ABSTRACTS

DAY 1: Tuesday, March 30th

Incidence of selective memory impairment after neonatal hypoxia/ischemia: cardiorespiratory cohort

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Objectives: Neonates with cardiorespiratory disease experience episodes of hypoxia/ischemia. The hippocampus, a structure essential for memory, is particularly vulnerable to such episodes. Hence, neonates undergoing treatment for cardiorespiratory disorders may be at risk of bilateral hippocampal pathology and emerging memory problems later in childhood.

Methods: Forty-four children treated for neonatal cardiorespiratory distress (mean age, 11.66, SD 1.63) and 64 age-matched controls (mean age, 10.94, SD 2.07) were examined on standardized measures of intelligence, memory, academic attainments, and semantic knowledge to characterize their cognitive profiles, and to determine the incidence of selective memory problems. Hippocampal volumes (mm^3) were measured using MRI corrected for intracranial volumes.

Results: The patient group scored in the average range on tests of academic attainments, semantic knowledge, and intelligence. However, on hippocampal-dependent tests of episodic and delayed memory, the patient group's mean socres were significantly lower than those of the control group, and the standard population mean (i.e. 100, SD 15). Ten patients (23%) had significant and selective episodic memory impairments (2 SDs below the standard mean), but otherwise normal cognitive profiles. In this subgroup, hippocampal volumes were reduced by 12% relative to controls, despite mean total grey matter, white matter, CSF, and intracranial volumes being comparable. A partial correlation controlling for age and IQ revealed a significant positive relationship between hippocampal volumes and episodic memory scores ($r=0.67$, $p<0.05$).

Conclusions: A significant proportion of children with neonatal hypoxia/ischemia associated with cardiorespiratory disease show selective memory deficits and hippocampal atrophy, but spared cognitive and neurological status.

Incidental recognition memory in developmental amnesia

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Incidental recognition memory is the spontaneous ability to discriminate novel objects from familiar ones. This type of recognition is reflected in the automatic attraction of one's gaze by novel objects, i.e. novelty preference, a behaviour consistently observed across species, including humans, monkeys, and rodents. However, whereas animal research points to the importance of

the hippocampus for incidental recognition, evidence in humans is more circumscribed. To test whether, like episodic memory, this type of memory requires the hippocampus in humans, we evaluated patients with developmental amnesia (DA), a syndrome caused by early episodes of hypoxia-ischemia and characterized by selective hippocampal damage and severe impairment of everyday memory, but with relatively preserved IQ. Patients with DA (n = 5) and normal controls (NC = 29) were examined in a visual paired comparison task. In each of the 64 unique trials of this task, a pair of identical stimuli (landscapes or fractal images) were presented for familiarization and, after a variable delay of 0, 5, 30, or 120 s, the familiarized stimulus was presented again but this time paired with a novel one. Preferential looking at the new stimulus, measured by monitoring eye fixations, signals recognition of the previously presented picture as "familiar". Patients and controls had similar mean looking times during familiarization trials (mean $\pm$ SD: DA, 5.9 $\pm$ 0.9; NC, 5.6 $\pm$ 1). In contrast, although preferential looking to novel stimuli by patients equalled that of controls at 0 and 5 s delays between familiarization and test, it was impaired relative to the controls' preferential looking at 30 and 120 s delays, falling to chance levels (ps > 0.01) for both stimulus types (interaction of delay x group $F[1,32] = 5.08$, $p = 0.031$ in ANOVA with repeated measures; independent t-test for 30 s delay: $t[32] = 2.71$, $p = 0.011$, and for 120 s ($t[32] = 2.37$, $p = 0.024$). The results indicate that the integrity of the hippocampus is required for delayed incidental recognition in humans. They also suggest that neural plasticity during development does not allow other structures or circuits to substitute for the damaged hippocampus in enabling this form of incidental recognition memory.

