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# Tapping, grasping and aiming in ideomotor apraxia

Magdalena Ietswaart [a,*], David P. Carey [b], Sergio Della Sala [c]

[a] *Cognitive Neuroscience Research Unit, Wolfson Research Institute, University of Durham, Queen's Campus, Stockton-on-Tees TS17 6BH, UK*
[b] *Neuropsychology Research Group, University of Aberdeen, Old Aberdeen AB24 2UB, UK*
[c] *Human Cognitive Neuroscience, University of Edinburgh, 7 George Square, Edinburgh EH8 9JZ, UK*

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**Abstract**

Very few studies have investigated sensorimotor control in apraxia using tasks that differ in movement complexity. Nevertheless, there is some evidence to suggest that spontaneous behaviour, although relatively preserved, can be rather clumsy or awkward, and that patients with ideomotor apraxia may have subtle kinematic abnormalities in movements made in the laboratory. It remains unclear whether patients with ideomotor apraxia perform normally on movements such as visually guided aiming, that may not depend on higher-order, more cognitive, processes and that are relatively unguided by overlearned contexts. In this study, three different sensorimotor tasks were given to the same sample of patients with quantified apraxic disturbance. Finger tapping, goal-directed grasping and aiming with and without visual feedback were examined in these patients. A clear dissociation was found between grossly impaired gesture imitation and intact motor programming of goal-directed movements with visual feedback. Apraxic patients were, however, impaired on aiming movements without visual feedback, suggesting that apraxia is associated with an increased reliance on integration of online visual information with feedforward/feedback somatosensory and motor signals. Furthermore, patients were impaired on single finger tapping which was a surprisingly good predictor of apraxia severity.


*Keywords:* Kinematics; Prehension; Reaching; Ipsilesional hand; Left hemisphere; Stroke

## 1. Introduction

Ideomotor or limb apraxia is generally a result of damage to the left cerebral hemisphere and is characterised by the inability to imitate gestures, to pantomime tool use or to perform gestures on verbal command (De Renzi & Faglioni, 1999; Goldenberg, 2003; Liepmann, 1900). Apraxia is considered a "higher-order" deficit of the praxis system beyond more elementary levels of sensorimotor control (e.g. Binkofski & Fink, 2005). However, the features of higher-order processing in relation to apraxia are generally rather ill-defined. Furthermore, the apraxia literature tends to ignore the fact that complex movements (particularly when they have to be copied and are meaningless) are also characterised by sensorimotor integration demands.

There is some evidence that these patients are awkward even in spontaneous movements. Anecdotal comments by Basso and Capitani (1985) and by De Renzi and Lucchelli (1988) suggest that spontaneous behaviour in ideomotor apraxia, although relatively preserved, can be rather clumsy or awkward (for discussion, see Cubelli & Della Sala, 1996). Furthermore, evidence suggests that patients with ideomotor apraxia also show subtle kinematic abnormalities in movements made within natural contexts (Clark et al., 1994; Poizner et al., 1995). For these reasons, it remains unclear whether patients with ideomotor apraxia are normal on movements, such as visually guided aiming, that may not depend on higher-order, more cognitive, processes that are relatively unguided by overlearned contexts.

Those types of movements have been the subject of some study, usually in investigations focussing on hemispheric contributions to visuospatial analysis and sensorimotor control using visually guided aiming. Such studies have examined the relative performance of left hemisphere damaged (LBD) and right hemisphere damaged (RBD) patients and tend to confirm a specialised role for the left hemisphere in online control of movement (cf. Goodale, 1988; Winstein & Pohl, 1995). However, these studies are focussed on gross differences between these two groups, hence they rarely examine the performance of any *apraxic* LBD patient sub-samples in detail. Very few studies have examined

* Corresponding author.
*E-mail address:* magdalena.ietswaart@durham.ac.uk (M. Ietswaart).

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apraxia by investigating how sensory inputs affect motor outputs.

Although the general population of LBD patients perform normally on single finger tapping (Haaland & Delaney, 1981; Haaland & Harrington, 1989, 1994; Kimura, 1977), an early study by Heilman (1975) linked tapping abnormality to apraxia. He found that tapping rate was significantly lower in 10 patients with apraxia compared to 10 aphasic patients without apraxia. This interesting claim has been left unexamined until Hermsdörfer and Goldenberg (2002) reported decreased regularity but not reduced finger tapping speed in a sample of predominantly apraxic LBD patients. This is a failure to replicate Heilman (1975); nevertheless some sort of abnormality in speeded finger tapping is suggested by both of these studies, although its nature remains to be determined.

Aiming movements have also been the subject of *some* experimental work in apraxia. Haaland, Harrington, and Knight (1999) found selective impairment in a group of 10 patients with ideomotor apraxia when reaching to targets without visual feedback of the hand or the target. Apraxic patients were less accurate compared to LBD patients without apraxia, when making aiming movements without visual feedback of the hand or target, but not when making movements with visual feedback. Although apraxics were less accurate without visual feedback, the temporal dynamics of their movements were not affected. Haaland et al. accounted for these findings by arguing for an increased dependence on target information and a "decoupling" of spatial and temporal representations in apraxia. One possible limitation of this study was that their participants held a hand-held stylus and the visual feedback and visual target positions were specified on a computer monitor in a different plane as the moving hand-stylus. In such set-ups, proprioceptive feedback about the effector, and the visual reafference consequent to those movements, are in extremely discrepant locations. These sorts of tasks may be more informative about the ability of participants to learn and utilise "non-standard sensorimotor mappings" (Wise, Di Pellegrino, & Boussaoud, 1996), but it is not transparent that they speak directly to mechanisms of open (hand invisible) and closed loop (hand invisible) control of the hand. It is therefore of interest to examine the performance of apraxic patients in a set-up where aiming movements require moving the index finger directly towards the actual position of a target.

