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THE CREATIVE POTENTIAL OF MICROGENETIC THEORY

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SUMMARY

This paper is devoted to illustrating how process neuropsychology and neurolinguistics, based on microgenetic theory and rooted in process thought, can help to explain the often baffling symptomatology of brain damage. Our purpose is to present an overview of this difficult and complex subject matter for readers, with particular emphasis on its creative potential. The essence of microgenetic theory in neuropsychology is an account of the phases in brain process through which successive mind/brain states arise and perish over the duration of the psychological present, measured in milliseconds. According to the theory, mental states are rhythmically generated out of a “core” in the anatomically deepest and phylogenetically oldest parts of the central nervous system, over phases to the outermost and youngest regions of the brain, the neocortex. The clinical applications are only one aspect of the creative potential of microgenetic theory. Indeed, the elegance of the theory consists in the way in which it can be extended into a number of different fields of endeavor, providing a kind of “unified field theory” for the explanation of often rather diverse phenomena. This provides an opportunity for neuropsychology and neurolinguistics to resume the interdisciplinary discourse they were founded to conduct.

Key words: neuropsychology, neurolinguistics, microgenetic theory, symptom, mental state

BACKGROUND

The terms neuropsychology and neurolinguistics emerged as the names of distinct branches of science after the Second World War, though both had appeared sporadically before, and the central issues of both disciplines also have a longer history. The post-war period was crucial, however, due to the confluence of a number of factors, ranging from theoretical (developments in neurology, psychology, and linguistics) to practical (the dramatic increase in the number of persons surviving gunshot wounds to the head during the war itself, with all the attendant symptoms and disabilities). Thus the Soviet neurologist Alexander Romanovich Luria, under the influence of the psychologists Pavlov and Vygotsky, and the linguists Jakobson and Leonteev, developed a body of systematic neuropsychological and neurolinguistic theory (Luria 1976a & b; 1996; Klimkowski 1976; Kądziaława 2003), supported by his own clinical practice with hundreds of World War II veterans. Although the claim that Luria “invented” neuropsychology and neurolinguistics is somehow redolent of Soviet propaganda, still, it is no easy task to find any earlier sources in Western literature for either term, used as the name of a putatively distinct science (Pachalska & Kaczmarek 2012). The work of Hecaen in France, Goodglass in Boston, and Poeck in Germany, at about the same time as the publication of Luria’s first books in English, all in the mid 1960s, suggests that (not for the first time in the history of science), many of the same ideas were emerging simultaneously in different places (Pachalska 1999; 2007; Kaczmarek & Markiewicz 2008). Thus establishing the priority of anyone’s claim to authorship of the terms in question would seem to be at this point neither possible nor particularly important.

The founding premises and basic character of both neuropsychology and neurolinguistics are implicit in the very names, whose compound form reflects a compound origin. Neuropsychology was to consist in an interdisciplinary discourse between behavioral neurology (focused on the brain) and cognitive psychology (focused on the mind). Neurolinguistics was intended to bring linguists into a mutually enlightening dialogue with clinicians (especially speech pathologists), much as psycholinguistics was to be a bridge between psychology and linguistics, sociolinguistics between sociology and linguistics, and so on. Each of the new sciences, then, was conceived as a sort of bridge between two separate bodies of knowledge with a common object of interest, but characterized by a different approach, and thus a different discourse. Knowledge about the brain, it was thought, would constrain the formation of theory in psychology and linguistics, while conversely, familiarity with psychological and linguistic principles would inform clinical practice in work with brain-damaged patients (Pachalska, Kaczmarek & Kropotov 2014). By the 1970s, both neuropsychology and neurolinguistics were well established as scientific disciplines, with clearly delimited fields of interest, generally accepted research methodologies, recognized academic centers, and official scientific journals. The “Decade of the Brain,” which ended in the year 2001, markedly increased both the store of knowledge and the prestige of both these two fields, now clearly counted among the “neurosciences.”

What has been lost, however, as these interdisciplinary projects have become separate sciences, is more than just a potential new perspective on the founding assumptions of psychology and linguistics, important as that would be. The points at issue in both neuropsychology and neurolinguistics are also deeply and intrinsically philosophical problems. No philosophy of mind can be expounded today without at least addressing the mind-brain problem, and any philosophy of language that does not include a reasonable account of how a mental concept or intention becomes a speech act, and vice versa, is manifestly incomplete. These are precisely the issues that neuropsychology and neurolinguistics were intended to address, but the present authors are compelled to confess, not without chagrin, that these philosophical preoccupations have largely disappeared from the discourse of the neurosciences. Rather, simplifying assumptions are made at the starting point of research, and the territory that lies beyond them is labeled “Here be dragons” (read: “Here be philosophy”) and carefully avoided. Thus the philosophy of mind is often done without much serious regard for brain work, while neuropsychology is typically done with a largely uncritical attitude towards the underlying concepts of cognition, perception, thinking, and so forth (Kaczmarek & Markiewicz 2008). Rather than a discourse unfolding at a meeting place where two roads intersect, we have built a cloverleaf exchange, with the mental vehicles whizzing past one another in opposite directions, meeting only with catastrophic consequences.

The absence of dialogue between the neurosciences and philosophy can be illustrated anecdotally. Several years ago, at very nearly the same time, Polish translations of Antonio Damasio’s *Descartes’ Error* and John Searle’s *Mind, Language and Society* appeared in many Polish bookstores, and were often displayed in the same window (Damasio 1999; Searle 1998). Most readers (like the booksellers who composed the window displays) can see at a glance that these two books are at many essential points devoted to the same topic, i.e. the relationship between the brain and the mind, and yet there would seem to be no point of contact between them. Not only do the two authors not cite each other, in fact they do not seem to have read any of the same books – with the interesting exception of Descartes, the favorite whipping boy of 20th century thought. In fact, scarcely a single book or author appears in the bibliographies of both books. Damasio and Searle are discussing the same phenomena, but within two realms of discourse, each of which seems oblivious to the other’s existence (see also: Searle 2008). More importantly, both these discourses are badly, not to say fatally flawed by the absence of precisely that awareness. Searle’s effort to find a via tertia between dualism and materialism would benefit greatly from contemporary insights into how the brain works, but in fact much of this information is still available only to a limited number of specialists who read the right journals and speak the lingo. As for Damasio, his assumption that developments in the neurosciences have simply rendered obsolete the concept of a mind as something other than a brain is to say the least philosophically naive (McGinn 2003).

Neuropsychology as a general rule has always tended to be about brains, not minds (Pachalska 2007; 2008; 2017; Paçhalska & Bidzan 2012; Paçhalska, Kaczmarek & Kropotov 2014; Ardila 2013; Kropotov 2016). The latter term is sometimes used, to be sure, in such stereotyped expressions as “changing one’s mind,” rather uncritically, or as a convenient way of talking about mentation, typically understood as an otherwise unspecified brain function, that which is left over when the other cognitive functions (memory, attention, perception, and so forth) are removed. More often, though, the mind (or the soul, or the psyche, or whatever term is used to designate the putative non-material dimension of human existence and cognition) is ignored or explicitly rejected as a concept irrelevant to an empirical science, such as neuropsychology is or aspires to be. A brain is a concrete object that can be measured, photographed (after a fashion), even removed from the skull and studied in the finest detail, while a mind is something whose existence we can infer, but cannot demonstrate (Ardila 2014). Not surprisingly, then, essentially mechanical idioms and metaphors representing the brain as a kind of biological computer dominate our thinking about thinking in the neurosciences, so that neuropsychology can begin to look like a certain kind of bioengineering. As perhaps inevitably happens (Lakoff 1987, Johnson 1987), the metaphor used to illustrate the concept begins to drive the concept, controlling what can and cannot be conceived. The brain is no longer like a computer, it is a computer, built of hydrocarbons instead of silicon, with nerves instead of wires and ganglia instead of circuit boards. Human language comes to be seen as an imperfect, quaint precursor of computational language, enabling the smooth flow of input and output. The mind is the software, and the brain is the hardware.

There are, of course, many reasons why the human brain cannot really be conceived as a computer. Even if we were to decide that what a computer does can be called “thinking,” certain stubborn biological facts remain. Computers do not grow, and when damaged, they do not repair themselves, but brains do both of these things (Cappa 1998, Papathanasiou 2003; Paçhalska, Kaczmarek & Kropotov 2014). Computers do not think (again, presuming that the operations a computer performs constitute “thinking”) in and for themselves, but rather react to the commands they receive from a user; brains, by contrast, execute commands they generate themselves – unless, that is, we conceive of the brain as a kind of instrument used by a distinct entity known as “the mind.” The mind, in this view, sits at the keyboard of the nervous system and looks at the screen of perception, using the brain exactly as the author of these words is using the computer. This seems a neat solution, but neither neuropsychologists nor philosophers are likely to find it particularly attractive, and in fact it begs the main questions posed by dualism and materialism. For the present purposes, however, the point is that a materialist account of the brain as a computer is confronted by the nearly insoluble problem of the “user,” without whom the computer has no input, and the output has no meaning.

Cognitivist theories of mind, on the other hand, can construct plausible, intellectually satisfying models of mental process (primarily cognition) precisely by setting aside the annoying details of brain and neuronal architecture. One postulates mental modules, each of which receives information from other modules, processes that information in a particular way, and then conveys the new information to the next module for further processing. Thoughts, behaviors, speech acts are broken down into elements in order to determine what transformations are needed to produce the observed articulations, which in turn indicates what “processors” or “modules” are needed in the model of the mind in order to account for these transformations (Jackendoff 2000). The difficulties in finding actual neural correlates for these processors, or the presumed connections between them, are taken as purely empirical problems, waiting for an answer until enough data have been amassed. The stubborn refusal of observable neural structures in the human brain to fall into a pattern of modules connected by transmission lines does not seem to have had much impact on the way thinking is modeled in cognitivist theory. Emotions, then, constitute the smoke, dust, and noise produced in the inner workings of the brain-machine, an approach which very much echoes the attitude towards emotion found in the works of many philosophers since at least the 6th century BCE. We are left with the unchallenged assumption that emotions and cognition belong to two entirely separate systems, as so eloquently stated by Pascal: “Le coeur a ses raisons, que la raison ne connait pas” [“The heart has its reasons, which reason does not know”; *Meditations* 4.277].

