

Neuropsychology

Although there is a very large literature on the neuropsychology of memory, and a well established literature on verbal short-term memory deficits, the existing neuropsychological literature is relatively light in its discussion of impairments of visuo-spatial working memory. There is certainly an established body of work on visuo-spatial deficits, some of which has explored deficits of imagery (e.g. Farah, 1984). Given my earlier discussion of the link between visual imagery and visuo-spatial working memory, a discussion of patients with imagery deficits as well as with other forms of visuo-spatial deficit should be informative. In this chapter I shall first discuss neuropsychological evidence for the dissociation between verbal and visuo-spatial working memory. This will be followed by a discussion of patients with varying forms of visuo-spatial deficits, and in particular visual imagery deficits. The chapter will conclude with a discussion of the neuropsychological case for a distinction between visual and spatial short-term memory as mooted in Chapter 4.

DISSOCIATIONS AND DEFICITS

There are a number of documented patients with reported visuo-spatial short-term memory deficits in the absence of equivalent deficits for verbal material. For example, De Renzi and Nichelli (1975) described two cases of patients with damage to the right hemisphere who had scores of 2.5 on the Corsi block span task. This is a performance level in the pathological

comb, a pencil, or some chalk. However he was not retaining information about the visual appearance of the pen because a few moments later he was unable to select the presented pen from among several other pens serving as distractors. He was also unable to recall the layout of objects in a room after a brief presentation, and after a delay of one minute during which his eyes were closed. Verbal short-term memory and long-term visual memory were unimpaired in both patients. For example, one of the patients could accurately recall and draw the layout of his parents' house.

Beyn and Knyazeva (1962) reported a single case of a patient with bilateral damage to the occipital lobe who was able to copy drawings of objects but was unable to continue copying after the original drawings had been removed. He also had considerable difficulty drawing objects from long-term memory.

There are in addition a number of patients who show the converse pattern. For example, patient K.F., mentioned in Chapter 1 (Shallice & Warrington, 1970; Warrington & Shallice, 1972) could recall a sequence of just two digits which he had heard. In contrast K.F. could recall a sequence of four visually presented digits. When he made errors with visually presented letters, these errors tended to be based on visual confusions rather than acoustic confusions. As I mentioned earlier, most normal subjects tend to produce acoustic confusions with visually presented, verbal material. This suggests that normal subjects usually translate the visually presented words, letters, or digits into a phonologically based code for retention in a phonologically based store. It appeared, then, that K.F. attempted to retain information using relatively intact visual short-term storage functions while his phonologically based, verbal short-term storage system was severely impaired. In contrast, normal subjects typically can retain more auditorily presented items than visually presented items (e.g. Conrad, 1964). Similar patterns have been reported for other patients with verbal short-term memory deficits (e.g. Basso et al., 1982; Saffran & Marin, 1975). In particular, patient P.V., to whom I referred earlier in the book, has been extensively studied as a relatively pure case of someone with a deficit in the phonological loop component of working memory (e.g. Basso et al., 1982; Vallar & Baddeley, 1984). Basso et al. reported P.V.'s performance on visual and auditory versions of the Brown-Peterson task where recall of verbal material is required after a filled delay. With one consonant for recall, after a three-second filled delay, P.V. achieved perfect recall performance with visual presentation, but recalled correctly on only half of the trials for auditory presentation.

One approach to interpreting patterns of sparing and impairment in neuropsychological patients is to suggest that impairments might be shown by tasks that are more difficult. That is, a damaged brain may have more difficulty with cognitively demanding tasks. It is clear from the

range. These same patients achieved scores of around 7 on digit span. They also had normal performance on tests of long-term visuo-spatial memory. In that same study De Renzi and Nichelli reported that among a group of 32 patients with right hemisphere damage (including the two patients just described), performance on Corsi span was notably poor in relation to their performance on digit span tasks. A contrasting group of 39 patients with left hemisphere damage showed the opposite pattern. De Renzi, Faglioni, and Previdi (1977) replicated the observed link between right hemisphere damage and Corsi span impairment in a further group of patients with right hemisphere damage.

More recently, Hanley, Young, and Pearson (1991) reported a single case, E.L.D., with right hemisphere damage who showed very poor performance on Corsi blocks, on the Brooks (1967) matrix task, on mental rotation, and in using visual imagery mnemonics. This same patient was unimpaired in her ability to recall letter sequences with auditory and with visual presentation. She also showed evidence of word length and phonological similarity effects.

In studies of visual rather than visuo-spatial short-term memory deficits, there is evidence for an involvement of the left hemisphere rather than the right. In a group study, Warrington and Rabin (1971) reported visual short-term memory deficits that were most evident in those of their patients who had damage to the posterior region of the left hemisphere. Performance was measured on a "visual span of apprehension" test. For this task, the patients were shown a sequence of letters, digits, or lines and curves for brief periods ranging from 50msec to 160msec, and were required to report as many of the presented items as possible. The left posterior damaged patients were impaired in their ability to report random strings of digits or letters, or of lines and curves. These patients were relatively unimpaired in reporting letter strings that were approximations to words. They also appeared to have auditory digit spans in the normal range. Patients with right hemisphere lesions showed no deficits in span of apprehension when compared with non brain-damaged control subjects.

