



The role of *Funktionswandel* in metamorphopsia

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Patients with metamorphopsia perceive objects or faces as being distorted and/or different in size. In most cases, recognition is not impaired. The stimulus specificity, particularly in the case of face perception, has led to the suggestion that metamorphopsia is a deficit at the entry level of category-specific recognition systems; in this case, the face processing system. An explanation in terms of a visuosensory deficit (i.e. *Funktionswandel*) that affects the perception of specific stimulus categories has not been systematically evaluated. In this study, we report two patients (MZ and CM) who experienced hemi-metamorphopsia for faces after a stroke in the posterior part of the brain. Despite the distortions, they could still match and recognize faces. We carried out a detailed evaluation of their visual-sensory status and found that both MZ and CM had specific problems with discriminating and estimating sizes and shapes, especially in the contralesional visual field. It was concluded that these cases, metamorphopsia was not due to a higher-order perception impairment specific for faces, but rather of a specific impairment in shape perception in the contralesional visual field that proportionally affects the perception of faces.

The term 'metamorphopsia' refers to the clinical condition where a patient perceives the outside world distorted as a result of neurological disease. It is usually classified under the umbrella of hallucinations and illusions. Simple distortions affect the perception of objects that might appear to have changed in size (macropsia/micropsia), shape (metamorphopsia), or colour and clarity. Borders may appear wavy (dysmorphopsia) and there may be loss of colour (achromatopsia). In more complex illusions, objects appear distorted in distance (macroproxiopia/microtelepsia) or appear to recur (palinopsia) (e.g. Tekin & Cummings, 2003).

Metamorphopsia usually lasts for minutes up to hours, and is only rarely observed as a stable phenomenon lasting days or weeks. It can be accompanied by (transient) hemianopia, when lesions approximate the visual cortex and the geniculostriate

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radiations, but do not involve these. Moreover, achromatopsia has been found in these patients as well. In patients with metamorphopsia, right-sided unilateral or sometimes bilateral lesions have been found in the occipital cortex¹ (Whitely & Warrington, 1977), with extensions to the temporal and parietal cortex (Brau, Lameiro, Liaguno, & Rifkinson, 1986; Brust & Brehens, 1977; Lance, 1976; Safran, Kline, Glaser, & Daroff, 1981; Taguchi, Akai, Watanabe, Kuroda, & Ioku, 1980), and to the retrosplenial area including the posterior part of the gyrus cinguli and the corpus callosum (Ebata, Ogawa, Tananka, Mizuno, & Yoshida, 1991). Patients with (hemi-)metamorphopsia may complain that face or object perception is distorted in such a way that usually (half of) the face or an object is perceived as being different in size (both smaller and larger), distorted (stretched, flattened, and vaulted), or that specific features are not on their appropriate location (e.g. eyes, mouth, and nose). Potzl (in Brain, 1941) described metamorphopsia as a spatial form of agnosia, along with visual disorders of spatial orientation and paralysis of gaze. Besides distortions in half fields, it has also been found in quadrants, or sometimes even smaller regions adjacent to homonymous scotomas (Bender & Teuber, 1949).

An intriguing observation concerns the stimulus specificity of metamorphopsia. Bodamer (1947) described a patient with metamorphopsia restricted to faces to argue for a specific face recognition system in the brain. This patient experienced episodes in which faces – but not other objects – looked ‘mouldy’ and ugly. Whitely and Warrington (1977) reported a patient who described human faces as fish heads. Finally, in some cases the distortions are experienced as positive; Seron *et al.* (1995) described a patient with metamorphopsia for faces who claimed that the distorted faces looked more pleasant.

An important distinction with visual agnosia is that, even though faces are perceived as being distorted, face recognition is still intact (Bodamer, 1947). Despite the sometimes grotesque distortions, patients with metamorphopsia can still recognize the objects or faces that they see (Bodamer, 1947, but see also Whitely & Warrington, 1977).

Given its stimulus specificity, metamorphopsia for faces has often been interpreted as a deficit at the entry level of a face-specific processing system. This claim, however, is controversial and reminiscent of the long-standing debate concerning higher-order recognition deficits. Several models have been proposed regarding the processes involved in face processing, of which the model by Bruce and Young (1986) have proven to be very influential (Figure 1).

