Capitol Sleep Medicine Newsletter

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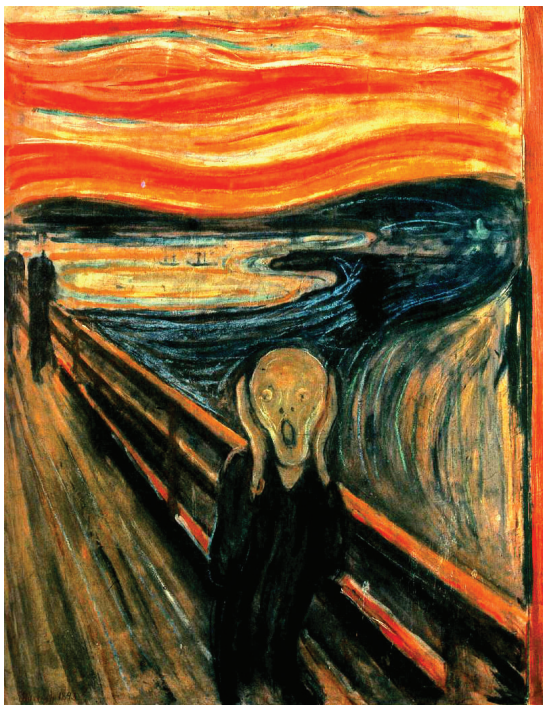
Dreaming though Depression – Part 1

What is exactly is different about the quality of sleep of depressed patients? Not surprisingly, insomnia is a common complaint. This may mean that patients have a difficult falling asleep initially, difficulty getting back to sleep after early morning awakenings, or both.[1] Differences may also be seen in the quality of sleep itself. Classic sleep study findings in depression that have sparked much debate and controversy in the sleep medicine community over the past several decades are a decreased latency in entering rapid eye movement (REM) sleep, an increased amount of REM sleep, and an increase in the amount of rapid eye movements seen per minute, or phasic density, of REM sleep.[2] When seen in patients with depression, once effectively treated, these patients may still have a persisting reduction in the latency to enter REM sleep for up to six weeks following recovery.[3]



Thus, some authors have concluded that these changes in REM sleep may represent trait markers that indicate a vulnerability of the individual for the development of a depressive illness.[4] Further, a decreased REM latency in family members of a patient may increase that patient's risk of depression three fold.[1]

So, if depressed patients go into REM earlier, get more REM, and have a higher power of REM (increased phasic density), then does that mean that REM causes depression? Restricting oneself to a cursory examination of the subject may lead one to believe this idea. In fact, the changes in sleep that can be seen with some of the older antidepressant medications initially seemed to support it. Some of the effective antidepressants have been shown to prolong the latency to enter REM sleep and decrease total REM sleep amount.[5]

However, not all antidepressant medications produce a clear suppression of REM sleep. Responders to desipramine may actually show more REM sleep than non responders.[5] Nefazodone and bupropion have both been documented to increase REM sleep.[6] Contrary to older theories, these findings suggest that an antidepressant medication's ability to decrease REM is not a prerequisite for its clinical antidepressant efficacy.[1] Futher, the polysomnographic finding of a reduced latency to enter REM sleep may be relatively non-specific for a diagnosis of depression, as it may also be seen in narcolepsy, chronic sleep deprivation, or recent withdrawal of a REM suppressing medication or alcohol.

Suicidality can be a dangerous side to depression. Is there a difference in sleep for depressed patients who are contemplating suicide? A study of 165 patients aged 18-68 years were interviewed with the Suicide Assessment Scale after a suicide attempt. This study concluded that frequent nightmares were associated with a fivefold increase in risk for a high suicidality score.[7]

As it has not been definitively demonstrated that decreased REM latency and increased REM are the cause of clinical depression, another consideration is that perhaps the brain is somehow attempting to repair itself through REM sleep. A few studies have shed some light on this.

To test how REM sleep may affect mood, 60 student subjects were tested with the Profile of Mood States (POMS) before and after two nights of laboratory sleep. Students with a pre-sleep depressed mood as determined by the POMS showed a pattern of decreasing negative and increasing positive affect in dreams reported from successive REM periods, when compared to other students. The authors concluded that dreaming may actively moderate mood overnight.[8]

One interesting study of 24 patients comparing REM latency in patients with unipolar from those with bipolar depression demonstrated that REM latency tends to increase as the mood improves in patients with bipolar depression but remains stable in those with unipolar depression. In other words, as a patient with bipolar depression moves away from depression and closer towards mania, the latency to enter REM sleep gradually prolongs towards what would occur normally. One possible explanation for this is that as the patient is less and less depressed, their brain needs less and less REM sleep.[9]

Part II of this newsletter will examine how neuroimaging during sleep has shed some light on the diffences in the brains of sleeping depressed patients before and after treatment.

[1] Biol Psychiatry 1995;37:85-98.
[2] Archives of General Psychiatry 1992;49:651-658.
[3] Arch Gen Psychiatry 1986;43:878-884.
[4] J Psychiatr Res 1984;18:467-475.
[5] Neuroscience and Biobehavioral Review 1990;14:49-63
[6] Am J Psychiatry 1995;152(2):274-276.
[7] Psychiatr Danub. 2006 Sep;18 Suppl 1:85
[8] Psychiatry Res. 1998 Oct 19;81(1):1-8
[9] J Affect Disord. 1996 Nov 25;41(2):125-33