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Piotr Lass[1]
Jarosław Sławek[2,4]
Hubert Wichowicz[3]

[1]Department of Nuclear Medicine,
[2]Department of
Neurological-Psychiatric Nursing
[3]Department of Psychiatry
Medical University, Gdańsk, Poland
[4]Department of Neurology
St. Adalbert Hospital, Gdansk, Poland

NUCLEAR MEDICINE IN SELECTED PSYCHIATRIC DISORDERS

SUMMARY

The paper reviews the applications of nuclear medicine techniques in selected psychiatric disorders. Nuclear medicine methods are most useful in the differential diagnosis of dementia, assessing the sequelae of head trauma and neuropsychiatric symptoms of systemic lupus erythematosus. It plays lesser role in mood disorders and parkinsonian syndromes. In other psychiatric disorders it plays mostly investigative role, although the meaning of radionuclide imaging in e.g. post-traumatic stress disorder, eating disorders and sleep disorders is growing.

The point of gravity in radionuclide brain imaging is turning from cerebral blood flow and metabolism studies towards receptors' imaging, which frequently enables a new insight in the underlying pathological mechanisms of psychiatric disorders.

Keywords: **radionuclide imaging, psychiatric disorders**

INTRODUCTION

From the second half of the XXth century, advances in biological and psychopharmacological research seemed to promise a swift discovery of organic basis of psychiatric diseases. Today, we know that biological research in psychiatry is not a success story. This may be due to the complicated character of psychiatric disorders, where it is difficult to establish a unified model of the disease, therefore in many cases difficult unfit to categorical classification. This secondarily affects the role of nuclear medicine in psychiatry.

Diagnosis in psychiatry

Today's diagnosis in psychiatry largely relies on a classification of diseases following the criteria of the Diagnostic and Statistical Manual of Mental Diseases (DSM-IV-TR-2000) (1) or International Statistical Classification of Diseases and Health Related Problems – Tenth Revision (ICD-10). This classification enabled better defining of nosological concepts, better communication and understanding between psychiatrists. On the other hand, DSM-IV or ICD-10 does not entirely fit the psychiatric diagnosis, due to stiff application of choice principle.

There are two major problems in psychiatric diagnosis as a whole:

- diversity of symptoms: e. g. depressive episode patients may be either agitated with insomnia, or psychomotor retardated with hypersomnia as well;
- symptoms overlapping (sometimes defined as „co-morbidity"); e.g. 30 – 60% of depressive patients may have co-morbid anxiety disorder, whereas 40% diagnosed with anxiety have depression (2).

Contemporary psychiatric criteria are a bit more focused on symptomatic aspects of illnesses,

Corresponding author. • E-mail: plass@amg.gda.pl

sometimes omitting the causal side.

Diagnosis in psychiatry – role of neuroimaging

The same way as the overlapping in symptoms between different diseases in psychiatry, functional brain research frequently shows the same patterns of changes across diagnostic borders; on the other hand, many other tests, e.g. psychological tests present the same problem, as mentioned above. Nuclear medicine report will rather confirm, less frequently exclude psychiatrist's diagnosis. Ideally, psychiatric patients should be rescanned after the treatment, than changes in perfusion and/or metabolism discussed between psychiatrist and nuclear medicine specialist.

Applications of NM can be divided into:

I. Mostly diagnostic/practical/clinical, e. g. concerning of:

- differential diagnosis of dementia;
- psychiatric sequelae of head trauma, including the late whiplash syndrome;
- neuropsychiatric lupus erythematosus.

II. With mixed/or indeterminate status:

- psychiatric disorders in parkinsonian syndromes

III. Mostly investigational/experimental:

- others/ most of receptor studies

Dementia and other organic diseases

There are five potential major roles for neuroimaging with respect to dementia:

- as a cognitive neuroscience research tool;
- for prediction of which normal or slightly impaired individuals will develop dementia and over what time frame;
- for early diagnosis of Alzheimer's disease (AD) in demented individuals, (sensitivity) and separation of AD from other forms of dementia (specificity);
- for monitoring the disease progression;
- for monitoring a response to therapy.

Alzheimer's disease – SPECT studies

Regional cerebral blood flow (rCBF) Single Photon Emission Computed Tomography (SPECT) reaches detecting AD in up to 81%; although the clinical criteria may be more sensitive (up to 81%), SPECT seems superior in differentiating AD from the other types of dementia (91% vs. 70%) (3). Applying novel methodological approaches like means clustering or principal component analysis improved rCBF SPECT accuracies even to 98% and 90%, respectively (4).

