REVIEW PAPER/*ARTYKUŁ POGLĄDOWY*

# Psychiatry and neurology: from dualism to integration

## *Psychiatria i neurologia – od dualizmu do integracji*

Jerzy A. Sobański[1], Dominika Dudek[2,3,4]

[1]Department of Psychotherapy, Jagiellonian University *Collegium Medicum*, Krakow, Poland
[2]Adult Psychiatry Department, University Hospital, Krakow, Poland
[3]Department of Affective Disorders, Department of Psychiatry, Jagiellonian University *Collegium Medicum*, Krakow, Poland
[4]Institute of Pharmacology, Polish Academy of Sciences, Krakow, Poland

Neurologia i Neurochirurgia Polska 2013; 47, 6: 577-583
DOI: 10.5114/ninp.2013.39069

## Abstract

The two objectives of the following paper are: to make few remarks on the topic absorbing neurologists, psychiatrists, and neuropsychiatrists – integration and division of their specialties; and to describe the situation in Poland, reflected in the latest literature. The authors describe the former and present processes of approaches and divisions in psychiatry and neurology. They indicate dissemination of mutual methods of structural and action brain neuroimaging, neurophysiology, neurogenetics, and advanced neurophysiology diagnostics. As it seems, even the effectiveness of psychotherapy, has recently been associated with changes in brain in functional and even structural markers. The authors indicate the value of the strive to join the still divided specialties, reflected worldwide in attempts of common education and clinical cooperation of physicians. It can be expected that subsequent years will bring further triumphs of neuropsychiatry – a field that combines psychiatry and neurology.

**Key words:** neuropsychiatry, neurology, psychiatry, dualism, integration.

## Streszczenie

Niniejsza praca ma dwa cele: pierwszy to poczynienie kilku uwag na temat od lat zajmujący neurologów, psychiatrów i neuropsychiatrów – integracji lub podziału ich specjalności; drugi to przybliżony opis stanu rzeczy w Polsce, odzwierciedlony przez najnowsze piśmiennictwo. Autorzy omawiają dawne i aktualne procesy przybliżania się i rozdzielania psychiatrii i neurologii oraz dokonują krótkiego przeglądu ich tła historycznego. Wskazują na upowszechnienie wspólnych metod strukturalnego i czynnościowego neuroobrazowania mózgu, neurofizjologii i neurogenetyki oraz zaawansowanej diagnostyki neuropsychologicznej. Jak się wydaje, nawet skuteczność procedury tradycyjnie umieszczanej tak daleko od neurologii, jak psychoterapia, już od kilkunastu lat próbuje się powiązać ze zmianami na poziomie mózgu, w zakresie czynnościowych, a nawet strukturalnych neurobiologicznych parametrów zaburzeń. Autorzy podkreślają wartość dążenia do połączenia obecnie rozdzielonych psychiatrii i neurologii, odzwierciedloną przez obserwowane na świecie próby wspólnego kształcenia i współdziałania klinicznego lekarzy obu specjalności, oraz omawiają konsekwencje podziałów i pominięć. Można się więc spodziewać, że kolejne lata przyniosą dalsze tryumfy neuropsychiatrii – dziedziny łączącej psychiatrię i neurologię, która stanie się przyszłością psychiatrii.

**Słowa kluczowe:** neuropsychiatria, neurologia, psychiatria, dualizm, integracja.

Correspondence address: dr med. Jerzy A. Sobański, Pracownia Diagnostyki Zaburzeń Nerwicowych i Behawioralnych, Katedra Psychoterapii UJ CM, ul. Lenartowicza 14, 31-138 Kraków, e-mail: molocko@poczta.fm
Received: 7.05.2013; accepted: 24.10.2013

*What Nissl and Alzheimer could find under their microscopes they called 'neurology'.*
*What they could not find was 'psychiatry'.*

*Edward Shorter (after [1])*

Even though the academic institutes have connected neurology and psychiatry (e.g. Department of Psychiatry and Neurology, initiated by professor Jan Piltz in Kraków in 1905 [2]), the basic interests of neurologists and psychiatrists were different. Neurology deals with treating organic diseases caused by identified reasons – damage or brain structure dysfunction. Such an assumption directed neurologists' interests into somatically-rooted problems such as: stroke, multiple sclerosis or Parkinson disease. Psychiatrists – in the contrary – turned to such disorders as psychotic, mood, anxiety and personality disorders etc., which are not likely to manifest in neurological examination, with slight or none movement and sensory symptoms.