Hermsdörfer, Blankenfeld, and Goldenberg (2003) examined aiming movements in LBD patients, of whom 85% were classified as apraxic. They found only a trend for impaired performance on some of their endpoint accuracy measures when reaching without visual feedback. Nevertheless, movement durations and peak velocities were statistically abnormal in this condition. The findings of Heilman (1975), as well as Haaland et al. (1999) and Hermsdörfer et al. (2003), are controversial in that they suggest a disruption of relatively low-level motor skills in apraxia.

In an earlier study, Hermsdörfer, Ulrich, Marquardt, Goldenberg and Mai (1999) found no evidence for disruption of other lower-level motor processes in apraxia. This study examined grasping movements with the ipsilesional hand in patients with left and right brain damage. Eight out of 12 of the LBD patients showed signs of apraxia. Hermsdörfer et al. (1999) found general slowing (increased movement duration and time to peak velocity, and decreased peak velocity) in LBD patients, but normal scaling of grip size to the size of the object and peak velocity to the distance of the object. However, no correlation was found between apraxia and performance on any of the kinematic parameters such as grip scaling, peak velocity and movement duration.

Tapping, aiming and grasping movements differ in at least three key respects. First, aiming and grasping require coordination of upper and lower arm segments while tapping does not. Tapping requires rapid transitions from extension to flexion, which are absent in aiming and grasping. Finally, grasping requires integration of transport and grasp components in time and space (Desmurget et al., 1996) while aiming does not. The current study examined tapping, aiming and grasping in a single sample of patients with ideomotor apraxia. This procedure allowed performance on these tasks to be compared *within* a group of apraxic patients. Furthermore, performance on these movements was explored in relation to severity as measured by a validated test of ideomotor apraxia. The kinematic features of performance on these tasks were examined utilising high-resolution recordings following the rationale suggested by Poizner et al. (1998).

## 2. Participants

Ten right-handed patients and 10 neurologically intact age- and sex-matched control participants were recruited for this study. Patients were recruited from the Aberdeen Royal Infirmary acute stroke unit. Controls were recruited from a general public panel of volunteers and were paid expenses. All of the patients had vascular lesions in the left cerebral hemisphere, as a result of a cerebrovascular ischaemic infarct or primary intra-cerebral haemorrhage. On the basis of CT scan evidence all patients in this study had damage in the territory of the left middle cerebral artery. In addition, all patients were impaired on the Goldenberg Ten Meaningless Gestures Apraxia Test (cut-off score < 17/20 correct; Goldenberg, 1995, 1996). The Goldenberg test requires patients to copy a series of meaningless gestures with the ipsilesional hand. Patients in this study scored in the range of 2–12 out of 20, when tested shortly after their stroke. At the time of testing of the laboratory tasks, 8 out of 10 patients still scored below the cut-off on the Goldenberg test, while 2 patients had a borderline score (see Table 1). All 10 patients had CT scans in the acute phase of their stroke. Scans were mapped onto standardised templates using the procedures recommended by Damasio and Damasio (1989). One of the scans (of patient BA) was performed too early for the lesion to be accurately localised. In a second patient (patient EL), the scan was reported to include damage restricted to the lentiform nucleus, but was not available to the study. Lesion size was estimated for the eight patients with suitable scans. Using the standardised templates the area corresponding to the lesion for each slice was calculated, and the areas were summed to arrive at a lesion size for that scan (Leibovitch et al., 1998). Lesion site was determined on the basis of the standardised templates (with the exception of

Table 1
Demographical and neuropsychological data, lesion information and experimental tapping data of the individual patients involved in this study

| Case | Age | Days post-stroke[a] | Apraxia score at bed-side[b] | Apraxia score at laboratory testing[c] | Aphasia score at laboratory testing[d] | Grip strength[e] | Tapping rate[f] | % inter-tap interval[g] | Damaged areas in the left hemisphere[h] |
|---|---|---|---|---|---|---|---|---|---|
| EL | 48 | 65 | 9 | 18 | 35 | 50/50 | 56 | 12 | Lentiform nucleus |
| ME | 67 | 154 | 2 | 8 | 23 | 18/12 | 21 | 28 | Inferior parietal lobe (IPL, area 39, 40) |
| FD | 72 | 81 | 4 | 8 | n.t | 30/8 | n.t. | n.t. | Broca's area, SMA, IPL (area 44, 45, 6, 39, 40), and post central gyrus |
| BA | 68 | 93 | 5 | 8 | 7.5 | 30/14 | 22 | 14 | White matter damage (periventricular ischaemia) |
| HE | 72 | 58 | 11 | 18 | 35 | 36/36 | 50 | 7 | Broca's area (area 44, 45) |
| HA | 64 | 80 | 10 | 11 | 26 | 15/15 | 36 | 8 | Broca's and part of Wernicke' area (area 44,45, part 22), and post central gyrus |
| CD | 65 | 51 | 10 | 15 | 30.5 | 19/n.t. | 37 | 16 | Broca's and Wernicke's area (area 44,45, 22) |
| CM | 67 | 194 | 12 | 15 | 21 | 18/22 | n.t. | n.t. | White matter damage (parietal lobe) |
| MC | 77 | 241 | n.t. | 2 | 33 | 22/18 | n.t. | n.t. | Wernicke's area, SMA, part of ILP (area 22, 6, part 39, 40) and post central gyrus |
| AM | 67 | 67 | 8 | 13 | 12.5 | 38/15 | 48 | 7 | Broca's area (area 44, 45) and white matter damage (frontal and parietal lobe) |
| Controls | 67 (S.D. = 7.6) | n.a. | n.t. | n.t. | n.t. | n.t. | 52 (S.D. = 7) | 11 (S.D. = 2.4) | n.a. |

In this table, n.t., not tested; n.a., not applicable. The bottom row presents the average scores and standard deviations of the 10 controls.

