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# Functional Anatomy, Physiology and Clinical Aspects of Basal Ganglia

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## 1. Introduction

Ensuring coordination of the nervous system functioning, communication between various structures, adjusting the functions to the changes in internal and external environment depends on processing of substantial amount of information (Groenewegen, 2007; Groenewegen & van Dongen, 2007).

The concept of cortico-subcortical loops is one of the explanations of the physiological control of the majority of motor, emotional and cognitive functions.

The most important elements are striatum and cerebral cortex. Especially in the pyramidal cells of the cerebral cortex and medium spiny neurons of the striatum there is capacity for plastic changes relating to the control of broadly defined mental functions (motor, emotional, cognitive).

The cerebral cortex is linked to the striatum via cortico-subcortical pathways, from where information is transmitted to the globus pallidus pars internalis or the substantia nigra pars reticulata (which physiologically and anatomically constitute one structure) or via the ventral globus pallidus reach the thalamus and the cerebral cortex subsequently.

The evidence of the anatomical and physiological brain research supported by clinical data and theoretical models suggests there are at least five loops (also called circuits) related to motor, emotional and cognitive functioning control (Alexander et al., 1986; DeLong et al., 1998). The loops division as well as the control of functions assigned to these loops has more model and didactic character rather than it reflects the real character and complexity of the functions controlling these loops.

The following cortico-subcortical loops have been described: 1. motor - between additional motor area of the cerebral cortex and the lateral part of dorsal striatum - putamen; 2. oculomotor - between the frontal visual eye field of the cerebral cortex and the corpus of the caudate (nucleus caudatus) belonging to the medial part of dorsal striatum; 3. prefrontal (associative) - between dorso-lateral prefrontal cortex and the dorso-lateral part of the head of caudate (nucleus caudatus) (the frontal part of the medial part of dorsal striatum); 4. latero-orbito-frontal - between lateral orbito-frontal cerebral cortex and the ventromedial part of the head of caudate (medial part of the dorsal striatum); 5. limbic (circuit of the anterior part of the cingulate gyrus) - between the anterior part of the anterior cingulate gyrus and the ventral striatum (of which the main part is the nucleus accumbens).

According to the classic description (Alexander et al., 1986) these circuits pass through various areas of cerebral cortex and subcortical structures and they have similar principles

of connections (Royall et al., 2002). As shown in Fig. 1, it was conventionally assumed that individual circuits pass through particular areas of the cerebral cortex, dorsal and abdominal striatum and thalamic nuclei, from where the information reaches the cerebral cortex. Moreover, structurally and functionally related areas of the anterior cerebral cortex and striatum are linked to the posterior parts of the cerebral cortex, e. g. the associative circuit processes information from the dorso-lateral prefrontal cortex and from premotor and posterior part of the parietal cerebral cortex (ibid.) (the areas responsible for the control of motor and spatial functions (ibid.)). The fronto-subcortical loops leaving various, distantly located from one other, regions of the cerebral cortex converge in relatively small, limited areas of the target basal ganglia, thalamic nuclei and frontal cerebral cortex (ibid.). Classic hypothesis that individual cortico-subcortical circuits are functionally separated and act simultaneously and independently (Alexander et al., 1986), does not explain the complexity of the nervous system functioning and is not confirmed by the results of investigations and clinical observations of the nervous system damages and disturbances in the loop functioning caused by mental disorders. The complex nature of cortico-subcortical loops functioning is most likely the result of functional connections between the loops. This would explain the control of functions related to integration and convergence of the information processed (Percheron & Fillion, 1991; Joel & Weiner, 1994; Parent & Hazrati, 1995).

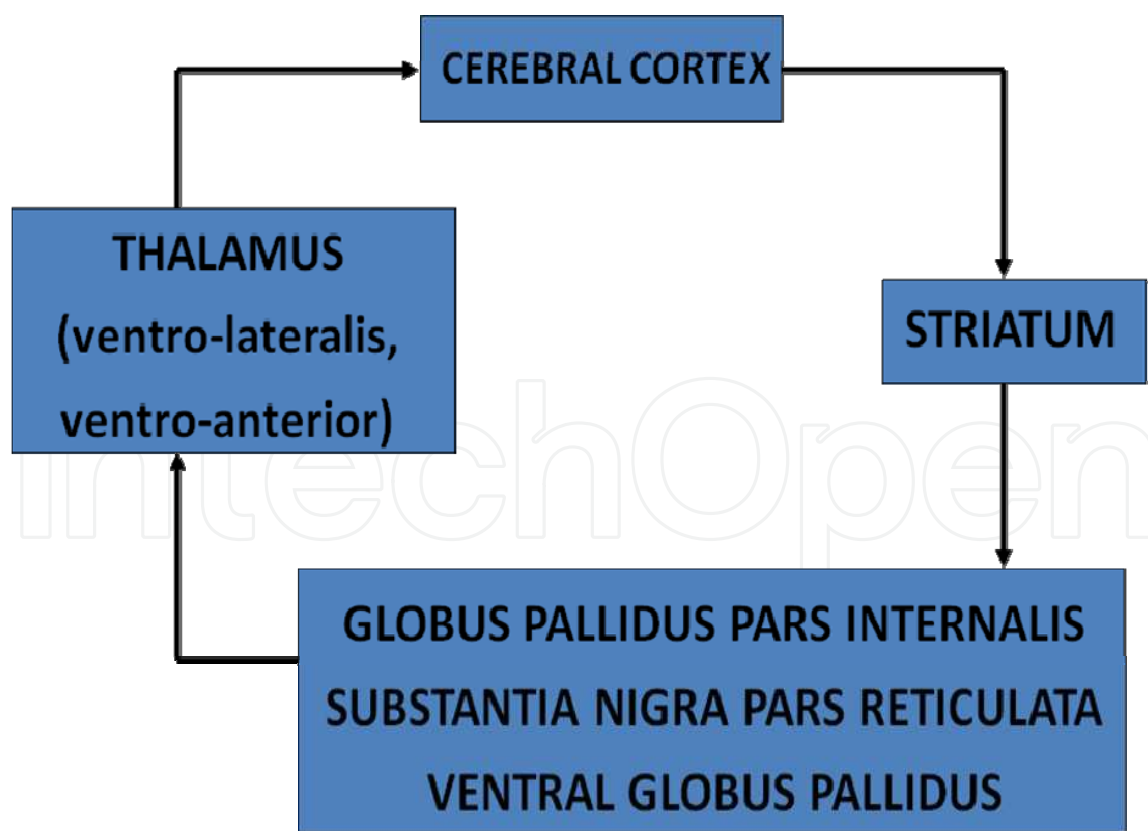


Fig. 1. General scheme of cortico-subcortical loops.

