GERALD SCHNEIDER: So a study of how these organized sequences of movement that we've been talking about are produced and how they can change has led to the development of some basic ideas about how the brain works and have influenced theory of the brain. I call them basic organizing concepts in theories of CNS function when we think of them at the level of connections and circuits, that is.

And this is just a summary of them, beginning with the straight-through processing concepts that are used in analysis of reflexes. And they're used in S-R models. It's the basic type-- it's the way we think of input analysis going from one stage to the next. You could call them the filters of the brain, the sensory filters, OK?

From retina through several stages to the geniculate body to the cortex and then through multiple stages and then to an association area and so forth. That type of analysis is basically a straight-through processing type of concept, the same-- based on the S-R model. And it's still a very dominant way to think about the brain.

But then we have the various kinds of feedback control, which play a major role in our models too. The old idea of reverberating circuits and positive feedback is still-- there is examples of that now in physiology. Reciprocal inhibition, we know, is critically important for movement control. And homeostats with negative feedback are also known to occur in the brain.

And then we have the spontaneous CNS activity of endogenous origin. The red there just means it wasn't on your original printout. So you can ignore that since you have the new printout.

But there's two types of spontaneous activity. We've talked about the rhythmic activity, which often leads to periodic bursts of action potentials. But also, we have what we call maintained excitatory states.

So for example, there are neurons that the hungrier you are, the more active they become. It's a central excitatory state. And it's called that in some models, OK? And that activity can be either endogenous or it comes from another part of the nervous system. At least it's not a direct result of a stimulus.

And finally, important to all these concepts has been the idea of plasticity. And that usually means a change in connections, though it can mean other things too. There are other kinds of plasticity that could affect neural activity.

But usually, when we talk about plasticity, we're talking about changes in connections, either involving the molecules at the synapses that can change the sensitivity of the synapse, or we can be talking about anatomical changes, like sprouting of various kinds-- collateral sprouting being one of them.

OK, so today, we're going to continue this topic of rhythmic output and timing. And we're going to switch to talking about circadian rhythms, OK? Sleep and dreaming. Circadian, about a day because the natural rhythm is not exactly a day in length.

These are the topics I'll talk about today. We may not finish the talk about sleep today, but we'll see how far we can get. So these three topics at the top are what we'll start with, focusing on proving the endogenous nature of a biological clock and then dealing with this question-- at least the first answers to that question about why we sleep.

Why does any animal sleep? And don't you wish you didn't have to? Oh, I don't think so. Isn't sleep fun? Yeah.

OK, endogenous versus periodic inputs. Whether sleep, the sleep-waking cycle, the rhythm of activity was primarily controlled by endogenous inputs or periodic inputs coming from outside was a major controversy in the middle of the last century, 1960s. It was resolved first by behavioral studies, and I'll talk about those, and then by brain research. And we'll be talking about those, also.

So the first proof of the endogenous nature of the body's clock mechanism came from behavioral studies of free-running rhythms of activity. That means, for example-- and hamsters are an animal commonly used for this because they have very precise and easy-to-measure circadian rhythms of activity. You put them in constant conditions. You put them in a room with continuous light-- continuous low light or continuous darkness or continuous lights on.

And similarly, humans have been studied, where they wanted to isolate them, even from cosmic rays. They wanted to isolate them from anything that might be periodic as the Earth turns. So they put them in underground bunkers. Was impossible, of course, to eliminate everything. But they did get free-running rhythms, OK?

So for hamsters, this was one common way. I've done these kinds of studies in hamsters, though I did not use a running wheel. I used an elongated cage where I measured breaking of a light beam. They had to break one and then the other. And if they did that, it would move the needle on a strip recorder like this.

At that time, I wasn't recording with computer memory. I was recording it on paper. But it was done very much like this, where here, every revolution of the wheel causes a blip on the paper. And then what they do is each 24-hour record is put just below the previous one, OK? So as you go down this chart, you're going from day to day to day, OK?

And you see there, this is a period of light, period of dark, OK? And right here, at the beginning, they cut the optic tract, OK? And you can see he still maintains a nice 24-hour activity cycle.

He's inactive in this period. And then he becomes active, OK? Then he goes back. You start the day again. He goes to sleep, or he's very inactive. Wakes up almost the same time every day, OK?

The optic tract is cut. But the important thing is where it was cut. It was cut above the optic chiasm but before it reached the geniculate bodies. So no axons were reaching the geniculate bodies, pretectal area, tectum, any of the areas we've talked about. And we'll be talking about more later on, OK?