One of the implications that emerges clearly from these arguments and counterarguments, models constructed and deconstructed, is that clinical material based on the behavior of persons with brain damage continues to be a largely untapped source of verification or refutation for most, if not all of the proposed models of both brain work and mind work. If we assume that the brain works in thus-and-such a way, then it should be possible to predict what the consequences for thinking would be if particular parts of the brain were to be “switched off,” i.e. destroyed or otherwise taken “off line.” If that which we are calling “the mind” is affected by something that happens to the brain, and if the effects of brain pathology seem to display certain patterns of regularity and predictability, then this fact seems to have important implications for the brain-mind relationship. Indeed, upon further reflection it seems clear enough that no theory of thinking which fails to account in some way for the clinical effects of brain damage can possibly be valid, especially since thinking, in both broader and narrower senses, is almost always affected by such damage. Thus the potential for neuropsychology to enrich and perhaps constrain philosophical discourse about the mind remains a goal worth pursuing, despite the obvious difficulties in initiating and maintaining dialogue (MacQueen 2018).

Both the creative potential and the daunting barriers entailed by dialogue between the neurosciences and philosophy are clearly revealed by a consideration of the history of microgenetic theory in neuropsychology. This history has a be-

ginning in the holistic thinking of such pioneer neurologists as J. Hughlings Jackson and Kurt Goldstein, the evolutionary thought of Paul Maclean and others, and the important work of Karl Pribram, Alexander Luria, and other prominent figures in neuropsychology, consistently voicing reasoned doubts about the usefulness of the essentially connectionist theories that have dominated neuropsychology since the days of Carl Wernicke (see also Pachalska, Kaczmarek & Kropotov 2014; 2018). For neuropsychology, however, the history of microgenetic theory consists primarily in the scholarly biography of Jason W. Brown, the American neurologist who first began the systematic application of process thought to behavioral neurology and neuropsychology in the 1970s (Brown 1988, Hanlon 1991). The result is a fascinating corpus of work that has had considerable difficulty finding readers able to follow the arguments, which begin in the arcana of neuroanatomy and aphasiology, and end in process philosophy, influenced at certain crucial points by Buddhist thought and German idealism. Almost every reader will inevitably get lost at least once along the way. Thus both the theory and its founder are treated with great respect among neuropsychologists, but alas, with growing actual understanding (Pachalska 2002; MacQueen 2003, 2018; Kaczmarek 2012; Pachalska 2017; Kropotov 2018).

This is all the more regrettable because microgenetic theory has the potential to become a veritable Copernican revolution in the neurosciences, and at the same time to renew the lapsed dialogue between them and philosophy.

A BRIEF INTRODUCTION TO MICROGENETIC THEORY

The essence of microgenetic theory in neuropsychology is an account of the phases in brain process through which successive mind/brain states arise and perish over the duration of the psychological present, measured in milliseconds. According to the theory, mental states are rhythmically generated out of a “core” in the anatomically deepest and phylogenetically oldest parts of the central nervous system, over phases to the outermost and youngest regions of the brain, the gray matter that constitutes the neocortex (Brown 2000; 2015; Pachalska, Kaczmarek, Kropotov 2014). The progression in each mental state, then, runs through layers deposited by millions of years of evolution, and by the growth processes of ontogenesis, which in a general way replicate the course of evolution.

It is essential in this context to bear in mind the nature of evolutionary change, since in microgenetic theory the principles that apply to phylogenesis over eons of evolution and ontogenesis over years of growth are equally applicable to the formation of a behavior over the milliseconds required for a neural impulse to travel up through the successive layers of the central nervous system. Evolutionary change occurs as new layers are constantly deposited on the surface, covering but not supplanting the older layers beneath, as the lava from successive eruptions builds up the classic volcanic cone. When higher species, then, begin to evolve from lower ones, the latter do not simply disappear. The appear-

ance of the human species did not render chimpanzees obsolete; rather, about 4-6 million years ago there took place a bifurcation in the evolution of the ape, so that one branch became chimpanzees, and the other human beings. Evolution is generally a matter of bifurcation and branching, characterized by splits and discontinuities, rather than the kind of gradual transformation commonly (but mistakenly) associated with the word “evolve.” The evolution of a species does not take place within a generation or a lifetime, but in the succession between generations and individuals, and in the discontinuities that prevent this succession from being an incessant repetition of the same patterns. That is why the branching process of evolution, the precise moment at which a split takes place, is seldom perceptible when it happens.

The layers deposited in the human brain by evolutionary and growth processes can be represented in several different ways, but the “triune brain” model of Paul MacLean is a good starting point (MacLean 1967, 1991).

The brainstem and midbrain of the human being differ rather little from that of reptiles, fish, and amphibians. In other words, any vertebrate has a structure at the upper end of the spinal cord that looks very much like a human brainstem, relative of course to the size of the animal (only the structure called the “pons” is slightly fatter, proportionally, in the human brain). The difference is that in the lower orders these structures constitute the entire brain, while in the higher orders there are additional layers. Thus the evolution of the earliest mammalian species was accompanied by the appearance of new neural structures that branched out from the midbrain, and organized themselves into the structures known as the limbic system (largely responsible for the basic emotions) and the cerebellum (whose Latin name, a diminutive from cerebrum ‘brain’, reflects its nature as a more or less self-contained center for movement and sensation). These structures constitute the middle layer on Fig. 1, the paleo-mammalian

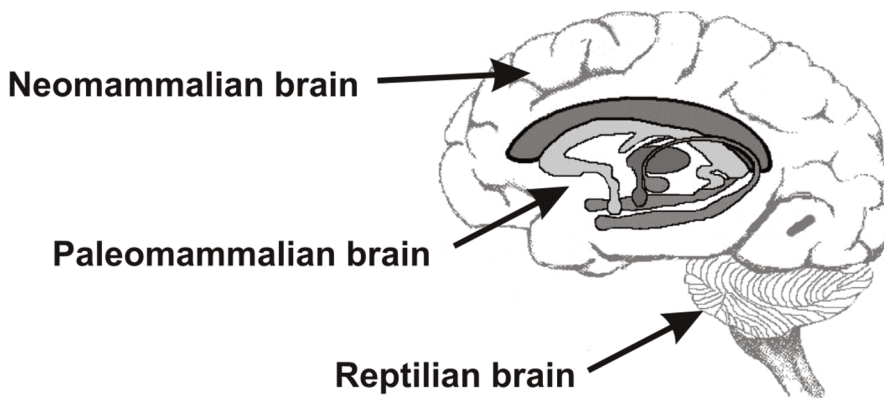


Fig. 1. Schematic representation of the three primary planes of evolutionary development in the human brain

Source: Pachalska (2008) with modification

brain. The important point, again, is that the paleomammalian brain does not replace or even strictly speaking subsume the reptilian brain that precedes it, but rather has been deposited on top of it. In the same way, then, the final layer of the human brain, the cortex (especially the forebrain), overlies the reptilian and paleomammalian brains (see also: Feinberg & Keenan 2005). The layering here is a matter of visible, anatomical structure, which can be revealed by dissection, and at the same time is reflected in function. Brain function is layered because the respective layers are not modules on a production line, but rather brains-in-the-making. This means that each of them can do what a brain does, i.e. produce behavior.

As Pachalska points out (2007, 2008), it is essential here to distinguish a sensori-motor reflex, which is linear and circular, like the inhale-exhale cycle, from an action-perception construct, which is simultaneous and parallel. As neural processing expands beyond the reflex level (i.e. above the lower brainstem), both sensation and movement are physical events that take shape outside cognition. On the level of the reptilian brain, then, the sensation-reaction cycle closes very quickly, differing little from a reflex arc. The brainstem is involved in many involuntary brain functions fundamental for life (pulse, respiration, the sleep-wake cycle), which is why damage to the brainstem almost always leads to either death or coma. Thus behaviors mediated primarily by the brainstem seem automatic, innate, and by the same token primitive. The frog, for example, has a rather fixed repertoire of reactions to stimuli and is capable of only a limited degree of adaptation. There is no perception in any meaningful sense of the word, but only a kind of gross sensation limited to detection of whole objects and motion, qualitatively undifferentiated for the most part; action is whole-body, immediate, algorithmic and inflexible (Pachalska, Góral-Pórola, Mueller i wsp. 2017).

The focus is upon survival, or perhaps more broadly, biological success: finding food and drink, avoiding threats, reproducing the species. Behavior is primarily approach-avoidance, modulated by drives and instinctive reactions. Upon hearing a sudden loud noise, the whole body tenses (producing the leap of the frog, the startle reaction in a higher animal) and the autonomic nervous system immediately raises the pulse rate, blood pressure, and respiration rate, long before the rest of the brain has taken in the stimulus, analyzed and identified it, and decided upon the appropriate reaction. Time here is the pulse and rhythm of heart and lungs, actions and reactions in a Newtonian system of balanced mechanics.

The limbic system, as previously mentioned, is the primary source of emotion, understood for the present purposes as a biochemical reaction to a stimulus. The brainstem works primarily with bioelectrical impulses, while the limbic system modulates the working of the nervous system through hormones, neurotransmitters, and the like. The world takes on color, in both a literal and figurative sense, and the approach-avoidance scheme of behavior is replaced by like-dislike, which in turn signals much more clearly the presence of a will and a self, entering into relations with objects that are liked and disliked (Heimer & Van Hoesen 2006).

There is an element of decision in the limbic system, as action here is less automatic and algorithmic. While the “reptilian” brainstem is oriented towards avoiding pain, i.e. avoiding danger and satisfying drives (the organism eats to assuage hunger, copulates to assuage tension, etc.), the “paleomammalian” limbic system is largely obedient to the pleasure principle. Perception on this level emerges from anxiety and desire, as objects are endowed with a highly subjective, emotional loading that takes precedence over their objective features, which in fact have not yet evolved into a perception. There is a different, more developed intentionality on this level, an “I-thou” that does not (yet) have a concept of the third person (Pachalska & MacQueen 2002). Limbic perception is thus the perception of memory, dream and hallucination, much more connected to the inner life of the perceiver than to any outer reality. Action is no longer restricted to whole-body movements, but begins to be goal directed, controlled by the cerebellum. The stiff and awkward movement of a patient with cerebellar damage is also the gait of a sleepwalker: arms stretched out, ankles stiff, eyes fixed forward (Bradford 2005).

