

# ANALYSIS OF THE MECHANISM OF DISTURBANCES OF THE CARDIOVASCULAR DYNAMICS IN HEMODIALYSIS

V. S. Sal'manovich, A. A. Triakashnyi,  
É. V. Tyurina, S. S. Mogoras,  
and G. P. Kulakov

UDC 616.61-78-07:/612.13+612.172.1

Experiments on healthy dogs showed that hemodialysis, while producing no substantial changes in the plasma electrolyte levels, nevertheless led to considerable changes in the cardiovascular hemodynamics. The principal change was a decrease in the circulating blood volume.

In patients undergoing treatment by hemodialysis, this procedure is often accompanied by lowering of the arterial pressure, tachycardia, extrasystoles, and dyspnea [2, 3]. In a previous report [4] the writers showed that these disturbances may result from the action of the hemodialysis procedure itself on the circulatory system.

In the present investigation the effect of hemodialysis was studied on the total oxygen consumption, blood oxygen saturation, cardiac output, arterial and venous pressure, the total peripheral resistance, and the circulating blood volume.

## EXPERIMENTAL METHOD

Experiments were carried out on 19 healthy dogs weighing 19-26 kg anesthetized with morphine and nembutal (30 mg/kg body weight). Fourteen of the animals were treated by hemodialysis and the other five acted as the control.

The artificial kidney designed by the Research Institute of Experimental Surgical Apparatus and Instruments was used for hemodialysis. The dialyzer was filled with dextran. The dialyzing solution contained (in g/liter): NaCl 6.3,  $\text{CaCl}_2 \cdot 2\text{H}_2\text{O}$  0.3, KCl 0.27,  $\text{MgCl}_2 \cdot 6\text{H}_2\text{O}$  0.2,  $\text{NaHCO}_3$  2.65, and glucose 2.0. The animal was connected to the apparatus by means of catheters inserted into the inferior vena cava. The volume velocity of the blood flow in the apparatus was constant at 75 or 100 ml/min.

The minute volume was determined by Fick's method, and the stroke volume of the heart, the external work of the myocardium, and the total peripheral resistance were calculated from the results obtained. The mean pressure was calculated from the aortic pressure curves recorded by an electromanometer on a type N-700 loop oscillograph. The pressure in the orifice of the superior vena cava or in the right atrium was measured by a water manometer.

The circulating blood volume (CBV) was determined in five dogs by the Evans' blue dilution method before the procedure and twice or three times during the first hour of hemodialysis.

---

Laboratory of Clinical Physiology, Institute of Normal and Pathological Physiology, Academy of Medical Sciences of the USSR, Moscow. Laboratory of Experimental Physiology of Resuscitation, Academy of Medical Sciences of the USSR, Moscow. Artificial Kidney Unit, S. P. Botkin Hospital, Moscow. (Presented by Academician P. K. Anokhin.) Translated from *Byulleten' Éksperimental'noi Biologii i Meditsiny*, Vol. 72, No. 8, pp. 11-14, August, 1971. Original article submitted January 29, 1971.

©1972 Consultants Bureau, a division of Plenum Publishing Corporation, 227 West 17th Street, New York, N. Y. 10011. All rights reserved. This article cannot be reproduced for any purpose whatsoever without permission of the publisher. A copy of this article is available from the publisher for \$15.00.

TABLE 1. Plasma Potassium and Sodium Concentrations of Healthy Dogs Undergoing Hemodialysis

	Statistical data	Potassium (in meq/liter)				Sodium (in meq/liter)			
		initial data	hemodialysis			initial data	hemodialysis		
			1h	2-2½ h	3h		1h	2-2½ h	3 h
Control	$M \pm \sigma$	3.5 0.30	—	—	3.8 0.30	147.6 3.26	—	—	147.2 3.75
Hemodialysis	$M \pm \sigma$	3.3 0.32	3.4 0.35	3.4 0.22	—	146.0 6.65	145.6 5.28	145.5 5.20	—
			Dialysis				Dialysis		

