Plasma and Whole Blood Viscosity Changes in Shock and after Dextran Infusion

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THE RELATION of viscous properties of blood to perfusion pressure and flow has received considerable attention.[4, 6-8, 17, 21] Many studies emphasize the relationship of viscosity alterations to the microcirculatory changes in disturbed circulatory states. Sludging, or as it is more properly termed, cellular aggregation, was described by Knisely.[12, 13] Gelin[6-8, 17] and others observed aggregation in bulbar conjunctivae and hepatic sinusoids by vital microscopy after burns, hemorrhage, trauma and high viscosity dextran administration. Moreover, reversal of cellular aggregation with return toward normal circulation was seen after low viscosity dextran (LVD) administration[6-8] and peripheral circulation of the frog hindlimb was improved after LVD.[9] Microcirculatory effects of high and low viscosity dextran have been observed in intact animals and correlated with whole blood viscosity alterations, hematocrits, sedimentation rates and hepatic morphologic changes.[17]

The therapeutic effects of dextrans have been attributed to increased plasma volume and associated reduction of hematocrit.[10, 14, 20] However, several other mechanisms such as reversal of cellular aggregation,[18, 19] changes of surface charge of the red cell membrane[2] and physiochemical properties of the protein envelope of the erythrocyte[15] are plausible explanations of improved flow after LVD administration.

In clinical studies, blood viscosity changes were reported by Gelin[1, 7, 11] and others[3, 5, 16, 22] following hemorrhage, trauma, surgical operation and burns. Dintenfass[5] reported ten-fold increase in blood viscosity of patients suffering from thrombosis and coronary occlusion as compared with normal subjects. The relation of hematocrit to viscosity of blood has been studied extensively by Wells *et al.*[22]

The present study was undertaken to investigate plasma and whole blood viscosity alterations in a series of normal subjects, and patients who were in traumatic shock. Secondly, the influence of LVD administration on blood viscosity of these two groups was studied. To correct for the influence of hematocrit changes (incident to either LVD infusion or the postoperative states) on viscosity, measurements were made on blood which has been reconstituted to a constant hematocrit.

Methods and Materials

Clinical Material. Studies were performed on 22 patients admitted to the surgical services of Cook County Hospital, six normal preoperative adult subjects, and 16 postoperative patients in shock.

Method of Viscosity Measurement. Relative viscosity measurements of 2.0 ml. sam-

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TABLE 1. *Reproducibility of Multiple Viscosity Measurements Made on a Single Blood Sample and on Multiple Aliquots of Blood from A Single Patient*

	Inverse Seconds			
	21	42	106	212
Data from 20 different measurements on a single sample of blood. (Mean and S.E.)	11.1 ± 0.6	7.7 ± 0.10	5.3 ± 0.14	4.3 ± 0.11
20 Different measurements on 20 separate aliquots of blood from a single patient. (Mean and S.E.)	6.7 ± 0.4	5.0 ± 0.5	3.9 ± 0.08	3.0 ± 0.12

ples were made on a Brookfield microcone-plate viscometer, Model LVT (Brookfield Engineering Laboratories, Inc., Stoughton, Mass.). Measurements of shear stress were made at 4 rotational speeds, representing shear rates of 21, 42, 106 and 212 inverse seconds. Comparisons were made to values obtained with 2.0 ml. distilled water. Temperature within the water-jacketed sample cup was maintained at 37.0° C. Readings were made after preliminary shearing of blood for at least four minutes at each shear rate. This interval was necessary for stabilization of the system. Scale readings were made at each rate in succession beginning with the lowest shear rates and allowing sufficient time for the system to reach equilibrium at each shear rate. This sequence of measurements was repeated three times and three values at each shear rate were averaged. If there were gross inconsistencies, the measurements were repeated from the beginning.

The reproducibility of twenty different measurements on the same blood sample and reproducibility of 20 different aliquots of blood taken from a patient are shown in Table 1. Reasonably good agreement was obtained. The extent of the artifacts incident to reconstituting blood samples was

TABLE 2

A. Viscosity measurements before and after reconstituting blood to constant hematocrit.

	Inverse Seconds			
	21	42	106	212
1. Observed Hematocrit (48%)	14.4	10.9	9.6	7.6
Reconstituted to 48% Hematocrit	14.5	10.9	9.6	7.6
2. Observed Hematocrit (49%)	14.3	8.8	7.0	5.7
Reconstituted to 48% Hematocrit	14.1	8.7	7.0	5.8
3. Observed Hematocrit (50%)	11.3	9.0	9.1	7.7
Reconstituted to 48% Hematocrit	11.1	8.9	9.1	7.6

