Upper and lower face apraxia: role of the right hemisphere

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Summary
The aim of this study was to evaluate face apraxia in left- and right-hemisphere-damaged patients both in the acute and chronic stage of their disease. Two newly devised tests that assess movements of the upper and lower face districts were employed. On the whole, the proportion of left-hemisphere-damaged patients showing face apraxia were 46 and 68% for upper and lower face, respectively. A substantial proportion of right-hemisphere-damaged patients also showed face apraxia, i.e. 44% upper face and 38% lower face. Concomitant variables such as general severity, locus of lesion, language or visuo-spatial impairments, presence of neglect, interval from stroke, peculiarity of clusters of items or scoring procedures did not account for these results. These findings suggest that face apraxia in some patients may affect movements of the upper face district and that the right hemisphere plays a significant part in both upper and lower face praxis.

Keywords: face apraxia; face movements; stroke; cranial nerves

Abbreviations: ANOVA = analysis of variance; R(L)/pts = patients with stroke in right (left) hemisphere

Introduction
Apraxia generally refers to the impaired ability to perform movements upon request in both an experimental setting and in daily life (Cubelli and Della Sala, 1996). It affects movements either when asked to use or mime the function of an object or when required to imitate meaningful or meaningless gestures (De Renzi and Faglioni, 1999). Apraxia can also affect gestures performed automatically in everyday life. Movement disorders observed in apraxics are not explained by elementary motor, cerebellar or sensory deficits. Rather, the term apraxia refers to a variety of disorders that are named after either the regions of the body affected, or the testing procedures employed or the theoretical framework of reference adopted. The focus of this paper is on face apraxia.

Face apraxia
Jackson was the first to observe a relationship between face apraxia and aphasia: ‘there are in some cases of loss of speech other inabilities; the most significant are that a patient cannot put out his tongue when he tries, or execute other movements he is told’ (Jackson, 1879 p. 319, see also 1932). Since then, face apraxia has been reported as almost exclusively resulting from lesions of the left hemisphere, often in association with non-fluent aphasia (Moutier, 1908; Nathan, 1947; Alajouanine and Lhermitte, 1960; De Renzi et al., 1966; Poeck and Kerschensteiner, 1975; Watamori et al., 1981; Kertesz, 1985; Square-Storer and Roy, 1989). This latter association is probably due to anatomical contiguity (Tognola and Vignolo, 1980; De Renzi, 1989). Face apraxia should be kept separate from ‘apraxia of speech’ (‘aparaxie de la parole’ or ‘aphemia’; Broca, 1861; Darley, 1968, quoted by Lebrun, 1989), and from ‘pure anarthria’ (‘anarthrie pure’ of Marie, 1907). These latter conditions are selective disturbances of word articulation rather than of mouth movements (for a review, see Lebrun, 1994; Square et al., 1997).

Testing face apraxia
Despite its high prevalence (Pramstaller and Marsden, 1996) and a discussion of it in virtually all neuropsychology
textbooks, there are only a few formal tests devised to assess oral apraxia (e.g. Kokmen et al., 1998). Short, informal and non-standardized test batteries (e.g. Liepmann, 1900, cited in Brown, 1988; Kertesz, 1985; Hodges, 1994; Lezak, 1995) are often proposed to assess different aspects of apraxia. They generally include the use of objects (e.g. the ability to suck from a straw), the performance of symbolic gestures (e.g. to blow a kiss) or other oral movements either on command or on imitation (e.g. ‘show your teeth’). These batteries comprise a small number of items, which are scored as pass/fail; rarely are normative data available (Lezak, 1995). The 10-item test of oral apraxia proposed by De Renzi et al. (1966) has been standardized and provided with norms (Spinnler and Tognoni, 1987), though the different items were not weighted according to their relative difficulty. Moreover, as is common practice, this test includes only items assessing the movements of the lower part of the face (mouth and throat).

**Apraxia of upper and lower face movements**

Since Jackson’s (1879) first observation and subsequent early reports (e.g. Wilson, 1908), face apraxia has been generally equated with ‘oral apraxia’ (Roy and Square, 1985; De Renzi, 1989), i.e., the inability to perform skilled movements of the lips, cheeks and tongue (Rogers, 1996).

The view that equates facial apraxia with oral apraxia contrasts with earlier reports of patients with facial apraxia who also showed deficits of movements of the upper part of the face (eyes and eyebrows). Jackson alluded to disturbances of intentional eye movements that aphasic patients would present when asked to move their eyes in a given direction, in the absence of overt oculomotor paralysis (Jackson, 1866). Subsequently, Liepmann reported the case of a 48-year-old man, patient T, who, after a left hemisphere stroke, showed oral apraxia and disturbances of ‘gaze motion’ (Liepmann, 1900; for further discussion see Lange, 1936). Similarly, Lewandowsky (1907), Mingazzini and Ciarla (1920), Pinéas (1924) and Bonhöffer (1914) each reported one case of apraxia involving impairment of the ability to close the eyelids. Lebrun reported anecdotally the difficulty that some facial apraxics have in raising their eyebrows on command (Lebrun, 1994). Items assessing apraxia of the upper face were also listed by De Ajuriaguerra and Hécaen in their apraxia test battery (De Ajuriaguerra and Hécaen, 1964). Interestingly, in the few observations of ‘lid-apraxia’ following focal damage, the lesion was in the right hemisphere (Johnston et al., 1989). A systematic investigation of upper face apraxia in a group study with a psychometrically sound measure has never been carried out.

**Role of the right hemisphere in face apraxia**

A few single case studies suggest that facial apraxia may appear following lesions in the right hemisphere (Kleist, 1934; Kramer et al., 1985; Mani and Levine, 1988; Marchetti and Della Sala, 1997). In contrast, results from the only systematic group study of oral apraxia available in the literature indicate that this disorder is extremely rare, if at all present, following right hemisphere lesions (De Renzi et al., 1966). Whether right hemisphere lesions play a part in causing apraxia is debatable (for further discussion see De Renzi, 1989; Marchetti and Della Sala, 1997; De Renzi and Faglioni, 1999).

The aim of the present study was to investigate the role of the left and right hemisphere in praxis of movements of the lower (mouth, tongue and throat) and upper (eyes and eyebrows) cranial nerve districts. The performance of acute and chronic left and right-hemisphere-damaged stroke patients is reported. Face apraxia was assessed by means of two new tests assessing upper and lower face apraxia, devised to minimize some of the psychometric problems considered above. These tests are reported herewith as an Appendix.

