

ABC of Antithrombotic Therapy

in vitro with increased platelet survival in vivo. Its effect is relatively short lasting, and repeated dosing or slow release preparations are needed to achieve 24 hour inhibition of platelet function.

Clopidogrel and ticlopidine

These thienopyridine derivatives inhibit platelet aggregation induced by agonists such as platelet activating factor and collagen, and also dramatically reduce the binding of ADP to a platelet surface purinoreceptor. The mechanism of this inhibitory action seems to be independent of cyclo-oxygenase. There is also impairment of the platelet response to thrombin, collagen, fibrinogen, and von Willebrand factor. The peak action on platelet function occurs after several days of oral dosing. Adverse effects include evidence of bone marrow suppression, in particular leucopenia, especially with ticlopidine.

Other receptor blockers

Signal transduction generally occurs when specific receptors on the surface are occupied by ligands such as ADP, leading to structural modification of the glycoproteins IIb/IIIa receptor on the surface of the platelet. This is the commonest receptor on the platelet surface and represents the final common pathway for platelet aggregation, resulting in crosslinking of platelets.

After intravenous administration of glycoprotein IIb/IIIa receptor inhibitors such as abciximab, platelet aggregation is 90% inhibited within two hours, but function recovers over the course of two days. The major adverse effect is haemorrhage, and concurrent use of oral anticoagulants is contraindicated. Eptifibatid is a cyclic heptapeptide that mimics the part of the structure of fibrinogen that interacts with glycoprotein IIb/IIIa. Thus it is a fraction of the size of abciximab and is targeted at the same structure on the platelet surface.

Clinical trials with oral glycoprotein IIb/IIIa receptor inhibitors have been disappointing, with no beneficial effects seen and even some evidence of harm.

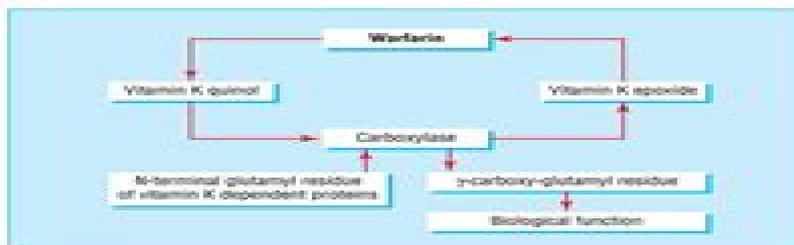
Anticoagulant drugs

Warfarin

This 4-hydroxycoumarin compound, the most widely used anticoagulant in Britain and the Western world, inhibits the synthesis of factors dependent on vitamin K (prothrombin; factors VII, IX, and X; protein C; protein S). Factor VII levels fall rapidly (in < 24 hours) but factor II has a longer half life and only falls to 50% of normal after three days. Warfarin is approximately 97% bound to albumin, and free warfarin enters liver parenchymal cells and is degraded in microsomes to an inactive water soluble metabolite that is conjugated and excreted in the bile. Partial reabsorption is followed by renal excretion of conjugated metabolites.

There is a considerable variability in warfarin's effect on patients, its effectiveness being influenced by age, racial background, diet, and co-medications such as antibiotics. Thus it demands frequent laboratory monitoring, the prothrombin time being compared with a standard to produce the international normalised ratio. The degree of anticoagulation required varies with clinical circumstance, but the target international normalised ratio usually ranges from 2 to 4. Phenindione is an alternative oral vitamin K antagonist, but concerns regarding the potential for hepatotoxicity, nephrotoxicity, and blood dyscrasias have reduced its role largely to individuals with documented hypersensitivity to warfarin.

Adverse effects of warfarin include haemorrhage, hypersensitivity and skin rashes, alopecia, and purpura.



Vitamin K metabolism and the effect of warfarin

Factors that influence the efficacy of warfarin*

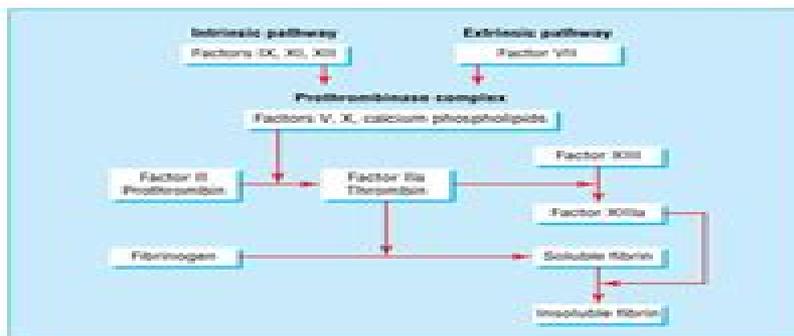
Patient factors

- *Enhanced anticoagulant effect*—Weight loss, increased age (> 80 years), acute illness, impaired liver function, heart failure, renal failure, excess alcohol ingestion
- *Reduced anticoagulant effect*—Weight gain, diarrhoea and vomiting, relative youth (< 40 years), Asian or African-Caribbean background

Examples of drug interactions with warfarin

- *Reduced protein binding*—Aspirin, phenylbutazone, salicypyrazone, chlorpromazine
- *Inhibition of metabolism of warfarin*—Cimetidine, erythromycin, sodium valproate
- *Enhanced metabolism of warfarin*—Barbiturates, phenytoin, carbamazepine
- *Reduced synthesis of factors II, VII, IX, X*—Phenytoin, salicylates
- *Reduced absorption of vitamin K*—Broad spectrum antibiotics, laxatives
- *Enhanced risk of peptic ulceration*—Aspirin, NSAIDs, corticosteroids
- *Thrombolytics*—Streptokinase, tissue plasminogen activator
- *Antiplatelet drugs*—Aspirin, NSAIDs

*This list is intended to be illustrative not exhaustive



Simplified coagulation cascade

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