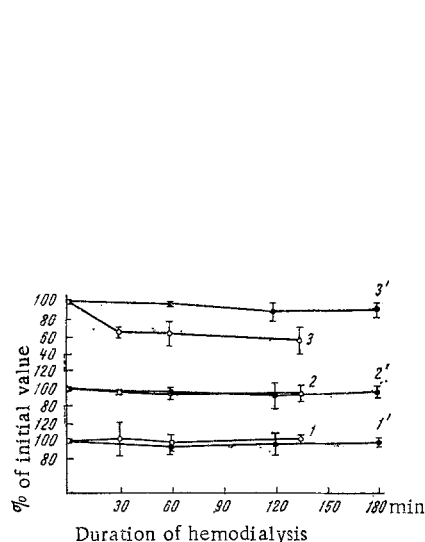


Fig. 1

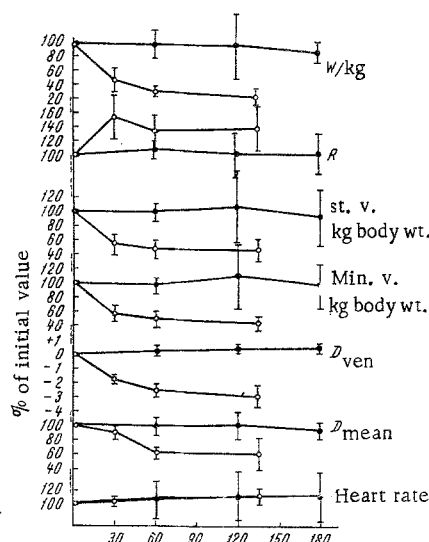


Fig. 2

Fig. 1. Dynamics of total oxygen demand (in ml/kg body weight, 1 and 1') and oxygen saturation of arterial (2 and 2') and venous (3 and 3') blood during hemodialysis (percentages of initial level). 1, 2, 3) Control group; 1', 2', 3') group with hemodialysis. Vertical lines show confidence interval for a 95% significant level.

Fig. 2. Changes in hemodynamic indices in dogs during hemodialysis. Dmean — mean pressure in aorta, Dven — central venous pressure, st.v./kg body weight — systolic volume per kg body weight; min.v./kg body weight — minute volume per kg body weight; R — total peripheral resistance; W/kg — external work of myocardium of left ventricle per kg body weight. Filled circles show control; empty circles show animals undergoing hemodialysis.

All the experiments were carried out while the heart contracted at an artificially imposed rhythm. Blood samples were taken periodically from the animals, and the potassium and sodium concentration in the plasma were determined by means of a Zeiss flame photometer.

#### EXPERIMENTAL RESULTS AND DISCUSSION

The oxygen saturation of the arterial blood remains unchanged during hemodialysis and reproduced the dynamics of this parameter in the control experiment (Fig. 1). The oxygen concentration in the venous blood, on the other hand, fell significantly during the first 30 min, and continued to fall subsequently during dialysis. The total oxygen demand did not differ by a statistically significant degree from its demand in the control animals, although during hemodialysis a tendency for the oxygen demand to be increased could be detected.

The minute volume fell considerably during the first 30–60 min of hemodialysis (Fig. 2), after which it remained essentially unchanged. The magnitude and dynamics of the decrease in systolic volume were very close to those for the minute volume of the heart.

In some experiments the heart rate was increased. This effect occurred 15–30 min after the beginning of hemodialysis and as a result of it, the heart rate was no longer bound to the original frequency of stimulation, so that this had to be increased still further. The mean increase in heart rate after 30 min of hemodialysis was 4% and after 90 min 10%. If these values were compared with the decrease in volume during the same times (46 and 58% of the initial levels respectively), it can be concluded that the increase in heart rate during hemodialysis does not compensate for the changes developing in the oxygen debt of the heart, and they cannot therefore be regarded as an effective mechanism for maintaining the minute volumes required. It must therefore be agreed with Rushmer [6] that the principal factors maintaining the minute volume of the heart are those which control the systolic output of the heart.

