Abstracts

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Alternative Smoking Cessation Aids: A Meta-analysis of Randomized Controlled Trials

Conclusion: Acupuncture and hypnotherapy both appear useful in smoking cessation. Additional evidence is needed to determine whether alternative interventions are as efficacious as pharmacologic therapies.

Summary: Smoking is estimated to cost the U.S. economy $210 billion each year and is the most preventable cause of morbidity and mortality in North America (Chandler MA, Renard SI, Chest 2010;137: 428-35; Rehm J et al, Stud Alcohol Drugs 2007;68:886-95). There are 51 million smokers in North America, and each year half of them attempt to quit at least for 1 day. (MMWR 2009;58:1227-82). Less than 50% of smokers attempting smoking cessation use pharmacologic aids such as nicotine replacement therapy, bupropion, or varenicline. Alternative methods of smoking cessation include acupuncture, stimulating specific acupoints on the ear, hypnotherapy, inducing an altered state of consciousness broadly defined as a cessation aids. They systematically searched the Cochrane Library, EMBASE, Medline, and PsycINFO databases through December 2010. They included trials that reported cessation outcomes as point prevalence or continuous abstinence at 6 or 12 months. The authors identified 14 relevant trials: five trials investigated acupuncture (823 patients), four trials investigated hypnotherapy (273 patients), and four investigated aversive smoking (99 patients). Estimated mean treatment effects were acupuncture (odds ratio [OR], 3.52; 95% confidence interval [CI], 1.03-12.07) hypnotherapy (OR, 4.58; 95% CI, 0.98-21.01), and aversive smoking (OR, 4.26; 95%, 1.26-14.88). The evidence concludes that is reasonable evidence that acupuncture and hypnotherapy are both useful as alternative stop smoking aids. The authors also feel the evidence is sufficiently strong that physicians should promote the use of acupuncture and hypnotherapy as alternative smoking cessation aids. The use of aspirin to reduce VTE events, however, is controversial. The Aspirin to Prevent Recurrent of VTE (ASPIRE) study is ongoing and has recruited, at the time of publication of this report, 822 patients with a first or recurrent VTE. Patients were randomly assigned to aspirin (100 mg daily) or placebo and are under follow-up for a first recurrence of symptomatic and objectively confirmed, nonfatal or fatal, deep venous thrombosis using an intention-to-treat analysis approach. This study is expected to report results in 2012. If this study confirms the current conclusion in the report abstracted here, we may be on the verge of a paradigm shift in how patients with unprovoked VTE are treated long-term.

Aspirin for Preventing the Recurrence of Venous Thromboembolism

Conclusion: In patients with unprovoked venous thromboembolism (VTE), who discontinue anticoagulation treatment, aspirin reduces risk of VTE recurrence without an increased risk of major bleeding.

Summary: Patients with unprovoked VTE who discontinue anticoagulation therapy with vitamin K antagonists have about a 20% incidence of VTE recurrence within 2 years (Schulman S, N Engl J Med 1995;332: 1661-5; and Pandolfi P, Ann Intern Med 1996;125:1-7). In studies of prophylaxis after VTE, aspirin has been associated with a risk reduction ranging from 20% to 50% (Antiplatelet Trialists’ Collaborative, BMJ 1994; 308:235-46). One small randomized trial of 39 patients has previously suggested a benefit of aspirin in reducing recurrence of venous thromboembolism (Steele P, Lancet 1980;2:1328-9). The goal of this study was to assess the potential benefit of aspirin to prevent recurrence of VTE after discontinuation of a vitamin K antagonist therapy. The authors performed a multicenter investigator-initiated double-blind study. Patients included in the study had a first-ever unprovoked VTE and had completed 6 to 18 months of vitamin K antagonist therapy after diagnosis. They were then randomly assigned to aspirin (100 mg daily) or placebo for 2 years. There was an option to extend study treatment. Primary efficacy outcome was recurrence of VTE. The primary safety outcome was major bleeding. VTE recurred in 28 of 205 patients who received aspirin and in 43 of 197 patients who received placebo (6.6% vs 11.2% per year; hazard ratio, 0.58; 95% confidence interval, 0.36-0.92; median study period, 24.6 months). With a median treatment of 23.9 months, 23 patients taking aspirin and 39 taking placebo had VTE recurrence (5.9% vs 11.0% per year; hazard ratio, 0.55; 95% confidence interval, 0.32-0.92). One major bleeding episode occurred in each group, and adverse events were similar in the aspirin-treated and placebo-treated groups.

Clinical Prediction Rule to Estimate the Absolute 3-Year Risk of Major Cardiovascular Events After Carotid Endarterectomy

Conclusion: Clinical risk models can be developed to predict major adverse cardiovascular events (MACE) in the first 3 years after carotid endarterectomy (CEA).

Summary: CEA is one of the most frequently performed procedures by vascular surgeons. Reducing adverse events in patients undergoing CEA has concentrated on the perioperative period. However, patients with carotid atherosclerosis have systemic atherosclerosis as well, and efforts should be made to reduce MACE in the years after CEA. The authors contend initiation of treatment and aggressiveness in treatment to reduce cardiovascular events after CEA should be determined by the absolute level of risk of cardiovascular events in this patient group. They note that prediction of secondary cardiovascular events after CEA is an area that has not been relatively well explored. In this report, they sought to develop a clinical score chart to stratify patients for absolute risk of MACE after CEA. The prediction model was developed in a consecutive cohort of 1138 patients who underwent a CEA between 2002 and 2009. The primary end point was MACE (myocardial infarction, stroke, and cardiovascular death). Potential predictors (n = 14) were entered into a Cox proportional hazard model. Backward stepwise regression and internal validation with boot strapping techniques was used to correct for over-fitting. A score chart was constructed in an effort to provide an applicable clinical tool. The chart divides patients into four risk groups. The performance of the model was assessed in terms of risk stratification, discrimination, and calibration. In the cohort studied, during a mean follow-up of 2.28 ± 0.95 years, there were 148 MACE. This corresponded to a cumulative incidence of 13%. Clinical predictors in the final model included were age, history of peripheral vascular disease, history of coronary disease, systolic blood pressure, smoking, clinical presentation, use of antihypertensive drugs, serum creatinine levels, and presence of a left internal carotid artery stenosis. Discrimination of the final model by C statistic was 0.69 (0.64-0.73). Calibration showed a good overall fit (Gronnesby Borgan, P > .39). The observed incidence of MACE in the four risk groups was 6%, 9%, 19%, and 35%, respectively, suggesting overall good risk stratification.

Comment: It is difficult to know what to do with this sort of information. All of the patients, included in this study that were stratified for additional MACE during the subsequent 3 years after CEA had, in fact, undergone CEA. Therefore, all the patients had serious atherosclerotic disease. Even if one can stratify risk of MACE in the subsequent 3-year period, why would one not offer “less-risk” patients the same level of aggressive treatment as “higher-risk patients? One can certainly argue that all patients who have undergone CEA, regardless of predicted further risk of MACE, should be offered maximal treatment of their atherosclerotic risk factors. The most important aspect of this report may not be the risk prediction score but that it highlights the fact that subsequent MACE after CEA are frequent. All patients with any form of atherosclerosis that has led to surgical intervention deserve maximal medical management of their atherosclerotic risk factors.