One exception was the interest of neurologists of the 19th and 20th century in 'nervous diseases' (neurosis). It stemmed from the common fear of psychiatric stigma [3] and of enormous psychiatric institutions, in which – often in inhuman conditions – mentally ill patients spent their whole lives. Neurologists of the times (even Zygmunt Freud, who was a coworker of Meynert) in such cases used sanatorium treatment, massages, even relaxation etc.

Psychiatry – in contrary to neurology – was limited to dealing with emotional and behavioral disorders, usually with unknown structural background.

Shortly after, the process of crossing borders, compared to dismantling of 'the wall', between the temporarily isolated specialties [4,5] was started. One of the leading Polish neuropsychiatrists – precursor of that process – was Leon Daraszkiewicz [6] (a student of Kraepelin), who in 1926 described neurological symptoms in patients with schizophrenia and formed a hypothesis about the correlation between progressive paralysis and vaccination against smallpox. The dichotomy neurology versus psychiatry did not survive the time trail. Nowadays, it seems to be completely unjustified – hopefully it will be proven in this paper.

One of the first and most exciting proves that organic brain damage causes changes in emotionality and personality is Phineas Gage's case. In 1848, he was hit below his left cheekbone with a rod which was one meter long, over 3 centimeters in diameter and weighed 6 kilograms. The hit damaged a large part of patient's frontal lobes. Phineas survived the injury, a few minutes after the accident he regained consciousness and was able to speak, what was more, after a short recuperation he came back to work. However, the event changed his personality traits and his temperament. Before the accident, Gage had a reputation of a hardworking and balanced person, after – he became impulsive, vulgar and incapable of achieving goals. The description of the impact of the so called 'organicity' on physical state of mind, since Gage's times and Alzheimer's [7] scientific exploration, has expanded. It is known that infections, medicines, amphetamine and related substances may cause psychosis similar to schizophrenia [8] (Bonhöffer 'exogenous' psychoses). These are just a few 'neurological' reasons of psychiatric symptoms. Another 'neurological' phenomenon, which affects lunching the first symptoms of affective disease (including stress factor) is called kindling, which is also blamed for resistance to treatment [9].

Mental state disorders, in turn, are common in many neurological diseases, which has been reflected in ICD-10 classification. Physicians dealing with neurology should pay special attention to the chapter 'Organic, including symptomatic, mental disorders' (F00-F09), which covers a group of mental disorders caused by brain dysfunctions due to its diseases, damages and injuries. Some of the diseases may by primary (diseases and damages directly affecting the brain), others – secondary (systemic diseases, indirectly affecting the brain).

General criteria for diagnosis of organic mental disorders assume, therefore, that there is confirmed (or known from medical record) disease, damage or brain dysfunction or general somatic disorders (except for the alcohol and psychoactive substances usage), for which there is probable link with mental disorders' emergence or exacerbation. Its removal or improvement may lead to a complete recovery (*ex iuvantibus*) or a significant improvement in mental disorder. Furthermore, the criteria assume no evidence of other cause of mental disorders.

On the other hand, one needs to be aware that identifying disorders defined as 'organic' does not mean that other mental states included in ICD-10 do not have

a brain background. For many years, localization studies of structural changes and functional patterns in mental illnesses, especially in schizophrenia or depression, have been carried out. More and more data from neuroimaging studies (computed tomography [CT], magnetic resonance imaging [MRI]) show the existence of structural changes in the brain in the course of psychoses traditionally considered as 'endogenous'. Other methods of examinations traditionally associated with neurology (or neurosurgery and neurotraumatology) such as: special electroencephalography (EEG) and the transcranial Doppler (TCD) were added to the collection of advanced methods of psychiatric diagnosis and research, for example in the treatment of schizophrenia, where they are used, for instance, to evaluate pharmacotherapy effects on changes in cerebral circulation [10]. At the moment, however, especially CT and MRI – primary neurological examinations – have become essential in differential diagnosis in psychiatry (as in the past EEG and the analysis of cerebrospinal fluid) [11]. They enable successful, absolutely crucial for good psychiatrists' and neurologists' cooperation differentiation of patients visiting (from many decades) one of the two medical specialists (despite the definite diagnosis), namely persons suffering from pseudoneurotic syndromes of such picture [11].