[a] Days post-stroke at time of laboratory testing.

[b] Goldenberg Ten Meaningless Gestures Apraxia Test; score achieved at bed-side (score out of 20, cut-off <17) (Goldenberg, 1995, 1996).

[c] Goldenberg Ten Meaningless Gestures Apraxia Test; score at time of laboratory testing (score out of 20, cut-off score <17 pathological; 17–18 are borderline scores) (Goldenberg, 1995, 1996).

[d] Language impairment aphasia score at time of laboratory testing assessed with the Token Test, cut-off <30 (De Renzi & Faglioni, 1978).

[e] Grip strength dynamometer assessment (kilograms force) of the left hand/right hand, at time of laboratory testing.

[f] Single finger tapping rate, mean number of taps per 10 s of the four best trials (experimental data task 1).

[g] Mean percentage tapping variability of single finger tapping of the four best trials (experimental data task 1).

[h] Damaged areas (Brodmann's numbers in parentheses) in the left hemisphere.

patients BA and EL, where the radiologist's report was used to describe the lesion), including the affected Brodmann areas (see Table 1). Depictions of the templates for the patients involved can be found in Ietswaart, Carey, Della Sala, and Dijkhuizen (2001). Language impairment was formally assessed at the time of laboratory testing using the shortened version of the Token Test (De Renzi & Faglioni, 1978).

None of the patients showed any signs of hemispatial neglect at any time, or evidence of misreaching behaviour either while foveating a target or while foveating the examiners nose while reaching in peripheral vision, tested acutely and in the laboratory setting. Immediately post-stroke all patients had mild to severe aphasia, and a weakness in the contra-lesional lower and/or upper limb. Different numbers of patients took part in each of the tasks of this study, ranging from 7 to 10 patients and controls, because not all patients were available for all of the testing sessions.

## 3. Task 1: single finger tapping

### 3.1. Procedure

Seven patients took part in this task (all patients but FD, CM and MC, see Table 1). Participants were instructed to tap a key attached to a mechanical counter (Lafayette Instruments Model #32726) as quickly as they could with the index finger of the ipsilesional hand for 10 s. The examiner demonstrated speeded tapping to the patients and provided them with practice, while carefully watching their performance and effort to ensure that the patient grasped the concept of speeded tapping and maintained the instruction. Throughout the session the examiner stressed the speeded nature of the movement. Five trials were collected after rest breaks. The performance with the ipsilesional hand of these right-handed patients was compared with seven right-handed age- and sex-matched healthy control participants tapping with the left (in patients' case ipsilesional) hand.

Movement characteristics were recorded with a MacReflex motion analysis system (Qualysis, Inc.). The cameras of this system project and detect infrared light reflected by markers attached to the participants' body. This optic-based system uses free-standing markers, allowing unrestricted movements by the participants. Three-dimensional coordinates of the markers were sampled at 60 Hz. Two markers were attached to the participants' hand—one to the dorsal surface of the index fingernail, and the second to the wrist (the second marker was used to search for not permitted forearm movements contributing to tapping speed). The inter-tap interval was assessed by calculating the time difference between the positive peaks of the velocity profile. Percentage variability of the inter-tap interval (the variance of the mean time difference between subsequent taps as a percentage of the mean inter-tap interval) was calculated to express the temporal variability of the tapping movements. The mean tapping rate was calculated after discarding the trial with the lowest number of total taps attained over the four remaining trials. Tapping rates and tapping variability of patients and controls was compared using *t*-tests and descriptive statistics.

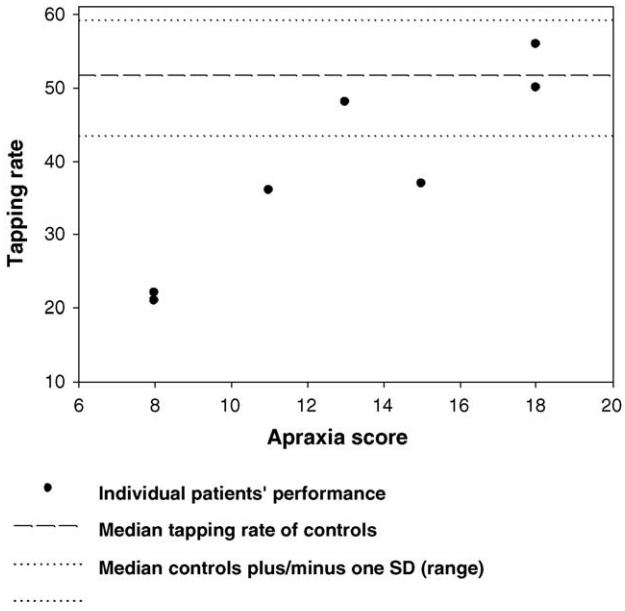


Fig. 1. The association between finger tapping rate and apraxia severity.

### 3.2. Results

Patients had an average tapping rate of 39 (S.D. = 14) taps per 10 s, while controls had an average tapping rate of 52 (S.D. = 7). This difference between patients and controls was found to be significant ($t(12) = -2.23$; $p < 0.05$). Four out of seven patients had a tapping rate more than 2S.D.s below the mean of controls. A high correlation was found between patients' apraxia severity and finger tapping rate (rho = 0.95; $p < 0.01$, see Fig. 1). The association between apraxia severity and lesion size was weak (rho = −0.39; n.s.). Furthermore, the association between lesion size and tapping rate (rho = −0.10; n.s.) suggests that tapping performance is selectively associated with apraxia.

