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## Letter to the Editors-in-Chief

# Hemorheological changes during venous stasis as result of tourniquet application

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We read with interest the paper of Rosenson and Tangney [1] on the effect of tourniquet application on plasma viscosity measurements. Hemorheological changes have been detected in various conditions in which there are variations in blood velocity and pressure. They have been demonstrated, for instance, in vascular diseases with the reduction of the blood supply to the tissue due to an obstruction or a stenosis in the arterial tree and the consequent increase in the viscosity of the blood coming from the ischemic area [2,3]. The important increase in venous blood viscosity that we have seen in these conditions was, in our opinion, dependent on relevant hemodynamic and metabolic changes in the capillary bed, with consequent variations in the rheological behavior of circulating blood cells [4].

As these hemorheological variations probably happened in microcirculation as the result of hemodynamic changes in the arterial tree, it seemed important to study if hemodynamic changes in the venous tree, capable of modifying the microcirculatory blood flow and velocity, could determine similar effects. In order to study this problem we prepared an experimental model of venous stasis in man. The results that are here presented have been already published in 1987 [5] and 1990 [6].

Venous stasis was performed in an arm we placed at heart level on a table with the subject in sitting position. We examined 10 volunteers (6 males, 4 females, mean age $56 \pm 8$). Venous stasis was obtained with a pneumatic cuff, inflated at a pressure 10 mm Hg lower than the diastolic pressure, around the arm. This model was prepared with the purpose of stopping venous blood flow only for a limited amount of time, as we saw that pressure in the veins, measured by means of a Quartz Transducer, is 5–10 mm Hg ahead of that in the cuff after no more than 30 seconds. So, after this first period, in the venous bed under stasis the blood continues to flow, even if at a higher pressure and at a lower velocity, and we collect the venous blood distally to the cuff when it leaves the microcirculatory bed.

Blood samples were withdrawn from the antecubital vein, before and after 1, 2, 3, 4, 5, 10 minutes of venous stasis and 1 minute after the release of the cuff pressure. Hematocrit (Wintrobe), Blood Viscosity (Haake Rotovisco), Whole Blood Filterability [7], pH, $pO_2$, $pCO_2$ (I.L. 1302 Gas Analyzer) were measured in the samples. Transcutaneous $pO_2$ and $pCO_2$ were measured simultaneously by means of a Kontron Tc Microgas 7640, placing the transducer on the forearm. Statistical analysis was made using Student's *t*-test for paired data.

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Table 1

| | Basal | 1 min | 2 min | 3 min | 4 min | 5 min | 10 min | Release |
|---|---|---|---|---|---|---|---|---|
| Blood viscosity shear rate 10 $s^{-1}$ (cp) | | | | | | | | |
| Mean | 8.908 | 9.732 | 9.788 | 9.707 | 10.36 | 10.716 | 10.17 | 8.505 |
| SD | 1.086 | 2.244 | 2.2S9 | 1.817 | 2.011 | 2.177 | 1.551 | 1.382 |
| "$t$" | | 1.811 | 1.823 | 2.673 | 3.463 | 3.909 | 3.241 | 2.597 |
| $p<$ | | 0.103 | 0.101 | 0.025 | 0.007 | 0.003 | 0.01 | 0.028 |
| Whole blood filterability VB (ml/min) | | | | | | | | |
| Mean | 0.7183 | 0.5896 | 0.5815 | 0.5899 | 0.5704 | 0.585 | 0.6206 | 0.7246 |
| SD | 0.1297 | 0.123 | 0.1156 | 0.1067 | 0.118 | 0.098 | 0.1099 | 0.1407 |
| "$t$" | | 9.1025 | 7.7053 | 3.559 | 4.8068 | 3.5057 | 0.1828 | 0.5503 |
| $p<$ | | 0.0001 | 0.0001 | 0.0061 | 0.0009 | 0.0066 | 0.8589 | 0.5954 |
| Hematocrit (%) | | | | | | | | |
| Mean | 41.4 | 40.7 | 40.8 | 40.3 | 41.5 | 42.3 | 41.95 | 41.7 |
| SD | 2.87 | 3.36 | 3.7 | 3.83 | 2.95 | 4.05 | 3.21 | 3.52 |
| "$t$" | | 1.9 | 0.97 | 1.63 | 0.42 | 1.44 | 2.53 | 0.63 |
| $p<$ | | 0.088 | 0.357 | 0.137 | 0.678 | 0.182 | 0.031 | 0.541 |
| pH | | | | | | | | |
| Mean | 7.347 | 7.361 | 7.349 | 7.353 | 7.349 | 7.352 | 7.34 | 7.349 |
| SD | 0.02 | 0.029 | 0.025 | 0.021 | 0.02 | 0.018 | 0.02 | 0.019 |
| "$t$" | | 2.791 | 0.615 | 1.354 | 0.457 | 1.328 | 1.235 | 0.591 |
| $p<$ | | 0.02 | 0.553 | 0.208 | 0.657 | 0.216 | 0.247 | 0.568 |
| $pO_2$ (mm Hg) | | | | | | | | |
| Mean | 39.3 | 41.1 | 37.3 | 34.5 | 32.6 | 32.5 | 27.8 | 40.3 |
| SD | 7.18 | 13.1 | 10.73 | 8.64 | 8.26 | 7.82 | 7.09 | 8.99 |
| "$t$" | | 0.781 | 1.285 | 3.582 | 4.789 | 3.631 | 6.211 | 0.88 |
| $p<$ | | 0.354 | 0.23 | 0.005 | 0.0001 | 0.005 | 0.0001 | 0.401 |
| $pCO_2$ (mm Hg) | | | | | | | | |
| Mean | 45.38 | 44.7 | 46.04 | 46.34 | 46.49 | 46.83 | 48.14 | 44.89 |
| SD | 2.16 | 2.71 | 3.36 | 3.56 | 3.25 | 3.37 | 4 | 3.33 |
| "$t$" | | 1.544 | 1.474 | 1.592 | 2.056 | 2.29 | 3.075 | 0.805 |
| $p<$ | | 0.156 | 0.174 | 0.145 | 0.069 | 0.047 | 0.013 | 0.441 |