In turn, among the most common neurotic symptoms, taken into account for example in the psychiatric screening, symptoms of traditionally neurological interest (e.g. various pains, dizziness, imbalances, sensory and movement disorders, memory disorders), occur numerously. They are obviously primarily presented by patients to neurologists. These cases require a special attention as conversion disorders simulate failure or loss of motor or sensory function ('neurological'), as well as the dissociative dysfunctions of integration of mental functions may also resemble neurological disorders. Of course, too late recognition of non-psychiatric causes of pseudo-conversion disorders carries a risk to a patient [11].

The situation is complicated by clinically observed phenomenon of patients who even after excluding 'biological background' of their disorders, are resistant to being referred to psychiatrist or psychotherapist, and demand successful treating of their psychogenic functional disorders [12] such as: conversion and dissociative disorders. Diagnostic and therapeutic problems will probably always be caused by symptoms placed on the crossing of both sciences – such as hysteria, which used to be called 'the greatest simulator', or the pseudo-epileptic seizures.

Another group of psychiatric-neurological disorders creating an unclear spectrum between obsessive-compulsive disorder and Gilles de la Tourette syndrome, are developmental disorders usually with limited and repetitive behaviors, of the type of stereotyped interests or specific routine activities, suggesting a significant part of neurological 'organicity'.

Also the analysis of the suicidal behaviors has recently led to a surprising for psychotherapy-oriented specialists conclusion, that they have a substantial genetic background [13]. In fact, neurobiology of emotion regulation has recently been raised as a key issue, even for such a psychodynamically conceptualized disorder (and treated with such psychotherapy) like borderline personality disorder [14]. Actually, its contemporary criteria reflect the picture of the consequences of non-adaptive emotion regulation, partly neurocognitively conditioned, due to the hyperactivity of noradrenergic system and hypothalamic-pituitary-adrenal axis (connected with, for instance, experiencing trauma) and changes in serotonin, dopaminergic, and glutaminergic systems' transition, leading also to structural differences in areas responsible for impulsivity (amygdala, hippocampus, orbitofrontal cortex and anterior cingulate).

However, the search for neurobiological mechanisms associated with dimensional descriptions of personality and its disorders is still inconsistent. Perhaps because it is difficult to identify a complex function of the brain, limited to one location or neurotransmitter [15].

Recently, the role of substances such as oxytocin and vasopressin has been revealed in the biological regulation of sexual behavior, in stress response, maternity response, regulation of anxiety, and also possibly in the pathogenesis of schizophrenia, depression, autism, and addiction (administration of oxytocin is considered to make the treatment with neuroleptics more effective) [16].

What is more, the possible lack or scarcity of data on structural changes in mental disorders does not necessarily imply the absence of such changes, but just still imperfect methods of neuroimaging (or heterogeneity of the investigated psychiatric syndromes of clinical symptoms). It can be suspected that with technical progress more detailed penetration into the structure of the brain will prove that other, strictly psychiatric, diseases also have their structural background.

Also neuropsychological research indicate certain similarities of psychiatric diseases and disorders resulting from organic damage to the central nervous system e.g. in cognition [17]. Such studies of association between neuropsychological functions, especially relation of memory dysfunction and executive function with the intensity of psychopathological symptoms e.g. in schi-

zophrenic patients, have been conducted for a long time, for example with the use of the Wisconsin Card Sorting Test, a test N-back and Stroop test. Nowadays, it is known that the evaluation of neurocognitive function has potential importance for the prognosis of schizophrenia emergence [18], because these dysfunctions occur sometimes even before its first episode.