In a study of two single cases, Ross (1980) reported what he termed loss of "recent visual memory" in patients with damage to the right occipital lobe. One of the patients also had damage in the left occipital lobe. These patients could copy drawings and could reproduce patterns if they were asked to draw them immediately after they had been removed. However they were unable to remember anything of visually presented patterns if there was a brief delay between removal of the pattern and the request to draw, or to recognise the pattern. At least one of the patients made extensive use of verbal codes to supplement his poor temporary visual memory. For example, if shown a pen for a few seconds, after a three-minute delay, he could correctly identify a pen from distractor objects such as a

patients discussed here that this cannot be the case, in view of the double dissociations observed between patients.

Impairments of both spatial and visual temporary memory have also been demonstrated in patients with dementia of the Alzheimer type and in patients with Parkinson's disease. Baddeley, Delia Sala, and Spinnler (1991) observed that one of their Alzheimer patients showed a pathological score on tests of visual short-term memory, but had verbal short-term memory performance within the normal range. One of their other patients showed exactly the converse pattern, demonstrating a double dissociation. In a study of 18 Alzheimer's disease patients, Perani and colleagues (1993) observed that five of their patients were below the pathological cut-off score on Corsi block span, while two of the patients were impaired on digit span. However in most studies of Alzheimer's patients, verbal short-term memory tends to be impaired more frequently than is visuo-spatial short-term memory (Cantone, Orsini, Grossi, & De Michele, 1978; Morris & Baddeley, 1988). In some studies, both visuo-spatial and verbal short-term and long-term memory deficits have been found in Alzheimer's patients (Spinnler, Delia Sala, Bandera, & Baddeley, 1988; Sullivan & Sagar, 1991). In the Sullivan and Sagar (1991) report, they noted that a group of Parkinson's patients were impaired on visuo-spatial short-term memory while long-term visuo-spatial memory was intact. In their group of Alzheimer's patients, both long- and short-term visuo-spatial memory were impaired.

Further evidence for a distinction between visuo-spatial and verbal short-term memory comes from studies of brain activity in normal subjects either by means of scanning techniques such as positron emission tomography (PET) which measures brain metabolism, or monitoring brain electrical activity such as via event-related brain potentials (ERP). Ruchkin *et al.* (1992) recorded ERPs from a group of young healthy subjects while they were asked to retain visuo-spatial material in the form of random two-dimensional displays, or to retain phonologically based material in the form of pronounceable non-words. The ERP patterns were quite different for the two different kinds of material. Indeed, Ruchkin *et al.* reported that these two distinct ERP patterns also differed from a third observed pattern which appeared to be associated with central executive functioning. Their results are supported by previous work showing, in separate studies, differential ERPs for phonological memory tasks (Rugg, 1984) and for tasks involving memory for visual features of faces (Barrett, Rugg, & Perrett, 1988).

Perani *et al.* (1993) carried out PET scans on 18 patients with Alzheimer's disease, and reported that scores on spatial short-term memory were best predicted by the scan-derived metabolic values from the right parietal and frontal associative areas. Verbal short-term memory was

more closely associated with the temporal, parietal, and frontal areas of the left hemisphere.

IMAGERY AND IMAGERY DEFICITS

Some hints about the role of various brain structures in imagery tasks have arisen from a wide range of studies of patients with imagery deficits, and from a few studies of brain activity during imagery tasks performed by normal adult volunteers. Examples of the latter were carried out by Goldenberg and colleagues (e.g. Goldenberg et al., 1991). They have used SPECT (single photon emission computer tomography) to study the differential patterns of blood flow in the brain during visual imagery tasks and during acoustic imagery tasks. The blood flow patterns were quite distinct in the two tasks. In particular the acoustic imagery tasks were associated with increased blood flow in both hippocampal regions and in the right temporal lobe, whereas both imagery tasks resulted in an increase in blood flow in the left inferior occipital region and the left thalamus. However on questioning the subjects Goldenberg et al. found that subjects reported using visual imagery for both the visual and the acoustic imagery tasks. They concluded that visual imagery was most likely associated with activity in the left hemisphere, in the occipital lobe, and in the thalamus.

Turning to imagery deficits, Farah (1984) has reported some very convincing evidence drawn from a re-examination of previously published single cases, each of whom appeared to have deficits of visual imagery. In her review she notes that different aspects of imagery ability are impaired in different patients. The patterns of impairment map well onto Kosslyn's model of imagery referred to in Chapter 2. For example, eight of the cases she described appeared to have an image generation deficit. That is they could perceive objects, describe and recognise objects, and draw objects that were present. Despite this they could not describe objects from long-term memory, or draw objects from long-term memory, and denied having visual imagery. All of these subjects appeared to have lesions in the left parieto-occipital area of the brain, and they seemed to have an image generation deficit in the absence of any evidence of visual agnosia. That is, they must have had access to information in their long-term memories about the link between the appearance of objects and the object name, in order for them to be able to identify those objects correctly. However they seemed unable to conjure up mental images of the objects. This points to a dissociation between the system responsible for retrieving information from long-term memory and for creating an image, and the processes involved in object recognition.

A further 13 patients in Farah's review could describe objects, draw

difficulty drawing a set of letter forms immediately after they had been removed from view. Performance was very similar when he was required to wait for a period of 10 seconds before drawing the stimuli. In other words, the patient appeared to have a difficulty generating images from long-term memory, but had access to long-term memory that allowed for normal object recognition. More important, he could retain visually presented information for at least 10 seconds after the stimulus had been removed. Riddoch interprets her data as suggesting that the system for generating and manipulating visual images might well be distinct from that involved in short-term visual storage.