Patients' percentage variability of the inter-tap interval was on average 13% (S.D. = 7.4), while controls' variability was on average 11% (S.D. = 2.4), and no significant difference between the groups was found ($t(12) = 0.731$; n.s.). Slow tapping rates appear to be associated with abnormally increased tapping variability in only one of the patients (patient ME was more than 2S.D.s more variable). All other patients with low tapping rates did not produce less regular tapping patterns, expressed in a weak association between tapping rate and tapping variability (rho = −0.63; n.s.). Furthermore, a weak association was found between tapping variability and apraxia score, which did not reach significance (rho = −0.49; n.s.).

The selectivity of the association between finger tapping rate and apraxia severity was assessed by correlating these measures with another neuropsychological deficit: aphasia measured with the Token Test. As expected, the correlation between apraxia and aphasia severity was very high (rho = 0.86; $p < 0.05$). However, the association between aphasia severity and tapping rate (rho = 0.70; n.s.) was not as strong as that between apraxia severity and tapping rate. Furthermore, partial correlation between apraxia severity and tapping rate controlling for aphasia severity

was high (partial $r = 0.91$; $p < 0.05$). In summary, apraxia appears to be selectively associated with slow finger tapping that cannot obviously be explained as irregular, poorly coordinated or "clumsy".

## 4. Task 2: visually guided prehension

### 4.1. Procedure

Nine patients took part in this task (all patients with the exception of patient CM). Participants were required to pick up an object placed in front of them on the tabletop using the thumb and index finger of the ipsilesional (left) hand. Three differently sized objects were placed at three different distances along the participants' body midline. The object used were blocks of 1 cm thick inflexible medium lightweight synthetic material. The blocks were Efron shapes (Efron, 1969) matched for area but differing in dimensions (sizes used here were 5 cm × 5 cm, 4 cm × 6.25 cm and 3 cm × 8.33 cm). Participants were asked to pick up the blocks along the sagittal plane where the blocks were 5, 4, or 3 cm wide. The blocks were placed along the sagittal midline on a distance of 40, 30, or 20 cm from the starting position of the hand. Participants were asked to pick up the object using thumb and index finger, and prior to moving to keep those fingers in a pinch position at the marked start position. Blocks were positioned on the table while participants had their eyes closed. At the auditory start signal participants were requested to open their eyes and to pick up the blocks as quickly and accurately as possible. Each of the objects was presented three times at each of the distances, resulting in 27 trials. Trial order was randomised, and the succession of trials was paced by the examiner. Patients' performance was compared with left hand reaches of control participants.

Movements were recorded with the motion analysis system described for task 1. Dependent measures extracted from each trial were peak velocity of the wrist, and maximum grip aperture (the maximum distance between thumb and index finger of the hand before picking up the object, typically related to object size and showing an overshoot before closing the fingers around the object). Maximum grip aperture was analysed in relation to object size, to determine whether grip of the hand was scaled according to the size of the object. Peak velocity was analysed in relation to object distance, to see whether the speed of movement was scaled according to object distance. A two-way analysis of variance assessed differences between patients and controls on grip scale formation (three object sizes) and distance scaling (three object distances).

### 4.2. Results

Both patients and controls showed clear grip scaling of the hand in prehension with visual feedback ($F(1,16) = 119.77$; $p < 0.001$). No differences between patients and controls were observed. Patients scaled the opening of the hand according to object size, as demonstrated by an individual grip aperture profile of the most severely apraxic patient in our sample (patient MC) and his matched control in Fig. 2. Group means for the effect of block size on maximum grip aperture are shown in Fig. 3.

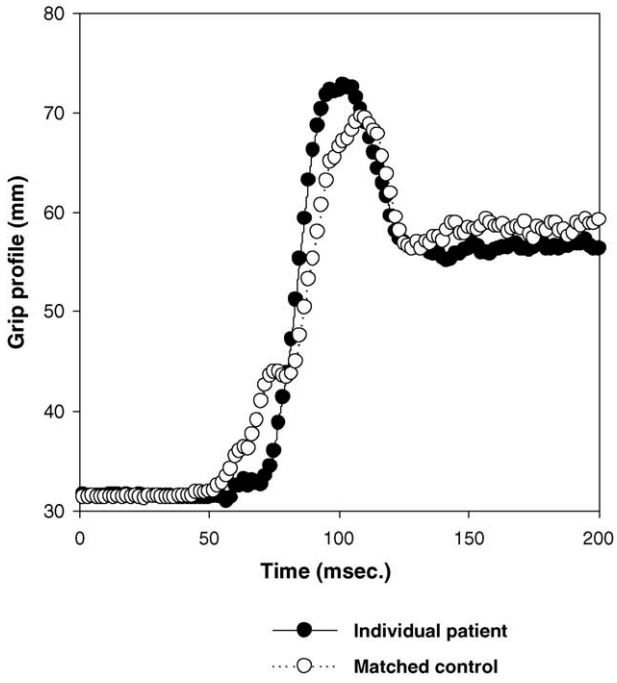


Fig. 2. Example of a patient's and matched control's individual trial grip scaling of the hand (distance between thumb and index finger) when picking up an object.

Furthermore, both patients and controls showed clear effects of distance scaling on peak velocity ($F(1,16) = 37.32$; $p < 0.001$), on time to peak velocity ($F(1,16) = 33.12$; $p < 0.001$) and movement duration ($F(1,16) = 38.85$; $p < 0.001$). Patients did not differ from controls on any of these measures. Patients showed preserved ability to increase the speed of the hand to increase in movement amplitude. No main effect of group was found on any of these measures, indicating that patients were neither slower in performing the movements overall. Fig. 4 shows the effect of maximum peak velocity distance scaling for each group.