B. Viscosity measurements on blood reconstituted to constant hematocrit by six separate pipettings.

1.	14.9	8.7	6.0	4.5
2.	14.0	8.5	5.9	4.4
3.	14.4	8.8	5.9	4.4
4.	13.7	8.3	5.9	4.4
5.	13.7	8.8	5.9	4.4
6.	14.1	8.6	5.9	4.48
Mean	14.1	8.6	5.9	4.4
Standard error of mean	±0.2	±0.1	±0.01	±0.01

TABLE 3. *Blood Viscosity before and after LVD Infusion in Normal Subjects*

Patient	Before LVD					Immediately after LVD				
	Hct. %	Inverse Seconds				Hct. %	Inverse Seconds			
		21	42	106	212		21	42	106	212
1.	41	7.3	7.8	5.5	5.3	39	7.2	8.1	4.8	4.7
	48	9.5	11.1	6.4	6.1	48	9.2	9.8	5.4	5.2
2.	39	4.2	4.7	5.3	4.4	35	4.4	4.0	4.4	3.9
	48	7.3	6.0	6.1	5.5	48	7.4	6.3	6.5	5.9
3.	51	13.7	7.2	8.3	7.7	44	10.7	6.1	7.7	7.5
	48	12.3	6.3	7.1	7.2	48	12.8	6.9	8.4	5.4
4.	44	7.4	5.7	5.8	5.3	38	6.1	5.0	5.3	4.6
	48	10.0	8.4	7.4	6.6	48	10.0	7.3	6.8	6.5
5.	32	6.3	5.4	5.3	4.2	26	4.2	4.6	4.8	3.7
	48	10.9	8.2	7.8	6.0	48	12.3	9.4	8.7	6.6
6.	44	8.4	7.6	5.8	5.6	41	9.0	8.4	6.7	6.3
	48	10.1	8.9	7.0	6.7	48	11.2	10.0	8.3	7.2
Observed Hematocrit (Mean S.E. of mean)		7.9 ±1.3	6.4 ±0.5	6.0 ±0.5	5.4 ±0.5		6.9 ±1.0	6.0 ±0.8	5.6 ±0.5	5.1 ±0.6
Hematocrit 48% (Mean S.E. of mean)		10.0 ±0.7	8.1 ±0.7	7.0 ±0.3	6.3 ±0.2		10.5 ±0.8	8.3 ±0.7	7.3 ±0.5	6.1 ±0.3

Measurements were made at the observed hematocrits and at 48% hematocrit.

appraised by measuring blood viscosity before and after reconstituting blood of three patients whose hematocrits were 48, 49 and 50. Finally, the reproducibility of viscosity measurements was observed in reconstituted blood obtained from six separate pipettings of plasma and packed red cells. These findings, summarized in Table 2, suggest that the reproducibility of the measurements, pipetting errors and artifacts associated with preparation of the constant hematocrit blood sample were not unreasonable.

Protocol. Twenty ml. of venous blood was withdrawn and immediately anticoagulated with 0.15 mgm. heparin sodium. An aliquot of whole blood was taken for viscosity analysis; the remaining blood was centrifuged at 2,000 *g* for 30 minutes. The plasma was separated from packed red cells and a plasma sample was taken for analysis. Blood was reconstituted at constant hematocrit by adding 1.0 ml. plasma to 1.0 ml. packed red cells using volumetric pipettes. Considering the amount of plasma in the packed cells (packing fraction) it was expected that the reconstituted blood would have a hematocrit of 48%. Measured hematocrits in every instance came to within 0.5% of this value. The relative viscosity of aliquots of whole blood, plasma and reconstituted (48% hematocrit) blood were measured before LVD and immediately after infusion of 500 ml. LVD. When appreciable changes occurred, viscosity measurements were repeated after 24 hours.

