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ReachMD
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info@reachmd.com
(866) 423-7849

Sleep Architecture and Neurotransmitters of Sleep

Announcer:
Welcome to CME on ReachMD. This episode is part of our MinuteCE curriculum.

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Dr. Yurcheshen:
Hi. I'm Mike Yurcheshen from the University of Rochester, and today I'm pleased to give you a talk about sleep architecture and the neurotransmitters of sleep.

This is a slide that I think does a great job in capturing what sleep macroarchitecture looks like across the lifespan. On the X axis, you'll see age from 5 to 85. And on the Y axis, there's minutes of sleep all the way up to 600. A few points about this slide, number 1, as we age, we require less sleep, so children get more sleep than older adults. Number 2, when you look at the different strata here, sleep latency stays roughly the same but WASO, which stands for wake after sleep onset, increases, so there are more disruptions to sleep as we age. Number 3, some of the percentages of sleep once we get there do change as well. REM sleep remains relatively constant, but slow-wave sleep goes down. It's quite common to see older individuals with essentially no slow-wave sleep at all. And stage N1 sleep tends to go up, which makes sense because if you're waking up more at night, you're spending more time getting back to sleep.

I've broken down the neurotransmitters that are relevant for our discussion today into three different slides. These are the four neurotransmitters that are generally highly active during wakefulness, there's glutamine, the monoamines, acetylcholine, and histamine. And some of these have clinical correlates that you may be familiar with. For instance, acetylcholine, the clinical correlate there is people who have Alzheimer's disease who generally have major disruptions in their cholinergic tone, often have difficulty with maintaining sleep and other behavioral aspects of sleep too, including sundowning. The monoamines, so these are dopamine and norepinephrine, and epinephrine for the purposes of our discussion. And traditional amphetamines, stimulants are highly active in these systems. So they either promote monoamine release or they prevent the reuptake synaptically. And then histamine is another wakefulness-promoting neurotransmitter. And this is centered mostly in the tuberomammillary nucleus clinical correlate there are for people who are taking traditional antihistamines, sedation and drowsiness are a common side effect.

So major neurotransmitters that are active during non-REM sleep, I've selected three here. Of these three, GABA is the main one that is primarily centered in your reticular activating system, which is a widespread neural network in your brainstem. So medulla and the pons, and sometimes into the midbrain as well. GABA is well known clinically, because many of your traditional hypnotics will focus on this, as well as anxiolytics. So benzodiazepines and benzodiazepine agonists are examples of this. And then adenosine, this is everybody's favorite, non-REM sleep regulating neurotransmitter because this is where caffeine impacts, so that's an adenosine antagonist. It's also really highly tied into this concept of sleep pressure. So as we're awake, your adenosine tone builds, and as you sleep, you pay this down. And so it's kind of a barometer of sorts.

The last slide I have is regarding the neurotransmitter regulating REM sleep. And the main one that we really leverage in clinical practice and think about is orexin. So this was discovered in the 1990s and has beyond just sleep-promoting properties. But this is centered in your hypothalamus, with wide-step projections, including into your prefrontal cortex, and into your locus coeruleus, into the thalamus and

others. And people who have type 1 narcolepsy, most of these people have low levels orexin, sometimes called hypercretin, you can measure this. But we've also developed a class of hypnotics, the hypocretin antagonists, which are also leveraged for people who have insomnia.

So that concludes our brief talk. I want to thank everybody for joining today. And I look forward to seeing you again.

Announcer:
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