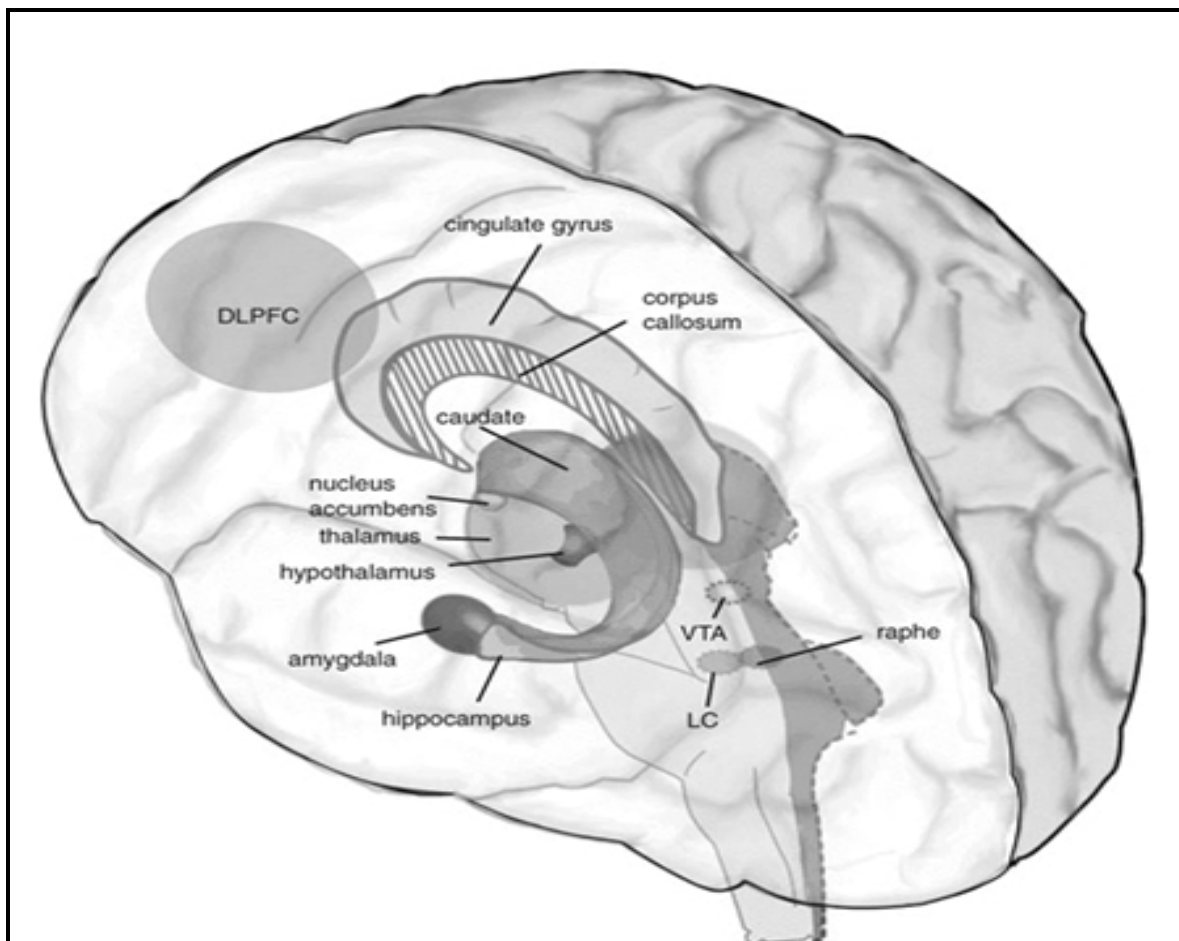


Fig. 2. The circuits connecting the basal ganglia with the cerebral cortex (Stahl, 2008).

The basal ganglia are connected not only with the motor areas of the cerebral cortex but they also influence the areas that are responsible for operating memory and executive functions (dorso-lateral prefrontal cortex and the anterior part of the gyrus cinguli) (Alexander et al., 1986; Middleton & Strick, 1994; Smith & Jonides, 1999; Hartley & Speer, 2000; Elliott, 2003). Prefrontal (associative), latero-orbito-frontal and limbic loops are particularly essential in the control of executive functions (Alexander et al., 1986; Royall et al., 2002; Haber, 2003).

Impairment of prefrontal loop functioning causes disturbances of verbal and spatial operating memory and executive functions (the choice of the aim, planning, accepting and changing of cognitive attitude, self-control and metacognition), involve, in particular, difficulties in executing tests such as Wisconsin Card Sorting Test, WCST) (Fuster, 1980; Cummings, 1995; Fuster, 1995; Goldman-Rakic, 1995a; Milner, 1995; Truelle et al., 1995).