Results

Normal Control Patients

Whole blood viscosity. Viscosity measurements of blood obtained preoperatively from 6 normal patients, who entered the hospital for elective hernioplasty, are summarized in Table 3. The dependency of whole blood viscosity on variations in shear

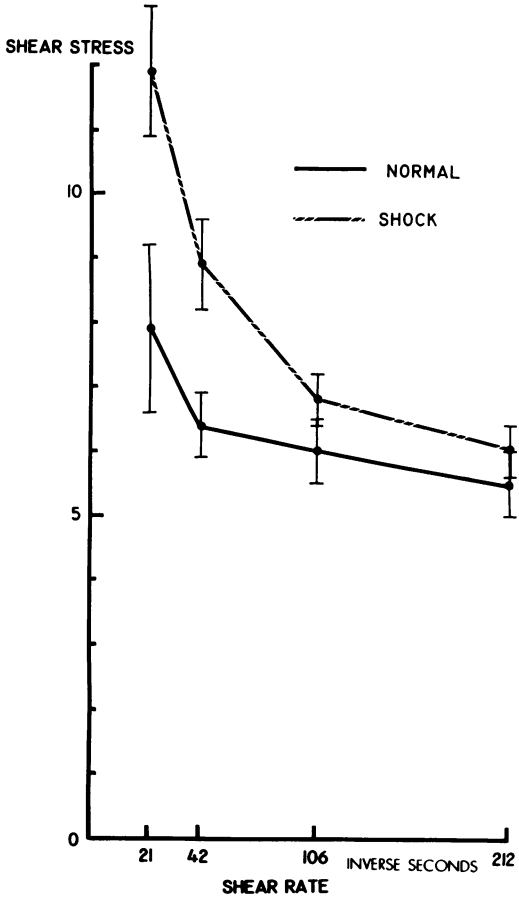

FIG. 1. Viscosity of whole heparinized blood obtained directly from a series of normal and shock patients. Observations were made at shear rates from 21 to 212 inverse seconds. The shear stress of the blood is expressed relative to values obtained with distilled water measured under comparable conditions. Dots represent mean values, bars the standard errors of means. The shear stress relative to distilled water is plotted against the shear rates in inverse seconds. The viscosity of blood obtained from normal subjects is significantly lower than that obtained from shock patients especially at the low shear rates.

rates is illustrated in Figure 1. Higher relative viscosities are seen with lower shear rates. Viscosity of whole blood was first measured at the observed hematocrit and was about 8 times that of water at shear rates of 21 inverse seconds; 5.4 times at 212 inverse seconds. Because of the wide range of hematocrits, the spread of viscosity values was wide. For these reasons, therefore, measurements were repeated with the same blood reconstituted to a 48% hematocrit. The values obtained from blood of a constant hematocrit are more consistent, the standard error of the means being smaller (Fig. 2). With hematocrits of 48%, the mean relative viscosity was ten times that of water at 21 inverse seconds and about six times that of water at 212 inverse seconds.

Plasma viscosity. The relative viscosity of plasma was much lower than that of whole blood; mean values at all shears were about twice that of water (Table 4). Large variations around these means result from calculations which represent small num-

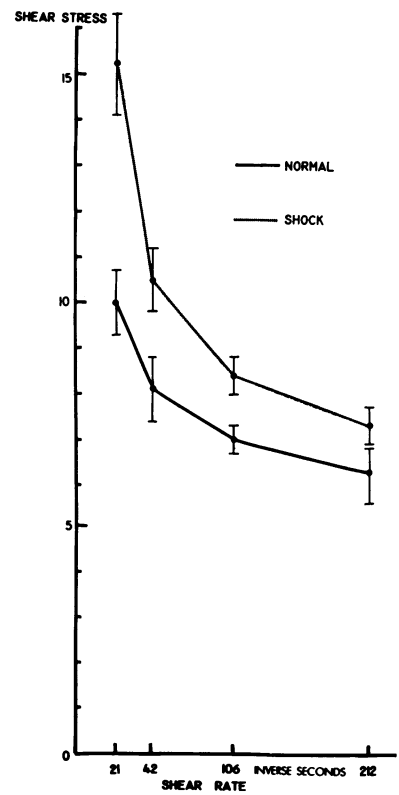

FIG. 2. Viscosity of whole blood reconstituted to 48% hematocrit obtained in the same series of normal and shock patients shown in Figure 1. The standard error of means of both control and shock groups are smaller and the differences between the groups are greater. Coordinate designations are the same as in Figure 1.

TABLE 4. *Plasma Viscosity of Normal Subjects before and after LVD Infusion*

Patient	Before LVD Inverse Seconds				Immediately after LVD Inverse Seconds			
	21	42	106	212	21	42	106	212
1. R. E.	1.8	2.6	2.0	1.9	1.5	2.5	1.6	1.6
2. H. H.	1.0	1.1	1.6	1.6	1.8	1.8	1.8	1.8
3. G. A.	2.7	1.2	2.0	2.0	2.6	1.3	2.1	2.1
4. G. G.	2.0	1.8	1.7	1.9	2.8	1.6	1.8	1.9
5. C. J.	2.3	2.0	2.0	1.6	1.6	1.8	2.1	1.7
6. F. G.	1.7	2.0	1.8	1.8	2.5	2.5	2.1	2.1
Mean and standard error of mean	1.9 ±2.4	1.8 ±2.3	1.8 ±0.8	1.8 ±0.7	2.1 ±2.3	1.6 ±1.3	1.9 ±0.8	1.8 ±0.8

bers expressed as a percentage of other small numbers.