But then the question was, well, is this because of light? So right here, they changed to continuous dim light. And now see what happens to the rhythm. Now he wakes up later every day, goes to sleep later every day.

Some students think that that is a powerful tendency in them. They always want to stay up later and get up later. It may actually be true because the studies of humans in underground bunkers-- and that was first done in Schwessin, Germany, by the father of one of our graduate students here, a former graduate student. The father's name is Ernst Poppel.

I met him in Germany when he was doing these studies. And he put humans in underground bunkers. Took all their clocks away. So they were isolated from time cues as far as he could do it. He did have a telephone there. And he just found out about their rhythm of activity.

Initially, they were students. They slept for about 48 hours initially. And then they started showing a regular rhythm of activity. But it was not exactly 24 hours. It was close to usually about 25 with humans.

Now, with hamsters recorded like this, the ones I've measured-- I've had maybe one or two hamsters with a rhythm less than 24 hours. Almost all of them are about 24 hours and 20 minutes or 30 minutes, roughly in that vicinity, OK? But it's free-running, OK?

And with the hamsters, the rhythm stayed free-running even though there was activity of people in the hallways that was regular, OK? The janitors came around regularly. They were fed regularly. And yet, none of those things influenced their activity rhythm. The only thing that influenced their rhythm of activity was the light-dark cycle. Question?

Yeah, I think that may be a mistake. I don't know. I noticed that, also. They noticed that there was-- I think something caused the phase to shift there. But in fact, if you keep the light-dark cycle, you might do something to cause a shift. But it will still stay 24-hour rhythm. They didn't. I don't remember how.

OK, now what about the brain studies? There was a long period of searching for a biological clock that can be entrained by the light-dark cycle. Some of those studies involved ablation of optic tract terminal areas.

Now, I mentioned that they could cut the whole optic tract. But they also made lesions of optic tectum, superior colliculus or geniculate body, or other regions. And they always got a persistence of the circadian rhythm, although they sometimes did get a shift in the phase.

And then it was a puzzle for a long time because we knew light was affecting the rhythm. And yet, we thought we had cut all the projections. But in fact, our anatomical tracing methods weren't sensitive enough to see a projection that was known from some studies of nonmammalian forms, namely a retinal projection to the hypothalamus.

And that projection is very easy to see with modern tract-tracing techniques. It goes to the Suprachiasmatic Nucleus, which, in textbooks, is often abbreviated SCN, OK? I usually put SCH because I use CH for the chiasm, so the suprachiasmatic nucleus.

If you remove the eyes, if they don't have any retina, if they don't have a suprachiasmatic nucleus, or you section that connection, you get a free-running circadian rhythm without that connection. If you actually remove the suprachiasmatic nucleus, you can get a disorganized rhythm, OK?

Now, if you look at those cells, you find that they do generate. Even if you isolate them from connections with the rest of the brain, they do show circadian changes in activity. They also show circadian changes and some in transcription, OK?

There are some cycles of molecules they're producing. But then there are other studies that show that there are other biological clocks in addition. We know less about the others than about this one.

OK, let's go to this question for a minute before we talk more about the brain mechanisms. The brain mechanisms will come at various points in this talk. Why do animals sleep? Why do we sleep?

Now, this kind of question-- anytime you get a question like that about human nature, it's got multiple levels of meaning. First of all, we can think of it in evolutionary terms. Why is sleep adaptive?

Then we can talk about it as an instinct that's triggered by a biological clock, which is entrained by the light-dark cycle, which is what I was implying in the previous slides. Then we can talk about specific aspects of that. How is that instinct represented in the brain?

There are neuromodulator systems with widespread axon connections. So we know that widespread parts of the brain could be affected at the same time by activation-- for example, serotonin axons, norepinephrine-containing axons, or acetylcholine-containing axons. Usually, I abbreviate that with a capital C. And then there's other types of neuromodulators-- chemicals in the cerebrospinal fluid that can influence sleep.

So let's talk about adaptive pressures. Why would sleep evolve in the first place? And of course, we always think first, we need rest and recuperation. There are certain processes that the body needs to recover from to change with rest. And there is evidence that, in fact, is important.

But we also know that there are other reasons to sleep. It's been proposed that animals need to avoid predators, especially a light-active animal that can't see well in the dark. And there are some predators that can see and hear very well in the dark.

