HAEMOSTASIS AND HIGH ALTITUDE. I.Singh and J.S.Chohan. Directorate General Armed Forces Medical Services, New Delhi, India.

There is a rise in platelet releasate and a decrease in antithrombin III (ATIII) which is reflected by decrease in prothrombin time (PT), bleeding time (BT), clotting time (CT), and serum glutathione peroxidase (GSH) activity. Platelet aggregability (PA) and platelet adhesiveness (PA) are also decreased. The decrease in PA and PA is reflected by a decrease in platelet count (PC) and platelet aggregation factor (PAF). The decrease in PA and PA is reflected by a decrease in platelet count (PC) and platelet aggregation factor (PAF).

After 7 days, platelet aggregation factor (PAF) is increased, platelet adhesiveness (PA) is decreased, and platelet aggregation factor (PAF) is decreased.

BLOOD COAGULATION IN HIGH ALTITUDE PULMONARY HYPERTENSION. I.Singh and J.S. Chohan. Directorate General Armed Forces Medical Services, New Delhi, India.

Blood coagulation studies were carried out in 36 Indian soldiers who were resident at altitudes between 3500 and 4500 m for 2 years. Compared with 10 sea-level controls, 6 of these 36 subjects who had developed pulmonary hypertension during their stay at high altitude showed a significant increase in plasma fibrinogen, fibrinolytic activity, platelet adhesiveness, platelet aggregation factor (PAF), factor V, and factor VIII. In the remaining 32 subjects who did not develop pulmonary hypertension there was a significant increase in plasma fibrinogen and fibrinolytic activity only.

The above differences between subjects who develop pulmonary hypertension at high altitude and those who do not develop pulmonary hypertension suggest that high altitude pulmonary hypertension is of thrombosis and fibrinolytic activity only.

THROMBOKINETIC STUDY TO PREDICT EPISODES OF THROMBOGENESIS. A.Kawamoto. Research Institute for Nuclear Medicine and Biology, Department of Medicine, Hiroshima University, Hiroshima, Japan.

To predict thrombosis and to evaluate anti-thrombotic agents, a thromboelastic study was done on subjects in thrombogenic state with cardio-vascular and hematologic diseases, artificial heart valve replacement, thrombocytopenia and thrombocytopenia. Platelets were labelled with 51Cr (Throm. Res. 8, Suppl. 11: 34, 1976). During thrombocytopenia (<10^4/mm^3), 2 of 4 cases with primary thrombocytopenia and 3 of 4 cases of chronic myelogenous leukemia (2 of whom developed thrombocytosis) had shortened survival. This increased turnover was reflected compensatory production of platelets and increased thrombocytopenia. Fourteen cases with artificial heart valve replacement showed short survival with proportionally higher turnover. Three cases with short survival despite high doses of diprydamole therapy developed thrombosis.

To evaluate platelet participation in the development of cerebrovascular lesion, cerebral infarction and/or bleeding in animal model, platelet survival was measured by 51Cr-methionine incorporation in the spontaneously hypertensive rat-stroke prone strain (SHR-SF, Okamoto-Yamori), which become hypertensive at age 5-23 weeks. Peak of 51Cr-methionine in platelets which develop hypertensive at age, 1-23 weeks occurs on 3rd. day after I.V. injection in SHR-SF and controls (SHR-15). Injection in SHR-SF and controls (SHR-15) was significantly higher in SHR-SF than the controls. Survival by 51Cr-methionine and by 51Cr labels (16th Int. Congr. Hemat. Sept. 1976, Kyoto, Abstr. Supp. 9-12-4, pp. 39) suggested shorter survival of SHR-SF in 10-12 weeks of age than controls. Short survival with increased turnover indicated increased consumption due possibly to vascular damage associated with the development of hypertension. Ability to predict strokes in adult rats has been examined.