Insomnia, Hypnotic Ability, Negative Affectivity, and the High Risk Model of Threat Perception

According to Morin (1993), insomnia is second only to pain in terms of the frequency of health complaints. According to The International Classification of Sleep Disorders Diagnostic and Coding Manual-Revised (American Sleep Disorders Association, 1997), psychophysiological insomnia is defined as “a disorder of somatized tension and learned sleep-preventing associations that results in a complaint of insomnia and associated decreased functioning during wakefulness.” Meaning associated with stressful events is generally denied or repressed, manifesting as increased physiological arousal (e.g., muscle tension). Based on the above criteria, psychophysiological insomnia may be regarded as a somatization disorder (American Sleep Disorders Association, 1997). Somatization is diagnosed by exclusion. The High Risk Model of Threat Perception (HRMTP) provides an approach to diagnosis by inclusion (Wickramasekera, 1988, 1993). It identifies specific and quantitative psychosocial and psychophysiological risk factors implicated in the perception of threat that are hypothesized to drive, consciously or unconsciously, somatic symptoms like psychophysiological insomnia (Wickramasekera, 1988, 1992b, 1995). The HRMTP proposes diagnosis of somatoform disorders by inclusion and provides quantitative and relatively modifiable risk factors that can be targeted in therapy. The identification of specific high risk psychosocial factors that unconsciously but independently drive somatic symptoms can have profound implications for diagnostic practice, as well as for therapy and for primary prevention.

Hypnosis is defined as a form of information processing in which voluntarily initiated suspension of peripheral awareness and critical analytic cognition can, in some people, readily lead to major changes in perception, memory, and mood that appear involuntary and may have important behavioral and biological consequences (Wickramasekera, 1986, 1988). Hypnotic ability is a normally distributed individual difference variable unrelated to gender (Barber, 1969; Hilgard, 1965), with a stable test-retest reliability of \( r = .71 \) after 25 years (Piccione et al., 1989), that is believed to have a partly genetic basis (Morgan, 1973; Morgan et al., 1970). The HRMTP states that people who score high (9 to 12) or low (0 to 4) on hypnotic ability are at risk, but for very different reasons (Wickramasekera 1979, 1986, 1988, 1993, 1995). Generally, patients with high hypnotic ability are at risk because they are hypnotized to be psychologically hypersensitive to the perception of threat and sympathetically hyperactive to threat (Wickramasekera, 1998; Wickramasekera et al., 1996a). In fact, highs (Das, 1958; Wickramasekera, 1970, 1976) condition very rapidly, operantly, and reportedly and, hence, are at risk for maladaptive learning (Wickramasekera, 1988, 1993). People low in hypnotic ability are hypothesized to be at risk because they appear to be verbally hyposensitive to emotional threats and prone to parasympathetic dysregulation during chronic threat (Wickramasekera, 1998; Wickramasekera and Price, 1997). A hypothesized deficit in psychological methods of coping may place lows at greater risk, during threat perception, of using external methods of self-soothing (e.g., substance abuse or overeating) or somatization to reduce negative affect (Wickramasekera and Price, 1997). It is also hypothesized that both high and low hypnotic ability are associated with low correlations (incongruence) between verbal report measures of threat perception on one hand and physiological measures of threat perception (e.g., high heart rate, high muscle tension, etc.) on the other (Wickramasekera, 1988, 1993, 1998). Hence, paradoxically, people of high or low hypnotic ability are more likely to be able to keep threatening secrets from their mind but not their body (Wickramasekera, 1988, 1998). Several controlled empirical studies show a relationship between hypnotic ability, physiology (Graffin et al., 1995), and chronic stress related disorders (Wickramasekera, 1988, 1993, 1995). For example, Wickramasekera et al. (1992) reported a relationship between EEG defined insomnia, with pathophysiology (e.g., apnea) excluded, and hypnotic ability. John et al. (1988) and Kelley (1984) reported a positive relationship between hypnotic ability and clinical phobias. Greenleaf et al. (1992) found that in 32 coronary bypass patients, those who scored high or low on hypnotic ability stabilized significantly more slowly (\( p = .05 \)) in an intensive care unit than those who scored in the middle range.

