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Kierownik: prof. dr hab. Wiesław Kawiak

Joanna IŁŻECKA

New Opinions of Pathomechanisms of Involuntary Movements

Nowe poglądy na patomechanizmy ruchów mimowolnych

Involuntary movements (hyperkineses) are the result of unintentional contractions of transversely striated muscles. Most often they are concomitant to the organic diseases of the central nervous system, chiefly of extrapyramidal system (10). Pathological hyperkinesia may be a result of disturbances of inhibitory neurons activity, coming out of the new striatum into the globus pallidus and black substance, which causes an excessive stimulation of the subsequent, lower system of neurons (4). Hyperkineses occur more seldom in other systems' diseases, they may also have psychogenic base (10).

Clinical division of involuntary movements: spasms, cramps, fasciculations, myokimias, myoclonus, tremor, chorea, athetosis, ballism, dystonia, tics (11).

Choreic movements are quick, irregular, extensive movements of limbs, facial grimaces distortedly imitating free movements. The occurrence of choreic movements testifies to the existence of disorders in the balance between dopamine, γ-aminobutyric acid, serotonine and acetylcholine. γ-aminobutyric acid is secreted by big cells of the caudate nucleus being under the inhibitory influence of the system, in which acetylcholine is the transmitter, and under the stimulating influence of the impulses from the neurons of the striatum small cells. The atrophy of small cells may lead to disturbances in releasing γ-aminobutyric acid. The deficiency of this neurotransmitter causes the release of lower motor centres from the inhibitory influence of the striatum (9). The share of membranous phospholipids affecting the affinity to the places of binding γ-aminobutyric acid has been suggested. The increased level of phospholipids may cause accessibility of γ-aminobutyric acid to the receptors (2).

Degeneration of the striatal cholinergic neurons causes functional prevalence of the undamaged system, in which dopamine is the transmitter. The role of acetylcholine in pathophysiology of choreic movements is to consist, at least partly, in the action it exerts on dopamine metabolism in the nuclei of the base (9).

Choreic movements may be caused by hypersensitivity of the striatum neurons to the normal quantity of dopamine inflowing through nigrostriatal tract. They may be also the result of the functional prevalence of the tracts leading from mesencephalon to the globus pallidus (9).

It is assumed that there are endogenous neurotoxins inducing pathomorphological and biochemical changes being the basis of choreic movements (7, 15).

Dystonic movements consist in stratification of free movements and involuntary spasm of both agonistic and antagonistic muscles (12). According to Dowżenko, anatomical changes found in cases showing this disturbance, are related to many structures. However, the essential anatomopathological basis of dystonia is the damage of the putamen or, of that part of the nucleus of the central thalamus, which controls the putamen. The damage of the latter causes the cancellation of the control of the abdominal segment of the globus pallidus, from where the impulses run through the thalamus to area 6 aα of the cerebral cortex. The area sends uncontrolled and irregular movements, most of which are directed towards the globus pallidus and to the posterior part of the black substance. From the globus pallidus the impulses may reach the peripheral neuron through the reticular matter. They are probably the chief cause of dystonic disturbances (3). So far biochemical irregularities of these disturbances have not been determined. Neurophysiological disorders, relating to blepharospasm described by Barardelli, allow only for suggesting a possibility of disturbing intracerebral neurotransmission (15).

Ballistic movements are vast, violent movements of the limbs, usually half-movements of throwing-up-kind, caused by the damage of hypothalamic nucleus and its junctions on the side opposite to hemiballism (4, 12). Ballistic movements are caused by cancellation of inhibitory effect of the Luys' nucleus to lower nuclei, mostly to the macrocellular part of the red nucleus, which is related to the adducent group of the skeletal muscles. The damage of hypothalamic nucleus cancels also its regulatory effect on the medial part of the globus pallidus, from where desorganised impulses come out through the thalamus to the cerebral cortex, especially to areas 6 aα and 4 s. From the cerebral cortex the irregular impulses run to the motor cells (3). Experimental studies carried out on animals confirm the possibility of provoking hemiballistic movements through administration of γ-aminobutyric acid to the Luys' nucleus (15).

Athetotic movements are slow fingers or toes movements, leading to unusual positions. They occur spontaneously, as co-movements or, under the influence of psychical impulses, they mostly concern distal parts of the limbs (12). The most frequent anatomopathological counterpart of athetotic movements is the diffuse damage of the striatum and of the globus pallidus, of its external part, more rarely — the foci located in the caudate nucleus region and in the thalamus. The damage of the globus pallidus inhibits the flow of impulses regulating the activity

of thalamic nuclei, in the consequence of which it comes to the cancellation of regulative effect of these nuclei on the prelocomotor areas (6 α and 4 s). Out of these areas come out uncontrolled impulses to the subcortical nuclei and to the medulla. Most of the pathological impulses run through the red nucleus and reticular matter in the anterolateral cords of the medulla. The system of the red nucleus and of the reticular matter is also devoid of regulative influence of the globus pallidus. Pathological enhancement of co-movements is the result of impulses running through the prelocomotor cortex and red-reticular-medullary fibres (3).

Myoclonus (clonic muscular contractions) are short, quick spasms of single muscles or a group of muscles, giving different locomotor effect. According to Helme, the base of myoclonus has not been sufficiently explained so far (5). Vinken distinguishes three types of myoclonus: the one deriving from epilepsy, extrapyramidal and medullar (16). Myoclonus is the result of irritation on different levels of the kinetic system: cerebral cortex, subcortical centres, brain stem and spinal cord (12).