Response to LVD infusion. After LVD infusion in normal subjects, there were small decreases in the relative viscosity of whole blood at all shear rates. However, these changes were not statistically significant. When the blood was reconstituted to a constant hematocrit, there were slight increases which also were not statistically significant (Table 3). Changes in plasma relative viscosity after LVD varied at different shear rates as illustrated in Figure 5.

Postoperative Shock Patients

Whole blood viscosity. Table 5 details results of studies of 16 postoperative patients who were in shock at the time of study. The relative viscosity of whole blood illustrated in Figure 1 again showed shear rate dependency: at shear rates of 21 inverse seconds, whole blood viscosity was about 12 times greater than water and at 212 inverse seconds it was about 6 times that of water. Values obtained from shock patients were increased over those from normal patients; these differences were statistically significant ($p < 1\%$). When blood was reconstituted to a constant hematocrit, the relative viscosity as the shear rate of 21 inverse seconds was about 15 times that of the water, while at 212 inverse seconds it was about seven times that of distilled water.

Plasma viscosity. Plasma viscosities in postoperative shock patients are summarized in Table 6. Values were slightly higher than normal, but the differences were not statistically significant. The plasma relative viscosities decreased slightly but not significantly after LVD infusion.

Response to LVD infusion. After administration of 500 ml. LVD, the relative viscosity of whole blood decreased at all shear rates. This was accompanied by pronounced decreases in hematocrit. Greater changes were seen at the lower shear rates. These changes were statistically significant (Table 5). The relative viscosity of blood which was reconstituted at a fixed hematocrit also decreased significantly (Table 5). Again, greater changes were observed at the lower shear rates.

The relationship between the initial viscosity of blood from patients in shock and the change in viscosity incident to LVD infusion is illustrated in Fig. 3. Also, the change of plasma viscosity after LVD is illustrated in Figure 4. In general, these findings suggest that the higher the initial whole blood or plasma viscosity value, the greater the decrease after LVD infusion. When preinfusion value is normal or lower,

TABLE 5. *Blood Viscosity before and*

Patient	Diagnosis	Hct. %	Before LVD Inverse Seconds 21	42	106	212
1	Gun shot wound, abdomen	48	14.4	10.9	9.6	7.6
		48	14.5	10.9	9.6	7.6
2	Perforated duodenal ulcer	37	4.4	4.8	4.8	3.6
		48	11.1	8.4	8.9	6.9
3	Gangrenous bowel obstruction	57	22.0	21.0	10.6	9.2
		48	12.7	8.0	6.6	6.3
4	Bowel obstruction with perforation and gangrene	42	12.1	7.1	5.3	5.2
		48	16.0	9.3	6.7	6.0
5	Stab wound, abdomen	36	11.0	7.2	5.6	5.6
		48	14.6	9.2	6.6	6.9
6	Strangulated ventral hernia	33	13.6	8.1	6.2	6.5
		48	18.6	11.2	8.0	7.6
7	Upper gastro-intestinal bleeding, perforated duodenal ulcer	50	11.3	9.0	9.1	7.7
		48	11.1	8.9	9.1	7.6
8	Septic pyelonephritis	49	14.3	8.8	7.0	5.7
		48	14.1	8.7	7.0	5.8
9	Small bowel obstruction with gangrene	36	16.0	9.6	7.7	6.9
		48	23.6	14.4	10.9	9.2
10	Stab wound, rupture of spleen	30	17.3	10.7	7.4	7.5
		48	23.6	14.3	9.3	8.7
11	Carcinoma, pancreas	34	8.8	5.7	4.6	4.2
		48	13.7	10.7	7.9	6.8
12	Diabetes	32	11.2	9.5	7.6	8.1
		48	22.0	17.6	12.4	12.1
13	Septic shock	38	8.0	5.3	5.5	4.3
		48	14.2	9.0	8.1	6.2
14	Gunshot wound, abdomen	37	8.4	7.8	6.5	6.0
		48	11.2	10.6	8.1	7.5
15	Gun shot wound, abdomen	45	9.0	6.9	6.4	5.1
		48	10.3	7.6	7.2	5.5
16	Multiple fractures	41	10.1	7.7	7.2	5.5
		48	11.5	7.8	7.4	5.9
Whole Blood at Observed Hematocrit Mean and standard error			11.9 ±1.0	8.7 ±0.9	6.8 ±0.4	6.1 ±0.4
p Values using statistical evaluations calculated on the basis of paired distributions where the control measurement of each experiment was compared with the corresponding measurement taken at each time period.						
Reconstituted to 48% Hematocrit Mean and standard error			15.2 ±1.1	10.5 ±0.7	8.4 ±0.4	7.3 ±0.4
p Value						

