31	21	10	24	10
Age [years]	71.0 (15.1)	75.0 (16.0)	64.1 (17.4)	68.3 (18.4)	64.4 (18.6)
Gender	18 ♂, 13 ♀	12 ♂, 9 ♀	6 ♂, 4 ♀	13 ♂, 11 ♀	6 ♂, 4 ♀
NIHSS at inclusion	5.0 (5.0)	6.0 (4.3)	4 (4)	n/a	n/a
Stroke type	23 ischemic, 8 hemorrhagic	15 ischemic, 6 hemorrhagic	8 ischemic, 2 hemorrhagic	n/a	n/a
Stroke localization	29 hemispheric, 2 pons	19 hemispheric, 2 pons	10 hemispheric	n/a	n/a
Stroke side	21 right, 10 left	13 right, 7 left	8 right, 2 left	n/a	n/a
Number of teeth	21.0 (11.0)	18.0 (10.0)	23.0 (8.0)	21.5 (18)	21.5 (19.0)
Number of occluding units	4.0 (8.0)	3.0 (5.0)	6.0 (7.0)	5.5 (10)	8.5 (12.0)
DMFT [n]	20.0 (11.0)	23.0 (10.0)	15.0 (7.0)	20 (7.5)	18.0 (13.0)

iPG initially recruited patients group, *dropoutPG* patients who dropped out of the study after first assessment, *fupPG* patients who returned for follow-up, *iCG* initial control group, *fupCG* control group with follow-up, *NIHSS* National Institute of Health Stroke Scale, *DMFT* number of decayed, missing, filled teeth

Of the initially recruited patient group [5], nine participants returned for the first follow-up assessment [T2, 213.0 (15.0) days after T1], ten for the second [T3, 395.0 (35.0) days post T1] and nine for the final examination [T4, 754.0 (32.0) post T1]. At T2, from the 31 patients, six had withdrawn their consent, three had moved abroad, six could not be contacted (and could not be traced down by the population registration office), three patients were too ill to participate and three had passed away. At T3, one patient had recovered sufficiently to return for the assessment and at T4 another patient had died. Ten [T3, 404.0 (36.0) post T1], respectively 9 [T4, 739.0 (38.0) post T1] matched control subjects were followed up. The time span between T1 to T3 and T1 to T4 was not different between stroke patients and controls ($p=0.623$ and $p=0.535$, respectively; Mann–Whitney). The NIHSS score did not predict the return for follow-up (OR=0.91; 95 % confidence interval [CI], 0.724–1.144; $p=0.419$; pseudo $r^2=0.0180$; logistic regression).

Patients had been hospitalized for a median time of 55 (68) days. They had received a normal unmixed hospital diet and followed an individualized rehabilitation program (Table 2).

For the T2 evaluation, all patients but one lived at home. The only exception was still hospitalized at T2, but returned for T3 and T4.

Dental state

At T1, patients and the controls were not different regarding dental state, age or gender [5]. However, patients who returned for the follow-up assessments had initially a significantly lower DMFT score ($p=0.0397$, Mann–Whitney), more teeth ($p=0.0419$, Mann–Whitney), and also more

occluding premolar units ($p=0.0484$, Mann–Whitney) (OU contralesional side 4.0 (6.0), OU ipsilesional side 3.5 (4.0), $p=0.3104$, Wilcoxon) than those who were not available for follow-up (Table 1). Although six of ten patients with follow-up underwent dental treatment after their stroke, according to the ANOVA model there was no significant change over time in occluding premolar units ($F_{3,72}=0.03$, $p_{\text{BOX}}=0.8708$, interaction term: $F_{3,72}=0.03$, $p_{\text{BOX}}=0.8708$).

Lower DMFT scores (OR=0.88; 95 % CI, 0.768–0.999; $p=0.047$; pseudo $r^2=0.1177$; logistic regression) and also the number of occluding units (OR 1.25; 95 % CI, 1.004–1.542; $p=0.046$; pseudo $r^2=0.1161$, logistic regression) predicted significantly the participation in the follow-up examinations. Thus, each additional occlusal unit increased the chance for follow-up by 24.5 % and each lower DMFT score by 14.2 %.

