REVIEW

THE RELATIONSHIP BETWEEN VISUAL PERCEPTION AND VISUAL MENTAL IMAGERY: A REAPPRAISAL OF THE NEUROPSYCHOLOGICAL EVIDENCE

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Abstract

Visual perception and visual mental imagery, the faculty whereby we can revisualise a visual item from memory, have often been regarded as cognitive functions subserved by common mechanisms. Thus, the leading cognitive model of visual mental imagery holds that visual perception and visual imagery share a number of mental operations, and rely upon common neural structures, including early visual cortices. In particular, a single visual buffer would be used "bottom-up" to display visual percepts and "top-down" to display internally generated images. The proposed neural substrate for this buffer consists of some cortical visual areas organised retinotopically, that is, the striate and extrastriate occipital areas. Empirical support for this model came from the report of brain-damaged patients showing an imagery deficit which parallels a perceptual impairment in the same cognitive domain. However, recent reports of patients showing double dissociations between perception and imagery abilities challenged the perception-imagery equivalence hypothesis from the functional point of view. From the anatomical point of view, the available evidence suggests that occipital damage is neither necessary nor sufficient to produce imagery deficits. On the other hand, extensive left temporal damage often accompanies imagery deficits for object form or colour. Thus, visual mental imagery abilities might require the integrity of brain areas related to vision, but at an higher level of integration than previously proposed.

Key words: visual perception, visual mental imagery, spatial imagery, object agnosia, prosopagnosia, achromatopsia, alexia, unilateral neglect, brain damage

Introduction

In a dramatic moment of Ian McEwan's novel *Enduring Love* (McEwan, 1997), the protagonist, Joe, has been aggressed by two individuals. He seeks protection at the police station, where a sceptical police officer asks him to describe the hands of his aggressors. Despite strenuous effort, Joe cannot recall the visual detail, thus loosing further credibility in the policeman's eyes. "I saw the sleeves of long black coats, as dim as blurred daguerreotypes and at the end of the sleeves – nothing. Or, rather, anything. Hands, gloves, paws, hooves" (p. 182). As Joe notes, his inability to explore an otherwise well-formed mental image in order to single out a precise detail is at sharp variance with current theories of visual mental imagery: "Neuroscientists report that subjects asked to recall a scene while under a magnetic resonance imaging scanner show intense activity in the visual cortex, but what a sorry picture memory offers, barely a

shadow, barely in the realm of sight, the echo of a whisper. You can't examine it for fresh information" (p. 182). The police officer concludes the exchange by suggesting a tranquillizer drug. This example underlines the wide diffusion of the idea of an anatomo-functional equivalence between visual imagery and visual perception, and also suggests the responsibilities of neuroscientists in shaping the way people think of their experiences.

This idea has indeed ancient roots. Hume (1739/1978), for example, underlined the "great resemblance" between percepts and mental images "in every other particular, except their degree of force and vivacity" (p. 2). Thus, mental images are "the faint images" of percepts (p. 1). Tye (1991) concludes that "by and large, philosophers historically held that mental images are picture-like representations similar to that occurring in perception" (p. 11). This tradition was sharply criticised by J-P. Sartre (1936), who argued that mental images have a radically different phenomenological status from percepts (Sartre, 1940).

In the domain of cognitive neurosciences, the idea of a strict equivalence between perception and imagery processes is exemplified by the model of visual mental imagery proposed by Stephen Kosslyn (1980, 1994), which is probably the leading cognitive account of visual mental imagery at present. Kosslyn has proposed that visual mental images are depictive, or "quasi-pictorial" representations. "Depictive representations convey meaning via their resemblance to an object, with parts of the representation corresponding to parts of the object" (Kosslyn, 1994, p. 5)[1]. Farah (1984) formalized the perception-imagery equivalency hypothesis in a componential model, whereby a single visual buffer is used "bottom-up", to display visual percepts, and "top-down", to display internally generated images (Figure 1). In long-term memory, the relevant information is represented in a non-pictorial, propositional format.

A great merit of this model (hereafter, for brevity, the Kosslyn model) is that, unlike most cognitive models, it makes an explicit proposal about the nature of the cortical regions subserving the model functions. To give mental images their quasi-pictorial character, the visual buffer shared by perception and

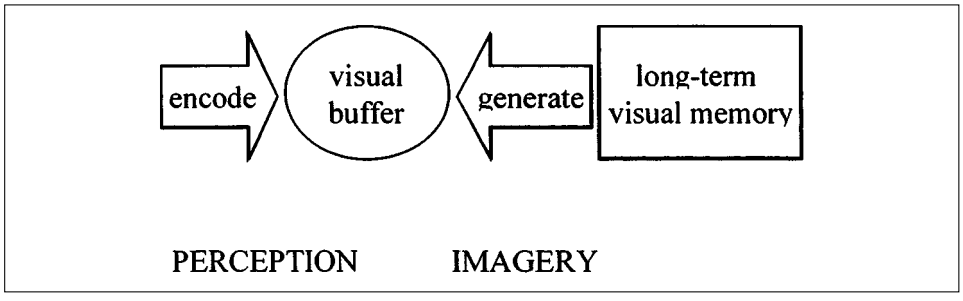

Fig. 1 – *The Kosslyn model of visual mental imagery as formalized by Farah (1984).*

[1]Note that, according to others (see, e.g., Pylyshyn, 1973), information processing in the brain is exclusively propositional-descriptive, in a non-pictorial, language-like fashion. We will not consider here this aspect of the "imagery debate" (see Thomas, 1999, for a discussion of the theoretical and empirical difficulties of both the pictorial and propositional theories and for the proposal of an entirely different theoretical perspective on visual mental imagery, based on the idea that imagery is experienced when perceptual exploratory procedures are put to work in the absence of a perceptual object).

imagery has to be implemented in retinotopically organized visual areas (Kosslyn, 1994; see Damasio, 1989, and Zeki, 1993, for related proposals). Given what we know about the topographical organization of the human visual cortex (Sereno et al., 1995), this hypothesis practically restricts the possible candidates to the striate and extrastriate visual cortices situated in the occipital lobe and in the posterior third of the ventral temporal lobe. As a matter of fact, Kosslyn argued that the most likely neural substrate for the visual buffer is V1, the primary visual areas (Kosslyn, 1994; Kosslyn et al., 1995, 1999). Thus, the Kosslyn model consists of levels of processing which are both functionally and anatomically specified. This remarkable feature offers a double opportunity for neuropsychology to test the model.