Further support for this view comes from an informal study reported by Frick (1987) who described two subjects with an apparent deficit in generating conscious visual images. The same individuals performed normally on tests of visual short-term memory. Frick's subjects were selected largely on self report and with no reported evidence of brain damage, and the data were somewhat informal. However together with data like those from Riddoch, there is a nagging doubt as to whether conscious visual imagery and visual short-term memory are as closely related as has been assumed. Within the rubric of working memory, one speculative suggestion is that conscious visual imagery may be more closely linked to central executive function than to a visuo-spatial temporary storage function. In this light of this, it would be interesting to test the central executive functioning of subjects with imagery deficits. This would allow a systematic exploration as to whether there is a segment of the architecture of working memory devoted to visual imagery, or whether imagery relies on a general purpose, flexible system. I shall explore this suggestion in more detail in Chapter 6 when discussing related data from normal adult subjects.

VISUAL NEGLECT

Among the disorders of visuo-spatial cognition, one of the most intriguing involves apparent loss of information from one half of visual space, normally on the left, and normally associated with right hemisphere damage. This disorder, which occurs in the absence of peripheral visual acuity problems, has generated a great deal of debate in that it touches on the nature of the visual representation of the world and of conscious experience (e.g. Bisiach, 1993). It has been interpreted both as a deficit of attention and as a deficit of representation. Although the disorder was first reported in 1876 by Hughlings Jackson, it was only in the mid 1970s that patients with this disorder appeared to any great extent in the research literature.

presented. However they could not recognise or identify objects, nor could they describe or draw objects when presented with the object name. Farah argued that this demonstrated an impairment in access to long-term visual representations of objects to allow identification, but left the patients with a means to process the physical features of presented objects in order to draw them. Presumably the process of drawing would require some form of temporary visual storage, in order to store the appearance of the part of the object while they looked at and produced the drawing (van Sommers, 1989). Even if the subjects frequently looked back at the object, there is still a requirement for temporary visual storage.

Farah's report is extremely useful in pointing out how previously published reports of neuropsychological patients can be used to inform contemporary theories of cognition. The disadvantage of course is that it is not possible to go back and re-test these patients. Nor can a great deal be gleaned about the neuroanatomical correlates of the impairments, because although the pattern of functional deficit was relatively homogeneous across these patients, there was no clear common anatomical locus for their lesions. Moreover, the initial investigations of these patients were not motivated by the model of visual imagery favoured by Farah, and by Kosslyn and colleagues, and the data are necessarily being interpreted *post hoc* (Sergent, 1989). This has the implication that a number of key questions cannot be asked. For example an inability to draw an object in response to the name coupled with a failure to identify an object could indicate a severing of the links between the name of the object and its visual form. That is, the patients might still have access to the long-term memory representation of the visual form of the object when presented with the object, but be unable to access that form via the name. This leads to the question as to whether the patients knew how to use the presented object as well as to draw it. That is, could they access object semantics, even if they were unable to name the object? Another important issue is whether the patients from either group could draw *a* picture of an object that had been presented for a short time and which was then removed from view. In other words, could these patients draw objects from visual short-term memory, without the opportunity to refresh the contents of that memory system by repeatedly looking back at the object?

This last question was addressed at least in part in a single case study reported by Riddoch (1990). Case D.W. suffered damage to the left temporo-parietal region as a result of a cerebro-vascular bleed. The patient was unable to draw objects from long-term memory, or to perform mental rotation when tested with letter forms or with the Manikin test (Benson & Gedye, 1963; Farmer et al., 1986; Ratcliff, 1979). However he showed unimpaired performance in object identification, and he could copy a picture that was physically present. In addition, the patient had little

One of the classic reports of this disorder was by Bisiach and Luzzatti (1978) who asked two Milanese neglect patients to imagine themselves standing in the Cathedral Square in Milan (Piazza del Duomo). When they imagined themselves facing the cathedral from the far end of the square, they accurately described the buildings on their "imagined" right. However they failed to mention buildings on the left side of the square. When asked to imagine facing in the opposite direction, standing on the cathedral steps with the cathedral building behind them, they described buildings on the other side of the square that they had previously failed to mention, but omitted descriptions of buildings that would have been to their left, and which they had successfully reported a few moments before. One of the patients was also asked to describe his studio, imagining himself sitting at his desk, or facing his desk. The studio was a room in which the patient had spent a great deal of time, and with which he was highly familiar. As before, he tended to describe items on the right of the studio, but omitted items that were on the left. Both for the Piazza del Duomo and for the studio, the patients omitted even very large and salient items on their imagined left such as the Royal Palace in the Square, and an upright piano in the studio.

