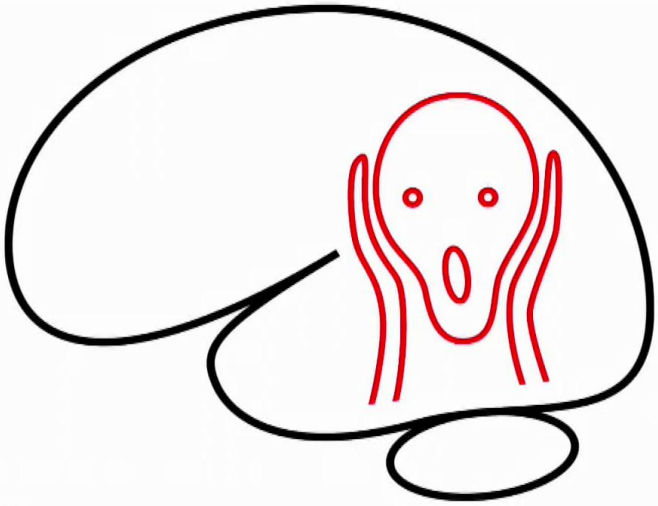
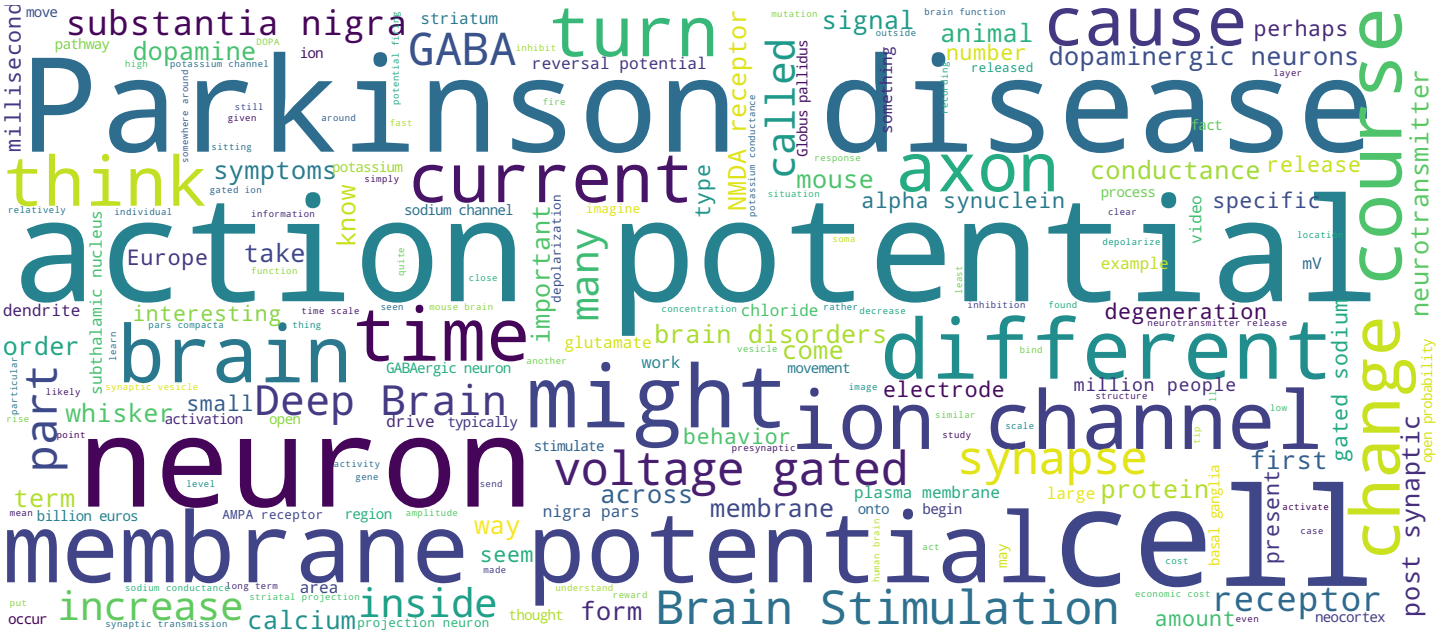


# 7.4 Brain dysfunction

## Cellular Mechanisms of Brain Function

Prof. Carl Petersen



# Prevalence of brain disorders

Gustavsson *et al.* Cost of disorders of the brain in Europe 2010. *European Neuropsychopharmacology* 21: 718–779 (2011).

Gustavsson *et al.* (2011) investigated the overall burden of brain disorders in 2010 across 30 European countries with a total population of ~500 million people.

Total financial burden : **~ € 800 billion = ~ $ 1 trillion**
Number of affected people: **> 150 million people**

Cellular Mechanisms of Brain Function

There are two important reasons for studying neuroscience. The first is to get a mechanistic understanding of how we think, feel and act. And that's the main thing that we focused on in this course. The second important reason for studying neuroscience is to try and alleviate the many brain diseases. That's what we're going to focus on in this video. It turns out that the prevalence and socio-economic costs of brain disorders are simply staggering. In 2011, in an important publication in the European Neuropsychopharmacology journal, Gustavsson et al. estimate the cost of disorders of the brain in Europe in 2010. Gustavsson et al. investigated the overall burden of brain disorders in 30 European countries, they have a total population of somewhere over 500 million people and the total economic gross domestic product of these European countries is on the order of 14 trillion euros. The brain disorders turn out to have an economic cost of somewhere around 800 billion euros. That's over 5% of the total GDP of Europe and that comes out to be somewhere around 1 trillion U.S. Dollars. Out of this cost, about 40% are direct medical costs, 20% are direct non-medical costs, and another 40% largely relate to the lost work hours.

