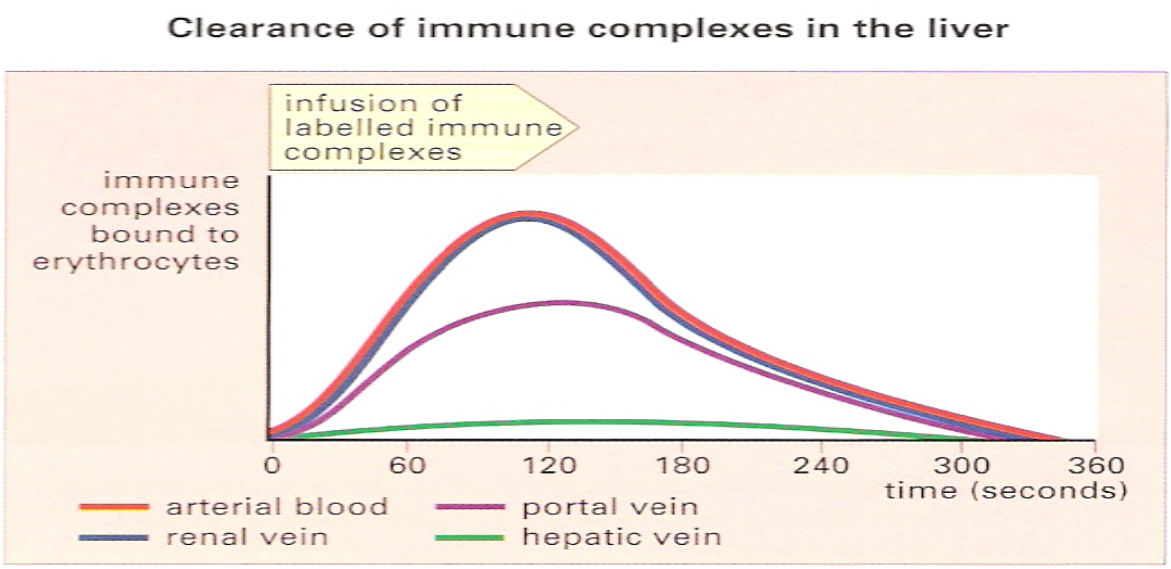

Figure-12 125|-BSA/anti-BSA complexes were infused into a primate over a period of 120 seconds. Blood was sampled from renal, portal and hepatic veins, and the level of immune complexes bound to the erythrocytes was measured by radioactive counting. The levels of complexes in the renal and portal veins were similar to that in arterial blood. However, complexes were virtually absent from hepatic venous blood throughout, indicating that complexes bound to erythrocytes are removed during a single transit through the liver.