Chewing efficiency

At T1, stroke patients chewed significantly less efficient than their corresponding controls [5]. One patient could not be assessed, because she wore no lower denture (Table 3). At T1, patients who later returned for follow-up chewed significantly better than those who dropped out ($p=0.0209$, Mann–Whitney). Lower UF scores, indicating a higher chewing efficiency, predicted significantly the participation in the follow-up (OR=4.15⁻¹⁰; 95 % CI, 1.28⁻¹⁸ to 0.1342; $p=0.031$; pseudo $r^2=0.1509$; logistic regression).

The ANOVA model showed no change in UF over time ($F_{3,72}=0.52$, $p_{\text{BOX}}=0.4792$, interaction term: $F_{3,72}=1.03$, $p_{\text{BOX}}=0.3192$) but a tendency to be influenced by the stroke ($F_{1,72}=3.30$, $p=0.0733$).

Table 2 Description of the study population who were available for follow-up assessments

Gender age [years]	NIHSS	Localization	Affected blood vessel	Etiology	Type of reeducation (in 5 out of 7 days)	Rehabilitation (days)	Neurological assessment at entrance
♂ 50	2	Parietal left, temporo-occipital right	MCA left, PCA right ischemic	Cardio embolic	Physiotherapy, occupational therapy, logotherapy, neuropsychological assistance	45	Hemiparesis and hypoesthesia of right side, anterior aphasia, executive function deficit
♀ 74	4	Frontal precentral right	MCA right, ischemic	Cardio embolic	Physiotherapy, occupational therapy, neuropsychological assistance	110	Hemiparesis of left side, hemispatial neglect, executive and mnemonic function deficit
♀ 86	2	Insula right	MCA right, ischemic	Cardio embolic	Physiotherapy, occupational therapy, neuropsychological assistance	30	Ataxic hemiparesis of left side, slight hemispatial neglect
♀ 63	4	Temporal and frontal right	MCA right, ischemic	Carotid occlusion	Physiotherapy, occupational therapy, neuropsychological assistance	7	Hemiparesis and hypoesthesia of left side, hemispatial neglect
♂ 44	12	Parietal right	Right hemorrhagic	Glioblastoma	Physiotherapy, occupational therapy, neuropsychological assistance	196	Left hemianopsia, hemiparesis and hypoesthesia of left side, hemispatial neglect
♀ 64	11	Capsulo-lenticular right	Right hemorrhagic	Hypertension	Physiotherapy, occupational therapy, neuropsychological assistance, psychology (depression)	100	Left hemianopsia, hemiparesis and hypoesthesia of left side, hemispatial neglect, executive function deficit
♂ 68	4	Internal capsule right	MCA right, ischemic	Small vessel disease	Physiotherapy, occupational therapy, neuropsychological assistance	37	Hemiparesis of left side, attention and executive function deficit
♂ 63	6	Left temporo-occipital, thalamus and cerebellum/old frontal lesion right	PCA left, MCA right; PICA left ischemic	Unknown	Physiotherapy, occupational therapy, logotherapy, neuropsychological assistance	50	Right hemianopsia, ataxic hemiparesis, and hypoesthesia of right side, aphasia, alexia, executive and mnemonic function deficit
♂ 57	3	Capsulo-lenticular right	MCA right, ischemic	Aortic atheromatosis	Physiotherapy, occupational therapy, neuropsychological assistance	105	Ataxic hemiparesis of left side, hemispatial neglect executive function deficit,
♂, 75	6	Temporo-occipital, thalamus right	PCA right, ischemic	Cardio embolic	Physiotherapy, occupational therapy, Neuropsychological assistance	60	Left hemianopsia, hemiparesis of left side, hemispatial neglect, executive function deficit

MCA middle cerebral artery, PCA posterior cerebral artery, PICA posterior inferior cerebellar artery

In the control group with follow-up, UF was negatively correlated with the number of occluding premolar units, thus the more premolar units were present, the better the chewing efficiency (T1: $\rho = -0.798$, $p = 0.0166$; T3: $\rho = -0.836$, $p = 0.0121$; T4: $\rho = -0.678$; $p = 0.0420$, Spearman rank correlation). In the patient group with follow-up, this effect was not present (T1: $\rho = -0.477$, $p = 0.6333$; T2: $\rho = -0.457$,

$p = 0.1701$; T3: $\rho = -0.250$, $p = 0.4532$; T4: $\rho = -0.616$, $p = 0.0647$, Spearman rank correlation).