In SPECT studies, the typical finding is decreased blood flow in the parietal or temporal lobe. In advanced cases with poor prognosis, decreased blood flow in the frontal lobes is also found. In mild cognitive impairment, rCBF SPECT studies may differentiate patients who will convert to AD from non-converters. As compared with non-converters, converters show reductions of rCBF in the bilateral temporo-parietal areas and the precunei. The logistic regression model reveals that reduced rCBF in the inferior parietal lobule, angular gyrus, and precunei has high predictive value and discriminative ability (5). The acetazolamide test in rCBF SPECT is helpful to differentiate AD from vascular dementia (6).

Following the therapy with acetyl cholinesterase inhibitors, (AChEI) rCBF increases or remains stable in AD patients with stabilized cognitive performance during therapy, whilst it decreases in nonresponders (7).

Alzheimer's disease – metabolic studies

$^{(18)}$F-FDG PET images of AD demonstrate focally decreased cerebral metabolism involving especially the posterior cingulate and neocortical association cortices, while largely sparing the basal ganglia, thalamus, cerebellum, and cortex mediating primary sensory and motor functions.

In a multicenter study comprising 10 PET centres (Network for Efficiency and Standardisation of Dementia Diagnosis, NEST-DD) that employed an automated voxel-based analysis of FDG PET images, the distinction between controls and AD patients was 93% sensitive and 93% specific, and even in very mild dementia (at MMSE 24 or higher) sensitivity was still 84% and 93% specificity (80). In very mild AD, both FDG-PET and voxel-based-morphometry (VBM –MRI) had high accuracy for diagnosis, but FDG-PET showed slightly higher accuracy than VBM-MRI. Combination of the two techniques will yield a higher diagnostic accuracy in very mild AD by making full use of functional and morphological images (9).

In addition to glucose metabolism, specific tracers for dopamine synthesis (^{18}F-F-DOPA) and for (^{11}C-MP4A) are of interest for differentiation among dementia subtypes. Cortical acetylcholine esterase activity (AChE) activity is significantly lower in patients with AD or with dementia with Lewy bodies than in age-matched normal controls. In dementia with Lewy bodies, there is also impairment of dopamine synthesis, similar to Parkinson's disease.

Alzheimer's disease – imaging amyloid deposits

Pathologically, AD is characterised by the excess accumulation of two types of protein aggregates: amyloid ß-peptide (Aß) plaques and neuro-fibrillatory tangles.

Amyloid imaging with SPECT is possible, with radioiodinated styrylpyridines and hydroxybenzthiazoles (10). However, today, practically only PET is the only currently available A-ß imaging technique with following agents:

- fluorophore derivative ^{18}F -FDDNP (11)
- benzothiazole derivative ^{11}C –PIB (12].
- stilbene derivative ^{11}C SB-13 (13)
- ^{18}F- fluoropegylated diphenylacetylenes (14).

Due to high costs, those modalities remain solely investigational.

Vascular (multi-infarct) dementia

Vascular dementia (VD) is caused by multiple cerebral infarcts. VD is usually associated with chronic hypertension, diabetes mellitus or/and other diseases associated with arteriosclerosis. Typical SPECT perfusion pattern is multiple focal defects, sometimes defined as a "patchy pattern". Similar changes can be seen in Creutzfeldt-Jakob disease and AIDS-related dementia (15).

Dementia with Lewy Bodies

Dementia with Lewy Bodies (DLB) is the type of dementia which morbidity probably can be underestimated. Core clinical features are: progressive cognitive impairment, visual hallucinations and parkinsonian symptoms and signs. In DLBD, there is a pronounced cholinergic deficit, therefore, cholinergic medication gives better results, than in AD. In early stages, DLBD brings difficulties in differentiation with AD.

On rCBF SPECT scanning, patients show hypoperfusion in parietal, occipital, also in frontal lobes, called „horseshoe" sign (16). Combined studies of MMSE and brain SPECT achieved a high discrimination between DLB and AD with a sensitivity of 81% and a specificity of 85%, suggesting that this is a useful and practical approach to differentiate DLB from AD (17). ^{18}F-FDG PET showed significant glucose metabolic reductions in the temporal, parietal, and frontal areas - including in the occipital lobe - as compared with those in the control group; in contrast, in AD patients, both the hippocampal volume and glucose metabolism were significantly decreased, whereas the occipital volume and metabolism were preserved (18).

At early stages of DLB when parkinsonian symptoms may not be present, the differential diagnosis in relation to AD may be difficult. New technique of imaging the dopaminergic transporting system (presynaptically) with the use of tracer called DaTSCAN may be useful. It has a practical significance due to hypersensitivity of DLB patients to classical neuroleptics which may deteriorate the outcome and are commonly used for treating psychiatric symptoms in AD (19).

Fronto-temporal dementia

Fronto-temporal dementia generally has a presenile onset. Behavioural problems dominate the clinical picture and cognitive functions are relatively intact.

rCBF SPECT studies show rCBF defects in frontal lobes and in the left temporoparietal-occipital areas, discriminates FTD and AD and with sensitivity 0.8, specificity 0.65 (20, 21).

