Reducing the Risk: Heart Disease, Stroke and Aspirin†

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Abstract

Aspirin used in cardiovascular disease is probably the best evaluated and the most highly cost effective prophylactic available in clinical practice today. It has been evaluated in over 150 randomised controlled trials and a small daily dose of around 100 mg has been shown to reduce the risk of myocardial infarction and stroke by about 30 per cent. The saving of lives and prevention of vascular events in patients judged to be at increased risk of a vascular event for any reason is large. In healthy subjects, however, the risk of a vascular event is so low that prophylaxis by any drug is inappropriate.

The earlier aspirin is given in the acute phase of a myocardial infarction the greater the saving of lives. Patients judged to be at increased risk of a vascular event, for any reason, should therefore be advised to carry aspirin and to chew and swallow one or two tablets immediately, if they experience sudden severe chest pain.

Aspirin is derived from a salicylate, and salicylates are widely distributed in nature, with many functions in plants. Its use in cardiovascular disease derives from an effect on blood platelets, but other possible effects have led to suggestions that it may be of benefit in conditions other than heart disease and stroke. Current research is now focussing on the possible reduction by aspirin in the risk of dementia, certain cancers, retarded foetal growth and cataract.

Key word : Myocardial Infarction, Stroke, 100 mg Aspirin

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The ultimate aim of medicine is not only to prolong life, but to postpone suffering and disability, and assist in their management when they do occur. Probably the most important pathological process associated with ageing is vascular disease. A major component of medical research should, therefore, be the identification and evaluation of factors involved in vascular disease. Prominent amongst

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these are life-style factors such as smoking, physical activity and diet, all of which are under the control of the patients themselves. The task of the physician is, however, to identify underlying pathological processes and then to provide means to limit them. In the case of vascular disease the most important processes are atherosclerotic disease of the vessel walls and thrombosis within the vessel lumen.

Haemostastic factors in heart disease

The huge rise in cardiovascular mortality in most Western countries during the first seventy years of this century, has been followed in more recent years by an equally rapid fall. What evidence there is, suggests that neither the prevalence nor the severity of atherosclerosis of the vessel wall has changed much during this period. On the other hand, infarction of the myocardium appears to have been unknown at the beginning of this century. Although there can be no certainty, all this suggests that changes in the incidence of cardiovascular disease have resulted from changes in factors involved in thrombosis, rather than due to any marked changes in the prevalence or severity of arterial atherosclerosis.

Fibrinogen, plasma viscosity, von-Willebrands factor and other factors involved in thrombosis are strongly predictive of thrombotic events such as coronary artery thrombosis(1).Work on these factors is now required to identify their determinants, that is, the dietary and life-style factors which determine their level, and hence the risk of a thrombotic event. The most powerful determinant which has been identified so far is smoking(2).

Blood platelets

Platelets play a key role in thrombosis and they constitute the link between atheroma and an occlusive thrombus in myocardial infarction and stroke (see Fig. 1). Evidence of the involvement of platelets comes from a wide variety of sources, including the histology of thrombi in the coronary vessels, the finding of platelet emboli in the micro circulation of the myocardium after sudden death and the finding of an occlusive thrombus on angiography shortly after infarction Platelet aggregation is enhanced after myocardial infarction and cross-sectional evidence shows an association between aggregation and prevalent IHD.

The strongest evidence that platelets are a key factor in thrombosis, is however, the effect of aspirin and other platelet active drugs on the risk of cardiovascular disease events(3). This is the main topic of this paper.

Blood platelets

Aspirin, (acetylsalicylic acid), has a remarkable effect on platelets. The usual dose recommended for pain, headache or fever is 600 mg or more, repeated as necessary up to three or four times a day. The dose needed to affect platelet function is around 100 mg. Furthermore, this action on circulating platelets is irreversible and lasts for the life of the platelets. The effect of a dose of aspirin is, therefore, lost only gradually as fresh platelets come into the circulation over the following seven to ten days. There is no evidence that prolonged taking of aspirin leads to either tolerance, or dependence.