Notes

Summary

0m 05s

# Prevalence of brain disorders

Gustavsson *et al.* Cost of disorders of the brain in Europe 2010. *European Neuropsychopharmacology* 21: 718–779 (2011).

Gustavsson *et al.* (2011) investigated the overall burden of brain disorders in 2010 across 30 European countries with a total population of ~500 million people.

Total financial burden : **~ € 800 billion = ~ $ 1 trillion**
Number of affected people: **> 150 million people**



The economic cost is enormous and perhaps more importantly, the number of people suffering from brain disorders is simply staggering. It's at around 150 million people and up, so well over a quarter of the population of Europe are in some way affected by brain disorders as estimated by Gustavsson et al. for the year 2010. These numbers for Europe are comparable and in fact, slightly lower than estimates for the United States so there's no hint of exaggeration and the impression is that with an aging population across the world and certainly in Europe, the number of people and economic costs are only going to go up in the future. So the scale of the problems of health related to brain disorders is simply enormous.

Notes

Summary

1m 55s

# Prevalence of brain disorders in Europe 2010

| | | € billion | People million |
|---|---|---|---|
| 1 | Mood disorders | 113 | 33 |
| 2 | Dementia | 105 | 6 |
| 3 | Psychotic disorders | 94 | 5 |
| 4 | Anxiety disorders | 74 | 69 |
| 5 | Addiction | 66 | 16 |
| 6 | Stroke | 64 | 8 |
| 7 | Headache | 44 | 153 |
| 8 | Mental retardation | 43 | 4 |
| 9 | Sleep disorders | 35 | 45 |
| 10 | Traumatic brain injury | 33 | 4 |
| 11 | Personality disorders | 27 | 4 |
| 12 | Child/adolescent disorders | 21 | 6 |
| 13 | Somatoform disorder | 21 | 20 |
| 14 | Multiple sclerosis | 15 | 0.5 |
| **15** | **Parkinson's disease** | **13.9** | **1.2** |
| 16 | Epilepsy | 14 | 3 |
| 17 | Neuromuscular disorders | 8 | 0.3 |
| 18 | Brain tumor | 5 | 0.2 |
| 19 | Eating disorders | 1 | 2 |

Gustavsson *et al.* (2011)

Cellular Mechanisms of Brain Function

Gustavsson et al. divided the analysis of brain disorders in Europe in 2010 into 19 different categories of brain disorders. Here, I've ranked them according to their estimated economic costs. On top of the list are mood disorders that affects some 33 million people and cost some 113 billion euros. In mood disorders, this is largely dominated by depression. Dementia, incorporating such diseases as Alzheimer's, dementia affects some 6 million people in Europe and costs around 105 billion euros in the year 2010. Psychotic disorders including schizophrenia affects some 5 million people and costs some 94 billion euros. Anxiety disorders including panic disorders, post traumatic stress disorder affects some 69 million people and costs 74 billon euros and addiction, largely drug and alcohol abuse affects 16 million people, costing some 66 billion euros. Just these top five brain related disorders affect more than 100 million people in Europe and cost over 400 billion euros as estimated by Gustavsson et al. in the year 2010. So the scale of the problems are rather significant. In this video, we're going to take a look at Parkinson's disease that affects perhaps some 1.2 million people in Europe so that's about 1 in 500 people and costs around 13.9 billion euros in terms of the direct and indirect costs for European countries.

Notes

Summary

2m 55s

# Parkinson's disease – Symptoms

Dr. James Parkinson (1817) *An Essay on the Shaking Palsy.*
published by Whittingham and Rowland, London.

Age-related, slow, progressive neurodegenerative disorder.

Early symptoms of Parkinson's disease:
slowness of movements, difficulty in walking, shaking, rigidity

Later other symptoms also become important:
dementia, depression

Cellular Mechanisms of Brain Function

Parkinson's disease was well described by Dr. James Parkinson in 1817 in his *Essay on the Shaking Palsy*. He described an age related, slow, progressive, neurodegenerative disorder and the early symptoms of Parkinson's disease include bradykenisia, so that's slowness of movements, difficulty in initiating movements, a problem with walking and stance, tremor, shaking. That's the shaking palsy that Parkinson described in his essay, and rigidity. That's contraction of muscles in an unwanted way. These motor symptoms gradually get worse as the neurodegenerative disease progresses and at later times, other symptoms also become important like dementia and depression. These are clearly very debilitating symptoms and make it impossible for the Parkinson's patient to have a normal life.