After some preliminary remarks on behavioural evidence on normal individuals, the present review will focus on reports about brain-damaged patients' performance on tasks tapping their perceptual and imagery abilities. Given the enormous amount of studies dealing with this subject, and the fact that a number of scholarly reviews are already available in the literature (see, e.g., Farah, 1984, 1988; Goldenberg, 1993, 1998; Trojano and Grossi, 1994), the present review will necessarily be selective, and will particularly favour studies that were either recently published, or not discussed in the available literature.

Interpreting Behavioural Evidence on Normal Individuals

Models of perceptual-imagery equivalence, such as the Kosslyn model, were originally motivated by empirical evidence of interaction between perception and imagery coming from experimental psychology, such as the Perky effect. In a now classic study, C.W. Perky (1910) asked participants to imagine an object (e.g. a banana or a leaf) while fixating onto a blank screen. At the same time, unknown to the participant, an image of that object was gradually projected onto the screen, with increasing definiteness, starting below the threshold for conscious perception. All the subjects continued to believe that they were just imagining the stimulus, even when its intensity was well above the perceptual threshold. The projected stimulus, however, influenced subjects' experience, in that they noticed that their images changed according to the picture they were presented with; for example, subjects remarked that the banana was vertically, and not horizontally oriented as they initially imagined; or found themselves imagining an elm leaf while they had been trying for a maple leaf. These effects demonstrated an interference of perception on imagery, and were consequently interpreted as evidence of shared function between these two domains. However, what Perky's subjects experienced was not, as usually claimed, a detrimental effect of imagery on perception; instead, they had an imagery-induced deficit of *awareness* of perception. Subjects did have a conscious experience of the relevant object; what was defective was their insight about the *source* of this experience as being perception or imagery. In other words, Perky's subjects were deceived about which psychological process was leading to their awareness of an object. This result is intriguing but hardly surprising, because subjects did not expect to see anything, and were instead actively trying to imagine

objects. Objects were projected near threshold, and the intensity of their perception was therefore too faint for subjects to be aware of the true source of their experience.

Over half a century later, another line of evidence that motivated models based on perception-imagery equivalence is the finding that the time employed by normal individuals to mentally rotate an imagined object is proportional to the angle of rotation (Shepard and Metzler, 1971). Similarly, the time employed to mentally scan an image increases with the distance to be scanned (Kosslyn, 1980). But, as Harman (1989, note 2) pointed out, it is a mistake to confound the properties of our imaginal experience with properties of the images themselves. When we say that we rotate or scan a mental image, we actually mean that we imagine ourselves while rotating or scanning that image. The positive correlations between real and imagined time to perform such activities only indicate that we can do so with reasonable accuracy, which, once again, is not so surprising.

Thus, these behavioural data on normal subjects do not provide unequivocal evidence about the format of visual mental images.

Domains of Visual Mental Imagery

According to Georg Goldenberg (1993), there are at least five kinds of visual entities whose imagery can be independently affected by brain damage: shapes of objects, colours of objects, faces, letters and spatial relationships. Unilateral neglect for mental images (Bisiach and Luzzatti, 1978; Bisiach et al., 1981; Bartolomeo et al., 1994; Coslett, 1997) might be considered as a special case of impairment of imagery for spatial relationships. Martha Farah and her co-workers (Farah et al., 1988a; Levine et al., 1985) have collected evidence suggesting that visual mental imagery – by and large, imagery for object form and colour, faces and letters – might mainly involve the ventral, or occipito-temporal, stream of visual processing, while spatial imagery (or movement-related imagery, Sirigu et al., 1996) might rather require the integrity of the dorsal, or occipito-parietal, stream of processing (see Mishkin et al., 1983; Milner and Goodale, 1995).

Neuropsychological Dissociations

From the functional point of view, the Kosslyn model of visual mental imagery has been supported by neuropsychological evidence showing imagery deficits which parallel perceptual impairments in the same cognitive domain. According to the Kosslyn model, in the case of a perceptual deficit, a corresponding mental imagery deficit should systematically co-occur, because "no other cortices, and certainly no other higher order, integrative cortices, are capable of supporting the recall of the perceptually impaired feature" (Damasio, 1989, p. 33). Indeed, such an association has often been observed, as described by several case reports reviewed and discussed by Farah (1988). However, it is commonly accepted that the logic of association of impairments warrants the inference of a single mechanism underlying the co-occurrence of multiple deficits only until patients are described who show only one component deficit in isolation. Once a double

dissociation is documented, interpretations can no longer postulate a unitary mechanism (Shallice, 1988). For example, patients showing an association between perception and imagery deficits might have sustained brain lesions large enough to damage several functionally independent but anatomically contiguous cortical regions. Indeed, reports of both preserved perception with impaired imagery and of impaired perception with preserved imagery can be found in recent literature, thus making the case for a double dissociation for these deficits. These studies, most of which were published after 1988, and were thus not covered by Farah's (1988) paper, constitute the primary focus of the present review.

It must be noted that both types of dissociation between perceptual and imagery abilities have been claimed to accommodate the Kosslyn model. Farah (1984) interpreted the isolated imagery deficits as coming from an impairment of the generation process (Figure 2).

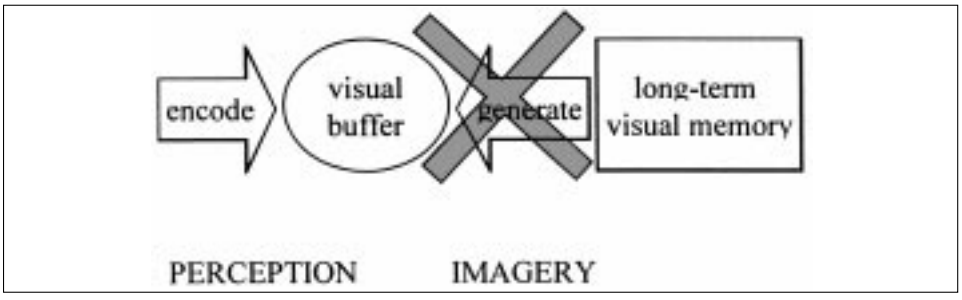

Fig. 2 – *Locus of impairment for isolated deficits of visual mental imagery, as proposed by Farah (1984).*

More problematic for the Kosslyn model seems to be the opposite dissociation, that of impaired perception with spared imagery. Kosslyn (1994) argued that this dissociation could come from an impairment of the sensory encoding system, prior to the visual buffer (Figure 3).

