Earlier observations of ours have suggested that, under in vitro conditions resembling those under which platelets function haemostatically in vivo, their activation is promoted by the red cells. Some of the evidence suggested that this is through limited haemolysis with release of ADP. However, newly determined time relationships make this uncertain. Could red cells provide ADP without haemolysis? Do their flow properties affect the process more? To analyse the problem, we are determining dependence of red cell deformability on membrane constitution; and release of haemoglobin and adeninenucleotides under different conditions. Ten percent human red cell suspensions in physiological saline flow under constant pressures through 2, 3, 4 and 5 μm micro pore filters, the flow rate measured continuously with an electronic balance. Initial flow rates are increased by fluidising agents, eg. ethanol, and decreased by agents with opposite effect. Our results are consistent with the new hypothesis of S.J. Singer on the mode of action of amphipathic agents, such as chlorpromazine, on red cell membranes.

Factors interfering with the enzyme system involved in the cleavage of Maillard-type compounds, may also interfere with haemostasis.

FREE ATP IN BLOOD DURING HAEMORRHAGE

When blood vessels are injured so that they bleed, circulating platelets adhere to the damaged vessel wall and aggregate within the first seconds. The mechanism of the initial platelet aggregation remains uncertain. To investigate the initiation stage of haemostasis, the carotid arteries of rats were punctured with a 100 μm needle and free ATP, as an indicator of CTP, was measured in the emerging blood. This was brought into contact with luciferin-luciferase in a polyethylene tube, internal diameter 0.8 mm. The light produced at the blood/ enzyme interface was measured with a sensitive photon - counting device which gave background counts of 1 photoelectron/sec. and could detect \( < 10^{-8} \) ATP in 2 μl blood.

When an artery was injured, the emerging blood contained about \( 10^{-7} \) M ATP in a first peak after about 2 sec. After about one min., the ATP concentration returned to a second peak of about \( 5.10^{-6} \) M. This was decreased by heparin (500 U/kg body weight) or by chlorpromazine (1 mg/kg). The observations suggest that the second peak represents ATP released from platelets. The source of ATP accounting for the first peak remains uncertain; possibly this ATP is released from red cells undergoing high shear stress from the haemodynamic effects of haemorrhage.