The orbito-frontal circuit is most probably responsible for socially adjusted behaviour and hampering not socially accepted one (Cummings, 1995; Truelle et al., 1995). Efficient functioning of this circuit is of importance for the estimation of the risk of the behaviour chosen (Rogers et al., 1999; Schiffer & Schubotz, 2011). The choice between behaviour in which the probability of a reward is large though the reward is small and behaviour in which the probability of a reward is small but the reward is significant depends on the activity of the inferior and preorbital areas of the prefrontal cortex. The damage to the orbital areas of the prefrontal cortex impairs "go/do not go" type tasks execution of in animals (Thorpe et al., 1983) and people (Drewe, 1975; Bunzeck et al., 2011).

		LOOPS				
loop unit		MOTOR	OCULOMOTOR	DORSO-LATERAL PREFRONTAL	LATERAL ORBITO-FRONTAL	LIMBIC (anterior cingulate)
cerebral cortex		premotor area primary motor cortex somatosensory cortex additional motor area	dorsolateral prefrontal cortex posterior parietal cortex frontal oculomotor area	posterior parietal cortex premotor area dorsolateral prefrontal cortex	superior and inferior temporal gyrus anterior part of the gyrus cinguli lateral orbitofrontal cortex	hippocampal cortex entorhinal cortex superior and inferior temporal gyrus
			CAUDATE (caudate nucleus)			
striatum		PUTAMEN	body	head		ventral striatum
			middle part	dorso-lateral part	ventro-medial part	
pallidum	globus pallidus pars internalis	ventro-lateral	dorso-medial (posterior part)	dorso-medial (lateral part)	dorso-medial (middle part)	anterio-lateral
	substantia nigra pars reticularis	postero-lateral	ventro-lateral	anterio-lateral	anterior-medial	anterio-lateral
	ventral globus pallidus					ventral globus pallidus
thalamus	nucleus					
	nucleus ventralis anterior <sup>2</sup>		pars magnocellularis (lateral part) <sup>6</sup>	pars parvocellularis	pars magnocellularis (medial part)	
	nucleus ventralis lateralis <sup>1</sup>	anterior part (rostral) <sup>4</sup> medial part <sup>5</sup>				
	anteriors (group)					
	nucleus medialis dorsalis <sup>3</sup>		pars paralamellaris <sup>7</sup>	pars parvocellularis	pars magnocellularis	postero-medial part

<sup>1</sup>(nucleus ventralis lateralis) = (nucleus ventralis intermedius)  
<sup>2</sup>(nucleus ventralis anterior) = (nucleus ventralis anteromedialis)  
<sup>3</sup>(nucleus medialis dorsalis) = (nucleus medialis)  
<sup>4</sup>(nucleus ventralis lateralis (medial part (rostral) = (nucleus ventrolateralis pars oralis)  
<sup>5</sup>(nucleus ventralis lateralis - medial part) = (nucleus ventrodorsalis pars medialis)  
<sup>6</sup>(nucleus ventralis anterior pars magnocellularis) = (nucleus anterior pars magnocellularis)  
<sup>7</sup>(nucleus medialis dorsalis pars paralamellaris (most lateral) = (nucleus medialis dorsalis pars paralamellaris)

Table 1. Five basalo-thalamo-cortical loop (Mink 1999; Smith et all., 2004; Bochenek & Reicher 2006; Laskowska et all., 2008, Laskowska & Gorzelańczyk, 2009).



It was shown that the anterior cingulate loop is responsible for correcting behaviour following a mistake (Peterson et al., 1999). During the Stroop Interference Color Test, which consists of inhibition of answers learned while choosing opposing answers, the activity of the anterior part of cingulate gyrus and its connections with the central part of the frontal cerebral cortex increases (ibid.).

Motor, emotional and cognitive functions are controlled by two neuronal pathways, being the part of cortico-subcortical loops: direct and indirect (Fig 1). These pathways are under control of connections of the nigrostriatal system of zona compacta nigra (Mandir & Lenz, 1998). The striatum is connected with the thalamus by pathways going through the internal part of the globus pallidus and the reticular part of the substantia nigra. The direct pathway runs from the striatum through the medial part of the globus pallidus, the reticular part of the substantia nigra and the ventral part of the globus pallidus to the thalamus (this causes activation of inhibitory neurons of the thalamus and in consequence activates the cerebral cortex) and further to the cortex of the brain (Longstaff, 2003; Morgane et al., 2005). The indirect pathway runs through the lateral part of the globus pallidus and reaches the subthalamic nucleus (inhibiting glutaminergic transmission of the subthalamic nucleus), from where axons reach the medial part of the globus pallidus and further the thalamus and the brain cortex (ibid.). Control of the stimulation of the cerebral cortex is held, among the others, by striatal neurons (medium spiny neurons) (ibid.). The axons of glutaminergic neurons from the cerebral cortex reach the medium spiny neurons (cortico-striatal pathway) (ibid.). The axons of striatal medium spiny neurons release gamma-aminobutyric acid (GABA) inhibiting activity of the globus pallidus (both: internal and external). Striatal spiny neurons, which are morphologically indistinguishable, can be divided into two populations: 1) medium spiny neurons with D1 receptors containing P substance (SP) and dynorphin (DYN) (GABA/D1/SP/DYN), reach the internal part of the globus pallidus (direct pathway) (Mink, 1999; Longstaff, 2003). 2) medium spiny neurons with D2 containing enkephalin (ENK) (GABA/D2/ENK), reach the external part of the globus pallidus (indirect pathway). Stimuli from the substantia nigra pars compacta neurons of nigrostriatal pathway are transmitted to both populations of the striatal medium spiny neurons. The nigro-striatal pathway increases the activity of the direct pathway and inhibits the activity of the indirect pathway (Mink, 1999; Groenewegen, 2003; Longstaff, 2003; Morgane et al., 2005). Dopamine released in the axon terminals of the nigro-striatal pathway causes in GABA /D1/SP/ DYN striatal medium spiny cells an increase, and in GABA/D2/ENK cells a decrease, in the concentration of the second transmitter: 3'5' - cyclic adenosine monophosphate (cAMP) (ibid.). The activity of the cerebral cortex is proportional to the concentration of cAMP (ibid.).

