BLOOD VISCOSITY: A NON-FACTOR IN SPONTANEOUS HYPERTENSION

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INTRODUCTION: The spontaneously hypertensive rat (SHR) in many respects mimics conditions in essential hypertension in man and has proved to be a useful experimental model. The mechanism responsible for the elevated total peripheral resistance in this species is unknown though factors thought to be implicated include vessel wall hypertrophy, enhanced vascular reactivity, and arteriole rarification. Recently, utilizing capillary viscometry to compare the blood viscosity between SHR and normal Wistar controls, it has been proposed that an elevated blood viscosity of the SHR contributes to the hypertensive state (1). The purpose of the work reported here was to measure and compare the whole blood viscosity of the SHR with both the genetically more similar but normotensive WKY strain of rat and the normal Wistar rat (NWC) to determine if, in fact, hyperviscosity is a viable component of the hypertensive state.

METHODS: In this study 25 SHR, 19 WKY, and 11 NWC rats with an age range of 8-12 weeks were utilized. Blood was obtained from anesthetized rats (Nembutal 6 mg/Kg I.P.) by direct cardiac puncture. An abdominal incision was made to gain access to the caudal surface of the diaphragm. A second incision was made around the peripheral attachment to the ventral body wall, thus exposing the apex of the heart. An #18 gauge needle fitted onto a 10 ml syringe (containing 100 units of sodium heparin) was inserted into the left ventricle. Negative pressure was slowly created by withdrawing the plunger, thereby removing the blood without collapsing the ventricle. The blood was transferred to a similarly heparinized tube for use in subsequent viscosity and other hematological determinations. All blood viscosity studies were done using a Brookfield cone-in-plate viscometer at 37°C. The viscometer was calibrated against a standard oil with a viscosity of 4.3 cp at 37°C. Blood viscosity was determined at shear rates of 230, 115, 46, and 23 1/sec. Two readings were obtained at each rate and the average used. Each one ml sample of blood was run at each speed for one minute. In addition to the viscosity measurements, standard hematology was done. One ml of whole blood was drawn by a pipette into a Coulter Counter Model S-Plus and tested for hemoglobin, hematocrit, red blood cell count, white blood cell count, mean corpuscular volume, mean corpuscular hemoglobin, and mean corpuscular hemoglobin concentration.

RESULTS: No statistically significant differences in any hematological parameter between the three groups was demonstrated. The results obtained from the blood viscosity measurements are shown in the Table with viscosities at each shear rate expressed as a mean ± (SD). As anticipated, all 3 strains of animals showed an increase in viscosity with decreasing shear rate. However, a comparison at each shear rate of the viscosities for the SHR with the WKY strains show no significant difference at any shear rate. Conversely, the normal Wistar control rat strain clearly has a significantly lower viscosity at the highest shear rate when compared against the pooled SHR/WKY data ($P<0.001$). This lower value of viscosity is evidenced at all shear rates except at the lowest value where the difference between the NWC group and the pooled SHR/WKY data becomes statistically not significant. These differences are present in spite of no difference in hematocrit among the 3 strains (SHR = 38.8±8.6, WKY = 41.0±4.4, NWC = 40.4±2.3).

Shear Rate	BLOOD VISCOSITY SHR	WKY	NWC
230	4.36 (0.47)	4.35 (0.64)	3.85[a] (0.21)
115	4.69 (0.54)	4.65 (0.74)	4.23[b] (0.19)
46	5.48 (0.72)	5.58 (1.04)	5.03[c] (0.31)
23	5.93 (1.03)	6.39 (1.54)	5.83[d] (0.56)

a) $p<0.001$; b) $p<0.01$; c) $p<0.05$; d) $p=0.16$

CONCLUSIONS: The results of the present study confirm the findings of De Clerck et al (1) that the SHR strain has a higher blood viscosity than the normal Wistar control strain. Though he showed a 17% higher viscosity the present findings indicate an average of 11.7% value. This may be accounted for by differences in shear rate at which viscosity was determined. It is clear that the difference between the NWC and the other two strains decrease as the shear rate decreases. However, on the central point as to whether the blood viscosity elevation is a component in the hypertensive state, the present findings indicate this not to be true. Since no difference between the blood viscosity of the hypertensive SHR and the normotensive WKY strains was demonstrated, it is concluded that hyperviscosity by itself is not a significant factor in the hypertension demonstrated by the spontaneously hypertensive rat.

REFERENCES:

1. De Clerck, F., M. Beerens, L. Van Gorp and R. Xhonneux. Blood Hyperviscosity in spontaneously hypertensive rats. Thrombosis Research 18:291-295, 1980.

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