In PET studies, the neurodegenerative process in early stages of FTD was found to be limited to the frontal lobes. During the progression of the disease, the pathological changes pass over the lobar borders and spread into the parietal and temporal cortices (22). In late stages, a significant hypometabolism is mostly found in extensive prefrontal areas, cingulate gyri, anterior temporal regions, and the left inferior parietal lobule. Frontal hypometabolism is usually more prominent in the left hemisphere than in the right one – in 79% of patients (23).

Dementia in Parkinson's disease/ Parkinsonian syndromes

Parkinson's disease is a neurodegenerative disorder characterised by progressive damage of the nigrostriatal dopaminergic neurons in the basal ganglia. It accounts for up to 85% of patients with parkinsonian symptoms.

Parkinsonian syndromes is a broader definition that encompasses other movement disorders with symptoms resembling PD and include progressive supranuclear palsy (PSP), multiple system atrophy (MSA) and corticobasal degeneration.

Confusingly, the symptoms of PD or PS can also be met in patients with essential tremor or secondary to some medications. Therefore, up to 25% of patients initially diagnosed as PD later have this diagnosis changed. 20 – 30% of patients with PD have dementia.

PD patients with dementia show left temporo-parietal hypoperfusion as compared to a

group of patients without dementia, which resembles perfusion deficits described in Alzheimer's disease. The hypoperfusion of the left temporal lobe with increase of rCBF within the left thalamus might be clinically useful in discrimination of Parkinson's disease patients with dementia against those without cognitive impairment (24).

Another aspect of RN diagnosis aims to differentiate PD and Essential Tremor by ^{123}I-ß-CIT or ^{18}F-DOPA uptake and differentiating PD and PS-plus syndromes with imaging of D_2 receptors with ^{123}I-IBZM (25 - 27).

Head injuries

After the head trauma, a number of neuropsychiatric symptoms and signs may follow, with considerable difficulties both in the treatment and in medical certification of posttraumatic neuropsychiatric disorders for the purpose of criminal and civil law proceedings.

Functional brain imaging data collected in a resting state can provide objective evidence of brain injury in mild blunt head trauma patients with persistent post-concussive somatic and/or cognitive symptoms, particularly in mild and moderate traumatic brain injury by: better sensitivity than MR/CT findings and important exclusion role (28, 29). RCBF SPECT scanning is particularly useful in imaging blood flow disturbances in basal ganglia following the head trauma, also in mild brain injury (28, 29). The results of rCBF scanning are important in rehabilitation counselling, medico-legal arguing and the evaluation of the ability of the patient to work. Its limitations are overlapping of diffuse axonal injury (DAI) invisible in PET/SPECT studies and in older patients overlapping of atherosclerotic vascular lesions.

Late whiplash syndrome

The prevalence of whiplash injury is 4 cases/1000 inhabitants per year, usually following rear-end car collision. This sometimes results in whiplash-associated disorder (WAD), a controversial condition with largely unknown pathogenetic mechanisms. Only small proportion (up to 5%) of whiplash injury patients develop late whiplash syndrome with cerebral symptoms: headache, dizziness, vertigo, disturbances of concentration, attention and memory, as well as peripheral symptoms: neck pain, neck rigidity, temporomandibular dysfunction.

As frequently secondary to traffic, sport and work accidents, late whiplash syndrome is a major medico-legal problem, attracting not only physicians, but also lawyers and insurance companies.

In 1997 - 2003 Otte et al. described an increased parieto/occipital hypoperfusion and hypothesized that whiplash injury may precede Alzheimer's disease following the prolonged hypoper-fusion in cortico-basal region (30 – 32).

This hypothesis has been heavily criticized by many opponents, e.g. Sundström and colleagues showed rCBF changes in patients with chronic back pain of non-traumatic origin, but not in ones following whiplash injury (33), whereas some data support it (34).

Systemic Lupus Erythematosus

The peak incidence of Systemic Lupus Erythematosus (SLE) occurs between 15 and 40 years of age. CNS manifestations have been described in 20 – 70% of cases. This dispersion alone illustrates the basic problem of cerebral involvement in SLE, i.e. differentiating organic brain lesion from functional disturbances, influence of medication, particularly steroids, feeling of social rejection following skin changes etc. Neuropsychiatric manifestations in SLE comprise: migraine, epilepsy and stroke; cognitive dysfunction, mood disorder, anxiety disorder, acute confusion state, psychoses.

Radionuclide studies help to establish organic brain lesion and to differ from functional/iatrogenic changes. The clinician expects a zero/one answer - is there a central nervous system (CNS) involvement or not?