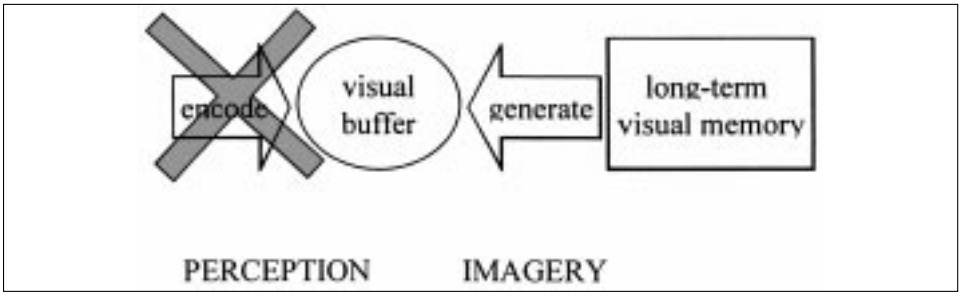

Fig. 3 – *Locus of impairment for perceptual deficits with preserved imagery, as proposed by Kosslyn (1994).*

If however, the visual buffer is subserved by occipital visual areas (Kosslyn, 1994; Kosslyn et al., 1995, 1999), one wonders where the anatomical locus of impairment should be located.

1. Isolated Deficits of Visual Mental Imagery

Probably the first report of a relatively isolated deficit of mental imagery is that concerning M.X… (Charcot and Bernard, 1883). This patient used to experience particularly vivid visual images, which he exploited as a mnemotechnics, until one day he found to have completely lost his visual memories of forms and colours as well as any visual experience in dreaming. The only other neuropsychological deficit in this patient was apparently a reading disorder without agraphia. No information is available about the anatomical locus of lesion in this early report.

More recent cases of imagery deficits, in whom the brain lesions were documented, point to the importance of the temporal lobes (and particularly of the left temporal lobe) for imagery abilities. In the patient with visual imagery deficit described by Basso et al. (1980), CT scan showed an area of reduced density involving the lower mesial region of the left occipital lobe and part of the juxtaventricular structures of the left temporal lobe with possible inclusion of the hippocampus. Patient RM (Farah et al., 1988b) was impaired in imagining object form and colour (despite his claim of being capable to picture objects in his mind). CT showed a left occipital and medial temporal infarct. Also patient DW, described by Riddoch (1990) as having a deficit of image generation, had a left temporal-parietal lesion. There was no occipital damage apparent on CT scan. Interestingly, this patient was able to perform a number of operations on perceptually derived images, suggesting that his visual buffer was intact. This led Riddoch to postulate the existence of "an imagery buffer, separated from (and perhaps more abstract than) a visual buffer" (p. 269). Extensive left temporal damage is also evident in the MRI scan of patient RG, described by Liliane Manning (2000). This patient presented an association of optic aphasia, pure alexia, associative agnosia and an imagery impairment for object form and colour. Although RG had problems recognising body parts, faces and objects, his imagery impairment seemed to be much more profound than this perceptual deficit, because he was unable to conjure up images of items which he could recognise (but not name on visual presentation, due to optic aphasia). Also patient KQu, described by Goldenberg (1992) had extensive left temporal damage and an imagery deficit for colour and form of objects. Sirigu and Duhamel (2001) recently reported on a patient (JB), who, after lesions to both temporal poles due to herpes simplex encephalitis, showed initially prosopagnosia and object agnosia, together with a complete inability to revisualise the shape or colour of objects and faces and the form of letters. Later, however, the perceptual deficits disappeared, leaving a profound and isolated impairment of visual mental imagery.

Thus, in the available cases of (relatively) isolated deficits of visual mental imagery, the left temporal lobe seems always extensively damaged.

2. Patterns of Dissociation between Perceptual and Imagery Abilities

Cortical Blindness and Hemianopia

A first domain of dissociation between impaired perception and spared imagery concerns elementary visual deficits arising from cortical damage. The

Kosslyn model would predict that cortically blind patients be unable to form visual mental images, because they lack the neural substrate for the visual buffer. Farah (1988) pointed out in her review of the literature that many cortically blind patients do indeed seem unable to use mental imagery, but also noted the lack of sufficiently detailed information to reach a firm conclusion from the reported cases. However, reports of cortically blind patients with spared visual imagery are not rare. Already the case reported by Anton more than a century ago (Anton, 1899) seemed to have spared visual imagery. Goldenberg (1995) reported another such case in which the visual images experienced by the patient apparently nourished her anosognosia. She claimed to "see" things that actually were her visual mental images. The author raised the possibility that the preserved visual imagery in his case could depend on the presence of small islands of spared visual cortex, but offered several arguments against the necessity of primary visual cortex for mental imagery. Particularly compelling about the relationship between cortical blindness and visual mental imagery is the evidence presented by Chatterjee and Southwood (1995). They studied three patients with cortical blindness. Two of these patients had bilateral medial occipital damage, but they nevertheless showed an intact capacity for imagining object forms. On the other hand, the third patient had damage extending well forward into the left temporal lobe, and he was unable to imagine object forms. Policardi et al. (1996) studied another cortically blind patient, and found impaired visual mental imagery. MRI showed no focal brain damage, but PET revealed reduced metabolism in the occipital and temporal regions.

Butter et al. (1997) reported on eight patients with unilateral visual field defects who were worse at imagining dots on the side of their visual field defect than on the other side, a replication of analogous findings obtained by Farah et al. (1992) in a single patient. Butter et al. (1997) interpreted their data as consistent with the claim, central to the Kosslyn model, that occipital visual areas are essential to visual mental imagery. However, there are several problems with this interpretation. First, three patients out of eight did not undergo any neuroimaging studies; consequently, a lesion extending beyond the occipital cortex could not be excluded (such a lesion was indeed present in two of the remaining patients). Second, one hemianopic patient performed the imagery task normally, contrary to the predictions of the Kosslyn model. Third, even in the case of lesions anatomically restricted to the occipital cortex, functional damage may extend to other cortical areas, as it actually happened in the patient described by Policardi et al. (1996). This possibility could not be excluded in the Butter et al.'s (1997) series, because no functional brain images were obtained. Moreover, in at least one patient of the series (MK), a functional impairment of the left temporal lobe is strongly suggested by the occurrence of an anomic deficit. Structural or functional damage to visual association areas might therefore account for the imagery deficit observed by Butter et al. (1997).

