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ABSTRACTS

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**at
INSTITUTE OF CHILD HEALTH, central London**

Language Disorders

Acquired aphasic syndromes. A primer for neurologists, psychiatrists and Neuropsychologists

Dr Argye Beth Hillis, Johns Hopkins University School of Medicine, USA

The classic aphasic syndromes, such as Broca's aphasia and Wernicke's aphasia, each consist of a collection of separate (dissociable) impairments of language processes supplied by a single major artery. Occlusion of the associated artery causing damage to the entire vascular territory, or sufficient stenosis to cause poor blood flow in the entire vascular territory, leads to the complete syndrome. When only part of the vascular distribution is affected, selective components of the syndrome can be observed. Recovery from stroke, which takes place via a variety of mechanisms from tissue restoration to reorganization of structure/function relationships, can also result in highly selective residual deficits in isolated components of language tasks. Aphasic syndromes due to conditions other than stroke (e.g., degenerative disease, tumor, infections) do not typically correspond to classic aphasic syndromes, because the damage is not limited to vascular territories. Aphasia due to these conditions, or due to small or chronic stroke, can be best described by identifying which cognitive processes underlying language tasks are impaired. For example, picture naming is often impaired in different degenerative diseases. However, these diseases may selectively affect different components of the complex process underlying picture naming. That is, the cognitive processes required for accurate oral naming of a picture include: access to a structural description of the object depicted in the picture, access to the semantic representation or meaning, access to the spoken word form, and motor programming of the speech articulators. Since each of these component processes may take place in different brain regions, neural dysfunction of a variety of brain regions can affect oral naming, but in distinct ways. These principles will be illustrated with cases of language deficits due to a variety of neurologic disorders.

Developmental disorders of language and reading. What should adult neurologists, psychiatrists, and neuropsychologists know about these?

Professor Maggie Snowling, University of York

It is well documented that children with a history of speech-language difficulty are at high risk of literacy problems, and that children with reading disorders (e.g., dyslexia) are characterised by language processing deficits. Indeed, the co-morbidity of reading and language impairments in childhood suggests that the oral language skills and written language abilities are causally related. This paper begins by considering how reading development builds on a foundation of spoken language development and proceeds to consider the impact of different forms of language difficulty on learning to read. In particular, it highlights the importance of differentiating problems of decoding skill in reading from problems of reading comprehension and discusses how the manifestations of reading disorders change with development from pre-school through adulthood. The paper outlines procedures for the assessment, management and treatment of reading disorders before discussing their cognitive, educational and psychosocial outcomes.

References that may be useful:

Snowling, M.J. (2003) Specific learning difficulties. In Skuse, D. (Ed.), *Child Psychology and Psychiatry: An Introduction*. Pp 67- 70. Abingdon; The Medicine Publishing Company.

Nation, K. (2003) Developmental language disorders. In Skuse, D. (Ed.), *Child Psychology and Psychiatry : An Introduction*. Pp 71-74. Abingdon; The Medicine Publishing Company.

Both reprinted from the journal *Psychiatry* (2002)

For a full list of references, refer to the Centre for Reading and Language, University of York
<http://www.york.ac.uk/res/crl>

Therapy for acquired language disorders: new and old evidence on effectiveness **Professor David Howard, University of Newcastle**

Data on the effectiveness of aphasia therapy has been being collected for more than fifty years. Recent meta-analyses of the literature identify more than fifty studies adopting a variety of methodologies addressing this issue. Yet there continue to be (influential) sceptics who argue that there is no persuasive evidence for the effectiveness of aphasia therapy.

The evidence on this issue will be briefly described and the reasons for misunderstanding of both the data and the issues will be explored. The evidence shows that with therapy there is greater improvement in language test scores than without, and that with more therapy there is greater improvement.

Recent evidence from people attending the Aphasia Clinic at Newcastle will be described showing that there is much greater improvement in language scores with intensive therapy than with much less intensive management in the community. This reinforces previous evidence that aphasia therapy can be effective.