Low doses of aspirin affect platelet function primarily by inhibiting platelet cyclo-oxygenase, an enzyme involved in the formation of the aggregating agent thromboxane A_2. In fact, salicylates have other effects on haemostatic mechanisms which may add to the protective effect of aspirin, some of these other effects are due to interference with vitamin K in the hepatic synthesis of the coagulation factors VII, IX and X.

Aspirin and myocardial infarction

The first randomised controlled trial of aspirin in the prevention of vascular events was reported in 1974. One thousand four hundred post infarction male patients were put on either a daily dose of one standard tablet of aspirin (330 mg) or matching placebo. A reduction by aspirin in all-cause mortality by aspirin of 24 per cent (95% CI-42.4% to 5.3%) was reported(4).

This first trial stimulated world-wide interest and over 145 randomised controlled trials of aspirin have now been reported in the medical press. These have involved a total of over one hundred thousand patients, amongst whom there were almost eleven thousand heart attacks and strokes. These trials have established aspirin, used in cardiovascular disease, as the most thoroughly tested drug available in clinical practice today(3).

The results of these trials have been examined in a number of overviews. They show a remarkable consistency and indicate that aspirin reduces the incidence of non-fatal myocardial infarction and strokes by between 30 and 40 per cent and reduces deaths from all causes by around 20

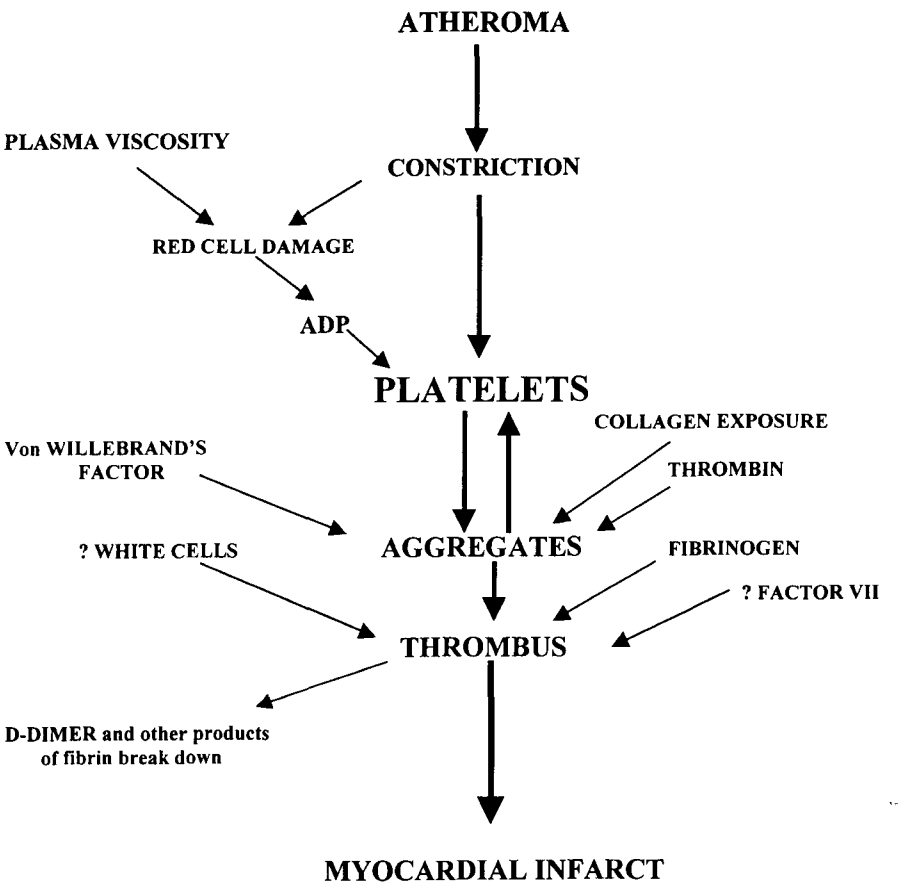

Fig. 1. The development of a myocardial infarct.