In this model, different processing routes are described for the analysis of facial expression, for the perceptual scrutiny of faces for short-term recollection of unfamiliar faces, for the perception of movements of the mouth (speech reading), and for recognition of faces. According to this model, at the first stage of normal face perception, structural encoding takes place, during which facial parts, such as size and the position of nose, eyes, and mouth are processed. At the second stage, a more abstract representation is created, in order to allow for recognition of the face under many different viewing conditions (i.e. Marr’s 3D representation). This idea is very similar to the proposals of, for instance Humphrey and Riddoch (1987), concerning object recognition. Subsequently, the retrieval of autobiographical information about the face can be initiated and this could result in access to the name of the person.

¹ It is important to note that, metamorphopsia has also been found after retinal pathology, such as macular holes (e.g. Saito et al., 2000). One of the methods used to detect functional visual changes is the Amsler chart test, with which distortions (e.g. pincushion distortions (Saito et al., 2000)) in a grid can be detected.

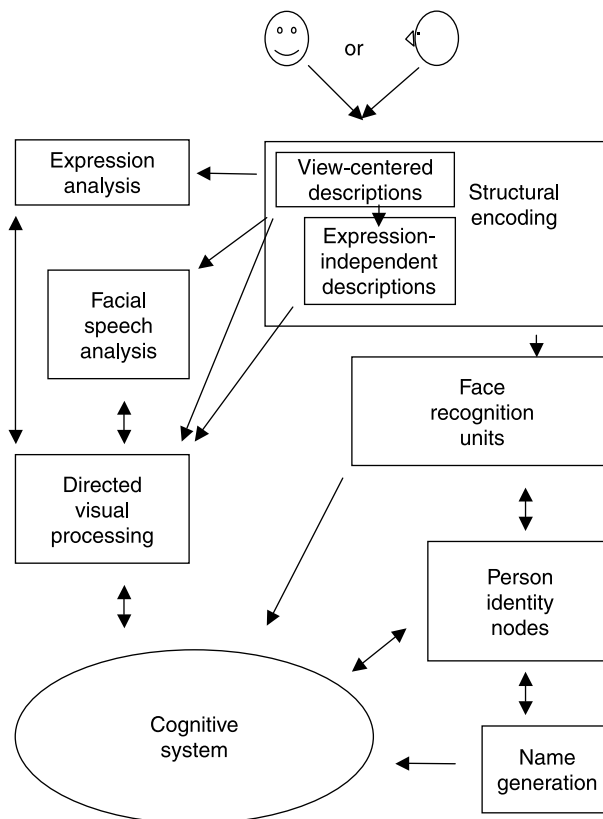


Figure 1. Overview of the model by Bruce and Young (1986).

In short, these models propose a number of separable subprocesses within the area of face processing. In the case of metamorphopsia with spared recognition of face recognition, it might be that the impairment is at the level of structural encoding or directed visual processing (Figure 1).

Historically, it was Bay (1953) who claimed that the concept of associative agnosia (Lissauer, 1890) was ill conceived. In Bay's view (see also Campion & Latto, 1985), these stimulus-specific recognition disorders (i.e. higher-order, cognitive disorders) resulted from low-level visuosensory impairments: 'on its way to perception, the peripheral stimulus undergoes (. . .) an enlarged and instable transformation, disturbing the constancy of visual objects (*Funktionswandel*)'. 'Low-level' visuosensory processing concerns the cortical processing of the incoming (optic radiation) information in the visual areas V1-V5. This concerns mechanisms for visual cues, such as shape, motion, and colour, in the contralateral visual field. 'Higher-order', more cognitive mechanisms are involved in more advanced processing, such as in reading and object perception and recognition. Although these levels of processing are obviously intrinsically interrelated, there is abundant evidence for suggesting that these levels are neuroanatomically and functionally distinct. According to Bay (1953), the constancies of visual objects are the first to be affected when transformations are disturbed and this *Funktionswandel* affects certain recognition skills more than others. This suggestion has been refuted by Ettliger, Warrington, and Zangwill (1957) and by De Haan, Heywood, Young, Edelstyn,

and Newcombe (1995). Although patients with a recognition deficit may have sensory impairments, other patients who do not experience recognition problems can show equal or worse sensory impairments. Therefore, sensory status alone could not explain the presence or the absence of recognition disorders. Despite these findings, Bay's explanation may still hold in individual patients. The central issue raised by Bay is also applicable to metamorphopsia. One should first exclude an explanation at the visuosensory level of perception, before higher-order impairments (e.g. recognition, associations) are inferred. This particular issue has not yet been addressed with respect to metamorphopsia for faces. In this study, we describe two patients (MZ and CM) who experienced face-specific distortions after a stroke in the posterior parts of the brain. Our aim was to chart their subjective visual experience of the outside world and to investigate their visual-sensory status in detail to see whether or not they suffered from low-level impairments that might explain this experience. In Bay's terms, it might be that *Funktionswandel* underlies face-specific distortions in metamorphopsia.

