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IMAGINARY VERSUS REAL LIGHT FOR WINTER DEPRESSION

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Pleurothotonus and the Pisa Syndrome

To the Editor:

The report of Yassa et al (1991) is one of only two systematic studies of the Pisa syndrome that have appeared very recently, the other being that of Suzuki et al (1990). Prior to these studies, there were only a few case reports of this phenomenon. Ekblom et al (1972) coined the term "Pisa syndrome" to describe the presentation of three patients who developed a postural abnormality consisting of leaning to one side (lateral torso flexion), usually accompanied by slight posterior rotation of the torso, while being treated with neuroleptics. Following Pilette (1987), Yassa et al (1991) employed the descriptive term "pleurothotonus" as synonymous with the Pisa syndrome in the title of their paper. Pilette (1987) had pointed out that the term "pleurothotonus" predated that of "Pisa syndrome," and stated that this particular postural abnormality is at least as common as opisthotonus even though it is mentioned less frequently in recent textbooks.

Strictly speaking, Pisa syndrome designates an entity more specific than pleurothotonus. Pleurothotonus delineates an anatomic deviation that could be observed prior to the advent of neuroleptic compounds. As with opisthotonus, it was, in the past, often considered to be a typical presentation of conversion hysteria. Pleurothotonus, also called "tetanus lateralis," designates a physical finding that could be related to any of a number of potential etiologies of dystonic phenomena (see DeSilva et al 1973). It is,

therefore, not quite synonymous with Pisa syndrome, which in the original report implicated neuroleptic treatment in the etiology of the clinical findings. When Ekblom et al (1972) stated that they had "christened this peculiar body posture . . . after the well-known Italian structure," they were designating a "new dystonic syndrome associated with butyrophenone therapy." Therefore, the appropriate synonym for the Pisa syndrome would be "neuroleptic-induced pleurothotonus," or simply "drug-induced pleurothotonus."

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Imaginary Versus Real Light for Winter Depression

To the Editor:

One of the unsettled questions in the research of Seasonal Affective Disorders (SAD) is the contribution of placebo effects to the therapeutic results (Eastman 1991). It is not clear to what extent the response to artificial light is due to nonspecific cog-

nitive reactions to the visual perception of light. We attempted to compare the effects of exposure to real bright light and to placebo bright light. The exposure to the placebo light was effectuated by inducing the imagination of perceiving light through hypnosis.

Fourteen patients were studied (7 men, 7 women, mean age 39.4 years (9.7 SD)). They were drug free before (>1 month) and during the experiment and met the criteria for SAD (Rosenthal et al 1984). The

criterion for entrance to the study was a score of ≥ 13 on the Beck Depression Inventory (BDI) (Beck et al 1961). Seven patients with scores ≥ 2 on the Stanford Hypnotic Clinical Scale for Adults (Morgan and Hilgard 1978) were treated with "placebo light" [i.e., imaginary light (IL)] from 9:00–12:00 AM under dim light conditions (< 50 lux). Another seven patients, who had lower scores, received real light (Vita Light, full spectrum, 2500 lux) from 9:00–12:00 AM (morning light, ML). After trance induction by hypnotic techniques, patients were made to imagine being exposed to bright light (Jenner et al 1990). Two patients did not complete the ML condition. After 4 baseline days, treatment was given during 3 consecutive days. The severity of depression was assessed with the BDI and the Hamilton Rating Scale for Depression (HRSD, 21-item version) (Hamilton 1967) on the days preceding (day 4) and following (day 8) each treatment condition and 10 days later (day 18). The mean BDI score of the total group at first assessment was 21.9 (7.5 SD), the mean HRSD score, 17.1 (5.6 SD). Furthermore, mood was monitored three times daily (7:30 AM, 3:00 PM, and 11:00 PM) by means of self-ratings on the Adjective Mood Scale (AMS) (Von Zerssen 1986). Averages of the self-ratings made on the base-line days (before), during therapy (during), on the 3 days immediately following therapy [after (1)] and on days 8, 9, and 10 after therapy [after (2)] were used as additional outcome variables. For the

analyses, analysis of variance with repeated measures was applied.

The three outcome variables show a significant improvement over time for the entire group (Figure 1) (AMS: $F(3,30) = 7$, $p = 0.002$; BDI: $F(2,20) = 15.4$, $p = 0.000$; HRSD: $F(2,16) = 8.0$, $p = 0.004$). A significant time $\times$ group interaction effect was observed in the AMS and HRSD scores (AMS: $F(3,30) = 4.1$, $p = 0.015$; HRSD: $F(2,16) = 7.2$, $p = 0.006$). Further analysis on the AMS scores showed that improvement disappeared in the withdrawal period after IL, but not after ML (before/during: IL: $F(1,10) = 8.0$, $p = 0.017$; ML: $F(1,10) = 12.4$, $p = 0.006$; before/after(1): IL: $F(1,10) = 0.6$, $p = 0.46$; ML: $F(1,10) = 13.1$, $p = 0.005$ and before/after(2): IL: $F(1,10) = 0.9$, $p = 0.36$; ML: $F(1,10) = 27.3$, $p < 0.001$). For the HRSD scores, there was a significant improvement only after ML (day 4/day 8 IL: $F(1,10) = 1.8$, $p = 0.21$; ML: $F(1,10) = 13.0$, $p = 0.005$ and day 4/day 18 IL: $F(1,8) = 0.2$, $p = 0.71$; ML: $F(1,8) = 12.9$, $p = 0.007$).

It should be noted that groups were small and differed with respect to hypnotizability. Maintaining a trance-like state for 3 consecutive hr turned out to be difficult, and patients often were not able to concentrate exclusively on the perception of the imagined light. Nevertheless, it is tempting to suggest that the data do not support the hypothesis that the long-term

Figure 1. Effects of imaginary light and morning light on AMS, BDI, and on HRSD. Hatched areas indicate treatment interval. Closed symbols correspond to seven patients treated with imaginary light, open symbols denote five patients treated with morning light. In this group, HRSD ratings are missing for two patients.

results of light treatment in SAD patients represent merely placebo effects. They do not disprove it either.

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Why Not ECT for Catatonia?

To the Editor:

The recent report by Ferro et al (1991) in this journal describes endocrinological studies in a 19-year-old woman with acute catatonia. The authors used supportive measures such as rehydration and parenteral feeding, with dantrolene as the only medication. After 15 days with little change, the patient was treated with antibiotics, antipyretics, and low doses of beta-blockers. They also gave benzodiazepines for episodes of agitation. The patient remained in the psychiatric unit for 42 days; she was discharged 2 months after admission "clearly improved."

Though the observations of changes in cortisol during an acute illness is of interest, the demonstration of a normalization of cortisol levels may be as much a reflection of the acute state of dehydration of the patient in the "catatonic" phase, and better hydration and feeding at the "remission" phase.

It is even more puzzling why a specific treatment for catatonia, electroconvulsive therapy (ECT), was not used during a period of extensive hospitalization. The specificity of ECT for catatonia is well docu-

mented (Taylor 1990; Fink and Taylor 1991), as is its life-saving properties in cases of lethal catatonia (Mann et al 1990). Is this another example of the adverse impact of social philosophy on medical practice? We understand that hospitalization of the severely mentally ill was interdicted in Italy by social regulations more than a decade ago, and that ECT is hardly available in that country except in some private hospitals. Is this another example of the impact of the anti-psychiatry movements throughout the world on patient care?

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