Limbic time, as Brown points out (1996), is the floating, recurrent time of dream consciousness (see also: Pachalska 2007; Pachalska, Kaczmarek & MacQueen 2014; 2018). Events have a before-and-after sequence, McTaggart’s “A Series” (1934/68), but are not placed in a progressive time frame consisting of past, present and future (the “B Series”). Things happen, but they have already happened, and will happen again, perhaps differently, perhaps the same. The inability of this subcortical brain to discriminate objects on the basis of their articulated features means that there is no psychological basis for saying that one event is different from another. Everything is blended into everything else, identities shift and flow, images fade in and out. The present is not only constantly compared to the past, it is the past, recurring yet again. More importantly, however, the action/perception cycle takes on a rather different character, so much so, in fact, that the whole distinction of action and perception, which seems so obvious, makes little sense at the limbic level. Feeling, a subliminal and perceptually informed affective engagement with images, is the fundamental action of the limbic system, not something prior to and motivating action, as we usually think of stimulus-response cycles. Both action and perception are guided by a pleasure orientation and unfold together, simultaneously (MacQueen 2018).

The level of conscious reasoning, of that which we usually call “thought,” is the cortex, the layer of grey matter (about a centimeter deep) that overlies and surrounds the entire brain (hence the name, “cortex,” which in Latin means “bark”). Although the cellular architecture of the cortex is in fact quite complex (several areas, phylogenetically and ontogenetically older than the rest, make up the “limbic cortex,” as opposed to the “neocortex”), the gross structure of the cortex, unlike that of the limbic system, does not consist of distinguishable clusters of tissue that form organ-like structures, but is rather a complex, ramified surface, whose anatomical divisions into lobes and regions are to some extent arbitrary (Rohen 2013). This fact, apparent to the naked eye, is not without sig-

nificance. The modular approach to cognition seems to imply that the cortex should be made up precisely of modules (the boxes in a cognitivist “flow-chart”) connected by “wires,” and yet nothing in the physical appearance of the cortex suggests that it is built in this way. To be sure, the evidence from more than a century of systematic study of the symptoms produced by damage to specific regions of the brain seems to indicate very clearly that the cortex is not a completely homogenous mass, not the sensorium commune of pre-19th century medicine. Yet attempts to “map” the brain have produced results of rather dubious value, and more recent studies using new neuroimaging techniques have complicated rather than simplified the task of the map-makers (Andrewes 2001).

It is in the cortex that perception and action reach the level of conscious decision. The brain forms articulated pictures or representations of what is out there in the world, and of what has been out there in the world, and the play of these images constitutes conscious perception. What is more – and this has only recently begun to be a subject of interest for neuropsychology (MacQueen 2002; 2018; Pachalska 2007; 2008, 2017) – the cortex is capable of forming pictures and/or images (see: Fig. 2) of what might be or could be out there, or could have been, or should have been, and was not.

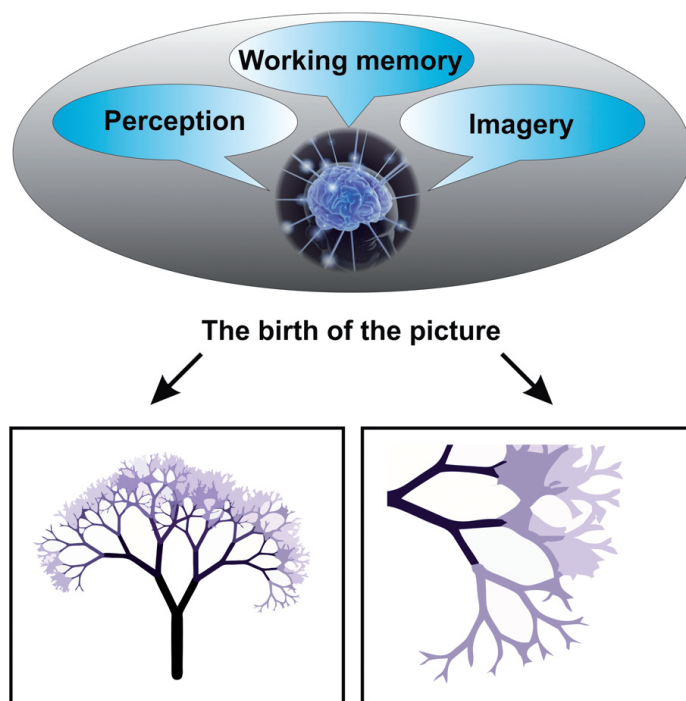


Fig. 2. The direction of brain and organism evolution, in full exteriorization, detachment and modulation of the process of perception.

Source: Pachalska, Góral-Pótróla, Mueller et al. 2017, with modification

It is not that hard to form a coherent theory of how the brain forms an image of something the eyes are seeing or have seen, but it is quite another thing to explain how the “mind’s eye” works in terms of brain structure and function.

For the present purposes, however, the most important fact about the cerebral cortex is that both perception and action at this stage are characterized by detail, discrimination, and analysis. The reptilian brain sees a large moving object, to be avoided, or seized, or ignored; the paleomammalian brain sees a human figure, producing an affect, positive or negative; the cortex sees features, details, a face, and can put a name to it, or not. The complexity of perception results from the fact that these three images come into existence independently and sequentially, though there is only one perceiver and one object, and the entire process takes milliseconds to complete. The conscious mind, then, typically experiences its perception as a single, simple act of seeing. According to microgenetic theory, however, this single act is a multi-layered actualization, the tip of an iceberg that floats to the surface and then subsides, containing within itself the traces of all that has gone before, in phylogeny, ontogeny, and microgeny (Pachalska 2002; 2007, 2017).

As objects take on their objective character in a sculpting process constrained by sensory data, time becomes a dimension of the subject-object system, along with space, which expands beyond the arms reach and the immediate visual field. The mind can conceive of places other than “here,” and time other than “now.” There is past, present, and future, which come into existence as concepts when the fluid before-and-after sequences of limbic time are projected out into the world and fixed to something that at least seems to be objective. This is not to say, of course, that the time of our consciousness is the same as clock time, or even that our ordinary assumptions about the three domains of time (past, present, future) are as natural or self-evident as they may seem. As Brown continually reminds us (1996, 2000, 2004), time is a central issue for microgenetic theory, where the crucial point is the duration or “thickness” of the “now.” Given that time-space is actually a continuum, the “forward” movement of time is more a psychological than a physical fact (Germine 2004). The past is a construct that is created and recreated at each moment of the now; the future is an extrapolation resulting from the experience of a certain “forward” momentum in the resurgence of the “now” over the rapidly receding past.

Thus from the physical (and metaphysical) point of view, the present is a dimensionless boundary between the fully actualized past, which having exhausted its potential no longer exists, and the potential future, which does not yet exist. When an arrow is shot from a bow at a target, its flight seems a single event, but this is a psychological fact, and not a physical one. Whether or not the arrow strikes the target at which it was aimed depends, of course, upon a number of variables: the skill of the archer, the distance, the wind, the movement of the target, etc. At the moment the archer releases the arrow, the range of possible outcomes is still very wide. With the proper video equipment, however, we can break this event down into a series of states, frozen on film, and at each of

these “nows” the number of possible trajectories is significantly reduced, as is the number of possible interfering factors. With each successive frame, then, it becomes easier to predict whether or not the arrow will strike the target, and at some point it becomes reasonably easy to predict exactly where it will strike the target. At 100 milliseconds before impact there is no real doubt what is about to happen.

The point of this example is that every mental act is in fact played out in the same way as the flight of this hypothetical arrow, and this is what forms the essence of time as the cortex learns to manipulate it. In fact, though the time in which cortex operates may seem more objective, closer to the fourth dimension of physical objects than the free-floating sequences of limbic time, the operations involved in creating past, present and future are ultimately subjective in nature. Patients with various kinds of brain disorders typically exhibit specific dysfunctions in the area of their experience of time, though for lack of theoretical groundwork there has been relatively little research done on this issue. Moreover, much of what has been done on the pathology of “orientation in time” is based on philosophically naive assumptions and has so far produced little of interest. The exception that proves the rule is Pöppel’s work (1988), which is much admired but seldom imitated. In most of the work on temporal orientation, the conventional past-present-future framework is taken as a self-evident reality against which the disturbances typical of, for example, Dementia of the Alzheimer Type (DAT) or Frontotemporal dementia (FTD) are measured (Harciarek 2012; Paçhalska & Bidzan 2012; Olszewski, Tłokiński & Lukaszewska 2012). Few if any have taken up the challenge of exploring how the brain creates time.

THE ARCHEOLOGY OF THE BRAIN

Sigmund Freud, as is generally known, was fond of remarking that psychoanalysis is “the archeology of the soul” (Jones 1960/62). The metaphor is often cited, but its meaning has seldom been explored. The image of the psychoanalyst digging in the rubbish of the past seems obvious, even stereotypical, but the notion of the present as a surface overlying successive deposits, and then itself subsiding to form yet another layer of substrate – this is a much richer metaphor. Memory is not a structure of interactive data bases in which information is shuttled through various connections and interfaces, but a layered, stratified structure made up of the “shells” of all the “nows” that have gone before. The deeper the layer, the more it is subject to a certain flattening, although the essential contours somehow persist.

The familiar Freudian models of the psyche (of which the “archeological” model is only one, and perhaps the least developed in Freud’s own work) have been replaced over the last century by more fashionable models, which sometimes proceed by mapping mental functions to neuroanatomical models, sometimes by mapping neuroanatomy to models of mental functions, but most often by mapping either neuroanatomy or mental functions with only a passing nod in the

other direction. The problem of the architecture of the psyche and its relation or lack of relation to the architecture of the brain and its billions of neurons has been largely marginalized, which may well be one of the primary reasons why microgenetic theory still struggles for a hearing, even though it presents an elegant solution to a problem that should be central to the neurosciences.