Object Form

One of the first reports suggesting spared imagery for object form in visual object agnosia was the study of patient HJA (Humphreys and Riddoch, 1987;

Riddoch and Humphreys, 1987), who had bilateral infarcts of the inferior temporo-occipital gyri, inclusive of the lingual and fusiform gyri, greater on the left than on the right side, with extension into the left anteromedial temporal lobe sparing the hyppocampus. BA 17 and 18 were relatively spared. Area 36 was affected bilaterally with extension to posterolateral area 28 and posterior area 20 on the left. These lesions provoked a dense visual agnosia, prosopagnosia, pure alexia, achromatopsia and topographical impairment.

Despite this, HJA was able to draw from memory objects that he could no longer recognise visually, thus suggesting preserved visual mental imagery for these objects. This, however, seems not to have been the case for colour imagery (see below, *Object Colour*). But other important issues emerged from the follow up of this patient. Riddoch et al. (1999) had the chance of retesting HJA after 16 years from lesion onset, and found that his perceptual impairment was stable; his visual knowledge for objects had, however, deteriorated. In drawing from memory and in verbal descriptions the patient gave fewer visual, and more functional details than before. With forced-choice testing, he still responded at ceiling. The authors thus made the important methodological point that current tests of visual imagery (usually based on forced-choice procedures) may not be sufficiently sensitive to detect subtle impairments of visual memory. However, it is not warranted from these considerations that all the reports of dissociations between preserved imagery and impaired perception are artifactual. For example, at least in the case of Madame D (Bartolomeo et al., 1998a), phenomenological evidence was consistent with the outcome of imagery tests; she claimed to experience perfectly vivid visual mental images. More generally, what is striking in some of the reported dissociations is the contrast between a profound impairment in perception and the spared imaginal capacities. Riddoch et al. (1999) concluded that the perceptual impairment prevented HJA from up-dating his visual knowledge[2]. These results suggest that visual semantic memory is not constructed once and for all, but needs perceptual practice to be maintained[3]. Drawing on these results, Riddoch et al. (1999) further speculated that "the conclusion that visual memory is intact in cases of apperceptive agnosia is perhaps premature", and that "[their] data are quite consistent with the notion that visual perception and long-term visual memory utilize common neural strata (e.g., in imagery tasks)" (p. 555). These last conclusions, however, do not seem to follow from the data. If, for example, a person who has been blind for years as a consequence of corneal damage had forgotten a number of visual details of objects, one might conclude that this depends on a lack of perceptual practice, but one could not infer from this that the cornea is a crucial structure for visual imagery.

Further evidence of sparing of object form imagery in visual agnosia came from the performance of patient MD (Jankowiak et al., 1992), who became

[2]A similar case might perhaps be made for patients MH (Ogden, 1993) and TC (Policardi et al., 1996), who, after several years of perceptual impairment, were found to have defective visual imagery. Unfortunately, no information is available about their visual memories at a time closer to the brain injury.

[3]Conversely, it has been shown that adult practice can shape even relatively basic aspects of visual perception, such as the ability of discriminating between digits and letters (Polk and Farah, 1995). Riddoch et al.'s (1999) results extend this notion to semantic memory.

profoundly agnosic after sustaining bilateral occipital lesions with extension into the inferior and middle temporal gyri and angular gyri. However, he had excellent visual imagery for object form, and could make very detailed drawings from memory. Patient DF (Servos and Goodale, 1995), who had bilateral damage of BA 18 and 19, with spared BA 37, showed a similar pattern of dissociation, with preserved visual imagery despite a profound visual form agnosia. Aglioti et al. (1999) studied another profoundly agnosic patient, who had a posterior brain atrophy, most marked in the occipital and parietal lobes. Also for this patient mental visual imagery for object form appeared intact.

Patient CK has been studied in great detail by Marlene Behrmann and her co-workers (Behrmann et al., 1992, 1994). CK developed a profound visual object agnosia after sustaining a head injury. He was also impaired in reading and in recognizing single letters. Face recognition and colour perception were intact. Despite his perceptual impairment, CK could produce detailed drawings from memory and showed preserved capabilities in an extensive series of visual imagery tasks. There was no brain damage detectable at MRI.

More recently, we had the opportunity to document a similar dissociation between impaired perception and preserved imagery in a patient with bilateral extrastriate lesions (Bartolomeo et al., 1998a). Madame D became severely agnosic, alexic, achromatopsic and prosopagnosic following two brain lesions which involved BA 18 and 19 bilaterally and extended to temporal area 37 on the left side. She was severely impaired in identifying visually presented objects, unless they were very simple forms, like polygons. Despite this, she produced plausible drawings from memory, but was unable to identify her drawings on subsequent testing. She performed perfectly on the object imagery tests. She claimed to have vivid mental images, and was astonished by our insistence at asking her questions that had nothing to do with her impairments. Her responses were always prompt and confident. A similar dissociation was present for orthographic material, and in the colour and the face domains (see below). Thus, this patient had intact mental imagery for the very same visual entities that she could not perceive. This clear-cut dissociation held across all the major domains of high-level vision: object recognition, reading, colour and face processing. As we have seen, according to the Kosslyn model, visual mental images would be produced by top-down activation of early visual cortices from more anterior areas. Such a mechanism could in principle explain Madame D's pattern of performance, as early visual areas were spared by the lesions. However, the retroactivation account would have predicted at least a relative impairment of mental imagery, because the anterograde flow of information was so massively disrupted, and the anterograde and reciprocal connections in the cerebral white matter are contiguous. Contrary to this prediction, Madame D performed the imagery tasks not only at ceiling, but also in such a rapid and easy way as to suggest that her imagery resources were entirely spared by the lesions.

Object Colour

A domain particularly important to study the relationships between perception and imagery is colour processing. As Farah (1988) notes, colour

imagery (tested, for example, by asking the patient to report the colour of common objects from memory[4]) has more chances of being visual in nature than, say, spatial processing, which could be nonvisual. Farah (1988) reviewed several case studies of acquired achromatopsic patients. She found a consistent association between impaired colour vision and impaired colour imagery, and concluded that the same neural representations are involved in seeing colours and imagining them.

