regions of interest (ROI's) was determined for 28 ROI's (14 left & 14 right) including the amygdala, hippocampus, parahippocampal gyrus, mediofrontal cortex (including anterior cingulate), caudate, lentiform nucleus, thalamus and prefrontal, temporal and occipital neocortex.

A significantly increased relative uptake of FDG in the right amygdala was found in all "psychotic" patients compared to the normal controls. In the schizophrenic patients, the significant increase above normal controls was limited to the right amygdala and right parahippocampal gyrus, whereas in the affective disorder groups (both psychotic and non-psychotic) there were widespread increases across other limbic structures. Significant decreases in relative FDG uptake were apparent only in the left mediofrontal cortex in the schizophrenic and manic groups in comparison to the normal controls.

These results might be accounted for by an increase in dopaminergic input into limbic areas, which is generalised to most limbic regions in the affective disorders and localised to the right amygdala and right parahippocampal regions in schizophrenia.

ANGER AND SADNESS: A PET STUDY OF AFFECTIVE MEMORY

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Introduction. Adverse life events can induce enduring mood states and precipitate overt psychiatric disorder. We have investigated the neurophysiological mechanism whereby the recollection of life events associated with anger and sadness rekindles the emotional experience.

Methods. Male volunteers were studied with $H_2{}^{15}O$ Positron Emission Tomography, images were analysed by Statistical Parametric Mapping.

Results. Recollection of neutral memories was associated with activation of the cortex of the medial temporal pole predominantly on the right. Recollection of events associated with anger activated the insula, anterior cingulate, inferior frontal and premotor cortex and the caudate nucleus. Recollection of sad events also activated the insula and caudate nucleus. Comparison of the anger and sadness conditions revealed activation of the ventro-medial striatum specifically associated with sadness and of the anterior cingulate and inferior frontal cortex associated with anger.

Conclusions. Pathways from the medial temporal cortex to the striatum and insula constitute a neurophysiological substrate for the association of affect and memory. Affective disorder may reflect a pathophysiological interaction between psychological and constitutional factors in this network.

A SELECTIVE INTERHEMISPHERIC TRANSFER CALLOSAL DEFICIT IN AUTISM

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Here we examined autistic children for lateralised and interhemispheric transfer abnormalities. Two studies were carried out, ten years apart, on groups of English and Welsh children. In the first children matched textures by touch. In the autistic group (N = 24) there was a selective impairment in contralateral matching between the hands but not in ipsilateral matching. The interhemispheric deficit was not found in four control groups consisting of mentally disabled children of either the same mental or chronological age as the autistics and normal children matched on the same criteria. In the second experiment 20 autistic children were compared with 20 mentally handicapped children of the same mental and chronological ages. The task involved matching geometric shapes by active touch, which is more clearly lateralised than the passive touch task above. Again no lateralised deficit was disclosed and in replication of the first study the autistic group was impaired in contralateral matching in both left to right and right to left directions. The results are discussed in the light of contemporary theories of neurodevelopmental anomalies in autism, here implicating the corpus callosum.

D2 DOPAMINE RECEPTOR BINDING BEFORE AND AFTER TREATMENT OF MAJOR DEPRESSION MEASURED BY SINGLE PHOTON EMISSION COMPUTED TOMOGRAPHY

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Previous *in vivo* studies demonstrated changes in regional cerebral blood flow and glucose metabolism as well as alterations of the opioid system within the frontal cortex in depression. The present study continues the search for specific biochemical alterations in depression and investigates the potential impact of serotonin reuptake inhibition on the dopaminergic system. As yet, 11 patients (age 53.0 ± 10.8 ys., mean ± SD, 8 f, 3 m) with major depression were investigated before and immediately following a six week treatment with the selective serotonin reuptake inhibitor (SSRI) paroxetine (40 mg/d) or fluoxetine (up to 60 mg/d). Dopamine receptor binding was estimated using the specific D2/D3 receptor antagonist 123I-iodobenzamid (IBZM, 185 Mbq) and SPECT (double head camera PRISM 2000, Picker Ohio) with high resolution collimation. Specific IBZM binding was calculated as the region of interest to cerebellum ratio.

The total score in the Hamilton Depression Rating Scale (HAMD) decreased from mean ± S.D. 27.6 ± 4.9 before treatment to 13.5 ± 9.1 after treatment.

Within the basal ganglia, the average IBZM binding remained unchanged in the group as a whole. However, there was a significant correlation between the change of striatal IBZM binding and the improvement in psychopathology ($p < 0.05$), i.e., responders demonstrated a 20% increase and nonresponders a 10–20% decrease or no change of striatal and cingulate IBZM binding.

These preliminary data of an ongoing prospective study suggest an increase of dopamine D2/D3 receptors during successful therapy of major depression with an SSRI, which is consistent with findings of dopamine D2/D3 receptor sensitization in animal studies.

RETROGRADE AMNESIA IN PATIENTS WITH TEMPORAL LOBE, FRONTAL LOBE AND DIENCEPHALIC LESIONS

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The aim of this study was to investigate retrograde amnesia in patients who had either temporal lobe, diencephalic or frontal lobe lesions. The groups contained patients with herpes encephalitis, Korsakoff syndrome and recent frontal tractotomy. Patients were assessed for background variables such as severity of anterograde amnesia, current IQ and performance on executive/frontal lobe tasks.

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Retrograde memory was assessed on measures of famous news events, famous faces, and autobiographical memory for facts and incidents. Quantified PET and MRI data were available on the patients.