Finally, I will argue that this debate misses the point. Aphasia therapy consists of a diverse set of behavioural therapies applied to a set of people with diverse forms of breakdown in their language processing. It is almost certain that different therapy methods will be appropriate for people with different patterns of breakdown in their language processing. The way forward would be to investigate the effects of specific therapy approaches, theoretically related to an understanding of the nature of the language impairment. Much progress has been made on this in the last twenty years, but the significance of this work is not widely appreciated outside the community of those with specific interests in aphasia and aphasia therapy.

An evolutionary perspective on language and its relationship to psychosis **Professor Tim Crow, University of Oxford**

Psychosis occurs in all human populations probably at approximately the same frequency. This suggests that the origin is intrinsic, ie genetic. In the absence of a significant environmental causation an evolutionary explanation is required. Why do the genes persist? Of the balancing advantages suggested, the most cogent is the human capacity for language. According to this theory schizophrenia is the price that *Homo sapiens* pays for language.

How old is the genetic predisposition? Perhaps as old as, but no older than, the origin of the species. As Broca suggested it now seems that cerebral asymmetry is the feature that defines the human brain. Anomalies of anatomical asymmetry in psychosis have been deleted in post-mortem and MRI studies and the core schizophrenic symptoms can be regarded as disorders of language. What is the genetic basis of cerebral dominance? There is evidence from sex chromosome aneuploidies for a locus in a region of homology on the X and the Y chromosomes. The gene pair ProtocadherinX and ProtocadherinY has been under domain-specific positive selective pressure in the course of hominid evolution. The chromosomal re-arrangements that provoked these changes are putative speciation events. It is argued that psychosis and language have a common origin in the genetic and epigenetic events that defined *Homo sapiens* as a species.

References

Crow TJ. Schizophrenia as the price that *Homo sapiens* pays for language: a resolution of the central paradox in the origin of the species. *Brain Research Reviews* (2000) 31: 118-129.

Crow TJ (Ed) *The Speciation of Modern Homo Sapiens*. Oxford University Press, Oxford (2002)

Members' papers & Guest Lecture

Sleep, altered states of consciousness and psychosis

Nikola N Ilankovic, Andrej N Ilankovic, Institute of Psychiatry, Belgrade, Yugoslavia

METHODS: By 60 patients with psychotic states (depression, mania, schizophrenia) after registration of sleep (PSG), scoring and statistical analysis, we estimated the Index of Endogenous Perturbation of Sleep (IEP=REM-1/NREM-1) and some discriminative models of sleep in different psychotic states.

RESULTS: 1. The Index of Endogenous Perturbation (IEP-P1=REM-1/NREM-1) was very HIGH (I): in 1a. MODEL - in endogenous DEPRESSED, MANIC, and PARANOID states ("DELTA DEFICITE TYPE", IEP-P1>2.40), and in 1b. MODEL - in DELIRIUM ("REM SUFICITE TYPE" ∞ >IEP-P1>2.40); or very LOW (II): in 2. MODEL in SCHIZOPHRENIA ("REM DEFICITE TYPE", IEP-P1<0.3).

CONCLUSION: The results of our investigation demonstrate that the INDEX OF ENDOGENOUS SLEEP PERTURBATION (IEP-P1=REM-1/NREM-1) is the highly reliable indicator of the endogenic perturbation of sleep in: depression, mania, schizophrenia, delusional state, delirium and other psychotic states (altered states of consciousness).

The impact of unilateral anterior temporal lobectomy on emotional perception and 'theory of mind' reasoning

P Shaw, E. Lawrence, C Radbourne, J Bramham, C Polkey, A S David, Institute of Psychiatry, London

AIMS: Structures within the temporal lobe such as the amygdala and temporal poles have been proposed as pivotal components of the 'social brain', supporting functions such as the detection of, and reasoning about the mental states of others. We explored the effects of a unilateral anterior temporal lobectomy (ATL) on these aspects of social cognition.

METHODS: Subjects were tested prior to and after ATL, performed as treatment for medically intractable epilepsy (RTL 12 and LTL 13 subjects). A comparison group of patients with temporal lobe epilepsy who did not undergo any surgical intervention (N=24) and healthy controls (N=25) were also tested on two occasions. Emotional perception was assessed by 1) forced choice labelling and judging the intensity of facial expressions of the basic emotions taken from Ekman's series; 2) forced choice labelling of complex emotional expressions taken from the 'Reading the Mind in the Eyes'. Theory of mind reasoning was assessed using false belief paradigm and tasks assessing the ability to reason about the motivations of others in everyday settings (Happé Strange Stories, faux pas task, and a novel test of joint reasoning about beliefs and emotions in others).

