Impairment of facial recognition in patients with right cerebral infarcts quantified by computer aided "morphing"

A Rösler, S Lanquillon, O Dippel, H J Braune

Abstract

Objective—To investigate where facial recognition is located anatomically and to establish whether there is a graded transition from unimpaired recognition of faces to complete prosopagnosia after infarctions in the territory of the middle cerebral artery.

Methods—A computerised morphing program was developed which shows 30 frames gradually changing from portrait photographs of unfamiliar persons to those of well known persons. With a standardised protocol, 31 patients with right and left sided infarctions in the territory of the middle cerebral artery and an age and sex matched control group were compared by non-parametric tests.

Results and conclusion—Facial recognition in patients with right sided lesions was significantly impaired compared with controls and with patients with left sided lesions. A graded impairment in facial recognition in patients with right sided ischaemic infarcts in the territory of the middle cerebral artery seems to exist.

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It is unlikely that we are able to distinguish any other object to such a degree as we can human faces. The idea that recognition of a human face could be a function which is independent of other recognition modalities of the brain, and therefore could also be independently disturbed, was finally established by Bodamer in 1947.¹ Previous to this reports of disturbances in facial recognition had been made by Willebrand² and Charcot.³ If we assume an isolated function of facial recognition, two substantial issues arise: (1) Where is it located anatomically? (2) Is there a graded transition from unimpaired recognition of faces to complete prosopagnosia?

Patients and methods

To elucidate these questions, 31 right handed patients with ischaemic infarcts in the supply area of the middle cerebral artery (15 left sided and 16 right sided infarcts) were studied and compared with 21 age and sex matched controls. The distribution of Barthel indices was the same for left and right sided infarcts (mean = 84 and median = 90 for left and mean = 84 and median = 95 for right sided infarcts). The following inclusion criteria were applied: cranial CT confirmed a single infarction in the supply area of the middle cerebral artery (table 1 shows the topographical distribution); no

Department of
Neurology
A Rösler
H J Braune

Department of
Psychiatry
S Lanquillon

Institute of Computer
Science, Philipps
University, Marburg,
Germany
O Dippel

Correspondence to:
Dr Alexander Rösler,
University Hospital
Marburg, Rudolf-Bultmann
Strasse 8, 35033 Marburg,
Germany.

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Figure 1 Selection of frames illustrating the morphing process.

Table 1 Distribution of infarcts by CT

Region	Right infarcts	Left infarcts
Cortical:		
Temporo-occipital	4	3
Temporoparieto-occipital	5	4
Frontotemporoparietal	1	2
Subcortical:		
Thalamus and internal capsule	4	3
Basal ganglia	2	3

Table 2 Results with frame Diana

	Attempts		
	≤ 25	26–30	Failed
Controls**	16 (76.2)	2 (9.5)	3 (14.3)
Left infarcts	6 (40)	4 (26.7)	5 (33.3)
Right infarcts**	3 (18.8)	6 (37.5)	7 (43.8)

Percentages are in parentheses.

**P = 0.002.

Table 3 Results with frame Kohl

	Attempts		
	≤ 24	25–30	Failed
Controls**	16 (76.2)	5 (23.8)	0 (0)
Left infarcts†	9 (60)	6 (40)	0 (0)
Right infarcts**†	5 (31.2)	7 (43.8)	4 (25)

Percentages are in parentheses.

†P = 0.034; **P = 0.004.

Table 4 Effect of cortical and subcortical lesions on results with frame Diana

	Attempts		
	≤ 25	26–30	Failed
Cortical:			
Left infarcts*	3	2	4
Right infarcts*	0	5	5
Subcortical:			
Left infarcts	3	2	1
Right infarcts	3	1	2

*P = 0.06 (NS).

