Respiratory disorders in neuromuscular diseases

(JUNE 2005)

TO THE EDITOR: In his review of respiratory disorders in neurologic diseases,[1] Dr. Aboussouan states that, because of the risk of hypercarbia, there is a limited role for oxygen therapy, and that it should probably be limited to patients already on ventilatory support or those in whom hypoventilation has been excluded.

Aside from its role in hyperbaric oxygen therapies, oxygen therapy has one main role, to correct hypoxemia. As the partial pressure of oxygen falls in the blood, there comes a point where the slope of the oxygen dissociation curve begins to fall precipitously, and oxygen-carrying by hemoglobin is adversely affected. This results in inadequate tissue oxygenation and its consequences. Hypercarbia may result from the oxygen therapy, but *not* treating hypoxemia because of the threat of hypercarbia is fraught with danger.

Therefore, it would be better to say that the role of oxygen in the therapy of neurologic diseases is to correct hypoxemia, and that oxygen therapy should be used judiciously, titrated carefully, and monitored closely. If hypercarbia develops, there are means such as noninvasive ventilation to correct this and continue to allow adequate oxygenation.

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IN REPLY: Dr. Hirsch's comments are certainly correct. However, the concern regarding oxygen supplementation in the setting of diverse neuromuscular diseases stems from experience and from reports that even low-flow oxygen supplementation has been associated with life-threatening elevations in the partial pressure of carbon dioxide (arterial).[1] Moreover, reliance on oxygen supplementation instead of ventilatory support in such patients has been associated with higher rates of pneumonia and hospitalization.[2]

We do not necessarily avoid oxygen because of the threat of hypercarbia. Rather, as stated in the review and based on the available evidence, we believe it is appropriate to either exclude hypoventilation as a cause of hypoxemia or ensure that ventilation is supported before oxygen supplementation is prescribed.

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Erectile dysfunction

(APRIL 2005)

TO THE EDITOR: In the April 2005 *Cleveland Clinic Journal of Medicine*, Dr. Mikhail reviewed the management of erectile dysfunction for the primary care physician.[1] It is true that the development of the selective phosphodiesterase type 5 (PDE-5) inhibitors has made primary care physicians—rather than urologists—the primary prescribers of these drugs,[2] and has made them the front line in the treatment of erectile dysfunction.

Most cases of erectile dysfunction are associated with diagnosed, and often undiagnosed, cardiovascular disease. It is associated with traditional risk factors such as diabetes mellitus, hypertension, dyslipidemia, or hypertension.[3] Similarly, it could point to a coexisting psychological or psychiatric disease.[4]

The effectiveness of PDE-5 inhibitors in various types of erectile dysfunction has made the previous exhaustive distinction between organic and nonorganic (psychogenic) erectile dysfunction less critical at times. However, the ability to make those distinctions is necessary when therapy with these agents fails. In a recent evaluation of 115 patients with erectile dysfunction, we noted that the presence of night or morning erection (nocturnal penile tumescence) and erection with foreplay ($P < .008$) but not with a sexual partner ($P < .036$) suggested a nonorganic (psychogenic) cause of erectile dysfunction. We feel that identifying these patients when therapy with PDE-5 inhibitors fails will help improve the management of these patients. We also feel that although PDE-5 inhibitors help both organic and nonorganic erectile dysfunction, appropriate identifi-

cation by clinical means may reduce the overuse of PDE-5 inhibitors.

The need to identify patients needing further evaluation when therapy fails should not be overlooked.

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2. **Wysowski DK, Swann J.** Use of medications for erectile dysfunction in the US. J Urol 2003; 169:1040–1042.
3. **Solomon H, Man JW, Jackson G.** Erectile dysfunction and the cardiovascular patient: endothelial dysfunction is the common denominator. Heart 2003; 89:251–253.
4. **Feldman HA, Goldstein I, Hatzichristou DG, Krane RJ, McKinlay JB.** Impotence and its medical and psychosocial correlates: results of the Massachusetts Male Aging Study. J Urol 1994; 151:54–61.

IN REPLY: I appreciate the comments of Drs. Akpunonu and Mutgi, and I agree with them that it is necessary to identify specific causes of erectile dysfunction when therapy with PDE-5 inhibitors fails. Thus, psychiatric evaluation is indicated if a psychogenic cause of erectile dysfunction (eg, persistence of nocturnal erection) is suspected during the patient's interview.

However, I do not share their feelings that the distinction between organic and psychogenic causes of erectile dysfunction by clinical means may necessarily reduce the overuse of PDE-5 inhibitors. Even if a psychogenic cause is identified, I believe it is reasonable to initiate treatment with PDE-5 inhibitors alone or in conjunction with psychotherapy for several reasons.

First, PDE-5 inhibitors were equally effective in patients with psychogenic and organic erectile dysfunction.[1] In addition, these agents proved efficacious in treating erectile dysfunction associated with the use of antidepressants.[2]

Second, as I mentioned in the article,[3] the etiology of erectile dysfunction is frequently multifactorial, and other causes such as occult atherosclerosis or cardiac disease may coexist with the psychogenic disorder.

Third, since the psychogenic symptoms may be further aggravated by the existence of erectile dysfunction, it is conceivable to assume that improvement of erectile dysfunction by the PDE-5 inhibitors could ameliorate the underlying psychogenic disease, although this concept was not investigated in clinical trials.

It should be emphasized that partner problems may also be a cause of nonorganic erectile dysfunction, and this issue should be addressed when taking the history, as I outlined in **TABLE 2** in the article.[3] Indeed, the patients described by Drs. Akpunonu and Mutgi might suffer from this problem, given the fact that their erectile dysfunction was evident only with their sexual partners, whereas their erection ability seemed intact at nighttime and with foreplay.

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3. **Mikhail N.** Management of erectile dysfunction by the primary care physician. Cleve Clin J Med 2005; 72:293–310.

CORRECTION

Migraine aura without headache

(JUNE 2005)

Due to a copy-editing error, the wrong authors were listed for reference 1, page 533, in the article by Dr. Robert S. Kunkel, "Migraine aura without headache: benign, but a diagnosis of exclusion" (*Cleve Clin J Med* 2005; 72:529–534). The correct reference is as follows:

1. **Headache Classification Subcommittee of the International Headache Society.** The international classification of headache disorders. 2nd edition. Cephalalgia 2004; 24(suppl 1):26–31.