EFFECT OF SULFINPYRAZONE AND ASPIRIN ON PLATELET LOSS INDUCED *IN VITRO* BY ACTIVATED CHARCOAL HEMOPERFUSION. James F. Winchester, Charles D. Forbes, James M. Courtney, Colin R.M. Prentice, Glasgow University, Dept. Medicine, Royal Infirmary, Glasgow G40SF, and Bioengineering Unit, Strathclyde University, Glasgow G40NW, Scotland.

Activated charcoal hemoperfusion is used for drug overdosage and is potentially useful in [illegible] and chronic uremia. While hemocompatibility of early devices has improved, platelet loss still occurs, and may be associated with hemorrhage.

We have developed an in vitro circuit consisting of hemoperfusion column and lines with a total blood volume of 60 ml for use with human blood. Citrated whole blood hemoperfusion is associated with platelet depletion of similar magnitude for coated or uncoated charcoal (20%) whereas polymer coating of charcoal improved the profound platelet loss (90%) noted with perfusion of heparinized whole blood (3 U/ml) over uncoated charcoal.

At 14 day intervals, in a double blind cross over study, we compared the effects of 2 days treatment with aspirin (ASA)(600 mg/day) or sulfinpyrazone (SP)(800 mg/day) alone or in combination against placebo, on platelet adsorption on 2% hydrogel coated or uncoated charcoal, and platelet retention on cuprophan membranes PT_{150} and PT_{250} in a test cell system, in 8 healthy male volunteers. Bleeding time was also measured before and after treatment using a spring loaded device.

In a heparinized system, ASA, SP, and combination reduced mean platelet adsorption on coated charcoal from 53% to 41%, and mean platelet retention to membranes from 20% to 10%, whereas ASA alone reduced platelet adsorption on uncoated charcoal. Bleeding time was prolonged by ASA but not by SP. These agents are capable of modifying platelet loss in vitro and may do so in vivo.

DECREASE IN PLATELET AGGREGABILITY AFTER TOTAL OOPHORECTOMY. H. Yamazaki, T. Motomiya, M. Sonoda and N. Miyagawa. The Tokyo Metropolitan Institute of Medical Science, Tokyo, Japan.

Substantial clinical evidence indicates that large doses of estrogen frequenly result in thromboembolic disorders. Effects of estrogen on platelet aggregability were examined in women with uterine myoma before and after oophorectomy. Bilateral oophorectomy on 15 cases (48.7±0.12 yrs, mean±SE) and unilateral or no oophorectomy on 18 cases (control group:42.2±0.18 yrs) were performed with myomectomy of the uterus. On one day before and one day, one week and one month after the operation performed, their platelet count by Coulter counter, platelet volume by Coulter channelyzer and platelet aggregability by Sienco aggregometer were measured. 24 hrs total estrogen in urine was also determined. In the control group, platelet counts were 85.1± 4.9 % of the preoperated value one day after, 127.9±9.0 % one week after and 98.1±7.6 % one month after the operation. In the bilateral oophorectomy group, these were 82.4±5.2 % one day after, 124.0±4.7 % one week after and 96.1±4.8 % one month after. Both the groups showed the same change. Platelet aggregability by 3 uM ADP were 76.9±14.3 % one day after, 203.0±57.1 % one week after and 193.4±59.0 % one month after in the control, while 55.0±13.6 % one day after, 102.5±12.9 % one week after and 60.6±14.7 % one month after the operation in the total oophorectomy group. There was a statistically significant difference in the values obtained one month after the operation between the groups ($p<0.05$). Characteristic changes in platelet volumes were also observed. A significant correlation was observed between the platelet aggregabilities and the daily urinary estrogen excretion levels. The above results suggest that estrogen may enhance platelet aggregability in vivo.

MILD DEPRESSION OF PLATELET COUNT AND ALTERED PLATELET FUNCTION IN ACUTE PLASMODIUM FALCIPARUM INFECTION. E.N. Essien and M.I. Ebhota. Department of Haematology, University of Ibadan, Nigeria.

We recently examined a possible role and extent of involvement of plasmodium falciparum parasitaemia in our earlier report of significantly lower circulating platelet numbers in healthy adult Nigerians ($100-300 \times 10^3$/ul) compared with matched Caucasians ($150-140 \times 10^3$/ml); ($P<0.001$), and in view of recently reported association of plasmodium parasitaemia with thrombocytopaenia, consumptive coagulopathy (DIC) and other haemorrhagic syndromes. Ninety-eight febrile children aged between 8 months and 10 years whose fever was attributed to malaria parasitaemia only were admitted into the study. Platelet count, platelet aggregation, Factor VIII and FDP were determined on each blood sample which was collected before treatment was started. Tests were repeated 10–14 days later. It was found that the mean platelet count of 132,000±61,620/ul (1 SD) during illness and immediately after treatment was significantly lower than the count of 234,400±98,480/ul, 10–14 days later, ($t=6.496$; $P<0.001$). Significant thrombocytopaemia (Platelets 70,000/ul) was observed only in 6% of the subjects and none had any haemorrhagic symptoms. The effect was independent of age or degree of parasitaemia. Pre- and post-treatment leucocyte counts were similarly different (7952±2043 vs. 9221±3071 $t=-2.81$; $P<0.05$). Haematocrit values did not change significantly. However, in the response to platelet aggregating agents, it was found that the second wave of ADP-induced aggregation was regularly abolished during the parasitaemia.

It is concluded that in hyperendemic plasmodium falciparum environment, the malaria may often lead to mild depression of platelet count but that severe thrombocytopaenia is uncommon. The platelets may not be optionally functional.