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R. Staff † H. Gemmell † A. Venneri ‡ M. Shanks * S. Pestell ‡ A. Murray †.

† Department of Nuclear Medicine, Aberdeen Royal Infirmary,
‡ Department of Psychology, University of Aberdeen,
* Old Age Psychiatric Unit, Royal Comhill Hospital, Aberdeen, UK.

rCBF SPET IMAGES OF AD PATIENTS WITH AN AUTOBIOGRAPHICAL CONTENT-SPECIFIC DELUSION

Delusional behavior and thinking are common symptoms in Alzheimer's disease (AD) and are thought to affect up to 50% of all patients at some point in their disease. In the past these symptoms have been considered to be a psychotic complication of some neurological dysfunction. More recently it has been suggested that, contrary to the earlier view, there is an organic deficit to which the onset of delusional belief in the context of AD can be attributed. The aim of this work is to discover if such a deficit can be identified in a specific type of delusion using rCBF SPET. **Methods:** 34 patients were investigated in this study. A group of 9 AD patients with a similar content-specific autobiographical delusion were compared to a group of 25 AD patients without any history of delusion. The autobiographical delusion was one in which the patient believed that a dead relative was still alive despite being presented with overwhelming evidence to the contrary. In each case the patient did not respond to treatment. Each patient underwent an extensive battery of neuropsychological testing and neuroimaging using 99mTc HMPAO. The reconstructed SPET data were compared using a Statistical Parametric Mapping (SPM) Technique. **Results:** The results showed that the deluded AD group had a significant area of hypoperfusion in the right frontal lobe when compared to the non deluded group. The area of hypoperfusion included parts of Brodman's areas 9 and 10. Region 9 has previously been identified as having a role in episodic memory retrieval. The neuropsychological results showed a significant reduction in executive control in the deluded group. **Conclusions:** This result suggests that autobiographical delusions such as this could be in part be a result of a failure in episodic memory retrieval and diminished executive function. If this is the case then the results also show that it is possible to identify an organic deficit to which this behavior can attributed using rCBF SPET.

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K. Nobuhara[1], C. Halldin[1], H. Hall[1], P. Karlsson[1], J. Hiltunen[2], D. McPherson[4], A. Savonen[2], K.A. Bergström[3], C.G. Swahn[1], S. Larsson[1], P.O. Schnell[1], L. Farde[1], G. Sedvall[1]. [1]Karolinska Institutet, Sweden; [2]MAP, Tikkakoski and [3]Kuopio University, Kuopio, Finland; [4]Oak Ridge National Laboratory, USA.

Z-IQNP: A POTENTIAL RADIOLIGAND FOR THE SPECT STUDY OF MUSCARINIC ACETYLCHOLINE RECEPTORS IN ALZHEIMER'S DISEASE

The M2 subtype of the muscarinic acetylcholine receptors (mAChR) is of interest for brain imaging in Alzheimer's disease (AD), where decreased densities have been reported. Z-IQNP is a muscarinic antagonist, which has high affinity to the M2. We labeled Z-IQNP with I-125 and I-123 and examined the distribution and selectivity of this radioligand using in vitro autoradiography and in vivo SPECT. Autoradiographic studies on human whole hemisphere cryosections showed a binding of [I-125]Z-IQNP in all brain regions and particularly high binding in regions known to have a high density of the M2 subtype. Competition studies with various reference ligands showed that [I-125]Z-IQNP specially labeled the mAChR. The addition of BIBN 99 (a compound with a high affinity for M2 subtype) specifically inhibited [I-125]Z-IQNP binding in the cerebellum (M2 receptor-rich region) with minor effects on the labeling of the other brain regions. SPECT examination demonstrated that about 5 % of the injected [I-123]Z-IQNP entered the monkey brain. There is high uptake of [I-123]Z-IQNP in all brain regions. The binding was markedly reduced in all brain regions after injection of dexetimide (non-selective muscarinic antagonist). The selective M1 antagonist biperiden had less effect in all brain regions. The marked reduction of [I-123]Z-IQNP binding by dexetimide in the cerebellum is consistent with a high density of M2-receptors in this region. The sigma compound DuP 734 had no effect on Z-IQNP binding, neither in vitro nor in vivo. It is concluded that iodine labeled Z-IQNP is a non-selective muscarinic antagonist which has potential for the exploration of M2-receptors in regions with a high density of this subtype.