Although Hermsdörfer et al. (1999) found no notable correlation between apraxia and performance on kinematic parameters of grasping speed, they found that LBD patients showed generally slowed grasping movements, differing from controls on peak velocity, time to peak velocity and movement duration. No significant group differences in grasping speed (either in peak velocity, time to peak velocity, or movement duration) were found in the present sample. Correlation coefficients between apraxia severity and grasping speed for the current sample of apraxic patients showed no significant association between apraxia score and peak velocity (rho = −0.20; n.s.), time to peak velocity (rho = 0.17; n.s.), or movement duration (rho = 0.21; n.s.). Distance scaling, expressed in the average scaling increments between the different object distances, also demonstrated no association with apraxia severity (rho = −0.16; n.s.). Finally, grip scaling was found to have a moderate association with apraxia score (rho = −0.52; n.s.), however, this relationship was contradictory (i.e. better grip scaling with more severe apraxia) and was not significant.

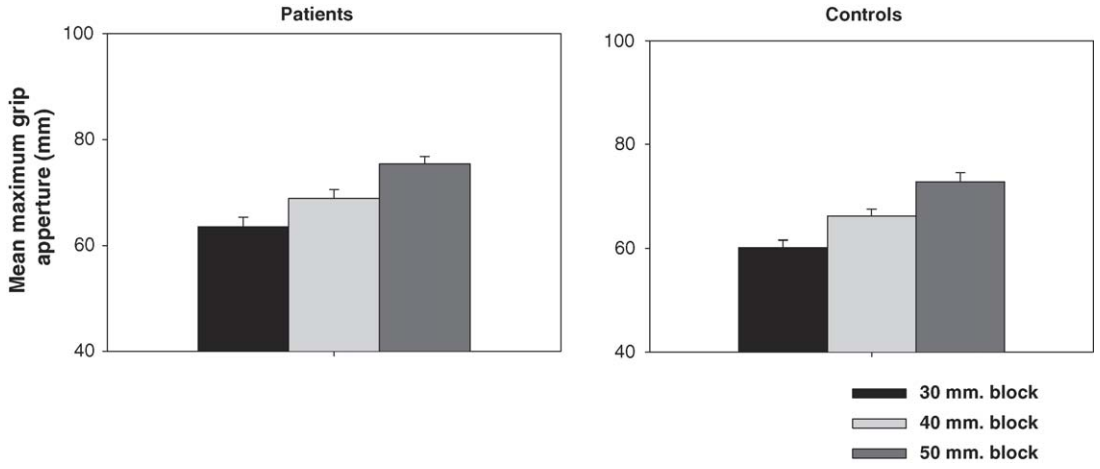


Fig. 3. Mean grip size scaling (maximum grip aperture in mm ± 1S.E.) of grasping with visual feedback in patients ($n = 9$) and controls ($n = 9$).

In summary, patients with apraxia showed normal motor programming of prehensile movements with visual feedback. Movements were not slowed, nor did they show different timing compared to movements of controls. Patients with apraxia were able to scale the maximum velocity of the hand moving towards the target to adapt to changes in movement amplitude. They were also able to scale the maximum aperture of the grasp to adapt to object size. No indications of impaired representations of hand or target were found in these movements.

## 5. Task 3: target-directed pointing and visual feedback

### 5.1. Procedure

Ten patients took part in this task (for details on these patients, see Table 1). They were compared with 10 age- and sex-matched controls. Some of the results of this experiment were reported in Ietswaart et al. (2001), where these target-directed pointing movements were the baseline condition in a memory-driven movement experiment. They are included here to explore the association with apraxia severity and for comparison with the findings by Haaland et al. (1999) and Hermsdörfer et al. (2003) who found impaired performance on some aspects of aiming movements in apraxic patients.

Participants pointed to six different target positions indicated by briefly illuminated LEDs, using the index finger of the ipsilesional (left) hand. They were asked to indicate the target position by moving their finger from the start position to the position where the target light was displayed, in a rapid and accurate movement. LEDs were fitted under a transparent Plexiglas sheet on the tabletop. The six possible target positions were horizontally aligned, three on each side of a central fixation point. The starting position was approximately 20 cm in front of the subject, along from the body midline. The fixation point was 29 cm further along this line. The lights were displayed 5, 10 and 15 cm to the left and right of the fixation point. Prior to target display, a green LED, representing the fixation point, was shown for 800 ms, subsequently one of the red target LEDs was displayed for 700 ms. There was an auditory movement start signal, while a second signal 2 s later indicated that the participant was to return the finger to the starting position before the next trial. The succession of trials was paced by the examiner, and target order was randomised. Each target position was presented four times, resulting in 24 trials per condition. Each condition was

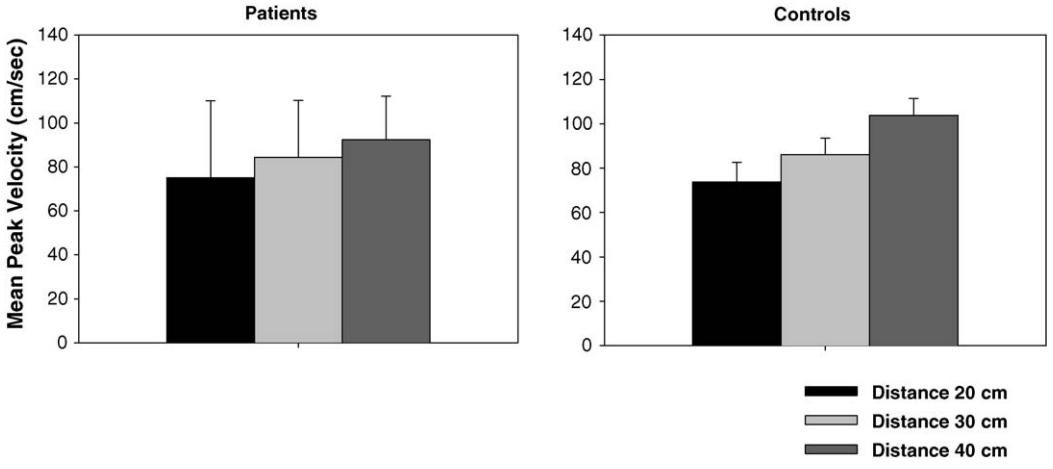


Fig. 4. Mean peak velocity distance scaling (maximum displacement in cm/s ± 1S.E.) of grasping with visual feedback in patients ($n = 9$) and controls ($n = 9$).