Table 1. The reduction in vascular events by long-term low-dose aspirin prophylaxis. Based on the combined results of 145 randomised controlled trials as reported in Antiplatelet Trialists Collaboration (1994).

Reduction in various clinical groups:			
Patients with prior MI	11 trials	25%	reduction
Patients with an acute MI	9 trials	29%	reduction
Patients with a prior stroke or TIA	18 trials	22%	reduction
Other high risk groups of patients	104 trials	32%	reduction
All patient groups		25%	reduction in further vascular events

per cent (Table 1). The consistency in the results of these trials give no evidence of any important differences in the proportionate benefit in patients with a variety of previous clinical conditions such as unstable angina, previous MI or stroke, peripheral vascular disease and artery and valve surgery (Table 2). Nor is there any evidence of significant heterogeneity in the reduction achieved by aspirin in different groups of patients such as male and female, older and younger, diabetic and non-diabetic patients etc (Table 2).

That is: aspirin substantially reduces the risk of vascular events, and of death in all patients and with all kinds of previous medical histories.

Table 2. The reduction in various outcomes by long-term low-dose aspirin prophylaxis. Based on the combined results of 145 randomised controlled trials as reported in Antiplatelet Trialists Collaboration (1994).

Reduction in the separate outcomes:		
Reduction in non-fatal MIs	122 trials	34% reduction
Reduction in non-fatal strokes	124 trials	25% reduction
Reduction in all vascular deaths	144 trials	17% reduction
Reduction in all-cause deaths	144 trials	16% reduction
Relative reduction in different patient groups:		
Males and females	similar reduction	
Older and younger	similar reduction	
Hypertensive and normotensive	similar reduction	
Diabetic and non-diabetic	similar reduction	

Of course there are failures and some patients experience a vascular event despite aspirin prophylaxis. Some of the failures in patients on aspirin are undoubtedly due to poor compliance. In the Physicians Health Study aspirin was to be taken on alternate days, and it was possible to assess compliance. Subjects who took aspirin on every day recommended, experienced a 51 per cent reduction in vascular events, whereas, those who took aspirin on less than half the days recommended showed only 17 per cent reduction[5].

Additional to this, platelets are not responsible for every vascular event, and aspirin, through its effect on platelets, could not prevent every infarction. However, a number of studies have compared infarctions in patients who had been taking aspirin and in control patients who had not been on aspirin at the time of their infarction. The MI appears to be modified by aspirin, and the likelihood is that it will be less severe and of the small, non Q-wave variety[6,7].

Aspirin and stroke

The use of aspirin in the prevention of stroke appears to be in line with MI prevention both in the degree of protection and in the dose that is appropriate[8]. Thus, aspirin reduces the incidence of cerebro-vascular events by about 30 per cent[3, 9]. Long-term aspirin prophylaxis should, therefore, always be considered in patients judged to be at risk of an ischaemic cerebral event.

The differential diagnosis between an ischaemic lesion and cerebral haemorrhage is of course impossible on clinical grounds alone. If computerised tomography (CT scan) can be performed and if this indicates an ischaemic lesion, aspirin should be given as early as possible, but if haemorrhage is demonstrated by CT, then aspirin should be withheld during the acute phase of the lesion.

The question naturally arises as to the advisability of aspirin prophylaxis in the acute phase of a stroke if CT cannot be performed. Evidence on this has been provided by two recent large randomised trials: CAST[10] and IST[11]. The results of these trials indicate that if aspirin is given in the acute phase of a stroke there is an additional prevention of 8 to 10 deaths during the first month (Table 3). That is, aspirin commenced immediately after the onset of stroke symptoms, when CT is not available, will lead to an additional decrease in the number of deaths and disabling strokes which is 'modest but worthwhile'[12].