Glutaminergic neurons of the subthalamic nucleus (indirect pathway) stimulate the external part of the globus pallidus, simultaneously reducing activity of the thalamus and cerebral cortex neurons. The striatum inhibits the neurons of the lateral part of the globus pallidus, what leads to disinhibition of glutaminergic cells of the subthalamic nucleus and stimulation of the globus pallidus pars interna. Dynamic changes of the activity of direct and indirect pathway make it possible to stimulate well defined areas of the cerebral cortex with simultaneous inhibition of the areas, which do not take part in execution of particular movement or mental action (Mink, 1999; Groenewegen; 2003).

The damage of various structures of basalo-thalamo-cortical loop can manifest with symptoms related to a definite loop (Alexander, 1986; Royall et al., 2002). According to this argumentation it can be assumed that pathology of the basal ganglia can cause symptoms

typical for the damage of a particular loop or cause typical symptoms for the damage of several of them (ibid.). The symptoms, being the consequence of, definitely localized in the brain, damages of the particular structures of cortico-subcortical loops, can overlap with the symptoms from different areas of encephalon connected with a specific functional system, which is not a part of this system (ibid.). The conceptual model of basalo-thalamo-cortical connections can be helpful in interpretations of the symptoms of mental disorders relating to basal ganglia pathologies, for example in Parkinson's disease (Tröster & Arnett, 2005).

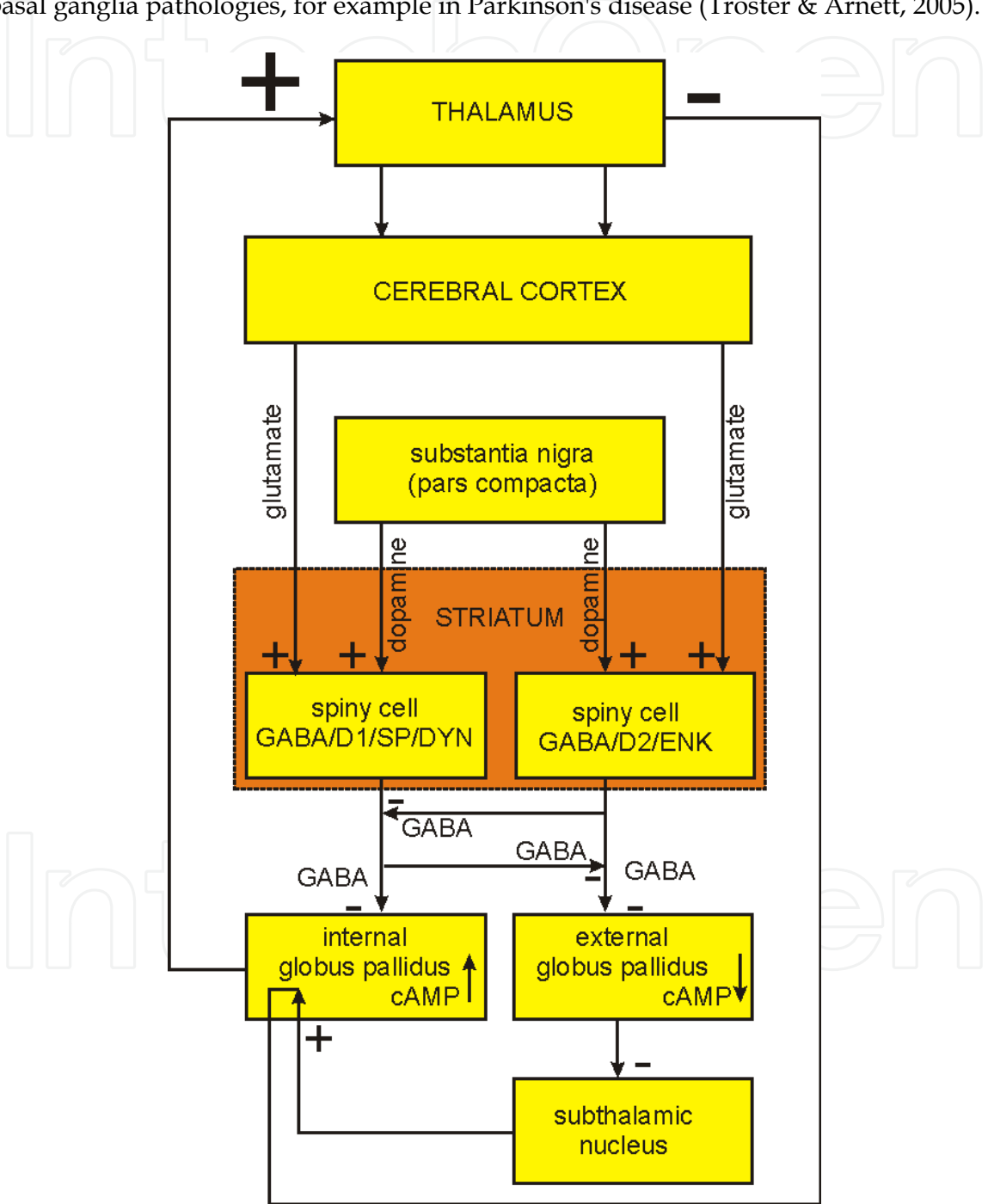


Fig. 3. The original conceptual model of the neuronal loop connecting the internal globus pallidus (GPI), subthalamic nucleus (STN) and thalamus with the cerebral cortex compiled on the basis of the literature (Fix, 1997; Longstaff, 2003; Groenewegen, 2003).

## 2. Motor loops (motor and oculomotor)

Motor control of skeletal muscles relates to the motor loop (motor circuit) and the oculomotor loop (oculomotor circuit) (ibid.). The dorso-medial prefrontal loop, orbito-frontal loop and the anterior part of the cingular gyrus loop are associated with the control of cognitive and emotional functions (ibid.).