Maximum restraining lip force

At T1, MLF was significantly lower in stroke patients than in the controls [5] (Table 4). The ANOVA model showed no

Table 3 Chewing efficiency expressed as ratio of unmixed azure color in an image of fixed size (UF)

UF	T1			T2	T3	T4
	Initial groups	Drop-outs	With follow-up			
Patients	0.0991 (0.0785), $n = 30$	0.1085 (0.0642), $n = 20$	0.0570 (0.0840), $n = 10$	0.0527 (0.0799), $n = 9$	0.0299 (0.0522), $n = 10$	0.0584 (0.0958), $n = 9$
Controls	0.0380 (0.0365), $n = 24$	0.0380 (0.0260), $n = 14$	0.0360 (0.0480), $n = 10$		0.0362 (0.0456), $n = 10$	0.0286 (0.0371), $n = 9$

Data presented as median (interquartile range). The higher the ratio, the lower is the chewing efficiency. Of the initial patient-group (iPG, $n = 31$), only ten participants were available for follow-up (fupPG). The initial control group (iCG, $n = 24$) was matched in age, dental state and number to the stroke patients. T2, T4, $n = 9$, last value carried forward for one patient

Table 4 Maximum lip restraining force (MLF, [N]), maximum voluntary bite force (MBF, [N]: at T1, four patients were excluded for MBF because of missing teeth), hand-grip strength (HGS, [N]) and masseter muscle thickness in contracted muscle condition (MMT, [mm])

	T1			T2	T3	T4
	Initial groups	Drop-outs	With follow-up			
MLF [N]: patients	6.5 (3.1), <i>n</i> =29	5.1 (2.1), <i>n</i> =19	7.6 (3.2), <i>n</i> =10	8.8 (3.5), <i>n</i> =9	7.8 (2.0), <i>n</i> =10	6.8 (2.8), <i>n</i> =9
MLF [N]: controls	8.4 (3.3), <i>n</i> =24	8.9 (3.6), <i>n</i> =14	7.5 (4.6), <i>n</i> =10		9.3 (2.4), <i>n</i> =10	7.8 (2.6), <i>n</i> =9
MBF [N]: patients, contralesional	145.0 (153.0), <i>n</i> =27	113.2 (103.6), <i>n</i> =18	283.3 (171.0), <i>n</i> =9	323.7 (433.3), <i>n</i> =9	405.9 (274.0), <i>n</i> =10	356.2 (344.6), <i>n</i> =9
MBF [N]: patients, ipsilesional	139.0 (128.0), <i>n</i> =27	91.5 (128.0), <i>n</i> =18	254.0 (300.7), <i>n</i> =9	260.7 (283.7), <i>n</i> =9	313.2 (408.0), <i>n</i> =10	271.0 (448.0), <i>n</i> =9
MBF [N]: controls, mean ri, le	146.9 (195.3), <i>n</i> =24	141.9 (91.5), <i>n</i> =14	176.1 (307.3), <i>n</i> =10		133.5 (264.5), <i>n</i> =10	140.3 (390.6), <i>n</i> =9
HGS [N]: patients, contralesional	22.6 (107.9), <i>n</i> =28	16.7 (49.0), <i>n</i> =19	117.7 (198.1), <i>n</i> =9	188.3 (95.1), <i>n</i> =9	196.1 (137.3), <i>n</i> =10	129.4 (166.7), <i>n</i> =9
HGS [N]: patients, ipsilesional	261.8 (151.0), <i>n</i> =28	255.0 (183.4), <i>n</i> =19	274.6 (130.4), <i>n</i> =9	342.3 (124.5), <i>n</i> =9	355.0 (156.9), <i>n</i> =10	341.3 (144.2), <i>n</i> =9
HGS [N]: controls, mean ri, le	274.6 (198.1), <i>n</i> =23	252.0 (199.1), <i>n</i> =13	297.1 (184.4), <i>n</i> =10		289.3 (221.6), <i>n</i> =10	297.1 (189.3), <i>n</i> =9
MMT [mm]: patients, contralesional	13.18 (4.00), <i>n</i> =30	12.43 (3.55), <i>n</i> =20	13.80 (2.15), <i>n</i> =10	14.85 (3.90), <i>n</i> =9	14.70 (3.1), <i>n</i> =10	14.95 (5.1), <i>n</i> =9
MMT [mm]: patients, ipsilesional	13.50 (2.40), <i>n</i> =30	13.18 (1.93), <i>n</i> =20	14.00 (3.75), <i>n</i> =10	14.25 (4.35), <i>n</i> =9	13.70 (3.40), <i>n</i> =10	14.7 (5.00), <i>n</i> =9
MMT [mm]: controls, mean ri, le	14.31 (4.49), <i>n</i> =24	14.31 (4.20), <i>n</i> =14	13.83 (6.75), <i>n</i> =10		13.78 (6.28), <i>n</i> =10	14.28 (6.28), <i>n</i> =9