An interesting aspect of the observations is that despite their disorder, both patients did report a small number of features on the neglected side after some delay or with some prompting from the experimenter. This might suggest that they have some residual information available on the neglected side, and that their disorder represents a bias rather than complete loss of access to the information. However it is also worth noting that the patients were very familiar with the *Piazza del Duomo*, and the second patient had used his studio for many years. The patients were not amnesic and there is no reason why they could not use verbal codes to help them recall either (a) their reports from the alternative imaginary perspective on the previous test session the day before, or (b) from verbally encoded information about the target environment. For example Bisiach and Luzzatti's second patient initially failed to mention a piano which was on his neglected side when sitting at the desk. He did however report the piano after some hesitation and after he had mentioned most of the objects on the other side of the room. Bisiach and Luzzatti noted that this patient had spent a great deal of time playing the piano in his lifetime, and that verbal knowledge of a highly familiar environment would be sufficient to allow a report of information from the neglected side. For example from my verbal knowledge of my own office, I can report that I have a desk, two bookcases, a round table, two filing cabinets, and a computer, without having to construct a visual image of the contents of my office or to consult a mental representation of their layout. It is also clear from other studies of neglect patients that when they are asked to draw a clock, some patients

although their drawing depicts only the right side of the clock, it is not uncommon (although this does not occur in every case) for numbers from the left side of the clock to appear squeezed into the right side shown in the drawing. One other possibility in the case of the Bisiach and Luzzatti patients is of course that the subjects attempted to alter their imagined orientation spontaneously, without instructions from the experimenter. That is, the delay in responding could have been sufficient for the subjects to imagine themselves walking back to the other end of the Cathedral square, and turning around!

Whether there is no information or just partial information available on the neglected side, it is clear that these patients have a severe asymmetric impairment, and since the seminal studies in the 1970s there is now a large literature on the topic with a range of associated and relatively well established findings. For example, when asked to draw everyday objects either from memory or when physically present, neglect patients tend to produce drawings of just one side of the object. When asked to cross out all the occurrences of a single letter on a sheet of randomly arranged letters, they tend to cross out only those letters on the right side. Similarly when asked to indicate the mid point of a line, their indication tends to be grossly skewed to the right. Many neglect patients also tend to ignore the left side of their body, and some deny that their left arm is actually part of their body. There are also cases of patients with neglect of the right side, but such patients are relatively rare. A recent excellent review of this area is given in Bisiach (1993).

The nature of the deficit could be thought of in at least two ways. One view is that the visual scene is adequately encoded, but that patients tend to ignore half of the encoded representation, with a substantial attentional bias towards the right (e.g. Kinsbourne, 1977, 1993; Riddoch & Humphreys, 1983). With a slightly different emphasis Posner, Cohen, and Rafal (1982) suggest that there is an attentional bias away from the left rather than specifically towards the right. In contrast, Bisiach among others has suggested that the deficit lies in the representation (see also Caramazza & Hillis, 1990), with information essentially omitted from the neglected side. He has pointed out that the attentional hypothesis runs the risk of requiring an homunculus which examines the visual representation displayed on some form of internal screen. This leads to questions about the nature of the homunculus and the possibility of an infinite regress. In support of the representational view, Bisiach, Luzzatti, and Perani (1979) asked their patients to look at a series of abstract cloud-like patterns, and to decide whether pairs of patterns differed or not. However the patterns could only be seen one part at a time while they were moved behind a narrow slit. Thus judgements could be made only if the subjects were able

the damaged neural functioning is preventing that information from reaching a conscious mental representation.

There is a continuing debate on this topic in the literature on cognitive neuropsychology. The attentional/representational issue remains as a focus of this debate. An additional issue concerns the representation of the relationship between egocentric and exocentric space. That is, does the cognitive system that is damaged in neglect patients hold information about the relationship between body parts and objects in the visual scene? Alternatively, does the disorder primarily affect the representation of the mutual relationships among objects in the world irrespective of the position of the body parts of the viewer? Bisiach has assumed the former (e.g. Bisiach, 1993), and moreover has stated explicitly that these spatial relationships are held in working memory. He does not give details as to the nature of the working memory system that he envisages might host this representation, but does refer explicitly to a visuo-spatial scratch pad referred to in the Baddeley and Lieberman (1980) paper. You may recall from Chapter 4 that Baddeley and Lieberman viewed the scratch pad as being primarily spatial rather than visual in nature. Bisiach suggests that visual neglect might be characterised as damage to that part of the visuo-spatial scratch pad that represents the left half of the spatial array. Presumably this would apply to a representation of the currently presented scene as well as for visuo-spatial representations generated from long-term memory.

In a more recent paper Halligan and Marshall (1992) argued strongly that it is highly misleading to think of visual neglect as a single form of disorder. They report for example that there are double dissociations among neglect patients, some of whom show an impairment in letter cancellation tasks but are unimpaired on line bisection tasks, whereas other patients show the converse. They conclude that there are likely to be a range of neglect disorders, each of which requires a cognitive explanation, and each of which would have its own set of neuropathological correlates. This raises complications for clinical approaches to hemi-neglect, for example in diagnosis and in development of possible neuropsychological rehabilitation. It also causes difficulties when developing general theories or explanations of hemi-neglect.

One way to tackle the problem of having different types of neglect patient would be to develop theories for each of the different forms of neglect. But this can be a perilous approach, potentially leading to the development of a different variant of the theory for each variant of the neuropsychological disorder. This gives the theories less generality and makes them less useful as a consequence. An alternative is to use the data derived from the neuropsychological patients to help develop general models of normal cognition. Thus we can attempt to develop a model of normal visuo-spatial cognition derived in part from studies of hemi-neglect

a small amount of changing information in central vision. The neglect patients failed to discriminate the items that differed only on the left side, despite the fact that all of the relevant information had been presented centrally. This experiment supports the notion that the phenomenon of neglect is based on the nature of the mental representation, although it does not rule out the possibility that the impairment is in attention to parts of the mental representation or due to an incomplete representation.