Case descriptions

MZ

Patient MZ is a 60-year-old woman who suffered haemorrhagic venous infarction of the left temporo-occipital cortex with sub-cortical extension after embolization of a dural arteriovenous fistula (see Figure 2). MZ was neuropsychologically examined 9 days post-stroke. She complained about (parts of) faces in her right visual field appearing to become smaller and to bulge. With confrontation testing, no signs of visual field defects, neglect, or extinction were found. When asked to describe what she saw when looking at a face, she came up with the following description: 'When looking at a face, the right side starts to distort within seconds; the right side becomes smaller and the eye starts to disappear outside the face. Nose, mouth, and eye brows are displaced as well and bulge, but are still seen inside the face'. She reported that she only saw these distortions with faces. However, when a cup with two spoons was shown at eye level, she reported that the right spoon became somewhat distorted as well, although not as much as the face. Despite the distortions, she could still recognize familiar faces. This was confirmed in an *impromptu* test in which she was asked to recognize members of her family or her neurologist.

CM

CM is a 59-year-old woman who suffered a haemodynamic TIA in the right posterior hemisphere (arteria cerebri media). CM was neuropsychologically examined 4 days post-stroke. CM complained that ever since the stroke, her perception of faces has altered; only faces, both real faces and faces in pictures (e.g. in magazines), become distorted within seconds after perceiving them. There are no signs of neglect, extinction, or hemianopia upon confrontation. When asked to describe the distortions, she explains that it was hard to look at the faces, as the distortions make the faces look ugly on one side only, namely the left. The left side of faces were seen 'as through a glossy mirage'. Despite the distortions, she could still recognize familiar faces. This was confirmed in an *impromptu* test in which she was asked to recognize members of her family or her neurologist. After about 5 weeks, CM reported that the facial distortions were not as obvious as before. It is unclear whether they had really become less obvious or whether CM had become acquainted to them.

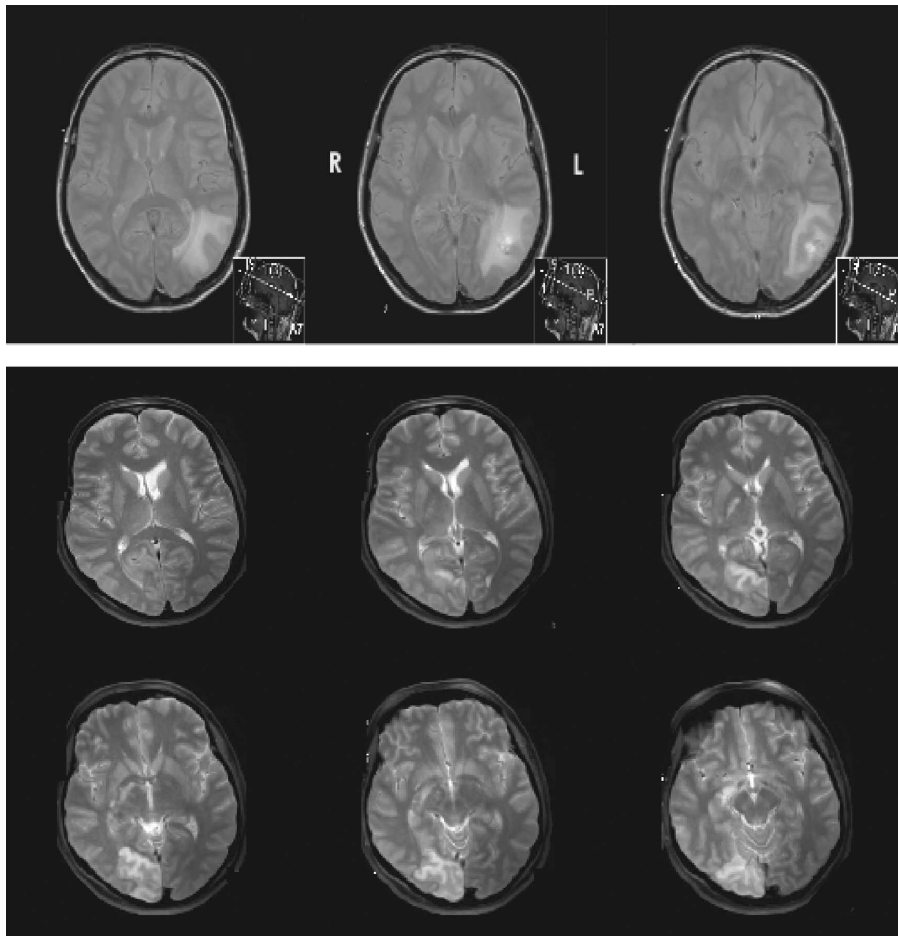


Figure 2. MRI scans of MZ's brain (upper panel) and CM's brain (lower panel).