RESULTS: Following a left temporal lobectomy there was a significant improvement in the detection of the basic emotional expressions and less deviation away from the intensity ratings given by healthy controls for each emotion. There was also a trend for improvement in the LTL group in the recognition of complex social emotions. There was no effect on theory of mind reasoning in either the RTL or LTL groups. The results were not explained by surgical outcome (which was similar in both TL groups), or post-operative changes in executive function, memory or IQ.

CONCLUSIONS: This preliminary data suggests that there are no deleterious effects on key aspects of social cognitive function following excision of the temporal lobe. Indeed after a LTL there may be partial normalization of emotional perception, perhaps reflecting a release of function in the right temporal lobe.

Not a laughing matter

D E Crompton, T D Griffiths, Newcastle General Hospital

AIMS: Video demonstration of woman with gelastic episodes 1) for the benefit of members who may not have seen this 2) as basis for brief discussion about the origins of mirth/laughter

METHODS: 1. Patient with SAH due to rupture of pericallosal aneurysm three years before assessment. 2. Frequent episodes laughing with minimal or inappropriate provocation 3. Surface EEG (normal) and structural imaging.

RESULTS: Video will show typical attacks

CONCLUSIONS: 1. Gelastic seizures can be caused by anterior cingulate damage. 2. They are often missed on surface EEG 3. This is a distressing disorder with dissociation laughter mirth consistent with other cases in literature with frontal foci 4. Gelastic seizures are not always caused by hypothalamic lesions

Catatonia in present day psychiatry – a postal questionnaire's study

D Hank, S Morgan, S Harris, D Rogers, The Burden Centre, Bristol

AIMS: To investigate psychiatrists' knowledge and understanding of catatonia, and how often they saw this condition over a two year period.

METHOD: A postal questionnaire was sent to 550 psychiatrists in the South-West of England and Wales and the collected data descriptively evaluated.

RESULTS: 158/550 (28.7%) questionnaires were returned. The majority of psychiatrists (96%) understood catatonia as a syndrome in its own right and/or a subtype of schizophrenia. More than 90% of the respondents felt able to give a definition for most of the 20 catatonia signs/symptoms listed. Two thirds of respondents (65%) indicated that they would require 3-5 of these signs/symptoms to be present in order to diagnose catatonia. 70 cases were reported for the period 2000-2002 of which 40% occurred

in the context of schizophrenia and 30% in patients with a learning disability. Treatment with ECT was given in 22 cases, with benzodiazepines in 44 cases.

CONCLUSION: Catatonia continues to be recognised in 21st century British psychiatry. However, clinicians' understanding of the condition differs which may result in under-diagnosing and sub-optimal treatment. To raise awareness of this often forgotten entity seems timely.

The neural systems that sustain sentence comprehension after left anterior temporal lobe resection for refractory epilepsy

Uta Noppeney, Matthias Koepp, Karl J. Friston, Cathy J. Price, Institute of Neurology, London

AIMS: The remarkable resilience of cognitive functions to focal brain damage indicates that multiple sets of neuronal regions can produce the same functional response. To reveal different sets of regions that can sustain sentence comprehension, this fMRI study investigated normal subjects and patients who had undergone left anterior temporal lobe resection for refractory epilepsy.

METHODS: Subjects read silently blocks of six 9-word-sentences (SOA 4.4 s) or viewed false fonts. Using a random effects analysis, we tested for activation that was (1) common to both groups, (2) decreased for patients, (3) increased for patients, (4) influenced by duration/onset of epilepsy.