The debate is taken forward by Halligan and Marshall (1991) who demonstrated that neglect patients have available information that appears to affect their responses even if they fail to report information present in the left visual field. For example, Halligan and Marshall showed neglect patients pictures of houses, and in one of the pictures the left half of the house was depicted in flames. The patients denied noticing anything unusual about the houses, and reported that the two pictures looked the same, but when asked about which house they would prefer to live in they rejected the picture showing half of the house on fire. This points strongly to an attentional interpretation, in that the information appears to be represented in some form, but is largely ignored by the patient.

Further evidence for this kind of implicit processing of information in the neglected field comes from a study by McGlinchey-Berroth and colleagues (1993). Their patients were given a lexical decision task, with the words or non-words presented centrally. Prior to the presentation of each letter string, pictures appeared in either the left or the right visual field. Lexical decision times for real words were faster when the picture matched the subsequently presented word, and this occurred even when the picture primes were presented in the neglected hemifield.

What is notable here is that the data on priming effects and implicit processing cause difficulty for the representational view of neglect only if it is assumed that these effects arise from information contained in a coherent visual representation. If instead we assume that priming involves the temporary activation of long-term memory representations in the lexicon or semantic memory system, the data are still consistent with an "incomplete representation" view of neglect. Thus the impairment could lie in the mechanism that constructs the conscious visual representation from activated long-term memory traces. For example, we know already that neglect patients fail to construct a representation of the neglected side of objects whose features are drawn from long-term memory. A similar argument can be made about the Halligan and Marshall experiments on implicit processing. Presentation of a picture of a house on fire could activate long-term semantic memory networks comprising links between houses and fires and unpleasant consequences, without this information being incorporated in a conscious mental representation of the complete picture. Thus the stimulus information is reaching semantic memory but

patients, and in part from studies of normal subjects and from other groups of neuropsychological patients. We should then be able to use such a model to provide putative explanations for at least some of the various forms of hemi-neglect.

Assuming this is a primary objective, then what can these data tell us about the nature of normal visuo-spatial working memory? One conclusion that is fairly well supported by these data is that there is indeed a distinction between the visuo-spatial information that subjects potentially have available, and the information they are able to report explicitly. Another way of looking at this would be to say that there is a distinction between conscious experience of visuo-spatial information, possibly in the form of visuo-spatial imagery, and some semi-independent visuo-spatial temporary memory. Moreover, I have already suggested that the data on priming effects in neglect patients indicates that visuo-spatial information presented to the neglected field has access to semantic information in long-term memory. This information does not appear to be part of the representation in working memory. One tentative interpretation would be to suggest that priming results in temporary activation of long-term memory representations and this activation is sufficient to make information detectable only in tests of implicit processing. In normal subjects, the activation makes the information available for construction of a mental representation in working memory. However some forms of brain damage prevent this information from being included in that mental representation. Such a model has a number of additional implications. In particular it suggests that the contents of working memory might result from activation of long-term memory traces, and this is in contrast to the view of working memory as some form of gateway between stimulus input and long-term memory. Far from being a gateway, the contents of working memory are available only *after* that information has been processed in long-term memory. This view is consistent with the argument outlined towards the end of Chapter 1, and referred to elsewhere in the book. I shall explore the model further in Chapter 6 (see Fig. 6.6, p.127) when giving more detailed treatment to theory development in the area of visuo-spatial short-term memory. In the meantime, I would like to return to another major theme of this book, namely the hypothesised distinction between visual and spatial working memory, with respect to relevant evidence from studies of neuropsychological patients.

VISUAL OR SPATIAL DEFICITS?

The possibility of a spatial versus visual distinction discussed in Chapter 4 echoes a similar distinction current in the neuropsychological literature on the nature of the cognitive mechanisms involved in *visual imagery*

I touched on neuropsychological evidence for the distinction earlier in this chapter. Dissociations among cognitive functions are convincing when double dissociations can be demonstrated (Shallice, 1988). Just such a double dissociation appears in the reports of two separate case studies, one reported by Farah and colleagues, and the other by Hanley et al. (1991).

Farah et al. (1988) described a neuropsychological patient who appeared to have a deficit in the performance of visual imagery tasks, but had spared function for spatial imagery tasks. Patient L.H. had damage to the right temporal lobe and the right inferior frontal lobe, in addition to damage to the temporo-occipital regions on both sides. The visual imagery tasks involved presenting the names of objects or animals about which the patient had to make some judgement based on the visual appearance of the object. Specifically, the tasks involved identifying the characteristic colour of objects, deciding on the relative size of two items that are similar in physical size, judging the length of animals' tails relative to their body size, and judging the similarity in the shapes of states of the USA. The spatial tasks involved mental rotation, mental scanning, Brooks matrix and letter corner tasks (Brooks, 1967; 1968; for a description see Chapter 3), and the locations of states of the USA. Subject L.H. showed significantly poorer performance on all four visual tasks. However he showed performance equivalent to that of controls on all of the spatial tasks, and in some cases (e.g. Brooks letter corner task) L.H. performed better than did control subjects.