Neuropsychological examination

The neuropsychological examination consisted of the Cognitive Screening Test (orientation in time and place), the Boston Naming Task (word finding), the Rey Auditory Verbal Learning Test (verbal memory), the Benton Facial Recognition Test (face matching and recognition), BIT Star Cancellation (neglect), the Visual Object and Space Perception Battery (VOSP, perception), and the Cortical Vision Screening Test (CORVIST, higher cortical visual function). An overview of the neuropsychological data is shown in Table 1A in the addendum.

MZ

The orientation in time and place was normal. There were no indications for problems in word finding. The recognition of faces was normal. There were no signs of neglect. Although MZ did not complain about her memory, her performance on the Rey Auditory Verbal Learning Test was defective on both direct and delayed recall, whereas recognition of was intact.

Object and space perception, as measured with the VOSP and CORVIST (shape detection, face perception, crowding, and dot counting), were intact. The shape discrimination and size discrimination tests of the CORVIST were defective. Interestingly, she was not able to indicate the right order from big to small of four squares. MZ also made several mistakes in the fragmented number test of this screening test.

In addition to the standard neuropsychological tests, MZ was asked to draw different objects: (a) a dog; (b) a house; (c) the face of the neuropsychologist who sat in front of her; (d) a cube; and (e) a clock. The drawing of the dog was relatively normal (Figure 3a), and the drawing of the face of the neuropsychologist was only mildly distorted: the right part of the face was drawn a bit lower and smaller than the left side (Figure 3c). The three-dimensional drawing of the house, however, was abnormal, as she was not able to draw the right perspective (Figure 3b). When she was asked to draw a cube, she could not remember how to do it (Figure 3d), but copying was intact. She was able to draw a face of a clock (Figure 3e), but was not able to put the hands in the right position, in this case 11.10 am. In sum, despite the distortions in perceiving faces, MZ was quite able to draw the face of a person sitting opposite to her, but she had more problems with drawing perspective from memory (i.e. the house and the cube).

Taken together, the neuropsychological examination showed disorders in (verbal) memory, visual function, and visuoconstruction.

CM

The orientation in time and place was normal. There were no indications for problems in word finding. The recognition of faces was normal. There were no signs of neglect, but there might have been problems with visual attention, as she missed some items throughout the test. Her performance on the Rey Auditory Verbal Learning Test was above average on the direct recall and average on the delayed recall, but recognition was (borderline) defective. On the Rey Complex Figure, her performance is defective on copying, suggesting an impairment of visuoconstruction. In the delayed recall of the Rey Complex Figure, her performance is also defective: size and location of the subfigures were clearly misjudged, especially on the left.

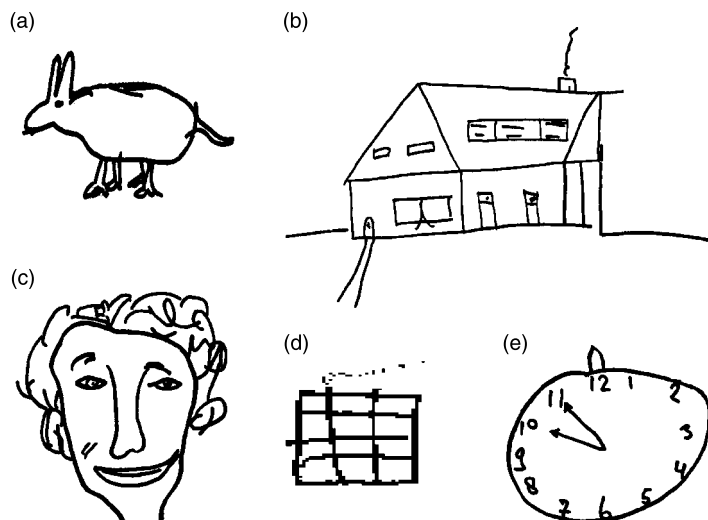


Figure 3. Different drawings of MZ: (a) dog; (b) house; (c) copy of a face; (d) cube; (e) clock.

Object and space perception, as measured with the VOSP and CORVIST (shape detection, face perception, crowding, and dot counting), were intact. The shape discrimination and size discrimination tests of the CORVIST were defective.

