LIGHT THERAPY: THEORY AND PRACTICE

BY
NORMAN E. ROSENTHAL, MD

Exposure to environmental light has been recognized for its salutary effects on mood for hundreds of years. Aretaeus noted in the second century AD., "Lethargies are to be laid in the light and exposed to the rays of the sun, for the disease is gloom." The solarium was regarded as a valuable room in any place of healing, and sunlight, along with fresh air, was widely recommended as a panacea for a variety of ills.

Following these lines of thought, it seemed quite logical for more modern minds to speculate that artificial light might also have health-inducing properties. For well over a century, physicians have suggested the use of artificial light for many different conditions, often without scientific backing.

Nevertheless, in some skin conditions, such as psoriasis and neonatal jaundice, light has been shown scientifically to have beneficial clinical effects, and the term "phototherapy" has become associated in the physician's mind with the cutaneous effects of ultraviolet light. In the past fifteen years, new discoveries about the biological and psychological properties of light have expanded our concept of phototherapy or light therapy to include the effects of visible light, along with ultraviolet or infrared light, on the brain.

A seminal development in the progression of our understanding of the new light therapy was the discovery by Lewy and colleagues that environmental light, if sufficiently bright, is capable of suppressing the nocturnal secretion of melatonin by the pineal gland. Animal studies had shown that melatonin is secreted in an endogenous circadian rhythm, generated by the superior cervical nuclei of the hypothalamus, the body's clock, and that this pattern of secretion can be modified by allowing light to impinge on the retina and produce results via the direct connections between the retina and the hypothalamus. The pincal gland was already known to orchestrate many seasonal rhythms in animals by its capacity to translate photoperiod, or day length, into a biochemical signal, such as the duration of nocturnal melatonin secretion.

In the early 1980s, Rosenthal and colleagues described a new condition, seasonal affective disorder (SAD), characterized by regular periods of depression in the winter alternating with non-depressed periods in the spring and summer. Unlike classic depressives, SAD patients generally oversleep, overeat, crave carbohydrates, and gain weight during the winter, and these vegetative symptoms often precede mood changes such as sadness or anxiety. The seasonal mood fluctuations of SAD patients resemble the seasonal rhythms of behavior in animals, and so it was logical for Rosenthal to hypothesize that their depressions might be influenced by seasonal fluctuations in environmental light and thus be treatable by the use of bright artificial light to counter the predictable decline of natural light during the winter months. A series of controlled light treatment studies conducted by Rosenthal and associates at the NIMH bore out this prediction, and light therapy is now recognized as a treatment of choice for SAD.

Initially, researchers used 2500lux light units, which deliver to the user's eyes about five times as much light as would be absorbed from ordinary in-

door lighting. Subsequently, Terman and colleagues made an important advance by showing that more intense light (10,000 lux), delivered by a box with an opening slanted towards the user's eyes, allowed therapy to be effective with short-duration treatment – as little as 30 minutes in some cases.

Commercially available fixtures are generally recommended over homemade devices because they are generally electrically safer, have demonstrated efficacy, and incorporate features that protect the eyes (such as light dispersion and screens that eliminate ultraviolet rays). Fluorescent light is generally preferred over incandescent, because the small point source of the latter is more conducive to retinal damage. Ordinary white fluorescent bulbs are sufficient; "full-spectrum light" is unnecessary. In fact, the higher levels of ultraviolet light emitted by some "full-spectrum" lamps are undesirable.

Patients frequently ask, "Can't I just replace all the light bulbs in my house with full-spectrum lights?" This is not feasible, however, because intensity, not spectrum, is the variable most critical for obtaining an antidepressant effect.

Much debate has centered around the timing of light therapy, but the latest studies suggest that light therapy can be effective regardless of when it is administered during the day. Since inconvenience is frequently cited as a reason for not complying with light therapy, it is important that the clinician emphasize the use of light at a time that fits in well with the patient's schedule, rather than focus on a time of day – such as the early morning – regarded by some as superior.