**Face apraxia in acute patients**

**Methods**

**Participants**

A consecutive series of stroke patients admitted from September 1997 to June 1998 as in-patients to the Neurology Ward of the S. Paolo Hospital in Milan were recruited. Inclusion criteria were as follows: CT-demonstrated unilateral ischaemic or haemorrhagic brain lesion affecting supratentorial brain regions that were not due to vascular malformations or aneurysms; no previously diagnosed neurological or psychiatric diseases, including earlier strokes or transient ischaemic attacks; no concomitant extracerebral metabolic impairments hampering their cognitive abilities; overall severity should allow testing, in particular patients should not show any sign of head or eye deviation to the side of the lesion; an interval from stroke not exceeding 30 days. Fifty patients were eligible to enter the study. Thirteen patients, nine with a right and four with a left stroke, had to be excluded for the following reasons: seven (all with cortical lesions) were in a stuporous state or were clinically too severe to allow reliable neuropsychological testing, one could not speak or understand Italian, and five refused testing. None of the patients had to be excluded because the test was considered too burdensome or too long.

Thirty-seven patients, 22 (eight men, 14 women) with a stroke in the left hemisphere (L/pts) and 15 (seven men, eight women) with a right stroke (R/pts), participated in the study. They all gave informed consent and were not paid for taking part in the experiment. All but two patients were right-handed and scored at ceiling in the Edinburgh Handedness Inventory (Oldfield, 1971). Of the two left-handed patients, one was R/pt (who scored 2/12) and one L/pt (who scored 9/12). To assess their overall clinical severity, all patients underwent a Standardized Neurological Examination (Bisiach et al., 1983), which included assessment of strength and tactile perception in both upper and lower limbs as well as visual field, subdivided into upper and lower quadrants. The score for each of the items ranges from zero (no impairment)
Face apraxia

Fig. 1 Percentage distribution of the scores achieved by L/pts and R/pts in the Lower Face Apraxia Test.

Table 1 Demographic, neurological and psychometric features of the acute brain-damaged patients

<table>
<thead>
<tr>
<th></th>
<th>L/pts (n = 22)</th>
<th>R/pts (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Range</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>Age</td>
<td>47–85</td>
<td>50–77</td>
</tr>
<tr>
<td>Education (years)</td>
<td>2–18</td>
<td>5–18</td>
</tr>
<tr>
<td>Days from stroke</td>
<td>2–15</td>
<td>6–30</td>
</tr>
<tr>
<td>Neurological Exam.</td>
<td>0–16</td>
<td>0–14</td>
</tr>
<tr>
<td>Token Test (0–36)</td>
<td>5–35</td>
<td>27–33</td>
</tr>
<tr>
<td>Line Orientation (0–30)</td>
<td>11–30</td>
<td>0–30</td>
</tr>
</tbody>
</table>

Anatomical templates
Lesions were mapped onto standard templates following the procedure described by Damasio and Damasio (1989). First, the angle of incidence in which tomographical cuts were obtained was determined; this was done by taking into account the relative position of anatomical, cerebral and bone landmarks. The set of best-fitting templates was then chosen and each patient’s lesion charted on the appropriate templates using a x/y plotting approach.

Finally, all individual templates were reported on four sets of templates subdividing the right and left-hemisphere-damaged sample according to the presence/absence of apraxia in the upper or lower face district. For the sake of clarity we adopted the most frequent CT inclination (orbito-meatal) as the standard template.

Tests
Each participant was tested with the Upper and the Lower Face Apraxia Tests, using item difficulty weighted scores. Administering procedures, scoring methods and normative data are reported in the Appendix. For the purpose of this study the more conservative cut-off score, i.e. the outer tolerance limit, was employed to determine impaired performance in both tests.

The assessment also included a verbal comprehension task, the Token Test (score range: 0–36, cut-off: 26.5) (De Renzi and Faglioni, 1978), as a measure of language impairment, and a visuo-spatial task, the Judgement of Line Orientation Test (score range: 0–30, cut-off: 19) (Benton et al., 1978), as an index of visuo-spatial impairment. Norms are available for both tests (Benton, et al., 1983; Spinnler and Tognoni, 1987).

Concomitant variables
Means and standard deviations of the performances of the two groups are shown in Table 1. Two one-way ANOVAs
were run to compare their scores on the Token Test and the Judgement of Line Orientation. As expected, the L/pts had greater language impairment. The score difference between the two groups on the Token Test was statistically significant \(F(1,35) = 7.373, P < 0.01\). Twelve (55%) L/pts performed below cut-off versus one R/pt (7%) only. There was no indication of a position preference in the choices of the tokens in the R/pts.

In the Judgement of Line Orientation Test, the mean score of the R/pts was significantly lower \(F(1,35) = 14.818, P < 0.001\) than that of the L/pts, implying a greater visuospatial impairment. Eleven (73%) R/pts, but only five (23%) of the L/pts, performed below cut-off in this visuo-spatial task. There was no indication of a position preference in the choices of the lines in the R/pts.

The two groups did not differ \(F(1,35) < 1\) in terms of Standard Neurological Examination score.

**Lower Face Apraxia Test**

Figure 1 shows the distribution of the patients’ scores. The percentage of patients scoring below cut-off (i.e. 400) was slightly higher in the L/pts damaged group \(n = 13, 59\%\) than in the R/pts group \(n = 7, 47\%\). The R/pt with left visual extinction was not apraxic.

An analysis of variance showed that there was no significant difference \(F(1,35) = 2.550, \text{n.s.}\) between the performance of the L/pts (mean 358.0, SD 90.1) and that of the R/pts (mean 396.2, SD 23.9).

In the L/pts group, Spearman correlation analyses between the scores on the Lower Face Apraxia Test and the Token Test, the Judgement of Line Orientation Test and the Standardized Neurological Examination all yielded non-significant coefficients \((0.19, 0.04 \text{ and } 0.18, \text{respectively})\). Similarly, low and non-significant correlations were found in the R/pts \((-0.04, 0.26 \text{ and } -0.35, \text{respectively})\).

To investigate the link between apraxia and aphasia, mentioned in the introduction, contingency between the performance in the Token Test and the Lower Face Apraxia Test was evaluated in the L/pts only. The number of L/pts scoring above or below cut-off in both tests is shown in Table 2A: three aphasic L/pts were not apraxic while four apraxics were not aphasic, Cohen’s \(\kappa = 0.32, \text{n.s.}\). To ascertain whether deficits in visuospatial processing may be held partly responsible for the poor performance of R/pts in the apraxia test, contingency between the performance in the Judgement of Line Orientation Test and Lower Face Apraxia Test was evaluated: Cohen’s \(\kappa\) yielded a value of 0.22, n.s. (see Table 2C).

**Upper Face Apraxia Test**

Figure 2 shows the distribution of the patients’ scores. The percentage of patients scoring below cut-off (38.43%) was higher in the R/pts \(n = 7, 47\%\) than in the L/pts \(n = 6, 28\%\). The R/pt with left visual extinction was not apraxic.

