PATHOLOGICAL PHYSIOLOGY AND GENERAL PATHOLOGY

DYNAMICS OF KIDNEY FUNCTION AFTER CLINICAL DEATH

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Several descriptions have been given of the functional state of the kidneys in dogs surviving clinical death. In such animals, a decrease in the renal blood flow [5] and the glomerular filtration and an increase in the tubular reabsorption of water [9] have been described. On the whole, however, insufficient attention has been paid in the literature to the state of the kidney function in dogs surviving clinical death.

At Professor V. A. Negovskii's suggestion, an experimental investigation of the kidney function in the recovery period after resuscitation has been carried out.

EXPERIMENTAL METHOD

Experiments were carried out on four female dogs weighing 16-22 kg. The ureters of the animals were first exteriorized by L. A. Orbeli's method [7] as modified by Sh. S. Kiguradze [4]. Clinical death was produced by exsanguination and it lasted for 5 min. Resuscitation was carried out by a combined method developed by Professor V. A. Negovskii and co-workers [6]. The kidney function was investigated in the original state and 2, 5, 10, 15, 20, 30, and 45 days after resuscitation. The following indices of kidney function were studied by methods developed by Smith and co-workers [11]: a) the effective renal plasma flow (EPF) in relation to diodone, b) the glomerular filtration (F) in relation to inulin, c) the maximal secretion (MS) in relation to diodone, and d) the urea clearance (C_u). The inulin in the plasma and urine was determined by Harrison's method [12] as modified by N. I. Ivanov [3]; diodone in the plasma and urine was estimated by the method of White and Rolf [16] as modified by Bak and co-workers [10]. The urea in the plasma and urine was determined by Borodin's method in a Kovarskii's apparatus. The percentage of water reabsorbed in the kidney tubules was calculated. In three dogs in which the left carotid artery was exteriorized subcutaneously, the arterial pressure was measured during the investigation of the kidney function. The animals received a constant diet. One hour before determination of the kidney function, a mixture of water and milk was given to the dogs internally in a dose of 60 ml/kg body weight. All the indices were expressed per square meter of body surface of the animal.

EXPERIMENTAL RESULTS

The following results were obtained for the indices characterizing the kidney function in the initial state: EPF 418-442 ml/min, F 97-197 ml/min, C_u 66-108 ml/min, MS 23.7-46 mg/min, reabsorption of water (P_{H_2O}) 90.5-96.8%. After determination of the initial indices, the resuscitation experiments were carried out, in the course of which the cardiac activity, the respiration, and the corneal reflexes were quickly restored (Table 1).

Investigation of the kidney function began 1-2 days after resuscitation, i.e., at the time of recovery of hearing, sight, and posture.

It is clear from Table 2 that the value of EPF fell 1-2 days after resuscitation. Later, on the 5th-20th day, the EPF rose to a level much higher than initially. Not until after the 20th day of the recovery period did the EPF fall again below the initial level, where it remained until the 45th day of the investigation. The changes in F and C_u were similar in character to the changes in EPF, but differed considerably in magnitude. For example, the decrease in the excretion of urea and in the glomerular filtration on the first-second days after resuscitation was much less than the decrease in EPF. The increase in these functions from the 5th-10th and until the 20th days of the recovery period was much greater than the increase in EPF.

The changes in P_{H_2O} also showed special features. Although a general tendency towards an increase in the reabsorption of water was present from the first until the 30th day, by the 45th day of the investigation fluctuations tending towards a decrease could be observed. So far as the MS is concerned, no strict relationship could be observed

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TABLE 1. Duration of Restoration of Several Functions Following Clinical Death for Different Periods

Experiment	Duration of clinical death	Duration of restoration of functions		
		Cardiac activity	Respiration	Corneal reflexes
1	5 min 10 sec	47 sec	3 min 37 sec	10 min 10 sec
2	7 "	45 "	3 " 40 "	13 "
3	6 " 15 sec	40 "	3 " 55 "	11 " 15 sec
4	13 "	30 "	5 "	12 " 20 "