Since the symptoms of SAD may be caused by light deprivation, and since light deprivation can be caused by cloudy days or windowless offices, light therapy may be helpful even outside the main season of risk. Side effects of light therapy, such as eyestrain, headache, fatigue, irritability or hypomania, generally respond to reduction of dosage (decreased duration of therapy or increased distance from the light source). Insomnia may occur if light therapy is used too late in the day. Although the theoretical possibility of retinal damage has been raised, to date there is no evidence that any retinal damage has resulted from properly administered light therapy. Light therapy can even be administered concornitantly with antidepressant medications, and such a combination may permit the use of lower medication dosages, resulting in fewer side effects.

There is no general consensus as to how light therapy works. There is evidence both for and against the following theories: (1) winter depressive symptoms result from abnormally delayed circadian rhythms, which are shifted to be earlier by light therapy administered in the morning; (2) winter depressive symptoms are due to abnormal patterns of melatonin secretion, which are normalized by light therapy; (3) winter depressive symptoms result from deficient serotonergic transmission, a situation that is helped by light therapy; and (4) winter depressive symptoms result from inadequate adjustment of the eyes to declining light levels, and increasing environmental light overcomes this inadequacy. Ongoing research is focusing on explorations of these theories.

The success of light therapy for SAD has encouraged researchers to extend this treatment to other conditions, with mixed results. Some studies have reported positive responses in patients with nonseasonal depression, premenstrual syndrome (late luteal dysphoria), bulimia, and alcohol withdrawal symptoms. Further work in these areas is clearly warranted. Beneficial results have also been obtained in certain cases by using the known capacity of light administration and light restriction to shift circadian rhythms. Cases where this therapy have been used include those of patients with stable circadian rhythm abnormalities such as delayed sleep phase syndrome (DSPS) or advanced sleep phase syndrome (ASPS).

Patients with DSPS have trouble falling asleep and waking up at conventional times; they can be helped by being exposed to bright light in the morning and having their environmental light restricted in the afternoon and evening. ASPS is most common in the elderly; their tendency to fall asleep and wake at inconveniently early hours can be alleviated if they are exposed to bright light at night.

Unstable and transient problems with circadian rhythms, such as those that occur with jet lag and shift work, are harder to treat. If light exposure or restriction are employed therapeutically, it is critical that they be used at the right time, as inappropriately timed interventions can actually make the symptoms of these conditions worse.

In conclusion, the physician should regard light as a drug with many potential indications, not all

of which are now fully recognized. Light is a versatile therapeutic tool that can be combined with other therapies and, if properly supervised, has few side effects. The biochemical effects of light on the brain are not well understood, but the discovery of these effects promises to allow new insights into the pathophysiology of disorders of mood and biological rhythms.

References

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Guidelines to Treatment of SAD

The following guidelines presume that the patient presents in the early fall, when symptoms are mild, and becomes progressively more depressed as the winter deepens. Treatments can be layered sequentially, as indicated by the patient's response. If the earlier steps suffice, there is no need to progress to the next level. If there is a need to progress to medication, factors such as ambient light levels outdoors and in the patient's indoor environment, as well as the patient's stress level, need to be taken into account at all times.

1. Encourage daily walks outdoors; the patient may look up at the sky but never directly at the sun. Enhance indoor lighting with regular lamps and fixtures.

2. Set a timer on a light to go on early in the morning in the patient's bedroom. Consider a dawn simulator for a more naturalistic artificial dawn.

3. Initiate light therapy with a 10,000-lux box, starting with 10 to 15 minutes in the morning or at any time of day that is convenient for patient. Increase the duration if symptoms become more severe, up to 45 minutes twice a day. Although there is no reason why this time should not be increased even further (provided side effects are not severe), 90 minutes per day tends to be the maximum practical amount for most patients. The light box can be left on for background lighting when it is not in active use.

4. Aerobic exercise, preferably in combination with exposure to bright light, may be quite helpful.

5. To help the patient manage stresses, suggest vacations (preferably in the south) at strategic times, and provide support, counseling and therapy. For example, remind the patient that the problem is not his or her fault, but a medical condition; correct cognitive distortions, eg, "I am no good, and I never was any good."

6. Start medication, generally an SSRI, though bupropion would be a reasonable alternative. If one medication is unhelpful or produces unacceptable side effects, try a different medication. Raise dosage until the optimum level is reached.

7. Consider medication combinations, eg, an SSRI plus bupropion, or an SSRI plus lithium. Lithium is generally of little use in itself, but it may be valuable as an enhancer of other antidepressants.