They're much more likely to get caught. So if they go to a secure place and sleep all night, they're going to be safer, even though they might be too stupid to outfox predators, which are often smarter animals than they are, OK? So sleep could evolve for that kind of function.

It's also been proposed in-- because infants sleep much more. And they have a lot more what we call active sleep, which we'll talk about in a minute, where the brain is very active. This could be very important, promoting development of neural circuits. And that has been proposed. And we'll look at a graph of how sleep changes with age.

More recent studies have focused on the role of sleep in memory functions and memory consolidation and in editing our memories. And we'll talk a little bit more about that, also. But first, let's talk more about this instinct that's triggered by the biological clock and entrained by the light-dark cycle. Let me give some more examples of experiments.

First of all, we had already mentioned the retina to suprachiasmatic nucleus studies. And I said that the suprachiasmatic nucleus cells seem to be very important for this rhythm. If you take them out, you can disrupt the rhythm.

The clincher there was, with hamsters, to get an animal that had no suprachiasmatic nucleus. So you didn't have a biological clock. He didn't have his natural rhythms. And you implant in his brain suprachiasmatic cells from another animal. And you can do that from using tissue of very young animals.

They were able to succeed in that. And they were able to restore the biological rhythm in some animals. Now, such transplant studies are difficult. But it has been possible to do that.

And there's other studies, then, that show molecular changes. And they find that sometimes, you get the left and right suprachiasmatic nuclei. There's a pair of them, one on either side of the midline. Sometimes, they oscillate out of phase. And I brought a few slides showing one of these studies.

First of all, look at the left here. I've described it here as wheel-running activity. And we saw an example of that before. Now, this animal is in constant light.

And notice what happens here around day 40. His activity splits into two rhythms, OK? One you see there. One here. OK, so now he's becoming active twice in a day. Sometimes, the two rhythms didn't have the same period, OK?

Then they looked at the proteins resulting from certain genes. I think, actually, they were looking at messenger RNA. So we're looking at transcription. OK, these are the proteins known to be important in circadian rhythm control because if you knock them out, you can disrupt circadian rhythms.

Notice that Per1 here, in this animal, at the time they killed him, was more active on the right side. Also, Per2. But Per3 didn't show such an extreme asymmetry. It was active on both sides.

And then they looked at that same animal. They looked at the amount of left-right asymmetry of these three genes. And they measured it as a function of the time the animal was killed after activity onset in hours, OK? So when he first became active-- four hours later, six hours later, and so forth. And you can see that they got differences among those genes.

Now, this was simply the asymmetry of those two genes. And they've seen that this antiphase oscillation is fairly common. Here's antiphase oscillation of these two genes.

Here's where they're oscillating together. And here's a case where they're in antiphase. We don't fully understand it, but this, we think, can explain some of these peculiar effects you get in abnormal conditions sometimes. And it happens in humans, too.

OK, now let's talk about the discovery of different-- we're just going to talk about sleep for a while now. And we'll talk about different sleep stages.

First of all, you have to know what these terms mean at the top. From your reading, you might know-- what does the EEG mean? Electroencephalogram. What about EOG? Electrooculogram for measuring eye movements.

OK, what about EMG? Electromyogram for measuring muscle tension, OK? I actually showed one when I was talking about movement controls, some electromyographs showing activity. Remember, the oscillating activity of limb muscles.

And also, so these are all used in sleep studies. And they make various measures of the autonomic nervous system as well, OK?

It was Hans Berger in the early part of the last century that first used EEG to look at sleep and waking. First of all, he knows, just in normal human beings during the day, they showed-- the EEG indicated two different kinds of waking.

If a person is sitting still with his eyes closed-- can even have his eyes open if there's nothing happening around him, and he can show a lot of alpha waves, which is slower. It's fairly synchronized activity, especially in the posterior, over the posterior part of the brain.

They're recording, actually, from the surface. They're scalp electrodes. But they're recording small currents induced by underlying electrical activity.

Berger also began to get evidence on different stages of sleep. But most of that work came later with the work of Dement and Kleitman. And Dement is still active in this work on sleep stages.

And I just want to note here that these EEG, as useful as it is in studying system properties of the brain, there are studies that show that in some cases, the CNS arousal indicated by the EEG doesn't correlate with behavioral states. You can give drugs, for example, that will give a lot of the slow activity. And yet, the animal can be quite active.