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Number of acute L/pts performing above or below cut-off score in the Token Test and Lower (A) and Upper (B) Face Apraxia Tests. Number of acute R/pts performing above or below cut-off score in the Line Orientation Test and Lower (C) and Upper (D) Face Apraxia tests</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>(A) Lower Face Apraxia Test</strong></td>
<td></td>
</tr>
<tr>
<td>L/pts with apraxia</td>
<td>L/pts without apraxia</td>
</tr>
<tr>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td><strong>(B) Upper Face Apraxia Test</strong></td>
<td></td>
</tr>
<tr>
<td>L/pts with apraxia</td>
<td>L/pts without apraxia</td>
</tr>
<tr>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td><strong>(C) Lower Face Apraxia Test</strong></td>
<td></td>
</tr>
<tr>
<td>R/pts with visuo-spatial impairment</td>
<td>R/pts without visuo-spatial impairment</td>
</tr>
<tr>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td><strong>(D) Upper Face Apraxia Test</strong></td>
<td></td>
</tr>
<tr>
<td>R/pts with visuo-spatial impairment</td>
<td>R/pts without visuo-spatial impairment</td>
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<tr>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td>0</td>
<td>4</td>
</tr>
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</table>

There was no significant difference \(F < 1\) between the performance of the L/pts (mean 40.3, SD 7.5) and the R/pts (mean 39.9, SD 4.8).

In the L/pts group, Spearman correlations between the scores on the Upper Face Apraxia Test and the Token Test, the Judgement of Line Orientation Test and the Standardized Neurological Examination resulted in the following coefficients: 0.58, \(P < 0.01\); 0.47, \(P < 0.03\); and –0.30, n.s., respectively. Low non-significant correlations were found in the R/pts group \((0.14, 0.01 \text{ and } 0.29, \text{respectively})\). The number of L/pts scoring above or below cut-off in the Token Test and the Upper Face Apraxia Test are shown in Table 2B: seven aphasic L/pts were not apraxic while two apraxics were not aphasic, Cohen’s \(\kappa = 0.16, \text{n.s.}\). Cohen’s coefficient (Table 2D) between the Judgement of Line Orientation Test
Face apraxia

Table 3 Percentages of acute L/pts and R/pts performing below cut-off in the Upper Face Apraxia Test, in the Lower Face Apraxia Test and in both tests, and those without apraxia

<table>
<thead>
<tr>
<th></th>
<th>L/pts (%)</th>
<th>R/pts (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 22)</td>
<td>(n = 15)</td>
</tr>
<tr>
<td>Pts with upper face apraxia only</td>
<td>5 (1)</td>
<td>20 (3)</td>
</tr>
<tr>
<td>Pts with lower face apraxia only</td>
<td>36 (8)</td>
<td>20 (3)</td>
</tr>
<tr>
<td>Pts with both upper and lower face apraxia</td>
<td>23 (5)</td>
<td>27 (4)</td>
</tr>
<tr>
<td>Pts without apraxia</td>
<td>36 (8)</td>
<td>33 (5)</td>
</tr>
</tbody>
</table>

Frequencies are given in parentheses.

and the Upper Face Apraxia Test in the R/pts was significant ($\kappa = 0.48, P = 0.029$).

Concordance between performance in the Upper and Lower Face Test

The degree of concordance of the performance of individual L/pts and R/pts on the two tests of face apraxia is shown in Table 3. Fifteen patients (41%) failed only one of the two tests.

Localization of face apraxia

Figure 3 gives a schematic representation of the site of the lesion in the L/pts and R/pts, grouped according to the absence or presence of lower and upper face apraxia. The templates show that several lesions were subcortical, both in the apraxic and non-apraxic subgroups. There is no apparent difference in gross localization and size of the lesions between apraxic and non-apraxic patients, between apraxic R/pts and L/pts, or between the localization of lesions resulting in upper and lower face apraxia.

Comment

The finding of a high proportion of R/pts showing apraxia was unexpected. To check whether the weighted scoring system might have spuriously increased the sensitivity of the tests, the data of the acute patients were also analysed using a traditional pass/fail scoring system, attributing one point to each item well executed and zero to a failure. The trend was similar to that obtained using the rank scoring system showing lack of significant difference between the performance of L/pts and R/pts, both in the Lower Face ($F(1,35) = 1.446, n.s.$) and in the Upper Face ($F(1,35) = 0.253, n.s.$) Apraxia Tests.

Furthermore, it should be noted that the asymmetry observed in several neuropsychological syndromes is not as clear-cut in the acute stage of the disease as it is when some time from the stroke has elapsed. For instance, the predominance of right-sided lesions in visuo-spatial neglect in chronic patients is not as obvious in acute stroke patients. Stone et al. found that neglect was equally common in R/pts and L/pts 3 days after the stroke, whereas 3 months post-stroke the frequency of neglect, as expected, was far higher in R/pts (Stone et al., 1991). Similarly, De Renzi et al. found that early motor impersistence tended to recover after 3 months (De Renzi et al., 1986). The findings of our study could reflect the relatively short interval post-stroke. The correlation between the scores in the Lower and Upper Face Apraxia Tests and the stroke/assessment interval was 0.0002 and –0.24 for the L/pts, and –0.12 and 0.31 for the R/pts,
Fig. 3 Templates depicting lesion locations derived from the superposition of CT scans of R/pts and L/pts on standard templates. (A) Non-apraxic patients; (B) lower face apraxia only; (C) lower and upper face apraxia; (D) upper face apraxia only. The right hemisphere is on the right of the figures.
which were all far from significant. However, the patients entering this experiment have all been tested within the acute phase of their stroke (i.e. within 30 days). Therefore, to verify whether R/pts might show a steeper slope of recovery than L/pts, the same face apraxia tests were administered to a group of chronic patients (stroke/testing interval > 3 months) in a subsequent experiment. Had we not found any chronic R/pts showing apraxia, the findings from the experiment with acute patients would be accountable for in terms of left hemisphere compensation. Patients entering this second experiment were tested by a different examiner trained to give the test battery but blind to the results of the first experiment. The choice of a different examiner was made to test for possible bias due to an individual examiner.

### Face apraxia in chronic patients

#### Methods

#### Participants

A series of chronic stroke patients were recruited. Inclusion criteria remained the same as for the acute patient group. Additional inclusion criteria were as follows: patients should be right-handed (Oldfield, 1971) and at least 3 months should have elapsed from the time of stroke.

Forty-one patients were eligible to enter the study. Three patients had to be excluded: one because of evidence of multiple lesions and infratentorial lesions, one because of illiteracy and one showing aphasia following a right lesion. Thirty-eight patients, 19 (eight men, 11 women) with a stroke in the left hemisphere (L/pts) and, 19 (13 men, six women) with a right hemispheric stroke (R/pts), participated in the study. To assess their overall clinical severity, all patients underwent the same Standardized Neurological Examination employed with the acute patients. The demographic and clinical features of the chronic patient group are summarized in Table 4.

A series of one-way ANOVAs showed that right- and left-hemisphere-damaged patients were reasonably well matched for age [F(1,36) < 1, n.s.] and education [F(1,36) < 1, n.s.]. The two samples did not differ in the interval between the time of stroke and the time of testing [F(1,36) = 2.138, n.s.].