Additional evidence of the association between perception and imagery deficits in colour processing was provided by Damasio et al. (1980, case 2), whose patient was achromatopsic and prosopagnosic. She reported to have colourless imagery, but coloured dreams. Her bilateral symmetric lesions involved the white matter of the posterior and inferior temporal lobes and inferior occipital lobes (BA 21, 36, 37, 19 and 18). BA 17 and 18 were intact. Also in other cases where a colour imagery deficit paralleled cerebral achromatopsia (Gomori and Hawryluk, 1984; Levine et al., 1985; Rizzo et al., 1993; Shelton et al., 1994), the lesions involved the temporal lobes bilaterally. The patient described by Shelton et al. (1994), for example, had apperceptive visual agnosia for objects and a deficit of colour perception. Imagery for object forms was normal, but imagery for colours was defective. Lesions were bilateral in the inferior temporal lobes (lingual and fusiform gyri), worse on the left side. PET showed reduced metabolism in the middle temporal regions bilaterally. Thus, from the anatomical point of view, when one considers these cases of association between colour imagery and colour perception deficits, the lesions extend almost invariably well into the temporal lobes. The agnosic patient HJA (Riddoch and Humphreys, 1987) was also severely achromatopsic, although he appeared to have some (non-conscious) residual colour processing on both direct (colour naming, matching, pointing, Farnsworth-Munsell 100-Hue and matching at isoluminance) and indirect (Ishiara plates, evoked potentials and the effects of colour on object identification) tasks (Humphreys et al., 1992). When questioned about the colour of objects his responses were sometimes surprising: he said that an elephant would be green and that a polar bear would be grey. He scored 27/30 naming colours of items with verbal associations (see Beauvois and Saillant, 1985) and 20/28 for items without verbal associations (Riddoch and Humphreys, 1987, Experiment 7). Thus, colour imagery was impaired, but not lost. Humphreys and Riddoch (1987) comment that this finding suggests "the intriguing possibility … that the brain mechanisms supporting colour perception are the same as those supporting colour memory – with the colour perceptual system perhaps being 'reactivated' when we remember the colour of an object" (p. 93).

However, also the association between acquired colour blindness and colour imagery deficit is not the rule. Patient EH described by Shuren and co-workers (1996), had cerebral achromatopsia, but could give the colour of an object named by the examiner, find from memory which object of a pair had more of one colour

[4]When asking questions about the typical colour of objects, one should of course avoid objects that are often verbally associated with their colour (e.g., snow/white), because verbal semantic memory might be sufficient to answer this type of questions (see Beauvois and Saillant, 1985, and Bartolomeo et al., 1997, for discussion of this issue).

than the other (e.g., "plum or eggplant, which has more red in it?"), and state from memory which pair among a triad of objects had the same colour. Unfortunately, in this report there is no hint about what the patient said of his imaginal experience. Another achromatopsic patient, Madame D (Bartolomeo et al., 1997), claimed to have vivid mental images of colours and could even perform subtle hue discriminations between imagined colours; for example, she was correct when stating from memory which item has the darker green, bay leaves or French beans.

The opposite dissociation, of normal colour perception with impaired colour imagery, had been described by De Vreese (1991, case II) in a patient with bilateral occipital lesions which in the left hemisphere extended to the temporal lobe, and by Luzzatti and Davidoff (1994) in two patients with predominantly anterior temporal lesions due to herpes simplex encephalitis. Also in these cases, the damage was more extensive on the left side. Also of interest in this context is patient KQu, described by Goldenberg (1992). After sustaining extensive left temporal damage, he showed intact colour perception, but had colour agnosia and colour anomia and an imagery deficit for colour and form of objects (but not for faces or topological relationships). Patients PCO and IOC, reported by Miceli and co-workers (2001), had a deficit of colour knowledge with normal colour perception and naming. They could not attribute the correct colour to objects, or select the correct crayon to colour them. Of note, IOC, but not PCO, had apparently normal knowledge of the form and function of objects. Although the issue of mental imagery was not explicitly addressed in this study, the pattern of performance of these two patients strongly suggests a selective deficit of visual mental imagery for colours for IOC, and an impairment of colour and form imagery for PCO. PCO had suffered from an encephalitis resulting in bilateral lesions (more extensive on the left than on the right) of the temporal pole and the inferolateral temporal regions (middle and inferior temporal gyri), prefrontal convexity and orbital gyri. Damage was less extensive in the parietal lobe and the superior-rostral insula. Both hippocampi were atrophic. IOC had lesions in the calcarine cortex, the lingual gyrus, the isthmus and the adjacent cingulated gyrus, the parahippocampal gyrus and the rostro-medial part of the fusiform gyrus, including part of the ventral portion of the temporal pole, with left hippocampal atrophy, as a consequence of a stroke in the territory of the left posterior cerebral artery. Interestingly, a control patient reported in the same study, SLA, who had more posterior lesions in the left hemisphere involving the posterior part of the lingual gyrus, the cuneus, and the calcarine cortex, had right homonymous hemianopia but no cognitive impairment. Thus, an extensive damage of the left temporal lobe, and particularly of its mesial part, seems both necessary and sufficient to produce deficits of colour imagery.

Faces

The prosopagnosic patient Bri… described by Hécaen et al. (1952) could easily evoke from memory the faces of his family members and of his doctor, but not his own face. The opposite dissociation seems to apply to M. X… (Charcot and Bernard, 1883), who had lost the ability to imagine faces, colours, forms, letters and topographical relationships, but could still visually recognise

his wife and sons. Information on the exact locus of anatomical lesion is not available for these two cases. The prosopagnosic patient HJA (Riddoch and Humphreys, 1987) performed well in a series of tasks tapping mental imagery of single faces and of facial features, but failed a test requiring comparisons between the global configurations of faces (e.g., 'Who looks more like Elisabeth Taylor: Joan Collins or Barbara Windsor?') (Young et al., 1994). Since HJA had perceptual problems in forming an integrated description (Riddoch and Humphreys, 1987), Young and co-workers concluded that the imagery deficit paralleled his perceptual impairment. Madame D, the already mentioned patient with bilateral lesions in the extrastriate visual areas (Bartolomeo et al., 1998a), was profoundly impaired in both overt and covert recognition of familiar faces, including her husband, but had intact ability to consult the configural as well as the componential aspects of faces in mental imagery.