MRI scanning with white matter hyperintensities (WMH) as a marker of vascular involvement is considered to be the „golden standard" of neuro-imaging in NP SLE. However, this appears in 30% of SLE patients without CNS involvement and is absent in early/intermediate stages of disease.

rCBF SPECT scanning is the most sensitive, although may not be entirely specific neuroradiologic assessment in NP SLE and other loose connective tissue diseases (35). The higher sensitivity of SPECT, as compared with MRI can be explained by the vasculopathy with microcirculation changes as a as a major pathological factor in SLE. ^{18}F-DG PET scanning shows as a typical finding a hypometabolism of „posterior' type (36).

Schizophrenia

Schizophrenia is a major source of morbidity worldwide, with a prevalence of about 1% and significant disabilitation. Radionuclide neuroimaging is focused on receptor research, with little results, mostly indicating the disturbances of

dopamine in schizophrenia. As new ligands are being developed, further insights will be gained in the underlying pathology in schizophrenia. New techniques combining functional imaging with genetic studies are likely to not only depict the state of receptor population, but also concentrate on long-term dynamic changes induced by the illness and its treatment.

Schizophrenia – the dopamine overactivity hypothesis

This hypothesis says that increased activity in the dopamine neurotransmitter system is responsible for the positive symptoms of schizophrenia. An increased density of D_2 receptors was found in post-mortem studies (37).

Early functional studies revealed a marked increase in D_2 receptors binding within the striatum. Further studies revealed two distinct families of dopamine receptors: D_1-like (D_1 and D_5) and D_2- like (D_2, D_2, $D_{4)}$. It is possible that antipsychotic drugs selectivity could be mainly due to preferential binding of dopamine D_2 – like receptors.

For measuring dopamine synthesis and transport in presynaptic function for the former, the most commonly used tracers are 6-[^{18}F]-DOPA and 6-[^{18}F]-MT, whereas for the latter several ^{11}C/^{18}F-labelled tropane analogues are being clinically used. Postsynaptically, dopamine exerts actions through several subtypes of the dopamine receptor (38).

In schizophrenia, dopamine studies of receptor competition of ^{123}I-IBZM – a dopamine D_2 ligand with dopamine showed that not only resting dopamine level matters, but also suggested that schizophrenia is a disorder of dopamine dysregulation indifferent parts of the brain. In treatment's follow up, increasing D_2 receptor occupancy on the 2nd SPECT was a predictive factor for the relapse. Therefore, D_2 receptor occupancy and its changes during quetiapine therapy is supposed to be related to the prognosis of the treatment efficacy (39).

Schizophrenia – 5HTreceptors

Serotoninergic agents, like LSD, lead to hallucinations. Initial SPECT and PET studies showed very high occupancy of $5HT_{2a}$ receptors for many atypical antipsychotic drugs including clozapine, olanzapine and risperidone, but not typical ones like haloperidol and chlorpromazine. The involvement of $5HT_{2a}$ receptors was undermined by the fact that its pure antagonists did not have any antipsychotic effect. More recent papers on $5HT_{1a}$ have showed in-creased binding in schizophrenics in the left medio-temporal cortex, but its meaning its unclear (40). The current view is that the change in this receptor population is unlikely to be causal in schizophrenia.

Schizophrenia – Glutamate and NMDA receptors

N-Methyl-D-aspartate (NMDA) receptors are calcium-permeable glutamate receptors that play putative roles in learning, memory, and excitotoxicity. There has been much interest over the years in the NMDA receptors in schizophrenia, as certain blockers of NMDAR such as phencyclidine and ketamine lead to transient drug-induced symptoms very similar to those reported by schizophrenic patients. First *in vivo* evidence of an NMDA receptor deficit in medication-free schizophrenic patients were published in 2006 (41).

Catatonia

Catatonia is a form of schizophrenic psychosis with prominent psychomotor disturbances. The symptoms of catatonia include e.g.: stupor, excitement, posturing, waxy flexibility.

In catatonic patients, SPECT and PET studies showed dysfunctions of prefrontal and parietal cortices that possibly account for its motor, affective and behavioural disorders. Dysfunctions in prefrontal cortex could account for some affective disturbances found in catatonia, whereas dysfunction in parietal lobes might participate in the disturbances of executive task planning.

In akinetic catatonia, SPECT and PET studies showed dysfunctions of medial, prefrontal cortex, lateral parietal cortex and precuneus, which is known to be involved in the conscious awareness.

Those studies support the argument for the existence of a neural network of conscious awareness that may be disturbed in patients with stuporous catatonia.

Mood disorders

Mood disorders are amongst the most prevalent in modern society and have an important socio-economical impact. The depressive episode may have various courses and clinical presentations. The severity of the episode can vary from mild to severe and the specific form of disorders, e.g. recurrent, with seasonal pattern or with rapid cycling pattern can be singled out. Other specifiers describe atypicality, postpartum onset, the presence of psychotic features or chronicity. The presence of manic/hypomanic or mixed episodes further specifies whether the disorder is unipolar or bipolar.