Data presented as median (interquartile range). T2, T4 *n*=9, last value carried forward for one patient

change of MLF over time ($F_{3,72}=0.40$, $p_{\text{BOX}}=0.5327$, interaction term: $F_{3,72}=0.28$, $p_{\text{BOX}}=0.6025$) but an influence of stroke ($F_{1,72}=5.43$, $p=0.0226$), with stroke patients having a lower MLF (-1.08 ± 1.18 N).

Maximum voluntary bite force

At T1, MBF was neither different between the contra- and ipsilesional sides nor different to the controls [5] (Table 4). Higher maximum bite forces on the contralesional side in the stroke patients predicted significantly their participation in the follow-up (OR=1.01; 95 % CI, 1.001–1.019; $p=0.024$; pseudo $r^2=0.3090$; logistic regression). Each Newton additional bite force on the contralesional side increased the likelihood for a follow-up by 1 %.

When comparing the MBF of the contra- and ipsilesional side in patients with follow-up, the ANOVA model demonstrated neither a significant change over time of the contralesional side ($F_{1,142}=0.05$, $p_{\text{BOX}}=0.8191$, interaction term: $F_{1,142}=0.03$, $p_{\text{BOX}}=0.8526$) nor an influence of the side (contralesional/ipsilesional) ($F_{1,142}=0.52$, $p=0.4716$).

For MBF on the contralesional side in participants with follow-up, the ANOVA model showed no change over time ($F_{3,71}=0.07$, $p_{\text{BOX}}=0.7963$, interaction term: $F_{3,71}=0.05$, $p_{\text{BOX}}=0.8210$) but a significant influence of the stroke ($F_{1,71}=6.95$, $p=0.0103$), with stroke patients having a

higher MBF on the contralesional side than the controls ($+104.7\pm 100.1$ N).

MBF on the ipsilesional side in the stroke patients did not change over time ($F_{3,71}=0.02$, $p_{\text{BOX}}=0.8968$, interaction term: $F_{3,71}=0.04$, $p_{\text{BOX}}=0.8521$) and was not different to controls ($F_{1,71}=2.58$, $p=0.1124$).

Two years after stroke [T4], MBF on the stroke patient's contralesional side was significantly correlated with the number of occlusal units on that side ($\rho=0.691$, $p=0.0398$, Spearman rank correlation). This effect was not present at any precedent time point (T1: $\rho=-0.051$, $p=0.8846$; T2: $\rho=0.506$, $p=0.1288$; T3: $\rho=0.500$, $p=0.1334$, Spearman rank correlation). In contrast, on the ipsilesional side, MBF was not correlated with the number of occlusal units at T1 ($\rho=0.336$, $p=0.3417$, Spearman rank correlation), but at all following assessments (T2: $\rho=0.838$, $p=0.0119$; T3: $\rho=0.820$, $p=0.0139$; T4: $\rho=0.809$, $p=0.0154$, Spearman rank correlation). In the control group, MBF was correlated with the total number of occlusal units at all time points ($0.792 < \rho < 0.862$, $0.0097 < p < 0.0175$, Spearman rank correlation).

