

ASPIRINE AS CHEAPEST DENTAL ANALGESIC FOR LOW INCOME GROUP

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Aspirin is mild analgesic and is effective against the pain of low to moderate intensity, e.g., dull throbbing pain of inflammatory origin. As an analgesic it is not effective in non-inflamed tissues and is also ineffective against sharp and stabbing pain which is caused by direct stimulation of sensory nerves. It is also not effective against deep visceral pain.

DISCUSSION

The analgesic effect of aspirin is mainly peripheral. Low concentrations of aspirin inhibit the synthesis and release of prostaglandins which are involved in the pathogenesis of inflammation and fever. Both peripheral and central nervous system factors appear to contribute significantly to the pain relief afforded by this class of drugs but according to Cawson and Spector (1985) the central inhibitory action of salicylates is controversial.

Salicylates are antipyretic and anti-inflammatory. They act in the hypothalamus and regulate to a lower level the set point of the temperature control which is elevated by fever. This may also be due to inhibition of prostaglandin synthesis which is a mediator of the febrile response to infection. Aspirin does not reduce the temperature when body temperature is normal or when temperature is increased due to exercise (Foster 1980). Salicylate poisoning can cause hyperpyrexia.

Salicylates are used as anti-inflammatory analgesic in rheumatoid arthritis, osteoarthritis and ankylosing spondylitis. They provide symptomatic relief from pain and inflammation but do not modify the basic pathological condition.

Aspirin reduces blood platelet adhesiveness and this results in prolongation of bleeding time. Therefore, it may be used in conjunction with anticoagulants for the prophylaxis of coronary and cerebral arterial thrombosis.

As little as 150mg of aspirin can modify platelet function for up to seven days (Hammond) but according to Farah and Rosenberg (1980) an inhibitory effect persists for the life of platelets (about 10 days). Mean plasma levels of acetylsalicylic acid of less than 0.25mg/l are sufficient to depress aggregation by approximately 50%. A low dose of acetylsalicylic acid taken daily significantly inhibits platelet aggregation. The clinical effect would be noted with one 650mg enteric coated tablet, or a 300mg effervescent tablet, taken daily (Ross-Lee et al, 1982). Paccioretti and Block (1980) found that a single 81mg chewable tablet will successfully inhibit platelet aggregation, and Burch et al. (1978) reached the same conclusion after testing doses of as little as 20mg/day.

Soluble aspirin has significantly greater effect on platelet aggregation and bleeding than tablets (Ross-Lee et al. 1982).

Salicylates can lower the blood sugar by increasing peripheral utilization of glucose and has been used in diabetes mellitus (Laurence and Bennett 1987). With heavy doses, however, hyperglycaemia may occur.

Methyl Salicylate is used externally for painful muscles and joints in ligaments or ointments.

Salicylic acid when applied topically acts as a keratolytic agent. It is also used as wart and corn remover (10%-20% collocation).

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Aspirin (acetylsalicylic acid) is a white powder poorly soluble in water available as tablets containing 65mg to 650mg, capsules 300mg and suppositories containing 65 to 1300mg of drug.

Sodium salicylate is a white water soluble powder. Tablets containing 300 or 600mg of drug are available.

The usual adult dose of aspirin is 300mg to 1g repeated 4 hourly. To minimize gastric irritation salicylates should be taken with a full glass of water.

The route of administration of aspirin is mostly oral. It is partly absorbed from the stomach and mostly from the upper small intestine. Effective concentrations are found in plasma within 30 minutes and peak level is reached in about 2 hours. Peak and total plasma concentration are greater after soluble aspirin than after the same dose of aspirin tablets.

Absorption is modified by the dissolution rate of tablets and gastric emptying time. Absorption will be decreased if the pH is high at the presence of food in GIT.

The absorption rate of aspirin has been said to be a factor in determining its analgesic efficacy (Levy 1965) but according to Seymour (1984) it is unlikely that variation in aspirin absorption accounts for variability in efficacy.

Aspirin and paracetamol in doses of 500-1000mg are equipotent as analgesics (Beaver 1981; Cooper 1981) and antipyretics and as effective as codeine (30mg), dihydrocodeine (30mg) and dextropropoxyphene (65mg). Increasing the dose of these drugs does not provide further analgesia but prolongs the duration of action at the expense of increased toxicity, i.e., gastric irritation with aspirin. According to Laurence and Bennett (1987) the analgesic effect of salicylates is less than that of codeine.

Combinations of an optimal dose of aspirin with a narcotic (codeine < hydrocodone or oxycodone) produce an additive analgesic effect greater than that obtained by doubling the dose of either constituent administered above (Beaver (1981).

