Anticoagulation Therapy for Deep Venous Thrombosis and Pulmonary Embolism

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INTRODUCTION

It has been estimated that 100,000 to 200,000 people die each year as a consequence of pulmonary embolism (PE). Although therapy for acute PE may be life saving, most deaths due to PE occur within half an hour of acute embolization. Therefore, the best means of preventing fatalities caused by PE is to prevent the occurrence of PE. Because almost all emboli arise from deep venous thrombosis (DVT) in leg veins, the critical clinical issue is the appropriate prevention and management of DVT.

This review considers the standard of care regarding the prophylaxis and management of DVT in medical and surgical patients. These standards undergo continuous review, and the recommendations in this review are based on the Fifth American College of Chest Physicians Consensus Conference on Antithrombotic Therapy, published in 1998. Additionally, because heparin and warfarin are the cornerstones of antithrombotic therapy, this review considers in detail the biology underlying the use of these agents in clinical practice. Because the availability of low-molecular-weight heparins (LMWHs) has had considerable impact on the therapy of DVT and has made outpatient therapy feasible for many patients, this review also considers the rationale for the clinical use of LMWH.

Other recent advances in the field of antithrombotic therapy, including new considerations regarding the duration of therapy in DVT and PE and the rational work-up for suspected hypercoagulable states, are also considered.

WARFARIN

BIOCHEMISTRY AND MECHANISM OF ACTION

Warfarin (Coumadin) is the most widely used oral anticoagulant. It exerts its anticoagulant effect by