A stroke in a patient with atrial fibrillation is likely to be embolic in origin. If such a stroke is suspected then anticoagulation, rather than aspirin, is the treatment of choice. Nevertheless, if facilities for monitoring are not adequate and it is decided not to use anticoagulants, aspirin should certainly be included in the management of patients with AF [13].

Table 3. Risks and benefits in aspirin prophylaxis. The figures shown are based on an overview of 145 trials (Antiplatelet Trialists Collaboration 1994) and other reports.

In every 1,000 patients, per year:	Prior MI	Prior stroke	First month after stroke*	Other high-risk patients	Low risk subjects
- the likely saving of clinical events	38	35	8	22	2 - 3
- major bleeds possibly due to aspirin	1.0	0.8	0.7	0.5	1.0
- intra-cranial bleeds possibly due to aspirin	0.5**	0.1**	2.0**	0.1**	0.5**

* The figures in this column are the additional savings during the first month after a stroke.

** These figures are not statistically significant but are the upper 99% confidence limits of estimates.

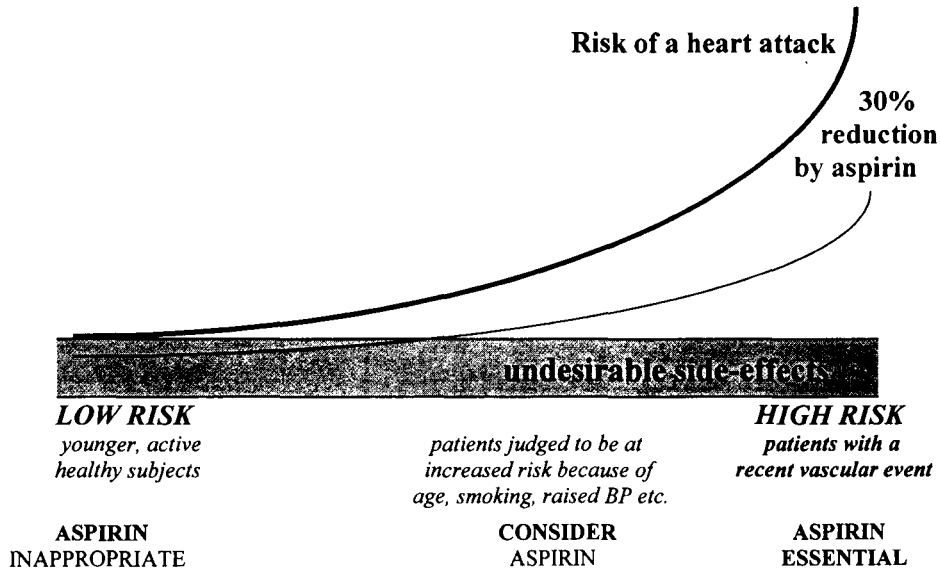

Fig. 2. Aspirin and the reduction of risk of vascular disease events.

Aspirin and so-called 'primary' prevention

Aspirin has been established as highly effective in all patients at high risk of a thrombotic event. However, all but a very few of the major trials have been based on patients who have already experienced a myocardial or a cerebral infarct. Questions, however, arise about aspirin use in primary prevention, that is, prevention in subjects who have not already had an MI, stroke or other vascular event.

Relevant data come from a number of trials. A trial of 22,071 American physicians who were given 325 mg aspirin on alternate days showed a relative reduction by aspirin of 44 per cent in the incidence of non-fatal myocardial infarction[14]. On the other hand, 5,139 healthy British doctors, half of whom were given 500 mg aspirin per day for six years gave no evidence of protection from aspirin [15]. Subsequent analyses of the complete results from both trials considered together indicate a significant reduction in non-fatal infarction by aspirin of 33 per cent and a non-significant reduction of all vascular deaths of 10 per cent[3,14].

The Thrombosis Prevention Trial[16] examined this situation further. This was based on over five thousand men who had not had a vascular event but were judged clinically to be at increased risk of infarction. A relative reduction by aspirin in incident events was found: 32 per cent fewer non-fatal events and 20 per cent fewer deaths. The Swedish Angina Pectoris Aspirin Trial[17] gives further evidence of protection in men who have not had a vascular event: a relative reduction of 34 per cent.