It is assumed that the base of myoclonus is an excessive synaptic excitability between the sensory tracts and motor centres, in the result of which even small impulses evoke motor reactions. The mechanism of myoclonus resembles epileptic discharges. Concomitant to the cortical myoclonus may be the changes in electroencephalographic record — multispike potential (15).

Pathogenesis of myoclonus, independently of the place of its origin, may be related to functional disturbances of the system, in which serotonine is the transmitter. After administration of agonistic substances there may occur, in relation to serotonine, hyperactivity of the serotoninergic system leading to myoclonus (6). According to Snodgrass, the main role in the genesis of myoclonus play serotonine receptors 1 A (5-HT 1 A). γ-aminobutyric acid receptors are also responsible for the generation of several types of myoclonus. The application of the agonists of γ-aminobutyric acid, as well as of some adrenergic blocking agents of this acid may evoke myoclonus. The role of glycynic receptors in pathogenesis of myoclonus was examined on animals, but it is not clear in relation to people. There are communications on the occurrence of myoclonus under the influence of opiates administered in big doses (14).

Tremor consists in rhythmical, mostly regular oscillating movements, mostly related to further segments of the limbs and to the head. The following kinds of tremor can be distinguished: physiological, pathological, among which static tremor can be included, postural, intentional and non-classified (12, 16). Helme states that anatomical postures and pathomechanism of tremor are not clear (5). Sanes is of the opinion that in pathogenesis of physiological tremor somatic centripetal impulses perform certain role (13). It is assumed that some forms of tremor are connected with the damage of the extrapyramidal system (e.g. resting

tremor), or with the damage of the cerebellum (intentional tremor); hysterical tremor is psychogenic.

It is believed that the basis of spontaneous tremor is islet-like atrophy of small neurons in the dorsal and lateral parts of the putamen and in the dorsal part of the caudate nucleus, as well as the loss of piriform neurons and of nuclei in the dentate nucleus. Through the ascertainment of the increase in blood flow in the cerebellum in the patient with idiopathic tremor, the role of neural activity in the generation of tremor has been shown. It has been suggested that idiopathic tremor may be dependent on the circulation of impulses on the tracts running from the cerebellum through the thalamus to the motor cortex, and then to the spinal cord (1).

Tremor in Parkinson's disease is numbered among static tremor. Hassler explains its mechanism in the following way: the antagonists' tremor is generated probably in the system of switch cells of the spinal cord. In the resting state it sends rhythmic discharge to the locomotor neurons, which through desynchronizing influence of black substance in normal conditions become suppressed. The impulses, coming out of the internal part of the globus pallidus, when running through the thalamus, exert a facilitating influence on the cortical-spinal neurons. At the same time the inhibitory impulses coming out of the striatum and running through the black substance, do not run any more through the black-reticular-spinal tract to the system of switch cells of the spinal cord. Tremor is supposed to be the result of facilitating influence of the synchronized cortical-spinal tract and of simultaneous lack of inhibition exerted by desynchronizing impulses coming out of the complex: the striatum — the black substance (4).

Tics are stereotypic, short lasting movements of the definite part of the body in the nature of a compulsion (12). They can be divided into functional and organic. Most often they are functional. Organic tics prove the damage of extrapyramidal system, they occur in tic disease (the syndrome of Gilles de la Tourette). Neuropathological examinations in this disease revealed the traits of underdevelopment of the striate body, together with the decrease in its volume. It is assumed that the syndrome is the result of an excessive activity or hypersensitivity of the dopaminergic system of the striatum. The concept of disturbances in noradrenaline metabolism and of purine metabolism did not gain support. Biochemical examinations point to disturbances within neurotransmitters and especially, to the disturbance of balance between the system, in which dopamine is the transmitter, and the system, in which serotonine is the transmitter (8).

Spasms consist in irregular contractions of muscles or groups of muscles, of different frequency and intensity.

Cramps are disorders of muscular origin, consisting in long lasting tonic contractions of the muscles or groups of muscles.

Fasciculations are irregular, short contractions of single bundles of muscular fibres without evoking any movement in the joints. Damage is localized in the peripheral motor neuron.

Myokimias are waves of contractions coming through particular muscular fibres or groups of fibres, without any visible motor effect. Localization of the damage and reasons for the generation of this disorder are unknown.

Myorythmias consist in rhytmical tremors in the whole muscular group with motor effect. The damage is localized in the central structures, it covers the tegmental-olivary tract (11).

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STRESZCZENIE

Opisano różne rodzaje ruchów mimowolnych będących wynikiem niezamierzonych skurczów mięśni poprzecznie prążkowanych. Towarzyszą one najczęściej uszkodzeniom ośrodkowego układu nerwowego, głównie układu pozapiramidowego, i mogą być następstwem zaburzenia czynności neuronów hamujących wychodzących z prążkowia do gałki bladej i istoty czarnej, co powoduje nadmierne pobudzenie niższego układu neuronów.

Nowe poglądy na patomechanizmy ruchów mimowolnych uwzględniają rolę zaburzenia równowagi pomiędzy neuroprzekaźnikami układu pozapiramidowego wskutek uszkodzenia różnych jego struktur. Zwrócono również uwagę na hiperkinezy związane z uszkodzeniem innych elementów ośrodkowego układu nerwowego oraz na ruchy mimowolne pochodzenia mięśniowego.