

Cardiovascular Effects of Common Analgesics

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SUMMARY

The cyclooxygenase (COX) enzyme forms locally active prostaglandins responsible for producing inflammation and pain. Classical non-steroidal anti-inflammatory drugs (NSAID) inhibit the COX-2 enzyme that produces inflammatory prostaglandins as well as the COX-1 enzyme that produces gastric mucosa protecting prostaglandins. By specifically inhibiting only the COX-2 enzyme, coxibs thus reduce pain but do not damage the gastric mucosa. However, COX-2 at the vascular endothelium produces antithrombotic prostaglandins, and so by inhibiting COX-2 enzyme, the coxibs promote thrombosis. Rofecoxib and valdecoxib have been withdrawn because of the adverse cardiovascular events they induce. Amongst presently available coxibs cardiovascular risk is highest with enterocoxib and lowest with celecoxib. NSAIDS also increase cardiovascular events, the risk is highest with diclofenac and lowest with naproxen. Paracetamol and corticosteroids induce hypertension, while steroids also adversely affect the heart from metabolic change as well as fluid retention. Aspirin is an anti-thrombotic agent because of its ability to inhibit the COX-1 enzyme that produces the pro-aggregatory thromboxane. However, it increases gastrointestinal bleeding, can promote fluid retention and is nephrotoxic, all of which may lead to adverse cardiovascular outcomes. Patients at especially high risk of cardiovascular events from analgesic use include the elderly, and those with heart failure, hypertension, rheumatoid arthritis, chronic renal disease, chronic obstructive airway disease and previous myocardial infarction, cerebrovascular disease or peripheral vascular disease. Adverse cardiovascular events can occur within a week of initiation of analgesic treatment.