Of the 20 'representative' cases of prosopagnosia reviewed by Shuttleworth et al. (1982), seven had had some assessment of imagery for known faces, which was normal or possible in five, and abnormal in two. One of these two patients, reported by Levine (1978), had a large bilateral lesion affecting the temporal lobes, resulting from repeated surgical removal of a midline meningioma. Although she claimed to be able to revisualise faces, she gave insufficient details on verbal description. The other patient with impaired revisualization was case 2 of Shuttleworth et al. (1982). This patient's verbal description of faces was "accurate but somewhat sparse" (p. 311); however, she claimed not to be able to revisualise faces, whose visual impression disappeared immediately after eye closure. In this patient, an aneurysm of the right posterior cerebral artery had been clipped, but the area of the operative site was subsequently damaged by a traumatic injury. Unfortunately, details on the precise locus of the brain lesion are not available. Patient LH, described by Etcoff and co-workers (1991), had an association of prosopagnosia and impaired revisualization of faces. As in other patients with imagery deficit, an extensive temporal lesion was present. MRI showed post-traumatic atrophy of frontal and temporal lobes, with loss of brain tissue in the right anterior temporal lobe, including partial damage to the amygdala and the hippocampus; the left temporal horn was also dilated. White matter in the right frontal and parieto-occipital region and to a lesser extent in the left parieto-occipital region was affected. Interestingly, unlike other prosopagnosic patients (see Tranel and Damasio, 1985), LH gave no sign of 'implicit' recognition of faces when tested with sympathetic skin responses, pupillometry or paired associated learning. This finding led Etcoff and co-workers (1991) to conclude that LH had lost his memories of faces[5].

Orthographic Material

Visually presented letters and words constitute a particular class of visual objects for literate people. Reading capacity can be disrupted by left posterior

[5]The association between face imagery impairment and lack of covert recognition of faces is not, however, obligatory. PH, another prosopagnosic patient with impaired face imagery (Young et al., 1994), did show signs of covert recognition of faces (De Haan et al., 1991).

brain damage in relative isolation from other impairments (and notably, with preserved writing), a disorder known as pure alexia. An impaired visual identification of letters has been repeatedly described in pure alexia (Arguin and Bub, 1993; Behrmann and Shallice, 1995; Perri et al., 1996; see Behrmann et al., 1998, for review). This might suggest an impairment of mental representation of letters in this disorder. Visual mental imagery for orthographic material is usually tested by asking questions about the physical appearance of letters or words. For example, one can ask to imagine a lower-case word presented auditorily, and to state whether it contains letters with an ascender (e.g., b, h), a descender (p, g), or neither (m, e) (Weber and Castleman, 1970). The same question can be asked for each letter of the alphabet. In addition, one can ask whether uppercase letters (again spoken by the examiner) contain any curved lines (Coltheart et al., 1975), or one can ask to identify letters to be constructed mentally following oral instructions (Behrmann et al., 1994). An example from the latter task is: Take the letter M. Cut it in half vertically. Drop off the right side and turn the remaining part upside down (the answer is V). Goldenberg (1993) pointed out that when asked this kind of question one may find oneself responding by either mentally reading or mentally writing the relevant letter, and suggested that the latter possibility be more common. Consistent with this proposal, an impairment of letter imagery has been described in agraphic patients with parietal lesions (Crary and Heilman, 1988; Friedman and Alexander, 1989; Levine et al., 1988)[6]. On the other hand, in the rare studies on pure alexic patients where imagery for letters was assessed, it has often been found intact. For example, KQu (Goldenberg, 1992), a pure alexic patient with impaired imagery for the form and colour of objects, performed in the normal range when asked to count the corners of upper-case letters, whose names were auditorily presented. SP (Perri et al., 1996), who made errors on visual identification of words and single letters, could accurately describe the form of letters from memory, or answer to specific questions about the structural characteristics of letters. Similarly, CK (Behrmann et al., 1994), who was unable to identify letters and words on visual presentation, could nevertheless perform at ceiling several difficult tasks requiring the processing of visual mental images of orthographic material. Madame D, a pure alexic patient whose residual reading capacities were disrupted by a second, right hemisphere lesion (Bartolomeo et al., 1998b), showed a similar pattern of dissociation between severely impaired visual identification of letters, words and digits, and intact visual mental imagery for these same visual entities (Bartolomeo et al., 1998a). A possible exception to this pattern of results is provided by the performance of patient VSB (Bartolomeo et al., 2002), an alexic patient with preserved writing who had lost the capacity of revisualising letters, both introspectively and when performing the above mentioned tasks of letter and word imagery. However, his performance improved dramatically when he was allowed to trace the contour of the relevant item with his finger. Thus, this patient may have primarily relied on a (defective) 'mental reading' strategy to imagine orthographic material; when a writing-based strategy was instead encouraged, he

[6]Note, however, that BT, an agraphic patient with parietal damage (Friedman and Alexander, 1989), had apparently normal visual imagery for letters.

could perform previously impossible tasks, consistent with his preserved writing capacities. A similar phenomenon occurred in patient JB (Sirigu and Duhamel, 2001), who had impaired visual mental imagery with preserved perception as a consequence of bilateral damage to the temporal poles. These patterns of performance are in agreement with Goldenberg's (1993) suggestion that two different codes, one visually-based and the other motor-based, may be used to solve tasks requiring visual mental imagery of letters. Interestingly, VSB's lesions, restricted to the left hemisphere, spared the occipital lobe but involved extensively the parietal and temporal lobes, similar to other cases of visual imagery deficits.

Spatial Imagery

Deficits of mental imagery for topographical relationships may or may not be associated with topographical disorientation (see Goldenberg, 1993, for review). As already noted, Farah and co-workers (Farah et al., 1988a; Levine et al., 1985) described a double dissociation between deficits of visual mental imagery, resulting from damage of the occipito-temporal stream of visual processing, and deficits of spatial imagery, determined by lesions of the occipito-parietal stream. Subsequent case studies confirmed the notion of a special status for deficits of spatial imagery, and its relationship with parietal lesions.