Notes

Summary

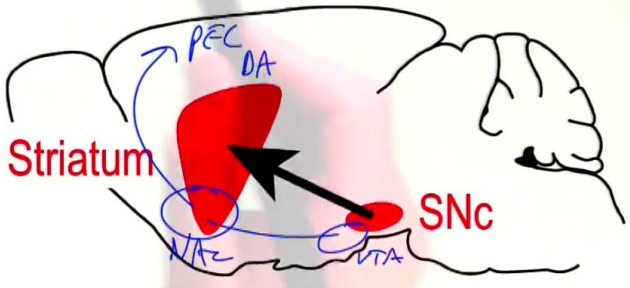
4m 55s

# Parkinson's disease – Loss of dopamine neurons

Symptoms of Parkinson's disease are caused by the degeneration of dopaminergic neurons in the *substantia nigra pars compacta (SNc)*. The degenerating neurons have cytoplasmic, protein-rich inclusions, termed Lewy bodies, which contain α-synuclein.

Clinical motor symptoms:

60% SNc cells lost
80% loss of striatal dopamine




Cellular Mechanisms of Brain Function

The symptoms of Parkinson's disease appear to be caused of the degeneration of dopaminergic neurons in the *substantia nigra pars compacta (SNc)*. So that's a part of the brain that we've already discussed in previous videos. Here you see it in the mouse brain, the location of the substantia nigra pars compacta, the dopaminergic neurons that send their axons to the stratum, and that's where they release their dopamine. This seems to be the major place of neurodegeneration that causes the motor symptoms of Parkinson's disease. Interestingly, the dopaminergic neurons of the ventral segmental area seem to be less affected when they send their axons to the nucleus accumbens and to the prefrontal cortex. They seem to be less affected by this disease. So there's some specificity it seems in the degeneration of the substantia nigra pars compacta neurons. These dopaminergic neurons have some cytoplasmic, protein-rich inclusions that have been termed Lewy bodies, and they contain the protein alpha-synuclein in quite a high concentration. That may be that alpha-synuclein aggregates are causally related to the degeneration of these dopaminergic neurons.

Notes

Summary

6m 04s

# Parkinson's disease – Loss of dopamine neurons

Symptoms of Parkinson's disease are caused by the degeneration of dopaminergic neurons in the *substantia nigra pars compacta (SNc)*. The degenerating neurons have cytoplasmic, protein-rich inclusions, termed Lewy bodies, which contain α-synuclein.

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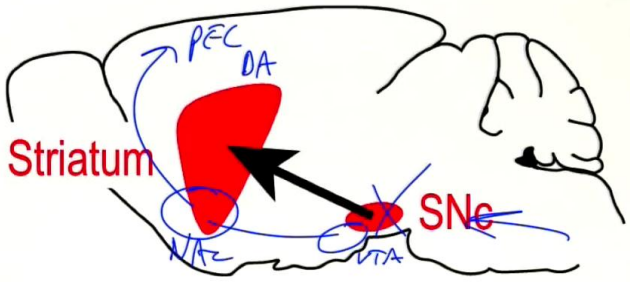


Cellular Mechanisms of Brain Function

By the time of clinical motor symptoms are diagnosed, it's estimated that about 60% of the dopaminergic neurons here in the *substantia nigra* are already dead. In terms of the amount of dopamine here, in the striatal, it's thought that there's about an 80% decrease in the dopamine content here of striatal dopamine. There's a massive loss of dopamine by the time clinical symptoms of Parkinson's are diagnosed. It's likely that they'll presumably be earlier symptoms that are more difficult to find by the clinicians and in the future research, it'll be important to try and study how early one can diagnose Parkinson's disease. In fact it seems that the neurodegeneration may not begin in the substantia nigra pars compacta but might be coming up from the brainstem or from other areas of the periphery and it's thought that maybe alpha-synuclein in a prion-like process might actually be spreading along different synaptic pathways. By the time the endstage of Parkinson's disease, it's clear that there are Lewy bodies, and degenerating neurons even in the neocortex, and so a large part of the brain is in fact affected by Parkinson's disease. Nonetheless, the most important symptoms of Parkinson's disease are thought to be caused by the degeneration here of these dopaminergic neurons in the substantia nigra pars compacta.

Notes

Summary

7m 26s

# Parkinson's disease – Environmental influences

Probably anything that causes degeneration of SNc dopaminergic neurons, will give rise to symptoms similar to Parkinson's disease.

Illicit use of intravenous drugs contaminated with methyl-phenyl-tetrahydropyridine (MPTP) has induced a Parkinson's disease-like syndrome in a number of people. MPTP crosses the blood-brain barrier, and in the brain is metabolised to the cation 1-methyl-4-phenylpyridinium (MPP+), which induces mitochondrial toxicity. In animal models, administration of MPTP causes Parkinson's like symptoms and a loss of dopaminergic neurons.

Cellular Mechanisms of Brain Function

The disease is probably generated by a mixture of the environmental influences and genetic susceptibility, just like most diseases that one can think of. It's likely that anything that causes the degeneration of these substantia nigra pars compacta dopaminergic neurons, will give rise to the same motor symptoms that are similar and common with Parkinson's disease. In the clearest demonstration of environmental influences, inducing Parkinsonian symptoms was through the illicit use of intravenous drugs that were unfortunately contaminated with *MPTP*, *Methyl-phenyl-tetrahydropyridine*, *MPTP* was then injected intravenously together with the illicit drug and that relatively rapidly induced Parkinsonian-like symptoms in quite a number of people who took this drug. The mechanism of action of *MPTP* has then been investigated. *MPTP* is lipophilic, it crosses the blood brain barrier so it can enter into the brain and in the brain, it's metabolized to another compound called *methyl-phenylpyridinium (MPP+)* it's a cation. And this compound, MPP+ is toxic and it appears to induce mitochondrial toxicity and in fact, a great deal of evidence suggests mitochondrial toxicity in the degeneration of the dopaminergic neurons from different perspectives.