The motor circuit is responsible, *inter alia*, for automatic motor activity connected with maintenance of body posture and reflexes (Fix, 1997), as well as for the control of muscular tension. The motor loop plays an essential role in initiating and fluent performing of motor actions executed by skeletal muscles especially during will dependent movements. The disorders of this loop can cause muscular stiffness, bradykinesia, akinesia and hypokinesia (e.g. in Parkinson's disease and parkinsonian syndrome) or excessively large and uncontrolled movements of limbs (e.g. Huntington's chorea, ballism) (Fix, 1997).

The oculomotor loop participates in the control of saccadic eyeball movements. Efferent connections to the superior colliculus (Sc) from the cortical areas of the brain and subcortical nuclei, especially the reticular part of substantia nigra (SNr) make it possible to control rapid eyeball movements through the inhibition of movements disturbing the execution of a task (Hikosaka, 2000). Presumably the neurons of ventro-lateral part of substantia nigra pars reticularis and caudate nucleus play essential role in external eyeball muscles movements, both through the neurons in which information on previously executed movements is remembered (memory-guided saccades), as well as neurons reacting on currently incoming visual stimuli (visually-guided saccades) (ibid.). In the result of oculomotor loop damage visual fixation can be impaired, and unilateral neglect syndrome, as well as attention deficits can be observed especially in the tasks requiring rapid movements targeted at stimuli (Hikosaka, 2000). The shortage of functions of external eyeball muscles caused by damages of basal ganglia (e.g. in Parkinson's disease, Huntington's disease) can impair saccadic movements of eyeballs depending on previously remembered information. In persons with basal ganglia disorders dysfunctions in intentional inhibition of eye movements, triggered by visual stimuli (ibid.), were observed.

## 3. Dorsolateral prefrontal loop

The dorsolateral prefrontal loop is responsible for the choice of aims, planning, programming of the sequence of mental actions and behaviours, switching between sentences (the ability to change attitude flexibly), verbal and spatial working memory, self-control and metacognition (self-consciousness) (Royall et al., 2002). The disorders of the loop functions can lead to incorrect order of linguistic behaviours what results in verbal fluency reduction (DeLong & Wichmann, 2007).

## 4. Lateral orbitofrontal loop

The lateral orbitofrontal circuit takes part in initiating social behaviours motivated by an award and in inhibiting behaviours, which can trigger punishment (Royall et al., 2002). Incorrect functioning of this circuit may result in disinhibition of behaviours, personality changes, lack of control and emotional liability, as well as irritability and gaiety (DeLong & Wichmann, 2007). The damage of this loop can cause perseverations, which make it difficult to process information from external environment and adaptation of behaviours to a particular situation (Royall et al., 2002).



## 5. Anterior cingulate circuit

The anterior cingulate circuit (limbic loop) is important in behavior control and adaptation of behaviours after making a mistake (*ibid.*). The damage of this circuit results in emotional disorders especially deep apathy and lack of spontaneity. Lowered mood is accompanied by weakening of affect and motor adynamy (*ibid.*).

On the basis of a pattern of basal ganglia connections, being a part of particular loops with cerebral cortex, similarity of motor and emotional functions can be deducted (Alexander et al., 1990). Information processed by various loops partly overlap in the striatum (*ibid.*). In the globus pallidus pars internalis and the substantia nigra pars reticularis and subsequently in the thalamus pieces of information from various circuits converge. Pieces of information from the thalamus reach limited areas of the cerebral cortex, which control motor, emotional and cognitive functions (*ibid.*). Such functional organization makes it possible to select motor and mental actions depending on information incoming from external and internal environment (Mink, 1999; Morgane et al., 2005; Groenewegen & Dongen, 2007).

The circuits, described above, functionally connect the basal ganglia with the cerebral cortex (Alexander et al., 1986; DeLong et al., 1998; Elliot, 2003; Haber, 2003; Saint-Cyr et al., 2003; Morgane et al., 2005; Olzak & Gorzelańczyk, 2005; Groenewegen & van Dongen, 2007; Laskowska et al., 2008; Haber et al., 2009). The assumption that the circuits connecting the cerebral cortex with the basal ganglia work independently and in a parallel way (Alexander et al., 1986, 1990; DeLong et al., 1998) has expired. Various consequences of the damages of particular loops activity depend on the cerebral cortex areas with which they connect and on the circumstances in which they are activated. The degree of co-operation of particular basalo-thalamo-cortical loops is not known (Longstaff, 2006; Laskowska et al., 2008; Haber et al., 2009; Sadikot et al., 2009), however more and more data indicate that the exchange of information between particular circuits takes place (McFarland et al., 2002; Groenewegen & van Dongen, 2007; Laskowska et al., 2008; Haber & et al., 2009; Sadikot et al., 2009).

Subcortical nuclei relate not only to motor control, but also to the processes of reminding as well as executive functions processes, short-term memory, the analysis of mutual setting of objects and to undertaking actions (Ślawek et al., 2001; Frank et al., 2001; Royall et al., 2002; Elliot, 2003; Haber, 2003; Laskowska et al., 2008; McNab et al., 2008; Haber et al., 2009).

The basal ganglia take part in the control of motor, emotional and cognitive behaviours by two pathways (indirect and direct) exerting contradictory effect on the stimulation of the thalamus and the cerebral cortex. (Albin et al., 1989; DeLong, 1990; Obeso et al., 1997, 2000b; Ślawek, 2003; Sobstyl et al., 2003; Groenewegen, 2003; Longstaff, 2006; DeLong & Wichmann, 2007; Groenewegen & van Dongen, 2007; Szolna, 2007). In the nigrostriatal pathway there are two kinds of dopaminergic receptors relating to striatal medium spiny neurons: D1, which activate GABA-ergic neurons of the putamen in the direct pathway; and D2, whose stimulation inhibits GABA-ergic neurons of the putamen in the indirect pathway (*ibid.*).