These disturbances in the course of the disease influence the subjective assessment of life quality and ability to perform roles by patients, that is why standardized tests (e.g. MATRICS) serve also to monitor changes of cognitive functioning in schizophrenia including processing rate, attention, working memory, learning visual and verbal material, reasoning, problem solving and social skills.

Cognitive functions disturbances are also used to assess the risk of recurrence of depression. Parameters of functions such as: attention, speed of psychomotor reactions, visual-spatial functions, working memory, and executive functions (including cognitive flexibility, response inhibition processes and decision making processes) but also emotion processing are today believed to be the measure of abnormal brain activity in states of depression, mania and even in remission [19].

Language efficiency is another neuropsychological competence seemingly distant from psychiatry. Still the clinical condition, the severity and course of schizophrenia appear to be linked with various language phenomena, verbal and nonverbal fluency, and abstracting ability [20,21].

Studies of organic disorders – post-stroke depression [22], and similar conditions frequent in the course (and sometimes even prior to the first episode) of neurodegenerative diseases, multiple sclerosis, amyotrophic lateral sclerosis, Huntington disease [23], dementia and others seem to be a mile step for neuropsychiatry.

Also in epilepsy, definitely neurological disorder, often (estimates vary greatly, from 9 to 62%) co-occurring depression is observed, attributed to neurochemical and neurophysiological changes in the limbic system, antiepileptic drugs, and reaction to chronic illness [24]. (What is particularly interesting, the course of neurologically confirmed epilepsy is sometimes entangled with an interaction of psychogenic seizures). The presence of depression in epileptic patients (similarly to other neurological illnesses) has a negative influence on survival rate, quality of life, it also increases the risk of suicide, and the cost of treatment.

In turn, despite the attempts to conceptualize the autism spectrum disorders (one of the disorders from the borderline of neurology and psychiatry) and a number of neuroimaging studies (structural and functional) [25] their etiology is still not completely defined.

Another group of neuropsychiatric symptoms of mixed etiology are aggressive behaviors (e.g. in the course of dementia), which usually are submitted to psychiatrists (on the basis of, for instance, The Law on Mental Health Protection, in connection with the regulation on hospitalization without content and the use of direct coercion).

However, recently it has turned out that in the pharmacological control of aggressive and impulsive behavior promising results are achieved not only with traditional 'psychiatric' antipsychotics, but also 'neurological' acetylcholinesterase inhibitors and valproic acid [26]. It is also worth adding that nowadays there is a generally increasing trend to research also other drugs from the borderline of these two specializations, such as S-adenosyl-L-methionine (involved in the synthesis of the neurotransmitters and melatonin, and mechanisms of epigenetic regulation) [27], in treating and prevention of a wide 'neuropsychiatric symptoms' profile. Hope for the reduction of drug-resistant depression, a common problem, has been incited by the NMDA receptor modulators (ketamine) [28].

To crown the general picture of neuropsychiatric linkage examples of psychiatric therapies referring to organic and neurological methods e.g. psychosurgery, transcranial magnetic stimulation (TMS) treatment or electroconvulsive therapy (especially in treatment-resistant states like catatonia or severe affective disorder) can be mentioned.

The last two decades has revealed that psychotherapy, the very difficult to 'biological' description and seemingly far from the neurology field, also has impact on the brain. It was predicted by the father of psychotherapy Z. Freud, who dreamed to make the knowledge of nervous system the basis of psychology and psychiatry. In his 'Project of scientific psychology' he wrote *What we experience as conscious and unconscious processing of information is reflected in the neuronal architecture of the brain and nervous system. 'Talking cure' can transform the neural connections and change the nature of mental experience* [29]. Since the 1990s, publications describing brain neuroimaging results in patients with various mental disorders (depression, compulsive syndrome, panic disorder, social phobia, arachnophobia, post-traumatic stress disorder, severe personality disorders) treated with psychotherapy, started to appear. Changes in their metabolism and blood flow in various regions of the brain, associated with the image psychopathological disorders, were

indicated. The main aspects of psychotherapy (e.g. impact on executive function and working memory, self-esteem, the way of regulating affective states) have the very biological effect in the relevant structures of the central nervous system (CNS) [30-33].