Notes

Summary

8m 59s

# Parkinson's disease – Environmental influences

Probably anything that causes degeneration of SNc dopaminergic neurons, will give rise to symptoms similar to Parkinson's disease.

Illicit use of intravenous drugs contaminated with methyl-phenyl-tetrahydropyridine (MPTP) has induced a Parkinson's disease-like syndrome in a number of people. MPTP crosses the blood-brain barrier, and in the brain is metabolised to the cation 1-methyl-4-phenylpyridinium (MPP+), which induces mitochondrial toxicity.
In animal models, administration of MPTP causes Parkinson's like symptoms and a loss of dopaminergic neurons.

Cellular Mechanisms of Brain Function

In animal models, it turns out that MPTP causes Parkinson's-like symptoms, and also a loss of dopaminergic neurons and indeed that's one of the experimental models that people try to mimic Parkinson's disease so that they can see what the possible treatments are that might work. Other environmental influences that have been recognized include pesticides and insecticides and in rural areas, it seems that there's in general, often an increase in the prevalence of Parkinson's disease. Now not everyone who lives in the environment where pesticides or insecticides are being used gets Parkinson's disease and so it's probably likely that it's an interaction between the gene and the environment that causes the prevalence of Parkinson's disease.

Notes

Summary

10m 32s

# Parkinson's disease – Genetic influences

Genome wide association studies reveal susceptibility loci for Parkinson's disease, e.g.

α-synuclein – many different mutations with varying strength of influence upon the chance of getting Parkinson's disease

- A53T mutation is dominant
- *REP1* dinucleotide repeat expansion has smaller effect

LRRK2 (Leucine-Rich Repeat Kinase 2)

- many variants R1441C/G/H, I2020T, Y1699C, G2019S
- aberrantly increased kinase activity?

Cellular Mechanisms of Brain Function

Indeed, it's very clear that there are genetic influences that affect Parkinson's disease. Somewhere between five and perhaps up to 50% of Parkinson's disease has some genetic element associated with it. And there have been a great number of studies carrying out so called genome wide association studies where they look at the prevalence of Parkinson's disease with different genetic mutations in the population. Many different so called susceptibility loci have been identified, regions where there are mutations that correlate with increased prevalence of Parkinson's disease. Here, I'll just highlight two genomic regions that have been associated with increased chances of Parkinson's disease. The first one is alpha-synuclein and that's an interesting target that's been identified in these genome wide association studies because alpha-synuclein is one of the key proteins that's sitting inside the Lewy bodies and might be causally related to the degeneration of these dopaminergic neurons.

Notes

Summary

11m 21s

# Parkinson's disease – Genetic influences

Genome wide association studies reveal susceptibility loci for Parkinson's disease, e.g.

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Cellular Mechanisms of Brain Function

So quite a number of different mutations have independently been found in alpha-synuclein so that it increases the confidence that this is real and some of the mutations turn out to be very powerful so there's a mutation here in Amino Acid 53 of alpha-synuclein that turns out to be dominant and that means that it's very likely that you get Parkinson's disease if you have this mutation, whereas other mutations for example REP1, a dinucleotide repeat expansion, it's sitting somewhere upstream of alpha-synuclein so it's a regulatory element and that has a much smaller effect and might just change the timing of when Parkinson's disease might happen or increase the chances in a small way. So there are different mutations in the genomic area surrounding the alpha-synuclein gene and that has varying strengths of influence upon the probability of a person getting Parkinson's disease. Another gene that's been implicated is *LRRK2 Leucine-Rich Repeat Kinase 2* again, many independent mutations have been found that associate with Parkinson's disease and there's some hints that some aberrant kinase activity might be responsible for the deficits induced by *LARRK2* mutations.

Notes

Summary

12m 34s

# Parkinson's disease – Genetic influences

Genome wide association studies reveal susceptibility loci for Parkinson's disease, e.g.

α-synuclein – many different mutations with varying strength of influence upon the chance of getting Parkinson's disease

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- *REP1* dinucleotide repeat expansion has smaller effect

LRRK2 (Leucine-Rich Repeat Kinase 2)

- many variants R1441C/G/H, I2020T, Y1699C, G2019S
- aberrantly increased kinase activity?

Cellular Mechanisms of Brain Function

However, I think it's important to point out that we don't know the normal physiological function of alpha-synuclein or LRRK2 and so it's clear that a great deal of basic research needs to be done before we can understand the link between these mutations and Parkinson's disease.