preceded by several practice trials, ensuring the examiner that the task instructions were understood and maintained. After an auditory start signal, participants were requested to point to the target position by moving the index finger of the ipsilesional hand from the start position to the position were the target light was displayed, in a rapid accurate movement. Their performance was compared with left hand reaches of 10 control participants.

Target-directed pointing was performed under full closed-loop and full open-loop conditions. In the closed-loop condition, full visual feedback of the hand was maintained throughout the trial (as well as the table surface, etc.). In the full open-loop condition, the room was darkened 1 s before the start of the trial and remained so throughout the trial till 3.4 s after target offset, by which time participants had returned their hand to the start position. The procedure of darkening the room allowed no visual feedback of the hand relative to the target throughout the trial. Room lights were switched on between trials to allow participants to visually orient before each trial and to avoid any short-term dark adaptation affecting the encoding of the hand position relative to the target (Rossetti, Stelmach, Desmurget, Prablanc, & Jeannerod, 1994) that may have occurred otherwise.

Movement characteristics were recorded with a MacReflex motion analysis system as described for task 1. The movement characteristics analysed were peak velocity (the point of maximum displacement over time); time to peak velocity (the time of maximum displacement relative to the start of movement); percentage time to peak velocity (the time used to reach maximum displacement relative to total movement time); reaction time and movement duration. Endpoint accuracy was measured as the absolute endpoint error (the two-dimensional radial distance between a finger on the target position and the actual landing position of the finger when reached to the target).

Median scores were analysed using a two-factor analysis of variance (ANOVA) for each of the six dependent measures, with factors of group (patients versus controls) and visual feedback (closed-loop versus open-loop conditions). Significant interactions were explored using the Newman–Keuls procedure (Kirk, 1982).

### 5.2. Results

Movement peak velocity of both patients and controls decreased when visual feedback of the hand was removed ($F(1,18) = 16.13$, $p < 0.001$, also see Ietswaart et al., 2001). Patients reached significantly lower peak velocities making target-directed reaches without visual feedback compared to controls ($F(1,18) = 5.03$; $p < 0.05$, see Fig. 5). Time to peak velocity, percentage deceleration, and reaction time did not differ between patients and controls. Movement duration was prolonged in these movements in patients; however, this effect did not reach significance ($F(1,18) = 4.30$; $p = 0.06$). Patients CM, HA and CD had median peak velocities and movement duration more than 2 S.D.s away from the mean of controls.

With regard to endpoint accuracy, all participants were less accurate when reaching without visual feedback of the hand moving towards the target ($F(1,18) = 178.92$, $p < 0.0001$). Patients were less accurate overall than the matched controls on the pointing task ($F(1,18) = 5.96$ $p < 0.05$). However, poor accuracy of the patients seems to arise from the removal of feedback (group × feedback: $F(1,18) = 6.25$, $p < 0.05$), as post hoc analysis revealed that the groups did not differ when reaching with the lights on (see Fig. 6). The median score of 4 out of 10 patients (patient BA, HA, MC and CD) was more than 2S.D.s away from the mean of the control participants. Separate analysis including only those patients whose mean endpoint accuracy was within the (normal) range of 2S.D.s from the mean of controls (i.e. patients EL, ME, CM, HE, AM and FD), comparing them to matched controls still suggested a group difference for endpoint accuracy expressed in a non-significant trend ($F(1,10) = 3.54$; $p = 0.09$). This suggests a fairly consistent pattern across individual patients of impaired accuracy when reaching without visual feedback. However, increased endpoint error as a result of

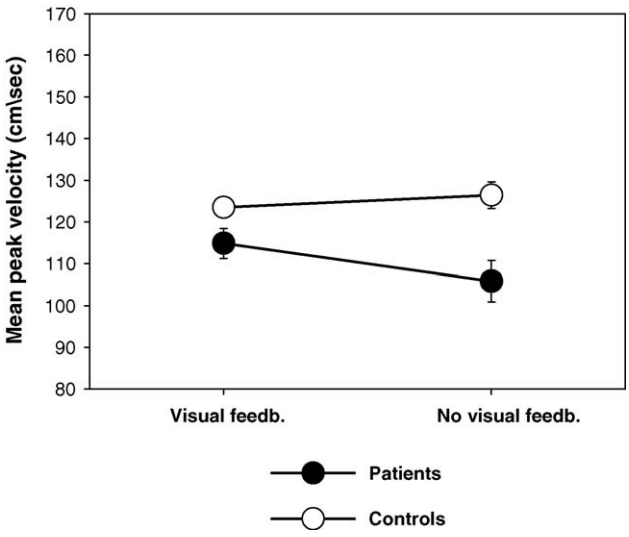


Fig. 5. Mean peak velocity (maximum displacement in cm/s ± 1S.E.) of aiming movements with and without visual feedback, for patients ($n = 10$) and controls ($n = 10$).