It is worth to quote here the words of a prominent American psychiatrist Nancy Andreasen: *Psychotherapy, often degraded to 'verbosity', is in a way as 'biological' as the use of drugs* [34]. The dichotomy neurology-psychiatry has no right to exist – to quote Joseph B. Martin, a neurologist, former Dean of Harvard Medical School: *The separation of these two categories (psychiatry and neurology) is arbitrary, it is often the result of convictions, not proven scientific observations. The fact that the brain and the mind are one makes the division artificial* [35]. Integration trends has existed since ever [4], it can be proven by The British Neuropsychiatry Association – multidisciplinary group (which members are psychiatrists and other professionals) and The Special Interest Group in Neuropsychiatry of the Royal College of Psychiatrist, which are constantly working on crossing the narrow models resulting from only one specialty. In the last decade the interest in association between psychiatry and neurology was intensified [36], which resulted in the formation of expert groups of psychiatrists and neurologists (enclosing neuropsychologists), together solving the theoretical and clinical problems that lie on the border between disciplines [37]. Such groups also run combined training at all levels of medical education – from undergraduate to advanced specialist [38], even in the form of combined programs of both specialization, e.g. the U.S. [39] under the supervision of the American Board of Psychiatry and Neurology. For the time being involving unfortunately a small number of doctors (1-2 residents per year). One may also may notice that in Polish specialists' education neurology and psychiatry barely take into account their mutual existence.

It must be pointed out that not all authors share the view of the inevitability or velocity of the connection of psychiatry and neurology [40]. If neurology and psychiatry really are separated by the mentioned by Martin *wall of differences at the level of philosophy, diagnosis, treatment, differences in approach to scientific research* the integration may not happen so fast, despite the intention to promote contact and cooperation.

Approaching psychiatry to neurology is indeed more visible on the side of psychiatry, which is also confirmed by studies on people in specialization training and their teachers [41]. Issues identified by respondents as being worth combining in teaching were somatization, dementia, chronic pain and medical treatment, and as the least purposeful were considered such efforts with eating disorders.

One of the biggest challenges for psychiatrists and neurologists are CNS tumors of atypical course [42,43], as they may cause a wide range of side effects in the area of cognitive deficits, resemble clinical dementia picture, mood and behavior disorders or reduce adaptive capacity. One of the most serious practical problems is therefore differential diagnosis. In case of its inaptitude, the likelihood of a successful treatment conducted in brain tumor (especially by a psychologist) is as low as a successful treatment of personality disorders by a neurologist working in the vegetative nervous system dysfunction clinic. Even more serious consequences may have a mistake in differentiating between 'non-neurological' schizophrenic psychosis and neurodegenerative disease such as Huntington disease [44]. Such diagnostic errors can be prevented by ensuring that in the diagnostic and therapeutic process both neurologists and psychiatrists are involved.

Differential diagnosis between psychiatric versus neurological disorders is necessary not only for a typical, primarily neurological and psychiatric disorders, but also in other somatic disorders causing secondary changes in the CNS, e.g. sarcoidosis, which can manifest in either simultaneous or alternate depressive and delusional syndrome or cerebellar symptoms, peripheral nerve neuropathy, and generalized seizures [45].

A lot of recently published works in Poland [46] concerns neurocognitive disturbances (visual memory, abstract thinking and fluency of verbal memory and subjective evaluation). The disorders are caused due to complications (microemboli, hypoperfusion of the CNS, systemic inflammation and other biochemical abnormalities, cerebral edema) associated with surgical procedures on the heart and coronary arteries [47,48], as well as with luckily much rarer cases of coma, stroke, seizures and blindness. Milder postsurgical cognitive dysfunctions are usually transient, but not in all patients, and modern clinical practice has not developed standard procedures for them.

Because of the major confusion or underestimation of co-occurrence of syndromes/symptoms of depression and dementia, a constant improvement in the neuropsychological testing methods of organic-psychic differentiation seems to be important [49].

Another group of conditions which secondarily expose the patient to neuropsychiatric complications are autoimmune diseases (especially lupus erythematosus) [50] affecting different systems and organs, including the nervous system, by vascular changes of inflammatory or

thrombotic, immune or atherosclerotic etiology. Their symptoms (cognitive distortions, emotional liability, and sleep disorders) may be complicated by the adverse effects of drugs (such as steroids), can occur separately or arranged in groups of diverse and changing forms including psychoses, seizures and other conditions. This makes it more difficult to differentiate if a given symptom is primary or secondary to treatment of the disease.