### Tests

Patients were tested with the testing material used to assess the acute patient group. Additional testing included the Line Bisection Test (Schenkenberg et al., 1980) and the Letter Cancellation Test (Diller and Weinberg, 1977) to test for the presence of unilateral visuo-spatial neglect. Two R/pts showed evidence of neglect.

### Results

#### Concomitant variables

ANOVA showed that the mean Token Test score of the L/pts was significantly lower than that of the R/pts [F(1,36) = 34.991, *P* < 0.0001] while the converse was true for the Judgement of Line Orientation Test [F(1,36) = 11.815, *P* < 0.002]. The neurological severity of the two groups was comparable [F(1,36) < 1].

#### Lower Face Apraxia Test

The percentage of patients scoring below cut-off (i.e. 400) was higher in the L/pts damaged group (n = 15, 79%) than in the R/pts (n = 6, 32%). An ANOVA showed that there was a significant difference [F(1,36) = 18.139, *P* < 0.0001] between performance of the L/pts (mean 300.7, SD 99.53) and that of the R/pts (mean = 406.6, SD 43.0). Four of the L/pts scored above cut-off on the Token Test and did not have aphasia, one of them fell below cut-off on the Lower Face Apraxia Test. The two R/pts with neglect scored above cut-off on this test.

#### Upper Face Apraxia Test

The percentage of patients scoring below cut-off was higher in the L/pts damaged group (n = 13, 69%) than in the R/pts (n = 8, 42%). There was no significant difference [F(1,36) = 1.387, n.s.] between performance of the L/pts (mean = 30.9, SD 12.2) and the R/pts (mean = 35.6, SD 12.3). Two of the L/pts without aphasia scored below cut-off on the Upper Face Apraxia Test. The two R/pts with neglect scored above cut-off on this test.

#### Comparison between acute and chronic state patients

A series of ANOVAs revealed that the chronic patients were significantly younger than the acute ones [F(1,73) = 6.410, *P* < 0.02], mean age 62.21 (SD 10.34) and 68.51 (SD 11.21) years, respectively. Their neurological impairment was more severe; the difference in the Standard Neurological Examination scores between the acute (mean 5.08, SD 4.23) and chronic (mean 6.92, SD 4.61) group approached significance [F(1,73) = 3.232, *P* = 0.07]. Moreover, there was a disparity between patients with subcortical lesions.
only [14/37 (38%) in the acute group versus 9/38 (24%) in the chronic group].

Lower face apraxia. A two-way ANOVA was run to verify whether there was any interaction between disease state (acute/chronic) and lesion side (right/left). This analysis showed no overall effect of disease state \(F(1,71) = 1.838, \text{n.s.}\), a significant effect of lesion side \(F(1,71) = 17.385, P < 0.0001\), with L/pts achieving lower scores than the R/pts, and an interaction between lesion side and disease state that was close to significance \(F(1,71) = 3.837, P = 0.0540\). A log-linear frequency analysis of right and left patients in the acute and chronic state falling above and below cut-off showed no significant differences between the groups \(\chi^2 = 2.649, \text{n.s.}\).

Upper face apraxia. A two-way ANOVA was run to verify whether there was any interaction between disease state (acute/chronic) and lesion side (right/left). This analysis showed a significant effect of disease state \(F(1,71) = 8.893,
with chronic patients scoring lower than acute patients but showing greater variability as indicated by larger standard deviations, no effect of lesion side \( [F(1.71) < 1, \text{n.s.}] \) and no significant interaction between lesion side and disease state \( [F(1.71) = 1.217, \text{n.s.}] \). A log-linear frequency analysis of right and left patients in the acute and chronic state falling above and below cut-off showed no significant differences between the groups \( (\chi^2 = 3.306, \text{n.s.}) \).

**Comment**

The predictions of the experiment with chronic patients were orthogonal. On one hand, chronic patients could show the same pattern as the acute patients, i.e. no difference in performance between R/pts and L/pts. On the other hand, a substantial proportion of the L/pts could still show apraxia, whereas the R/pts would all perform above cut-off, as predicted from the literature. The actual findings are somewhere in between. Indeed, more chronic L/pts than R/pts showed either upper or lower face apraxia. However, a discernible number of R/pts were still apraxic after a longer interval from stroke.

Some caveats should be considered. The period of recovery might not be enough for compensation of right hemisphere apraxia to be complete. However, the correlation between apraxia scores and interval from onset was small and non-significant for both lower and upper face (0.14 and 0.13, respectively). The two samples (acute and chronic patients) were not perfectly comparable in severity (the chronic group was on the whole more severe) nor for the locus of lesion (more cortical cases within the chronic group). Although both these variables might have partly influenced the outcome, they cannot alter the fact that some acute and chronic R/pts do show upper or lower face apraxia.

A further possible interpretation of the difference between failing particular items, for the errors are scattered across the distribution (see Fig. 1). Indeed, the mean ranking of the items common to both test-batteries is rather high (18.2), implying that the items used by De Renzi et al. (1966) were among the easiest of our test.

The high sensitivity of our test might have carried the unforeseen effect of the presence of clusters of items selectively biased to the consequences of left or right-hemisphere damage. To address this issue, errors of the acute and chronic samples to each individual item were totalled and the resulting data were plotted for both the Upper Face and Lower Face Tests (see Fig. 4A and B). The graphs demonstrate that the apraxia of R/pts is not due to them failing particular items, for the errors are scattered across the whole test. Therefore, we could exclude the charge that our findings were due to a potential bias of considering apraxic patients who failed individual items that are particularly sensitive to the effect of right hemisphere lesion.

**Discussion**

Acute and chronic stroke patients with a lesion either in the left or in the right hemisphere were evaluated with two new tests of face apraxia assessing the upper and lower face movements, respectively. A summary of the main findings is shown in Fig. 5.

Together with the expected performances of L/pts showing lower face apraxia, two other sets of data are of interest: the substantial proportion of R/pts showing apraxia and the high proportion of patients showing upper face apraxia. While the latter finding is interesting and had not been reported in the literature, the role of the right hemisphere lesions in face apraxia was unexpected. The above findings will be discussed in turn.
Apraxia in L/pts
Approximately 70% of all L/pts participating in the study performed below cut-off on the Lower Face Apraxia Test. These findings lend further support to the evidence that attributes a key role to the left hemisphere in praxis for the movements of the lower face (e.g. De Renzi et al., 1966; Rothi et al., 1994). Some of the L/pts performed well on the Lower Face Apraxia Test but poorly on the Token Test; some other L/pts showed the converse profile. This double dissociation extends to face apraxia (see the case of the patient showing face apraxia but not aphasia reported by Sittig, 1931) the distinctness of praxis from language observed in studies of limb apraxia (Selnes et al., 1982; Papagno and Della Sala, 2000).