Are there multiple-memory systems? Tests of models of recognition, priming, and fluency

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Objectives: To test single-system and multiple-systems versions of a signal-detection model of recognition, priming and fluency proposed by Berry, Shanks, and Henson (2008), and to also test predictions of the related dual-process model of recognition (Yonelinas, 1994).

Methods: Three behavioural experiments were conducted with normal adult participants using a continuous identification with recognition (CID-R) paradigm. This paradigm allows recognition judgments and identification reaction times (RTs; which form the basis of priming and fluency measures) to be measured concurrently for each item at test. Models were fit to the data using maximum-likelihood methods.

Results: The models differ in the extent to which they predict that recognition judgments are related to RTs. The results largely favoured the single-system model predictions: RTs were related to recognition judgments when the judgments were either old-new (Experiment 1), six-point confidence (Experiment 2), or remember-know (Experiment 3). Furthermore, in all experiments, a priming effect occurred for items not recognized, and this effect was smaller than the overall priming effect. Also, in Experiment 1, priming for unattended items reduced to chance as recognition approached chance-levels. Finally, priming and recognition were weakly (but not reliably) correlated across experiments.

Conclusions: The results are largely consistent with the notion that one memory strength signal drives recognition, priming and fluency, rather than multiple independent implicit/explicit signals. The results are also consistent with the notion that Remember and Know judgments index memories of high/low strength. The results demonstrate the potential to move the single- versus multiple-memory systems debate forward by the exploration of formal models.

Berry, C. J., Shanks, D. R., & Henson, R. N. A. (2008). A unitary signal-detection model of implicit and explicit memory. Trends in Cognitive Sciences, 12, 367–373.
Yonelinas, A. P. (1994). Receiver operating characteristics in recognition memory: Evidence for a dual process model. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 20, 1341–1354.

SYMPOSIUM: ORGANIC AMNESIA: WHAT DOES IT IMPLY ABOUT THE NEURAL BASES OF MEMORY? (Organiser: Andrew Mayes)

Retrograde amnesia - are we nearer an understanding?

Michael Kopelman
Institute of Psychiatry, Kings College London

There are three main theories of retrograde amnesia. (i) Consolidation theory states that there is a long-term process of consolidation of memories, combined with structural reallocation, such that they eventually become independent of medial temporal structures, and thereby spared from the effects of medial temporal damage. (ii) Episodic-to-semantic shift theory states that episodic memories acquire a more semantic form as they get older, protecting them from the effects of brain damage; this may be associated with structural reallocation. (iii) Multiple trace theory states that the hippocampi/medial temporal lobes are always involved in the storage and retrieval of episodic memories, and sparing of early episodic memories is related to the number of traces that have been laid down. This debate, particularly between consolidation and multiple trace theory, has been continuing for at least 13 years, and there are conflicting findings within both the lesion and the brain imaging literatures. These findings will be reviewed. After brief consideration of earlier studies, three recent findings from my team will be discussed: (i) A comparison between performance on a test of remote semantic memory and tests of autobiographical memory, which indicated that the relative preservation of remote autobiographical memories in Korsakoff patients did not result from an episodic-to-semantic shift. (ii) A re-analysis of findings in patients with focal medial temporal lobe atrophy, indicating that there was a temporal gradient in their recollection of remote autobiographical memories. (iii) A functional imaging investigation indicating that residual tissue in the hippocampus is activated in autobiographical memory retrieval, even when atrophied; but that, as the atrophy progresses, bilateral frontal and right lateral temporal structures are recruited in autobiographical memory retrieval. The implications of these various findings will be considered.

