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Abstracts

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A POSSIBLE ROLE FOR ASPIRIN IN THE PREVENTION OF THE COAGULOPATHY OF HEAT STROKE. AMA Gader, S. Al-Mashhadani; S. Al Harthy; College of Medicine and King Khaled University Hospital, Riyadh-11461, Saudi Arabia.

Heat stroke is frequently complicated by activation of the clotting system which may progress to the often fatal DIC. The trigger to the activation process is obscure but the direct activation of platelets by heat is a possibility we set out to study. A series of experiments were undertaken in which platelet rich plasma (PRP), prepared from blood donors, was incubated at increasing temperatures: 38° to 45°C and then platelet aggregation was undertaken in response to decreasing low doses of ADP at 7.5 minute intervals. Hyperaggregability was manifested only when the incubation temperature reached 43°C and was maximum at 44°C before complete inhibition of responses at 45°C. Besides hyperaggregability was recorded upto 30 minutes incubation at 43° and 44°C and thereafter responses were totally inhibited.
THE EFFECT OF COOLING: After platelets have shown hyperaggregability at 44°C PRP was reincubated at 37°C and aggregation was undertaken thereafter at 15 minutes intervals for up to two hours. Hyper-responses remained unchanged in most samples and in others were enhanced further.
EFFECT OF ASPIRIN: When subjects ingested 100 mg of aspirin the night before, or when aspirin was added to PRP before starting the aggregation procedure, heat no longer induced platelet hyperactivity. However if aspirin was added to PRP after platelets display the heat-induced hyperaggregability, the responses were partially inhibited and significant activation remains. These findings lead us to conclude: (1) platelets can be activated directly by heat(2) this mechanism can be the trigger to the coagulopathy of heat stroke and is unaffected by cooling (body cooling being basic in the management of heat stroke)(3)early institution of antiplatelet therapy (aspirin) blocks the heat-induced platelet hyperactivity and may, therefore, be useful in the prevention of the coagulopathy of heat stroke

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LOW DOSE ASPIRIN: EFFECT ON PLATELET FUNCTION IN NORMAL SUBJECT. P. Prayoonwiwat*, T. Srichaikul*, C. Pulket*, U. Chantharaksri†, and M Tanyawannon†. Division of Hematology, Department of Medicine, Pramongkutklao College of Medicine, Bangkok, Thailand*, Department of Pharmacology, Faculty of Science, Mahidol University, Bangkok, Thailand†.

We did a double blind randomized placebo controlled trial at Pramongkutklao Hospital to determine the effects of different doses of aspirin on platelet function and prostaglandins in normal subjects and to find the optimal dose of this drug for inhibition of platelet function. The normal subjects were sixteen healthy volunteer medical students of Pramongkutklao College of Medicine who had normal blood chemistry, complete blood counts and platelet function, and who had not received any other drugs within a period of two weeks before the period of study. They were randomly divided into four groups. Each group received a single dose of either placebo or 20 mg, 40 mg, or 60 mg of aspirin at 7:00 a.m. for seven consecutive days. Bleeding time, platelet aggregation, plasma level of adenosine triphosphate (ATP) and thromboxane B_2 were measured before and one hour after ingestion of the dose on days 1, 2, and 7. In the aspirin group, there was no secondary aggregation with adenosine diphosphate (ADP) and no ATP release. The percentage of maximum inhibition of thromboxane B_2 generation by 20 mg, 40 mg, and 60 mg of aspirin was 74.3%, 95.1%, and 97.7% on the first day and 95.8%, 98.9%, and 98.9% on the seventh day, respectively. In control subjects, all platelet responses before and after placebo ingestion showed no difference. Conclusion: In normal subjects, a daily dose of 60 mg of aspirin can inhibit the platelet function as early as one hour after ingestion of the drug. The effect of 20 mg and 40 mg of aspirin lasted for 24 hours. Thus, a daily dose of 20-40 mg of aspirin effectively inhibited platelet function in normal subjects.

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ASPIRIN IN PATIENTS WITH RAYNAUD PHENOMENON ASSOCIATED WITH PERIPHERAL EMBOLI.
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Background: Raynaud phenomenon (R.P.) is characterized by monophasic or triphasic discolouration after cooling of the fingers. It may be associated with systemic or autoimmune diseases and its pathophysiological mechanism is unclear. Angiographic studies in patients with R.P. sometimes reveal peripheral emboli.
Study objectives: I: To determine the prevalence of peripheral emboli in patients with R.P.; II: To study the efficacy of 500 mg ASA daily on the signs and symptoms of R.P.-patients with emboli as compared to patients without peripheral emboli.
Design I: Angiography in all patients.
Design II: Prospective double blind placebo-controlled cross-over study of 3 weeks ASA versus 3 weeks placebo.
Patients: Forty one consecutive patients with R.P., excluding patients with systemic lupus erythematodes, scleroderma, rheumatoid arthritis, diabetes mellitus or hyperlipoproteinemia.
Results: Ten of all 41 patients showed peripheral emboli. In 5 of these patients an obvious isolated segmental stenosis of the brachial artery was found, while the remaining patients had a normal angiogram. The efficacy of ASA was studied in these 10 patients with peripheral emboli as compared to 10 patients with R.P. and a normal angiogram. None of the clinical parameters (duration, intensity and frequency of attacks) changed during ASA treatment (power > 80%).
Conclusions: R.P. may be associated in nearly 25% of the cases with peripheral emboli, and in half of these patients an isolated stenosis of the proximal artery can be found. There are no clinical methods to distinguish patients with emboli from patients without emboli. ASA 500 mg daily produced no significant effect on the frequency, duration and severity of the attacks.

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INHIBITORY EFFECT OF CORDYCEPS SINENSIS ON AGGREGATION OF HUMAN PLATELETS
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The effects of CsB,an extract of mycelium from Cordyceps Sinensis,on the inhibition of platelet aggregation induced by optimal concentration of ADP,collagen,epinephrine,U46619(a thromboxan A_2 agonist),A23187,and arachidonic acid (AA) were evaluated. Addition of CsB to platelet rich plasma (PRP) resulted in the inhibition of platelet aggregation induced by all of the above aggregating agents except A23187 and U46619. The effect of CsB inhibitive to AA was most magnificent and the effect to inhibit collagen and ADP can next, the inhibitory effect of CsB to epinephrine was different from person to person. The herb inhibited platelet aggregation and ATP release in a dose dependent manner. The I_{50} of CsB on inhibition of collagen induced platelet aggregation was approximately 11 ug/ml. 10-40ug/ml CsB can markedly increase the intracellular level of cyclic AMP(cAMP) up to 200-1800 P moles/10^9 platelets while 50 ng PGI_2 can rise the cAMP level to 110.17±36.20 P moles/10^9 platelets. Agonists differ in their sensitivity to inhibitory effect of CsB and PGI_2 on platelet aggregation induction. U46619 was most sensitive to PGI_2 and least sensitive to CsB. It suggests CsB might have some new mechanisms involved in the inhibition of platelets activation.