

Haemorrhologic and Haemostatic Factors in Ischaemic Heart Disease

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Clinicians specially dealing with cardiovascular or circulatory problems, generally overlook the importance of blood and its constituents which are therapeutically modifiable as compared to other anatomical cardiovascular structures. Measurements of the flow behaviour of blood or plasma in vitro can be performed for one of the two reasons; for the diagnosis and monitoring of disease, or for the prediction and study of blood flow in vivo¹. Routine haemorrhological tests for the former include ESR and plasma viscosity. For prediction and study of the blood flow in vivo measurement of whole blood viscosity and the deformability of red and white cells in small bore capillaries can be performed. The arterial and venous occlusive diseases being of major medical importance haemorrhology is establishing its rightful place along side perfusion pressure and vessel diameter in the triad of determinants of blood flow and tissue perfusion^{2,3}.

Most workers accept that in the aetiology of coronary heart disease, there are two elements, atherogenesis and thrombogenesis. However, for more than 30 years atherosclerotic research became almost synonymous with researches on lipid and lipoprotein metabolism but part played by thrombi and haemostatic factors have not been the main-stream topics for investigations and most clinical trials and experimental tools were designed to produce gross hypercholesterolaemia⁴. Lately this imbalance was partly redressed by the publication of early results of the North Park Prospective Heart Study (NPHS) on haemostatic function and cardiovascular death⁵, which showed the association of cardiovascular death with raised fibrinogen level was at least as strong as that with raised cholesterol. Association of IHD with fibrinogen is also shown by some other authors⁶⁻⁹. It is known that elevation of fibrinogen levels increases blood viscosity which may further enhance the risk of thrombus formation. Fibrinogen is a major determinant of blood viscosity which if increased has a causal role in IHD¹⁰. Fibrinogen levels are also found to be higher in smokers^{4-8,11-13} and hence the increase in blood viscosity. Much of the association between smoking and IHD may be mediated by raised fibrinogen levels and increased blood viscosity.

Primarily haematocrit and plasma viscosity are important determinants in the rate of oxygen transport and delivery in the circulation and coronary blood flow¹⁴. According to the Gordon's equation¹⁴ oxygen delivery is inversely proportional to the haematocrit and plasma viscosity. Other haemostatic factors⁷ as well as haematocrit¹⁵⁻¹⁸ have also been found to be associated with IHD. Hypercoagulable states as well as thrombogenic stimuli can be reversed easily as compared to relatively irreversible atherosclerotic changes. Despite adequate control and increasing efforts to reduce the elevated levels of lipids, the incidence of LHD is constantly on rise. Therefore, inclusion of rheologic and haemostatic factors in coronary risk profile will have salutary effects.

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