The problem, of course, is much older than either microgenetic theory or psychoanalysis. Indeed, there are ancient precedents in philosophy for MacLean's "triune brain" structure, of which one of the most interesting is surely Plato's "tripartite soul," particularly in the version developed in the Republic, which in turn influenced St. Augustine and those medieval philosophers whose thought was shaped by him. As the population of the ideal city is divided into three classes – workers, warriors, and rulers – so the soul, Socrates argues, is divided into three elements:

1. the desiring part (*to epithymetikon*), the seat of bodily appetites, corresponding to the workers, whose *raison d'être* is the consumption and production of goods;
2. the spirited part (*to thymoeides*), consisting of the "social" emotions characteristic of warriors, who must be prepared to sacrifice their lives for the common good;
3. the rational or calculating part (*to logistikon*), the ruling element, shaped by philosophy to strive for self-control and ever greater understanding.

Though of course Plato cannot have had any idea of the evolutionary development of the brain (indeed, in the fifth century BCE it was not at all clear to anyone that the brain, the contents of the skull, had any essential role in thinking), it is not hard to see that the elements of cognition, perception and action have been arranged in much the same way as the sequence from reptilian (brainstem) to paleomammalian (limbic system) to neomammalian (cortex) proposed by MacLean and developed by Brown in microgenetic theory.

In the Republic, Socrates argues that in a well-ordered city (or soul), good order and harmony depend on the ability of the *nous* to keep the lower impulses of *epithymia* and the "spiritedness" of the *thymos* under control. Yet the *epithymos* and the *thymoeides* are essential parts of the soul (*psyche*): for all the purity of its thought, the Platonic *nous* does not exist separately from the physical self. Thus the goal of philosophy for Socrates is not to dispose of emotions and passions, but rather to somehow harness them, or more precisely, to bring sensation, feeling and cognition into a "well-ordered state." Later, the Stoics consigned emotions to the category of *pathemata*: that is, an emotion is an event, something that happens to the soul, and not something the soul does, which would belong to *praxis*. When an adverse event occurs, as for example when one's house burns down, the event itself is a *pathema*, and so is one's emotional reaction to the event, that which we would today call "affect." Thus the Sage must be on his guard not to allow *pathemata* to steer *praxis*, a perversion of the natural order as the Stoics conceived it. This leads by a series of steps to the formation of that tradition in Western philosophy enthroning reason as the Good King and con-

signing the “passions” (*passio* is Latin for *pathema*) to the role of Evil Pretender. Pascal demurred, Nietzsche and Schopenhauer rebelled, the twentieth century went to the barricades, but even in the works of Freud, who is generally credited with disenthroning Reason and giving power to the lawless Id, the relative valuation of conscious reason and surging passion is constantly in the background. Freud’s volcanic Id and the “dark horse of the soul” in Plato’s *Phaedrus* are clearly drawn from the same source (MacQueen 2018).

This same problem, though couched in different terms, arises in microgenetic theory. The perception and action of the later phases of a mental act would appear at first glance to be superfluous, since the stimulus-response arc is closed very quickly in the brainstem. In order to allow for behavior shaped by higher levels of processing, it is necessary that the brain possess a mechanism to prolong, retard, suspend, or even interrupt the flow of the process, to allow successively higher (and slower) levels to operate. In evolutionary theory, one speaks of “neoteny,” the prolongation of a phase in development, which is often necessary in order to allow for elaboration of function; the prolonged helplessness of the human infant, for example, is both cause and effect of the fact that a much greater percentage of our behavior is learned, rather than innate. In microgenetic terms, neoteny in behavior suspends the immediate closure of the stimulus-response arc, an essential precondition for the heuristics of thoughtful behavior. This explains why the psychological time frames are increasingly broader as the mental act moves upward through the evolutionary planes. The “now” of the reptilian brain is the handful of milliseconds that elapses from the moment the frog sees a fly until it thrusts out its tongue to catch it; the limbic “now” is an envelope of cyclical dream time, in which hours seem like minutes and minutes seem like hours; the “now” of the cortex is the sum of everything that lies within the envelope of consciousness, created, as Brown suggests (1996), by the interval of time that exists between the arising of a psychological moment and its extinction, prolonged by the fact that the self and its objects do not disappear from one moment to the next, but rather linger, more or less successfully resisting decay.

This overlapping of “nows” is illustrated by Fig. 3.

Each of the three “Gothic arches” in this figure represents a behavioral cycle completed within one phase of the “triune brain” described above. The first cycle, A, is closed so quickly that in essence it differs little from a reflex arc. The reaction at time T_1 results directly and immediately from the nature of the stimulus (at T_0), consistent with a limited repertoire of instinctive or learned automatic behaviors. The second cycle, B, which begins at the same moment (T_0) in the “paleomammalian” brain, takes somewhat longer to be realized, at T_2 , which also means that cycle A as a whole is part of the environment to which the brain in cycle B (the limbic brain) is responding. In the same way, cycle C (the neocortex) takes in the two previous cycles, since it requires yet more time before a behavior emerges at T_3 . To some extent, the inhibition (or at least temporary suspension) of behaviors issuing from cycles A and B at T_1 and T_2 is a precondition for any behavior to be realized by cycle C at T_3 , since otherwise cycle C is perfectly su-

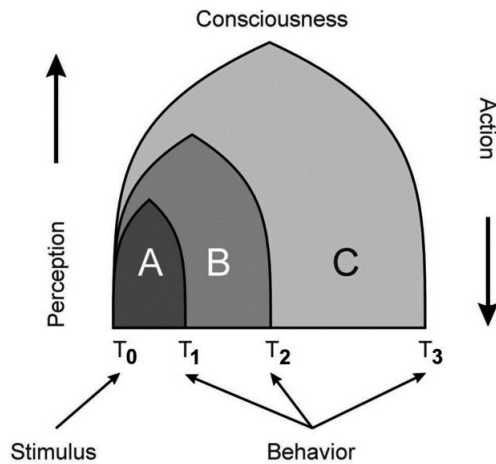


Fig. 3. A microgenetic model of behavior
 Source: Pachalska (2008) with modification

perfluous. This suppression function, as is well known, is especially associated with the frontal lobes, which have many projections back to the limbic system and the basal ganglia. At the same time, however, cycle C never appears completely independently of cycles A and B, which always form a part of the sub-structure for cycle C.

An important qualification is necessary at this point. We do not have three brains inside one skull, but rather one, vertically layered brain, in which at least three phases, corresponding to stages in phylogenesis, can be identified. The human brainstem may grossly resemble the brain of a frog, but unlike a frog, a human being cannot survive, even in a coma, if the cortex and subcortical structures are stripped away. If we actually had a self-sufficient frog brain inside our skulls, we would leap from lily-pad to lily-pad and catch flies with our tongues, and the higher levels of the nervous system would be no more than superfluous spectators to behaviors that have already been realized. In an evolutionary system, newer forms appear on the base of older forms, which may or may not persist. Thus in the ecosystem of which the newer form is now an element, the older form can exist in two ways:

1. as a continuation of itself, though as part of a changed system, as lemurs and macaques persist in the presence of more advanced ape species;
2. as an archaic layer in the substance of the newer form, as in the 95% of the human genome that does not differ from that of the chimpanzee.

The same applies, then, to mental process: the functions of the lower, deeper, older layers of the nervous system persist as behaviors that in certain circumstances emerge directly, and as the archaic substrate of everything that is done at higher levels. The force-fields that allow the respective layers of the brain to interact with each other, inside or outside the field of consciousness, are as yet

only beginning to be understood, which explains why reductionist models of behavior (such as that of behaviorism) come and go so quickly.

Action and perception in the cortex take distinct neural pathways, unfolding from memory (hippocampus and association cortex) outward to the anterior and posterior parts of the brain. It is not the case – the point must be stressed – that action in the anterior brain is bound to or emerges from perception in the posterior brain in a cause-effect relationship, or even that (as the “S-R” cycle of behaviorist psychology would have us believe) in a fixed temporal sequence. Action unfolds from memory, through the frontal lobes to the precentral gyrus and then out to the peripheral nerves and muscles, while perception, as Brown points out (2004), moves from parietal association cortex (again, memory) to primary visual and acoustic cortex in the occipital and temporal lobes, respectively. This is no longer anything like the reflex arc; it results, rather, from the specialization of areas of the cortex to control the formation of percepts or acts. This parcellation of tasks on the motor level is what enables the fingers, under the control of the motor cortex, to move independently; on the cognitive level, parcellation means that the whole brain does not do everything that the brain does. Just as animals and plants in an ecosystem find a niche for themselves, or perish, so neurons in the cortex find a niche, a particular task that they can do better than other neurons can do them, or they perish.

The part of the cortex that is involved in movement can be mapped with considerable precision to the various parts of the body, but it is important to recall that the motor regions of the cortex are not “circuit boards.” It is not the case that every muscle in the body is directly wired to (and thus controlled by) the motor cortex, which by the same token is not the point of origin for gross motor behavior. Rather, the “body map” in the motor cortex is mostly devoted to those parts of the musculo-skeletal system that perform delicate, articulated movements. Not surprisingly, then, the face and the hands (from forearm to fingers) are disproportionately represented in the motor cortex. When a person plays the violin, the cortex sculpts the grasp reflex into the fingering of the strings. The difficulty most people have in moving the little finger without moving the adjacent fingers or the whole hand is a result of incomplete sculpting in the motor cortex, not a mechanical problem in the hand itself.

Fully developed, rational behavior is thus formed by the elimination of excess, the trimming or parsing of the irrelevant, the inappropriate, the undesirable. The suspension of brainstem and limbic impulses enables the cortex to make its analysis of wholes to parts, or of potential to actual, in a process of creation that is more like the sculpting of a block into a figure than the modeling of a figure from a shapeless mass of elements. The essential notion here is of constraints, the factors that allow a thing to become what it is by not allowing it to become something else. Constraint, as Hume argued in his essay *On liberty and necessity* (1909-14), is not the same thing as necessity, or causation. The banks of a river constrain, but do not cause its flow. The river flows because of the force of gravity and the pressure of the water, but it flows here or there because of the

constraints of the landscape. Analogously, then, the cortex (especially the frontal lobes) does not so much cause behavior as constrain it, shape it, sculpt it to fit more precisely the situation into which the behavior evolves.