The second amplifying risk factor is high overt or high covert (repressed) negative affectivity (NA) or neuroticism (Wickramasekera, 1988, 1993). This dimension of personality is found in people who frequently report negative emotions and distress across time, cultures, and situations independent of objective stress (Clark and Watson, 1991; Costa and McCrae, 1986). It is related to the number of somatic complaints people present independent of age and pathophysiology (Costa and McCrae, 1985). There is evidence that neuroticism, or negative affectivity, is stable (\( r = .64 \)) across 20 years (Clark and Watson, 1991; Costa and McCrae, 1986) and is also partly genetically based in monozygotic twins reared apart (Tellegen et al., 1988). Wickramasekera (1979, 1986, 1988; Wickramasekera and Atkinson, 1992; Wickramasekera et al., 1992) has hypothesized that high overt or covert NA is a risk factor for threat-related disorders because this chronic negative bias in perception and memory may alter the hypothalamic-pituitary-adrenal axis and immune function. An interaction between hypnotic ability and NA is hypothesized to dysregulate the autonomic nervous system driving clinical symptoms (Wickramasekera, 1988, 1998, in press; Wickramasekera et al., 1996).

Methods

Subjects: The subjects of this study were volunteers obtained from a subset of the patients referred to the Sleep Disorders Centers at Eastern Virginia Medical School.
N scale of the EPI was employed as the measure of neuroticism, as defined in the HRMTP. Eysenck (1983) claims that N is closely related to the degree of lability in the autonomic nervous system (ANS). This lability is an important component of the HRMTP and is theorized to interact with hypnotic susceptibility and catastrophizing to predispose a person to enhanced threat perception and to generate psychological and somatic symptoms.

**Hypotheses:** The prevalence of high and low hypnotizability in insomnia patients will be greater than in the normative group. Insomnia patients will show a bimodal distribution of hypnotizability scores. That is, there is expected to be a predominance of lows (0 to 4 on the HGSHE:A) and highs (9-12 on the HGSHE:A), with very few moderates (5 to 8 on the HGSHE:A). The prevalence of high overt neuroticism scores on the EPI will be significantly greater in the insomnia group than in an age-matched normative group. That is, there is expected to be a predominance of high neuroticism (75th percentile).

**Results**

The numbers of patients in the low, moderate, and high score ranges of hypnotizability for the insomnia patients were 8, 3, and 6, respectively. The distribution for clinical insomniacs was bimodal, as predicted, as opposed to the normal distribution that was expected based on published HGSHE:A norms. Based on a normal distribution of scores, a sample of 17 individuals should have resulted in 2 lows, 13 moderates, and 2 highs. (The estimate was based upon 12.5% low, 75% moderate, and 12.5% high expected distribution in each category.) The difference between the observed frequencies and the expected frequencies, based on published norms, was significant at $\chi^2 = 30.76$, $df = 2$, $p = .00000021$ (Fig. 1).

The numbers of patients in the low, moderate, and high score ranges of neuroticism for the insomnia patients were 0, 4, and 13, respectively. Based on a normal distribution of scores, a sample of 17 individuals should have resulted in 4 low neurotics, 9 moderate neurotics, and 4 high neurotics. (The estimate was based upon scores in the 25th percentile indicating low, 25th to 75th percentiles indicating moderate, and 75th percentile indicating high levels of neuroticism.) The difference between the observed frequencies and the expected frequencies, based on published norms, was significant at $\chi^2 = 24.65$, $df = 2$, $p = .0000445$.

**Discussion**

This study found a bimodal distribution of high and low hypnotic ability people, in a sample of patients with a psychophysiological insomnia diagnosis, referred to a sleep disorders center. A previous study, using three nights in a sleep EEG laboratory on each patient to definitively rule out pathophysiology (e.g., sleep apnea) also confirmed the bimodal prediction from the HRMTP (Wickranauskerka et al., 1992). As predicted by the HRMTP, this study also found that patients with psychophysiological insomnia are likely to score high on a measure of neuroticism, or NA. In fact, it has been hypothesized and found that an interaction between hypnotic ability and cognitive stress can drive up the level of sympathetic
reactivity as measured by skin conductance in a dose response, or linear, fashion (Wickramasekera et al., 1996).

Because both hypnotic ability and neuroticism are stable and partly genetically based (Clark and Watson, 1991; Morgan et al., 1970; Piccione et al., 1989; Tellegen et al., 1988), it is likely that these personality features existed pre-morbidly (before the onset of psychophysiological insomnia) and contributed as risk factors in interaction with other unmeasured HRMTP risk factors (major life changes, accumulated hassles, low support systems, low coping skills, high catastrophizing), to the development and onset of insomnia.

These two risk factors for psychophysiological insomnia are confirmed by independent studies using first EEG and now clinical diagnostic criteria. In terms of therapy, it is hypothesized (Wickramasekera, 1988, 1993) that recruiting the hypnotic ability of the highs, with a hypnotic induction, and reversing its direction of action through training in self-soothing self-hypnosis and desensitization will significantly reduce insomnia. In the case of the lows, biofeedback (Wickramasekera, 1976, 1988, 1993; Zillmer and Wickramasekera, 1987) and cognitive behavioral therapy (Wickramasekera, 1976, 1988, 1993) is predicted to be the most effective short-term form of therapy.

References

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