6 Interaction of Vasomotion and Blood Rheology in Haemodynamics

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6.1 Introduction

The rheology of blood as an extremely non-Newtonian fluid has attracted the interest not only of physiologists but also of physicists, bioengineers and of course of clinicians. Unfortunately, the conclusions drawn from the in vitro measurements of apparent viscosity of blood in health and disease are often gross over-simplifications, ignoring the established facts about haemodynamics in general, let alone the important organ-specific differences in haemodynamics. It is therefore not surprising that the reaction of the general biomedical public is one of neglect or of almost emotional rejection — as evidenced in a recent editorial in an influential journal (Anonymous, 1977), in which the biological significance of the shear-rate dependence of apparent blood viscosity was totally denied.

At the other end of this credibility spectrum are markedly overenthusiastic “simplificateurs terribles” who attribute pathogenetic mechanisms (and thence disease processes) solely to an alleged “hyperviscosity” of blood, which they claim to have established by measuring apparent blood viscosity in large-bore viscometers. Unfortunately, classic viscosity concepts in the sense of continuum mechanics are not applicable to a haemodynamic analysis of the circulation, i.e.
for the flow analysis of blood perfusing a complex network of vessels of grossly
different diameter under extremely variable driving pressures.
Anomalous flow behaviour is a feature primarily of mammalian, and
especially human, blood. The rheology of blood containing nucleated red cells is
far more "conventional" in that it primarily depends on the viscosity of plasma,
the haematocrit value, and the temperature (Chien et al., 1971; Gaehgens and
Schmid-Schönbein, unpublished). In addition to these factors, in mammalian
blood the driving pressure and the vessel radius have pronounced effects on the
apparent viscosity. This "anomaly" of the macroscopic flow behaviour is a
consequence of the flow-dependent change of the mechanical behaviour of the
non-nucleated red cells. These can either passively participate in flow (by which
mechanism they maximize the fluidity of blood) or they can be united into
rouleaux and rouleaux networks. The latter give rise to "structural viscosity of
blood", a rheological state that can potentially interfere with or abolish
altogether the fluidity of blood. To make the situation even more complicated,
the cells' ability to be deformed in flow depends not only on the prevailing driving
pressures but also upon the micromechanical integrity of the red cells, a feature
subject to changes in the metabolism of the cells themselves or of the
parenchymal cells. Likewise, the tendency of aggregation can be grossly enhanced
by local or general changes in the protein composition of the plasma, particularly
the concentration of fibrinogen and other high-molecular-weight macro-
molecules. Last but not least, the locally effective haematocrit level in the micro-
circulation is subject to vast variations. These are brought about by vasomotion
(v.i.) and by the effect of white cells, which are much stiffer, on local blood
velocity. In summary then, the composition of the blood and hence its fluidity
can be vastly different in the microvessels (where all the important haemo-
dynamic events take place) and macrovessels (from which the blood is taken for
viscometry).

6.2 Present State of Blood Rheology

Most authors working in the field of blood microrheology agree to a remarkable
extent about the underlying microrheological causes of the non-Newtonian flow
behaviour of blood. As can be seen in a large number of recent review articles
(Bicher, 1972; Braasch, 1971; Charm and Kurland, 1974; Chien, 1972; Dintenfass
1971; Larcan and Stoltz, 1970; Merrill, 1969; Schmid-Schönbein and Wells, 1971;
Schmid-Schönbein, 1976; Wells, 1973) there is no more debate that red-cell
deforation and red-cell aggregation are prime determinants of the shear-
dependent apparent viscosity in large-bore rheometers in vitro, as well as
phenomena associated with normal rapid and abnormally retarded flow in the
microcirculation in vivo.

In an attempt to summarize the content of the above-mentioned work, the
present author, an experimental haemorheologist with the bias of a physiologist
with a medical background, has condensed our present ignorance as follows.
Blood is an "anomalous fluid", the viscosity of which cannot be defined, as it
varies with flow conditions. As blood is a dispersion of cells in plasma, the
computed coefficient of apparent viscosity primarily depends on the true
viscosity of the plasma and the effect of the dispersed cells on the flow of plasma.
In addition forces associated with high-molecular-weight plasma proteins, e.g.
fibrinogen and $\alpha_2$-macroglobulin, aggregate the red cells into rouleaux and