Hanley et al. (1991; Hanley, Pearson, & Young, 1990) described a patient who appeared to have the opposite deficit, namely a sparing of visual imagery but with a deficit in spatial processing following a right hemisphere aneurysm. Patient E.L.D. showed poor performance relative to controls on the Brooks matrix task and on Corsi blocks. However she performed well on versions of the four tasks used by Farah et al. (1988) to test visual processing.

Although these two patients provide an apparent double dissociation, the neuroanatomical evidence is less clear, as both L.H. and E.L.D. had right hemisphere lesions, with L.H. having an additional lesion in the posterior left hemisphere. Moreover, E.L.D. has fairly widespread damage across the frontal area of the right hemisphere, and this provides little information as to the neuroanatomical corollaries of her functional impairments.

Other studies have indicated the contrasting result that right anterior temporal damage results in impairments in recognition and recall of photographs of faces, of geometric designs, and of complex visual scenes (Kimura, 1963; Milner, 1968; Pigott & Milner, 1993). The extent of the impairment in each of these studies did not appear to be related to whether

involved brightness judgements (Baddeley & Lieberman, 1980). The primary spatial tasks were disrupted by concurrent movement but were unaffected by concurrent visual processing. In contrast, visual retention tasks were disrupted by secondary visual tasks but not by secondary spatial tasks (Baddeley & Lieberman, 1980; Farmer et al., 1986; Logie, 1986; Logie & Marchetti, 1991). It would be interesting then at this stage to explore whether there are patients with disorders of spatial representation and processing who have spared visual processing and retention functions.

One candidate pattern of impairments has been reported in studies of patients suffering from Optic Ataxia. Patients with this disorder appear to have normal visual acuity and to have normal perception and representation of visual space, but they are unable to use visual information to coordinate their arm movements when reaching out for objects or other visual targets. This is true for both familiar and for unfamiliar targets. For example, when reaching out to grasp an object, the patients initially may open their hand too far and may close their grip prematurely, thereby missing the object completely or knocking it over (Jakobson, Archibald, Carey, & Goodale, 1991; Jeannerod, 1986). Despite this they are unimpaired on general motor ability or strength of grasp, and are able to reach accurately in response to an auditory or kinaesthetic cue. Therefore the inability is in using visual information to guide movement rather than in movement control.

A very clear report of 10 such patients was published by Perenin and Vighetto in 1988. Their patients had lesions in the posterior parietal cortex on either the left or right hemisphere. All the patients appeared able to perceive visual scenes accurately, but were unable to orient their arm movement correctly to visually presented targets when the movement involved the arm contralateral to that of the lesion. When using the arm on the impaired side, the patients made significant errors in reaching for familiar objects such as a large pencil. In another task, the patients were shown circular cards, with a rectangular hole cut out of the middle. Their goal was to reach out and orient their hand so as to place it in the rectangular gap. None of the patients was able to do this successfully with the hand on the side opposite to that of their lesion. In all cases patients made successful targeted movements using the hand that was on the same side as the lesion. Other cases have been reported where the reaching impairment affects both hands in the same patient (e.g. Damasio & Benton, 1979; Rondot, De Recondo, & Ribadeau Dumas, 1977; see also De Renzi, 1982, p. 113 ff). In these patients, then, there is evidence that is consistent with a dissociation between visual information about an object and the processing of the spatial information necessary for physically interacting with the object.

been shown to be closely linked to hippocampal damage (Corkin, 1965; Milner, 1965, 1971; Petrides, 1985; Pigott & Milner, 1993; Smith & Milner, 1989). For example the recent study by Pigott and Milner (1993) reported visual and spatial memory deficits in a group of 65 patients with right anterior temporal damage. The patients were shown complex visual scenes derived from Mandler and Johnson (1976), and tested for recognition and recall of the figurative details of the objects or for their spatial locations. All of the patients were impaired in their ability to remember details of the visual characteristics of objects in the presented scenes. However only those patients with extensive damage to the hippocampus, or removal of the hippocampus, were impaired in their ability to remember the location of objects in the scene. Pigott and Milner go on to argue that spatial memory depends on an intact hippocampus whereas visual memory appears to rely on the right anterior temporal lobe.

Further evidence from patients has reported that cortical excisions for temporal cortical epilepsy in the right hemisphere impairs spatial memory, but leaves verbal short-term memory intact (Corkin, 1965; Kimura, 1963; Milner, 1965). In other studies of groups of patients, visuo-spatial memory deficits are most frequently associated with lesions in the posterior part of the right hemisphere, specifically in the posterior parietal lobe close to its junction with the occipital lobe (Alajouanine, 1960; Butters, Barton, & Brody, 1970; Warrington & James, 1967). The distinction is also clear when considering the patients described by De Renzi and Nichelli (1975; De Renzi et al., 1977), and by Hanley et al. (1991) all of whom had deficits in the right hemisphere, and all of whom showed deficits of primarily spatial tasks such as the Brooks matrix or the Corsi Blocks. Patients with deficits in retention of primarily visual information such as the Warrington and Rabin (1971) "span of apprehension" test tended to have lesions in the posterior left hemisphere.