The direct pathway relates to striatal medium spiny neurons (SP/DYN) releasing GABA and inhibiting GABA-ergic neurons running from GPi and SNpr to the thalamus. Cortical stimulation of this pathway causes activation of thalamus neurons (Groenewegen, 2003; Sobstyl et al., 2003; Longstaff, 2006; Groenewegen & van Dongen, 2007; Szolna, 2007). The direct pathway connects the putamen, where the connections from the cerebral cortex and the substantia nigra (SNpc) meet, with the exit structures - the internal part of the globus pallidus and reticular part of substantia nigra pars reticulata - (SNpr) (*ibid.*).

The direct pathway is connected with striatal medium spiny neurons (ENK) secreting GABA and it leads through the external part of the globus pallidus (GPe) to STN, which stimulates inhibitory neurons in GPi and SNr structures. Cortico-striatal stimulation of the indirect pathway decreases thalamus stimulations and inhibits motor cerebral cortex (Albin et al., 1989; DeLong, 1990; Obeso et al., 1997, 2000b; Herrero et al., 2002; Sobstyl et al., 2003; Longstaff, 2006; Groenewegen & van Dongen, 2007).

Dopamine is a neurotransmitter in SNc neurons (Groenewegen, 2003; Sobstyl et al., 2003; Groenewegen & van Dongen, 2007). During repose occasional discharges of small frequency, which do not affect movement, appear in these neurons (Longstaff, 2006). The frequency of neurons firing changes when a stimulus rewarding a movement is present (ibid.). They influence the reply of striatal medium spiny neurons to the stimulation from the cerebral cortex. GABA/SP/DYN neurons increase responsiveness in the direct pathway, however GABA/ENK neurons reduce responsiveness in the indirect pathway due to the SNc influence. As a result, the nigro-striatal connection increases the activity of the direct pathway, inhibiting the indirect pathway (ibid.).

The basal ganglia are responsible, inter alia, for executing motor sequences (Alexander et al., 1990; Mink, 1999; Sadowski, 2001; Herrero et al., 2002; McFarland et al., 2002; Longstaff, 2006; Haber et al., 2009). Each sequence is represented by striatal medium spiny neurons creating motor or oculomotor microloops in the cortico-subcortical circuit, which can be activated or inhibited (Alexander et al., 1986; Longstaff, 2006). A considerable part of striatal medium spiny neurons has low frequency of electric discharge at rest (0,1-1Hz). In other subcortical nuclei - GPi and SNr, the frequency of electric discharge at rest is considerably higher and it amounts to about 100Hz (ibid.). According to a contemporary model of motor control carried out by the basal ganglia, movements are initiated by activation of the motor cerebral cortex, which consequently activates the striatum (Groenewegen, 2003; Sobstyl et al., 2003; Longstaff, 2006; Groenewegen & van Dongen, 2007; Sadikot et al., 2009). The activity of striatal medium spiny neurons increases during the execution of movements being a result of functioning of cortico-striatal neurons (Longstaff, 2006). When a discharge frequency of GPi and SNr decreases, a reduction in the thalamus inhibition follows making it possible to execute a movement or other mental action (Albin et al., 1989; DeLong et al., 1990; Obeso et al., 1997, 2000; Sobstyl et al., 2003; Longstaff, 2006).

Motor, emotional and cognitive functions are controlled by the system of structures and connections of the central nervous system, among which three levels of co-operation were distinguished: lower, middle and upper (Schotland & Rymer, 1993; Longstaff, 2006). Experimentally induced damages of the nervous system centers in animals result in the lack of the function subjected to the place of the damage and the appearance of new or enhancement of until now executed actions. A damage of the motor center of the cerebral cortex causes limbs paresis, an increase in muscles tension, intensification of tendinous reflexes in these limbs as well as appearance of Babiński's reflex (Babinski, 1986). The occurrence of such symptoms indicates that lower in hierarchy structures are inhibited by higher ones and that the lack of this inhibition causes unblocking of the physiologically inhibited centre. Clinical observations of persons with the basal ganglia damages indicate that these structures do not initiate movements, but they control the course of skilled sequence of movements (ibid.). An increase in the activity of the direct pathway increases, and of the indirect pathway decreases, stimulation of particular areas of the cerebral cortex (Groenewegen, 2003; Longstaff, 2006; Groenewegen & van Dongen, 2007; Szolna, 2007).

Cooperation between the direct and indirect pathways changes the activity of the cerebral cortex in such a way that the activity increases in the areas which control execution of a particular motor action and inhibits the areas which are not involved in the executed activity (ibid.). Planning of motor activity relates to the functioning of the cerebral prefrontal cortex controlling the process of scheduling and execution of a particular sequence of movements (LeDoux, 1996; Groenewegen, 2003; Longstaff, 2006; Groenewegen & van Dongen, 2007). The control of implementation of the actions scheduled is possible when the centers of the cerebral cortex and basal ganglia (especially the caudate) cooperate properly (Alexander et al., 1986; LeDoux, 1996; Herrero et al., 2002; Groenewegen & van Dongen, 2007). The nucleus caudatus – a part of the ventral striatum is connected with a substantial part of the frontal, mainly with the associative cerebral cortex (ibid.). Pieces of information incoming from the cerebral cortex to the caudate make it possible to control and implement a particular sequence of movements (ibid.). A program relating to the implementation of a specific sequence of movements is sent from the caudate through the thalamus to the premotor prefrontal cerebral cortex, which controls voluntary movements (ibid.). The basal ganglia in connection with ventral nuclei of the thalamus and frontal areas of the cerebral cortex play an essential role in instrumental conditioning (Petri et al., 1994; Smith's Petri et al., 2006; Rueda-Orozco et al., 2008). The results obtained in investigations on animals confirm the meaning of the basal ganglia in the procedural memory (Petri et al., 1994; Akhmetelashvili et al., 2007; Hartman et al., 2008; Rueda-Orozco et al., 2008).