Brain perfusion and metabolism studies in depressive disorders

Early studies by Baxter et al. described a global decreased supratentorial brain activity in bipolar disorder, with metabolic rate increasing while going from depression to euthymic or manic state. Further studies showed a significant left-right prefrontal asymmetry, resolving after treatment (42). Hypoperfusion in the recurrent depressive disorders was considerably greater (nearly significant) in comparison with the first depressive episodes (43).

Further studies refined the findings of prefrontal hypoperfusion/metabolic hypoactivity, as most important, delineating those findings to areas of prefrontal cortex and its functionally separate areas: dorsolateral (DLPFC), the orbito-frontal and the anterior cingulate, left amygdala, parahippocampal gyrus (44, 45), which with subcortical circuits have separate behavioural functions: DLPFC mediating executive functions, orbito-frontal object-affect associations, anterior cingulate mediating motivation.

Mood disorders – treatment effects

Nearly all available antidepressants have been studied. Normalization of the frontal hypoperfusion/ hypometabolism and/or asymmetry appears to be the most replicative finding (46, 47).

In many studies, a pattern of cortical flow/metabolism increases and limbic/paralimbic decreases was seen in association with chronic treatment, which suggest a primary subcortical and limbic effects of pharmacological treatment with neocortical effects as secondary (47).

Electroconvulsive therapy (ECT), a powerful tool where pharmacological intervention fails, initially in a short-term reduces the CBF and metabolism, suggesting the reduction of neural activity, in a long-term normalizing perfusion/metabolism in depressed patients (46, 49). ECT flow/metabolism results may be however atypical, as this therapy is mostly applied in treatment-resistance patients.

Repetitive transcranial magnetic stimulation (rTMS) of the prefrontal cortex may be useful in refractory cases. High-frequency rTMS of the left prefrontal cortex and possible of the opposite physiological effects low frequency rTMS of the right prefrontal cortex produce a significant decrease in Hamilton depressing scale scores; it increases metabolic rate in prefrontal cortex, amygdala, basal ganglia, hippocampus and cerebellum; a post-treatment decrease in cingulate (50).

Vagal nerve intermittent electrical stimulation which is used mostly in epilepsy treatment also reduces depressive symptoms. rCBF changes share features with changes of rCBF previously associated with the administration of selective serotonin reuptake inhibitors. Similarities to other brain-stimulation strategies in antidepressant treatment were less pronounced (51).

Radioligand receptor studies in depression

Those studies are limited mainly by the availability of suitable ligands. Currently available ligands allow the investigation of 5-HT_{1A} -[^{11}C-WAY-100635], 5-HT_{2A} - [^{123}I -ketanserin, ^{18}F-altanserin, and ^{11}C-methylpiperone] and D_2 receptors - [^{123}I-IBZM]. The results are rather discordant (52, 53). Most, albeit not all studies find an increased 5-HT_{2A} binding after treatment, the same effect in D_2 binding, decrease in HT_{1A} binding.

The current status of ligand studies in depression does not yet allow for the tailoring of pharmacotherapeutic status in an individual patient. They allow at least some agreement on the effect of anti-depressant agents on the HT and dopamine receptors and on serotonin transporter.

There are two conclusions about radionuclide studies in depression: good news: brain imaging is a powerful tool to explore various aspects of brain function in depression; bad news: the clinical psychiatrist should not ask the nuclear medicine psychiatrist to confirm the diagnosis of depression in his patients – hypofrontality is quite unspecific finding. Performing functional imaging, however, should stimulate both specialists to look at functional abnormalities on the scan and to link them to the patients behavioural abnormalities and/or symptoms.

Future research should be devoted to finding the basics of treatment regimens and finding predictors of treatment response.

Neurotic and other stress-related disorders

Obsessive-compulsive disorder

Obsessive-compulsive disorder (OCD) is characterised by repetitive thoughts, impulses, images or behaviours. The obsessions or compulsions interfere significantly with the person's normal life, as well as occupational and social functioning.

Neuroimaging studies have proved the evidence of the dysfunction. The fronto-subcortical

circuit might be involved in the pathophysiology of OCD. In SPECT and PET studies, hyperactivities of those circuits including orbito-frontal cortex, anterior cingulate and/or basal ganglia have been a consistent finding (54).

Serotonin transporter studies indicate the reduced serotonergic input into the fronto-subcortical circuits in OCD, thereby diminishing the inhibitory regulation of serotonin on these circuits (55).

Fluvoxamine treatment significantly improves clinical symptoms and increases D_2 ligand [^{11}C]-raclopride binding potential (BP) in the basal ganglia of OCD patients. (Chronic treatment with fluvoxamine induces a slight but significant increase in striatal [^{11}C]-raclopride binding of previously drug-naïve OCD patients (56).