## Conclusions

For over twenty centuries, medicine had not separated neurology from psychiatry. The division which took place at the beginning of the twentieth century had a number of reasons and was probably inevitable. Now it starts to be a burden for us due to its multiple, serious consequences. Because of the contrary assumptions on the pathogenesis of numerous disorders (or lack of sufficient data), seemingly opposing groups of researchers and clinicians dealt with them, pushing aside the others as well as the idea of combination of both fields of medicine – the peak of this phenomenon occurred in the mid-twentieth century. Current neuroscience removes many divisions and blurs the boundaries, which can be exemplified by frequent co-occurrence of, inter alia, depression states and Parkinson disease, amyotrophic lateral sclerosis, multiple sclerosis, stroke and related dementia, and similar association of dementia depressive disorders [3].

*The belief that mental health is more than the absence of organic pathology of the brain is essential in the practice of both psychiatrists and neurologists* [3]. Psychiatry and neurology once again proved to be bound to each other like Siamese twins [51], regardless of the attempts to separate them.

## Disclosure

Authors report no conflict of interest.

## References


1. Angel K. Defining psychiatry: Aubrey Lewis's 1938 report and the Rockefeller Foundation. *Med Hist* 2003; 47 (Suppl. S22): 39-56.
2. Bomba J., Pilecki M. [eds.]. Mózg i Psyche. W stulecie utworzenia Katedry Psychiatrii i Neuropatologii w Uniwersytecie Jagiellońskim. Pokłosie Jubileuszowego Sympozjum. *Biblioteka Psychiatrii Polskiej KRW PTP*, Kraków 2007.
3. Baker M.G., Kale R., Menken M. The wall between neurology and psychiatry. *Br Med J* 2002; 324: 1468-1469.
4. Ring H.A. Wall between neurology and psychiatry. Neuropsychiatry is alive and well. *Br Med J* 2002; 325: 778.
5. Peralta V., Lang A.E. Crossing the borders between neurology and psychiatry in functional neurological disorders. *Mov Disord* 2011; 26: 1373-1374.
6. Marcinowski F. Leon Daraszkiewicz i jego monografia o hebefrenii. *Psychiatr Pol* 2012; 46: 123-131.
7. Goedert M., Ghetti B. Alois Alzheimer: his life and times. *Brain Pathol* 2007; 1: 57-62.
8. Rabe-Jabłońska J., Mirek M., Pawełczyk T. Czynniki ryzyka rozwoju schizofrenii u pacjentów uzależnionych od amfetaminy i jej pochodnych z psychozą (pointoksykacyjną lub schizofrenią) oraz bez psychozy. *Psychiatr Pol* 2012; 46: 571-584.
9. Ferensztajn E., Rybakowski J. Etapy przebiegu choroby afektywnej dwubiegunowej. *Psychiatr Pol* 2012; 46: 613-626.
10. Pawlak W., Szafrański T. Przezczaszkowe badanie dopplerowskie w schizofrenii – przegląd piśmiennictwa. *Psychiatr Pol* 2011; 45: 405-417.
11. Pawełczyk T., Pawełczyk A., Rabe-Jabłońska J. Zanim rozpoznasz u pacjenta zaburzenie konwersyjne, dokładnie zbadaj jego stan somatyczny i neurologiczny. Opis przypadku. *Psychiatr Pol* 2012; 46: 483-492.
12. Peters S., Stanley I., Rose M., et al. Patients with medically unexplained symptoms: sources of patients' authority and implications for demands on medical care. *Soc Sci Med* 1998; 46: 559-565.
13. Jaeschke R., Siwek M., Dudek D. Neurobiologia zachowań samobójczych. *Psychiatr Pol* 2011; 45: 573-588.
14. Pastuszak A. Regulacja emocji u pacjentów z zaburzeniem osobowości borderline – aktualne kierunki badań. *Psychiatr Pol* 2012; 46: 401-408.
15. Grabski B., Gierowski J.K. Zaburzenia osobowości – różne spojrzenia i próby ich integracji. *Psychiatr Pol* 2012; 46: 829-844.
16. Wójciak P., Remlinger-Molenda A., Rybakowski J. Rola oksytocyny i wazopresyny w czynności ośrodkowego układu nerwowego i w zaburzeniach psychicznych. *Psychiatr Pol* 2012; 46: 1043-1052.
17. Dudek D., Siwek M., Grabski B. Zaburzenia psychiczne w neurologii. *Termedia*, Poznań 2009.
18. Hintze B., Borkowska A. Nasilenie objawów psychopatologicznych a zaburzenia pamięci operacyjnej i funkcji wykonawczych u chorych na schizofrenię w okresie częściowej remisji objawowej. *Psychiatr Pol* 2011; 45: 457-467.
19. Kałwa A. Zaburzenia funkcji poznawczych w chorobie afektywnej dwubiegunowej. *Psychiatr Pol* 2011; 45: 901-910.
20. Waszkiewicz J., Wciórka J., Anczewska M., et al. Zaburzenia językowe a inne wybrane funkcje poznawcze u osób chorujących na zaburzenia schizofreniczne. *Psychiatr Pol* 2012; 46: 553-570.
21. Talarowska M., Florkowski A., Orzechowska A., et al. Zastosowanie baterii RHLB do oceny funkcji językowych i komunikacyjnych pacjentów psychiatrycznych – opis przypadku. *Psychiatr Pol* 2012; 46: 1089-1098.
22. Wichowicz H.M., Wieczorek D. Badanie przesiewowe depresji poudarowej z użyciem Hospital Anxiety and Depression Scale (HADS). *Psychiatr Pol* 2011; 45: 505-514.
23. Dubas-Ślemp H., Tylec A., Michałowska-Marmurowska H., et al. Choroba Huntingtona zaburzeniem neurologicznym czy psychiatrycznym? Opis przypadku. *Psychiatr Pol* 2012; 46: 915-922.