Measurements were made at observed hematocrits and at 48% hematocrit.

after LVD Infusion in Shock Patients

Immediately After LVD					24 Hrs. After LVD				
Hct. %	Inverse Seconds				Hct. %	Inverse Seconds			
	21	42	106	212		21	42	106	212
39	7.0	6.6	7.3	5.9	43	9.5	8.4	8.3	6.2
48	11.5	9.5	8.7	7.3	48	12.1	10.5	9.8	8.0
25	3.3	3.8	4.2	3.4	28	5.0	4.2	5.4	4.2
48	9.8	8.2	7.8	6.3	48	11.4	9.3	9.1	7.1
37	7.0	4.4	4.1	3.8		Patient expired			
48	9.2	5.4	4.8	4.4					
30	7.5	5.0	4.0	4.0		Patient expired			
48	14.3	8.1	6.0	5.9					
29	9.3	6.3	4.8	4.8	29	9.3	6.3	5.1	5.3
48	13.3	8.7	6.2	6.4	48	21.0	12.5	8.3	8.0
24	7.6	5.0	4.2	4.3	25	7.6	4.7	4.5	3.9
48	16.0	9.5	6.8	6.3	48	15.1	10.0	6.6	6.8
38	7.0	6.8	6.3	5.5	35	4.8	5.3	6.0	5.0
48	11.0	8.8	8.6	7.3	48	9.6	8.8	8.2	7.0
39	16.6	8.1	6.0	4.9		Patient expired			
48	16.9	10.0	7.3	6.0					
30	15.0	8.1	6.9	6.2	31	14.9	8.0	6.8	6.3
48	24.3	14.4	10.0	8.8	48	24.8	14.4	10.4	9.0
21	9.3	5.7	5.8	5.3	22	9.3	6.8	5.7	5.7
48	22.6	13.2	8.8	8.7	48	23.6	13.6	9.2	8.7
28	6.5	5.3	4.3	4.2	23	7.6	6.5	5.2	4.3
48	12.7	10.0	6.9	6.9	48	11.5	9.0	6.7	6.0
29	10.0	9.4	8.6	8.4	32	11.7	11.1	9.2	9.5
48	18.8	16.4	14.0	12.2	48	19.5	17.8	14.2	12.4
31	6.4	4.8	5.0	3.8	37	8.2	6.1	6.3	5.0
48	10.0	7.1	7.0	5.5	48	11.8	8.0	7.7	6.1
33	5.5	5.6	4.7	4.9	40	8.8	8.1	6.4	6.3
48	11.2	10.0	7.8	7.6	48	11.3	10.3	7.5	6.9
41	7.4	5.7	5.8	4.6	40	8.1	6.1	6.4	4.9
48	10.6	8.1	7.4	6.0	48	11.6	8.5	8.2	6.4
36	6.8	6.0	6.4	4.9	32	11.3	8.4	8.0	6.0
48	11.5	8.3	7.7	6.3	48	15.1	10.7	10.3	7.9
	8.2	6.0	5.4	5.0		8.9	6.9	6.3	5.0
	±0.8	±0.4	±0.3	±0.3		±0.7	±0.5	±0.4	±0.3
	0.005	0.01	.05	.05		0.025	0.1	N.S.	N.S.
	14.0	9.8	7.9	7.0		15.2	11.0	9.0	7.7
	±1.1	±0.7	±0.5	±0.5		±1.4	±0.8	±0.6	±0.5
	0.01	0.01	0.025	N.S.		N.S.	N.S.	N.S.	N.S.