Although present imaging techniques have a limitation as a way of brain dysfunction in OCD, nuclear neuroimaging may be used as an objective tool and predict response to the treatment.

Social phobia

Social phobia (SP) is characterised by fears of social interaction and performance situations. A person fears that he/she will act in a away that will be humiliating or embarrassing [DSM-IV]. In a course of disease, secondary depression or subsequent alcohol abuse and dependence may develop.

The presence of manic/hypomanic or mixed episodes further specifies whether the disorder is unipolar or bipolar.

Regional CBF SPECT scanning data are similar to ones of other anxiety disorders and are consistent with previous work demonstrating the importance of limbic circuits in this spectrum of disorders. These play a crucial role in cognitive-affective processing, are innervated by serotonergic neurons, and changes in their activity during serotonergic pharma-cotherapy seem crucial (57).

Serotonin receptor studies showed the lower 5-HT1A binding in the amygdala and mesiofrontal areas of SAD patients was consistent with a previous PET study in healthy volunteers showing an inverse correlation between 5-HT_{1A} BP and state anxiety, and the other human PET studies in patients with panic disorder showing reduced 5-HT_{1A} binding, thus corroborating the potential validity of 5-HT_{1A} receptors as targets in the treatment of human anxiety disorders (58).

Dopamin receptor studies showed that striatal dopamine reuptake site densities were markedly lower in the patients with social phobia than in the age- and gender-matched comparison subjects. Those results indicate that social phobia may be associated with a dysfunction of the striatal dopaminergic system (59).

Hysteria (conversion disorder)

Hysteria, or conversion disorder is defined as a psychiatric illness which symptoms or deficits, affecting voluntary motor or sensory function, cannot be explained by a neurological or general medical condition.

Functional neuroimaging has revealed selective decreases in the activity of frontal and subcortical circuits involved in motor control during hysterical paralysis, decreases in somatosensory cortices during hysterical anaesthesia, or decreases in visual cortex during hysterical blindness (60). An inhibition of normal neural networks seems to be a common marker also to the tunnel vision or hysterical deafness.

Post-traumatic stress disorder (PTSD)

PTSD is a complex psychobiological disorder which develops in the aftermath of severe and/or life-threatening trauma (serious accident, disasters, burns, sexual abuse or rape). Functional imaging in PTSD is in its infancy, but number of evidence is growing and through these insights, PTSD has changed from "traumatic neurosis" to a biologically based psychological disorder. Most probably, this anxiety disorder is associated with changes in neural circuitry involving frontal and limbic systems. Altered metabolism in these brain structures after a traumatic event is correlated to PTSD. Developments in the field of neuroimaging have allowed researchers to look at the structural and functional properties of the brain in PTSD (61).

Neuroimaging studies are usually performed as emotional activation studies, utilizing e.g. combat-related sounds, images of great fire etc. (62, 63). Therefore, those studies sometimes are difficult to perform for ethical reasons.

Emotional activation studies demonstrate flow/metabolic activation most frequently in the precentral gyrus, posterior cingulate, amygdala and cerebellum, while deactivation is seen in the middle temporal gyrus, inferior and middle frontal and parietal region.

Preliminary data show the circuits in the brain connecting regions of the medial prefrontal, medial temporal, frontal and parietal cortices. Future of those studies will probably help in understanding what makes some people more vulnerable to PTSD, and how treatment influences and predicts the changes we see in brain imaging studies.

Eating disorders

Eating is one of the basic human behaviours. There are two major pathological entities: anorexia nervosa and bulimia nervosa. On the other hand, a separate major issue is investigating the CNS in binge eating and obesity, a major epidemic problem. Many obese patients probably suffer from binge eating disorder, but certainly, this is not always objectified and it is even overlooked.

Functional brain imaging in eating disorders have been available since more than one decade. Generally, there is an enormous differentiation between studies, which may be secondary to methodological variations. Therefore, they are of limited, if not nil clinical value, at least at present, although may give some guidance to future pharmacological studies.

Anorexia nervosa

Anorexia nervosa (AN) is devastating and life-threatening disease, among females its prevalence is approximately 0.27 – 1% (64). There are two subtypes of AN: in restrictive subtype dieting, fasting or excessive exercise is used to achieve weight loss, in binge eating/purging type a combination of binge–eating, purging by means of self-induced vomiting, misuse of laxatives, diuretics and enemas is met.

In AN, patients show either the hyperperfusion in frontal, fronto-temporal and parietal cortex or the hypoperfusion in anterior cingulate. Activation tests showed rCBF increase in the left inferior frontal lobes. Following treatment, changes of rCBF in right DLPFC, ACC, MPFC, PCC and precuneus were related to the AN recovery process and might be associated with improvement of interoceptive awareness (65).

Receptor [^{18}F-altanserin] HT_{2A} studies showed reduced tracer binding with altered neurotransmission during recovery. The question remains, whether it is a cause or consequence of AN.