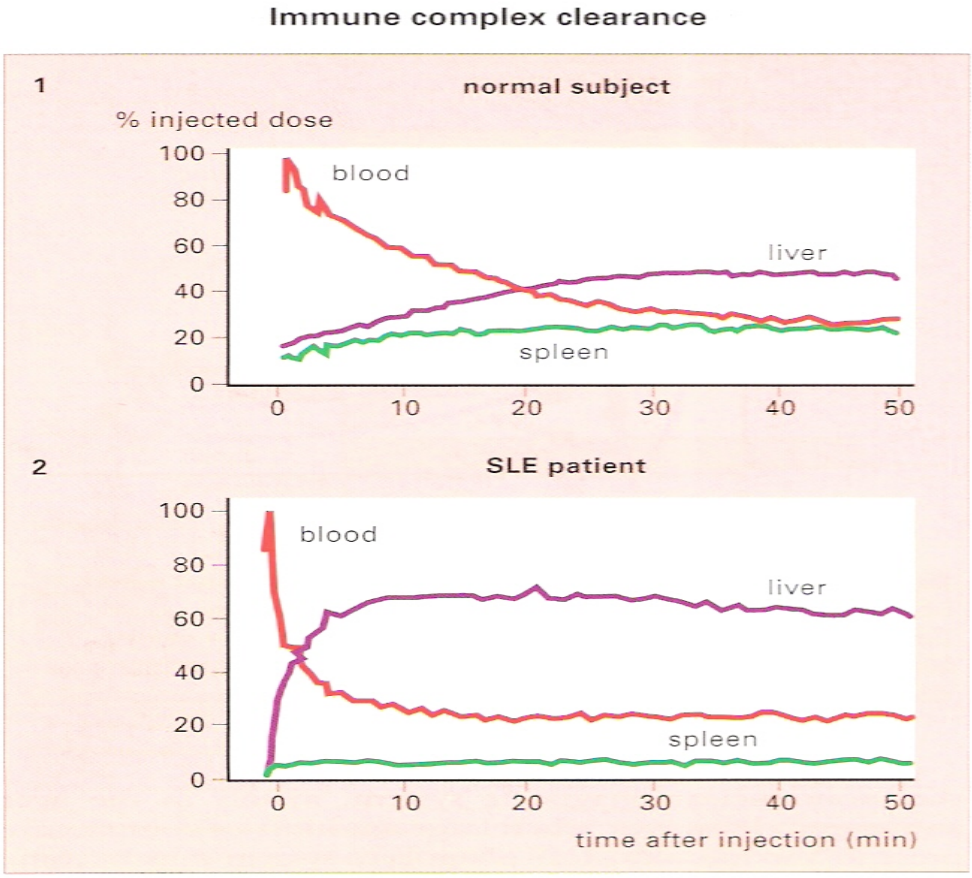

Figure-13 (1) Immune complex clearance in healthy normal subject. (2) Immune complex clearance in patient with SLE. Radiolabelled soluble complexes were injected intravenously and immune complex localization monitored by dynamic imaging. In the normal subject complexes remained longer in the blood through binding to CR1 on red cells, followed by clearance to the liver and the spleen, where immune complexes take part in immunoregulation. In the hypercomplementaemic SLE patient there was little binding to red cells but rapid clearance to organs such as the liver, with little localization to the spleen, leading to impaired immunoregulation which may be a factor in the persistence of autoimmunity.