Role of the right hemisphere in lower face apraxia
The findings of the present study indicate that the right hemisphere plays a part in face praxis. Almost half of the acute R/pts and a third of the chronic R/pts performed below cut-off in the Lower Face Apraxia Test. Their poor scores in this test could not be explained in terms of general neurological severity nor in terms of size or location (e.g. Brodmann area 8) of the lesion (Fig. 3B and C). Similarly, their poor performance on the apraxia test could not be easily accounted for by spatial impairment, since their performance on the Judgement of Line Orientation Test correlated quite poorly with that on the apraxia tests. Poor performance in the praxis tests could not be accounted for by neglect, since there was no patient with neglect in the acute sample and the chronic patients with neglect showed no apraxia.

The nature of the role of the right hemisphere in lower face apraxia, and indeed in apraxia in general, is far from clear. The legacy left by Liepmann’s seminal paper, in which the author observed limb apraxia only in those patients with a lesion in the left hemisphere in a series of 89 brain-damaged patients (Liepmann, 1920), had a strong influence on authors subsequently investigating apraxia (e.g. Junque et al., 1986).

The influence of the presumption of a left sided dominance in praxis is even more overt in studies of oral apraxia, perhaps due to its frequent association with language impairments. This argument has been supported by the evidence that oral apraxia is more frequent than limb apraxia in crossed aphasics (Brown and Code, 1987; Alexander et al., 1989). Indeed, the proportion of limb apraxia versus face apraxia reported in review papers is 17 versus 52% (Castro-Caldas et al., 1987), 11 versus 46% (Alexander et al., 1989) and 17 versus 34% (Coppens and Hungerford, 1998). However, double dissociations between face apraxia and language deficits can also be culled from the literature (Heilman et al., 1974; Fisher et al., 1991; Rey et al., 1994).

In group studies of oral apraxia, right-hemisphere-damaged patients have been used as controls, on the assumption that they were exempt from apraxia (e.g. De Renzi et al., 1966). Several authors have taken the worst score achieved by R/pts as the cut-off score to diagnose apraxia in L/pts. If one follows this procedure it would be particularly arduous to support any role of the right hemisphere in causing apraxia.

However, findings from neuroimaging studies give support to the theory of an involvement of the right-hemisphere damage in apraxia. For instance, Foster et al. assessed 17 right-handed patients affected by Alzheimer’s disease with PET while the participants were performing a limb apraxia imitation test (Foster et al., 1986). These authors found that the right parietal cortex was activated during the task. Moreover, an extensive literature exists, in which observations of loss of dexterity of the ipsilesional hand in both left and right-hemisphere-damaged patients has been reported (Jebsen et al., 1971; Spaulding et al., 1988; Desrosier et al., 1996). However, in the R/pts this impairment is less frequent and is only observed soon after the stroke (Sunderland et al., 1999). Similarly, Harrington and Haaland (1992) reported a deficit in parsing serial postures in R/pts.

A further argument pointing to an active role of the right hemisphere in praxis control comes from studies of patients who have undergone commissurotomy, which have shown that ‘both hemispheres can independently exert a praxis control’ (Berlucchi and Aglioti, 1999, p. 658). Commissurotomy patients show the expected left hand apraxia (De Renzi and Faglioni, 1999). This evidence has often been proffered as support for the existence of a praxis centre, lateralized to the left hemisphere. However, left hand apraxia surfaces only on verbal command, not on imitation (Zaidel and Sperry, 1977; Gazzaniga, 1983), which indicates that when the right hemisphere understands what is required, it can be efficient in executing praxis commands. The role of the right hemisphere is even more overt in the case of face movements that are not impaired by commissurotomy (Zaidel and Sperry, 1977).

Our test involves only one modality of item presentation, i.e. imitation. Haaland and Flaherty reported that the L/pts and R/pts did not differ in their performance in a limb apraxia test when they were asked to imitate intransitive gestures (performance without objects) (Haaland and Flaherty, 1984). However, when the same patients were tested with a transitive version of the same task, i.e. they were asked to pretend to use an object, a clear hemispheric difference emerged: the L/pts performed more poorly than the R/pts. Therefore, Haaland and Flaherty maintained that the results of the studies investigating the link between apraxia and the side of the lesion could be biased by the modality of stimulus presentation. Moreover, some cases have been reported of patients who could imitate limb gestures yet could not comprehend or discriminate between them (Rothi et al., 1986). This crucial evidence led Rothi et al. to develop a cognitive model of limb praxis which could account for the aforementioned and other discrepancies observed in apraxic patients (Rothi et al., 1991, 1997; see also Cubelli et al., 2000).
Recently, Goldenberg proposed that R/pts might fail finger posture tasks for reasons different from those of L/pts (Goldenberg, 1999). Whilst L/pts show a conceptual deficit, R/pts would fail the task due to visuo-perceptual processing deficits. In Rothi et al.'s frame of reference (Rothi et al., 1991, 1997), this is akin to stating that R/pts might fail praxis tests because of faulty visuo-spatial processing. From the present data, some indication that this might be the case comes from the observation that some patients in our sample showed associations between apraxia on both upper and lower face tests and poor performance on the Line Orientation Test (Table 2C and D).

It could be suggested also that motor impersistence (Fisher, 1956), frequently reported in R/pts (Maas and Sitting, 1929; Joynt et al., 1962; Levin, 1973; Nutt, 1977; Colombo et al., 1982; De Renzi et al., 1986), might account for some of the observations summarized above. Motor impersistence is defined as the inability to sustain movements initiated on command and by some authors is considered a ‘form of apraxia’ (Benton et al., 1983; see also Schilder, 1924; Zutt, 1950). The errors shown by the patients reported in the present study cannot be interpreted as motor impersistence because the patients were not instructed to hold any movement for a given time (Berlin, 1955; Fisher, 1956; De Renzi et al., 1986). Finally, errors cannot be accounted for by simultanapraxia (Sakai et al., 2000), because patients were never asked to perform two motor acts simultaneously.

Finally, several cases have been reported of right-handed individuals who show an impairment of gesture production after a lesion in the right hemisphere, so called ‘crossed apraxics’ (e.g. von Monakow, 1914; Rapcsak et al., 1987; Motomura et al., 1990; Alexander and Annett, 1996; Marchetti and Della Sala, 1997; Raymer et al., 1999). Some case reports of ‘crossed oral apraxia’ have also been published. Hartmann reported the case of a 55-year-old man with oral apraxia, whose post-mortem demonstrated a right frontal haemorrhagic stroke (Hartmann, 1907). Kleist reported two cases of right-handed war-wounded patients with lesions confined to the right inferior precentral gyrus, who showed oral apraxia (Kleist, 1934). More recently, a few more cases have been reported (Kramer et al., 1985; Mani and Levine, 1988; Rapcsak et al., 1987; Marchetti and Della Sala, 1997; Papagno and Della Sala, 2000). In a group study with epileptic patients, Kolb and Milner observed lower face apraxia in a sequence imitation test following either left or right corticectomy (Kolb and Milner, 1981). Similarly, Alexander et al. reported that 13 out of 18 right hemisphere stroke patients had problems with an item from their face apraxia test, namely ‘frowning as if angry’ (Alexander et al., 1992). Finally, Foundas et al. observed that two out of nine right-handed patients showed transient oral apraxia while undergoing the Wada amytal test on the right side (Foundas et al., 1995).