RESULTS: In neurologically normal subjects, sentence comprehension activated a primarily left lateralized fronto-temporal system extending into the left temporal pole. The patients showed decreased activation in undamaged areas of the normal left hemispheric system but increased activation in several right frontal and temporal regions that are not usually engaged by normal subjects. Critically, activation several right-hemispheric regions was significantly predicted by the duration and the onset age of epilepsy highlighting the influence of the epileptic process on the language system especially during early neurodevelopment

CONCLUSIONS: These results demonstrate that in addition to the normal left hemispheric system, in patients sentence comprehension can be sustained by a right hemispheric system that has emerged primarily due to functional reorganisation during early neurodevelopment.

Presymptomatic focal atrophy precedes speech production impairment in familial frontotemporal lobar degeneration

JM Schott, JC Janssen, L Cipolotti, NC Fox, RI Scahill, KA Josephs, JM Stevens, MN Rossor, Institute of Neurology, London

AIMS: To demonstrate the progressive pattern of in vivo atrophy in familial frontotemporal lobar degeneration (FTLD) using fluid registered MRI.

METHODS: An asymptomatic 51 year-old woman from an autosomal dominant FTLD pedigree was studied over a period of 69 months with annual clinical, MRI and neuropsychological assessments. Non-linear (fluid) registration of serial MRI scans was performed to reveal areas undergoing significant regional atrophy.

RESULTS: Over the first 26 months of the study, the patient remained asymptomatic. She subsequently developed progressive speech production difficulties, and latterly severe orofacial dyspraxia, dyscalculia, frontal executive impairment and limb dyspraxia. Regional atrophy was present prior to the onset of symptoms, and was initially centred on the left dorsolateral prefrontal cortex and the left middle frontal gyrus. Latterly, there was increasing asymmetric left frontal and parietal atrophy. Neuropsychological evaluation revealed mild intellectual impairment prior to the onset of these clinical symptoms; frontal executive, left parietal and eventually widespread cognitive impairment subsequently emerged.

CONCLUSIONS: Focal atrophy, in this case centred around Broca's area, appears to precede and underlie the development of focal deficits in FTLD. Fluid registered MRI may be a useful tool allowing delineation of the pattern and progression of neurodegenerative diseases in vivo

**Please note that an abstract reporting similar data was presented in poster form at the World Alzheimer Congress, Stockholm, 2002. These data have not been presented at a UK meeting.*

Magnetic Resonance Spectroscopy as a diagnostic tool in frontotemporal dementia

EJ Coulthard, M Firbank, PT English, D Birchall, J Welch, J O'Brien, TD Griffiths, Royal Victoria Infirmary, Newcastle

AIMS: Frontotemporal dementia (FTD) affecting the frontal lobe is often difficult to distinguish from psychiatric conditions on the basis of the phenomenology, neuropsychology, and conventional structural imaging using MRI. This study was carried out to assess whether MRI spectroscopy might allow parenchymal changes in the frontal lobes to be demonstrated at a stage before frontal lobe atrophy is apparent.

METHODS: MRI spectroscopy was performed on the temporal, parietal and cingulate cortices of five patients with the semantic dementia form of FTD and ten age-matched controls. Naa/Cr ratio (as a marker of neuronal integrity) and Mi/Cr ratio (as a marker of gliosis) were measured. Structural MRI and neuropsychometry were also carried out.

RESULTS: Group comparison of Naa/Cr ratio showed significant differences at the $p < 0.05$ level in the experimental group compared to the controls for cingulate and temporal but not parietal volumes. No consistent group effect on Mi/Cr ratio was demonstrated.

CONCLUSIONS: These data show that MRI spectroscopy can demonstrate parenchymal abnormalities in the frontal lobe of patients with FTD. This pilot work assessed subjects with established frontal lobe syndromes; it will be of considerable future interest to assess how early these changes can be detected

Language & memory impairments in a case study of semantic dementia

J J Tree, J M Kay, R Baron, C Haslam, C Williams, University of Exeter

AIMS: This case study explores the pattern of language and memory impairments in a longitudinal case study of a gentleman with semantic dementia (EP) in order to understand the nature of impairment in such cases in greater detail.

METHODS: A battery of language based neuropsychological testing was conducted, which included reading, writing, spelling, repetition tasks and measures of semantic memory. This testing was conducted over an 18-month period and mapped the progressive decline of performance on these tasks and the degree of consistency in terms of errors across language production tasks and measures of semantic memory.