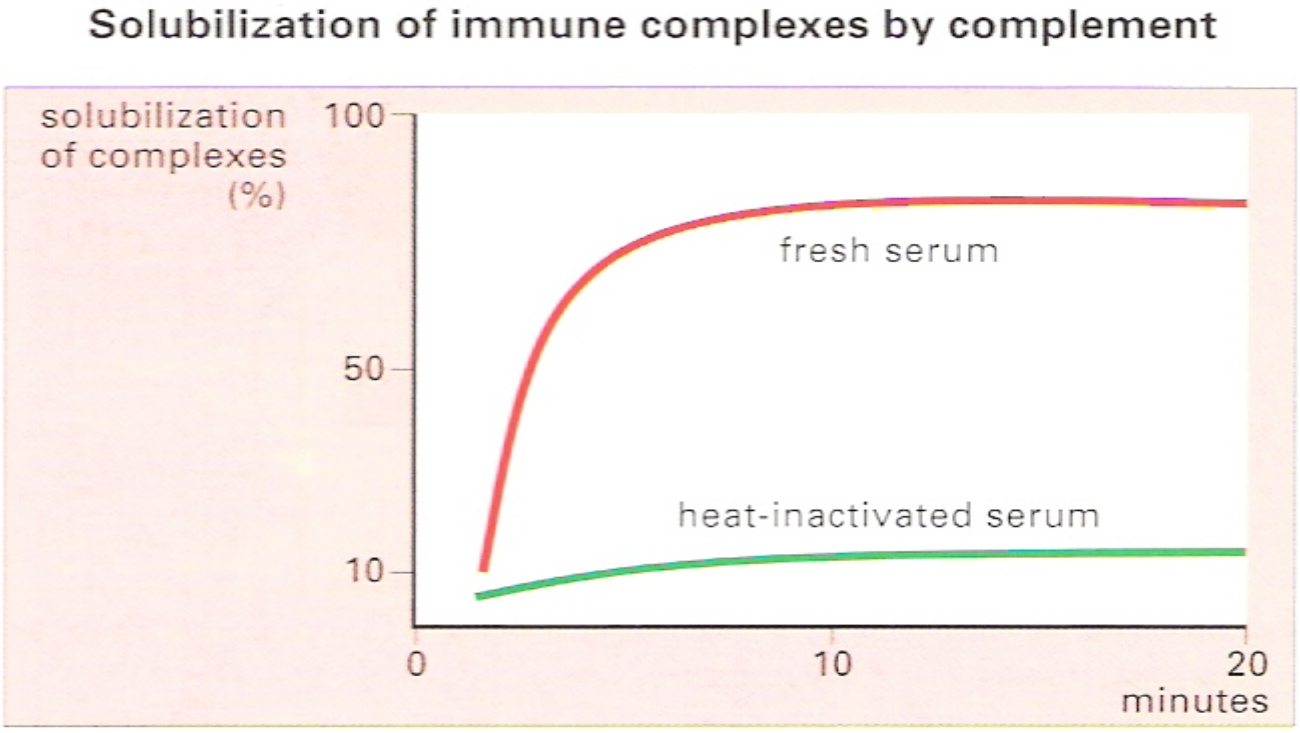

Figure-14 Complement can solubilize precipitable complexes in vitro. Addition of fresh serum containing active complement to insoluble complexes includes solubilization over about 15 minutes at 37^0C. Some of the complexes resist resolubilization. Heated serum (56^0C for 30min) lacks active complement and cannot resolubilize the complexes. Interaction of complement components C3b and C3d into the complex causes their solubilization by disrupting antigen-antibody bonds. Complexes that have been artificially connected by covalent bonds cannot be solubilized by complement.