Though the role of the right hemisphere in praxis is usually considered trifling, several authors have postulated a bi-hemispheric representation for the production of skilled limb movements (e.g. Lange, 1936; Haaland and Flaherty, 1984; Kertesz et al., 1984; Berlucchi and Aglioti, 1999, p. 248). De Renzi maintained that ‘while the dominance of the left hemisphere in movement planning is likely to be the rule, the degree of its intensity is variable from subject to subject and there can be cases of equipotentiality’ (De Renzi, 1989). This hypothesis echoes that of Rose who, reporting a case of left apraxia in a left-hander following a callosal lesion, stated that both the left and the right hemisphere play a role in face movements (Rose, 1911). First Goldstein (1915) and later Mingazzini and Ciarla (1920) reconsidered the idea of a bilateral contribution to face movement praxis. Mingazzini and Ciarla also maintained that the individual contribution of each hemisphere might vary in different people. To support the view that indeed the right hemisphere does play a part in face movement praxis, they reported the case of a 75-year-old man, C.G., presenting with a left paresis and face apraxia, whose post-mortem showed the presence of an anterior callosal lesion. Some involvement of the right hemisphere in apraxia was suggested also by Lange (1936), translated in Brown (1988) who, discussing motor engrams, stated that ‘we cannot totally deny [their presence in] the right hemisphere despite the superiority of the left hemisphere’. Even Liepmann, who strongly believed in the dominance of the left hemisphere for praxis, did not reject the possibility that the right hemisphere might sometimes play a part: ‘Let us remember once and for all that the right hemisphere also takes part in eupraxis . . . ‘ (Liepmann, 1920, p. 531). Furthermore, recent case reports have demonstrated that the hemispheric side of praxis can be dissociated from the side of hand dominance (Marchetti and Della Sala, 1997; Raymer et al., 1999). Our findings subscribe to this concept, expanding it to the facial domain.

The discrepancy between the present findings and some of the earlier group studies (e.g. De Renzi et al., 1966) might be due to different inclusion criteria, i.e. acute versus more stabilized patients. Therefore, it could be entirely accounted for in terms of differential recovery of functions between R/pts and L/pts. However, this possibility could be discounted on the grounds that face apraxia was observed among the chronic R/pts. It cannot, however, be entirely dismissed at the present stage. First, the interaction between lesion site and state of disease indicates that face apraxia, at least lower face apraxia, recovers less in L/pts than in R/pts. Secondly, a cross-sectional comparison may not be the best approach to address this issue; only a longitudinal study would give a definite answer. Ochipa and Rothi (1989, quoted by Rothi et al., 1994) followed up a patient with right hemisphere lesion and showed that face apraxia recovered with time.

The fact remains though that a considerable proportion of chronic R/pts in our sample showed either lower or upper face apraxia or both. There is some evidence of patients in whom face apraxia persisted after a long interval from their stroke. Marchetti and Della Sala reported the case of a right-handed 63-year-old female general practitioner whose lesion encroached upon the right mesial frontal region (Marchetti
and Della Sala, 1997). She showed limb ideomotor apraxia coupled with oro-facial apraxia 8 years after her brain lesion, though no distinction was made between lower and upper face movements. Finally, Papagno and Della Sala recently reported the case of a 55-year old right-handed artist who still showed, 2 years after his right fronto-parietal stroke, a very severe upper and lower face apraxia both on imitation and on command (Papagno and Della Sala, 2000).

Upper face apraxia

The second aim of this paper was to assess movements of the upper face and to ascertain whether the prevalent role of the left hemisphere was to be sustained for the planning of these movements as well. Our data show, on the whole, about half of the L/pts and R/pts performed below cut-off on the Upper Face Apraxia Test.

Although the topic has never been systematically investigated, the observation of upper face apraxia following a brain lesion is not entirely new. Kleist described a 36-year-old man with multiple bilateral strokes who showed severe apraxia both for limbs and face, which included eyelid closure apraxia (Kleist, 1907). Riese reported the case of a left-handed patient with left hemisphere stroke and right hemiplegia who displayed apraxia of lid closure (Riese, 1930). Lewandowsky reported the case of R.L., a 64-year-old waiter, who, after a sudden left hemiplegia due to a right hemisphere stroke, permanently lost the ability to close his eyelids on command (Lewandowsky, 1907). R.L. also showed a transient inability to move his eyes leftwards on command, but retained automatic and reflex eyelid closure as witnessed by normal blinking, blink reflex and eyelid closure during sleep. Lewandowsky surmised that eye closure disorder was apraxic, and accordingly labelled it ‘Apraxie des Lidschlusses’ (lid-apraxia) (Lewandowsky, 1907). He maintained that a right hemisphere dominance was at work for these eyelid and oculomotor disorders.

Localization of apraxia

The analysis of the anatomical localization of the lesions (Fig. 3) does not provide critical differences linked to the presence or absence of face apraxia, or any apparent dissociation between upper and lower apraxia, or right and left hemisphere.

It appears that 45% of apraxia among acute patients had lesions limited to subcortical regions. This observation indicates that, as for limb apraxia (Della Sala et al., 1992; Pramstaller and Marsden, 1996), face apraxia can also be due to damage confined to deep brain structures sparing the cortex. Our crude mapping showed that some apraxic patients had isolated anterior lesions and that the parietal lobe was spared in several cases of upper or lower face apraxia. This is in agreement with previous literature on the localization of face apraxia (Tognola and Vignolo, 1980; Basso et al., 1987; Mintz, 1989; Raade et al., 1991; review Rothi et al., 1994). However, the results from the above mentioned group studies denied the possibility that posterior lesions could be held responsible for face apraxia; yet a few of our apraxic patients had lesions confined to posterior cortical structures. This result matches clinical observations (Benson et al., 1973; Geschwind, 1975; Heilman et al., 1983) as well as functional neuroimaging data (Kareken et al., 1998). Indeed, from the templates shown in Fig. 3 it emerges that a distributed neuroanatomical network in both hemispheres appears to be involved in face praxis (see also Raade et al., 1991; Kareken et al., 1998; Leiguarda and Marsden, 2000).

Final remarks

The classic view of face apraxia is rooted in two assumptions: the left hemisphere dominance and the existence of a specific praxic centre. This view runs into some difficulty when attempting to account for the data reported in this paper. The present findings support the notion of a more distributed left hemisphere network for face praxis and point to a strong involvement of the right hemisphere also. The hypothesis of a bilateral distribution of the network subserving face praxis poses the question of why patients with unilateral lesions become apraxic, which remains unanswered at present. It may well be that the left and the right hemisphere contribution to face praxis do not overlap, and therefore the respective deficits are not entirely compensated. Longitudinal analyses of patients’ performance and a qualitative account of their difficulties are needed to address the issue of a possible differential role of the right and left hemisphere (e.g. Platz and Mauritz, 1995; Goldenberg, 1999). This would possibly lead to the mapping of the respective roles of the two hemispheres within a cognitive framework (Rothi et al., 1991, 1997).

