Time 16.00 cont.

1030 PROPHYLAXIS OF VENOUS THROMBOEMBOLISM (VTE)

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A major objective in management of VTE is prevention of the initial or recurrent episodes. Patients at high risk of VTE are identifiable, thus allowing application of prophylactic methods (proph.). Factors contributing to the development of VTE include stasis, vessel wall damage, or activation of platelet or coagulation systems, and proph. methods are available to influence each of these. Success of proph. requires proper selection and application of efficacious method, or combination of methods. Implicit is that consideration be given to the risk as regards; time of onset and duration; its mechanism; its intensity; and the type and intensity of measure required to inhibit it. About treatment must be known; the proper method of application; the type and frequency of complications and ways to prevent them. Beginning treatment prior to initiation of thrombosis and continuing throughout the period of risk is crucial to success. Clinical results indicate that patients at minimal to moderate risk of VTE are protected by pneumatic cuffs, low-dose heparin, dextran, oral anticoagulants, and possibly platelet suppressants, while higher risk requires oral anticoagulants, larger amounts of heparin, or possibly a combination of pneumatic devices with other modalities. "Secondary proph." to prevent recurrence may require more potent measures than primary proph. Thus, efficacious proph. approaches are available for most patients, if properly chosen and applied.

1031 CONTROL OF HEPARIN THERAPY - IS THERE A NEED?

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The argument for laboratory control of heparin treatment rests on 2 issues: 1, that the response to heparin varies from patient to patient and from time to time, and 2, that the clinical results are improved by laboratory control.

The theoretical basis for lab control is strong. AT III levels vary and are reduced in thrombotic states. Heparin's absorption and rate of degradation vary; its survival is shorter after pulmonary embolism.

The clinical evidence that lab control prevents bleeding is weak. When heparin is given by intermittent injection, bleeding is not avoided by lab tests. Even with continuous infusion, coagulation tests may identify a group at high risk for bleeding, but lower values are no guarantee against hemorrhage.

The case for lab control to assure an antithrombotic effect is stronger. Clotting test values correlate with recurrent thromboembolism, particularly when heparin is given by continuous infusion. Thromboembolism recurs more ofter after smaller doses or after a poor APTT response to a larger dose. Other clinical settings are analogous: e.g., cardiopulmonary bypass and hemodialysis. New tests may correlate better with clinical events by measuring end products of heparin's antithrombotic activity rather than its action on coagulation kinetics: e.g., fibrinopeptide A, fibrin monomer, thrombin-antithrombin complexes, prothrombin fragments, and products of platelet activation.

1032 ROLE OF THROMBOLYTIC THERAPY IN THROMBOTIC DISEASE

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Commercially available thrombolytic agents have been tried in many clinical conditions with convincing evidence of benefit in few. The clearest indications are in the venous circulation. Controlled clinical trials have shown that streptokinase dissolves extensive recent deep venous thrombi more often and much more completely than other drugs; there is emerging evidence that early lysis of a venous thrombus prevents destruction of the valves, the main cause of the postphlebitic syndrome. Massive and recent deep venous thrombosis extending above the knee in younger patients appears to be the best indication for streptokinase or urokinase. Urokinase (UK) or streptokinase (SK) are useful in the few patients who have acute massive pulmonary embolism with obstructed right-heart outflow but who do not appear to require surgical embolectomy to prevent immediate death. Other clinical conditions can be marginally improved by thrombolytic treatment as central vein occlusion, medium-risk myocardial infarction and acute obstruction of peripheral arteries in whom vascular surgery is contraindicated or failed. The search still continues for a more effective dose regimen given either continuously or intermittently with or without plasminogen or plasmin or after defibrination.