Unfortunately, CM was unable to participate on the additional drawing tasks, as she became very tired. However, her performance on the Rey Complex Figure illustrated that she had problems with perceiving sizes and locations of subfigures in the figures, especially on the left.

Taken together, the neuropsychological examination showed disorders in (verbal) recognition, visual function, and visuoconstruction.

In order to investigate the visuosensory status in more detail, we adapted a methodology developed by De Haan *et al.* (1995). In this study, we wanted to examine the visual thresholds of MZ and CM in an odd-one-out, 3-up-1-down paradigm with different visual features, e.g. shape, luminance, and colour. In an experimental set-up, we were able to compare the thresholds of the different visual features presented in the patients' contralesional visual field with the same stimuli in their ipsilesional visual field. Moreover, we were able to compare their thresholds per hemifield with the mean thresholds of a control group of healthy participants.

Visual sensory evaluation

Apparatus

Data collection and stimulus presentation were controlled by a PC and two 17 inch monitors; one for presenting the stimuli to the participants and the other for the experimenter. Fixation control was managed with an eye-tracking system, iView X, that was interfaced with the experimental setup. The delay between an eye-movement and the stimuli disappearing from the screen was 2 frames. The experiment was programmed using Visual Basic 6.

Stimuli were presented on one side of the monitor, while participants were fixating on the other side (for examples of the stimuli at start, see Figure 4). Stimuli for the shape discrimination (Figure 4a) task were two squares (3.2×3.2 cm) and one rectangle (6.4×1.6 cm, at start), resulting in different shapes with the same area (10.24 cm²). After three correct responses, the shape of the rectangle changed in such a way that it resembled the squares more.

Stimuli for the luminance discrimination (Figure 4b) were three squares of the same size (3.2×3.2 cm), one of which differed in brightness. At the start, two of the stimuli were black ($xyY = .304, .319, \text{ and } .480$) and one was white ($xyY = .318, .330, \text{ and } 105.33$), or vice versa. After three correct responses, the Michelson contrast between the stimuli became smaller, resulting in more greyish stimuli.

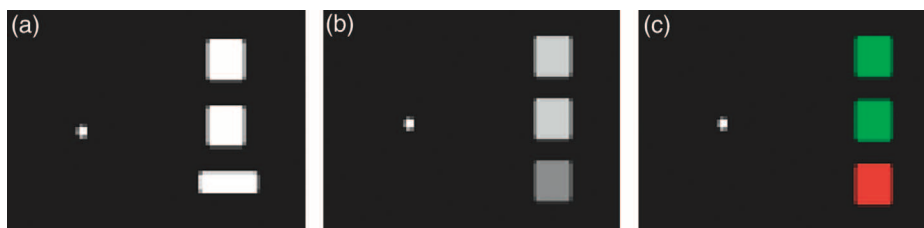


Figure 4. Examples of the stimuli in (a) the shape, (b) luminance, (c) and colour discrimination tasks. Fixation is on the left and stimuli are presented on the right.

Stimuli for the colour discrimination (Figure 4c) were three squares of the same size (3.2×3.2 cm), one of which differed in colour; two of the stimuli were green ($xyY = .287, .585, \text{ and } 64.30-92.40$) and one was red ($xyY = .622, .339, \text{ and } 23.30-32.00$), or vice versa. The two stimuli with the same colour differed in luminance, to rule out the possibility that the task could be done based on luminance differences between the two different colours alone. After three correct responses, the colour contrast between the stimuli became smaller, resulting in more yellowish stimuli.

Viewing distance was 89 cm, and stimuli were presented 14.2° left or right from fixation.

Procedure

For fixation control, a 9-point calibration procedure was done first. The participants were instructed to fixate the fixation cross throughout the experiment. Whenever participants made an eye-movement towards the stimuli, the stimuli disappeared and only when they were fixating again, the stimuli re-appeared on the screen. Eye-movements were recorded with an iView X system to control fixation and disappearance of the stimuli.

The experiment started with the shape discrimination, followed by the luminance and colour discrimination. The participants were instructed to indicate which of the three stimuli was different, either based on shape, luminance, or colour. Before each task, they were told which feature to discriminate. Responses were given by pressing a specific button on a response box: when it was the upper stimulus that was different, they were told to press the upper button; when it was the lower stimulus, they should press the lower button; and when it was the stimulus in the middle, they should press the button in the middle. There were 45 trials per task and after each incorrect response the differences between stimuli became 50% bigger, making the judgments easier (reversal). After three correct responses, the differences between stimuli became 50% smaller, making the judgments more difficult (continuation). In this manner, a threshold per task could be obtained, based on the mean of the last six reversals.