How distinct are the neural bases of recollection and familiarity? Evidence from amnesia

Daniela Montaldi
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The dual process model of recognition proposes that recognition can be supported by two kinds of memory; recollection and familiarity. The critical difference between these is that the former involves some form of recall while the latter does not. It is generally agreed that the hippocampus plays a key role in recall memory. However, the extent to which it only plays this role rather than also contributing to the processes of familiarity is a strongly debated issue. While recent fMRI research has started to contribute to the resolution of this debate, neuropsychological data focusing on selective hippocampal patients has remained inexplicably conflicting over the last decade. A summary of these data and possible reasons for the lack of convergence will be presented as will data from a recent study exploring recognition memory performance in a large group of patients with damage to the fornix and mammillary bodies – part of the extended

hippocampal system. Critically, this study draws on four methods of distinguishing recollection and familiarity performance combined with detailed MRI measurement to reveal a selective role for the hippocampus. These and other data, including those from animal lesion studies, will be used to support a view of medial temporal lobe function that stresses differentiation on the basis of processing rather than other differences.

Memory, perception, and the ventral visual-perirhinal-hippocampal stream: thinking outside of the boxes

Lisa Saksida and Tim Bussey
Department of Experimental Psychology, University of Cambridge

The prevailing paradigm in cognitive neuroscience assumes that the brain can be best understood as consisting of modules specialised for different psychological functions. Within the field of memory, we assume modules for different kinds of memory. The most influential version of this view posits a module called the "medial temporal lobe memory system" which operates in the service of declarative memory. This system can be contrasted with a separate "perceptual representation system" in the ventral visual stream, which is critical for perceptual learning and memory, an example of nondeclarative function. In this talk, we will suggest that a potentially better way to understand the ventral visual-perirhinal-hippocampal stream is as a hierarchically organised representational continuum. This representational-hierarchical view makes specific predictions related to classic issues in amnesia research, namely whether amnesia is due to a deficit of encoding, storage or retrieval, and the related issue of the role of interference in amnesia. It further suggests that, in general, rather than trying to map psychological functions onto brain modules, we could benefit by instead attempting to understand the functions of brain regions in terms of the representations they contain, and the computations they perform.

Extending the 'pathology' in diencephalic amnesia

John Aggleton
School of Psychology, Cardiff University

Understanding how selective brain injury might affect memory depends on accurate assessments of the pathology responsible for the cognitive changes. Descriptions of structural atrophy and overt cellular loss provide a vital start point, but there remains the potential for other, more subtle cellular changes that could also contribute to the cognitive changes. This possibility was explored in the context of diencephalic amnesia, starting with the assumption that damage to the anterior thalamic nuclei is a consistent feature of this condition.

Selective lesions were made in the anterior thalamic nuclei of rats. The expression of c-*fos* and zif*268* (two immediate-early genes) was used to assess the status of other brain sites. A loss of immediate-early gene activity was noted in various structures, including multiple subfields within the hippocampus, but was particularly extreme in the retrosplenial cortex. This same cortical region showed no evidence of neuronal cell loss but slice-recording studies showed that anterior thalamic lesions blocked retrosplenial synaptic plasticity (long-term depression). These cortical dysfunctions, which appear to be permanent, may prove to contribute to the pattern of cognitive changes associated with anterior thalamic damage and so extend our concept of diencephalic amnesia.

Thalamic amnesia: a reappraisal

Augusto Carlesimo
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It has been long recognized that focal lesions of the thalami, mainly of vascular origin, may produce in humans a memory disturbance (e.g., Castaigne et al., 1980; von Cramon et al., 1985). It has been generally hold that an amnesic syndrome in thalamic patients results from a damage to connections between mesio-temporal structures (MTL) and gray matter nuclei located in the anterior portions of the thalami. There is both autoptical (Castaigne et al., 1980) and neuroradiological (Van Der Werf et al., 2003) evidence that because an amnesic syndrome is observed, the critical structure to be lesioned in this area is the mammillo-thalamic tract (MTT) which connects the medial and lateral nuclei of the mammillary bodies with the anterior thalamic nuclei. Graff-Radford et al. (1990) also pointed out the existence of an other MTL-thalamic connection (known as ventroamygdalofugal pathway) which conveys fibers from the perirhinal cortex (and the adjacent amygdala) to the medio-dorsal (MD) nucleus of the thalamus. The fact that two parallel but distinct fiber pathways, the first originating from the hippocampus proper, the other from the perirhinal cortex in the parahippocampal gyrus, connect the MTL to the thalamus raises the question as to whether the functional specialization hypothesized for the different components of the MTL can also extend to different portions of the thalami. In particular, it is discussed whether damage to the MTT-anterior nuclei could mainly affect the recollective component of declarative memory while a damage to the ventroamygdalofugal pathway and related nuclei (mainly the MD) could particularly compromise the familiarity-based component of memory (Aggleton and Brown, 1999; Yonelinas, 2002). Evidences either supporting (e.g., Carlesimo et al., 2007) or challenging (Cipolotti et al., 2008) this proposed dissociation will be reviewed. Theoretical as well as methodological issues relevant for this controversial topic will be addressed.