Table 5 Effect of cortical and subcortical lesions on results with frame Kohl

	Attempts		
	≤ 24	25–30	Failed
Cortical:			
Left infarcts**	5	4	0
Right infarcts**	0	6	4
Subcortical:			
Left infarcts	4	2	0
Right infarcts	5	1	0

**P = 0.001.

Table 6 Effect of cortical brain region involved in right sided infarcts with frame Diana

	Attempts		
	≤ 25	26–30	Failed
Temporoparieto-occipital	0	1	4
Temporo-occipital	0	3	1

Table 7 Effect of cortical brain region involved in right sided infarcts with frame Kohl

	Attempts		
	≤ 24	25–30	Failed
Temporoparieto-occipital	0	1	4
Temporo-occipital	0	4	0

Figure 2 Group comparisons of attempts to identify Diana.

previous or multiple infarcts; tests were done in the second week after the stroke; exclusion of patients with moderate or severe dementia by mini mental status examination⁴; and exclusion of patients with receptive aphasia by the Aachener aphasia test.⁵

Each patient was shown two series of 30 pictures on a video screen (fig 1). In the first series a photo of Jil Sander turned into a portrait of Lady Diana; in the second Michael Douglas was transformed into Helmut Kohl. Patients were given 30 seconds with each frame to recognise Lady Diana and Helmut Kohl. The number of frames needed to identify the target either by name or social function was counted. Characteristics which could have aided in the recognition of the prominent person—for example, hair, glasses, or a tie—were not shown in the photo.

Based on the results of the controls for both series of pictures, categories were developed for normal, late, and non-identification. Seventy five per cent of the controls recognised Diana within 25 and Kohl within 24 attempts; therefore these numbers were set as arbitrary upper boundaries for “normal” recognition. More attempts up to the end point of 30 were considered “late” and no identification at all as “failed”. Patients or controls who did not know the target persons even after being told their names, were excluded from analysis. Results were analysed by maximum likelihood χ^2 tests.^{6,7}

Results

Healthy controls recognised Helmut Kohl early in 16 out of 21 cases and late in the remaining five. Diana was recognised early in 16, late in two, and not at all in three cases.

Patients with left sided infarcts recognised Diana and Kohl later than controls but this was not significant ($P = 0.086$ for Diana; $P = 0.58$ for Kohl).

Patients with right sided infarcts identified Diana and Kohl significantly later than the controls ($P = 0.002$ for Diana and $P = 0.004$ for Kohl).

Figure 3 Group comparisons of attempts to identify Kohl.

Group comparisons between left and right sided infarcts for Diana did not differ significantly ($P = 0.42$), whereas patients with right sided infarcts identified Kohl significantly later than those with left sided infarcts ($P = 0.034$) (tables 2 and 3, figs 2 and 3).

Subanalysis of cortical involvement by CT showed significant impairment of facial recognition in patients with right sided infarcts for Kohl ($P = 0.001$) and did not reach significance for Diana ($P = 0.06$), whereas isolated subcortical lesions did not have a significant effect on the recognition task (tables 4 and 5).

Involvement of the parietal lobe in right sided lesions was associated with more recognition failures compared with temporo-occipital lesions alone (tables 6 and 7). No statistical analysis was carried out because of the few patients in these subgroups.

Discussion

Our patients with right sided infarcts seemed to have more difficulties in identifying familiar faces than healthy controls and patients with left sided infarctions.

The neuroanatomical localisation of facial recognition and its disturbances still remain controversial. Kolb and Wishaw postulated that the damage which leads to prosopagnosia must be bilateral. Brodman's areas 18 and 19, and additionally 20, 21, and 37 seem to play a part in facial recognition.⁸ Kandel too argued that "lesions that cause prosopagnosia are always bilateral".⁹ In a comprehensive article by Meadows¹⁰ there were seven postmortem results available from patients who had had prosopagnosia. Five of these had bilateral occipitotemporal lesions, mostly symmetric, in accordance with the study by Damasio *et al.*¹¹ The two other patients had right sided occipitotemporal lesions.

Young *et al* reported a 51 year old woman who showed a deficit in recognising faces and matching photographs of facial emotional expression after bilateral partial amygdalotomy.¹²

On the other hand there is little doubt that the right hemisphere plays an important part in facial recognition: Hecaen and Angeluerges made a list of neurological signs which were positively associated with prosopagnosia.¹³

The disorder showed associated signs such as left visual field defects in 91% of the cases, disturbances of spatial orientation, constructive apraxia, and neglect. This enumeration alone indicates that right sided lesions are obviously more often related to impaired facial recognition. The high number of combinations with left upper quadrantanopia led to the assumption that the underlying lesion could be in the right basal occipital lobe. Miller described delayed recognition of faces in patients who had undergone right temporal lobectomy.¹⁴ In a more recent article, Evans *et al* reported the case of a 68 year old woman with progressive atrophy of the right temporal lobe developing progressive prosopagnosia.¹⁵ In a recent review Boeri and Salmaggi favoured a right posterior lesion in what they called true prosopagnosia,¹⁶ and a patient reported by Sergent and Poncet had a large right parietotemporal lesion sparing the medial posterior temporal cortex.¹⁷

These in part contradictory results suggest the possibility of different forms of impaired facial recognition.