Hand-grip strength

At T1, contralesional HGS was significantly weaker than ipsilesional HGS ($p<0.0001$, Wilcoxon) and weaker than in controls ($p<0.0001$, Mann–Whitney). No difference was

found between controls and the patient group's ipsilesional side ($p=0.6225$, Mann–Whitney) (Table 4).

When comparing contralesional and ipsilesional HGS of patients with follow-up, the ANOVA model demonstrated no significant change over time of the contralesional hand ($F_{3,142}=0.26$, $p_{\text{BOX}}=0.6121$, interaction term $F_{3,142}=0.12$, $p_{\text{BOX}}=0.7263$) but an significant influence of the side (contralesional/ipsilesional) ($F_{1,142}=14.73$, $p=0.0002$) with lower strength on the contralesional side (-197.1 ± 82.87 N).

Stroke patients had lower contralesional HGS compared to controls (-202.0 ± 57.86 N, $F_{1,71}=30.13$, $p<0.0001$, ANOVA) without improvement over time ($F_{3,71}=0.14$, $p_{\text{BOX}}=0.7096$, interaction term $F_{3,71}=0.33$, $p_{\text{BOX}}=0.5692$, ANOVA).

According to the ANOVA model, there was no change over time in HGS on the ipsilesional side ($F_{3,71}=0.16$, $p_{\text{BOX}}=0.6888$, interaction term: $F_{3,71}=0.05$, $p_{\text{BOX}}=0.8168$) and no difference to the controls ($F_{1,71}<0.01$, $p=9805$).

Masseter muscle thickness

At T1, there was a significant difference in MMT between contra- and ipsilesional sides, but not between cases and controls [12] (Table 4).

During follow-up, this difference was no longer present. When comparing contra- and ipsilesional MMT, the ANOVA model demonstrated no change over time ($F_{3,144}=0.01$, $p_{\text{BOX}}=0.9178$, interaction term: $F_{3,144}=0.02$, $p_{\text{BOX}}=0.9017$) and no influence of the side (contralesional/ipsilesional) ($F_{1,142}=0.09$, $p=0.7610$).

The ANOVA model did not show a change in MMT on the contralesional side over time in the participants with follow-up ($F_{3,72}<0.01$, $p_{\text{BOX}}=0.9494$, interaction term $F_{3,72}=0.01$, $p_{\text{BOX}}=0.9427$) and no influence of stroke ($F_{1,71}=0.28$, $p=0.5974$). For the ipsilesional side there was also no change over time ($F_{3,72}=0.02$, $p_{\text{BOX}}=0.8848$, interaction term $F_{3,72}=0.01$, $p_{\text{BOX}}=0.9179$) and no influence of stroke ($F_{1,71}=0.01$, $p=0.9300$).

Discussion

Chewing efficiency and maximum lip restraining force

The current study demonstrates that the chewing efficiency of stroke patients, when compared to a control group with similar dental state, is reduced. This confirms and refines previous findings in the literature [4]. The high drop-out rate weakened the statistical power; the ANOVA model however confirmed a tendency for lower chewing efficiency within the patient group during the 2 years.

In healthy subjects, chewing efficiency is largely depended on the number of occluding units and thus the

total surface of posterior antagonist tooth contact [29]. Furthermore, maximum bite force, saliva flow rate, the function of lip, cheek, tongue and the soft palate play a distinct role [30, 31]. Six out of ten patients in this study underwent dental treatment during the first 12 months following stroke. Restorative measures were expected to improve masticatory efficiency but in this present cohort it was not the case. In contrast to the control group their chewing efficiency was not correlated to the number of occlusal units. This supports the hypothesis that the reduced chewing efficiency in stroke patients might rather be dominated by neurophysiological impairment of the oro-facial structures [32, 33] than the dental state [2].