That's somewhat of a problem. But people tend to ignore that. We like our correlations. It doesn't mean we fully understand them.

So now, I want to talk about the two major kinds of sleep. People divide sleep up into more stages than this, but there's really two major kinds of sleep-- REM sleep, Rapid Eye Movement sleep, and non-REM sleep. REM sleep, you probably know that corresponds to the mental state we call dreaming, though it doesn't mean you don't have any images occurring in non-REM sleep.

But first, before we talk much about dreaming, let's talk about phasic versus tonic activities in REM sleep. OK? REM sleep, we call paradoxical sleep for the simple reason that the brain waves-- that's the term we use for electroencephalograph recordings. The EEG is the EEG of a waking state, an aroused state.

Remember, I said there's two kinds of waking. The person could be sitting in a relaxed state, and you get these slower waves. But if you attract his attention, you get him to pay attention to something. You rouse him. He gets the low-voltage desynchronized activity, OK?

Well, that's what you get in REM sleep, OK? That's here. You get rapid eye movements. In addition to the desynchronized activity, now you have the rapid eye movements, some twitching, especially of the distal muscles.

And you get a particular kind of wave in that affects the visual system. It originates in the pontine region of the hindbrain. It goes to the geniculate body and then appears in the acceptable neocortex. OK, and they have found that those waves of activity are correlated, at least to some degree, with the eye movements.

There's also tonic states in REM sleep-- that is, not just momentary states. Whenever you enter REM sleep, your muscles become relaxed, muscle atonia. You lose muscle tone, OK?

Along with the EEG desynchronization, in the hippocampus shows a theta rhythm, a regular rhythm that's a little bit different than the alpha. It's characteristic of hippocampus in certain behavioral states.

OK, in addition, there's an autonomic dysregulation, we will call it, because it's not a normal state of the autonomic nervous system. Some regulatory functions are either changed or lost.

For example, you get exothermia. So if it's very cold in the room, your body temperature will drop when you're in REM sleep. OK, you don't have normal control. It won't stay that way because you'll come out of REM sleep, and then you'll get better autonomic regulation, OK?

There's vascular dilation in the genital organs. So that's one reason that it was common to think of dreams as having something to do with sex because you would always get the sexual arousal during-- it appears to be sexual arousal.

But I think if we think of it more as vascular, as a autonomic dysregulation characteristic, it's probably a little more accurate because you can get that symptom of arousal, even when the dream has nothing to do with sexuality. In addition, you get irregularities in the heart rate and in breathing that are characteristic of that state.

So here's how these states change over a human lifetime. They've just taken the two major stages of sleep-- REM sleep in yellow there, non-REM sleep, and here's waking. And this is divided up into total amount in 24 hours, OK?

So non-REM sleep and REM sleep, you can see both go down with age. And you see how very high they are in babies. A newborn here is sleeping almost 16 hours a day, like a cat, OK? But it rapidly declines, OK?

So by the time you're in your late teens, you're down to below nine hours. And that's an average. Of course, people will differ. And I should say right here that there's, in fact, a lot of variability in sleep times, especially in adults. And I mean the amount that people seem to actually need, the amount that they will naturally sleep if they're not under constraints of MIT assignments and exams, OK?

It's also very common for sleep to continue to decline in amount with age. So by the time people are over age 70, they're falling below six hours of sleep. And also note that the amount of rapid eye movement sleep is decreasing as a percentage of the total sleep time.

We had no real good explanations for this decline until recently, when we're beginning to find out more about functions of REM sleep, other than simple recuperation during sleep. That probably is more a function of slow-wave sleep.

So let's deal now with the questions about why we have REM sleep at all. Why do you have these two different stages? It's been an area of a lot of controversy. And there were studies of REM sleep deprivation.

And the early reports indicate that if you deprive animals or people of REM sleep over a long enough period of time, they either go crazy or die. Of course, they didn't do it long enough to kill anybody. But that was the report until somebody realized they'd left out the control group. You better wake people up in the other stages of sleep, also, an equal number of times.

And when they did that, then the effects of deprivation, like greater irritability and so forth, were the same. People don't like to be woken up at night so often. So it doesn't much matter whether you wake them up in dream sleep or non-dream sleep. I still see people report these drastic effects of REM sleep deprivation, however. But they are at least not what people initially reported, OK?

So what is the function of the paradoxical part of sleep, the rapid eye movement sleep? One was that in slow-wave sleep, you have-- neurons are becoming less active. The brain seems to be shutting down.