Anatomically, this is reflected in the extensive connections (visible in Fig. 4) between the frontal lobes and the limbic system (Pachalska 2003). The frontal lobes impose a kind of neoteny in behavior, a suspension or even interruption of algorithmic stimulus-response mechanisms, to allow parcellated perception and action to develop. This mechanism surely accounts, at least in part, for the intuitive conviction that “good” behavior is characterized by restraint (read: self-imposed constraints), and for psychological theories from Socrates to Freud, according to whom there is a constant struggle within the psyche between impulse and inhibition. The same thing goes on within each cell of the organism, as changes in the chemical and physical environment cause various genes in the DNA strands to be activated or deactivated, and specific receptors in the cell walls are opened by agonists or closed by antagonists. Nature displays a great conservatism of principle, constantly repeating the same themes at different levels of organization, from atoms and molecules through cells and tissues up to human societies and the cosmos.

In microgenetic theory, the flow of mental process is from the archaic to the recent in forebrain evolution, from the continually re-activated past to the present moment, which is born and immediately dies and decays. Momentary cognitions

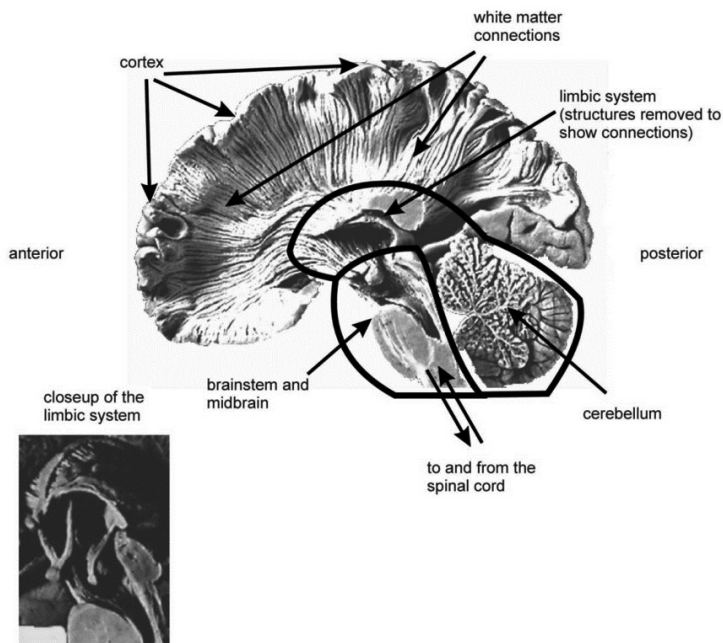


Fig. 4. Medial cross-section of the human brain
Source: Pachalska (2008) with modification

are extrapolated from memory to perception, and from the intrapsychic to the extrapersonal in spatial and temporal representation. Mental process is uni-directional, obligatory and recurrent, like the flow of a fountain, whose water continually circulates in such a way that the stream, though in reality it is constantly changing, seems to be constantly the same. What is fixed in a given state of mind is the cumulative series of phases traversed in a given actualization as it rises and falls in this "fountain" structure. Continuity, the ability to sustain a thought, an argument, a train of associations, even a mood, results from the constant repetition of these actualizations, which to the last moment traverse the same routes, and from the fact that a new actualization always appears before the last has faded. This overlapping of past and present gives the thickness of the psychological present, which for William James (1890) was one of the central questions of psychology (cf. Brown 1996, 2000, 2004).

Each present moment, then, is derived out of the memory and past experience of the antecedent state. It appears, perishes and is replaced, like the overlapping pulses or drops of experience described by James, or the succession of *ksana* ("point-instants") in Buddhist metaphysics. External and internal constraints on this process, such as sensation and habit, determine the degree of novelty in the mental contents. Concepts and categories may control the process in an algorithmic manner, virtually a reflex, and be deduced all the way down to objects perceived in the world without pausing to consider alternatives, or the cognitive process may undergo neoteny at an earlier, imaginal or conceptual phase, to serve as a springboard for the propagation of novel content. This is, once again, the evolutionary principle of branching: novelty does not appear from a relentless forward movement, but from a branching that occurs at some point before the main process reaches its natural endpoint. The content of the neotenuous image may be reproductive, as in the case of a memory image, or productive, as in imagination (or hallucination). There is, to be sure, some novelty in every act of cognition (no two moments are ever exactly alike), but what is productive should be interpreted as a deviation from a more general trend to repetition. Without the stability of this repetition the mind/brain would jump from one world to the next in each blink of the eye, which is exactly what seems to be occurring in some kinds of psychosis.

Most of us are aware that when we look at a film sequence, we are actually looking at a series of still photos, projected in such rapid succession that the images seem to flow together, creating the illusion of motion. The illusion is dependent, however, on there being relatively little change between one frame and the next; otherwise, the images jump and flash, spoiling the illusion of reality. According to microgenetic theory, all our perception in fact occurs in an analogous manner. Memory is a film to which new frames are continually being added, but each frame (that is, each perception) is itself the result of a series of transformations up through the evolutionary layers of the nervous systems. Thus there seems to be movement in two planes, horizontal and vertical, time and space, though in reality there is one movement, up-and-out. The brainstem's Gestalt

becomes the limbic system's image, and finally the articulated object of the cortex, which does not mean that we see three different objects in three different worlds. Rather, the object being perceived flows through these phases in a process of continuous change, more or less what one would expect from a nervous system that seems to consist entirely of connections (i.e. neurons) without any fixed point of destination for the information that is being conveyed along the "wires." The principle is one of movement and change, where stability is provided by reiteration.

One essential point that emerges from this discussion is that the conventional distinction made in neuropsychology (and elsewhere) between structure and function is not as sharp as it may seem in naive intuition.

The infant brain, which is still only to a limited extent capable of exercising voluntary control over the rest of the body, exhibits a pattern of exuberant neuron growth, creating a veritable jungle of synaptic connections. As maturity approaches, the development of new synapses slows markedly, and the superfluous ones begin to be trimmed away. Once again, the principle is one of sculpting, removing from a shapeless mass whatever is unnecessary, so that the desired form, whether of structure or of function, can emerge untrammelled. Maturity, in fact, is marked by a kind of shift from process that deposits structure to process that deposits function, though there is no real discontinuity between these moments in time, and the same process of discarding what is superfluous is still the guiding principle (Brown & Pachalska 2003). This sculpting process – or rather, to use the more precise term, parcellation – continues to the end of life, unless the atrophic, involutory processes associated with dementia (as in persons with Alzheimer's Disease) erode the neural architecture of the brain to such an extent that the elaborate dance of impulse and constraint breaks down. From the microgenetic point of view, then, dementia is precisely the result of involution, or perhaps more strictly, demented behavior is the result of "microphthisis," as normal behavior results from microgenesis.

SYMPTOMS

The claim was made earlier that any theory of thinking has to be able to account for the disturbances of thinking that occur when the brain is damaged. In the light of the foregoing, all-too-brief sketch of how the brain works, from the standpoint of microgenetic theory, it is possible to account for a great deal of what one actually observes in clinical practice with persons who have suffered brain damage, whether localized to one particular place (in what is called a "focal lesion") or spread out over much or all of the brain.

Each of the three levels of brain structure exhibits a similar relation of symptom type to lesion and anatomical phase. For example, evolutionarily recent, or surface, formations are associated with the analysis of form in expanded realms of space and time, while older, deeper, formations are associated with concepts and meanings in personal space, that is, within the immediate field of vision or the arm's reach. Typically, in the deeper structures it is bilateral lesions that pro-

duce the characteristic symptoms, with increasing laterality and specificity closer to the surface. The higher the point at which the lesion occurs, the more specific the symptoms, where by “higher” we mean, at one at the same time, “more advanced” and “farther removed from the base,” i.e. the brainstem. Both structure and function in the brain “branch out” from the brainstem (*truncus cerebri* in Latin), and the degree of elaboration and specification is to a large extent a function of the distance from that “trunk.”

These general principles are illustrated by the pathological phases in the realization of an action. In the condition known as akinetic mutism, a common early phase in recovery from prolonged coma after head injury, the patient lies motionless and does not vocalize, but there is no paralysis. The interpretation is that the envelope or kernel of the action is disrupted at the phase of its initiation. This in turn results from the fact that coma is usually caused by an insult to the brainstem, which is responsible for, among other things, the sleep-wake cycle. In akinetic mutism, then, the limited, stereotyped reactions which the “reptilian” brainstem originates are suspended, even though the cortex remains theoretically capable of moving the limbs. What the cortex cannot do unassisted, it would seem, is to set the body in motion. Many patients later report that during this phase of their recovery, they seem to remember conversing with people and even walking around the room, and then being puzzled that no one seems to react; in reality, they have been all this time in “akinetic mutism,” and their memory has recorded what they meant to do or say as being what they actually did or said. The converse situation arises when there is damage to the motor areas of the cortex (a band of gray matter about 2 centimeters wide just in front of the central fissure that divides the frontal lobes from the parietal and temporal lobes). The ultimate effect may seem at first glance to be the same, in that the patient is unable to move the affected limb, but the differences between paralysis and akinetic mutism are extremely significant. To begin with, in akinetic mutism there is no initiation of movement in any limb, while the patient with a paralyzed limb can usually move the other limbs. Even more importantly, the patient with paralysis resulting from damage to the cerebral cortex can actually perform gross movements of the limb (e.g. the arm moves at the shoulder), but is unable to make the finer movements needed for any kind of manipulation (e.g. is completely unable to move the fingers).

Indeed, the sequence of purely motor symptoms caused by nerve damage reveals very clearly how actions (in this case, movements) are created microgenetically, as one moves the point of damage higher and higher in the central nervous system, from the spinal cord through the base of the skull into the brain.

To begin with, if there is serious damage to the spinal cord in the lumbar or thoracic segments, the result is paraplegia, when the patient cannot walk; if higher, the arms are also affected, a condition which is called “tetraplegia” (or “quadriplegia”). In either case, the patient cannot move or feel either of the affected limbs below the level corresponding to the point where the cord is damaged. The mental image of what motion and sensation actually feel like is

unaffected, but the programmed activation of the effectors in the musculo-skeletal system to produce movement (a process not directly accessible to consciousness) is interrupted. Tetraplegics and paraplegics know all too well what they would do with their limbs, if only they could. The action (wiggling the fingers, moving the arm, walking) remains a potential object of cognition, even though the physical movement has become impossible.