Results

The thresholds of MZ and CM were compared with the thresholds of a group of 26 healthy control participants per task per side, as well as to their own thresholds on the other side.

For comparing the thresholds of the patients with the mean thresholds of the control group, we used Crawford and Howell's (1998) significance test on differences between individual's score and control sample. We used a Bonferroni correction for multiple comparisons. Mean thresholds for the healthy controls are shown in Figure 5.

As can be seen in Figure 5, the thresholds for the luminance and colour discriminations are comparable for left- and right-sided presentations for the group of control participants ($t(25) = -.761, p > .2; t(25) = -.013, p > .5$). The threshold for the left-sided presentation of the shape stimuli is lower than the threshold for the right-sided presentation ($t(26) = 5.22, p < .001$). It seems that the right hemisphere is somewhat better in discriminating shapes than the left hemisphere. The thresholds of MZ and CM are illustrated in Figures 6 and 7, respectively.

MZ

When comparing the thresholds on the three different tasks with left-sided presentation of the stimuli, the thresholds of MZ on the shape, luminance, and colour discriminations

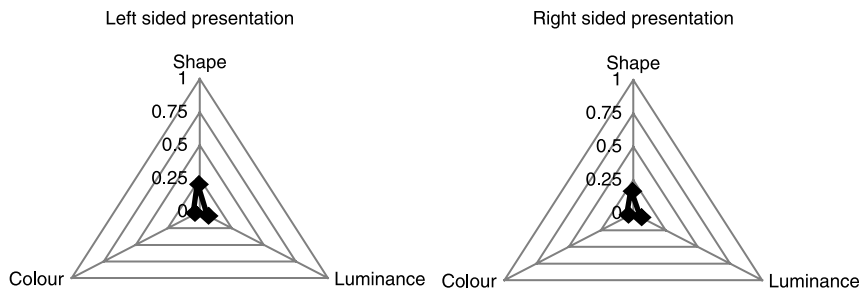


Figure 5. Mean thresholds of the age-matched controls on the shape, luminance, and colour discrimination tasks, split on side of presentation.

are comparable with the thresholds of the controls ($t(25) = .378, p > .5; t(25) = -.244, p > .5; t(25) = -.195, p > .5$, respectively). For right-sided presentations, there was a significant difference for the threshold on shape discrimination ($t(25) = 7.531, p < .001$), whereas there were no differences between thresholds of the healthy controls and MZ on the luminance and colour discriminations ($t(25) = -.502, p > .5; t(25) = .268, p > .5$, respectively). An important observation during the shape discrimination with right-sided presentation is that especially when the odd-one-out was presented in the upper right position, incorrect judgments were made. When asked to describe what the stimuli looked like, MZ explicitly responded by stating that: ‘most of the time the upper square is much smaller’. When asked to indicate the width and height of the upper right square, it became apparent that a rectangle of 6.4×1.6 cm as well as a square of 3.2×3.2 cm appeared much shorter in width and only mildly shorter in height (approximately 4.1×1.4 cm and 2×2.6 cm). With presentations on the left, size estimations were much more adequate: approximately 6×1.5 cm and 3×3 cm.

CM

When comparing the thresholds on the three different tasks with right-sided presentation of the stimuli, the thresholds of CM on the shape, luminance, and colour discriminations are comparable with the thresholds of the controls ($t(25) = -1.312, p > .1; t(25) = -1.12, p > .1; t(25) = -.221, p > .1$, respectively). For left-sided presentations, there was a significant difference for the threshold on shape discrimination ($t(25) = 4.011, p < .001$), whereas there were no differences between thresholds of the healthy controls and CM on the luminance and colour discriminations ($t(25) = -.846, p > .1; t(25) = -.221, p > .1$, respectively).

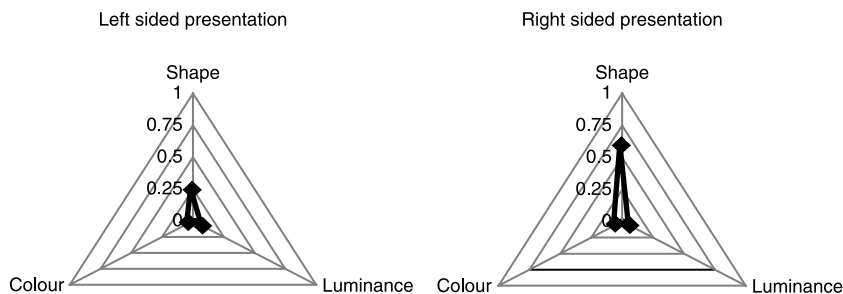


Figure 6. Mean thresholds of MZ on the shape, luminance, and colour discrimination tasks, split on side of presentation.