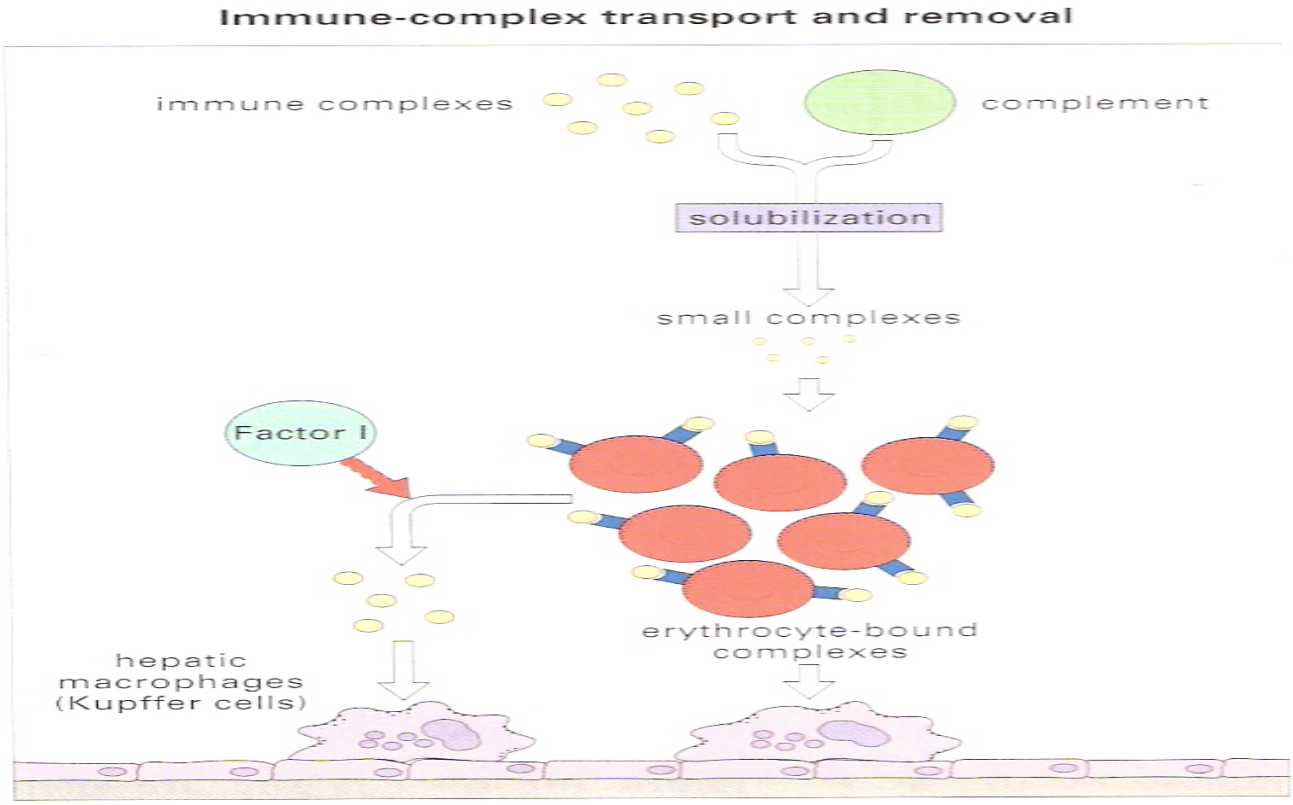

Figure-15 In primates, complexes solubilized by complement are bound by CR1 on erythrocytes and transported to the liver where they are removed by hepatic macrophages. Complexes released from erythrocytes by Factor 1 are taken up by cells (including macrophages) bearing receptors for Fc and complement.

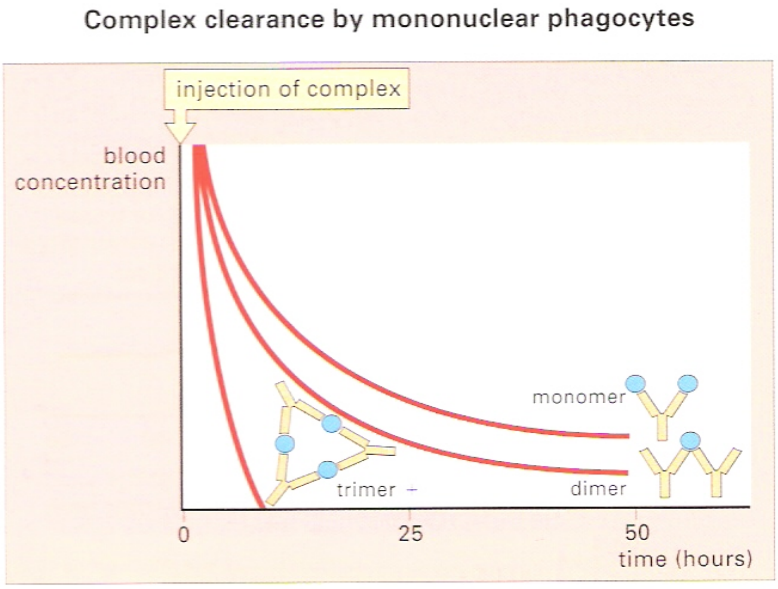

Figure-16 Large immune complexes are cleared most quickly because they present an IgG-Fc lattice to mononuclear phagocytes cells with Fc receptors, permitting higher avidity binding to these cells. They also fix complement better than small complexes.