TABLE 6. *Plasma Viscosity Before and after LVD Infusion in Shock Patients*

Patient	Diagnosis	Before LVD				Immediately After LVD				24 Hrs. After LVD			
		Inverse Seconds				Inverse Seconds				Inverse Seconds			
		21	42	106	212	21	42	106	212	21	42	106	212
C. R.	Gun shot wound, abdomen with sepsis	2.4	2.4	2.6	2.2	4.4	4.0	2.7	2.2	2.3	2.4	2.6	2.3
H. C.	Perforated duodenal ulcer	1.8	1.5	1.6	1.5	1.8	1.8	1.8	1.6	1.8	1.9	2.0	1.8
H. J.	Gangrenous small bowel obstruction with gas gangrene of abdominal wall third postop. day	3.2	2.4	2.3	1.9	3.1	2.3	1.7	1.5	Patient expired			
Z. E.	Bowel obstruction with peritonitis	2.1	1.8	1.5	1.8	2.6	1.8	1.7	1.7	Patient expired			
R. L.	Stab wound, abdomen	3.1	2.5	1.9	2.2	3.1	2.2	2.1	2.3	3.3	2.2	1.9	2.1
K. G.	Strangulated incisional hernia	7.6	4.0	3.2	3.1	2.6	1.9	1.7	2.0	Patient expired			
H. R.	Massive gastro-intestinal bleeding, perforated duodenal ulcer, cardiac arrest	3.0	2.1	2.1	2.0	3.0	1.7	2.0	1.8	2.1	1.9	2.3	1.8
E. N.	Septic pyelonephritis	2.6	3.1	2.4	1.9	3.5	2.2	1.9	1.9	Patient expired			
D. E.	Gangrenous small bowel obstruction	5.1	2.8	2.5	2.5	4.3	2.8	2.4	2.3	4.2	2.6	2.4	2.5
G. C.	Stab wound, abdomen, ruptured spleen	2.6	2.0	1.5	1.8	2.3	2.1	1.9	2.1	3.3	2.5	1.9	2.1
H. R.	Carcinoma, pancreas with obstructive jaundice	2.2	2.2	1.6	1.9	2.0	2.2	1.7	1.9	2.2	1.9	2.4	2.1
B. V.	Diabetes with bleeding	4.5	3.2	3.2	2.6	3.3	2.9	2.8	3.0	2.8	2.5	2.9	3.0
C. S.	Septic shock	2.1	1.6	1.8	1.6	2.0	1.6	1.9	1.6	2.6	1.9	2.2	1.9
A. B.	Shot gun wound, abdomen	2.0	2.1	1.9	2.1	1.7	1.9	1.8	1.9	1.7	1.9	1.7	1.8
T. J.	Shot gun wound, abdomen	1.7	1.8	1.8	1.5	2.5	2.1	2.0	1.7	2.6	2.4	2.5	1.4
Mean and standard error of mean		3.0 ±3.8	2.3 ±1.6	2.1 ±1.3	2.0 ±1.0	2.8 ±2.0	2.2 ±1.5	2.0 ±0.9	2.0 ±0.9	2.6 ±2.0	2.2 ±0.8	2.2 ±1.1	2.1 ±1.2

no appreciable decrease in viscosity occurs with LVD administration.

Discussion

Measurement of blood viscosity is complex. Blood viscosity may be affected by temperature, concentration of cells, physiochemical properties of the red cell membrane, concentrations of the various plasma proteins, especially those of high molecular weight, the presence of chylomicrons, the concentrations and composition of the various lipoproteins, the presence of abnormal or denatured proteins, perfusion pressures, the velocity of blood flow, velocity gradients between layers of the flowing blood, the geometry of the vessels particularly the diameters of the small vessels and the state of red cells (e.g., cellular aggregation). Many of these factors are not directly measurable *in vivo* and most are not measurable in patients. More-

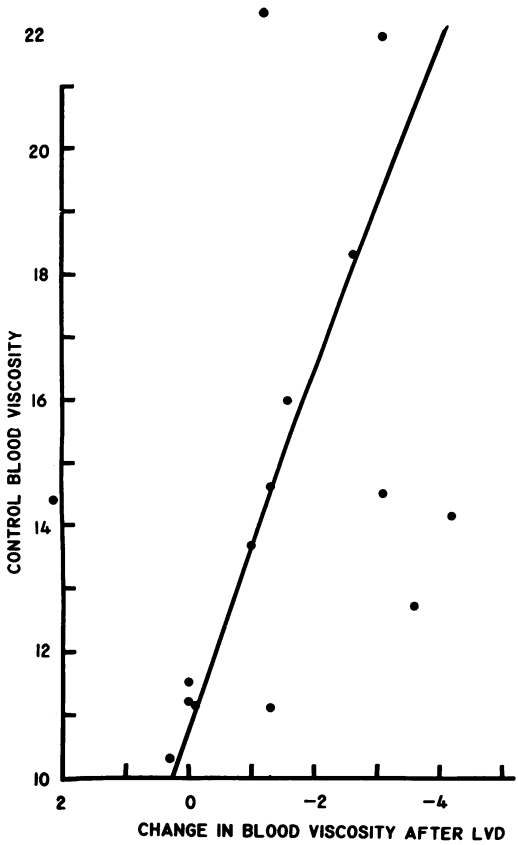

FIG. 3. The changes in viscosity of 48% hematocrit blood after low viscosity dextran administration are plotted against control viscosity measurements at 21 inverse seconds. Data show greater decreases in whole blood viscosity after LVD administration with higher control whole blood viscosity measurements.