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Appendix

The Upper and Lower Face Apraxia Test

Description of the test

Two tests of facial apraxia have been devised, assessing the upper and the lower face movements, respectively. The Upper Face Apraxia Test included nine items, while the Lower Face Apraxia Test was made up of 29 items, according to the territory of the cranial nerves involved. The upper face items comprise movements subserved by the superior or inferior division of the facial nerve (VII) and by the three oculomotor nerves (III, IV and VI). The lower face items comprise movements subserved by the inferior division of the facial nerve, the motor component of the 3rd division of the trigeminal (V), glossopharyngeal, vagus and hypoglossal (IX, X and XII) nerves. Most of the items require a visual input only, though seven items in the Lower Face test (e.g. ‘whistle’) also have an acoustic component.

Test scoring

The items for the lower and upper face tests are listed in Tables A1 and A2, respectively. Each item was demonstrated by the examiner and the participant had to respond by imitation immediately after presentation. This procedure was employed in order to minimize the confusion between apraxic and verbal comprehension errors. Items were scored as pass or fail according to a set of rules derived from previous authors’ attempts to define errors in facial apraxia (De Renzi et al., 1966; Poeck and Kerschensteiner, 1975). In particular an item was considered ‘failed’ (i.e. apraxic) if the participant produced random and continuous amorphous movements (Square-Storer and Roy, 1989), if the response was preceded by protracted pauses, during which additional, unsolicited movements may be present (De Renzi et al., 1966), if the performances was scattered with conduites d’approche analogous to the word finding approach of some aphasic patients (Poeck and Kerschensteiner, 1975), or if the required movement was incomplete or not performed at all (Lange, 1936). Mirror movements were not considered as errors. The testee was never asked to hold a given movement for a period of time, therefore it is unlikely that an error due to motor impersistence could have been mistaken for apraxia. Of course, allowance was made for the paralysis of the lower half face contralateral to the brain lesion. The actual test proper was preceded by a run-in item (‘puff out both your cheeks’) repeated until the examiner was sure that the participant had understood the task.

For both the Upper and Lower Face Tests, scoring procedures based on the rank order score were employed, whereby the score for each item was weighted taking into account its relative difficulty. Relative difficulty was determined according to the total number of failures observed in the sample on each individual item. Items were ranked from the one, the most difficult, which yielded the largest number of failures (to which the rank score of 1.0 was assigned) to the one, the easiest, producing the smallest number of errors (to which the rank score of 25.0 was assigned) the one, the easiest, producing the smallest number of errors (to which the rank score of 25.0 was assigned). This ensured that if a patient were failing an item that was more likely to be failed by a large number of controls, his/her total score would not be affected disproportionately. Should, however, the patient fail in an item that is unlikely to be failed by controls, this will have a big impact on his/her total score. Rank scores for each item included in the Upper and Lower Face Tests are given in Tables A1 and A2, respectively. Items were given in the randomized order set forth in the tables. The total score ranges

Table A1 Items of the Lower Face Apraxia Test and relative ranking

<table>
<thead>
<tr>
<th>Item</th>
<th>Rank score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Open your mouth</td>
<td>25.0</td>
</tr>
<tr>
<td>2. Move the jaw left to right (and right to left) three times</td>
<td>10.0</td>
</tr>
<tr>
<td>3. Show your teeth</td>
<td>25.0</td>
</tr>
<tr>
<td>4. Push the tip of your tongue against the inside of your right cheek</td>
<td>12.0</td>
</tr>
<tr>
<td>5. Pass the tip of your tongue over your lower lip</td>
<td>25.0</td>
</tr>
<tr>
<td>6. Pass the tip of your tongue over your upper lip</td>
<td>17.5</td>
</tr>
<tr>
<td>7. Puff out your right cheek</td>
<td>3.0</td>
</tr>
<tr>
<td>8. Stick out your tongue to the left</td>
<td>17.5</td>
</tr>
<tr>
<td>9. Clench your jaws together (keeping lips shut)</td>
<td>17.5</td>
</tr>
<tr>
<td>10. Stick out your tongue</td>
<td>17.5</td>
</tr>
<tr>
<td>11. Push out your lower teeth (prognathism)</td>
<td>6.0</td>
</tr>
<tr>
<td>12. Push the tip of your tongue against the inside of your left cheek</td>
<td>10.0</td>
</tr>
<tr>
<td>13. Puff out your left cheek</td>
<td>4.0</td>
</tr>
<tr>
<td>14. Stick out your tongue to the right</td>
<td>17.5</td>
</tr>
<tr>
<td>15. Push your tongue against the inside of your lower lip</td>
<td>5.0</td>
</tr>
<tr>
<td>16. Whistle</td>
<td>7.5</td>
</tr>
<tr>
<td>17. Blow</td>
<td>25.0</td>
</tr>
<tr>
<td>18. Make your teeth chatter</td>
<td>17.5</td>
</tr>
<tr>
<td>19. Bite the inside of your right cheek</td>
<td>2.0</td>
</tr>
<tr>
<td>20. Smile</td>
<td>25.0</td>
</tr>
<tr>
<td>21. Pretend to give a kiss</td>
<td>25.0</td>
</tr>
<tr>
<td>22. Stick in your cheeks (together)</td>
<td>7.5</td>
</tr>
<tr>
<td>23. Stick out both lips</td>
<td>17.5</td>
</tr>
<tr>
<td>24. Make a clip-clop noise with your tongue</td>
<td>10.0</td>
</tr>
<tr>
<td>25. Bite the inside of your left cheek</td>
<td>1.0</td>
</tr>
<tr>
<td>26. Swallow</td>
<td>13.5</td>
</tr>
<tr>
<td>27. Make a high sound</td>
<td>25.0</td>
</tr>
<tr>
<td>28. Make a low sound</td>
<td>25.0</td>
</tr>
<tr>
<td>29. Clear your throat</td>
<td>25.0</td>
</tr>
</tbody>
</table>

Table A2 Items of the Upper Face Apraxia Test and relative ranking

<table>
<thead>
<tr>
<th>Item</th>
<th>Rank score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Close your eyes</td>
<td>8.0</td>
</tr>
<tr>
<td>2. Look down</td>
<td>3.0</td>
</tr>
<tr>
<td>3. Look towards the right (keeping head motionless)</td>
<td>8.0</td>
</tr>
<tr>
<td>4. Wrinkle your nose</td>
<td>8.0</td>
</tr>
<tr>
<td>5. Wrinkle your forehead</td>
<td>4.0</td>
</tr>
<tr>
<td>6. Look towards the left (keeping head motionless)</td>
<td>6.0</td>
</tr>
<tr>
<td>7. Blink your left eye (tight)</td>
<td>1.5</td>
</tr>
<tr>
<td>8. Look up (at the ceiling)</td>
<td>5.0</td>
</tr>
<tr>
<td>9. Blink your right eye (tight)</td>
<td>1.5</td>
</tr>
</tbody>
</table>
Table A3 Correction grids: (A) Lower Face Apraxia Test; (B) Upper Face Apraxia Test

