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Nicotine Reduction and Bupropion

To the Editor:

In a recent article in CHEST, Dale and colleagues (May 2001) reported on factors that are predictive of successful smoking cessation and noted that the first 2 weeks of treatment were the “most crucial.” We also noticed this time period and found no significant immediate, 3-month, or 6-month reduction or quit rate differences between 1 month and 2 months of use of bupropion SR, 300 mg/d.2 The results of the study of smoking cessation by Dale et al.1 as well as the work of others in antidepressant therapy,3 suggest very limited differences between bupropion SR dosing schedules of 150 and 300 mg/d.

We recently completed a treatment program evaluation to compare the relative effectiveness of 150 mg/d vs 300 mg/d bupropion SR as part of nicotine reduction therapy with older American veterans. Seventy-three patients completed all project requirements and were the focus of this study. Treatment condition assignment (150-mg group, 59 patients; 300-mg group, 39 patients) was random, with patients in the 150-mg group having the option to receive 1 month of bupropion therapy at 300 mg/d (our standard dosage) after the 2-month follow-up. All patients signed consent forms and received the same behavioral treatment during the first session. Patients returned after 2.5 weeks to discuss their progress and difficulties, to receive more behavioral change information, and to receive bupropion SR. Subjects were followed-up by mail 2 months after the end of the second session.

The typical veteran was a white male, aged 57 years. There were no significant (p < 0.05) differences between groups as a function of demographic factors, treatment motivation variables, or health habits. About 26% of patients reported quitting all nicotine use at follow-up, and 50% of those still using tobacco claimed nicotine reduction. Neither the group that quit (χ² test, 1.35; p = 0.25) showed significant differences; however, side effect profiles tended to be different. In response to a yes-no question concerning the presence of side effects, a higher percentage of patients in the 300-mg group complained of side effects than did patient in the 150-mg group (χ² test, 3.47; p = 0.06), with the total number of side effects averaging 2.6 per patient in the 300-mg group vs 1.2 per patient in the 150-mg group (t test, 1.92; p = 0.06). Weight change did not vary significantly between groups.

Larger studies with fewer methodological limitations are needed. However, this program evaluation indicates that 1 month of bupropion SR therapy at 150 mg/d may provide the maximum benefit for minimal expense and risk.

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Significant Omission in Antithrombotic Supplement

To the Editor:

In reviews of the treatment of venous thromboembolism, Hyers and colleagues1–3 and the researchers in the 263 articles that they referenced neglected to mention a randomized trial by Nielsen et al.4–5 comparing heparin and a phenprocoumon anticoagulation therapy with phenylbutazone therapy in patients with deep venous thrombosis (DVT). This is the only published randomized controlled trial comparing the treatment of DVT patients using standard anticoagulation therapy with control subjects who had not received anticoagulation therapy. It was a negative study with 1 of 45 anticoagulated patient dying of pulmonary emboli (PEs) and 0 of 42 patients receiving phenylbutazone therapy experiencing fatal PEs.

In the 1995 review, Hyers et al.1 stated, “Patients with DVT or PE should be treated with IV heparin or adjusted-dose subcutaneous heparin sufficient to prolong the activated partial thromboplastin time to a range that corresponds to a plasma heparin level of 0.2 to 0.4 U/mL. This grade A recommendation is based on level 1 studies in patients with PE6 and DVT.7–9 The placebo-controlled randomized trial by Barritt and Jordan6 of patients with PE is too old, small (n = 35), and flawed to be considered as proof of the efficacy of anticoagulant therapy.10 Barritt and Jordan6 made the diagnosis based on clinical suspicion. We now know that only about 27% of patients suspected of having PEs actually have the diagnosis confirmed by angiogram or lung scan.11 Of the five deaths in the control group, cases 1 and 4 had chronic septic cavitation of the lungs, with no recent embolization. The patient in case 2 had thrombophlebitis and thrombosis of the hepatic veins but no documented autopsy evidence of PE. In the patient in case 5, the cause of death was cerebral infarction. Consequently, only case 3 would meet a modern definition of fatal PE: “massive fresh emboli present in the main pulmonary artery, or in at least two lobar arteries, demonstrated post mortem in patients in whom no other cause of death was found.”12 None of the other randomized trials cited in the later reviews concerning DVT or PE had control subjects who were not anticoagulated.7–9,13–16 Therefore, they do not provide “level 1” evidence of the efficacy of anticoagulant therapy in patients with PEs or DVT.

Heparin and vitamin K antagonists became the standard treatment for DVT and PE patients in the 1940s before the