Most of the current patients suffered from lesions in the region of the primary motor cortex M1 supposedly affecting the cortical facial representation area [34]. Although bilateral innervation of the lower facial muscles was suggested, the most frequent abnormality found in patients with central facial paresis is the contralesional loss of motor evoked potentials [35] which was indirectly confirmed by the current study. However, central paresis of the lower facial muscles is often discrete [32].

At T1, chewing efficiency could be significantly predicted from the maximum lip force [5], hence the latter might have essentially contributed to the poor mastication following stroke. In the current analysis, patients demonstrated a significant lower lip force than their controls with no significant improvement over the whole observation period. This confirms the findings from Hägg et al. [36], who employed a similar method to evaluate lip force as used in the present study. They reported mean lip forces of 7 N (0–27 N) in patients with central facial palsy which was significantly lower than in a healthy control group [36]. The current results suggest a recovery plateau that might, as a consequence, lead to a chronic disability when no specific rehabilitation measures are prescribed.

Even in chronic stroke patients, functional improvement can be achieved [37]; however, oro-facial training procedures are still scarce [36]. The current results suggest that conventional dental rehabilitation procedures with the goal to increase the number of occluding dental units as a unique intervention will not significantly improve the masticatory function in stroke patients. Strengthening and increasing the co-ordination of the facial and chewing muscles is more likely to improve the masticatory performance. Malnutrition is an issue with stroke patients [38], and a good chewing efficiency is a prerequisite for a better food comminution and a wider food choice [39].

Maximum voluntary bite force

The current study reveals contradictory evidence to the maximum bite force in stroke patients. The results at T1

support findings from earlier studies which show no significant differences in bite force either between the contra- and ipsilesional sides [10, 40–43] or between stroke patients and controls [10].

Furthermore, the present results suggest a considerable difference in the motor impairment between jaw adducting- and upper limb muscles following unilateral stroke. Whereas contralesional jaw closing forces were not reduced, the corresponding HGS was significantly compromised. This confirms findings of Kemppainen et al. [42], who detected significant side differences in finger grip force but not in maximum bite force, in their study involving 16 stroke patients with hemiparesis. The current study helps extending their findings through longitudinal observations. The different impairment patterns may be a result of dissimilarities in the cortical control of the jaw closing muscles and those of the upper limb. The contralateral primary motor cortex M1 is the main origin of fibre pathways ultimately descending to the spinal cord neurons which are engaged into unilateral hand movements [8]. In contrast, corticobulbar fibres project from the cortical masticatory area to trigeminal motor nuclei in the pons of both sides [6]. Using focal TMS of the cortical representation area of the masseter muscle, bilateral muscle evoked potentials can be induced, providing functional proof of bilateral corticobulbar projections to the trigeminal motoneuron pools. Contralateral responses are larger than those evoked in the ipsilateral muscle, suggesting a stronger contralateral projection [7].

The ANOVA model demonstrated in follow-up patients significantly higher forces on the contralesional side than in their controls. Jaw closing forces are generated through the jaw closing muscles and peak bite force measurements depend significantly on the dental state [44, 45]. The high number of occluding posterior teeth of the patients with follow-up might partly explain the results, although there was no significant difference in occluding units between patients and controls. However, six out of ten patients had teeth replaced in between T1 and T2, whereas the controls did not undergo any restorative treatments. This might have contributed to the difference in bite force, even though the chewing efficiency remained unchanged. It has to be born in mind that the assessment of unilateral biting forces is difficult, since the mandible is a solid bone and acts as a lever. Exact unilateral forces between teeth are difficult to measure, because activation of the contralateral jaw closing muscles will always influence ipsilateral forces.