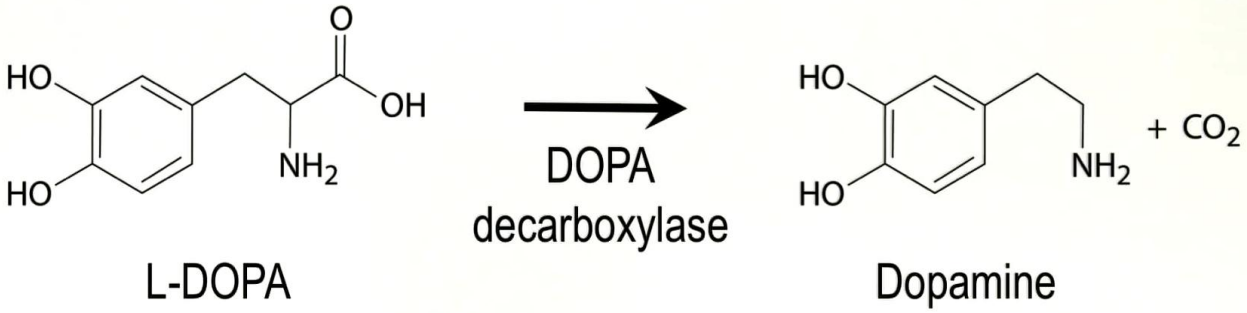
Notes

Summary

13m 53s

# Parkinson's disease – L-DOPA treatments

L-DOPA is a precursor for the synthesis of dopamine. L-DOPA crosses the blood-brain barrier and increases brain dopamine thus alleviating symptoms of Parkinson's disease. However, high doses and long-term use are associated with serious side-effects.




L-DOPA Dopamine

Cellular Mechanisms of Brain Function

One of the good things about Parkinson's disease is that there are relatively efficacious treatments for many of the symptoms. *L-DOPA* treatment is the first line of treatment in Parkinson's disease and this was something that was developed in the 50's by Arvid Carlsson and in the year 2000 he won the Nobel Prize for his work both in terms of identifying dopamine as a neurotransmitter and also for developing what ultimately is still the first and most successful treatment against Parkinson's disease, which is *L-DOPA*. And *L-DOPA* treatment makes a lot of sense. We know that Parkinson's disease results from the degeneration of dopaminergic neurons so there's less dopamine in the brain than there should be and one way which one might go and try to alleviate the symptoms is to increase the amount of dopamine in the brain. Now dopamine doesn't pass the blood brain barrier so you can't just give a patient dopamine but you can give the natural precursor in the synthetic pathway of dopamine, *L-DOPA* that does cross the blood brain barrier, it enters into the brain and the normal enzyme used in synthesizing dopamine *DOPA decarboxylase* can then act on the *L-DOPA* that's been administered just as a pill that you can take.

Notes

Summary

14m 12s

# Parkinson's disease – L-DOPA treatments

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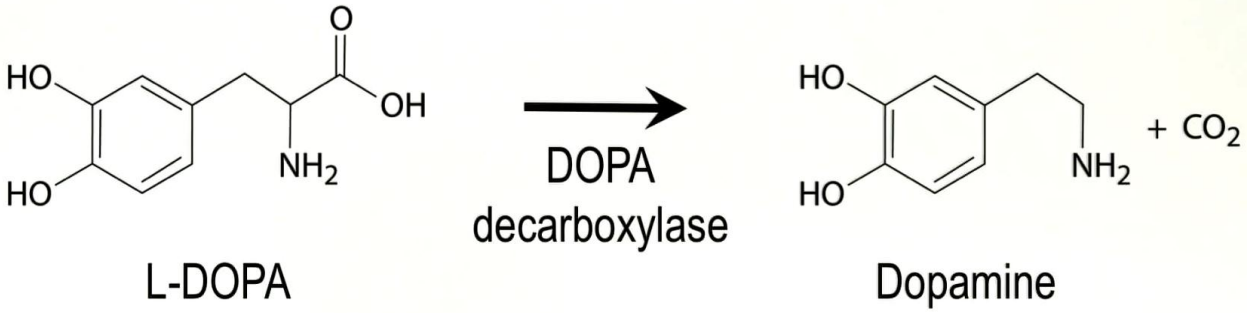



L-DOPA Dopamine



It releases dopamine into the brain and it causes a remarkable benefit and alleviates the symptoms of Parkinson's disease. However, high doses and long term use of *L-DOPA* are associated with quite serious side effects. Including dyskinesia where unwanted movements are initiated and in some cases also, overall changes in character of the person.

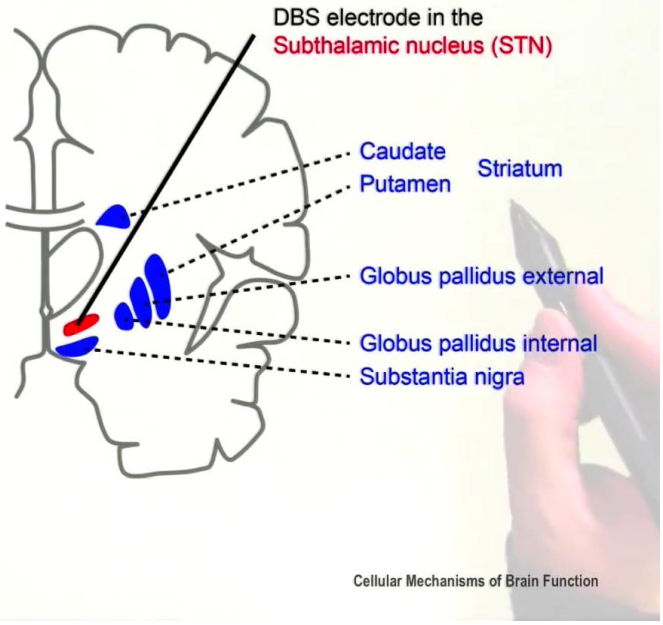
Notes

Summary

15m 43s

# Parkinson's disease – Brain stimulation treatment

When drug treatments fail, then some symptoms of Parkinson's disease can be alleviated by '*Deep Brain Stimulation*' (DBS). DBS consists of bilateral implantation of electrical stimulation electrodes in the subthalamic nucleus (STN). High frequency electrical stimuli are then continuously applied to the STN, and provide immediate relief of symptoms.