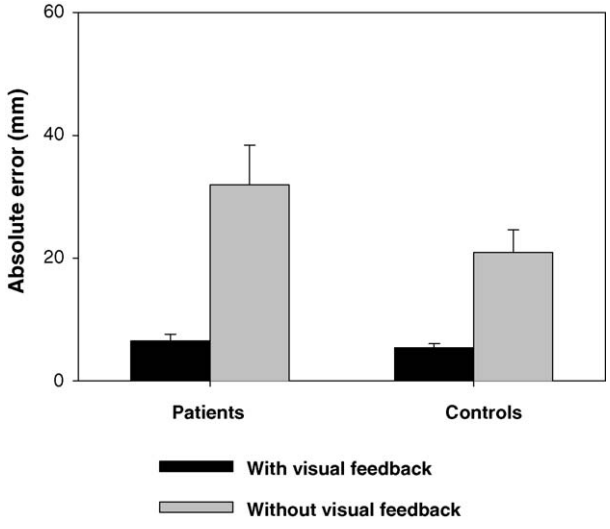


Fig. 6. Endpoint accuracy (absolute error in mm ± 1S.E.) of aiming movements with and without visual feedback, for patients ($n = 10$) and controls ($n = 10$).

removed visual feedback, expressed in the difference in accuracy between pointing under closed-loop and open-loop conditions, was not particularly strongly associated with apraxia severity (rho = −0.41; n.s.) or lesion size (rho = 0.21; n.s.).

## 6. Discussion

Apraxic patients were impaired on aiming movements made without visual feedback, and on overall tapping rate but not tapping variability. Visually guided grasping in our sample did not differentiate between patient and control groups. Remarkably, finger tapping rate was the best predictor of apraxia severity.

We found that normal spatial and temporal programming were clearly apparent in apraxic patients reaching towards targets and grasping objects with visual feedback, as previously reported by Haaland et al. (1999) in apraxic patients, and by Hermsdörfer et al. (2003) and Hermsdörfer et al. (1999) in a sample of LBD patients with a high apraxia incidence. We found no association between reaching and grasping performance indicators and apraxia severity.

However, when visual feedback was removed, apraxic patients were clearly impaired on target-directed aiming movements. This pattern of results is similar to that found by Haaland et al. (1999). However, there are some differences between the procedures of this particular study and the current one. In the study of Haaland et al., aiming movements were made holding a hand-held stylus, and position and movements of the pointing device were monitored by participants via a video screen. Such movements may depend more on learned non-standard sensorimotor mappings, as discussed above. Only when natural goal-directed movements with removal of true *online* visual guidance of movement is examined, as was the case in the current study, can it be said that the difficulties arise from disrupted sensorimotor integration rather than from impaired motor learning classically associated with apraxia (Geschwind, 1975).

The apraxic patients in Haaland et al. (1999) were less accurate than controls when visual feedback (from either hand or target) was unavailable. Other movement characteristics, however, were not affected when visual feedback was removed. Haaland et al.'s main conclusion from their findings was that apraxic patients showed a decoupling of spatial and temporal components, as movement duration and peak velocity were normal, but accuracy was impaired when visual feedback was removed. From their examination of complex movements, Poizner et al. (1995) also argued for decoupling of spatial and temporal representations in apraxia. The current findings challenge such a hypothesis. The present study did find impaired accuracy in apraxic patients on aiming movements made without visual feedback of the hand moving towards the target. However, movement duration and peak velocity were also affected in these patients. On the basis of these findings, we conclude that apraxic patients may rely more on integration of online visual information with feedforward and possibly feedback motor signals, rather than Haaland et al.'s favoured inference of decoupled temporal and spatial processing.

The current study also found impaired finger tapping in apraxic patients. It was previously reported that the general population of LBD patients performed normally on single finger tapping (Haaland & Delaney, 1981; Haaland & Harrington, 1989, 1994; Kimura, 1977). Heilman found decreased finger tapping rate to be selectively impaired in apraxia (Heilman, 1975), however, impaired finger tapping in apraxia had not been replicated since. Our findings mirror those of Heilman in a similar sample of apraxic patients. In addition, kinematic analysis of tapping variability and the association between tapping measures and apraxia severity and lesion size are reported in the current study. A strong correlation (rho = 0.95) between tapping rate and apraxia score was found. This association, although weaker ($r = 0.53$), was previously reported by Hermsdörfer and Goldenberg (2002). In contrast to our study these authors found no group differences between LBD patients and controls. The correlation between apraxia severity and tapping rate that Hermsdörfer and Goldenberg did find in their sample was mainly explained by the performance of the five most apraxic patients. The fact that the current study finds marked differences between apraxic patients and controls on finger tapping rate (which is strongly associated with apraxia severity but not with temporal variability) further clarifies that impaired finger tapping originally reported by Heilman (1975) cannot be explained as merely 'clumsy' tapping.

The results of the current study suggest that impaired single finger tapping may be related to apraxia. Indeed, Kimura (1977) admitted that although her left hemisphere aphasic group as a whole presented with normal single finger tapping, the three apraxic patients within that group did tap more slowly. In this respect, it is interesting that those patients in our sample with severe apraxia performed particularly poorly at this task, as confirmed by the highly significant correlation of finger tapping rate with apraxia score. This association cannot be explained by the presence of a few outliers, and furthermore, seems independent of lesion size.

The present replication of the finding of slowed single finger tapping in apraxia by Heilman (1975) should be seen in the context of our null findings of intact motor programming of goal-directed movements in the first ever within-subjects examination of tapping, reaching and grasping. Heilman claimed to test Liepmann and Maas' (1907) notion that the left hemisphere stores motor memories or engrams, which are lost or disconnected in apraxia. Heilman suggested that if Liepmann's hypothesis is correct, then, in addition to poor performance on imitation, there should also be poor performance on a motor task like finger tapping. However, if finger tapping relies on intact motor engrams, then presumably goal-directed movements or any other movement would as well. Heilman's finding of reduced finger tapping in apraxics was replicated in this study, but such impairments were not apparent in goal-directed movements with visual feedback.

