April 2002
Vol. 12 No. 2

American Journal of Pain Management

The Official Journal of the American Academy of Pain Management

TEXAS TECH UNIVERSITY
HEALTH SCIENCES CENTER
AT AMARILLO
School of Pharmacy

To the Editor:

In response to the letter by Mazin Ellias, MD. . . he is absolutely correct in that Ambien® and Sonata® are not benzodiazepins but a new group of sleep medications of the pyrazolopyrimidine class. The sentence on page 146 of the article actually should have read, "New generation non-benzodiazepines, such as Ambien® and Sonata®, have been found not to interrupt the normal sleep structure, but they do not increase delta wave sleep." In the last sentence he adds that "the addition of a tricyclic antidepressant can also help for both the pain and for the modification of sleep." We specifically left this out as the article mainly focused on fatigue. Tricyclic antidepressants are well known to help with pain modification. Initially, they will improve sleep. However, the improvement in sleep is often short-lived and wears off in three to six months. In addition, the improvement is in stage I or II sleep; and tricyclic antidepressants interfere with delta wave sleep actually leading to an increase in fatigue.

–Randall L. Oliver, MD
Evansville, Indiana

To the Editor:

Approximately 45% of patients over the age of 60 develop neuralgia following herpes zoster infection (1). Current treatments for postherpetic neuralgia include topical capsaicin, anticonvulsants, and tricyclic antidepressants (2); however, treatment with these drugs is often less than optimal due to potential systemic side effects, drug interactions, temporary worsening of pain, and/or delayed onset of action.

This report describes a 78-year-old female who presented with herpes zoster dermatitis on the left side of her face without ocular involvement. The patient complained of frontal and periorbital pain in the areas of the lesions, which she rated as a 3.5 or 4 in intensity on a scale ranging from 1 = *mild pain* to 5 = *excruciating pain*. She had obtained no relief from approximately 2 weeks of treatment with carbamazepine (Tegretol®). The patient had a history of hypertension, cholecystectomy, and carotid disease, and was taking nifedipine (Procardia®) and enalapril (Vasotec®).

Based on reports of headache pain relief with selective local chemodenervation (3,4), it was decided to inject botulinum toxin type A (Botox®) into the affected regions. Botulinum toxin type A was diluted with nonpreserved normal saline to a concentration of 20 units/mL. A total of 40 units of botulinum toxin type A was injected subdermally into the left frontal area in a 4 x 4 grid pattern, with injections placed 1 cm apart. Injections were administered at least 1.5 cm above the brow to avoid brow and lid ptosis.

At the patient's first follow-up one month after treatment, she reported an 80% improvement in pain. Her pain intensity rating at this visit was 1 on the 1 to 5 scale. Two weeks later (6 weeks after the initial injection), the patient reported a recurrence of pain and returned for retreatment. At this visit, 30 units of botulinum toxin type A were injected into the same sites. Again the patient reported improvement.

The patient subsequently returned for 4 more injections spaced at approximately 2-month intervals. At the patient's most recent visit, the total dose was increased to 70 units injected into 16 sites in a 4 x 4 grid pattern in an attempt to increase duration. To date, the patient has not reported any adverse side effects from the medication.

The mechanism of action of botulinum toxin type A is the inhibition of acetylcholine release from motor nerve terminals; however, the mechanism by which it may exert its apparent effects on postherpetic pain is unclear. A neuromuscular effect of botulinum toxin type A in this patient can, of course, not be ruled-out. However, results of one preclinical study suggest that botulinum toxin type A may inhibit substance P release from trigeminal nerve terminals of the isolated rabbit iris sphincter muscle (5). Results of another preclinical study suggest that botulinum toxin type A may have a nociceptive effect when injected subcutaneously into the subplantar surface of mice in the absence of overt neuromuscular weakness (6). Further scientific exploration of the putative benefits of botulinum toxin type A in