The pattern of basal-thalamic-cortical connections carrying out motor, emotional and cognitive functions is mutual for these functions (Alexander et al., 1996; DeLong et al., 1998; Herrero et al., 2002; Groenewegen, 2003; Saint-Cyr, 2003; Groenewegen & van Dongen, 2007; Laskowska et al., 2008), and the control of these functions consists in the choice of the sequence of behaviours adapted to a particular situational context (Heilman et al., 1993; Royall et al., 2002; Longstaff, 2006).

The control of motor, emotional and cognitive functions is hierarchical and the processing of information connected with these functions can be executed sequentially or simultaneously in various structures of the brain. Clinical observations and the results of the investigations carried out in persons with a damage of the basal ganglia confirm the participation of subcortical structures not only in motor but also cognitive functions (Oberg and Divac, 1979; Berns and Sejnowski, 1995; Frank et al., 2001). Also the results of computer simulations of cognitive actions such as: maintenance of information in operating memory, remembering, planning of sequences of behaviors and making decisions indicate a participation of the basal ganglia in the control of cognitive functions (Prescott et al., 2002; Jessup et al., 2011). The function of the basal ganglia in the control of motor, emotional and cognitive functions relates to cooperation with frontal cerebral cortex areas (Brown et al., 1997). The basal ganglia cooperating with the prefrontal cerebral cortex make it possible to initiate motor, emotional and cognitive actions. A complex system of stimulations (inhibition and disinhibition) limits the flow of excessive information which disables the initiation of an activity. A complex choice mechanism makes it possible to initiate a particular kind of movement without detailed determination of the whole sequence of the movements executed (Bullock & Grosberg, 1988; Hikosaka, 1989; Chevalier & Deniau, 1990; Passingham, 1993).

According to the concept of mental (motor, emotional, cognitive) functions control carried out by cortico-subcortical loops a proper action of all the structures of these loops is a condition necessary for their efficient functioning (Brown et al., 1997). Mental processes,

such as for example: making decisions, the choice of motor action, the change of behavior, working memory can be disturbed in a similar degree, independently from which structure of a particular loop is damaged (*ibid.*). Because of a specific structure of striatal medium spiny neurons (having a very large quantity of synaptic connections) and synaptic processes like long-term synaptic potentiation (LTP) and long-lasting synaptic depression (LTD), similar to those carried out in the cerebral cortex, the striatum (including the ventral striatum) is functionally the main structure of cortico-subcortical loops (Picconi et al., 2005) that controls motor, emotional and cognitive functions. Due to numerous inter-striatal and cortico-striatal connections and described above plastic mechanisms enabling enhancement or reduction of stimulations, the striatum together with the cerebral cortex controls the functions of particular cortico-subcortical loops, substantially influencing mental processes (Brown et al., 1997; Prescott et al., 2002).

The results obtained from the experiments on a model of shoulder movement simulation indicate that the higher dopamine concentration is in the striatum the shorter is the time necessary to initiate a movement (*ibid.*). This means that the time necessary to initiate a movement becomes shorter simultaneously with an increase of the striatum stimulation (*ibid.*). Moreover, the simulation of a decrease in dopamine concentration in the striatum in the same model causes bradykinesia and akinesia, similar to that observed in the Parkinson's disease (Prescott et al., 2002). The key function of the striatum is to produce signals which reach, through the thalamus, the cerebral cortex (*ibid.*). It was suggested that the choice of a particular pattern of action takes place in the striatum due to so called gating mechanism. Activation of the neurons responsible for processing of a particular pattern leads to inhibition of other striatal neurons. The mechanism makes it possible e.g. to process efficiently information introduced to the working memory with simultaneous inhibition of the inflow of new information before completing a presently executed task (Frank et al., 2001). If the gate is closed, new information does not influence the memory essentially and that is why it allows a stable maintenance of information being processed (*ibid.*). Opening of the gate enables data updating (*ibid.*). The inhibition of new information inflow prevents their interference with previously gathered data (*ibid.*). This mechanism enables continuous selection of the processed information optimizing in this way execution of mental actions (*ibid.*). The disturbance of the mechanism of selective actualization of data in proper time (both too frequent and too rare data updating) increases the number of errors made in various tasks. This disorder relates to initiation of both movement and thought processes. A confirmation of the concept equity is psychic akinesia observed in persons with a damage of the striatum (Brown and Marsden, 1990). However, inhibition of the internal part of the globus pallidus leads to disinhibition of frontal loops activity, which results in continuous updating of data. Continuous updating of data is caused by the lack of blocking in access to the working memory (Frank et al., 2001). Results of these disorders can be Absent-mindedness, impulsivity and hyperactivity (occurring in the disorders such as: Huntington's disease, Tourette syndrome, attention deficit hyperactivity disorder) can be the results of the above mentioned disorders (*ibid.*). Diminishing of dopamine concentration in the striatum causes that the gate regulating the access to the working memory is opened and closed in the wrong time, and in permanent inhibition of the internal part of the globus pallidus it is opened all the time (*ibid.*).

The gating model does not fully explain the selection of data, which arises from the fact that dopamine is secreted in large areas of the prefrontal cortex. In this model, different areas of the prefrontal cortex are activated simultaneously, making it impossible to select one



particular piece of information (ibid.). This mechanism does not meet the functional requirements of selective updating of data, which is, as explained above, a simultaneous updating of certain information and maintaining the remaining data unaltered (ibid.).

The gating model is complemented by the concept of selective gating mechanism based on the functioning of parallel basal-thalamic-cortical loops (Frank et al., 2001). Inhibitory nature of the connections leading from the striatum to the internal part of the globus pallidus (GPi), the reticular part of the substantia nigra (SNr) and the thalamus has a decisive influence on the processes of gating. For this reason, the activity of neurons in the striatum causes the stimulation of neurons in the thalamus (via dual inhibition) (Deniau & Chevalier, 1985, Chevalier & Deniau, 1990). Disinhibition of the thalamus evokes gating function that enables emergence of other functions, although it does not induce them directly.