If the damage is very high in the spinal cord, in the top two vertebrae or near the area where the cord passes into the brain at the base of the skull and becomes the lower end of the brainstem, the result is the “locked-in” syndrome, where the patient is fully conscious but unable to initiate any movement. Sometimes the eyelids are spared, sometimes the facial muscles, sometimes the vocal apparatus as well, all depending on how high the point of damage is and how completely the cord is severed. There is still full consciousness of what motion is, despite the complete inability to perform it, which makes the locked-in syndrome a “fate worse than death” for most patients and a major ethical dilemma for physicians and families.

If the brainstem itself is damaged (serious damage here is invariably fatal, since the brainstem controls pulse and respiration), there is akinetic mutism, in which, as described above, the patient lies motionless, and may even be “conscious” of movement that is not actually occurring in the limbs. Consciousness at this stage is fitful, as the patient drifts in and out, and the danger of dying or falling into a persistent vegetative state is very great.

If there is damage to the cerebellum, movement is possible, but it is dis-coordinated, awkward, jerky, often out of sequence (a condition referred to by neurologists as “cerebellar ataxia”). The patient can often learn to walk again, though slowly and with difficulty. Many patients report a persistent fear of falling, or even a sensation of falling when walking, which causes them to lunge forward or backward to regain balance, when in fact they have not lost their balance at all. Interestingly, we have observed in our own clinical practice that patients at this stage are unable to walk and talk at the same time: if one speaks to them while they are learning to walk, they will either ignore the interlocutor, or stop the effort to walk in order to enter into conversation.

Clearly, at this level, movement and language are using many of the same (compromised) brain resources, so that the two activities must compete for access.

When the basal ganglia are compromised, the patient’s motor behavior is slowed, becoming stiff and stereotyped. The face assumes a mask-like, impassive expression that gives little outward clue to what the patient is actually feeling or thinking. Trembling of the hands is common; as some readers will doubtless have noticed, this is the classic picture of Parkinson’s Disease, which tends to attack the basal ganglia first. If the limbic system is attacked, as in Tourette’s Syndrome, facial mimicry is exaggerated, motor behavior is disorganized by sporadic jerks and violent tics, and the patient often exhibits a symptom known as “coprolalia”: the involuntary utterance of foul words. Interestingly enough, coprolalia in one form or another is also encountered in stroke patients with subcortical

damage, and in many persons at an early stage in regaining consciousness after general anesthesia.

If there is damage only to the motor area of the cortex, the result is hemiplegia, that is, paralysis affecting one side of the body, left or right. The paralysis can be spastic or flaccid, depending on the exact nature of the damage and several other factors, but this is a peripheral issue for the present purposes. The main point is that the cortex is primarily engaged in complex, articulated actions, and damage to this area disrupts primarily (though not exclusively) the ability to operate with the muscles of the face and hands. In some cases, cortical damage produces a symptom called “apraxia,” in which the patient loses the ability to execute voluntary, learned movements, especially of the hands and fingers (sometimes the facial muscles as well), despite intact neurological function.

From this brief description of motor symptoms resulting from nerve damage at various levels, it becomes possible to infer how a normal action unfolds, since the symptoms here described constitute derailments of normal process at specific points, with consequences as predicted by microgenetic theory. The picture of action that emerges here begins with an incipient action that is first prefigured in body space, then initiated, and then projected out into extrapersonal space. The act unfolds from gross movements along body axes to fine distal articulations, from egocentric body space to extrapersonal space, finally to motor implementation, as actions in the mind become movements in the world. The act develops over tiers of spatial representation, from the space of the body through the action perimeter, the space of the arm’s reach (Bradford 1992), to an effectuation on objects in extrapersonal space.

The speech act, though obviously far more complex than limb motion, unfolds in an analogous manner, and the range of speech disturbances associated with damage occurring at specific points along the older-to-newer, inner-to-outer, self-to-world lines of microgeny can be explained in much the same way. As Brown points out (1988), the word selection errors seen in patients with aphasia do not represent a pattern of random mistakes:

- Some patients regularly commit errors in word choice that show wide semantic distance from targets, e.g. “wheelbase” for chair. This is called “semantic jargon” or “jargon aphasia,” a form of Wernicke’s aphasia, in which patients use words that bear little or no actual relation to the intended word, and not infrequently are not words at all.
- With “anomic” or “amnesic” aphasia, the errors tend to be in the same object category as the target word, e.g. “table” for chair, or the word may be semantically constrained but not evoked, the so-called “tip-of-the-tongue” state, often leaving the patient very frustrated.
- In “conduction aphasia,” the lexical item is available but does not achieve an adequate phonemic encoding. For example, these patients say “chore” for chair, “predisent” for president, and so on, and cannot repeat a sentence immediately after hearing it; they seem unaware of their errors and seldom correct themselves.

- In “apraxia of speech,” the phonemic realization of the words is likewise distorted, but there are frequent efforts to correct; the patient seems “tongue-tied,” as though every sentence were a tongue-twister, and struggles to be understood. The patient is frustrated by the fact that what is coming out of his mouth is not the speech act formed in his mind.
- In “dysarthria,” the patient is unable to make the speech apparatus obey the intention to speak. There is a fully formed and completely normal speech act in the mind, but its physical execution is faulty because one or more of the components of the peripheral speech apparatus (mouth, lips, tongue, palate, voice box, diaphragm) does not “fire” properly or at the proper time.

When these errors are correlated with the location of the lesions, it becomes clear that the progression from word meaning to word form is again occurring over growth planes in forebrain evolution (Pachalska 2007; 2008; Chantsoulis 2015& 2017). Respectively, then:

- In jargon aphasia, the lesion most often occurs in limbic-temporal cortex, the oldest part of the cortex, richly connected to and arising from the limbic structures (see Fig. 3).
- In anomic and amnesic aphasia, the lesion is likely to be in association or integration neocortex in the left hemisphere (phylogenetically and ontogenetically younger than limbic cortex but older than focal cortex);
- In conduction aphasia, apraxia of speech, and dysarthria, very specific areas of focal cortex in the language-dominant hemisphere (usually the left hemisphere in right-handed persons) are affected, so that specific functions involving the transition from lexical selection to phonetic realization are knocked out or disorganized by the lesion.

What emerges from the symptoms is progressive specification within the lexicon, a zeroing-in on the target item, using first semantic criteria, and then phonological. In other words, this is parcellation on the microgenetic scale. The major forms of what is usually called “receptive” aphasia (primarily affecting the understanding of speech acts, including one’s own) can be interpreted as disruptions at successive phases in the perceptual actualization of an utterance. The same holds true for the various forms of “expressive” aphasia (primarily affecting the ability to produce speech acts), where again (though the demonstration is lengthy, cf. Brown 1988) the various syndromes we see in the clinic reflect varying points in normal processing where the interference occurs. There is normal processing up to a certain point, then a derailment or gap, and then a resumption of normal process with some elements missing or out of place (Brown & Pachalska 2003).

In language, as in action, microgeny actualizes through a sequence of context-item transformations. The symptom reveals correlation with evolutionary growth, and is also the key to understanding the process. What then is a symptom? Instead of the unreflective assumption that a lesion destroys or disables a center, an operation or a representation, brain process should be conceived in terms of wave-fronts or recurring fields, and a lesion as a disruption in the flow, comparable to a rock in a stream. The rock produces an eddy or whirlpool that

retards flow but does not stop it (which is to say, neoteny occurs, a heterochrony of flow in the water above, below, and beside the lesion). The extent to which the process is restored to normal flow depends, then, on how large the rock is, and where it occurs in relation to the point where the stream is measured. What emerges as a symptom is not a deficit, not an empty place where a behavior used to be, but a fragment of a normal process, prematurely brought to the surface by the “rock in the stream.” This is one of the fundamental problems of neuropsychology, which remains to be solved: so long as we use tests only to measure what the patient cannot do, we do not really understand what the symptom is, or what it means. Microgenetic theory (Brown & Pachalska 2003) suggests how symptoms reveal the inner structure of behavior.

To summarize: the mental state develops out of reflex systems in the brainstem, where the purely sequential nature of sensorimotor reflex arcs undergoes a shift to a simultaneous act-object. This cognitive core is surrounded by a multi-tiered layering of physical input and output. The pattern of a continuous sheet of thinking that flows from self out to world, characterized by a wave of whole-to-part transformations sculpted by sensory constraints, activating a series of motor keyboards, becomes the model for successive levels of derivation. The progression is from unity to multiplicity, with reiteration of the sequence within the state and across states in overlapping waves. The model requires that events within the same phase are simultaneous from one domain to another (for example, movement and speech), and that all events pass through each phase in the series.

THE NEUROPSYCHOLOGY OF CONSCIOUSNESS

Although consciousness has always been an important topic for both philosophy and psychology, neuropsychology until recently has not ventured into this territory (Brown 1996, 2000). There are several reasons for this, one of which is surely the problem that consciousness is so variously defined. A scientist or clinician who is trying to solve a particular concrete problem can perhaps be forgiven for attempting to sidestep this difficult issue. In neurology, consciousness is normally defined as the nervous system’s readiness to receive sensory stimuli and react to them on an on-going basis. In psychology, on the other hand, there are as many definitions of consciousness as there are schools of psychological thought, but at the risk of some simplification it can be said that consciousness from a psychological perspective is most often defined in a somewhat circular fashion, as the individual’s awareness of being aware: if I know that I know something, then I am conscious of knowing something, and thus I am conscious. Even Freud’s discovery of the unconscious is at some level based on just such a definition, since the fundamental doctrines of psychoanalysis emerge from the premise that we can know something and not know that we know it, which is precisely what the unconscious is.

Although Freud was a neurologist by training and generally very interested in the brain (Wallesch 2004; Pachalska et al. 2018), his views on the nature of the psyche and the inner life put him at odds with the pioneers of neuropsychology, such as Carl Wernicke. He never tried to place the unconscious (or the Id) in the brain, and despite his lifelong fascination with Darwinian evolution he made only sporadic and not very systematic efforts to apply evolutionary thinking to his psychological doctrine until his last great work, *Moses and Monotheism*. Nowadays, the idea of applying neuropsychological methods to such fundamental problems of psychoanalysis as consciousness, or conversely, of applying psychoanalysis to neuropsychological research, will strike many specialists on both sides of this divide as a very dubious undertaking. The result of this mutual suspicion, however, is that an extremely important area of inquiry has remained largely a no-man's land (or perhaps, to update the metaphor, a "free-fire zone"). There is, to be sure, an admirable journal called *Neuropsychoanalysis*, but it has been dogged by marketing problems from the beginning, which perhaps serves to prove the point. This is much to be regretted. Neuropsychology, by focusing largely on specific cognitive processes (leaving emotion and even mentation for separate treatment) without having a coherent theory of consciousness, identity, or personality, may well be making bricks without straw (Feinberg & Keenan 2005; Pachalska et al. 2018). On the other hand, theorizing about consciousness without taking the brain into account would seem to be an undertaking at least equally dubious.