RESULTS: Consistent with other cases of semantic dementia, our testing demonstrated that EP showed a marked performance decline on measures of semantic memory with disease progression. The impact of this decline in conceptual knowledge had a marked effect on picture naming performance, which declined in a similarly rapid manner. However, other language production tasks (i.e., reading, repetition and spelling) were not as severely affected. These differences in performance decline are discussed with reference to current neuropsychological models of semantic memory and word retrieval.

CONCLUSIONS: This study demonstrates the importance of detailed case study assessment of patients presenting with focal cognitive impairment. The work has allowed us to test predictions concerning the progressive impairment of particular language skills (semantic comprehension and name retrieval), in the context of apparently unimpaired phonology. We intend to continue monitoring performance in our present case study as well as recruiting further such cases in the future.

Guest Lecture - The Neuropsychiatry of Gulf War Syndrome

Professor Simon Wessely, Institute of Psychiatry

It is now well known that despite the immediate medical and military success of the 1991 Gulf War, the aftermath has proven rather more complex both politically and medically. At some unspecified date after the end of hostilities, large numbers of members of the Armed Forces in the United States, United Kingdom, Denmark, Canada, and latterly Australia, began to complain of a change in their subjective health. Numerous claims and counter claims were made. It was alleged there was no problem, or an epidemic on a vast scale. It was claimed that this was the result of variety of toxic insults, such as exposure to depleted uranium, vaccinations against biological warfare, chemical weapons, smoke from oil fires, pesticides and so on. Or that this was a psychological condition, such as post traumatic stress disorder. Some said this was new condition, others that this was a variant of shell shock or Agent Orange syndrome.

We began a large scale programme of research on members of the UK Armed Forces beginning in the mid 1990s, comparing several thousand UK gulf veterans with members of the UK military who had served elsewhere. After a large epidemiological study, we then carried out more detailed physical, psychological, neurological and immunological studies, and have also continued to follow the cohort up. At the same time, we carried out several historical studies of previous post conflict syndromes.

In this talk I will use the old English proverb to show that Gulf War Illness was indeed a reality, but one with many complex causes. It is a story of the new hazards of warfare, but also the unchanging realities of sending men into war. It is also a story of our new health fears and cultural concerns. And finally, given the theme of this conference, I will conclude that we should start thinking about depression once more in the military context, and not be quite so obsessed with PTSD.

Parietal Lobes

The neurology and neuropsychiatry of parietal damage

Professor Bob Rafal, University of Wales, Bangor

It is a sad and provocative fact that neurological patients with damage to the right temporal and parietal lobes are more likely to require residential care outside the family home than their more apparently disabled aphasic patients with comparable lesions of the left hemisphere. This talk will attempt a synthesis of the right hemisphere syndrome that explains why this is the case. It will consider several distinctive components of the syndrome that, conjointly, confound rehabilitation: loss of emotional communication, failure of global perceptual processing, spatial inattention, and degraded insight into disability and its impact on others. I will consider whether there are common pathophysiological mechanisms underlying these diverse component symptoms. I'll also consider the degree to which processing outside of awareness in patients with hemispatial neglect influences the feeling and behaviour of patients and contributes to social disability.

Distinguishing between spatial neglect and extinction and neglect in right hemisphere damage

Professor Hans-Otto Karnath, University of Tuebingen, Germany

Brain-damaged patients suffering from spatial neglect show profound deficits in everyday tasks, spontaneously gazing and exploring space predominantly to the ipsilesional side, and ignoring people or objects located on their contralesional side. Traditionally, the disorder has been associated with damage to the right inferior parietal lobule (IPL) and the right temporo-parietal junction (TPJ). Thus, it was surprising when a recent study found that the centre of lesion overlap covered the right superior temporal gyrus (STG) and planum temporale (PT) suggesting that the superior temporal cortex rather than the inferior parietal lobule is the critical substrate for spatial neglect in humans.¹ This conclusion is fundamentally different from the conclusions of previous studies and offers a new perspective regarding the function of intact superior temporal cortex.