The terms 'primary' and 'secondary' relate, however, to the past history of a patient, and not to his or her future risk. They are, therefore, misleading and the concept of 'primary' prevention is not in the best interests of patients. In relation to the risk of a future event, subjects simply form a continuum, ranging from subjects at exceedingly low risk, such as younger healthy subjects, to patients who have recently had an MI. Ranged between these extremes are subjects who are older, who smoke, who have raised BP, raised cholesterol etc. Fig. 2 displays this continuum of vascular risk. Aspirin reduces the risk of a future vascular event by about one third right across the continuum. On the other hand, the average risk of undesirable side effects is constant in all subjects, whatever their vascular risk. Clearly, aspirin prophylaxis is mandatory at the high-risk end of the continuum, but whether or not aspirin is recommended at lower levels of risk is clearly a clinical decision, taking into account the likely risk of a future vascular event and the likely

occurrence of serious side effects from the aspirin. The occurrence of a past event is only one element in this judgement.

'Early' and 'immediate' aspirin

There are two special situations in which the use of aspirin would seem to be most reasonable. The first of these is what has become generally known as 'early' aspirin: that is, aspirin given by a doctor or paramedic on first contact with a patient who has chest pain and is judged to possibly be experiencing an MI. Early aspirin in such situations is now accepted clinical practice in most Western countries.

An extension of this measure would be for patients who are judged to be at high-risk of infarction, to be advised to carry their own aspirin and chew and swallow one or two tablets immediately they experience severe chest pain. This 'immediate' aspirin has been recommended and would certainly seem to represent a very reasonable and simple extension of present practice for a number of reasons:

- The initial mortality after myocardial infarction is high and this rapidly decreases with time. The earlier aspirin is given after symptoms commence, the greater the savings in death and disability are likely to be.

- Platelet emboli have been found within the coronary circulation in subjects who have died suddenly[18] and it is likely that aspirin, taken very early, will limit the growth of a developing thrombus and possibly lead to the disaggregation of the platelets in emboli.

- There is a peak in the occurrence of myocardial infarction in the early morning between about 04.00 and 10.00 hours and this coincides with an increased sensitivity of platelets to aggregating agents[19]. The readiness with which a patient is likely to call a doctor, and the availability of help during these hours is probably less than later in the day. Furthermore, it is of relevance that in the US Physicians trial it was found that the reduction in myocardial infarction by aspirin was significantly greater during the early morning (59%), than during the rest of the day (34%)[20].

- A high proportion of patients in whom an infarct is proven are likely to go on to receive thrombolytic therapy. There is a 'marked' or 'considerable' heightening of platelet activity after thrombolysis[21]. Prior treatment with aspirin abolishes the excess in re-infarction that otherwise follows fibrinolytic therapy[22].

If, therefore, a subject is judged to be at increased risk because of raised levels of any of the risk factors for cardiovascular disease - age, smoking, blood pressure, cholesterol level - then the giving of advice about 'immediate' aspirin should be considered. Even patients who have already had a thrombotic event and are taking daily low-dose aspirin should be advised to carry a few standard aspirin tablets. The half-life of aspirin in the circulation is only about 30 minutes and if a thrombus develops despite daily exposure to aspirin, then some sensitive platelets which have not been acted upon by aspirin have probably come into the circulation. An extra 'flush' of aspirin could, therefore, be beneficial. Such patients will have already been screened for intolerance to aspirin, and so a single dose of, say, 300-600 mg of aspirin, taken in addition to a daily dose of perhaps 100 mg, could be beneficial and is unlikely to do any harm.

The appropriate dose of aspirin for prophylaxis

There is little evidence that the degree of protection in long-term prophylaxis is related to the dose of aspirin. Overviews of trials give no evidence of any significant differences in the protection given by doses between about 75 and 300 mg. The WHO recommendation is for 100 mg daily in long-term prophylaxis.