The primary reason why neuropsychology has not produced a theory of consciousness (and has thus remained largely a marginal contributor to the philosophy of mind) is that the dominant views of brain function in the neurosciences make such a theory all but impossible to conceive. A modular mind/brain made up of discrete processors shuttling bits of data back and forth does not need to be conscious in order to do its job. If computers were to become conscious they would by the same token cease to be useful as computers, and if we conceive of our brains as organic computers, as is fashionable nowadays, then the same applies to them. For Damasio, for example (2000; 2003), all mental states (emotions, cognitions, and consciousness) are perceptions of the body, so that the very notion of consciousness becomes superfluous, an anachronistic and misleading name for the fact that a mental state exists in a given person at a given time. As McGinn points out, however (2003), Damasio has neglected the intentionality of mental states and reduced subjects to objects (as one might expect from the author of *Descartes' Error*), avoiding the "homunculus" trap but falling straight into another, yet more insidious. Even if I say, "My consciousness is a perception of my body," what is the meaning of the first person singular "my"? One should say, "This consciousness is a perception of this body," as a computer would say, since the very word "my" brings the homunculus right back out of the waste basket. But if first-person-ness is not simply a verbal artifact left over in the language from a long rejected paradigm, like "sunrises" and "sunsets," if the brain-damaged patient who says "he" in reference to himself is in fact displaying

a loss of some essential element of consciousness, and not just language competence (MacQueen 2017), then the simple reduction of consciousness to perception and perception to bodily sensation propounded by Damasio gives us no basis whatever for understanding the basic problem. If neuropsychology is to make a contribution to philosophical discourse about consciousness, then we need to find another way to do it.

To the present authors, it seems clear that microgenetic theory, based on process thinking on the one hand, and acute observation of clinical phenomena on the other, is precisely the path that we should be taking. Consciousness is not simply a particular state of the mind/brain, something predicated of the brain as one of many possible attributes. It is, rather, the mental process by which the self creates itself. As perception and action percolate upwards through the nervous system, through the evolutionary planes of brain growth, consciousness emerges in three primary stages:

In the midbrain core, perception and action are indivisible and fully complementary parts of a single event, and the self functions automatically, purely ecologically, as a part of the environment. There is no distinction between inner and outer worlds, self and other, so that even the notion of “self” at this stage is an interpretation from the perspective of the observer. There is the natural selfishness of the organism striving to survive at all costs, reacting instinctively to threats and drives, but this is not the same thing as self-consciousness.

In the next stage, the limbic self turns inward from the outer environment and creates a dream world of pure feeling (Bednarek 2017). The world consists of the self and the objects which lie within its cocoon, similarly as in a dream (see Fig. 5). This is an animist world, in which the objects perceived by the self are all “thou,” seen as sentient beings endowed with intentionality.

In the cortical self, perception and action are constrained by an awareness of the existence of self as subject and a world populated by objects. There is a theory of mind which enables the self to distinguish between objects that are sentient and those that are not.

Consciousness is usually seen by neurologists as a phenomenon of the cortex. When we sleep, it is precisely the electrical activity of the cortex that is most affected. In dream, the limbic system is in active dialogue with the sensory cortex, but the pons (a brainstem structure), having set the dream process in motion, shuts down the flow of sensation (in dream, after all, we “see” with our eyes shut). This does not mean, however, that this “cortical” consciousness is something entirely separate from or antagonistic to the lower layers of the self. Extensive bilateral damage to the cortex also produces coma, even when the brainstem is untouched. A patient in the final stages of a neurodegenerative disease, such as Alzheimer’s disease or the MELAS syndrome (Pachalska & MacQueen 2001), which gradually destroys the cortex, is mostly conscious in the strictly neurological sense, since the central nervous system is in a state of readiness to receive sensory stimuli and react to them, but in any other sense of the word consciousness has been lost. What this means is that the cortex, with its characteristic



Fig. 5. The limbic self in dream: a picture entitled “Lost Self,” presenting a dream after the imposition of Martial Law in Poland in 1981, associated with a situation at the airport in August 1981, painted by artist Zbigniew Perzanowski after a Transient Ischaemic Attack (TIA).
Source: clinical material provided by Maria Pachalska

functions of analysis, discrimination, and articulation, is no longer able to control either perception or action. There are other neurodegenerative diseases in which the pathological process begins at a lower level, such as Parkinson’s Disease, and the cortex is largely spared until the disease progresses. Functions deteriorate and there is steady decline, but until the brainstem ceases to function the cortex does its work. Although dementia does sometimes occur with Parkinson’s Disease, its symptoms and course are quite different from those of Alzheimer’s Disease, since the derailment occurs at a deeper level and does not have much direct effect on consciousness until the disease is far advanced.

Consciousness is not purely a cortical phenomenon, from the microgenetic point of view, but emerges precisely from the process of evolution, passing from an undifferentiated core, through an animist dream world, to a world of self and objects. It is the whole process, not its just endpoint, that constitutes and creates consciousness. This is illustrated by Fig. 6.

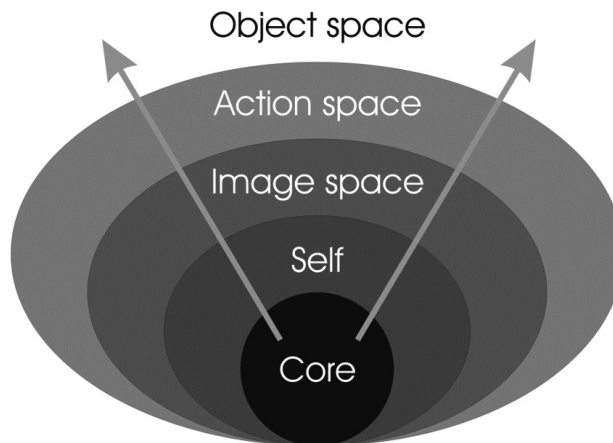


Fig. 6. Levels in the mental state. Self, image, and objects are stages in the unfolding between an intrapersonal core and the representation of extrapersonal space.

Source: Pachalska, Kaczmarek & Kropotov 2014, with modification

The self arises at a phase in the forming of the object prior to the resolution of clear mental images in sensory cortex, but it is not reducible to an image, even a reflective one. The duration of brain process creates an envelope of time and space; the self gazes at this envelope and feels itself present in the moment, which is the essence of consciousness. The overlapping of these moments and the continuing presence of the conscious self over the flow of time creates identity, which in this way can be approached as a neuropsychological problem. Consciousness, in the microgenetic concept, is not a sort of homunculus sitting upon the rumbling volcano of the id and praying that it will not erupt, as in the Freudian psyche, but rather the product of the self pushing its way up through the evolutionary planes and perceiving itself in action.

CONCLUSION

This paper is devoted to illustrating how process neuropsychology and neurolinguistics on the basis of microgenetic theory, rooted in process thought, is supported by the observable symptoms of brain damage. What often happens in clinical practice is that the bewildering array of symptoms conceals the regularities and patterns that underlie them (Pachalska 1999; Pachalska, MacQueen & Brown 2012a &b). Most clinicians will immediately recognize the symptoms we have already discussed here, in terms of motor dysfunction and language disturbances, and could easily add a host of additional categories and sub-categories (Pachalska, Kaczmarek & Kropotov 2018). Our purpose was to present an overview of this difficult and complex subject matter for readers and to venture into this dangerous territory, fully aware of the perils, and also of the enormous potential. Of course the clinical applications are only one aspect of the creative potential of microgenetic theory. Indeed, the elegance of the theory consists in

the way in which it can be extended into a number of different fields of endeavor, providing a kind of “unified field theory” for the explanation of often rather diverse phenomena (MacQueen 2003). High time, then, for neuropsychology and neurolinguistics to resume the interdisciplinary discourse they were founded to conduct.

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REFERENCES

- Ardila, A. (2013). A new neuropsychology for the XXI century. *Arch Clin Neuropsychol*, 28(8), 751-62.
- Andrewes, D. (2001). *Neuropsychology: From theory to practice*. Hove and New York: Psychology Press.
- Ardilla, A. (2014). *Aphasia Handbook*. Miami: Florida International University.
- Bednarek, S. (2017). Świat marzeń sennych: w stronę interpretacji neurokulturoznawczej. In: G. Jastrzębowska, J. Góral-Pórola, A. Kozołub (red.), *Neuropsychologia, neurologopedia i neurolingwistyka in honorem Maria Pałchalska* (s. 433-458). Opole: Wydawnictwo Uniwersytetu Opolskiego.
- Bradford, D.T. (1992). *Interpretive reasoning and the Halstead-Reitan tests*. North Tonawanda, New York: Multi-Health Systems.
- Bradford, D.T. (2005). Autoscopic hallucinations and disordered self-embodiment. *Acta Neuropsychologica*, 3(3), 120-189.
- Brown, J. (1988). *Life of the mind*. Hillsdale, N.J.: Erlbaum.
- Brown, J. (1991). *Self and process*. New York: Springer-Verlag.
- Brown, J. (1996). *Time, will and mental process*. New York: Plenum.
- Brown, J. (2000). *Mind and nature. Essays on time and subjectivity*. London: Whurr Publishers.
- Brown, J. (2004). A microgenetic approach to time and memory in neuropsychology. *Acta Neuropsychologica*, 2 (1), 1-12.
- Brown, J.W. (2015). *Microgenetic theory and process thought*. Exeter: Imprint Academic.
- Brown, J., & Pachalska M. (2003). The nature of the symptom and its relevance for neuropsychology. *Acta Neuropsychologica*, 1(1), 1-11.
- Cappa, S.F. (1998). Spontaneous recovery in aphasia. In: B. Stemmer, H.A. Whitaker (eds.), *Handbook of neurolinguistics* (pp. 536-544). San Diego: Academic Press.
- Chantsoulis, M., Mirski, A., Rasmus, A., Kropotov, J.D., Pachalska, M. (2015). Neuropsychological rehabilitation for traumatic brain injury patients. *Ann Agric Environ Med*, 22(2), 556-563.
- Chantsoulis, M., Pórola, P., Góral-Pórola, J., Hajdukiewicz, A., Supiński, J., Kropotov, J.D., Pachalska M. (2017). Application of ERPs neuromarkers for assessment and treatment of a patient with chronic crossed aphasia after severe TBI and long-term coma – Case Report. *Ann Agric Environ Med.*, 24(1), 141-147. doi: 10.5604/12321966.1232770.
- Damasio, A. (1999). *Błąd Kartezjusza: emocje, rozum i ludzki mózg*. Poznań: Dom Wydawniczy Rebis.
- Damasio, A. (2000). *Tajemnica świadomości*, Poznań: Dom Wydawniczy REBIS.
- Damasio, A. (2003). *Looking for Spinoza: Joy, sorrow, and the feeling brain*. New York: Harcourt.
- Feinberg, T.E., Keenan, J.P. Eds. (2005). *The Lost Self: Pathologies of the Brain and Identity*. Oxford: Oxford University Press.
- Germine, M. (2004). Why time moves forward. In: A. Combs, M. Germine, & B. Goertzel (eds.), *Mind and time: The dynamics of thought, reality and consciousness* (pp. 227-233). Creskill, NJ: Hampton Press.