TABLE 2. Dynamics of Kidney Functions after Clinical Death Lasting 5 min

Experiment No.	Kidney function	Initial state	Days after resuscitation						
			1—2	5	10	15	20	30	45
1	F (in ml/min)	142	137	265	240	160	143	122	124
	EPF (in ml/min)	438	246	348	552	560	570	340	350
	C_u (in ml/min)	66	40,5	79	89	80	65,0	68	65
	P_{H_2O} (in %)	96,8	97	98	97,5	97	96,5	97,5	96,5
	MS (in mg/min)	23,7	25,4	22	15,4	18	23,5	18	19,2
2	F (in ml/min)	112	58	145	190	200	200	90,5	89,5
	EPF (in ml/min)	425	148	217	282	398	622	282	299
	C_u (in ml/min)	108	94	117	135	144	133	111	102
	P_{H_2O} (in %)	94,5	90	93	95	96,4	96,4	92	92
	MS (in mg/min)	34	47	30	25,5	21,5	21	25,5	25
3	F(in ml/min)	197	163	151	206	202	134	174	187
	EPF (in ml/min)	442	185	558	501	440	375	333	329
	C_u (in ml/min)	80	50	87	89	105	112	85	48
	P_{H_2O} (in %)	96,6	98,2	90,8	95	94,2	91,5	96,5	95,2
	MS (in mg/min)	38	25	50,8	46	39	47	27	28,8
4	F (in ml/min)	97	—	126	173	159	271	146	107
	EPF (in ml/min)	418	—	271	346	529	200	246	252
	C_u (in ml/min)	88	—	61	71	80	103	80	82
	P_{H_2O} (in %)	90,5	—	95	96,4	94	97,3	95	93
	MS (in mg/min)	46	—	44	42	50	39	35	36

between its changes from the first until the 20th days of the recovery period. The MS could be either higher or lower than the original level irrespective of the changes in the renal blood flow. In the later stages of the recovery period, starting with the 20th day, the MS fell considerably below its initial level and remained at these values until the end of the investigation (45th day).

The results of this investigation demonstrate considerable disturbances in the renal activity of animals surviving clinical death. It might be expected that one reason for the depression of renal activity in the resuscitated animal would be a decrease in the general blood pressure. However, systematic measurements of the blood pressure in these experiments showed that its level was the same both in the initial state and in the recovery period after resuscitation. Since clinical death is one form of deep hypoxia, it may be supposed that the mechanisms of the disturbance of renal activity after clinical death are like the mechanisms of kidney damage in other severe forms of hypoxia. Probably one of the reasons for the depression of kidney function in the state described above was a protective reaction of the body consisting of a redistribution of the blood during clinical death to the organs more sensitive to hypoxia and more important for the vital activity of the organism, i.e., to the brain and heart [6,15].

The response reaction of the kidney to severe oligemia is known to be vasoconstriction of the organ, leading to depression of its function [2,14]. It may also be assumed that the disturbances of kidney activity between the

first and 45th days of the investigation were based on a marked disturbance of the regulatory mechanisms of urine secretion, including such important factors as the osmotic pressure and the composition of the blood and the hormones controlling the transport of water and sodium (mineralocorticoids, antidiuretic hormone). So far as the disturbances actually in the glomerular and tubular portions of the renal nephron are concerned, an increase in the permeability of the glomerular membrane and depression of the processes of reabsorption in the renal tubules may be postulated. The fact that the inulin and urea clearances were reduced during the first days after resuscitation much less than the diodone clearance confirms this hypothesis concerning the disturbance of the reabsorption function of the kidneys. In normal conditions, inulin and urea, filtered through the glomeruli, are known to be partially reabsorbed into the blood stream in the renal tubules.

Since these substances are excreted by the kidneys to a greater degree than diodone, which normally is not reabsorbed into the blood stream in the renal tubules, a decrease in the reabsorption of these substances in the renal tubules may be postulated. Evidence in favor of this was given by the greater increase in the excretion of these substances later in the recovery period.

Finally, another probable cause of the kidney disturbances after resuscitation was the development of degenerative changes in the kidneys, leading to impairment of the nutrition of the glomeruli of the nephrons [1,8,13]. It may be concluded from the results described above that in the recovery period after clinical death the functional capacity of the kidneys is considerably reduced. The treatment of an animal or a patient surviving clinical death must therefore follow the same lines as the treatment of renal failure.

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