It seems to be widely believed that the dose of aspirin in stroke prevention should be larger than that which is adequate for MI prevention, and 200 mg per day is often quoted. A number of trials have randomised patients to two doses and these have yielded evidence that favoured the lower dose[8].

The formulation of aspirin seems to matter little. There is, however, evidence that the absorption of enteric coated tablets may be low if taken with food[23]. Furthermore, absorption from enteric coated tablets is not only greatly delayed[24] but some elderly subjects may absorb very little of the drug from such preparations[25]. However, as some large and successful trials have been based on enteric coated tablets these formulations are clearly effective in the majority of subjects.

The aim of 'early' and 'immediate' aspirin is to get the drug into the circulation as quickly as possible. Soluble aspirin should, therefore, be used

Table 4. Overall balance in long-term low-dose aspirin prophylaxis.

In every 1,000 patients given low-dose aspirin, per year...
- 30 to 40 patients with a recent MI or stroke will avoid a vascular event
- perhaps 15 to 20 patients with angina will avoid an event
- between about 10 and 30 patients judged to be at high-risk* will avoid an event
- probably only two or three subjects at low-risk* subjects will avoid an event

AND, in every the same 1,000 subjects on aspirin, per year...
- one or two will suffer a major bleed
- around 5% will experience minor bleeding and/or gastrointestinal symptoms

* 'High' and 'low' risk are clearly clinical judgements based on the levels of all cardiovascular risk factors and not just the occurrence of a prior vascular event.

Note: these estimates are based on trials from which patients with contraindications for aspirin have been excluded – as is done in all good clinical practice!

if available, and if not, standard aspirin should be used and the patient instructed to chew and swallow one or two standard tablets. Clearly, coated tablets are unsuitable if swallowed whole. Significant quantities of aspirin have been detected in the plasma within a few minutes of the ingestion of soluble aspirin, together with a total inhibition of aggregation, again within minutes[26-28]. Regarding dose, it would seem to be advisable to use a relatively high dose as there is evidence that absorption can be impaired in patients during the acute phase of infarction[29]. Based on studies of platelet aggregation, Berglund and Wallentin[30] recommend that the 'loading dose in unstable conditions should exceed 300 mg'... and it can reasonably be added - soluble aspirin.

Side-effects of low-dose aspirin

Aspirin, in whatever formulation, and even at low doses, can occasionally cause gastric irritation, increased occult blood loss and rarely, serious gastric bleeding. These effects are dose related and while evidence from trials suggests that the relative risks of gastric irritation and intestinal bleeding are increased by 50 to 100 per cent, the absolute risk for gastric irritation with continuing low-dose aspirin is only a little higher than that which occurs with placebo tablets, and for clinically significant blood loss the excess incidence is probably at most 1 in every hundred subjects[14].

Concerns have been expressed regarding the increased risk of cerebral haemorrhage, and in particular, during aspirin prophylaxis in stroke. The summary statistics in Tables 3 and 4 give reassurance. Clearly, the number of cerebral bleeding events that might be attributable to aspirin is very small (and technically non-significant, despite huge numbers in the trials). Furthermore, even in the acute phase of a stroke, the absolute number of bleeds is very considerably exceeded by the numbers of events prevented by aspirin. It is this balance that led to the judgement that the use of aspirin prophylaxis during the acute phase of stroke is 'modest but worthwhile[12].

Relative and absolute reductions

There are two ways of stating risk. The relative risk is the difference between two groups expressed as a proportion of the risk in one of the groups. Thus the relative, or proportionate reduction in aspirin trials conducted in post MI patients is about 33 per cent, that is a reduction by aspirin to an incidence of perhaps 8 per cent compared to perhaps 12 per cent in those who received no aspirin. The absolute reduction (assuming the same incidence rates apply each year) is only 4 per cent. That is, although the relative risk is reduced by one third, of every hundred patients given low-dose aspirin prophylaxis, four will avoid a clinical event each year. And, of course, the other 96 patients are exposed to the costs and the side effects of the aspirin.