- Hanlon, R.E. (ed.). (1991). *Cognitive microgenesis. A neuropsychological perspective*. New York: Springer Verlag.
- Harczarek, M. (2012). Językowy wariant otępienia czołowo-skroniowego (lvFTD). W: M. Pączalska, L. Bidzan (red.), *Otępienie czołowo-skroniowe: ujęcie interdyscyplinarne* (s. 145–173). Kraków: Wydawnictwo KA AFM.
- Heimer, L., Van Hoesen, G.W. (2006). The limbic lobe and its output channels: Implications for emotional functions and adaptive behavior. *Neuroscience & Biobehavioral Reviews*, 30: 126–147.
- Hume, D. (1909-14). *An enquiry concerning human understanding*. Part VII: On liberty and necessity. Harvard Classics. Cambridge, Massachusetts, USA: Harvard University Press.
- Jackendoff, R. (2000). Fodorian modularity and representational modularity. In: Y. Grodzinsky, L. Shapiro, & D. Swinney (eds.), *Language and the brain. Representation and processing*. San Diego: Academic Press.
- James, W. (1890). *Principles of psychology*. New York: Holt.
- Johnson, M. (1987). *The body in the mind. The bodily basis of meaning, imagination and reason*. Chicago: University of Chicago Press.
- Jones, E. (1960-62). *Das Leben und Werk von Sigmund Freud*. In 3 vols. Bern: H. Huber.
- Kaczmarek, B.L.J., Markiewicz, K. (2008). The brain and the mind. W: M. Pączalska, M. Weber (red.), *Neuropsychology and philosophy of mind in process. Essays in honor of Jason W. Brown* (s. 98–109). Frankfurt–Lancaster: Ontos Verlag.
- Kadzielawa, D. (2003). Zaburzenia językowe po urazach czaszkowo-mózgowych. W: A. Herzyk, B. Daniluk, M. Pączalska, B.D. MacQueen (red.), *Neuropsychologiczne konsekwencje urazów głowy: Jakość życia pacjentów* (s. 17–24). Lublin: Wydawnictwo UMCS.
- Klimkowski, M. (1976). *Pamięć człowieka i jej mechanizmy. Analiza neuropsychologiczna*. Lublin: Wydawnictwo UMCS.
- Kropotov, J.D. (2016). *Functional neuromarkers for psychiatry*. San Diego: Academic Press, Elsevier.
- Kropotov, J.D. (2018). Functional neuromarkers for neuropsychology. *Acta Neuropsychologica*, 16 (1): 1-7
- Lakoff G. (1987). *Women, fire and dangerous things*. Chicago: University of Chicago Press.
- Luria, A.R. (1976a). *Podstawy neuropsychologii*. Tłum. Danuta Kadzielawa. Warszawa: PZWL.
- Luria, A.R. (1976b). *Problemy neuropsychologii i neurolingwistyki*. Tłum. E. Madejski. Warszawa: PWN.
- Luria, A.R. (1996). *Basic problems of neurolinguistics*. Hague: Mouton.
- MacLean, P. D. (1967). The brain in relation to empathy and medical education. *Journal of Nervous and Mental Disease*, 144, 374-382.
- MacLean, P. D. (1991). Neofrontocerebellar evolution in regard to computation and prediction: Some fractal aspects of microgenesis. In: R.E. Hanlon, ed. *Cognitive microgenesis: A neuropsychological perspective* (pp. 3-31). New York: Springer Verlag.
- MacQueen, B.D. (2002). Imagining the brain. *Medical Science Monitor*, 7(4), 564-566.
- MacQueen, B.D. (2003). How a sentence unfolds. *Acta Neuropsychologica*, 1(4), 382-406.
- MacQueen B.D. (2017) Tożsamość jako tekst. W: G. Jastrzębowska, J. Góral-Pórola, A. Kozołub (red.) *Neuropsychologia, Neurologopedia i Neurolingwistyka* in honorem Maria Pączalska. Opole: Wydawnictwo UO: 296-322.
- MacQueen, B.D. (2018). Microgenetic theory of consciousness. Lecture presented at the Workshop: *Neuropsychology of consciousness in honor to Professor Maria Pachalska*. Krakow, Poland: 28–30 May 2018.
- McGinn, C. (2003). Fear factor. *New York Times Book Review*, 23 February 2003, p. 11.
- McTaggart, J. (1934/1968). *Philosophical studies*. New York: Books for Libraries.
- Olszewski, H., Tłokiński, W., Łukaszewska, B. (2012). Otępienie czołowo-skroniowe – rzadziej spotykane warianty. W: M. Pączalska, L. Bidzan (red.), *Otępienie czołowo-skroniowe: ujęcie interdyscyplinarne* (s. 183–208). Kraków: Wydawnictwo KA AFM.
- Pachalska, M. (1999). *Afazjologia*. Cracow and Warsaw: PWN [in Polish, with English summary].
- Pachalska, M. (2002). The microgenetic revolution. Reflections on a recent essay by Jason Brown. *Neuro-psychoanalysis, An Interdisciplinary Journals for Psychoanalysis and the Neurosciences*. 4(1-2), 108-116.

- Pachalska, M. (2003). Neuropsychological rehabilitation for post-traumatic frontal syndrome in patients recovering from prolonged coma: A preliminary report. *Acta Neuropsychologica*, 1(2), 164-227.
- Pachalska, M. (2007). *Neuropsychologia kliniczna. Urazy mózgu*, T.1. Warszawa: Wydawnictwo Naukowe PWN.
- Pachalska, M. (2008). *Rehabilitacja neuropsychologiczna: Procesy poznawcze i emocjonalne*. Lublin: Wydawnictwo UMCS.
- Pachalska, M. (2017). Diagnostyka i rehabilitacja neuropsychologiczna osób z chorobami otępienymi. [w:] M. Cybulski, E. Krajewska Kułak, N. Waszkiewicz, K. Kędziora-Kornatowska (red.), *Psychogeriatrics* (s. 261-298). Warszawa: PZWL.
- Pachalska, M., Bidzan, L. (2012). *Otępienie czołowo-skroniowe: ujęcie interdyscyplinarne*. Kraków: Wydawnictwo KA AFM.
- Pachalska, M., Góral-Pórola, J., Mueller, A., Kropotov, J.D. (2017). *Neuropsychology and the neurophysiology of perceptual microgenesis*. *Acta Neuropsychologica*; 15 (4): 365-389.
- Pachalska, M., Kaczmarek, B.L.J. (2012). Alexander Romanovich Luria (1902 – 1977) and the microgenetic approach to the diagnosis and rehabilitation of TBI patients. *Acta Neuropsychologica*, 10(3), 341-369.
- Pachalska, M., Kaczmarek, B.L.J., Kropotov, J. D. (2014). *Neuropsychologia kliniczna. Od teorii do praktyki*. Warszawa. Wydawnictwo Naukowe PWN.
- Pachalska, M., Kaczmarek, B.L.J., Kropotov, J. D. (2018). *Neuropsychologia tożsamości. Ja utraczone i odzyskane*. Warszawa. Wydawnictwo Naukowe PWN.
- Pachalska, M. & MacQueen, B.D. (2001). Episodic aphasia with residual effects in a patient with progressive dementia resulting from a mitochondrial cytopathy (MELAS). *Aphasiology*, 15(6), 599-615.
- Pachalska, M. & MacQueen, B.D. (2002). The collapse of the US-THEM structure in aphasia: a neuropsychological and neurolinguistic perspective. (In:) Duszak A. (ed.), *Us & others* (pp. 481-503). Amsterdam: John Benjamins.
- Pachalska, M., MacQueen, B.D., Brown, J.W. (2012a). Microgenetic Theory: Brain and Mind in Time. In: R.W. Rieber (ed.), *Encyclopedia of the History of Psychological Theories*, XXVI (pp. 675-708). Frankfurt: Springer.
- Pachalska, M., MacQueen, B.D., Brown, J.W. (2012b), Microgenesis of Consciousness. In: H. Pashler (eds.), *Encyclopedia of the Mind*. (pp. 513-515). Los Angeles, London, New Delhi, Singapore.
- Papathanasiou, I. (2003). Nervous system mechanism of recovery and plasticity following injury. *Acta Neuropsychologica*, 1(3), 345-354.
- Pöppel, E. (1988). Time perception. In: *Encyclopedia of Neuroscience*, vol. 2. Boston: Birkhauser.
- Rohen, J.W. (2013). Functional anatomy of the brain. Paper presented at the Osteopathie Kongress: „Faszien und Osteopathie”. Berlin, Germany: 21–22 November 2013.
- Searle, J.R. (1998). *Mind, Language and Society: Philosophy in the Real World*. Cambridge: Cambridge University Press.
- Searle, J.R. (2008). *Philosophy in a New Century: Selected Essays*. Cambridge: Cambridge University Press.
- Wallesch, C.-W. (2004). The history of aphasia: Freud as an aphasiologist. *Aphasiology*, 18(4), 389-399.

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