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[Intervention Review]

# Vitamin K antagonists versus antiplatelet therapy after transient ischaemic attack or minor ischaemic stroke of presumed arterial origin

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## ABSTRACT

### Background

People who have had a transient ischaemic attack (TIA) or non-disabling ischaemic stroke have an annual risk of major vascular events of between 4% and 11%. Aspirin reduces this risk by 20% at most. Secondary prevention trials after myocardial infarction indicate that treatment with vitamin K antagonists is associated with a risk reduction approximately twice that of treatment with antiplatelet therapy.

### Objectives

To compare the efficacy and safety of vitamin K antagonists and antiplatelet therapy in the secondary prevention of vascular events after cerebral ischaemia of presumed arterial origin.

### Search methods

We searched the Cochrane Stroke Group Trials Register (last searched 15 September 2011), the Cochrane Central Register of Controlled Trials (CENTRAL) (*The Cochrane Library* 2011, Issue 3), MEDLINE (2008 to September 2011) and EMBASE (2008 to September 2011). In an effort to identify further relevant trials we searched ongoing trials registers and reference lists. We also contacted authors of published trials for further information and unpublished data.

### Selection criteria

Randomised trials of oral anticoagulant therapy with vitamin K antagonists (warfarin, phenprocoumon or acenocoumarol) versus antiplatelet therapy for long-term secondary prevention after recent transient ischaemic attack or minor ischaemic stroke of presumed arterial origin.

### Data collection and analysis

Two review authors independently selected trials, assessed trial quality and extracted data.

### Main results

We included eight trials with a total of 5762 participants. The data showed that anticoagulants (in any intensity) are not more efficacious in the prevention of recurrent ischaemic stroke than antiplatelet therapy (medium intensity anticoagulation: relative risk (RR) 0.80, 95% confidence interval (CI) 0.56 to 1.14; high intensity anticoagulation: RR 1.02, 95% CI 0.49 to 2.13).

There is no evidence that treatment with low intensity anticoagulation gives a higher bleeding risk than treatment with antiplatelet agents: RR 1.27 (95% CI 0.79 to 2.03). However, it was clear that medium and high intensity anticoagulation with vitamin K antagonists, with an INR of 2.0 to 4.5, were not safe because they yielded a higher risk of major bleeding complications (medium intensity anticoagulation: RR 1.93, 95% CI 1.27 to 2.94; high intensity anticoagulation: RR 9.0, 95% CI 3.9 to 21).

### **Authors' conclusions**

For the secondary prevention of recurrent ischemic stroke after TIA or minor stroke of presumed arterial origin, there is sufficient evidence to conclude that vitamin K antagonists in any dose are not more efficacious than antiplatelet therapy and that medium and high intensity anticoagulation leads to a significant increase in major bleeding complications.

### **PLAIN LANGUAGE SUMMARY**

#### **Vitamin K antagonists versus antiplatelet therapy after transient ischaemic attack or minor ischaemic stroke of presumed arterial origin**

People who have a stroke due to a blockage of an artery have a higher risk of having another possibly fatal stroke, or a heart attack. Treatment with antiplatelet drugs (like aspirin) definitely reduces this risk. Blood thinning treatment (anticoagulation by vitamin K antagonists) was believed to provide added protection. We reviewed eight trials involving 5762 participants that compared anticoagulants with antiplatelet agents for preventing recurrent stroke and found no benefit of low intensity anticoagulation over aspirin, and an increased risk of bleeding with high intensity anticoagulation.