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Castaigne P, Lhermitte F, Buge A, Escourolle R, Hauw JJ, Lyon-Caen O. Paramedian thalamic and midbrain infarct: clinical and neuropathological study. Ann Neurol, 10:127-48, 1981.
Cipolotti L, Husain M, Crinion J, Bird CM, Khan SS, Losseff N, Howard RS, Leff AP. The role of the thalamus in amnesia: a tractography, high-resolution MRI and neuropsychological study. Neuropsychologia, 46:2745-58, 2008.
Graff-Radford NR, Tranel D, Van Hoesen GW, Brandt JP. Diencephalic amnesia. Brain 113:1-25, 1990.

Decoding memories: evidence from neuropsychology and high resolution fMRI

Eleanor Maguire
Wellcome Trust Centre for Neuroimaging, Institute of Neurology, University College London

In this talk I will consider the role of the medial temporal lobe, particularly the hippocampus, in supporting memory. I will draw on neuropsychological evidence from patients with primary damage to the hippocampus bilaterally and amnesia, whilst also describing some recent high resolution fMRI findings. The latter studies employed multivariate pattern analysis, a technique that allowed us to predict which specific past experience a participant was recalling, solely from the pattern of fMRI signals in the hippocampus. Our recent extension of this work into hippocampal subfields in vivo in humans is starting to open up a range of novel opportunities to explore what information the human hippocampus represents, and how and why its contribution is so crucial for memory.

Do parietal lobe lesions cause amnesia?

Jon Simons
Department of Experimental Psychology, University of Cambridge

An intriguing puzzle in cognitive neuroscience in recent years has been the common observation of parietal lobe activation in functional neuroimaging studies of human memory. These findings have surprised neuropsychologists who have traditionally associated the parietal lobe with spatial attention rather than memory. However, direct empirical investigation of whether circumscribed parietal lobe lesions might indeed be associated with human memory impairment has been lacking. In this talk, I will describe some recent experiments investigating memory function following unilateral and bilateral parietal lobe damage. These studies indicate that the lateral parietal lobe is not necessary for accurate performance on many memory tasks but that damage to this region can impair the subjective experience of rich episodic recollection.

PRESIDENT'S INVITED LECTURE
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Conscious and unconscious memory systems of the mammalian brain

Larry Squire
University of California, San Diego, USA

The medial temporal lobe includes a system of anatomically related structures that are essential for declarative memory (conscious memory for facts and events). Other brain systems support a heterogeneous collection of nondeclarative, unconscious forms of learning and memory including the ability to acquire skills and habits. The function of these systems has been illuminated by explorations of spatial cognition, remote memory, and the constructs of recollection and familiarity.

DAY 2: Wednesday, March 31st

SA-squared: Semantic ambiguity accounts for the missing word frequency effect in semantic aphasia

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Objectives: Multimodal comprehension impairments can result from degraded conceptual knowledge representations (semantic dementia - SD) or impairment to control processes that bias activation towards task-relevant aspects of knowledge (multimodal semantic deficits following stroke, termed semantic aphasia - SA). This distinction can account for the differing comprehension profiles of these two groups. One difference that remains puzzling is the lack of word frequency effects in SA, compared with robust frequency effects in SD. We hypothesised that high frequency words have greater control demands because they are more semantically ambiguous – they tend to have more variable meanings and to appear in a broader range of linguistic contexts – explaining why frequency effects are not observed in SA.