24. Bosak M., Dudek D., Siwek M. Depresja u chorych z padaczką. *Psychiatr Pol* 2012; 46: 891-902.
25. Bryńska A. W poszukiwaniu przyczyn zaburzeń ze spektrum autyzmu – neuroobrazowanie funkcjonalne (część II). *Psychiatr Pol* 2012; 46: 1061-1071.
26. Bidzan L., Grabowski J., Dutczak B., et al. Wpływ farmakoterapii inhibitorami acetylocholinesterazy, kwasem walproinowym i lekami przeciwpsychotycznymi na zachowania agresywne w przebiegu otępienia typu Alzheimera. *Psychiatr Pol* 2012; 46: 361-372.
27. Krzystanek M., Pałasz A., Krzystanek E., et al. S-adenozylo-L-metionina w schorzeniach OUN. *Psychiatr Pol* 2011; 45: 923-931.
28. Gosek P., Chojnacka M., Bieńkowski P., et al. Zastosowanie antagonisty receptorów NMDA (N-metylo-D-asparaginianu) – ketaminy w leczeniu depresji lekoopornej. *Psychiatr Pol* 2012; 46: 283-294.
29. Freud S. Project for a scientific psychology (1895). The Standard Edition of the Complete Psychological Works of Sigmund Freud. Vol. 1. *Hogarth Press*, London 1953; pp. 283-397.
30. Baxter L.R., Schwartz J.M., Bergman K.S., et al. Caudate glucose metabolic rate changes with both drug and behavior therapy for obsessive-compulsive disorder. *Arch Gen Psychiatry* 1992; 49: 681-689.
31. Brody A.L., Saxena S., Stoessel P., et al. Regional brain metabolic changes in patients with major depression treated with either paroxetine or interpersonal therapy: Preliminary findings. *Arch Gen Psychiatry* 2001; 58: 631-640.
32. Furmark T., Tillfors M., Merteinsdottir I., et al. Common changes in cerebral blood flow in patients with social phobia treated with citalopram or cognitive-behavioral therapy. *Arch Gen Psychiatry* 2002; 59: 425-433.
33. Rok-Bujko P. Neurobiologiczne podstawy psychoterapii (neurobiological basis of psychotherapy). In: Murawiec S., Żechowski C. [eds.]. Od neurobiologii do psychoterapii. *Instytut Psychiatrii i Neurologii*, Warszawa 2009; pp. 235-266.
34. Cozolino L.J. Neuronauka w psychoterapii. *Zysk i s-ka*, Poznań 2002, p. 224.
35. Martin J.B. The integration of neurology, psychiatry, and neuroscience in the 21st century. *Am J Psychiatry* 2002; 159: 695-704.
36. Barcia D. Acerca del reencuentro entre la neurología y la psiquiatría. Reflexiones de un viejo neuropsiquiatra. *Rev Neurol* 2007; 45: 746-754.
37. Cunningham M.G., Goldstein M., Katz D., et al. Coalescence of psychiatry, neurology, and neuropsychology: from theory to practice. *Harv Rev Psychiatry* 2006; 14: 127-140.