Hand-grip strength and masseter muscle thickness

The current study revealed a significant weakness of the paretic hand and no significant improvement over time. Carin-Levy et al. [46] investigated longitudinal changes in 17 stroke patients. They did not detect differences in HGS of

the ipsilesional side and control patients, but significantly lower forces of the paretic hand with no significant improvement over 6 months [46]. Patients of the present study have been examined with a median delay of 40 days after stroke. It is known that most functional recovery is spontaneous or can be observed within the first 2 months after stroke [47]; hence this initial recovery is not considered in the present T1 assessments. During the subsequent 2-year observation period, no further significant improvement of HGS was noted. Patients might have adapted to their disability through using the contralesional arm as best as they can while compensating for functional deficits with the intact arm, which might have led to long-lasting learned non-use [48].

The results of the current study may allow comparing the morphological effects of unilateral stroke on the masseter muscle with literature findings on the upper limb muscles. In stroke patients, a significant muscle atrophy of the upper limb can be observed [13], which may be attributed to disuse or altered central neural innervation, yet morphological evidence is scarce. A recent systematic review combined results from four studies and reported significant lower lean tissue mass in the paretic compared with the non-paretic upper limb in patients at least 6 months post stroke [13].

As reported previously [12], a small, yet significant side difference in MMT was observed in the current study population at T1. After 6 months, this difference was no longer present, but it has to be borne in mind that at this stage the study might have been underpowered. However, Uematsu et al. [49], who compared the cross-sectional area of masseter and medial pterygoid obtained by CT in 24 hemiplegic stroke patients support the present findings. They also did not find significant differences between contra- and ipsilesional sides more than 3 months after stroke [49]. Morphological recovery of the contralesional masseter muscles may have occurred within the first 6 months. Therefore, stroke seems to affect the upper limb muscles and the masseter muscle differently which supports the functional results of the currents study on a morphological level.

Study strengths and limitations

Mortality is high after acute ischemic stroke, about 25 % of patients die within the first 30 days after the event, and about half of the stroke patients within 1 year [50]. One-month mortality rates for hemorrhagic stroke are even worse with almost 50 % [51]. Following rehabilitation only 50–60 % of stroke survivors will regain independence in activities of daily living [47, 50, 52, 53], with most improvement being seen in the first 6 months post stroke [11].

Thus, it is “disease-inherent” that drop-out rates are very high such as in the study of Carin-Levy et al. [46], who

reported on a 6-month observation period in stroke patients and showed drop-out rates similar to the current study (52 % to 88 % [46]).

Patient drop-out may be due to active refusal or passive loss to follow-up, such as death or severe impairment. Of the 21 patients who dropped out of the study, only six withdrew their consent and further three had moved abroad (active drop-out). The remaining 12 drop-outs were at the 6-months evaluation either dead, too disabled to participate or could not be traced down by means of the Swiss population registry and consequently have to be considered passive drop-outs.

The drop-out rate of the current study is 68 % which may preclude avoiding type II errors. This has to be considered an unavoidable, yet severe limitation. However, the current study seems the first report on longitudinal observations in stroke patients with regard to oro-facial impairment. Thus, no sample size calculation could be performed. Nevertheless, the final number of participants was sufficiently high to demonstrate significant differences in HGS, maximum lip force and a tendency for chewing efficiency.

Although an unexpected finding, drop-out could be predicted from poor dental state, low chewing efficiency and low maximum bite forces. These factors are symptoms of oral-phase dysphagia [2, 3, 54] and thus might predict quality of life or even mortality. Further research is needed to study in depth the role of oral health in stroke rehabilitation.

In the current study the NIHSS did not predict a follow-up, although it was described to be significantly associated with long-term functional outcome after stroke [55, 56]. This might be due to the high drop-out rate which might have impeded statistical significance.

Summary and conclusion

Impaired chewing efficiency and reduced lip force are quantifiable symptoms of impairment following stroke. In absence of functional rehabilitation, these symptoms seem not to improve. Rehabilitation after stroke should also seek to improve the strength and co-ordination of oro-facial muscles to improve chewing efficiency and help avoiding weight loss and malnutrition. Additionally it might be concluded that stroke affects the upper limb- and the masseter muscle differently on a functional and morphological level. Further research is needed to evaluate the predictive value of oro-facial parameters on functional outcome after stroke.

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