Together with the changes described above, hemodialysis also led to a marked decrease in the mean pressure in the aorta (Fig. 2). One of the main causes of this decrease is considered to be a decrease in the systolic output of the heart since the action of other factors participating in the maintenance of the mean pressure was directed during hemodialysis toward compensation of the changes arising. These factors include the tachycardia developing during hemodialysis, and also the increase in the total peripheral resistance (Fig. 2). The increase in peripheral resistance was more marked during the first 30 min of hemodialysis, after which it fell slightly and was maintained at a steady level throughout the rest of the procedure.

The decrease in mean systolic pressure and in the systolic output of the heart was accompanied by a decrease in the external work of the myocardium, so that after 1.5 h it had fallen to 26% of its initial level.

Besides changes in the arterial part of the circulatory system, hemodialysis also produced disturbances in the venous blood flow. The central venous pressure fell considerably during hemodialysis, and the character of its changes was similar to that of the minute and systolic volumes of the heart (Fig. 2).

The concentrations of potassium and sodium in the plasma at all stages of hemodialysis were virtually indistinguishable from their original values and they corresponded on the whole to their concentrations in the plasma of the control animals (Table 1). It can accordingly be concluded that hemodialysis itself, unaccompanied by changes in the electrolyte composition of the blood, produces profound disturbances of the circulatory system.

Analysis of the character of the circulatory disturbances during hemodialysis suggested that they may be due to a decrease in CBV. Direct determination of the CBV showed that hemodialysis is in fact accompanied by a decrease in this parameter. The initial CBV was  $81 \pm 12.1$  ml/kg, and after 1 h of hemodialysis it fell to  $18 \pm 3.9$  ml/kg ( $21 \pm 6.2\%$  of the initial level). A tendency for the CBV to fall could be detected during the first 5–10 min of hemodialysis, and it increased progressively during the first hour of the procedure.

The results indicating a decrease in the CBV during hemodialysis correlate well with the dynamic of the early decrease in central venous pressure [4]. They are also confirmed by the observation that the veins are emptied during hemodialysis, as is shown by the effect of suction of the catheter collecting the blood against the wall of the vein. From this point of view the results of this investigation provide a ready explanation of the morphological findings obtained by Gol'dina and Gorbovitskii [1] on dogs undergoing hemodialysis. These workers found hyperemia of the liver, lungs, and spleen – the blood depots of the body – after hemodialysis. A decrease in the circulating blood mass after treatment of patients with renal failure by hemodialysis was also observed by Braun et al. [5].

The decrease in CBV during hemodialysis observed in the present experiments was undoubtedly one of the chief reasons for the disturbances of the hemodynamics during this procedure. A decrease in CBV leads to a decrease in venous pressure, and through a decrease in the venous return to the heart it also decreases the volume. This disturbance, in turn, gives rise to a decrease in arterial pressure, reduces the work of the myocardium, and causes the development of an insufficient blood supply to the tissues, which, in the present experiment, was expressed as a decrease in the oxygen saturation of the venous blood.

#### LITERATURE CITED

1. B. G. Gol'dina and E. B. Gorbovitskii, Arkh. Pat., No. 2, 70 (1962).
2. A. Ya. Pytel', S. D. Goligorskii, M. D. Dzhavad-Zade, et al., The Artificial Kidney and Its Clinical Application [in Russian], Moscow (1961).
3. N. N. Skachilova and V. A. Agranenko, Ter. Arkh., No. 7, 73 (1963).
4. A. A. Trikashnyi, V. S. Sal'manovich, S. S. Mogoras, and G. P. Kulakov, Kardiologiya, No. 5, 94 (1970).
5. L. Braun, P. H. Krasemann, and H. J. Michalke, Z. Kreisl.-Forsch., 57, 1190 (1968).
6. R. F. Rushmer, Cardiovascular Dynamics, Philadelphia (1961).