(A) Lower Face Apraxia Test

<table>
<thead>
<tr>
<th>Education</th>
<th>Age (years)</th>
<th>20</th>
<th>25</th>
<th>30</th>
<th>35</th>
<th>40</th>
<th>45</th>
<th>50</th>
<th>55</th>
<th>60</th>
<th>65</th>
<th>70</th>
<th>75</th>
<th>80</th>
<th>85</th>
<th>90</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>–3.75</td>
<td>–2.75</td>
<td>–1.75</td>
<td>–0.75</td>
<td>0.50</td>
<td>1.50</td>
<td>2.50</td>
<td>3.50</td>
<td>4.50</td>
<td>5.50</td>
<td>6.75</td>
<td>7.75</td>
<td>8.75</td>
<td>9.75</td>
<td>10.75</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>–4.75</td>
<td>–3.75</td>
<td>–2.75</td>
<td>–1.75</td>
<td>–0.75</td>
<td>0.50</td>
<td>1.50</td>
<td>2.50</td>
<td>3.50</td>
<td>4.50</td>
<td>5.75</td>
<td>6.75</td>
<td>7.75</td>
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<td>9.75</td>
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<td>8</td>
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<td>–5.25</td>
<td>–4.25</td>
<td>–3.25</td>
<td>–2.25</td>
<td>–1.25</td>
<td>0</td>
<td>1.00</td>
<td>2.00</td>
<td>3.00</td>
<td>4.00</td>
<td>5.25</td>
<td>6.25</td>
<td>7.25</td>
<td>8.25</td>
<td></td>
</tr>
<tr>
<td>13</td>
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<td>–8.00</td>
<td>–6.75</td>
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<td>–4.75</td>
<td>–3.75</td>
<td>–2.75</td>
<td>–1.75</td>
<td>–0.50</td>
<td>0.50</td>
<td>1.50</td>
<td>2.50</td>
<td>3.50</td>
<td>4.75</td>
<td>5.75</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>–11.00</td>
<td>–10.00</td>
<td>–9.00</td>
<td>–8.00</td>
<td>–6.75</td>
<td>–5.75</td>
<td>–4.75</td>
<td>–3.75</td>
<td>–2.75</td>
<td>–1.75</td>
<td>–0.50</td>
<td>0.50</td>
<td>1.50</td>
<td>2.50</td>
<td>3.50</td>
<td></td>
</tr>
</tbody>
</table>

Score = \(-0.21 \times (\text{age} - 56.361)\) – \([0.517 \times (\text{education} - 10.389)]\).

(B) Upper Face Apraxia Test

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>20</th>
<th>25</th>
<th>30</th>
<th>35</th>
<th>40</th>
<th>45</th>
<th>50</th>
<th>55</th>
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<th>75</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>–1.00</td>
<td>–0.75</td>
<td>–0.75</td>
<td>–0.50</td>
<td>–0.50</td>
<td>–0.25</td>
<td>–0.25</td>
<td>0</td>
<td>0</td>
<td>0.25</td>
<td>0.25</td>
<td>0.50</td>
<td>0.50</td>
<td>0.75</td>
<td>0.75</td>
</tr>
</tbody>
</table>

Score = \([0.025 \times (\text{age} - 56.361)]\).

from a worst of zero to a best score of 45.0 in the Upper Face Apraxia Test and to a best of 435.0 in the Lower Face Apraxia Test.

Normative data

Sample

The sample included 180 healthy volunteers (122 female and 58 male), 96 of whom were Italian and 84 British. The participants were not paid. Their age ranged from 20 to 94 years (mean 56, SD 21); their education ranged from 1 to 21 years (mean 10.4, SD 4.66). None of the participants had a history of head injury or any other neurological disease. Forty-one of the volunteers who entered the study (10 male, 31 female) were re-tested by the same examiner after 5–7 days. The age range of this group was 20–94 with a mean age of 65 (SD 21).

Norms

Multiple regression analyses were performed to assess the influence of sex, age and educational level on the participants’ performance in the Upper Face Test \([F(3,176) = 9.885, P < 0.0001]\) and the Lower Face Test \([F(3,176) = 26.024, P < 0.0001]\) apraxia tests. Sex did not influence performance in either of the tests (Upper Face: \(t = -0.459, \text{n.s.}\); Lower Face: \(t < 1, \text{n.s.}\)), while age had a significant effect on both the Upper \((t = -3.012, P < 0.003)\) and Lower Face \((t = -5.091, P < 0.0001)\) scores. Education was shown to significantly affect performance in the Lower Face Test \((t = 2.735, P < 0.007)\) but not in the Upper Face Test \((t = 1.819, \text{n.s.}\)). Correction grids (Table A3a and A3b) were derived to adjust the performance of each new individual tested for age and education.

Because of the skewness towards the top score, inner and outer tolerance limits were computed by means of a non-parametric procedure (Ackermann, 1985). A score is considered normal if it lies within the highest 95% of the population and abnormal if it falls within the lowest 5%. The inner tolerance limit indicates the score at which or above which, with a confidence level of at least 0.95, the probability that an individual does not belong to the normal population is \(<0.05\). The outer tolerance limit defines the score at which or below which the probability that an individual belongs to the normal population is \(<0.05\). Scores between the inner and outer tolerance limits are considered borderline. The inner and outer tolerance limits for the Upper and Lower Face Apraxia Tests are displayed in Table A4.

Table A4 Inner and outer tolerance limits for the Upper and Lower Face Apraxia Tests

<table>
<thead>
<tr>
<th>Test</th>
<th>Tolerance limits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Inner</td>
</tr>
<tr>
<td>Upper Face</td>
<td>42.29</td>
</tr>
<tr>
<td>Lower Face</td>
<td>419.18</td>
</tr>
</tbody>
</table>

Reliability

Test–retest reliability for the Upper and Lower Apraxia Face Tests was 0.93 and 0.95, respectively.

Conclusions

Two tests of facial apraxia, assessing upper (eyes and eyebrows) and lower (mouth and throat) face movements have been presented. The psychometric workout of either test allows the experimenter to account for the relative difficulty of the test items, by means of an item ranking scoring system. Moreover, it permits adjustment of the score for the contribution of normal ageing and educational level. The normative data provide future investigators with age- and education-adjusted scores to carefully assess new individual patients with population-rather than sample-based cut-off scores. Finally, these tests have a high test–retest reliability, and proved easy to administer to brain damaged patients.