The use of absolute rates is most important in the evaluation of treatment or preventive measure. In relation to low-dose aspirin prophylaxis the absolute rates indicate the following general conclusions:

- of every thousand post-MI patients put on aspirin about forty will avoid a thrombotic event each year. One or two may experience a serious

bleed, requiring transfusion. Many more will experience minor, or trivial side effects, perhaps several in every hundred.

- of every thousand healthy subjects, with no clinical evidence of increased cardiovascular risk, perhaps one or two will avoid a thrombosis if put on aspirin, while one or two may experience a serious bleed.

- of every thousand subjects with evidence of increased cardiovascular risk (older and/or smoking and /or raised blood pressure and/or raised cholesterol level) perhaps 10 to 30 subjects (depending on their level of risk) will avoid a thrombosis if put on aspirin, while one or two may experience a serious bleed, requiring transfusion.

Alternatives to aspirin

In most studies it has been found that aspirin is inappropriate for about 8-10 per cent of subjects. In some cases, aspirin causes abdominal discomfort or other symptoms and for these, enteric-coated tablets should be tried. In other subjects aspirin is definitely contra-indicated because of sensitivity, or a history of peptic ulceration etc. There are alternatives to aspirin which, although very much more expensive, should be considered in such patients.

- Dipyridamole may give a small additional benefit if given along with aspirin, but is an inadequate substitute for aspirin.

- Ticlopidine: A reduction in vascular events similar in magnitude to aspirin has been demonstrated(31), but the drug causes severe neutropenia in almost 1 per cent of patients(32). It should, therefore, only be used if the facilities for white cell monitoring are adequate.

- Clopidogrel (Plavix): This derivative of Ticlopidine gives just about the same benefit as aspirin(33,34). It is, therefore, best reserved for use as an alternative to aspirin in the few patients who cannot tolerate aspirin in any formulation(35).

Cost/effectiveness of aspirin

When used in the reduction of cardiovascular risk, aspirin is certainly the most cost-effective of all drugs available at present. During the year or two after a myocardial infarction or a stroke, only about 40 patients have to be prescribed low-dose aspirin to prevent one cardiovascular event each year. This cost of prophylaxis is therefore about $110 per vascular event or death prevented. This contrasts somewhat with around $27,000 for every event prevented by Clopidogrel, and around $150,000 with cholesterol lowering with a statin.

It must, however, be emphasised that aspirin is not in competition with other drugs, nor is it an alternative to other therapies. Thus, if a patient has hypertension, this should be treated. If it is judged that the cholesterol level is too high, this should be treated. But in both these and in any other situations in which there is evidence of an increased risk of a vascular event, long-term, low-dose aspirin prophylaxis should also be considered.

The future of aspirin

When used in vascular disease, aspirin has really become the victim of its own success and further placebo-controlled trials in vascular disease are neither acceptable nor necessary, the cost benefit of aspirin prophylaxis is so well established and so highly attractive. At the same time, concern has repeatedly been expressed that, despite convincing evidence of its effectiveness, the knowledge of the benefits of aspirin and its use by doctors is much less than desirable. Everything possible should, therefore, be done to promote aspirin prophylaxis in appropriate subjects, and to achieve a high level of compliance in tablet taking(5).

Salicylates occur widely throughout nature and have a wide variety of essential functions in plants. It is perhaps not surprising, therefore, that new uses of aspirin other than in vascular diseases are being found.

On present evidence perhaps the most hopeful new use of aspirin is in cognitive decline and dementia. Clearly numerous factors are involved in cognitive decline, and as yet few of these are understood. The most obvious cause is of course a stroke, but it is now accepted that a less dramatic, and often unrecognised process of small repeated infarcts can contribute to cognitive decline in the elderly. This has become known as 'vascular dementia' or, 'multi-infarct dementia', and the fact that aspirin reduces the risk of stroke makes a reduction of cognitive decline through the prevention of multi-infarct lesions by aspirin a most reasonable expectation.