Patient RT (Farah and Hammond, 1988), who had a large lesion involving the frontal, temporal and parietal lobes of the right hemisphere, with left neglect and hemianopia, was impaired on three tests of mental rotation, despite performing well on a variety of tests of visual mental imagery for object shape, size and colour. He could also recognise misoriented numbers, letters and drawings. The authors concluded that orientation-invariant object recognition does not depend upon mental rotation. Patient MG (Morton and Morris, 1995), whose lesion impinged on the left parietal lobe and extended posteriorly into the occipital lobe and anteriorly to the posterior mesial region of the temporal lobe, had no perceptual impairment on an extensive series of tasks, but performed poorly on tasks of mental rotation and mental transformation. She performed well on tasks tapping visual and spatial short-term memory, as well as on tasks exploring visual mental imagery for colour, form and faces. The authors suggested that these last abilities were unimpaired because the lesion did not extensively involve the temporal lobe. Spatial imagery was also recently assessed in patient HJA, who, among his other deficits, shows topographical disorientation (Riddoch et al., unpublished data). HJA performed relatively well on a number of tasks (e.g., judgement of the angle between the imagined hands of a clock, maintaining visual patterns over relatively long inter-stimulus intervals, the Moscovitch letter manipulation task); however, he was strikingly impaired on other tasks (such as recalling the spatial layout of his room, judging the spatial directions between two cities, the Brookes matrix task, reproducing both possible and impossible figures). This pattern of performance led the authors to argue that his imagery deficits mirror his perceptual deficits; that is, performance is poor when he has to respond to the spatial relations between the local parts of objects.

Imaginal Neglect

Finally, I address the issue of unilateral neglect for visual mental images, a phenomenon which seems to stand rather apart from the deficits reviewed so far. The observation that unilateral neglect may not only occur during activities requiring the processing of sensory input, but also during tasks less directly involved with perception, such as the description from memory of places, is another line of evidence taken to support the Kosslyn model of perceptual-imagery equivalence. In their seminal paper, Bisiach and Luzzatti (1978) reported two left neglect patients who, when asked to imagine and describe from memory familiar surroundings (the Piazza del Duomo in Milan), omitted to mention left-sided details regardless of the imaginary vantage point that they assumed, thus showing representational, or imaginal, neglect. Bisiach et al. (1981) replicated this finding in a group study with 28 neglect patients, of which 13 had to be excluded from analysis because they misplaced the imagined details (e.g., they said that a left-sided detail was on the right side); the remaining 15 patients showed a bias toward mentioning more right-sided than left-sided details of the Piazza del Duomo. Bisiach et al. (1979) asked 19 neglect patients to perform same/different judgements over pairs of cloud-like shapes that moved horizontally and could only be seen while passing behind a narrow slit, so that their form had to be mentally reconstructed to perform the task. Performance was particularly impaired when the shapes differed on the left side. Rode et al. (1995) reported eight neglect patients who gave poor descriptions of the left part of an imagined map of France. The number of items reported on the left side increased after vestibular stimulation of the left ear with cold water (Rode and Perenin, 1994). Grossi et al. (1993) asked 24 neglect patients to judge the angle between imagined or seen clock hands. The ten patients who were able to complete the experiment made more errors when one of the hands was in the left hemispace, both in the imagery and in the perceptual condition.

In these group studies, imaginal neglect co-occurred with visual neglect, thus supporting the Kosslyn model. However, also in this domain later single-case reports demonstrated both the possible dissociations between visual and imaginal neglect; visual neglect in the absence of imaginal neglect (Anderson, 1993; Coslett, 1997), and imaginal neglect without visual neglect (Beschin et al., 1997; Coslett, 1997; Guariglia et al., 1993). Also, a patient has been recently described who, after a left parieto-occipital haemorrhage and a right thalamic stroke, showed *right* visual neglect together with *left* imaginal neglect (Beschin et al., 2000).

To study the relationships between these two forms of neglect, in the group of Guido Gainotti, in Rome, we asked subjects to describe from memory three Roman piazzas, a map of Europe centred on Italy, and the Italian coast as it could be seen from the Sardinian coast (Bartolomeo et al., 1994). We studied 30 patients with right brain damage, 30 with left brain damage and 30 normal individuals. Seventeen patients with right brain damage and two left-brain damaged patients had contralesional visual neglect. Imaginal neglect was present only in five right brain-damaged patients, all showing signs of visual neglect. Thus, although imaginal neglect was always associated with visual neglect, the

most frequent finding in right brain-damaged patients was that of a visual neglect in isolation. We concluded in favour of an important role of right visual objects, as opposed to imagined scenes, in triggering neglect behaviour, presumably because these objects attract patients' attention, which is biased rightward (D'Erme et al., 1992; De Renzi et al., 1989; Gainotti et al., 1991; Mark et al., 1988). We found no instance of imaginal neglect in isolation. However, we found such a dissociation when we followed up one of our patients with imaginal and visual neglect. This patient recovered from visual neglect, while imaginal neglect persisted longer. Later, we identified another patient who presented a similar pattern of selective recovery for visual, but not for imaginal, neglect (D'Erme et al., 1994). This patient had so little visual neglect at onset, that it would probably have passed undetected without formal testing. It is possible that these patients learned first to compensate for their neglect by exploring their visual space, which is more ecologically relevant and subject to feedback than mental images. Had these patients been tested only after recovery from visual neglect, they would have shown imaginal neglect in isolation. The performance of another neglect patient, tested by Sylvie Chokron, exemplifies the power of visual details, as opposed to mental images, to trigger neglect behaviour. When asked to draw a butterfly from memory, he completely omitted the wing on the left side of the figure. Soon afterwards, the patient was blindfolded and asked to draw a butterfly once again. The result was a less detailed, but two-winged butterfly (see Figure 9 in Bartolomeo and Chokron, 2001). Our interpretation of this finding is that, in the first case, patient's attention was attracted by the right-sided details that he had just drawn (see D'Erme et al., 1992; De Renzi et al., 1989; Gainotti et al., 1991; Mark et al., 1988), thus provoking the failure to draw the left-side parts. In the second case, this attentional attraction was not possible because the patient had no visual input. This evidence is of course anecdotal, but it confirms that mental representations can be intact in unilateral neglect, and cautions against the direct use of drawing from memory to infer the status of these representations.