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M. PAGANI, H. JACOBSSON D. SALMASO, C. RAMSTRÖM, C. JONSSON, P.O. SCHNELL, L. THURFJELL, R LUNDQVIST, A WÅGNER AND S.A. LARSSON. Section for Nuclear Medicine, Dept of Hospital Physics, Dept of Diagnostic Radiology, Dept of Neurology, Karolinska Hospital, Stockholm, Sweden; Center for Image Analyses, Uppsala University, Sweden. Inst of Exper Med, Inst of Psychology, CNR, Rome, Italy;

MAPPING PATHOLOGICAL rCBF IN ALZHEIMER DISEASE AND FRONTAL LOBE DEMENTIA USING A STANDARDISED BRAIN ATLAS

Alzheimer Disease (AD) and Frontal Lobe Dementia (FLD) are well-characterized pathological with regard to cortical rCBF SPECT. The purpose of this study was to investigate the additional diagnostic information that can be given by visual and statistical evaluation of SPECT comparing pathological standardised data sets to aged matched normal individuals.

17 AD pts, 8 FLD pts and 20 aged matched normal subjects (NOR) were examined. AD and FLD were diagnosed according to clinical presentation, EEG pattern and Mini Mental Score Examination. 99m-Tc-HMPAO SPET was performed with a three head gamma camera. and the A Computerised Brain Atlas allowed for spatial normalisation of all SPECT data sets. Hence it was possible to average across the subjects of the same group, compare data between groups and determine the recovered activity in all cerebral lobes, hippocampus, thalamus and Basal Ganglia.

ANOVA resulted in a significant overall difference in all considered regions with the exception of the talamus (Table). The p-values were:

REGIONS	AD-CRT	FLD-CRT	AD-FLD
Frontal Lobe	N.S.	0.000	0.000
Insular Lobe	0.000	0.000	N.S.
Occipital Lobe	0.042	0.000	0.000
Parietal Lobe	0.000	N.S.	0.010
Temporal Lobe	0.000	0.000	0.043
Hippocampus	0.000	0.001	N.S.
Caudatus	0.003	0.000	N.S
Putamen	N.S.	0.014	0.019
Thalamus	N.S.	0.013	N.S.

AD differed significantly from NOR in all lobes but the frontal one. FLD did the same with the exclusion of parietal lobe. FLD and AD differed in all lobes but the insular lobe. Subtracting the AD and FLD images from the NOR one resulted in highlighted caudatus and insular lobe for both diseases and temporo-parietal lobes and frontal lobes respectively.

We conclude that standardising SPECT in a common space and subtracting data from a control group results in a better visual interpretation of data. Standardising SPECT in a common space may be a useful quantitative tool in the diagnosis of early AD and FLD.

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R. Linke[1], I. Eisensehr[2], S. Noachtar[2], J. Schwarz[2], G Pöpperl[1], K. Hahn[1], K. Tatsch[1]

Departments of Nuclear Medicine[1] and Neurology[2], University of Munich, Munich, Germany

[I-123]IPT-BINDING TO THE PRESYNAPTIC DOPAMINE TRANSPORTER IN PATIENTS WITH IDIOPATHIC REM SLEEP BEHAVIOR DISORDER

Idiopathic rapid eye movement sleep behavior disorder (RBD) is a rare disease and characterized by complex behavior and lack of skeletal muscle atonia during REM sleep. The underlying cause is yet unknown; but a recent study showed that about 40% of pts with RBD developed Parkinson's disease (PD) or a multiple system atrophy. This fact suggests, that RBD could be an early sign for the development of parkinsonism. Therefore, we studied the striatal presynaptic dopamine transporter with the cocaine analog IPT and SPECT in pts with RBD.

In 5 polysomnographically confirmed RBD-pts, 8 age matched controls and 14 PD-pts with Hoehn & Yahr stage I SPECT-studies were performed using a triple head gamma camera and [I-123]IPT. For semiquantitative evaluation of specific IPT binding ratios (S) between striatum and a reference region were calculated.

	N	S (right)	S (left)
Controls	8	4.4±0.3	4.3±0.3
RBD	5	3.0±0.4*	3.0±0.4*
		S ipsi	S contra
PD (H&Y I)	14	3.1±0.5*	2.4±0.4*°

* p<0.01 compared to controls
° p<0.01 compared to RBD-pts

None of the RBD-pts showed any abnormalities in neurological examinations. However, image analyzes showed that the specific IPT-uptake was reduced in all pts with RBD compared to controls. The extent of reduced IPT binding was comparable to the one observed in the striatum reflecting the clinically yet unaffected side of early PD-pts. The contralateral striatal ratios reflecting the affected body side of PD-pts were significantly decreased compared to RBD-pts.

Our results demonstrate a striatal dopaminergic deficit as a possible pathophysiological mechanism for RBD. Furthermore, the reduced binding ratios suggest that pts with RBD could develop parkinsonism later on. In this case RBD would precede the onset of the typical parkinsonian motor symptoms. The latter will occur, if the loss of striatal dopaminergic nerve terminals continuously progresses. Therefore, IPT-SPECT may be recommended as a useful tool to document a nigrostriatal deficit in various disorders even in an asymptomatic preclinical stage of a progressing disease.

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