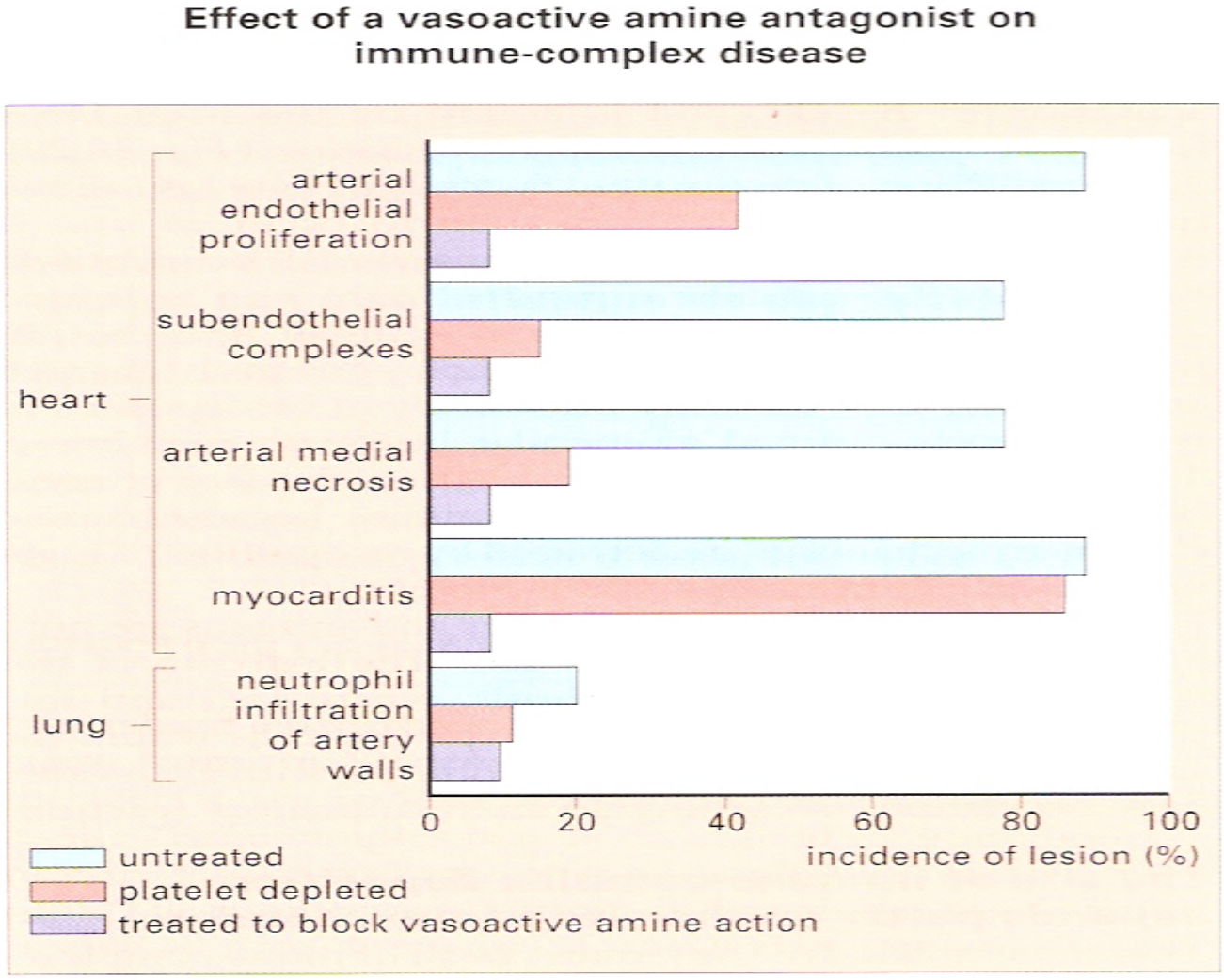

Figure-17 Serum sickness was induced in rabbits with a single injection of bovine serum albumin. The animals were either untreated, platelet depleted or treated with drugs or block vasoactive amine action. The incidence of serum sickness lesions in the heart and lung was scored. Drug treatment considerably reduced the signs of disease by lowering vascular permeability and thus minimizing immune-complex deposition.