VISUAL INFORMATION IN MOVEMENT CONTROL

The discussion of neuropsychological data so far has concentrated on mental representation of visual and/or spatial information. In Chapter 4, the discussion of separate visual and spatial components of working memory led to the association between spatial processing and the control of movement. The argument was based on dual task studies comprising for example some form of spatial processing or spatial memory task, coupled with visual or spatial secondary tasks. The spatial secondary tasks involved arm movement such as tracking a moving target or sequentially tapping an array of unseen switches, while the visual secondary tasks

VISUO-SPATIAL REPRESENTATIONS AND NEUROANATOMY

It will be clear from the discussion so far that although there is reasonably strong neuropsychological evidence for a dissociation between visual and spatial short-term memory, there is no clear agreement as to the neuroanatomy that might be linked with this dissociation. Some further clues have been derived from some detailed neuroanatomical studies of non-human primates (Derrington & Lennie, 1984; DeYoe & Van Essen, 1988; Maunsell & Newsome, 1987; Mishkin, Ungerleider, & Macko, 1983; Ungerleider & Mishkin, 1982; Van Essen, Anderson, & Felleman, 1992). The thesis from these studies is that visual information is projected via a common pathway to the occipital lobe in a region of the striatal cortex known as V1. From that point, there are two independent routes, one of which goes via area V5 in the occipital lobe, ending up in the parietal cortex. This dorsal pathway known as the **magnocellular** or "**M**" stream appears to deal with object location, or "where" an object is. This pathway appears to be sensitive to low spatial frequencies and has high contrast sensitivity as well as being sensitive to high temporal frequencies. This last property also associates the pathway with the processing of movement. The second, ventral pathway known as the **parvocellular** or "**P**" stream ends up in the temporal cortex via area V4. This pathway is sensitive to high spatial frequencies, to low temporal frequencies, and to spectral information such as colour and shade. This combination of properties is associated with object identification, object form, and object colour, or "what" an object is.

However, despite the initial appeal of the "what" and "where" pathways, the situation is actually rather more complex. Zeki and Shipp (1988) have noted that although area V4 projects to the temporal cortex, it also projects to an area of the parietal cortex. This area is in turn linked to the destination in the parietal cortex of the "where" (V5) route. Similarly V5 projects to both the temporal and the parietal cortex, with subsequent interconnections between the destinations.

We can cope with this further complication by noting that even though visual and spatial information may be handled separately at one level of processing, it is clear that normal subjects can remember the location of particular objects, and at some level of the processing system these two items of information have to be coordinated. For example in following a moving object across the visual scene we are aware of the movement and can retain information about that movement independently of the identity of the moving object. However normal adults are quite capable of associating object identity with object trajectory. For example we can note that we saw a bird or a rugby ball and observe that they followed the same

scored if we saw a crow fly between the upright posts on the rugby field! Zeki and Shipp specifically suggest that the scheme of having separate pathways which later converge may be one that is repeated across the cortex, providing the combination of specialised, parallel processing together with coordination of the products of that processing.

A key aspect of representing visual and spatial information is that it cannot of course be entirely dependent on the mapping of object position to retinal coordinates. The eyes move about continuously and rapidly, and yet most of the time we are left with the impression of a stable environment. The repeated fixations of the eyes must feed into some internal representation of the environment, and that representation must incorporate not just visual information, but also information concerned with the relationship of the body to objects in space. There is a large literature on possible neuroanatomical correlates of this representation, some of which I have already described. For example Pigott and Milner (1993) and others have argued that the hippocampus hosts the representation of spatial information.

Stein (1992) has suggested that the parietal cortex, and in particular the posterior parietal cortex, is a much better candidate as a locus for the representation of space in relation to the body, or egocentric space. He argues that the hippocampus appears to deal with information about the spatial relationships among objects in space, but does not incorporate the information about the positions of body parts. He points out that the posterior area of the parietal lobe receives inputs not only from the primary visual cortex via the "what" and "where" pathways as noted by Zeki and Shipp, but also from the auditory system, the proprioceptive system, the vestibular system, and the somatosensory system. Moreover, there are projections to the posterior parietal cortex from areas dealing with information about movement of the eyes, head, limbs, and body. Thus the information that is coordinated in the parietal cortex concerns not only the association between where an object is and what it is, but also the relation between the position and movement of parts of the body and the relative positions of body parts to objects in space.

Stein's views are not wholly new (e.g. De Renzi, 1982) but are still controversial, as is evident from the commentaries that are included in the same issue of the journal in which his article appeared. However his arguments are persuasive and they have considerable appeal for theories of normal cognition that are concerned with mental representation. Moreover, damage to the posterior parietal cortex has also been linked to impairments in visuo-spatial imagery (Bisiach & Luzzatti, 1978; Bisiach et al., 1979). Some researchers are rather less specific, linking visual imagery with the posterior region of the left hemisphere (e.g. Farah, 1984),

suggestion is that each type of information projected onto the posterior parietal area weights the connections differentially according to the nature of the source, and this allows the PDP network to represent egocentric space topographically. When one information source is damaged, the weights of the connections are then determined by information arriving from intact sources, and a topographic representation can still emerge. It is not as rich a representation, but may be adequate for interacting with objects that are within reach. This demonstrates an attractive feature of PDP networks in that they degrade gracefully rather than catastrophically when they encounter loss of data. The network also degrades gracefully if the damage is to the network rather than to an information source that feeds into the network. Thus a pattern of impairment might arise from damage to the network, or from damage to a pathway that projects to that network. In principle, it might be impossible to tell which bit of the system is damaged by referring to behavioural data. In one sense this makes life difficult for cognitive neuropsychology, but it provides a very neat explanation for why similar patterns of impairment can arise from lesions with very different neuroanatomical locations.