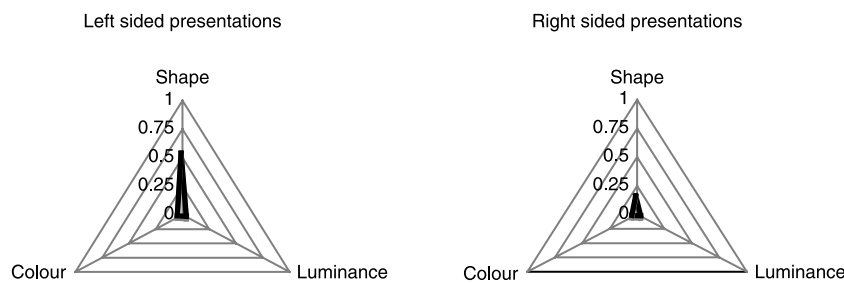


Figure 7. Mean thresholds of CM on the shape, luminance, and colour discrimination tasks, split on side of presentation.

Discussion

We examined two patients (MZ and CM) with hemi-metamorphopsia, who reported selective distortions in perceiving faces. MZ had suffered haemorrhagic infarction in the left temporo-occipital cortex, which has been suggested to be involved in complex hallucinations. CM suffered a haemodynamic TIA in the right posterior hemisphere. Metamorphopsia is usually found after right-sided unilateral lesions and lasts from minutes up to hours. MZ, however, experienced face-specific distortions after a left-sided lesion in the temporo-occipital cortex. The face-specific distortions lasted up to 6 weeks. Also, CM suffered from the distortions for about 5 weeks. We investigated the visual-sensory status in detail to see whether or not the patients suffered from low-level impairments that could help to explain this experience. Both patients complained only about face-specific distortions. However, when a cup with two spoons was shown at eye level, MZ reported that the right spoon became somewhat distorted as well, although not as much as the face. Moreover, she told us that she never saw distortions in objects before, probably as the distortions were very small. The main point here is that the metamorphopsia is more general in the sense that it also affects other objects. This observation is in line with a visuosensory basis. It is interesting that the subjective complaints mainly concerns face perception. Thus, a low-level visual deficit may cause a relatively category-specific perception problem. We can only speculate about the reason for this. Perhaps, it is the fact that faces need to be scrutinized continuously to detect subtle changes in expression.

We found that both MZ and CM had specific problems with discriminating and estimating (sizes of) shapes. For MZ, elevated thresholds were observed in shape discrimination when stimuli were presented in the right visual field, especially in the upper right quadrant. For CM, elevated thresholds were observed in shape discrimination when stimuli were presented in the left visual field. Colour and luminance perception were unimpaired in both patients. The fact that we found differences in thresholds for shape perception suggests that metamorphopsia is not merely an illusion or hallucination, but might be a result of a selective deficit in shape and size perception. In other words, low-level impairments in shape perception were found that might help to explain the experience of distorted face perception. This finding seems to be in line with Bay's proposal (1953) that the perception of an object in a visual field depends on the integration of the whole visual field. Bay (1953) divided perception in two different processes: first, purely physiological for elementary sensations; second, psychological for integrating and transforming elementary sensations into perceptions. During these transformations, which Bay called

Funktionswandel, constancy of visual objects can be disturbed due to instable transformations. As a result, disorders of visual function, e.g. agnosias, can be found in absence of any clear visual field defect. Bay (1953), however, suggested that an impairment in the sensory processing could explain gnostic disturbances in neurological patient. For instance, contrast perception might be unimpaired, while the perception of subtle pattern differences and fine shades of texture could be impaired. These perceptual skills are very important for face recognition, and therefore, these low-level deficits might have caused prosopagnosia. Bay (1953) therefore states that when methods are used which are sufficiently sensitive, low-level perception deficits will be found.

The finding that both in MZ's and CM's low-level impairments in shape processing were observed in addition to their metamorphopsia supports Bay's proposal (1953) in the sense that low-level impairments in shape processing found in the patients might have caused the metamorphopsia. In contrast, Bay's suggestion is not completely supported as MZ and CM did not have any difficulty in recognizing or matching faces, despite the distortions in face perception.