38. Oertel W.H., Kircher T.T. Das Verhältnis von Psychiatrie und Neurologie. *Nervenarzt* 2010; 81: 1281-1282, 1284, 1286-1288.
39. New York University website; http://psych.med.nyu.edu/education/residency-training/combined-psychiatryneurology-program [cit. 2013.02.20].
40. Pies R. Why psychiatry and neurology cannot simply merge. *J Neuropsychiatry Clin Neurosc* 2005; 17: 304-309.
41. Schon F., MacKay A., Fernandez C. Is shared learning the way to bring UK neurology and psychiatry closer: what teachers, trainers and trainees think. *J Neurol Neurosurg Psychiatry* 2006; 77: 943-946.
42. Pawełczyk A., Łojek E., Rabe-Jabłońska J., et al. Depresja czy apatia? Pułapki diagnostyczne: olbrzymi oponiak prawego płata czołowego rozpoznany i leczony jako epizod depresji umiarkowanej atypowej – opis przypadku. *Psychiatr Pol* 2012; 46: 903-913.
43. Bilikiewicz A., Smoczyński S. Psychopatologia guzów śródczaszkowych. *PZWL*, Warszawa 1989.
44. Grabski B., Siwek M., Dudek D., et al. Objawy psychotyczne sugerujące schizofrenię u pacjenta z potwierdzonym rozpoznaniem choroby Huntingtona – opis przypadku. *Psychiatr Pol* 2012; 46: 665-675.
45. Gaweł M., Domitrz I., Dziewulska D., et al. Objawy psychotyczne i zaburzenia poznawcze w przebiegu neurosarkoidozy – opis przypadku i przegląd piśmiennictwa. *Psychiatr Pol* 2012; 46: 1099-1108.
46. Sobański J.A. Współczesne kierunki badawcze w polskiej psychiatrii na podstawie publikacji w Psychiatrii Polskiej w latach 2010–2012. Doniesienie wstępne. *Psychiatr Pol* 2012; 46: 691-707.
47. Stążka J., Szepietowska E.M., Barańska E., et al. Funkcjonowanie poznawcze osób z chorobą niedokrwienną serca leczonych kardiochirurgicznie – ocena przed i po zabiegu. Badania pilotażowe. *Psychiatr Pol* 2012; 46: 757-769.
48. Szwed K., Bieliński M., Drożdż W., et al. Zaburzenia funkcjonowania poznawczego po zabiegach kardiochirurgicznych. *Psychiatr Pol* 2012; 46: 473-482.
49. Talarowska M., Zboralski K., Mossakowska-Wójcik J., et al. Wykonanie Testu Łączenia Punktów przez osoby z depresją i organicznymi zaburzeniami depresyjnymi. *Psychiatr Pol* 2012; 46: 273-282.
50. Celińska-Löwenhoff M., Musiał J. Zaburzenia psychiczne w chorobach autoimmunologicznych – problemy diagnostyczno-terapeutyczne. *Psychiatr Pol* 2012; 46: 1029-1042.
51. Ramsay A.R. Neurology and psychiatry: Interface and integration. *Psychosomatics* 1979; 20: 269-271, 275-277.