There are two basic technical problems to assess AN patients: rCBF changes are either small and space-restricted or diffuse and unspecific. This requires either good quality statistical parametric mapping (SPM) or activation paradigms utilising food stimuli in well-standardized environment.

Bulimia nervosa

Bulimia (Greek – *bull- hunger*) is generally considered a condition in which the subject engages in recurrent binge eating followed by an intentional purging. This purging is done in order to compensate for the excessive intake of the food and to prevent weight gain. Purging typically takes the form of vomiting; inappropriate use of laxatives and diuretics, enemas and other medication and/or; and excessive physical exercise. Historically, initially it was considered a variant of anorexia nervosa. Sometimes AN can be preceded by bulimia and vice versa.

SPECT studies showed decreased right inferior frontal and left temporal lobes. It may suggest the hypoactivity of the putative feeding suppression mechanism in frontal lobe, resulting in hyperphagia. Alterations in rCBF during the ill state of BN may be a state-related phenomenon that remits with recovery (66).

PET studies data showed - contrary to depressive patients – maintained basal ganglia metabolism. Some PET studies showed lower rCMRGlu pattern similar to that of obsessive-compulsive disorder symptoms.

Receptor studies suggest decreased serotonergic transmission in bulimia nervosa. Studies using PET with serotonin specific radioligands implicate alterations of 5-HT1A and 5-HT2A receptors and the 5-HT transporter. Alterations of these circuits may affect mood and impulse control as well as the motivating and hedonic aspects of feeding behaviour (67).

Obesity

Obesity is an epidemics of our age, at least in some countries, therefore functional brain studies in obesity seem very promising. rCBF SPECT studies in the obese women, showed higher CBF in the right parietal and temporal cortices during the food exposure than in the control condition. In addition, in the obese women, the activation of the right parietal cortex was associated with an enhanced feeling of hunger when looking at food. No such changes or associations were seen in the normal-weight women (68).

In PET studies, the higher metabolic activity was showed in the area of parietal cortex where the somatosensory maps of the mouth, lips and tongue are located, suggesting the role of reward component in the aetiology of obesity. Other study showed an involvement of enhanced sensitivity of frontal regions following the food stimuli, suggesting their specific response.

Receptor studies with ^{11}C-raclopride studies showed the negative correlation of BMI and D_2 receptor availability (69).

Other disorders and indications

Forensic psychiatry

PET and SPECT studies are increasingly used as a tool in forensic medicine, particularly in the USA, since start-90'ies. The mental disorders which attract most legal attention are those where a connection between an injurious stimuli, e.g. post-traumatic brain injury or post-traumatic stress disorder exists. The brain changes are not clear in all patients, therefore functional neuroimaging cannot be safely used to make a diagnosis of psychiatric disorder or to exclude/confirm psychiatric disorder. For that reason, it can be used as an auxiliary tool together with CT, MRI and psychometric testing, although some specific reports exist (70, 71).

To be accepted as the evidence, the Supreme Court of the USA ruled that the following elements should be taken into account assessing the diagnostic method (72):

- Can particular method be tested?
- Are the known/potential pitfalls of the method established?
- Is there a sufficient scientific evidence on the method?
- Is there a wide acceptance for a method in the scientific world?

In most cases, PET and SPECT fulfils above mentioned demands at least incompletely due to scarce number of controlled studies, low sensitivity and specificity and insufficient standardization, therefore found utilization in few situations: traumatic brain injury following car accidents or criminal attacks, in confirming/excluding brain injury in victims with unspecific complaints such as persistent headaches, amnesia, emotional disorders etc., organic basic brain dysfunction e.g. the frontal syndrome in suspects or post-traumatic stress disorder (PTSD) in war veterans.

Brain function during hypnosis

Hypnosis may be defined as a state of focus attention, concentration and inner absorption with a relative suspension of peripheral awareness.

PET studies demonstrated a vast activation in occipital parietal, precentral prefrontal and cingulate cortex. This suggests that hypnotic state is not relied on simple evocation of episodic memory, but a reminiscent of mental imagery (73).

PET studies gave a neuroimaging support for nociceptive effect of hypnosis; the hypnotic state significantly enhances the functional modulation between midcingulate cortex and a large neural network involved in sensory, affective, cognitive and behavioural aspects of nociception (74). In fibromyalgic patients under hypnosis, the cerebral blood-flow was bilaterally increased in the orbitofrontal and subcallosial cingulate cortices, the right thalamus, and the left inferior parietal cortex, and was decreased bilaterally in the cingulate cortex. The observed blood-flow pattern supports notions of a multifactorial nature of hypnotic analgesia, with interplay between cortical and subcortical brain dynamics (75).