So when the drug treatments fail, then some of the symptoms of Parkinson's disease can be alleviated by so called deep brain stimulation. *DBS*. Deep brain stimulation consists of implanting electrodes into the brain, and at the tip of these electrodes one can then drive currents and stimulate the area of the brain that's targeted by this electrode that's implanted into the brain. What turns out to be successful in the treatment of Parkinson's disease, is targeting these deep brain stimulation electrodes to the so called *subthalamic nucleus (STN)* that's colored in red here, in the schematic coronal section of the human brain. Also indicated are other parts of the basal ganglia. So the sub thalamic nucleus that we haven't discussed yet are the glutamatergic excitatory neurons of the basal ganglia, whereas these other blue colored structures are gabaergic neurons of the basal ganglia. So we've already discussed the striatum and in the human brain this can be divided into two parts the Caudate and the Putamen they're both part of the same striatum and they're just divided in two, by axonal pathways of cortical parametal tract with the internal capsules, so these are largely the same types of cells and both are part of the striatum.

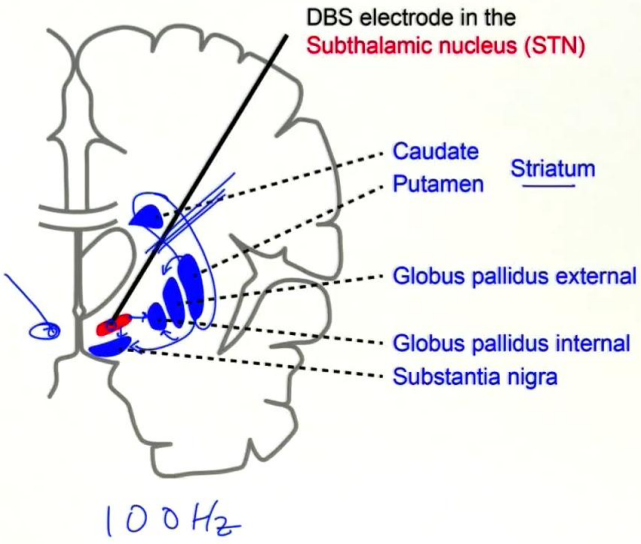
Notes

Summary

16m 12s

# Parkinson's disease – Brain stimulation treatment

When drug treatments fail, then some symptoms of Parkinson's disease can be alleviated by '*Deep Brain Stimulation*' (DBS). DBS consists of bilateral implantation of electrical stimulation electrodes in the subthalamic nucleus (STN). High frequency electrical stimuli are then continuously applied to the STN, and provide immediate relief of symptoms.




Cellular Mechanisms of Brain Function

The striatum as we know, projects both to the Globus pallidus and it projects to the substantia nigra and also the so called internal segment of the Globus pallidus. The subthalamic nucleus provides an excitatory drive onto other parts of the basal ganglia. So these electrodes are implanted bilaterally into the human brain where they are placed in the subthalamic nucleus and neurophysiologist helps the neurosurgeon guide this electrode to the right place in the brain, this is a small structure, it's a difficult surgery to do, but when it's successful, the recovery in terms of alleviating the symptoms of Parkinson's disease is really quite remarkable. So after implantation surgery and recovery from the surgery, then high frequency stimulation is applied to the tips of these electrodes and there's 100 Hz stimulation, so every 10 milliseconds there's a stimulus delivered here at the tip of these electrodes and that's done continuously throughout the life of the Parkinson's patient. That turns out to provide an almost immediate relief of the symptoms.

Notes

Summary

17m 41s

# Parkinson's disease – Brain stimulation treatment

You can learn more about '*Deep Brain Stimulation*' from many remarkable YouTube videos, *e.g.*

Prof. Hagai Bergman, Hebrew University of Jerusalem
http://www.youtube.com/watch?v=mMRX_Noco7g

Patient Andrew Johnson, Auckland
http://www.youtube.com/watch?v=uBh2LxTW0s0

Cellular Mechanisms of Brain Function

You can learn more about Deep Brain Stimulation from looking at YouTube videos which I think are really quite informative and one that I like is by Professor Hagai Bergman of the Hebrew University of Jerusalem. He's one of the inventors of Deep Brain Stimulation and neurophysiologist who's studied Parkinson's disease and the basal ganglia for a long time. You may also be interested in the self report of Andrew Johnson a patient who has been implanted with Deep Brain Stimulation electrodes shows how efficacious the brain stimulation is. What's remarkable about Deep Brain Stimulation in general is that there are more than 100,000 patients already implanted with stimulation electrodes and so clearly, this is a successful treatment in late stages of Parkinson's disease that can help patients dramatically.