And the idea was that if that goes too far, it could lead to plastic changes that could damage brain function. So you needed to wake it up periodically. I don't think it was-- I think people come up with theories like that when they're frustrated coming up with anything else.

So then studies were done on memory consolidation-- teaching people, for example, things just before they go to sleep, testing their memory. And if you do deprive them of REM sleep, they will not remember as well as if you don't. But it's still not very clear what that role is.

There are recent ideas beginning to appear. And I've been teaching this hypothesis for a few years that I developed in the course of teaching this class, that this kind of active sleep is a kind of-- involves editing our internal model of the world. That's the basis of our ability to predict our perceptions and events.

I point out here that the model includes the social world. And we encounter things during the day where sometimes there's drastic changes, novelties that affect that model. And yet, if that model is highly overlearned, our old memory of it is still much stronger than the newly encountered thing. So we need to do something about that old memory, OK?

So I think in many cases, and there's evidence for this, if you encounter very novel things, a new building goes up that suddenly wasn't there before, and suddenly you see it. You have to change. Somebody that you thought was happily married suddenly gets divorced and you didn't know about it, some big change in the social world-- people frequently will dream about those things.

It doesn't explain all dreams, but that is a correlate of dreaming. So it could involve a kind of editing of the model so that the older memories would not be stronger than new ones. We just edit them. We change them so they correspond to current realities.

And there are some hypotheses now beginning to be studied of this nature. This is just to point out how controversial this is, about how sleep scientists disagree. And this was a report couple years ago from the Associated Press. It began with, "When you're asleep, your mind uses dream time to process information for use when you're awake or not."

He published these views. One was of Robert Stickgold, who's a professor at-- a man that I know at Boston University and Harvard Medical School Department of Psychiatry. He says that the brain is taking information, helping us put it into a form we can understand. Understanding the complexity of the world is one of our brain's most difficult tasks. It needs more than our hours of awake time to get the job done. He was talking to us through a reporter.

And this, another researcher at the Center for Sleep Research of the Department of Veterans Affairs. This guy, Jerome Siegel, says he's looked into dozens of studies on dreams and learning. Found no evidence that the sleeping mind does anything important.

I should point out that work with Stickgold and his group has come up with additional evidence that, in fact, something important happens in sleep. They found that certain memories actually improve. Things you don't remember well before sleep, you'll remember better by morning. And some of that looks pretty good to me.

Let's say a little bit about sleep pathologies and irregularities of sleep patterns before we say more about the brain systems of sleep. I want to say more than just talk about the suprachiasmatic nucleus. So these are some of the problems that occur during sleep.

Sleep apnea. What is that? People that stop breathing while they're sleeping. That can be pretty dangerous. It usually happens when they enter dream sleep and their muscles become very relaxed, OK? And because of the relaxation, they get a blockage in their throats, OK?

And it can be corrected. People with that kind of sleep apnea can be corrected by surgery. It's usually in people that are a little overweight but not always, OK? There are several forms of sleep apnea.

People with severe sleep apnea, the normal course of it is that they don't die. In fact, CO2 levels go up. There are autonomic changes that kick in, and they wake up. So these people are getting very bad sleep because they keep stopping their breathing. Then they wake up to correct it.

If they have a partner that notices that they stop snoring-- and often, these people are snorers. They'll stop snoring. The person learns to nudge them. And then they'll get better sleep because they'll start breathing again.

So sleep apnea is, in a sense, related to insomnia because these people have very bad sleep. But insomnia, of course, is people that aren't sleeping or have a great deal of difficulty sleeping. And some kinds of insomnia include failure of entrainment of the activity cycle. And insomnia, there are ways to treat it. Of course, there's several different sleep medications.

Narcolepsy is another one. And I have a video clip that I will try to remember to bring to class next time to show you a little bit of these things. There's a narcoleptic dog, for example, on the film and also a narcoleptic human, just so if you've never seen any of this, you'll see it.

So let's just say a little bit about narcolepsy. Narcolepsy, broadly defined, means excessive daytime sleepiness. It doesn't mean just because you didn't get enough sleep, OK? I mean, we've all experienced that, at least most of us, if we're academics.

These people have an abnormal sleepiness that can have some really interesting characteristics. Usually, people that are narcoleptic, when they first go to sleep, have REM sleep. They fall into REM sleep. That is not normal. Normal person does not have his first REM period until after he's been asleep for a little while, OK? These people have REM sleep right at the beginning.

