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GUEST EDITORIAL

Secondary insomnia: a myth dismissed

It took the recent NIH State-of-the-Science Panel[1] two days to conclude what has taken the sleep community decades: the traditional concept of secondary insomnia (SI) is poorly understood and lacks adequate scientific basis. They recommended using the term comorbid insomnia (CI) when insomnia coexists with another condition that is salient for sleep disturbance.

Stepanski and Rybarczyk's paper,[2] though conceived long before the appearance of the State-of-the-Science Conference on Insomnia, is very timely. They dissect secondary insomnia with the precision of a skilled surgeon; their scalpel: the deliberate, methodical scrutiny of data. Their comprehensive review points to the conclusion that SI and CI (and primary insomnia) are largely indistinguishable disorders that exhibit comparable treatment response to comparable treatments. Rather than repeat the authors' nicely orchestrated recounting of persuasive articles, my comments will focus on amplifying aspects of SI that I think deserve emphasis.

Causal inference

To fully appreciate the meaning and implication of SI, one must recognize the critical distinction between SI and CI. Causal inference is what separates the two. SI asserts that stimulus A (a disease, disorder, or substance) causes event B (poor sleep), and *causes* is not a casual term. It requires all of the following:

1. Stimulus A must precede event B.
2. Variation in frequency, severity, or duration of A is closely mirrored in all dimensions by variation in B.
3. In the absence of variation of A, B is invariant.

If all three conditions prevail, the requirements of SI are satisfied, and I have labeled such disorders absolute SI.[3] If any one of these conditions is lacking, it would be logically impossible for A to exclusively own causal influence over B. We would have to acknowledge there is at least one other causal agent also impinging on B or part of B has acquired functional independence, conditions I have called partial SI. If these requirements are substantially absent, then A and B are comorbid. Unless the observer takes the trouble to inquire about these requirements, this last scenario can easily be anointed with the SI title (I have called this specious SI), and the likelihood of this misdiagnosis is elevated if the "primary" condition is a high profile SI candidate like pain or depression.

It is also salient to point out that with true CI, the relationship between the sleep disturbance and the other condition is likely to be dynamic. The two conditions may be independent, at other times one may affect the other, and each may assume the primary role at different times. The interconnectedness between the comorbid conditions will be strengthened during spells of flare-ups of either one.

The diagnostic murkiness of SI is compounded by the chronic nature of the syndrome. Indeed, if the primary condition were not a refractory, chronic problem, it would be dispatched (as I have never encountered the argument that treatment should be withheld from the primary condition), and the topic of SI would not arise. The entirety of the diagnosis of SI rests on the ability of the patient to recreate the history of the primary condition/insomnia relationship whose origin may have blossomed many years ago. For example, consider the task difficulty of recalling which came first, depression that is about 3 years old or

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insomnia that is about 3 years old, and recalling how insomnia varied over the years with variation in the depression. The reliability of these data is suspect. In practice, the health care provider usually has a near impossible task of discriminating absolute, partial, and specious SI.

SI Concept Does Harm

The traditional concept of SI does harm. Once providers embrace the concept that A (primary condition) causes B (insomnia), it would be foolhardy to squander health care resources to treat the insomnia directly. Any sleep treatment gains would be cancelled by the primary condition refueling sleep disturbance. Motivated by beneficent intentions that are nonetheless misguided by invalid theory, health care providers have been inclined to deny treatment to people experiencing presumably absolute SI. In an unknown proportion of cases, this extends insomnia suffering which may be severe, aggravates the assumed primary psychiatric/medical condition (as Stepanski and Rybarczyk have shown that the sleep disturbance can assume the role of primary condition), and beckons subsequent incremental psychiatric/medical disability. Untreated insomnia is a health risk factor for depression, anxiety, drug abuse, and other conditions.[4]

Secondary depression

Morawetz[5] published a paper in an obscure Australian journal that has received little attention. The study can claim only modest methodological rigor and the dependent measures do not constitute a robust evaluation, but the results are so stunning, the study is instructive. The author reports on 86 patients in his practice who presented with severe, chronic insomnia. About two-thirds of these individuals also experienced depression spanning the mild to severe range. He proceeded with a self-help format of cognitive behavior therapy targeting insomnia only, ignoring the depression. After about two months of treatment, most (87%) patients demonstrated clinically significant sleep improvement. 70% of the patients who exhibited pretreatment depression and attained insomnia improvement also showed clinically meaningful depression improvement as measured by pre–post change on the Beck Depression Inventory. Among patients who did not show sleep improvement, none showed depression improvement.

Stepanski and Rybarczyk also cited a study that reported (less dramatic) depression improvement in comorbid individuals when only the insomnia was treated.[6] Additional reports of this same phenomenon are available; i.e., depression and other comorbidity improvement follows insomnia gains.[7–9] Indeed, there are more data to support the assertion that depression is secondary to insomnia by virtue of correlated depression improvement with insomnia change, than the reverse. Remarkably, after all these years of asserting that insomnia is secondary to depression, there is virtually no evidence that if you selectively treat depression and ignore the insomnia, the latter improves. In cases of comorbid depression/insomnia, shall we advocate that depression treatment be withheld till the associated insomnia is resolved?

Does secondary insomnia exist?

The answer is an unequivocal yes, but on a much more restrictive scale than has previously been believed. During acute onset of intrusive disorders such as cancer, depression, and pain, there appears to be a clear insomnia response in some individuals that does parallel the course of the primary disorder. And the presence or absence of this causal influence is often more obvious to the patient than the provider. Patients know when their sore back is disturbing their sleep, and these individuals are unlikely to show up at a sleep disorders center. They are going to grab the first doctor they see and ask for a sleeping pill. By the time a patient with SI makes it to a sleep disorders center, they have a chronic condition that is probably more like CI. Sleep specialists are the least likely type of health care provider to treat true SI. I have been studying SI for 10 years, and I do not believe I have encountered a single case of absolute SI.

But even in "clearcut", acute cases of SI, the patient's "nature" for want of a better term plays an important role. There is no disorder that produces a universal insomnia response. Further, SI is not a static condition. Following Spielman's model,[10] insomnia secondary to some precipitant acquires self-sustaining momentum by virtue of the patient's cognitive/-behavioral response to the sleep disturbance. After some unknown period of time, what was SI is now CI.

Conclusion

When plausible theory achieves broad audition, it may attain untouchable status and become highly

resistant to revision despite mounting disconfirming data.[11] Revered theory can become treasured mythology, and allegiance to mythology even among scientists, can be slow to fade. The interests of science and patient welfare are both served by dismissing the myth of SI.

Like traditional conceptualizations of SI I am criticizing, a fair number of the assertions herein have not been empirically demonstrated. We do not exactly know what varieties of SI and CI exist, what is the genesis of these disorders, and what is the long-term clinical import of these relationships. This conclusion speaks to the wisdom of the NIH State-of-the-Science Panel. These relationships are poorly understood and represent a fertile area of much needed scientific inquiry.

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