Week 6 Answer to Question 3
Step 6.8 Ask Mark

So now we turn to the third question for this final week, and the question is as follows:

Question 3: earlier in the course you hinted at the connection between dreams and the limbic system. You mentioned eye movements and seeking in dreams. Please can you explain this?

Well, that's a big question. There's a lot to be said about the limbic system in dreams, and there's a lot to be said about dopamine in dreams, and seeking, and eye movements. But I'll try to distill my answer down to the essentials.

If you look at the dreaming brain, which we can do with for example PET imaging - you can literally look at what's going on in the brain while you're dreaming - what you see is that the cortex, and in particular association cortex, and especially prefrontal association cortex - prefrontal association cortex, and association cortex in general, is deactivated in dreaming sleep. Conversely, the limbic system is hyperactivated. So there's, as it were, a disinhibition of the limbic system by the cortex during sleep. What goes to sleep is your frontal lobes - your limbic system doesn't go to sleep. Your limbic system generates dreams. But it's not the whole of the limbic system that generates dreams - or rather I should say all the different component parts of the limbic system do different things in dreams. And we've done studies - now PET imaging doesn't tell you anything other than that the whole limbic system is activated during dreams - but we've done studies using another method, which is the lesion method, where you look at what happens when the different parts of the limbic system are damaged, and you ask what is taken away from the dream process by damage to different parts of the limbic system, and that tells you what those different parts are doing.

Before I tell you what we've found, let me mention that when the frontal lobes are damaged it has no effect on dreams, because the frontal lobes are deactivated during dreams - they're not part of the process. When the limbic system is damaged, there are big effects on dreams. For example, when patients have damage to both of their amygdalae - there's a structure called the amygdala, if it's damaged on both sides, in other words, there's no amygdala - what happens is the patients continue to dream, but they have very simple dreams, very pleasant dreams, they never have nightmares. So what that tells us is that the amygdala is contributing something of the affective quality of dreams, perhaps particularly the negative affective quality of dreams, and this in turn has something to do with the complexity of the dream thoughts. If you take away the

hippocampus, as happened in the famous case of HM, where his hippocampus was by bilaterally removed - was removed on both sides surgically - you might think, well that's the memory part of the brain, how can you have dreams without the memory part of the brain? Well, as it happens, you do. HM had lots of dreams. So we know that the hippocampus does not generate dreams. What would be more interesting would be to look at how is the character, the quality, the content of the dreams of hippocampal patients different from those of normal people. And those studies, amazingly, have not yet been done. So there's something that one of you can do - compare the content of dreams in patients with bilateral hippocampal lesions, with those with intact hippocampi. That's a study that I'm sure will be done fairly soon.

But by far the biggest effect - I haven't exhausted the whole of the limbic system there, I'm just giving you examples - by far the most dramatic effect of limbic lesions on dreaming, is lesions of the mesocortical mesolimbic dopamine system, also known as the seeking system - a part of the medial forebrain bundle - and when that part of the brain is damaged on both sides, you stop dreaming completely. Now this does not apply to any other part of the limbic system. So this is the big ticket item. The mesocortical mesolimbic dopamine system drives the dream process - it's the generator of dreams.

So that's what the lesion method tells us. The lesion method by itself is not enough, you want to test that conclusion by other methods. And that's been done with single-cell recordings, with microdialysis which measures the amount of dopamine release. But also it's been done with pharmacological studies where you can increase dopamine and decrease dopamine. And what you find if you block dopamine - for example by haloperidol, an antipsychotic - you block dreaming. If you increase dopamine with a dopamine agonist - with levodopa, the drug that Parkinson's patients take - you increase dreams. A lot. In fact, if you give enough levodopa, you'll start dreaming while you're awake, but that's another story. So that confirms what the other methods - the lesion method, and the imaging method - suggests, which is that the - and the single-cell recordings, and the microdialysis - it suggests that dopamine - mesocortical, mesolimbic dopamine - is the thing that drives the dream. So that's the the single biggest discovery we've had in recent years about the role of the limbic system in dreaming.

I might add, as an aside - although it's an important one - that we used to think that REM sleep, which is driven by a completely different part of the brain - not the limbic system, but rather the upper brain stem - we used to think that REM sleep caused dreams, was the causal mechanism of dream generation, and that's not true. Damage to the part of the brain that generates REM sleep does not lead to loss of dreaming it leads to loss of REM sleep. REM sleep co-occurs with dreaming, but it is not the same thing as dreaming. Damage to the dopamine system leads to loss of dreaming, but it does not lead to loss of REM sleep. But these two things have important interactions with each other, although they're not the same thing. Now the question of eye movements comes up.

You know, from what I've said before, that the seeking system is the basic motivational - it's the most basic motivational mechanism of the mammal brain, our own brains included. And what it

does, is it makes you do stuff, it makes you believe that if you interact with the world, that's where your needs, that how your needs will be met. So it energizes us, it motivates us, it initiates spontaneous activity, particularly what's called exploratory activity, that is to say feelings of curiosity, interest, expectancy - also optimism, enthusiasm. This is the seeking system.

If this is the system that drives the dream process - as we know it is - this has massive implications for our understanding of what dreams are all about. For one thing, it tells us that dreams are a motivated state - that it's not just random firings of neurons in your brain while you're sleeping. It's what your brain does about your needs while you're sleeping, and it's a motivated form of - whereas during waking life we forage, animals forage, that's what the seeking system does, we look, we explore the world to find the things that we need to meet our needs - in sleep you can't do that. In sleep you can't forage, otherwise you can't sleep.

So the drive to sleep, in order to satisfy that drive while all these other ones are still active while you're sleeping, in other words, the seeking system is still active while you're sleeping - and by the way, it's just as active while you're sleeping as it is during waking life - you can measure the firing of those cells, they don't fire any less during sleep than they do during waking life. That I believe is why we dream - dreaming is imaginary activity. It's virtual activity, it's activity in this - in mental space. All of that seeking, all of that foraging, all of the curiosity, interest, expectancy, initiation of activity that occurs in the imagination during dreams, enables you to give vent ,as it were, to your seeking needs, without having to wake up.

So that's how we understand - that's at least how I understand - the role of dopamine and the seeking system in dreaming. Now the role of eye movements - you know, we humans, although we are animals and we forage - we have to forage just as much as any other animal - we don't forage in the same -we don't always forage concretely, wondering about looking for a good time, cruising. We also forage with our eyes - we sort of explore the world, searching, finding meaning, working out what's going on in this much more inhibited way through eye movements. So eye movements - circadic eye movements, searching eye movements - are a proxy of dopamine activity.

And I believe that, when I said that dreaming and REM sleep interacting complex ways, I believe that this is an important line for future research. We know for example in rats that measure of the twitching of their whiskers is a proxy for dopamine activation. In other words, you can show experimentally that the more dopamine there is in the brain - especially in that dopamine system - the more twitching there is in the whiskers, and therefore you can measure the twitching in the whiskers as a proxy of measuring dopamine activation. I suspect we can do the same thing with the eye movements. Our eye movements are like the rat's whiskers twitching.

So those were the sorts of things I was alluding to when I made the remarks about dreaming and the limbic system, and seeking and eye movements, that the questioner's referring to. As I said, there's a lot more that I could tell you about the dreaming brain - it's a very deep interest of

mine. In fact, it's the it's the thing that I've researched probably more than anything else - brain mechanisms of dreaming. So Google that, and you'll find out more.