Methods: We developed a new measure of semantic ambiguity based on latent semantic analysis that took into account the similarity between the different contexts in which a word can appear. Regression analyses were used to investigate how ambiguity and frequency influenced comprehension in both groups.

Results: SD patients were highly sensitive to frequency and ambiguity had no effect in this group, in line with their representational deficit. SA patients were poorer at comprehending more ambiguous words and, most importantly, they did show normal frequency effects, but only when ambiguity was taken into account.

Conclusions: SA patients tend not to show the expected comprehension advantage for high frequency words because these words are associated with greater semantic ambiguity, which place greater demands on their impaired control systems.

Syntactic comprehension following stroke: right-hemisphere recruitment cannot compensate for damage to left inferior frontal gyrus

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[2]School of Clinical Medicine, University of Cambridge

Objectives: For 150 years, the role of sub-regions (Broca's area) of left inferior frontal gyrus (LIFG) in syntactic processing has been hotly debated. Here we combine measures of functional activity, tissue integrity and performance in left-hemisphere damaged patients and healthy participants to ask whether sub-regions of LIFG are essential for syntactic processing.

Methods: In a functional neuroimaging (fMRI) study, using a task that minimises working memory and cognitive demands, participants heard spoken sentences which differentially loaded on syntactic and semantic information. Task performance and fMRI activation were related to tissue integrity in patients using voxelwise correlations with T1-weighted structural images.

Results: While healthy controls activated a left-hemispheric network including Brodmann area (BA) 45/47 and posterior middle temporal gyrus (LpMTG) during syntactic processing, patients activated BA 45/47 bilaterally and RMTG. However, only tissue integrity in LBA45 was correlated with activity and performance. Decreased tissue integrity in LBA45 was associated with reduced functional activity and increased syntactic deficits.

Conclusions: These results argue for the essential contribution of regions of LIFG in syntactic analysis and highlight the functional relationship between LBA45/47 and LpMTG. In healthy participants, but not in patients, activity in these regions was correlated, suggesting that when this relationship breaks down, through damage to LIFG or through its effects on the connectivity between frontal and temporal regions, syntactic processing is impaired.

Distributed frontotemporal connectivity supports semantic and episodic processing in older adults

Simon Davis and Roberto Cabeza
Center for Cognitive Neuroscience, Duke University, USA

Objectives: An ongoing debate in the aging literature is whether age-related increases in frontal and temporal activity represent functional compensation for the effects of aging, or instead reflect non-specific increase in effort. However, differences in behavioral performance may complicate

comparisons between young and old adults. Semantic tasks, which tend not show large age effects, represent an ideal way to investigate systematic changes in the structural (SC) and functional connectivity (FC) that reflect true age-related neural reorganization, and offer a means to interpret age-related increases in other tasks affected by age.

Methods: In our study we used a combined FC (single-trial correlations) and SC (DTI tractography) analysis as a basis for investigating age-related changes in networks subserving semantic retrieval and episodic encoding.

Results: Older adults showed preserved semantic and impaired episodic memory performance when compared to younger adults. In the semantic task, age-related increases in FC between frontal and anterior temporal regions increased with task difficulty in older adults, and, frontotemporal SC predicted both FC during the task as well as successful behavioral performance in older adults. In the episodic task, frontotemporal SC and FC predicted subsequent memory performance in older but not younger adults. Critically, there was significant overlap in FC patterns in both semantic and episodic tasks in older adults.

Conclusions: These results support the idea that age-related increases in frontotemporal connectivity supports both semantic and episodic memory processes, and that overlap between these networks represents common processing operations that are task relevant, and thus may represent compensatory function.