Thus Bruce and Young developed a functional model of face processing which divides facial processing into different modalities. After a basic process of structural encoding, there should be different pathways to the analysis of facial expression, the recognition of familiar faces, and the matching of unfamiliar faces.¹⁸

We know that object recognition is processed by a ventral pathway constituted by occipito-temporal cortices. On the other hand, visuospatial processing of the visual scene is comprised of parieto-occipital pathways. Different modalities for the recognition of faces play a synergistic part. When healthy persons have to discriminate elementary features like colour, the prestriate cortex and in particular the fusiform gyrus is activated. Perceptual integration takes place in the posterior part of the lingual gyrus and, in a third step, especially important for the recognition of faces, mainly the anterior part of the lingual gyrus and the parahippocampic gyrus are activated in attributing meaning to the "percept".¹⁹

Despite this well established topographical association between the visual recognition process and the brain region involved, there is still no consensus on the question of lateralisation of different facial recognition modalities. For instance, results of analysing emotional facial expression are controversial:

Young *et al* examined 32 servicemen 40 years after they had sustained a penetrating missile injury to the brain in the second world war and found that expression analysis was selectively impaired in five patients with left hemispheric lesions,²⁰ although earlier this task had mainly been thought to be located in the right hemisphere.^{21 22}

Superiority of the right hemisphere was also claimed for the function of visual attention according to Dimmond and Heilmann.^{23 24} It could therefore be argued that the impairment of facial recognition in our patients with right

sided lesions might be partly due to failure of arousing visual interest in the emotional expression of the face shown. However, the lateralisation of visual attention remains controversial: Posner showed that patients with left or right parietal lesions were equally impaired in directing their attention to stimuli in both visual fields.²⁵

The studies mentioned were all carried out on patients with relatively longstanding brain defects, whereas in our patients the interval of one week between the event of infarction and the morphing task was presumably too short to allow selective testing for single modalities of facial recognition. This view is supported by the well known finding in other neuropsychological syndromes—for example, aphasia or apraxia—that a final distinction of different subforms is not usually possible in the acute phase of brain damage.

In general we may state that the complex function of facial recognition requires the integrity of the right temporoparieto-occipital region. This anatomical region was damaged to different extents in our patients. Three of five patients with right temporoparieto-occipital lesions failed to recognise both target pictures, the remaining two of this group received lower scores (28 for Diana and “failed” for Kohl, 28 for Kohl and “failed” for Diana) than the group of patients with temporo-occipital involvement, in which only one of four patients failed with Diana and all four recognised Kohl. Despite the small case numbers in these subgroups, these results suggest a possible key role for the parietal region in facial recognition.

In accordance with the results of Young and Tupler,^{20,26} our patients with lesions involving cortical regions were significantly impaired in our test of facial recognition, whereas the six patients with subcortical lesions only were not (tables 2 and 3).

Our patients, like others with right hemisphere damage, did not complain about difficulties recognising faces. Probably these deficits are easy to compensate for, because recognising a certain person “behind” a face in daily life is made easy by the perception of further visual and acoustic cues.²⁷

Among the tested patients with right sided cortical infarctions there were none with prosopagnosia in a “pure” sense, although there was a clear impairment in recognition of familiar faces; graded disturbances in the sense of a “prosopagnosia” in this common neurological disease do seem to exist.

With the exception of the Benton-Van Allen facial recognition test there are only experimental tests available for the function of facial recognition.²⁸

As Puschel and Zaidel showed, the Benton-Van Allen test is not able to discriminate between patients with damage in the left or right hemisphere.²⁹

Morphing programs with a variable number of features are widely available for personal computers and enable sensitive testing for impairment of facial recognition.

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