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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