Anticoagulating Elderly Patients in Atrial Fibrillation

To the Editor:

The recent article in CHEST, by Drs. Marine and Goldhaber (April 1998),1 is well-reasoned. There are indeed great hazards in anticoagulating elderly patients. However, significant hazards exist in any group of patients requiring anticoagulation. Unfortunately, aspirin is only about 20% effective in preventing platelet aggregation compared to coumadin. New drugs are or will be available that may add a modest increase to the efficacy of aspirin.

In my own experience, most elderly patients cardioverted from atrial fibrillation will not maintain sinus rhythm or will require one or more antiarrhythmic drugs that also have very serious adverse effects and may be very expensive. Since a number of patients do not recognize recurrence of atrial fibrillation, they are then exposed to the risk of stroke if not anticoagulated.

Obviously, in any patient on coumadin, close supervision of the prothrombin time and international normalized ratio (INR) is essential. I have always experienced a great deal of anxiety with my anticoagulated patients. Over many years, I have had two patients under 60 years of age with prosthetic valves, with anticoagulation carefully controlled, die of massive cerebral hemorrhage, and one patient under 70 years of age with a prosthetic valve recover from a massive subdural hemorrhage. In the last 2 years, in clinical research projects, I have lost one 74-year-old patient with a massive cerebral hemorrhage with INR maintained about 2.2. During this same period, I have seen two patients who succumbed to cerebral embolism with atrial fibrillation before they could be brought under anticoagulation.

AFFIRM (atrial fibrillation follow-up study sponsored by NHLBI), a 5-year, two-arm study comparing rate control vs rhythm control with all rate control patients anticoagulated and probably the majority of rhythm control patients anticoagulated (age ≥65 unless having a major risk factor), may provide valuable information to help answer the questions posed by the authors.

I certainly agree with the need for a major study looking at aspirin (with or without one of the newer antiplatelet drugs) vs coumadin in the very elderly group discussed.

Gordon L. Maurice, MD, FCCP
Associate Clinical Professor Emeritus of Medicine
(Cardiology) OHSU
Associate Director E.A. Chiles Research Institute
Co-Director Cardiovascular Research Department
Portland, Oregon

REFERENCES

Respiratory Effects of the Dead Sea
A Historical Note

To the Editor:

I read with interest the report by Kramer and colleagues about their experiences performing pulmonary rehabilitation at the Dead Sea (March 1998).2 I would like to bring to light an early proposal for the possible therapeutic effects of the Dead Sea environment on pulmonary function. Sir Richard Francis Burton, the 19th century explorer, linguist, and early anthropologist, had numerous experiences visiting and living in cities which served as sanatoriums for patients with respiratory problems. He observed that the geographic preferences of physicians had seen an ever-changing variety of locations. In his memoirs, dictated to his wife in 1876, he made a suggestion: “I proposed to utilize the regions about the beautiful Dead Sea, about thirteen hundred feet below the level of the Mediterranean, where oxygen accumulates, and where, run as hard as you like, you can never be out of breath. This will be the great Consumptive Hospital of the future.”2 Burton, who had a lifelong interest in medical affairs, would be gratified to see that the valid basis for his proposal has now been demonstrated.

V. Theodore Barnett, MD, FCCP
Section of Respiratory and Critical Care
University of Illinois at Chicago
Chicago, Illinois

REFERENCES

Levels of Soluble L-Selectin and E-Selectin in Heatstroke and Heatstress

To the Editor:

We have recently shown that lymphocyte expression of several adhesion molecules is altered in heatstroke and heatstress and not normalized after cooling3 and that circulating levels of endothelial cell activation markers are elevated in heatstroke.2 Several studies have shown that the levels of soluble adhesion molecules, such as sL-selectin and sE-selectin, correlate with the concentration of molecules expressed on the cells and with the activity of various diseases.4,5 We determined serum levels of sL-selectin and sE-selectin (ELISA; Bender MedSystem; Vienna, Austria) in 25 consecutive heatstroke patients before and after cooling. 14 subjects who were exposed to the same living conditions of heatstroke patients but did not develop heatstroke (heatstress), and 13 normal control subjects. The characteristics of the study population have been published.3 Student’s t test with Bonferroni correction for multiple comparisons was used for comparing the three groups, and Pearson’s correlation coefficient was used for measuring correlation.

The mean (±SE) sL-selectin level was elevated in heatstroke (867±60 ng/mL, p=0.14) and heatstress (1129±35 ng/mL, p=0.0003) compared to normal control subjects (676±30 ng/L).

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