Module:
Biological Foundations of Mental Health

Week 5
Reward, emotion & action

Topic 2
The structure and function of the Basal Ganglia – part 4 of 5

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Lecture transcript

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Here, you see a list of behavioural abnormalities that are seen in basal ganglia dysfunction. They include motor abnormalities, impaired memory formation, attention deficits, affective disorders, sleep disturbances. Now interestingly, those dysfunctions can be found either in isolation or sometimes together in a variety of basal ganglia-related disorders, such as Parkinson's disease, Huntington's disease, or even dystonia, abulia, dementia, but also attention deficit hyperactivity disorder or even schizophrenia. In case you know a little bit about these diseases, it is very interesting that the very same brain region can lead to so different behavioural outcomes.

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Let's look into one of the diseases, which is Parkinson's disease. This is characterised by the specific loss of dopaminergic input into the striatum. Here, you see the summary scheme, where you have dopaminergic input from the substantia nigra pars compacta, the SNC.

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Now what happens in Parkinson's disease? On the left hand side, you see a cross-section through the human brain, where you can see where the substantial nigra pars compacta is located. On the right-hand side, you see that SNC specifically innervates the putamen and the caudate, which constitute, as you now know, the striatum, and this connection is called the nigrostriatal pathway.

Now what is important in Parkinson's disease is that because of the specific loss of dopaminergic neurons in the pars compacta, you lose the nigrostriatal pathway. That is, dopamine does no longer have an impact onto the striatum.

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What is the consequence of that, if you no longer have dopaminergic modulatory input onto the striatum? That is, the direct pathway becomes less active, whereas the indirect pathway becomes more active. Remember, this is due to the fact that the D1 and D2 receptors respond differently to dopamine.

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Because of this absence of dopamine, action selection via the direct pathway is suppressed, whereas action inhibition via the indirect pathway is facilitated.

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And this causes the very typical symptoms of Parkinson's disease, which is tremor, rigidity, bradykinesia. But please be also aware that Parkinson's disease is characterised by non-motor symptoms that also can account for basal ganglial dysfunction. And one of the most important is sleep disturbances, which occur even before any motor symptoms are visible.

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But as mentioned, we also have other disorders that are related to the basal ganglial dysfunction, and I will illustrate it with another example.

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What you see here are MRI scans through the human brain. On the left-hand side, you can see highlighted the different regions, the caudate and the putamen, which together form the striatum. Then the globus pallidus, the nucleus accumbens, which also belongs to the striatum, and the anterior cingulate.

On the right-hand side, you see the scan of a patient which has been diagnosed with abulia, also called Athymhormia. This is caused by lesions of the globus pallidus and the connections between the striatum and the globus pallidus. Remember, this is part of the indirect pathway where you have an inhibitory connection from the striatum to the external segment of the globus pallidus. Now what is interesting is that those patients have been characterised by a profound inertia. Action is impaired in its initiation and in maintenance as well. Action is also impaired in its progress, since it tends to stop unless kept up by external stimulation.

Now this is intriguing. Those patients which have defects in the globus pallidus and the striatal pathway, they have great difficulties to initiate actions. And that, of course, directly relates to what we call voluntary actions or voluntariness. Now voluntariness, as you know, is a central point when we regard our freedom of activity, what we want to do and when we want to do it.

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