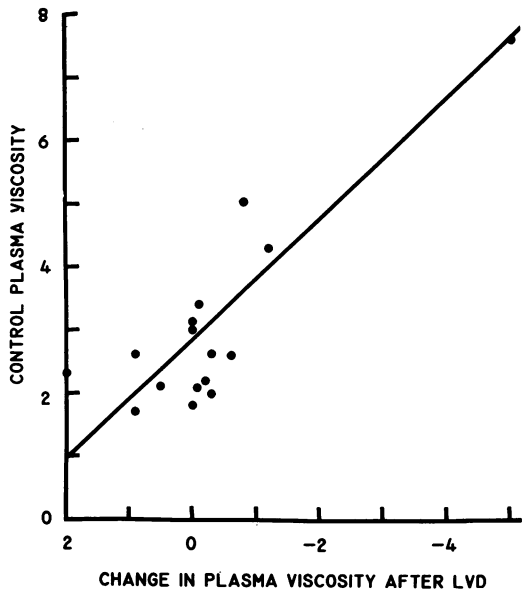

FIG. 4. The changes in plasma viscosity after low viscosity dextran administration are plotted against the control plasma viscosity measurements at 21 inverse seconds. Data show greater decreases in plasma viscosity after LVD administration with higher control plasma viscosity measurements.

over, when *in vitro* measurements are used, different values may be obtained by different methods. Although *in vivo* methods of measurement are not currently available, an index of the relative viscosity of blood samples observed *in vitro* may be obtained by measuring the shear stress at various shear rates and comparing these values to those obtained under comparable conditions using distilled water. In this way, viscosity of blood relative to water is calculated and presumably may reflect actual viscosities in the *in vivo* system.

The data of the present study show significantly increased relative viscosity of whole blood from patients in postoperative shock. The greatest changes were observed in the lowest shear rates which were measured in this study. Gelin [1] emphasized the marked alterations at low shear rates. This may have special significance in the disturbances associated with low flow states during postoperative shock.

A major uncertainty in the interpretation of viscosity measurements is variation in hematocrit, since viscosity of whole blood is hematocrit dependent. Thus, it becomes important to differentiate influences incident to hematocrit changes from those attributable to viscosity alterations when comparisons are made between blood obtained from normal and shock patients. Similarly, cognizance must be made of the hematocrit change which occurs after administration of plasma expanders.

Comparability is enhanced when correction is made for obvious hematocrit altera-

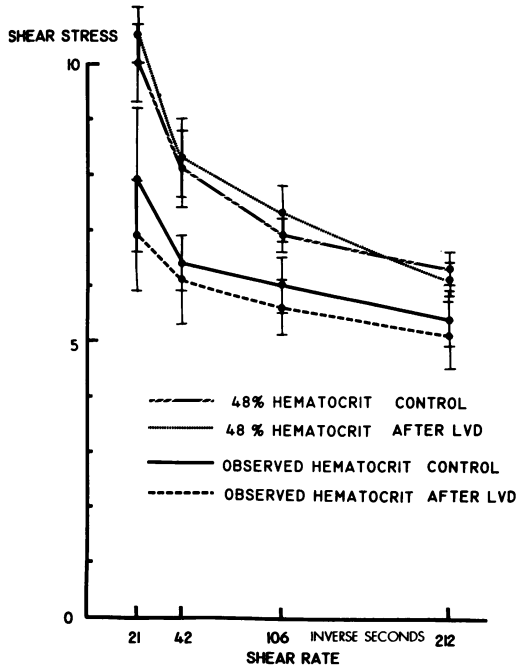

FIG. 5. Responses of whole blood viscosity at both observed and 48% hematocrits to low viscosity dextran administration in normal subjects. As indicated by the lower 2 lines, the viscosity of whole blood at the observed hematocrit decreased after low viscosity dextran administration. As indicated by the upper 2 lines, however, the same blood, when reconstituted to 48% hematocrit, showed no significant changes after LVD administration.

tions. The method proposed in the present study is to reconstitute whole blood to a fixed hematocrit by centrifugation and pipetting plasma and packed red cells in a specified manner. The artifacts produced when this is carefully done do not appear to alter greatly measurements of relative viscosity by the standard technic employed.