An anti-inflammatory effect of low-dose aspirin has been reported and this gives a possible biological basis for claims that anti-inflammatory

drugs, including aspirin, may reduce progression in Alzheimer's disease[36]. Clearly, more trials are urgently needed in this most important area.

Perhaps the most exciting possible new uses of aspirin is in colon and other cancers. A number of major cohort studies and smaller studies have reported a substantial reduction in colon and other gastro-intestinal cancers in habitual aspirin takers[38,39]. A number of randomised controlled trials have been set up to examine this further.

SUMMARY

Many folk remedies used since pre-historic times have depended upon salicylates for their effect. Their first recorded mention was by Hippocrates around 400 BCE, who recommended an infusion of willow leaves for the relief of pain in childbirth. One hundred years ago, in 1897 aspirin was formulated from salicylic and acetic acids. It was the first drug to be synthesised and its formulation is regarded as the foundation of the modern pharmaceutical industry.

The benefit of aspirin as a prophylactic after a thrombotic event was first reported twenty-five years ago and its use after coronary or cerebral thrombosis, and in patients judged to be at increased risk of a thrombotic event is now virtually mandatory, unless there are signs of intolerance.

The current phase of the aspirin story is, however not over, and its possible use in new conditions seems likely to ensure that it will long continue to play a remarkable part in clinical practice.

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แอสไพรินกับการลดความเสี่ยงในการเกิดโรคหัวใจและลมปัจจุบัน†

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แอสไพริน น่าจะเป็นยาที่ใช้ป้องกันโรคของหลอดเลือดหัวใจที่มีประสิทธิภาพและราคาถูกที่สุดในปัจจุบัน การศึกษาผลของการให้แอสไพรินขนาดต่ำวันละ 100 มก. ในผู้ป่วยกว่า 150 คน แบบ randomised controlled trial ในการลดอัตราเสี่ยงของการเกิดกล้ามเนื้อหัวใจตาย และลมปัจจุบัน (stroke) ได้ประมาณ 30 เปอร์เซ็นต์ การช่วยชีวิตและการป้องกันการเกิดโรคของหลอดเลือด (vascular event) มีประโยชน์มากในผู้ป่วยที่มีความเสี่ยง แต่สำหรับคนปกติที่สุขภาพแข็งแรงนั้น โอกาสเสี่ยงต่อการเกิดโรคหลอดเลือดต่ำจนไม่สมควรที่จะใช้ยาใด ๆ ในการป้องกัน

สำหรับผู้ป่วยกล้ามเนื้อหัวใจตายเฉียบพลันนั้น การให้แอสไพรินยิ่งเร็วจะยิ่งเพิ่มอัตราการรอดชีวิต ผู้ป่วยที่ได้รับการวินิจฉัยว่ามีความเสี่ยงในการเกิดโรคหลอดเลือดจากสาเหตุใดก็ตาม ควรได้รับคำแนะนำให้พกแอสไพริน เพื่อเคี้ยว และกลืน 1 หรือ 2 เม็ด ทันที เมื่อมีอาการเจ็บหน้าอกรุนแรง เฉียบพลัน

แอสไพรินมาจากเกลือซาลิซิเลตซึ่งพบทั่วไปในธรรมชาติ การใช้แอสไพรินในโรคหัวใจและหลอดเลือดนั้นสืบเนื่องจากฤทธิ์ของแอสไพรินต่อเกร็ดเลือด แต่จากงานวิจัยปัจจุบันพบประโยชน์อื่น ๆ ของแอสไพรินนอกเหนือจากการใช้ในโรคของหัวใจและลมปัจจุบัน (stroke) ด้วย คือ การใช้เพื่อลดความเสี่ยงในการเกิด dementia มะเร็งบางชนิด การเจริญของตัวอ่อนที่ผิดปกติและต้อกระจก

คำสำคัญ : กล้ามเนื้อหัวใจตายเฉียบพลัน, ลมปัจจุบัน, แอสไพริน 100 มก.

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