It was speculated that methodological reasons due to (i) patient selection (excluding patients with primary visual field defects), or to (ii) the manual transfer procedure (using a standard MRI template for lesion analysis) might have led to this unsuspected finding.² To address this criticism, we have conducted two new studies. The first one was designed to rule out the selection bias argument. We used a very large, unselected 7-year sample of 140 consecutively admitted patients with right hemisphere lesions (with or without visual field defects) and applied voxelwise statistical testing for lesion analysis. The results demonstrated that the right STG, PT, and the insula are the cortical structures most frequently damaged in neglect patients.³ A second study addressed the MRI template argument. We used a lesion analysis method that did not rely on the manual transfer of lesions to standard template slices, but rather where lesions are drawn directly on the patient's own MRI scan and SPM normalization is used to transform the data into stereotaxic space. A new sample of 15 consecutively admitted patients with acute right-hemisphere ischaemic strokes and spatial neglect (with or without visual field defects)⁴ was studied. Again, we found the lesion overlap centering on the STG, the insula and the operculum.⁴

The data of both investigations thus clearly argue against the traditional view that the IPL and TPJ are the critical substrates for spatial neglect in humans. A recent study revealed that the TPJ rather seems to be the neural substrate of visual extinction,⁵ i.e. of the inability of brain-damaged patients to detect a contralesional target in the presence of a competing ipsilesional stimulus. The data of this study argue that spatial neglect and extinction are dissociable phenomena with dissociated neural substrates. The TPJ seems to play a crucial role for conscious detection of distinct stimuli or changes in the environment. In contrast, spatial neglect is associated to the STG, PT and insula, areas that underlie the spontaneous exploration of the environment.

1. Karnath H-O, Ferber S, Himmelbach M. Spatial awareness is a function of the temporal not the posterior parietal lobe. *Nature* 2001; 411: 950-953.
2. Mort DJ, Malhotra P, Mannan S, Rorden C, Pambakian A, Kennard C, Husain M. The anatomy of visual neglect. *Brain* 2003; 126: 1986-1997.
3. Karnath H-O, Fruhmann Berger M, Küker W, Rorden C. The anatomy of spatial neglect based on voxelwise statistical analysis – a study of 140 patients. Submitted.
4. Karnath H-O, Fruhmann Berger M, Zopf R, Küker W. Using SPM normalization for lesion analysis in spatial neglect. *Brain* 2004; 127: in press.
5. Karnath H-O, Himmelbach M, Küker W. The cortical substrate of visual extinction. *NeuroReport* 2003; 14: 437-442, Erratum 14: 1189.

The spatial and non-spatial functions of the parietal cortex

Dr Masud Husain, Imperial College London and Institute of Cognitive Neuroscience, University College London

Traditionally, both the monkey and human posterior parietal cortex have been considered to have a privileged role in spatial perception or action. Lesions to this region of the human brain, particularly of the right hemisphere, undoubtedly lead to spatially lateralised deficits such as visual extinction or neglect. However, although studies in monkeys have revealed much about the spatial functions of the parietal lobe, the monkey parietal cortex may not be a good model system with which to understand fully the disorders of attention that follow damage to the human posterior parietal cortex. Several lines of evidence, from functional imaging as well as investigations of patients with parietal damage, demonstrate that parts of the human inferior parietal lobe IPL have non-spatial functions. Here, I argue that it is important to distinguish spatially lateralised from spatial deficits. Both spatial and non-spatial impairments might, in principle, contribute to a spatially lateralised behavioural syndrome such as neglect. In this talk, I will consider the evidence for such a proposal and suggest that a better understanding of human parietal cortex may emerge from considering both the spatial and non-spatial functions of this region.

The Parietal Lobes and Consciousness

Dr Geraint Rees, Institute of Neurology, University College London

The immediacy and directness of our conscious experience belies the complexity of the underlying neural mechanisms, which remain incompletely understood. It is well established that functionally specialised areas of the human visual system represent properties of objects in the visual environment. This has led recently to claims that activity in visual cortex alone is both necessary and *sufficient* for a visual stimulus to be represented in consciousness. However, such a notion is challenged by the clinical syndrome of parietal neglect, which suggests that normal visual awareness relies at least partly on top-down signals from parietal cortex. This presentation will explore the role of parietal cortex in human visual awareness, drawing on recent functional MRI studies in both patients with focal parietal damage and normal subjects. This work suggests that activity in ventral occipital and temporal cortex is not sufficient to support conscious vision without a contribution from parietal and prefrontal areas. Such a contribution may reflect processes such as selective attention and working memory. Reciprocal interactions between parietal and ventral visual cortex may thus serve to selectively integrate internal representations of visual events in the broader behavioral context in which they occur. Such network interactions may account for the richness of our conscious experience, and provide a fundamental neural substrate for conscious visual experience.