The relationship between episodic long-term memory and white matter integrity in normal aging

Rebecca Charlton[1], Thomas Barrick[1], Hugh Markus[1], and Robin Morris[2]
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[2] Department of Psychology, King's College, Institute of Psychiatry, University of London

Objectives: Although is it well known that episodic long-term memory (LTM) declines in normal ageing there is less agreement on the neurobiological mechanisms that underlie such changes. Unlike neurodegenerative disorders that are linked to neuropathological changes in grey matter, in normal ageing decline in LTM may be affected by disruption of white matter networks.

Methods: The role of white matter in LTM was explored in 104 healthy adults aged 50-90 years in the GENIE study. White matter integrity was assessed using diffusion tensor imaging (DTI) in large regions of interest, with additional measures of white matter hyperintensities (WMH), normalised brain and hippocampal volumes. The mediating effects of executive function, working memory and information processing speed on the LTM-DTI associations were explored.

Results: Memory correlated significantly with DTI, WMH and whole brain volume, but not with hippocampal volume. Using linear regression, DTI measures explained the variance (approximately 15%) in LTM. Meditational analyses demonstrated that the association between LTM function and white matter integrity was partially mediated by executive function and working memory, not at all mediated by verbal IQ, and fully mediated by information processing speed.

Conclusions: These results suggest that in normal aging, memory deficits may be related to reduction in the integrity of the distributed network that normally supports LTM, in particular the white matter connections that integrate memory processing in the prefrontal and temporal lobes. Our findings do not support a simple causal chain in which white matter change exerts an effect on memory via executive function.

Identifying patients' "good" days and "bad" days

Pat Rabbitt[1], Mary Lunn[2], and Dan Lunn[2]
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[2] Department of Statistics, University of Oxford

Decision Times are the most common performance index that is used in experimental tasks developed to test whether functional models for human cognition can be used to interpret differences in performance due to old age, brain injury, clinical depression, or differences in intelligence. So we need reassurance that any differences in decision times reflect local, rather than global differences in brain function. Unfortunately comparisons invariably show that decision times obtained from individuals who are elderly, damaged, drunk or less able are simple multiplicative products of those of their controls. A natural hope is that though means of decision times are simply scaled up their variability may differ between groups of interest. This is also contradicted by consistent findings that distributions obtained from damaged or less able individuals are simple linear transforms of those obtained from the intact and more able.

This causes further inconvenience because greater variability from trial to trial during a single administration of a task necessarily implies greater variability between mean decision times on the same task between different sessions, at different times of day, or from day to day or before and after critical events. Greater session to session variability on individual tasks also necessarily implies greater variability in performance between different tasks performed on the same or different experimental sessions, and is a confounding factor if we try to test whether some individuals experience more "good" or "bad" days on which they perform better or worse than usual.

To resolve this statistical quandary we offer a new, non-parametric statistic that evades these problems, allowing us to test whether individuals experience "good days" when they perform relatively well and "bad days" on all of several tasks that they attempt. Applying this to archived data we find that individuals who are older, or have lower intelligence test scores, or who are suffering from cardio-vascular disease or diabetes experience more frequent exceptionally "bad days" than those who are young, more able and well. We suggest that colleagues can now confidently compare the extent of fluctuations in performance across time in clinical and normal individuals, and to identify individuals who experience greater or less performance variability.

SYMPOSIUM: THE GREAT DECEPTION(S): ANOSOGNOSIA FOR HEMIPLEGIA AND OTHER DISORDERS OF MOTOR AWARENESS (Organiser: Paul Jenkinson)

Introduction to symposium

Paul Jenkinson
Department of Psychology & Mental Health, Staffordshire University

Leonardo da Vinci was remarkably astute when he declared that 'The greatest deception men suffer is from their own opinions'. This idea is inherent in current comparator/forward models of motor awareness, which claim that our knowledge of the body is a representation of external reality, constructed from sensory predictions. Unfortunately, brain injury can damage the processes whereby these internal predictions are compared with actual sensory feedback to provide true and accurate awareness. When this happens, the motor system may continue to operate by deceiving awareness. This symposium considers empirical data relating to this proposal in patients with

disorders of motor awareness, and discusses the mechanisms of normal and pathological awareness.