Impulsive aggression and suicidal behaviour

Impulsive aggressive behaviour including deliberate self-harm and impulsive aggression towards others as well as suicidal behaviour is a major problem in health care. It is believed that its anatomical correlates are prefrontal and temporal limbic regions and the serotoninergic dysfunction play the crucial role.

In impulsitivity, PET studies indicate that the impulsivity and chronic stress are associated with amphetamine-induced striatal dopamine release. PET studies with high specific activity [^{11}C] raclopride was associated with blunted right ventral striatal DA release. Dopamine release was greater in low vs. high impulsivity subjects under conditions of low or moderate stress (76).

In suicidal behaviour, direct in vivo functional imaging with PET or SPECT demonstrated a reduction in 5-HT_{2A} binding index in suicide attempts in anxious and depressed suicide attempters and an increase in 5-HT_{2A} binding in impulsive suicide attempters (77). The importance of those studies is two-fold: first, this may increase insight into the pathophysiology of impulsive aggression; secondly, this may enable to develop new pharmacologic approaches.

Sleep disorders

One-third of our lives is occupied by sleep. The utilization of nuclear medicine in sleep disorders is still in infancy; this may be however a promising area for future development.

In healthy sleep, there is an increase in function in limbic and anterior paralimbic cortex in REM sleep and decreases in function in higher cortical regions in known thalamocortical networks during NMREM sleep. Serotonin $5HT_{1A}$ receptor PET studies showed a significant increase in their ligand - [^{18}F]MPPF binding in sleep compared to wakefulness in the whole brain and all regions of interest examined: temporal cortex, mesial temporal region and cingulate cortex (78).

There is a collection of papers on diverse sleep disorders such as: sleep deprivation, insomnia, dyssomnia, narcolepsy, sleep apnoea, sleepwalking.

Perfusion abnormalities in patients with REM behavior disorder are located in the brainstem, striatum, and cortex. These abnormalities are consistent with the anatomic metabolic profile of Parkinson disease (79).

In primary insomnia, a pattern of hypoperfusion in the frontal medial, occipital, and parietal cortices was found with particular deactivation in the basal ganglia (80).

In narcolepsy, SPM analysis of brain SPECT showed hypoperfusion of the bilateral anterior hypothalami, caudate nuclei, and pulvinar nuclei of thalami, parts of the dorsolateral/ventromedial prefrontal cortices, parahippocampal gyri, and cingulate gyri in narcoleptics, also reduced cerebral perfusion in subcortical structures and cortical areas in narcoleptics. The distribution of abnormal cerebral perfusion is concordant with the pathway of the cerebral hypocretin system and may explain the characteristic features of narcolepsy, i.e., cataplexy, emotional lability, and attention deficit (81).

In sleepwalking, activation of thalamocingulate pathways and persisting deactivation of other thalamocortical arousal systems were found (82).

CONCLUSIONS

As showed above, practical applications of nuclear medicine are few due to low specificity and low spatial resolution, although in the aspect of functional imaging still superior to CT/MRI, even in their functional modalities.

On the other hand, its investigational potential is still growing, as there is no insight into imaging technique, which could replace nuclear medicine metabolic and receptor studies. Secondly, that the scope of functional imaging is psychiatric diseases is spreading from its traditional applications like dementia or depression towards many poorly investigated fields like e.g. hypnosis, suicidal behaviour or sleep disorders.

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PRIMENA NUKLEARNE MEDICINE KOD ODABRANIH PSIHIJATRIJSKIH POREMEĆAJA

Piotr Lass[1], Jaroslaw Slawek[2,4], Hubert Wichowicz[3]

[1]Odeljenje za nuklearnu medicinu
[2]Odeljenje za neurološko-psihijatrijsku negu
[3]Odeljenje za psihijatriju, Medicinski fakultet, Gdansk, Poljska
[4]Odeljenje za neurologiju, Bolnica St. Adalbert, Gdansk, Poljska

SAŽETAK

U radu je predstavljena primena tehnika nuklearne medicine kod odabranih psihijatrijskih poremećaja. Metode nuklearne medicine su najkorisnije u uspostavljanju diferencijalne dijagnoze demencije, u proceni sekvela kod povreda glave i neuropsihijatrijskih simptoma sistemskog lupusa. Manja uloga joj se dodeljuje kod poremećaja raspoloženja parkinsonovih sindroma. Kod ostalih psihijatrijskih poremećaja uloga nuklearne medicine je uglavnom istraživačka, premda je značaj radionukleidnog snimanja u slučajevima poput posttraumatskog stresnog poremećaja, poremećaja ishrane i spavanja u stalnom porastu.

Trend radionukleidnog snimanja mozga se od studija o cerebralnom protoku krvi i metabolizmu kreće ka snimanju receptora, što često daje novi uvid u postojeće patološke mehanizme psihijatrijskih poremećaja.

Ključne reči: **radionukleidno snimanje, psihijatrijski poremećaji**