Acknowledgements: This work is supported by the Wellcome Trust.

Consciousness

The concept of consciousness

Dr Adam Zeman, University of Edinburgh

The key question for a 'science of consciousness' is: how can neural processes generate our experiences - how does brain give rise to mind? This question is sometimes referred to as the 'problem of consciousness', and was recently described by EO Wilson as 'the master unsolved problem of biology'. Some contemporary neuroscientists believe that we are approaching a solution using conventional methods, others doubt that the problem will ever be fully amenable to scientific enquiry while a third camp argues that the 'problem' is ill-posed and in need of redefinition. These disagreements stem at least partly from the complexity of the concept of consciousness, and its hinterland of 'associated beliefs' which remain strongly influenced by religion: a recent survey of Edinburgh students indicates that a majority believe that the mind and the brain are separate, that each of us has a soul which is separate from the body and that some spiritual part of us survives after death. This talk aims to clarify some dimensions of the concept of consciousness which often generate confusion, by way of a series of contrasts.

First 'consciousness' comprises two partly distinct functions: 'wakefulness' and 'awareness'. The former, arousal, function has been clarified by studies of the electrical correlates of conscious states – distinguishing wakefulness, slow wave (stages 1-4), rapid eye movement sleep and pathological variants – and of their regulation by the activating systems of the brain stem and thalamus. The neural basis of awareness, the 'content' of consciousness, has been the more or less explicit target of a vast programme of research in neuroscience involving all our major psychological capacities but especially perception (from which I will draw examples). A quite separate, second, set of contrasts is opened up by the distinction between 'consciousness' and 'self-consciousness': the latter is used to refer, among other things, to the 'idea of me' (possession of a concept of self) and the 'awareness of awareness' (possession of a theory of mind). A third contrast, between 'consciousness narrow' and 'consciousness broad' highlights the distinction between the full-blooded adult human expression of consciousness, permitting self-report and the control of action, and a variety of unconscious or preconscious relatives of consciousness: much current research focuses on the 'contrastive analysis' of these two categories. A fourth distinction, between consciousness 'inner' and consciousness 'outer' picks out the distinction between consciousness considered as a fundamentally private, solipsistic phenomenon and consciousness considered as the result of a process of exploration and interaction with our physical, social and cultural surroundings. The strength of our attachment to the 'inner' view is likely to determine our reaction to the final contrast I shall draw, between the 'easy' and 'hard' problems of consciousness. This distinguishes questions amenable to objective science (for example which brain events subserve normal vision and which subserve blindsight?) from a question which some consider to be of a different kind (how do the brain events subserving normal vision give rise to the conscious experience of sight?).

A wide of ranges of disciplines and interests converge on the multi-faceted problem of consciousness: the study of conscious states, content-rich psychological processes, unconscious states, artificial intelligence, religion, the Arts, the philosophy of mind. This convergence creates a rich opportunity for cross-fertilisation - and for cross-purposes. Recent work supplies ample evidence for the feasibility of a science of wakefulness and conscious states, a science of conscious processes, such as perception, and a science of self-awareness (for example of theory of mind). The feasibility of a comprehensive science of consciousness depends on our view of its target: are we studying a private event or an objective set of interactions? To clarify this goal, scientists and philosophers need to renew their old alliance.