Some of them will have these hypnagogic imagery, which is a extremely clear kind of imagery that-- this really amounts to a hallucination. It's so clear to them that they cannot tell it from normal, from reality. Some people will have this only once or twice in their lifetime. Some people have it regularly when they daydream or when they first wake up.

How many of you have had hypnagogic imagery? Probably a number of you. You might be reluctant to admit it. But believe me, it's not that abnormal.

I've had them where I can't-- I haven't had them since college days. But when I was your age, I had them a few times. And I can remember-- of course, my sleep patterns were pretty irregular, too, in college. But I do remember being so aware while I was sleeping that I wasn't quite sure if I was really sleeping because the images were so clear.

I noticed that nobody was talking to me, but maybe that was just-- they were all studying for a test, you know? But I did various things in the dream to test this image, like pinching myself, and it hurt. So that test failed. I didn't wake up in all this.

I don't know if other people are so conscious during this imagery as I was, but it was quite fascinating. And I finally, when I did wake up, then of course, ah, I was greatly relieved. See, I was worried that I wouldn't be able to get out of it, just like a science fiction story.

But now let's talk briefly about cataplexy. Many narcoleptic people, when they become-- and usually, when they become emotional. In the Doberman dogs, it's like that. And there's another species of dog that's also narcoleptic. When they get excited, they can suddenly collapse because their movement, their muscles become relaxed, just like in dream sleep.

There is a mechanism in your brain for relaxing the muscles, even when-- well, why is that so important that we have that? Because in dream sleep, our brain is active. If it was driving movement, we would be active too, OK? So you need to relax the muscles.

But here, it happens in an abnormal situation. It happens during the day. They get a little excited. They'll start to laugh or something. Somebody will tell a joke at the dinner table. And bang, this person's in his soup or on the floor.

So that's a cataplectic attack. And it can be dangerous because it can happen if they're doing a sport. For example, they'll be running rapidly, and something is very exciting. And bang, they fall.

Yes. Sorry? That's a good question. I'm not sure. The lady in the film, when she comes out of the cataplectic attack, it's like she's waking up. She seems sleepy. I don't if that's always true. It's a good question.

There are some other even more bizarre syndromes of sleep, disturbances of the sleep mechanisms. One of them is called automatic behavioral syndrome. It happens in people with sleep apnea and narcolepsy. And in these periods, they have no awareness, no memory, complete amnesia. So to them, the behavior that they did seems to have been automatic without consciousness, OK?

I can tell you what it's like because I've had it in influence of hypoglycemia. I don't have it normally, OK? But when I've had that, for example, I'm climbing a stairway, OK? And I look at my clock. I'm having a little trouble moving. My blood sugar is very low. I'm not quite sure what's wrong.

I look at my clock, and it's 10:00 at night. And then I say, oh, I forgot already what time it is. I look again, and it's 10:20. What happened to those 20 minutes? It seemed like it was instant. So I look again. It's 10:20.

And suddenly, I'm on the upper landing suddenly. Well, of course, I've walked up there. But because it was automatic behavior syndrome, I don't remember a thing. So my perception of time is that no time has passed.

So you can see how some of the stories in folk literature and things arise because of these brain abnormalities that can occur occasionally in people. I could have said, well, I went on a magic carpet or whatever.

OK. Other things that you're probably more familiar with-- nightmares, night terror, somnambulism. Let's talk about these. Nightmares. They happen in dream sleep. They're frightening dreams. Sometimes, they wake you up, OK?

But night terror-- oh, do we need to stop? I'll define these things because I've already excited their curiosity. And then I'll stop, OK? Night terror is you become totally frightened. Sometimes, you don't even know what you're frightened of. The images in your mind can be simple.

I actually still remember-- we don't need that slide anymore. You can turn it off. I still remember a period of night terror when I was a child. That is not dream sleep. That's slow-wave sleep. And yet, the images can be very, very vivid, OK?

And finally, sleepwalking is also an abnormal state that occurs in slow-wave sleep. So you see, even though the brain is showing slow waves, no arousal, indicated by the electroencephalograph, but still, we can get up and walk and do things. And we're actually asleep. My brother gives me vivid descriptions of what I tried to do to him when I was sleepwalking. And of course, I was asleep, so I said, I am not responsible.

OK, we'll take up the discussion of sleep and dreaming next time. And I'll try to remember the video clip.