Selective gating mechanism explains the role of the basal ganglia in the initiation of the process of collecting new information in the working memory and the possibility of its inflow of new information before completing a presently executed task (Frank et al., 2001). If the fast update (Frank et al., 2001). In the absence of stimulation of striatal neurons the gate remains closed and the frontal cerebral cortex maintains the information currently being processed. This is possible due to existence of multiple parallel loops (ibid.).

In a classical division five parallel basal-thalamic-cortical loops were proposed (Alexander, 1986). However, on the basis of anatomical characteristics, existence of many subloops inside this five basic circuits was assumed. Due to this a relatively precise control of the working memory is possible (Beiser & Houk, 1998). It was assumed that control of the working memory depends on maintaining constant stimulation of neurons of the prefrontal cortex (Frank et al., 2001), which is the result of a continuous self-stimulation via a feedback loop (ibid.). Maintaining of this state is possible due to active feedback connections between the neurons of the frontal cerebral cortex and the thalamus. (ibid.).

The importance of the basal ganglia in senso-motor processes consists in the fact that they decide on the choice of a particular activity (Prescott et al., 2002). In the computational model, which was based on the observation of two different, competing with each other behaviors, it is assumed that coded is only one of them (i.e. the one whose execution by the motor system is being currently more important) (Redgrave et al., 1999). At the cellular level, a complex choice mechanism that allows solving a conflict between competing behaviors through fast and decisive switch between pre-selected actions, may be related to the degree of polarization of the neuron cell membrane. Resting membrane potential of striatal spiny neurons fluctuate from the values close to depolarization state so-called "up" state to hyperpolarization - "down" state (Calabresi et al., 2007). The selection mechanism consists of four steps: 1) selection of the cells in "up" state and exclusion of the cells in "down" state; 2) local inhibition within the striatum, which selectively increases the probability of stimulation of some spiny neurons and decreases the probability of stimulation of the other ones (accordingly to the cognitive model it increases the probability of the flow of information through specific channels (perceived as groups of spiny cells) (Redgrave et al., 1999); 3) localized inhibition induced by striatal medium spiny neurons (D1) together with dispersed stimulating impulses, incoming from the subthalamic nucleus, which act like a feed- forward loop, controlling the information coming out of the basal ganglia (from the internal globus pallidus and the substantia nigra reticularis); 4) local mutual inhibition occurring in the output basal ganglia (thalamus), which additionally restricts the selection criteria (ibid.).

According to the described above hypothesis on action selection, an alternative explanation for the functional organization of the basal ganglia was proposed. Instead of traditionally selected pathways (*direct* and *indirect*) the existence of neuronal circuits responsible for the choice (called selection circuit) and control (control circuit) of the activities undertaken is postulated. The selection circuit (traditionally called: direct pathway), leading from D1 dopamine receptors of striatal medium spiny neurons to the entopeduncular nucleus (EP) (or internal globus pallidus in primates) and a reticular part of the substantia nigra pars reticularis (SNr), receiving also the stimulation from the subthalamic nucleus reaching the EP and SNr, creates a selection mechanism acting as the feedforward selection circuit, which allows, on the basis of information coming into the system, the choice of motor or cognitive schema, before it starts to be executed. In this perspective, the neural connections of the striatum with the globus pallidus (Gp) and the nucleus subthalamicus (traditionally called indirect pathway) are included in the loop responsible for the choice control (control circuit) (Gurney et al., 1998, Prescott et al., 2002). Based on the analysis of the results of the experiments simulating the described above way of the striatum function regulation, two functions of the control loop were distinguished: 1) inhibition of STN by the GP using negative feedback allows to regulate the number of impulses outgoing from STN through the channels of information flow (associated with the activity of striatal medium spiny cells) (ibid.); 2) inhibition of EP/SNr by the GP as a part of a selection support mechanism. The increase in concentration of dopamine in the striatum facilitates the choice of information channels, which will be disinhibited, while the decrease in concentration of dopamine hinders the choice (ibid.). The reason of errors occurring in the selection of behaviors adjusted to a particular situation or difficulties in completing already chosen behaviors in the Parkinson's disease, in which concentration of dopamine in the nigrostriatal pathway is decreased, are tried to be explained with the above presented model (Prescott et al., 2002).

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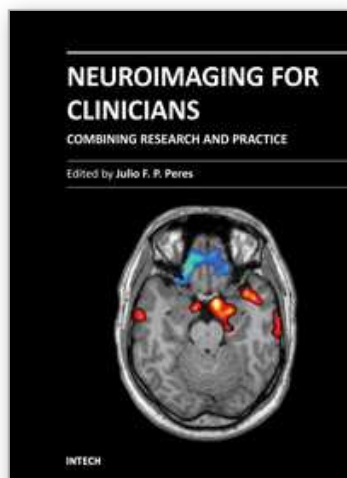
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Neuroimaging for clinicians sourced 19 chapters from some of the world's top brain-imaging researchers and clinicians to provide a timely review of the state of the art in neuroimaging, covering radiology, neurology, psychiatry, psychology, and geriatrics. Contributors from China, Brazil, France, Germany, Italy, Japan, Macedonia, Poland, Spain, South Africa, and the United States of America have collaborated enthusiastically and efficiently to create this reader-friendly but comprehensive work covering the diagnosis, pathophysiology, and effective treatment of several common health conditions, with many explanatory figures, tables and boxes to enhance legibility and make the book clinically useful. Countless hours have gone into writing these chapters, and our profound appreciation is in order for their consistent advice on the use of neuroimaging in diagnostic work-ups for conditions such as acute stroke, cell biology, ciliopathies, cognitive integration, dementia and other amnesic disorders, Post-Traumatic Stress Disorder, and many more

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