Conscious states, wakefulness, sleep, coma, the vegetative state and hypnosis

Dr Steven Laureys, University of Liege, Belgium

Functional neuroimaging techniques represent a useful tool to objectively quantify the residual brain function in patients with altered states of consciousness. We will here compare the cerebral function in patients who survive a severe brain injury (i.e., coma, vegetative state, minimally conscious state and locked in syndrome) with that observed in the resting conscious state, sleep and general anaesthesia. The interest of this work is twofold. First, severely brain injured patients represent a problem in terms of diagnosis, prognosis, treatment and daily management. Second, these patients offer the opportunity to explore human consciousness. Indeed, they present a complete – nearly graded – range of conscious states from unconsciousness (coma) to full awareness (locked-in syndrome). In what follows, we will put a special emphasis on the vegetative state. This condition represents a unique and complete dissociation between the two main components of consciousness: *wakefulness* -which is preserved- and *awareness* -which is abolished.¹

Compared to the conscious resting state, global brain metabolism has been shown to be significantly reduced in the vegetative state (approximately 40 to 50% of normal values). Similar values have been observed in coma, slow wave sleep and general anaesthesia. However, the recovery of consciousness from vegetative state is not always associated with substantial changes in global metabolism.² This finding led us to hypothesize that some vegetative patients are unconscious not just because of a global

loss of neuronal function, but rather due to an altered activity in some critical brain regions and to the abolished functional connections between them. In the vegetative state, the most dysfunctional brain regions are bilateral frontal and parieto-temporal associative cortices.³ Interestingly, a similar fronto-parietal network is the most active during wakefulness and the least active in coma, sleep and general anaesthesia.⁴ Despite the metabolic impairment, external stimulation still induces a significant neuronal activation (i.e., change in blood flow) in vegetative patients as shown by both noxious⁵ and auditory⁶ stimuli. However, this activation is limited to primary cortices and dissociated from higher-order associative cortices, thought to be necessary for conscious perception. Finally, we show that vegetative patients have impaired functional connections between distant cortical areas and between the thalami and the cortex and, more importantly, that recovery of consciousness is paralleled by a restoration of this cortico-thalamo-cortical interaction.⁷ Consciousness seems to rely on the functional integrity of a critical frontal-parietal 'global workspace' network and its intra- and subcortical connections.

1. Zeman A Consciousness *Brain* 2001, 124:1263-89
2. Laureys S et al Cerebral metabolism during vegetative state and after recovery to consciousness. *J. Neurol. Neurosurg. Psychiatry* 1999, 67:121
3. Laureys S et al Impaired effective cortical connectivity in vegetative state. *Neuroimage* 1999, 9:377-382
3. Baars B, Ramsay T & Laureys S Brain, conscious experience and the observing self. *Trends in Neurosciences* (in press)
4. Laureys S et al Cortical processing of noxious somatosensory stimuli in the persistent vegetative state. *Neuroimage* 2002, 17:732-41
5. Laureys S et al Auditory processing in the vegetative state. *Brain* 2000, 123:1589-1601
6. Laureys S et al Restoration of thalamocortical connectivity after recovery from persistent vegetative state. *Lancet* 2000, 355:1790-1

Conscious contents: change blindness, sensory experience and sensory substitution

Professor Kevin O'Regan, Laboratoire de Psychologie Experimentale, CNRS, University of Paris
Recent experiments in "change blindness" and "inattention blindness" seem to show that we see much less than we think we see. These findings suggest it may be time to abandon the classic model in which seeing consists in activating an internal representation of the outside world. Instead I suggest that seeing is an exploratory skill in which the outside world plays a role similar, but not identical, to memory.

The experienced quality of visual sensation derives, under this approach, from the particular skills that are put to work when we see. The "realness" or "presence" of visual experience, as compared with other mental experiences like imagining or dreaming, can be explained in terms of specificities of the agent-environment relationship involved in the skill of seeing.

Such a skill-based approach to seeing can be extended to other sensory modalities, and makes predictions about the possibility of "sensory substitution", that is, of creating visual-like sensations from, say, auditory or tactile input.

Is the mind in the head

Professor Alva Noë, University of California, Berkeley

Philosophical and empirical issues intertwine in the theory of consciousness. One idea -- that neural activity is sufficient for consciousness -- commands a strong consensus. Recent work on perceptual plasticity (by Hurley, Noë, O'Regan and others) provides a standpoint from which to rethink this assumption. The talk will explore this line of criticism. It will also provide a survey of important problems and themes in the theory of consciousness, focusing on the question: whether the study of consciousness?