In normal subjects, LVD appeared to reduce whole blood viscosity when measured at its observed hematocrit. But when the blood was reconstituted to fixed hematocrit, the diminution in viscosity was not apparent. Contrarywise, there was increased viscosity in whole blood as well as 48% hematocrit blood of the majority of patients in shock. Moreover, viscosities of whole blood as well as 48% hematocrit blood from patients in shock significantly decreased after LVD infusion. The latter changes were not attributable to hematocrit changes. These findings suggest that there are blood viscosity alterations in patients in shock which may be corrected in part by LVD administration.

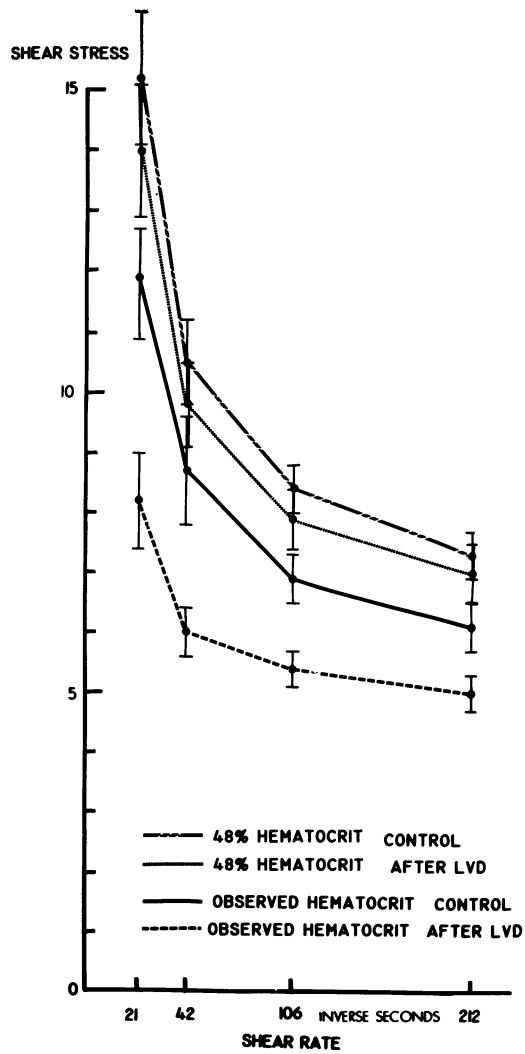

FIG. 6. Response of whole blood viscosity at both observed and 48% hematocrit to low viscosity dextran administration in shock patients. As indicated by the lower 2 lines, whole blood viscosity decreased markedly at all shear rates after low viscosity dextran. As indicated by the upper 2 lines, the same blood reconstituted to a fixed hematocrit showed decreases which were less marked.

Summary

Plasma and whole blood viscosity were measured in a series of normal subjects and in a series of postoperative patients who were in a state of moderately severe shock.

Viscosities increased in blood from patients in shock especially at low shear rates.

Whole blood viscosity, when measured at the observed hematocrit, decreased after low viscosity dextran administration in both normal subjects and in patients in shock.

Because whole blood viscosity measurements are hematocrit dependent, interpretation of these observations was obscured by associated changes in hematocrits. To obviate the influence of hematocrit changes, whole blood was reconstituted to a constant hematocrit of 48%. When carefully done, manipulations of the blood necessary to obtain constant hematocrits did not appear to produce appreciable artifacts in viscosity as measured by the system which was used. Reconstitution of blood to a fixed hematocrit was carried out to differentiate the direct and specific effect of lowered viscosity from the influence of diminished hematocrit associated with plasma volume expansion.

When 48% hematocrit blood was analyzed, viscosity alterations associated with shock were more evident.

When 48% hematocrit blood was analyzed, no significant change was seen after low viscosity dextran administration to normal subjects, but significant viscosity decrements were seen with infusions to patients in shock.

Decreases of viscosity in both plasma and 48% hematocrit blood were related to the magnitude of the control viscosity measurements. The higher the initial viscosity, the greater the decrease after LVD administration.

The data are consistent with the interpretation that low viscosity dextran administration decreases whole blood viscosity under conditions of shock, but not under normal conditions. This LVD effect may occur because of a capacity to expand plasma volume and concomitantly decrease hematocrit as well as by a more direct and specific viscosity lowering capacity.

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