Combinations of acetylsalicylic acid (500mg) with codeine phosphate (30mg) provided better pain relief than 300mg acetylsalicylic alone in cases of post-

operative dental pain (Dahl et al. 1985). According to Cooper and Beaver, the analgesic effect for relief of post-operative dental pain is superior with aspirin (650mg) and paracetamol (600mg) than with codeine (60mg).

The analgesic action is faster, more effective and its duration is greater with soluble aspirin than aspirin tablets for post-operative dental pain (Dixit et al 1984; Seymour et al 1986).

Side effects of Aspirin

High doses of salicylates may cause exacerbations of peptic ulcer, erosive gastritis and G.I.T. hemorrhage (Langman 1983) accompanied by secondary anemia due to blood loss. According to Rees (1980) aspirin ingestion rarely causes clinically significant gastric damage in normal subjects and then usually only in large doses or when taken frequently. Gastric bleeding due to salicylate is usually painless. In 70% of people who regularly take aspirin, about 10ml of occult blood per day is found in their stools and about 1 in 15 have G.I.T. symptoms (Laurence and Bennett 1987). Bleeding is aggravated by taking alcohol and diminished by administration of alkalis or using buffered aspirin (Cawson and Spector 1985; Laurence and Bennett 1987). However, according to Pennington et al (1978) there is no evidence that soluble or buffered aspirin is any less irritant to the gastric mucosa.

Aspirin prolongs bleeding time even with small doses, e.g., a dose of 0.65g approximately doubles the mean bleeding time for 4 to 7 days (Goodman and Gilman 1980). Aspirin causes hypoprothrombinaemia with large doses over 6g/day (Pennington et al 1978) and potentiates hemorrhagic disorders and increases post-operative blood loss.

Aspirin has fibrinolytic activity (Cawson and Spector 1985). It binds firmly to plasma proteins and displaces other drugs from binding sites (Goodman and Gilman 1980). This can cause a problem for a patient who is receiving Warfarin, or sulphonylurea as a hypoglycemic agent.

Aspirin has been implicated in several drug interactions, notably with ethanol, anticoagulants, phenytoin, diuretics, sulphonylureas and methotrexate (Hayes 1981).

Moderate overdoses stimulate the respiratory centre causing increased rate and depth of respiration and ultimately respiratory alkalosis (Cawson and Spectro 1985) but when poisoning is severe respiration and metabolic acidosis follow associated with hypernatraemia (Goodman and Gilmans 1980). Children under 4 years tend not to exhibit respiratory alkalosis but readily develop severe metabolic acidosis (Laurence and Bennett 1988). In children up to 15 years of age with respiratory tract infection, chicken pox, influenza and other viral infections aspirin may rarely precipitate liver damage with encephalopathy (Starko et al 1980; Scully and Cawson 1987).

After large doses, peripheral vessels dilate and toxic amounts depress the circulation directly and by central vasomotor paralysis. With large doses of salicylates congestive cardiac failure or pulmonary oedema may occur in patients with acute rheumatic fever and carditis due to increased plasma volume and increased peripheral oxygen uptake necessitating greater cardiac output.

Salicylates have a deleterious effect on liver function (Zimmerman 1981) and with toxic doses fatty infiltration of liver and kidney may occur. Aspirin may cause liver damage, particularly in young patients with collagen diseases such as systemic lupus erythematosus, in whom the incidence of salicylate hepatitis is 20-70% (Zimmerman 1981).

There have been numerous reports of impairment of renal function and analgesic nephropathy associated with aspirin (Gokal 1979; Plotz 1981).

In hypersensitivity to salicylates, anaphylactic shock, rashes, urticaria, angio-oedema and bronchospasm may occur.

Women taking salicylate chronically may give birth to babies of reduced weight. There may be increased perinatal mortality, anaemia, antepartum and postpartum hemorrhage, prolonged gestation and complicated deliveries (Coffins et al³⁵. 1975).

Aspirin may have undesirable effect on male fertility (Goodman²⁴ and Gillmans 1980).

Moderate overdose causes, nausea, vomiting, epigastric discomfort, vertigo, tinnitus, headache, temporary deafness and mental confusion. Large overdose can cause along with above symptoms, hyperpyrexia, pulmonary oedema, mania, convulsions, and coma with severe dehydration and ketosis.

CONCLUSION

Aspirin taken before the extraction interferes with platelet function, which may prolong bleeding as a result of decreased platelet aggregation and platelet plug formation in small arterioles. If the patient is already on aspirin before invasive procedure, proper assessment, consultation with the physician and the surgeon will be needed.

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