The required rapid transitions in speeded finger tapping may be somewhat unique, however, compared to the larger ballistic-type movements generated in aiming or grasping. In spite of its apparent simplicity, rapid finger tapping may be lateralised differently in the two hemispheres of left- and right-handed neurologically intact participants (Herve, Mazoyer, Crivello, Percehy, & Tzoutio-Mazoyer, 2005) in a way consistent with

probable lateralisation of praxis (Goodale, 1990). Furthermore, a recent neuroimaging study speaks to a potentially complex neurobiology underlying tapping movements. Agnew, Zeffiro, and Eden (2004) looked at left- and right-handed thumb tapping movements in phase with a visual cue in a 1.5 T fMRI experiment. They found more activational increase in the left hemisphere motor cortex with increases in tapping rate of the right hand than in the right hemisphere with increased tapping rate of the left hand, in spite of perfectly accurate performance in both conditions. The tapping rates in Agnew et al. (2004) were very low, by design, so their data is not directly comparable to what our patients and neurologically intact controls did. Nevertheless, additional activations of corticostriatal and corticocerebellar structures were found, but only in the left hemisphere. These authors emphasised interactions between motor cortices and subcortical loops that are more pronounced in the left hemisphere of neurologically intact participants. The role of subcortical motor circuits in apraxia remains relatively understudied in the mainstream apraxia literature (Classen et al., 1996; Leiguarda, 2001; Pramstaller & Marsden, 1996). From our data, it is difficult to know with certainty that tapping deficits are related specifically to the damaged mechanisms that produce apraxia and are not a consequence of damage to overlapping circuits in the left hemisphere. Nevertheless, although aphasia and apraxia "overlap" neurologically, the relationship between tapping rate and aphasia severity is weaker ($r = 0.70$) than that between tapping rate and apraxia severity ($r = 0.95$). Furthermore, partial correlation between tapping rate and apraxia controlling for aphasia still demonstrates a highly significant association. These data convince us that tapping should be the target of additional experimentation by scientists interested in apraxia.

Our 60 Hz records do not have the necessary temporal resolution to determine any unique cause of slower tapping rates (i.e. lower velocities, longer transitions between flexion and extension, etc.). For this purpose, a replication with either direct digital recording of the tapping device or an opto-electronic recording system with higher temporal resolution would be an important next step in identifying if there is a specific, reliable cause of slowed tapping after left hemisphere lesions and/or apraxia. These latter recording systems would be ideal for additional experiments on tapping, such as control of increasing tapping rate in these patients and their limitations in following an increasing tempo specified by a metronome (as suggested by helpful comments of one of the reviewers). Furthermore, our small sample size limits our ability to try and link the poorest tapping performance to specific lesion locations. For this later purpose, a finger tapping device and a stopwatch would certainly suffice for a larger sample, multi-centre study.

Regardless of the specific neurobiological substrate, finger tapping might tax motor sequencing systems to some extent. Kimura (1993), like Heilman (1975), tried to keep close to Liepmann's original accounts, and has stressed this aspect of apraxia. However, recent data suggest that the simplest tapping tasks may be the most asymmetrical in neurologically intact participants (Hausman, Kirk, & Corballis, 2004). These authors found simple index finger tapping rate favoured right hand performance more than a simple and complex multi-digit sequence. They conclude that these data as well as several neuroimaging studies support the notion that more complex sensorimotor tasks may require the participation of wider networks less likely to be restricted to circumscribed regions of one cerebral hemisphere.

The strong relationship between impaired finger tapping and apraxia severity reveals that apraxics may have deficits at the level of elementary, non-higher-order movements. However, impaired finger tapping in the presence of preserved goal-directed reaching and grasping movements does not suggest a general motor programming deficit. Relatively recent claims (Leiguarda, 2005; Leiguarda & Marsden, 2000; Marsden, 1998) that apraxia is likely to be associated with misreaching or misgrasping, such as that seen in optic ataxia or after damage to the dorsal stream (Galletti, Kutz, Gamberini, Breveglieri, & Fattori, 2003; Rossetti et al., 2005), cannot be confirmed by the current findings. The present study does not find evidence of misreaching that would indicate disrupted dorsal stream processing in patients with apraxia. Movement characteristics of goal-directed movement like size and distance scaling as well as timing of aiming movement and accuracy are thought to be important and sensitive indicators of intact processing of the dorsal stream. This study provides clear evidence that visual information is appropriately integrated into movement programming of goal-directed movements in the current sample of patients. Even if visuomotor transformations are impaired in higher-order movement programming in apraxia, they cannot be related in any obvious way to impairment at the level of the dorsal visual pathway.

Impaired selection of motor programs may indeed underlie apraxia. However, such a deficit was not apparent in our patients on this level of relatively simple visuomotor transformations. The present study found a clear dissociation between grossly impaired gesture imitation, but intact motor programming of goal-directed movements with visual feedback. Patients were slower compared to controls executing the movements. But this effect is unlikely to be specific to apraxia and has been found in many other studies of patients with left hemisphere damage (Fisk & Goodale, 1988; Haaland & Harrington, 1989, 1994; Haaland, Harrington, & Yeo, 1987; Winstein & Pohl, 1995; Wyke, 1967). However, indicators of normal spatial and temporal programming were clearly apparent in these patients reaching towards targets and grasping objects with visual feedback, suggesting intact motor programming of goal-directed movements in apraxia. Paradoxically, slowing in finger tapping is the best predictor of apraxia severity.

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