Vascular surgery to a large extent depends on the use of drugs that influence blood coagulability. Heparin, introduced in the 1920s, is widely used by vascular surgeons during the perioperative period, but seems to have lost some of its popularity probably because its long term use is cumbersome. In the late 1970s and early 1980s the distal arterial tree has increasingly been the target of reconstructive surgery or reopening procedures. As a result it became necessary to maintain patency over a long period of time by employing pharmacological measures. "Old fashioned" drugs, such as acetyl-salicylic acid (ASA) have attracted renewed interest in the last decade because of their potentially beneficial action as platelet stabilising agents. Indeed ASA is easy to prescribe and exact postoperative patient surveillance is not necessary, although the best dosage regimen is a matter of continuing debate.

The role of oral anticoagulants, such as coumadin and warfarin, has never been fully established and other drugs that influence either blood viscosity or red cell deformability are currently under investigation. Prostacyclin analogues are interesting not only for their vasoactive potential but also because of their interaction with the intrinsic clotting system. Finally, fibrinolytic agents have attracted considerable interest for the treatment of both arterial and venous thrombosis.

The economic drive of pharmaceutical companies plays an important role in the widespread use of several drugs that have never been proved to be really beneficial in the field of peripheral vascular surgery. Controversies exist concerning the type of drugs, the value and risk of combinations, dosages, optimal time to start treatment and methods of administration.

Times and fashions change, and a renewed interest can currently be noted in the use of oral anticoagulants. Recent discussions at the meeting of the American Society for Vascular Surgery and other international meetings seem to indicate the rediscovery of oral anticoagulants for their possible protective action on the short-term and long-term patency of femoro-popliteal and femoro-crural bypasses.

International attention has been drawn to the peculiar and unique situation in the Netherlands, where the widespread use of coumadine has been possible because of a network of "Thrombosis Services", a non-profit organisation to control the individual dosage of some 35000 patients with peripheral arterial disease, with or without vascular reconstructions.

Both in Europe and in the U.S.A. vascular surgeons treat the majority of their patients postoperatively with platelet inhibitors, usually for a period of about 1 year. The same can be said for the regimen after PTA. However, increasingly and for an obvious reason, for the "difficult" and "thrombosis prone" (redo) patients, protection with oral anticoagulants is seen to be preferable, in spite of a general acceptance that the risk of bleeding complications is increased with the use of these drugs.

At this point it seems reasonable to ask the question: What beneficial action of anticoagulant drugs has really been proven? And what in fact is the risk of haemorrhagic complications associated with the use of these drugs? The answers are not easily provided. The majority of publications in the 1960s and early 1970s on the use of oral anticoagulants in peripheral vascular disease have one thing in common: the studies have no control group or are compared with historical controls.

In 1976 and 1977 three review articles by van Vroonhoven and Bruins Slot, Verstraete and Cristol independently come to the same conclusion: the beneficial effect of long-term treatment with oral anticoagulants on the course of peripheral atherosclerotic disease—with or without surgical reconstruction—has not been demonstrated; the methodology of all previously published studies is unsatisfactory and
there is a definite need for prospective randomised studies, based on well defined objective criteria and study endpoints.

In 1979 a publication by Schneider et al. seemed to fulfill these criteria when they reported a prospective randomised series, using oral anticoagulants or antiplatelet drugs after TEA or vein-bypass surgery. The follow-up period was 2 years and the patient’s condition was evaluated objectively by Doppler pressure measurements and patency confirmed by angiography. They came to the surprising conclusion that patients with a TEA fared significantly better with antiplatelet drugs, whereas patients with a vein-bypass procedure did better on oral anticoagulants. The authors have no explanation for these peculiar results, but make a plea for anticoagulants after bypass surgery and antiplatelet agents after endarterectomy.

The American College of Chest Physicians in cooperation with the National Heart, Lung and Blood Institute in 1984 started a working group to evaluate the indications for, and the efficacy of, treatment with anticoagulants and platelet aggregation inhibitors and their conclusions were published by Genton et al. in 1986. They reported that the available data from the literature did not provide convincing evidence that treatment with antithrombotic agents has a favourable influence on the natural history of peripheral atherosclerotic disease, nor does it prevent or delay arterial occlusion after reconstructive vascular surgery. They advised against the use of anticoagulants after reconstruction in arteries of a diameter of more than 6 mm, with a high flow and low resistance.

Notwithstanding the lack of evidence for a beneficial effect, many vascular surgeons have continued to treat their patients with oral anticoagulants, either as a routine postoperatively or for “extra safety” in high risk patients.

If benefit has not been proven what are the detrimental effects of oral anticoagulants? Here again, there is surprisingly little information in the literature. In the few publications where a relationship is noted between the level of anticoagulation and positive results in peripheral arterial disease, hardly any bleeding complications are mentioned. Consequently, most data on bleeding complications are derived from studies on the use of anticoagulants in coronary and carotid artery disease. In the Sixty-Plus Reinforcement Study Research Group (1982) the incidence of intracranial bleeding is one per 92 treatment years, compared with one per 67 treatment years in the placebo group. Wintzen (1984) calculates, for patients over 50 years of age and treated with anticoagulants, a ten times higher risk of developing an intracranial bleedin-
beneficial effects of long-term anticoagulant treatment: progression of atherosclerotic disease was significantly diminished as shown by a decreased need for further vascular surgical reconstructions, and coronary protection was shown, evidenced by a decreased number of myocardial infarctions in the treatment group. This result is in accordance with the results of the Viennese trial, where oral anticoagulants seem to prolong postoperative survival. Last, but not least, the risk of bleeding complications appears to be very moderate, which is even more important because a synchronous protection for T.I.A.s and cerebral ischaemic infarctions is found.

So what is the role of oral anticoagulants in peripheral vascular surgery? The answer regrettably is that we do not know. Further prospective studies are mandatory to confirm the studies mentioned above. A new prospective trial is underway in the Netherlands, randomising oral anticoagulants against platelet inhibitors. Naturally, if both drugs have a proven beneficial effect, and one is not significantly more effective than the other, oral anticoagulants have the obvious disadvantages of the inconvenience and extra cost of a control system.

Therefore, in conclusion, it can be stated that perhaps there is an important role for oral anticoagulants but this will have to be corroborated by future multicentre clinical studies of high quality. Hopefully this statement may be an incentive for many European colleagues.

References
5. Genti E, Glagett GP, Salzman EW. Antithrombotic therapy in peripheral vascular disease. Chest 1986; 89(12): 75S–81S.

Accepted 30 July 1990