Notes

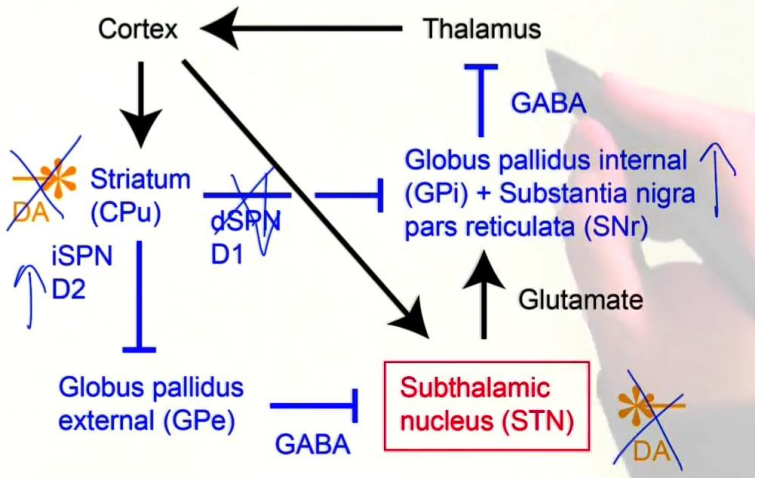
Summary

19m 01s

# Parkinson's disease – Mechanisms of DBS

Several different mechanisms have been proposed to account for how DBS of the subthalamic nucleus (STN) alleviates some of the symptoms of Parkinson's disease.

Further research is needed.




Cellular Mechanisms of Brain Function

Unfortunately, the mechanisms by which Deep Brain Stimulation works is more or less mysterious. Here we put the neural circuits that connect the sub thalamic nucleus to basal ganglia, Thalamus and Cortex, to try and put together some idea of how it might be working. So the basic deficit with Parkinson's disease is the degeneration of the dopaminergic inputs to striatum and also the subthalamic nucleus. Now we've previously noted that dopaminergic input tries to potentiate the *D1* pathway, the pathway from Cortex, running through the direct striatal projection neurons but then inhibits Substantia nigra pars reticulata and the Globus pallidus internal [inaudible] output of the basal ganglia. So if we now remove the dopamine then that'll cause a decrease in the efficacy of this pathway, so in Parkinson's disease, we think that there's going to be a deficit in this pathway, and conversely, the *D2* pathway may well be upper regulated. So if we remove the inhibition here, the Globus pallidus internal neurons and Substantia nigra pars reticulata neurons are perhaps going to be overactive. That means that they're going to inhibit the thalamus more than they should be doing and they'll then shut down the overall motor output equally of course.

Notes

Summary

20m 03s

# Parkinson's disease – Mechanisms of DBS

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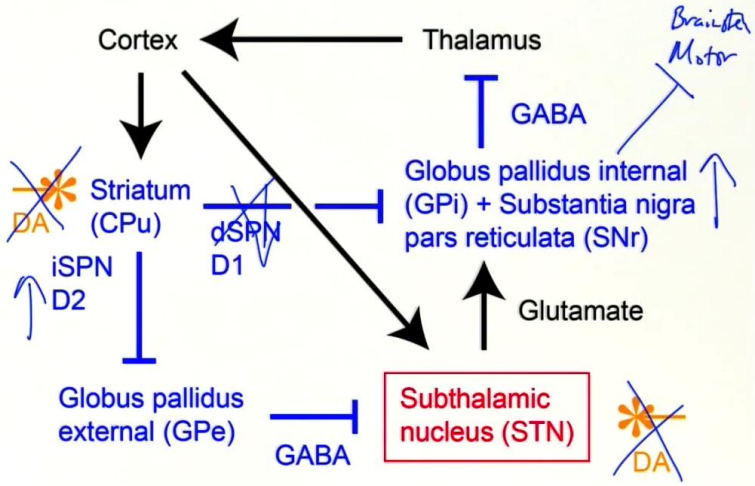


Cellular Mechanisms of Brain Function

Substantia nigra has descending projections to the brain stem motor areas and again, this will then cause a decrease in movement. And that's then consistent with much of what we know about Parkinson's disease. There's Bradykinesia, difficulty in initiating movements and that could then, reflect this decreased importance of the direct striatal projection neurons mediated via a loss of dopamine, and then a lack of stimulation of the *D1* receptors. Now, if we stimulate the subthalamic nucleus, and that's what we do with the Deep Brain Stimulation. The first notion would be that we stimulate these neurons, cause axon potentials in the subthalamic neurons and that would then release glutamate upon it's target structures. That would be stimulating the Globus pallidus and Substantia nigra and these GABAergic neurons would then be causing further inhibition and would then apparently be doing the exact opposite of what one would want. So apparently that's not how Deep Brain Stimulation works and so other ideas have come up. One idea is that the 100 Hz stimulation of the subthalamic nucleus, instead of stimulating this nucleus, actually inactivates it, so high frequency stimulation might be acting to inactivate this nucleus and that would then reduce the excitation of the Globus pallidus and that would then allow motor output to take place.