The PDP approach has considerable appeal, but of course does not provide an explanation at a functional, psychological level. In this case, the argument can be translated fairly readily by suggesting that neuropsychological patients may develop strategies to help them cope with their damage. Moreover, different individual patients with the same damage might develop different coping strategies. Thus patterns of sparing and impairment might reflect the nature and efficiency of the coping strategies, and not the nature of the damaged system. It is widely known that normal subjects develop strategies to cope with the requirements of an experimental task (e.g. Delia Sala et al., 1991; Siegler, 1987), although this fact is rarely acknowledged in studies of normal cognition (Logie et al., submitted). It is also well known within cognitive neuropsychology that patients develop strategies, but here again, the fact is rarely acknowledged. Caramazza (1986; Caramazza & McCloskey, 1988) has argued that by their very nature, patient groups are rarely homogeneous with respect to their lesion site, even among patients who have apparently the same neuropsychological profile. He points out that group studies of patients are therefore very misleading in that the aggregate data from the group cannot reflect the nature of the damage suffered by any one patient. Moreover, he argues that if the patient group is homogeneous, then there is little point in conducting a group study, because the data pattern from any one patient should reflect the pattern of the group as a whole.

The way forward, according to Caramazza, is to conduct multiple single case-studies of patients, with each patient being the subject of a series of experiments. The cumulative record across patients then provides us with

there appears to be quite strong **neuroanatomical** evidence for the conflation of several sources of information about egocentric space, and that these sources of information may function more or less independently of one another, until such time as the various information types are brought together.

An interesting corollary of this form of organisation is that the loss of any one source does not cause a complete breakdown of the representation, because other complementary sources are available. So, for example, if purely visual information is unavailable, a sense of egocentric space can be gained from kinaesthetic, auditory, somasthetic, and proprioceptive information. This kind of scenario would readily account for the reports of a form of spatial imagery in the blind (e.g. Cornoldi et al., 1991; Kerr, 1983; Millar, 1990). One argument, then, could be that the visual image is not merely a visual and spatial representation, but also contains information as to some form of egocentric frame of reference.

An emergent consequence of this argument is that it complicates the interpretation of neuropsychological studies. Specifically, when observing a pattern of impairment, there is no guarantee that this pattern arises from the functioning of some global representation that simply has a chunk of missing data from a damaged pathway. The pattern of impairment and sparing might arise from a set of algorithms (or strategies) which attempt to form a functional, global representation derived from the information that is available. So, for example, if the "where" pathway from the visual cortex is damaged, information may reach the posterior parietal cortex via the "what" pathway, and this would be combined with data that were available from tactile, vestibular, and auditory sources. In other words, do the behavioural, neurophysiological, neuroanatomical, and event-related potential data from brain-damaged patients reflect the operation of a damaged system, or of a "coping algorithm" that has arisen in response to the damage? This algorithm may not be evident in normal cognition. Performance of a brain-damaged subject, then, is not simply normal performance that has a bit missing.

Some insight into this problem in theory development can be gained if we return to a section of the article by Stein (1992) where he briefly describes a PDP type model of the functioning of the posterior parietal lobe, and how such a model might account for how a coherent representation of egocentric space could be derived from information from very different sources, and which use different coordinate systems. For example the retinal coordinate system is not entirely linear, with an over-representation of data from the fovea, and under-representation from peripheral vision. The coordinate system based on tactile and proprioceptive input relies on information about the distances between body parts, and their mutual spatial relationships. In essence, his

some insight into the disorders. I should like to go further and argue that even when patients have the same lesion site, they may not attempt to cope with their damage in the same way. In addition, some patients may change their strategies from one occasion to another. Therefore in testing any patient there should be measures of test–retest reliability on each test performed, to ensure that the patient is at least performing the test reliably in the same way. By this means we might get some notion as to the pattern of performance that arises when patients use a particular strategy. The hope is that this strategy reliably uses a particular combination of components of the available cognitive architecture.

The issue remains as to whether studies of neuropsychological patients can actually tell us anything at all about normal human cognition. What we end up with is a set of theories about the functioning of damaged systems, and no coherent way to link these to theories of normal cognitive function. The method that best seems to deal with this apparent impasse is to attempt an approach based on converging operations, looking for common conclusions derived from a wide range of data sources rather than relying solely on behavioural, neuropsychological data. This brief review indicates that the neuropsychological data, brain scan data, and event-related potential data, do appear to converge on the main theme of the last two chapters. There does indeed appear to be strong evidence that spatial and visual information are processed separately, but that they are brought together to form some global representation of egocentric space. This global representation does also appear to be available for manipulation in the form of visual images, and there are links between that representation and the planning and control of movement to targets.

This conclusion hints at the functional model, in that visual short-term memory may be considered a separate system from spatial short-term memory, but that both kinds of information are contained in visual images. It provides a basis for the dichotomy between visual and spatial working memory, and for a dissociation between both of these specialist systems and the imagery system. A detailed characterisation of this view is the topic of the final chapter of the book.