The results are shown in the Table 1.

Blood viscosity significantly increased, reaching a peak at the 5th minute, while whole blood filterability decreased just from the first minute. These values remained practically unchanged till the 10th minute. No variations were seen in hematocrit.

Regarding the acid-base and the blood gas equilibrium no significant changes were detected in pH, even if there was a slight tendency to reduction. At the same time we observed a gradual slight decrease of $pO_2$ and a parallel gradual increase of $pCO_2$. The same behavior was shown in $TcpO_2$ and in $TcpCO_2$, as can be seen in the Fig. 1.

All the variations we have observed disappeared after the release of the pressure in the cuff.

Our observations demonstrated that, during venous stasis, provoked with an hindrance in the venous outflow, blood fluidity significantly decreased; so we have one more demonstration that blood viscosity can be modified if we act on the blood flow, not only in the arterial but also in venous tree; beside, we

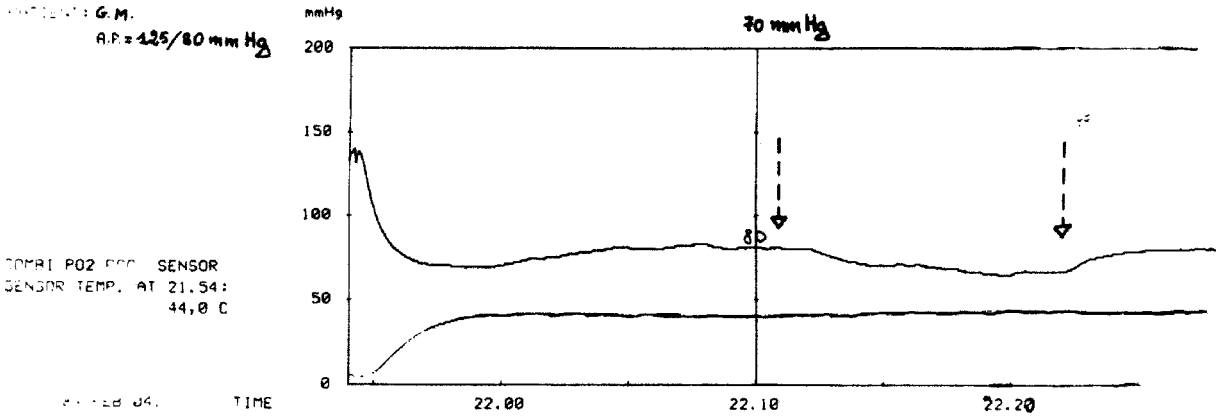


Fig. 1. Example of a measurement of transcutaneous $pO_2$ (upper tracing) and $pCO_2$ (lower tracing) during venous stasis.

have another simple experimental model, in man, where we can study these important viscosity changes, with the purpose of detecting which viscosity factor (in blood cells, or in endothelium, or in plasma) is affected. We believe that, in microcirculation under stasis, something occurred capable of modifying the rheological behaviour of blood; we gave a similar interpretation to the viscosity changes of the ischemic states of arterial vascular diseases [8]. But, while in arterial vascular diseases the viscosity variations of ischemia are accompanied by important hypoxia and lactic acidosis, during venous stasis the blood and the transcutaneous gas variations (that have the same behaviour, demonstrating that the gas changes in venous blood reflect what happened in the tissues) are very small, even if there is a slight tendency to hypoxia.

In our opinion, differently from ischemia, venous stasis due to the tourniquet application provokes deep modification, more than in the $O_2$ transport, in the interrelationship between blood cells (red, white and platelets), endothelium and plasmatic factors of coagulation and of fibrinolysis. The rheological changes that we see in venous blood reflect what happens in microcirculation and must be considered during samples acquisition, as stated by Rosenson.

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