Thrombosis prophylaxis in hip arthroplasty

Sir: We wish to comment on the article by Swierstra and associates (1) on the thromboprophylactic efficacies of two different oral anticoagulant regimens in patients undergoing total hip replacement.

We question that oral anticoagulation should be continued for 3 months in all patients. Their suggestion is based on the observation that scintigraphically verified nonfatal pulmonary embolism occurred after discontinuation of prophylaxis in 3 patients in whom no deep vein thrombosis had been detected during the study. As stated by the authors and others (2), the screening method for deep vein thrombosis (radionuclide venography) used in the study is not reliable in the detection of calf vein thrombosis, and isolated distal thrombi may therefore have escaped detection in a number of patients, some of whom may also have had nonfatal pulmonary emboli. The authors do not state whether or not all the patients were routinely examined by an objective technique (ventilation/perfusion lung scintigraphy) during the study in order to rule out the presence of pulmonary embolism before discontinuation of prophylaxis. Anticoagulation therapy is expensive and troublesome because close monitoring is needed, and it is potentially dangerous because of the risk for bleeding (3, 4), though this can be minimized by low anticoagulant dosage.

Furthermore, the reported incidence of postoperative proximal deep vein thrombosis in patients receiving anticoagulants was not different from the incidence we have previously observed in patients receiving a placebo after total hip replacement (5).

The study has therefore not convinced us that there is enough evidence to recommend routine anticoagulation for 3 months after total hip replacement.

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Sir: The comment of our Danish colleagues forces us to clarify once more the twofold basis of our suggestion that oral anticoagulation should be continued for 3 months after total hip replacement in all patients. First, even with the best primary prophylactic method around 20 percent of the patients still develop deep venous thrombosis. In their recent study (5), our Danish colleagues reported an incidence of even more than 30 percent. Some of these patients will develop (fatal) pulmonary embolism if no further protection is given. Secondly, there is growing evidence of a delayed thrombogenesis after different preventive measures. We gave a few references in our article, and can add another (6). Our own observations fit well in this picture, despite the possibility of false-negative venographies.

Our Danish colleagues seem to be convinced of the minimal risk of low-dose oral anticoagulation. It is therefore confusing that they still refer to publications that deal also with complications during inadequately controlled intravenous heparin therapy.

Regarding the reported incidence of proximal vein thrombosis, we found 14–24 percent (depending on the subgroup) versus 29 percent in the Danish placebo group.

In our country, with a well-functioning thrombosis service, anticoagulant therapy is not expensive. Also in the United States, our suggested policy has a favorable cost effectiveness (7).

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