Imagining the impossible: motor representations in anosognosia for hemiplegia

Paul Jenkinson
Department of Psychology & Mental Health, Staffordshire University

Anosognosia for hemiplegia (AHP) is characterised by poor insight for motor impairment after brain injury. Recent explanations of AHP have used an established 'forward model', which proposes that normal motor awareness involves comparing the predicted and actual sensory consequences of movements. These accounts propose that AHP patients may be able to form representations of their intended movements (i.e., motor representations), but fail to register discrepancy between intended and actual movements. The present experiment provides direct evidence regarding the ability of AHP patients to generate these motor representations. Results are discussed in relation to current explanations of AHP which capitalise on forward models.

The breakdown of the comparator: action awareness for the non-paralysed limb in anosognosia for hemiplegia

Catherine Preston
School of Psychology, University of Nottingham

Most previous work investigating anosognosia for hemiplegia (AHP) has focused on awareness for the paralysed limb only. The current experiment, however, presents evidence for the first time that suggests AHP patients also have impaired motor awareness for movements performed with the intact limb. Unlike healthy and brain-damaged controls, AHP patient GG was unable to detect computer-generated manipulations as large as 20 degrees applied to his real movements. GG was also unaware of large on-line corrective movements and movement inaccuracy. These results are discussed along with previous findings from the literature in relation to current explanations used to model AHP.

Somatoparaphrenia, anosognosia, and the neglect syndrome

Giuseppe Vallar
Department of Psychology, University of Milano-Bicocca, and Neuropsychological Laboratory, IRCCS Instituto Auxologico Italiano, Milano, Italy

The symptom-complex of somatoparaphrenia (SCS) is reviewed. The deficit typically occurs in the context of the syndrome of unilateral spatial neglect, is associated with many of its manifestations, and largely shares its hemispheric asymmetries, and neural correlates. SCS' main manifestation is a reported feeling of estrangeness – disownership of contralesional body parts, that is frequently associated with deficits of tactile sensation and proprioception, extra-personal neglect, and anosognosia for hemiplegia, although dissociations are on record. Particularly, the feeling of disownership may occur independent of the patient's ability to explore the contralesional side of the body (personal neglect or emisomatoagnosia), suggesting that (dis)ownership of body parts relies on mechanisms independent of those concerned with awareness of those body parts. A case study

of a right-brain-damaged patient with left STS is presented, investigating the role of perceptual and intentional components in the shaping of the SCS.

Is this my hand I see before me: experimental studies on action agency and body ownership in anosognosia and hemiplegia

Katerina (Aikaterini) Fotopoulou
Institute of Cognitive Neuroscience, University College London

Introduction: When stroke causes paralysis, a variety of striking neuropsychiatric symptoms related to the paralysed limb also occurs. These include 'anosognosia for hemiplegia' (AHP; the apparent unawareness of paralysis) and somatoparaphrenia (SP; the belief that one's limbs belong to someone else). The investigation of these symptoms can inform theories of motor awareness and body representation.

Objective: Three studies are presented which investigate a total of 16 right-hemisphere lesioned patients with AHP and SP and assess the following hypotheses (1) Does motor intention influence action awareness? (2) Do 1st and 3rd perspectives on the body dissociate and can the latter be used to treat AHP and somatoparaphrenia? (3) Do implicit and explicit representations of the body dissociate?

Methods: Included creating visual illusions of movement via realistic rubber-hands, video recordings and mirror-viewing in experimentally controlled conditions. A modified verbal inhibition task was used to measure implicit emotional awareness of deficit. Lesion analysis and overlay aimed at characterising the patients' lesions.

Results: The results suggest that motor intention has a profound influence on the on-line representation of one's actions, 3rd person perspective on one's body representation is intact in AHP and SP, and emotional factors may have a top-down influence on one's body-representation.

Discussion: The relation of these findings to critical determinants of bodily representation and awareness will be discussed. These studies enrich traditional theories on bodily awareness and representation.