Notes

Summary

21m 40s

# Parkinson's disease – Mechanisms of DBS

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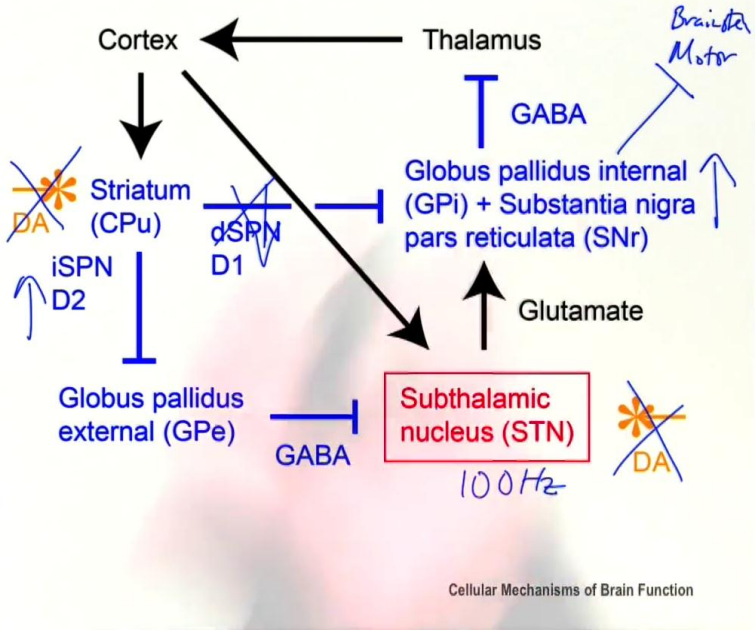
Further research is needed.




And so that's one possible way in which the Deep Brain Stimulation might be working. Other hypotheses are that actually it's not the stimulation of the subthalamic nucleus itself, but rather of the axons that are terminating in that region that mediate the positive effects of Deep Brain Stimulation. So it could be that it's actually stimulation of the cortical neurons that project here that cause the positive effects of Deep Brain Stimulation, or perhaps it's because of the Globus pallidus axons that are being stimulated or dopaminergic innovation here. There are many possible ideas as to how the Deep Brain Stimulation occurs whether it's an activation of these neurons or an inactivation of these neurons, or whether it's affecting the other components that are sending the axons here or perhaps it's because there are unusual rhythms in the Parkinsonian patient, there are high frequency, 10 to 20 Hz, so called BETA isolations that are prominent in Parkinsonian patients and perhaps stimulating subthalamic nucleus disrupts those BETA osolations and that might be how it works.

Notes

Summary

23m 20s

# Parkinson's disease – Mechanisms of DBS

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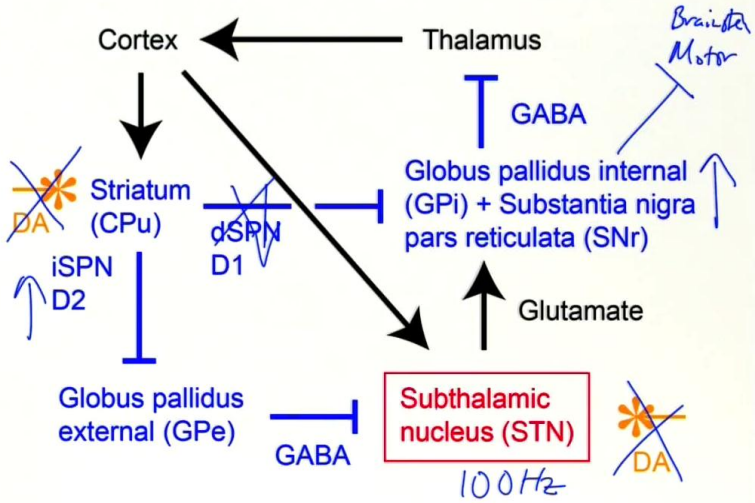
Further research is needed.




Cellular Mechanisms of Brain Function

I think what's clear is that further research is needed to understand how Deep Brain Stimulation works, and of course, if one gains an understanding of that, one may be able to target better therapies or better stimulation methods to be more specific, because of course, the Deep Brain Stimulation also comes with a variety of side effects.

Notes

Summary

24m 34s

# Brain dysfunction

- The brain is a delicate biological organ with many disorders.
- Neuroscientists are developing therapies for brain disorders.
- For example, Parkinson's disease is associated with loss of dopaminergic neurons, and the symptoms can be treated by drugs and deep brain stimulation.

Cellular Mechanisms of Brain Function

So in this video we have seen that there's a variety of brain disorders that make a large socio-economic impact upon the world. Neuroscientists are working hard to try and understand these brain disorders, try to come up with cures, and at least to alleviate the symptoms. That's what we see in the context of Parkinson's disease where we know some of the things that go wrong in the brain, that are associated with Parkinson's disease. We know about environmental influences, we know about genetic susceptibility loci. There's still a lot of work to be done for Parkinson's disease and also of course in terms of alleviating the symptoms, and of course, most importantly, would be to try to cure the disease and prevent the degeneration of the dopaminergic neurons. The same of course is true for the many other brain disorders and I think what's clear is that the scale of brain disorders in both the social and economic costs are such that it's really important that a vast amount of brain research is carried out in the future so that as rapidly as possible, we can help those suffering from various brain diseases.

Notes

Summary

24m 57s