In conclusion, imaginal neglect can double-dissociate from visual neglect, even if visual neglect in the absence of imaginal neglect seems to be a more frequent and perhaps more empirically robust pattern than the opposite dissociation (which could result from a difference in recovery rate rather than from a true difference in the underlying mechanisms). In all the cases reported in the literature, patients show imaginal neglect for the left side of imagined scenes, as a result of a right hemisphere lesion. This pattern seems thus at sharp variance with other mental imagery deficits, which preferentially occur after left hemisphere lesions (with the possible exception of mental imagery for faces, see above).

CONCLUSIONS

Despite the great variety of the methods used to assess perceptual and imagery abilities, recently published case studies have repeatedly confirmed that every type of dissociation is possible between these functions. The studies in

which an accurate assessment of perception and imagery in different domains was carried out, moreover, suggest that dissociations can occur even between different imagery domains.

From the functional point of view, the present review of neuropsychological evidence concerning visual mental imagery abilities concurs with previous reviews (Trojano and Grossi, 1994; Goldenberg, 1998) in showing that the Kosslyn model of perceptual-imagery relationship, in its present form, fails to predict the observed patterns of performance in brain-damaged patients. In particular, it seems that Goldenberg's (1998) challenge to the notion that a single visual buffer is used for object recognition and object form imagery (see also Riddoch, 1990) may now be extended to imagery for colours, letters, and faces. From the anatomical point of view, despite the fact that precise information about the locus of lesion is not always available, the neuropsychological evidence indicates that occipital damage can determine perceptual deficits, but it seems neither necessary nor sufficient to produce imagery deficits[7]. These results challenge the anatomical underpinnings of the Kosslyn model, which contends that activity in occipital visual areas would be essential to visual mental imagery. On the other hand, the available neuropsychological evidence strongly suggests that a rather extensive damage of the left temporal lobe (and perhaps especially of its mesial part) is necessary in order to produce visual imagery deficits for object form or colour. For face imagery, the available anatomical evidence is really scanty, but it seems to suggest that extensive bilateral temporal damage (perhaps including preferentially the right temporal lobe, as in the case of LH described by Etcoff et al., 1991) is necessary to observe an impairment of this ability.

The present conclusions need to be confirmed by further case and group studies carefully exploring each of the domains of visual mental imagery and the lesional correlates of the observed deficits. They are, however, in broad agreement with the idea that structures including the infero-temporal cortex, the mesial temporo-limbic regions and perhaps the temporal pole might be critical for processing of knowledge based upon visual information (Miceli et al., 2001; Gainotti, 2000)[8]. Selective deficits for visually-based knowledge of object form, object colour, and faces might thus result from damage to domain-specific regions (Kanwisher, 2000) or circuits (Ishai et al., 1999) in the ventral temporal lobe. Thus, the evidence coming from lesion neuropsychology strongly suggests that cortical areas which are related to vision, but at a higher level of integration than previously proposed, might be crucial for visual mental imagery abilities.

[7]In agreement with lesion neuropsychology, also PET and fMRI studies (recently reviewed by Mellet et al., 1998) often failed to demonstrate an increase of occipital activity during visual mental imagery.

[8]Although the implications for visual mental imagery are by no means clear, it is perhaps worth recalling here that Penfield and Perot (1963), in their review of 1,288 cases of focal electrical stimulation of the human cerebral cortex, found that highly organized visual or auditory events, such as seeing people in the room or hearing a song, were exclusively evoked by stimulations applied to the cortex of the temporal lobe. Similar evidence was recently obtained Lee et al. (2000), who also stimulated epileptic patients with subdural electrodes. Only when the temporal lobe was stimulated did patients experience complex visual forms, such as animals, people, landscapes and scenes from one's memory. More posterior stimulation of the occipital lobe evoked either simpler forms, such as spots or blobs, or simple geometric shapes such as triangles or diamonds. Kreiman et al. (2000) directly recorded the activity of neurons in the human medial temporal lobe, and found selective changes in firing rate of neurons located in the hippocampus, amygdala, entorhinal cortex and parahippocampal gyrus when subjects viewed figures and when they were visually recalling the corresponding images with closed eyes.

More specifically, the cortical areas that can be identified as being necessary for visual imagery abilities on the basis of the available neuropsychological evidence are situated at a distance of two or more synaptic levels from V1. In Mesulam's terminology (Mesulam, 1998), they would be classed as 'downstream' visual association areas[9] and transmodal association cortices. This anatomical evidence suggests that imagery abilities maintain only high-level 'visual' characteristics (which would explain the parallelism found in some cases between imagery impairments and high-level perceptual deficits). This view seems consistent with Goldenberg's proposal (Goldenberg, 1998) that knowledge about the visual appearance of objects is represented at two distinct levels, within perceptual mechanisms and in semantic memory, and that only semantic representations can subserve imagery.

But where does the 'quasi-visual' character of visual mental images come from? This question is related to the broader (and largely unresolved) issue of the origin of our phenomenological experiences. Recently, O'Regan and Noë (in press) have proposed that perception does not result from the construction of an internal representation of the perceived object, but from the peculiar actions associated with perception in that particular sensory modality. Seeing would be a particular way of exploring the environment, characterised by peculiar sensorimotor contingencies, which are different from, say, those associated with hearing. Thus, when we look at an object, the visual quality of its shape is the set of all potential distortions that this shape undergoes when it moves relative to us or when we move relative to it. Knowledge of these sensorimotor contingencies constitutes visual consciousness. No percept is created in the brain, but perception is an ongoing activity of exploration of the environment (see also Gibson, 1966, 1979; Ballard, 1991; Ballard et al., 1997). In this 'active perception' framework, mental imagery could be conceived as the implementation of perceptual exploratory procedures in the absence of an external object (Thomas, 1999). In support of this view, it has been shown that eye movements during visual mental imagery reflect the content of the visualized scene (Brandt and Stark, 1997). If the application of these procedures can be constrained either by the external environment or by memory processes, with distinct neural correlates subserving these occurrences, then double dissociations between perceptual and imagery abilities are expected to arise in brain-damaged patients.

As Sartre (1940) pointed out, the imagining consciousness achieves full liberty, because it can reach beyond reality. The much greater level of abstraction and flexibility of mental images with respect to percepts lends support to the suggestion that mental imagery is one of the abilities that "help to loosen the rigid stimulus-response bonds that dominate the behaviour of lower animal species" (Mesulam, 1998, p. 1014).

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[9]As opposed to the 'upstream' visual association areas (V2 to V5), monosynaptically connected with V1.

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