Korsakoff patients showed a retrograde memory loss extending back 20 or 30 years with a 'temporal gradient' ie relative sparing of early memories. However, a small group of patients with irradiation to the diencephalon causing anterograde amnesia showed spared performance on retrograde memory tasks, suggesting that the problem in the Korsakoff group results from concomitant frontal lobe or cortical atrophy. Patients with temporal lobe lesions also showed severe retrograde memory loss extending back many years, but their 'temporal gradient' was flatter than in the Korsakoff group, largely attributable to the herpes encephalitis patients. Patients with frontal lobe lesions also performed very badly in the recall of autobiographical incidents and news events, performing somewhat similarly to the Korsakoff patients on these two tests. Performance on a cued recall and a recognition task indicated a retrieval component to the deficit across the patient groups. It is concluded that pathology in the temporal cortex and frontal cortex produces a retrieval deficit from remote or retrograde memory.

PSYCHIATRIC ILLNESS ONE YEAR FOLLOWING HEAD INJURY

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Objective: Although neuropsychiatric morbidity following head injury is known to be common, no comprehensive population-based study of outcome following head injury exists. We have set out to study all the admissions following acute head injury within a year between 1st April, 1994 and 31st March, 1995 in the South Glamorgan Health District of the U.K. (general population 400,000) to estimate the incidence of morbidity one year following head injury.

Method: All patients have been assessed one year following head injury using a purpose-designed questionnaire for data collection, Mini mental state (MMS) and other supplementary neuropsychological tests to assess cognitive deficit. The Clinical Interview Schedule-Revised (CIS-R), Psychotic Screening Questionnaire, GHQ-28 and a Post Traumatic Stress Disorder Questionnaire were used to screen for psychiatric illness. Those who reached caseness according to the initial interview were further assessed by the Schedule for Clinical Assessment in Neuropsychiatry (SCAN) to get an ICD-10 and DSM-4 diagnosis of Psychiatric illness.

Results: Of the 122 patients approached so far, 28 are either untraceable or have refused to take part. Of the remaining 94 patients, 12 are deceased, 4 are in persistent vegetative state, 8 have severe disability according to Glasgow outcome scale and could not be assessed. Fifteen patients had a diagnosis of psychiatric illness (mainly affective disorder). The MMS score of 32 patients was below 24. Another 29 patients showed deficit in other neuropsychological tests (mainly in information processing) although they scored 24 or more in MMS.

AFFECTIVE DISORDER, TEMPORAL LOBE SYMPTOMATOLOGY AND MIGRAINE. A STUDY OF MIGRAINE CLINIC ATTENDERS

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There is evidence from migraine clinics, psychiatric populations and epidemiological surveys that migraine and affective disorders are linked. About a third of migraine clinic attenders report migraine aura symptoms which may represent temporal lobe dysfunction. The aims of this study were to assess temporal lobe symptomatology and its relationship to mood disturbance between migraine attacks.

To this end we carried out a comprehensive assessment of aura symptomatology, an evaluation of mood (past and present) using the Schedule of Schizophrenia and Affective Disorder-Lifetime version (SADS-L), the Hamilton Depression Rating Scale (HDRS) and the Hospital Anxiety and Depression Scale (HAD). 102 patients from the hospital's migraine clinic were assessed. The null hypothesis was that there would be no relationship between temporal lobe symptomatology and mood disorder in migraineurs.

Almost 60% of patients had a past psychiatric history as evaluated using the SADS-L. 40% had a present definite affective diagnosis on the HAD and 25% were depressed as measured by the HDRS. A third of patients had more than five temporal lobe symptoms (tls's) during their typical attack and the presence of such tls's, both in the aura phase of the migraine and the headache itself, was significantly correlated with scores on the ratings for affective disorders.

Conclusion: not only is affective disorder common in patients attending a migraine clinic but it is also linked to the presence of temporal lobe symptoms during the attack.

A NEUROPSYCHOLOGICAL AND SPECT STUDY OF EPILEPSY AND SCHIZOPHRENIA

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Patients with temporal lobe epilepsy (TLE) are at increased risk of developing schizophrenia. Early studies of this association made some oIJ the first contributions to the growing body of evidence implicating temporal lobe dysfunction in schizophrenia. However, the overwhelming majority of patients with TLE never develop schizophrenia and clearly a more discriminating account of the relationship between temporal lobe abnormalities and the pathophysiology of schizophrenia is required. We have studied 3 groups of patients: those with chronic interictal schizophrenia-like psychosis of epilepsy (SLPE), those with schizophrenia, and those with epilepsy, together with a normal control group (25 subjects in each group). All subjects were investigated with a battery of psychometric tests which included tests of memory and executive function. A proportion of the 3 patient groups also underwent functional neuroimaging with SPECT using a split-dose, verbal fluency activation technique. Patients with SLPE and those with primary schizophrenia were impaired on psychological tests of episodic memory when compared with the epileptic and normal control groups. The greatest memory deficits in the two psychotic groups were seen for verbal material. Both psychotic groups also showed impairment in tests of executive function. These results implicate dominant temporal lobe and prefrontal dysfunction in both SLPE and schizophrenia. By contrast, the SPECT study revealed anterior cingulate abnormalities in the schizophrenic group but dominant temporal lobe dysfunction in SLPE. The findings of these two sets of investigations might be reconciled if schizophrenia involves a functional disconnection between the prefrontal cortex and temporal lobes: while the neuropsychology results implicate dysfunction in both regions in the two psychotic groups, the SPECT findings suggest that this may have a different pathological basis in the two conditions.

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