Our finding argues against the interpretation that metamorphopsia is in these cases a consequence of impairments at the entry level of a face-specific processing system, as both patients did not show signs of problems with recognizing and matching faces, despite the selectivity of their perceived distortions. So even though faces are special and seem to have their own processing system, specific impairments in face processing might also result from impairments in shape perception. Even though one might argue that at the entry level of normal face perception, structural encoding takes place, during which non-changing facial parts are processed, it seems unlikely that this encoding is defect in metamorphopsia, but rather that processing of face-independent shapes of sizes is impaired. Exactly why it is that faces are disproportionately affected by these visuosensory deficits, one can only speculate about. It may be that a system specifically processing faces is more vulnerable for impaired visuosensory input, as faces are changing all the time (e.g. blinking of the eyes, movement of the eyes, emotional expressions, and speech).

We do not want to suggest that this explanation will hold for all metamorphopsic patients. Our data do not preclude the possibility that metamorphopsia may also arise as a consequence of a higher-order deficit. In this context, it is interesting to note that in both of our patients the metamorphopsic episodes lasted much longer than is usually described in the literature. This might have been the result of the (additional) problems with discriminating sizes and shapes.

In summary, we have described two patients with hemi-metamorphopsia for faces as a result of a stroke in the posterior parts of the brain. We observed a specific impairment in low-level sensory processing that affects the perception of shape in contralesional visual field. On the basis of these findings, we surmise that the metamorphopsia in both patients, which also lasted longer than usually described, may be best explained by their low-level sensory impairments. The fact that similar (or even more severe) shape/size perception deficits are commonly observed in patients with damage to the posterior temporo-parieto-occipital cortex who do not complain of metamorphopsia shows that there is no one-to-one relationship between low-level visuosensory deficits and metamorphopsia. In fact, the same logic has been used by Ettlinger *et al.* (1957) and De Haan *et al.* (1995) to argue that metamorphopsia may occur as the result of higher-order impairment, i.e. a deficit in the face perception route of the Bruce and Young (1986) model. However, this observation does not refute the possibility that subtle

visuosensory deficits may cause metamorphopsia in certain patients. The reason why we suggest that this is the case here is based on two arguments. First, the visuosensory deficits in these two patients are restricted to shape, while colour and motion are perceived normally. The metamorphopsic distortions also concern only the shape of the face and not, for instance, the colour. Second, the visuosensory deficits only affect one-half of the visual field. In both patients, this half-field coincides with the side of the face where the distortions are seen. In contrast, if the functional locus of the metamorphopsia is assumed to be in the higher-order face perception system, one would not expect the metamorphopsia to be modulated by the position within the visual field. In fact, there is evidence that face selective cells in the macaque are not spatially sensitive (Tovee, Rolls, & Azzopardi, 1994). The reason that the possible relationship between subtle sensory deficits and metamorphopsia has not been put forward before may be explained by the fact that the method that we have devised to test half-field visuosensory processing is new, i.e. the methodology was lacking to assess these impairments in detail. Finally, the fact that two patients with similar complaints show comparable elevated thresholds on shape/size discriminations further supports the suggestion of a causal relationship. Further investigations into visual half-field defects are needed to be able to explain these relationships in the future.

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Addendum neuropsychological data of MZ and CM**Table 1A.** Overview of neuropsychological data of MZ and CM. Their performance is compared to a normalized control group (age and the level of education)

Neuropsychological tests	MZ's performance	CM's performance
Cognitive Screening Test	17/20	19/20
Boston Naming Task ^a	160/180	152/180
Rey Auditory Verbal Learning Test: total trial 1–5	22*	43
Rey Auditory Verbal Learning Test: delay	3*	7
Rey Auditory Verbal Learning Test: recognition	28/30	27*
Benton Facial Recognition Test	43	42
Star Cancellation	55/56	49/56* ^b
VOSP Screening Test	20/20	20/20
VOSP Incomplete Letters	19/20	20/20
VOSP Silhouettes	18/30	21/30
VOSP Cube Analysis	8/10	8/10
CORVIST Symbol Acuity Test	36/36	36/36
CORVIST Shape Discrimination Test	2/4*	1/4*
CORVIST Size Discrimination Test	1/2*	1/2*
CORVIST Shape Detection Test	8/8	8/8
CORVIST Hue Discrimination Test	4/4	3/4
CORVIST Scattered Dot Counting Test	4/4	4/4
CORVIST Fragmented Numbers Test	5/8*	na
CORVIST Face Perception Test	8/8	8/8
CORVIST Crowding Test	4/4	4/4

*defective score.

^aPlease note that Dutch norms (Van Loon-Vervoorn, 1996) are used for the Boston naming test (4-point scale: 3, correct answer; 2, semantically appropriate answer; 1, semantically related answer; 0, incorrect answer), which results in a total of 180.

^bNo signs of neglect, as the missed stars were on the left as well as on the right, in more or less the same amount.