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EFFECT OF CONTROLLED HYPOTENSION WITH PENDIOMID (AS USED IN SURGERY) ON RENAL HEMODYNAMICS AND WATER AND ELECTROLYTE EXCRETION— A COMPARISON WITH HEXAMETHONIUM AND ARFONAD AND THE EFFECT OF NOREPINEPHRINE ON THESE RESPONSES * † ‡

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SINCE controlled hypotension is being used more widely for maintaining hemostasis during surgery, an evaluation of the renal hemodynamic response to the agents used for this procedure is indicated. The purpose of the authors in writing the present report was to study the renal hemodynamic response to hypotension produced by a continuous infusion of pendiomid (pentamethyl-diethyl-3-aza-pentylene-1,5-diammonium dibromide) and to compare these observations with observations made on a similar group of patients to whom hexamethonium or arfonad (*d*-3,4-(1',3'-dibenzyl-2'-keto-imidazolido)-1,2-trimethylene thiophanium *d*-camphor sulfonate) was administered by the same method. Additional observations were made on water excretion, electrolyte excretion, and the effect of blood pressure elevation with an infusion of norepinephrine.

METHODS

Observations on renal function and on water and electrolyte excretion were made on 9 normotensive subjects whose ages ranged from 17 to 52 years. None of them had a history of renal disease or hypertension. One of the patients had a mean blood pressure of 113 mm. of mercury which is slightly above normal. However, this was probably a result of the excitement associated with having the renal function tests performed. Glomerular filtration rate was measured with inulin and renal plasma flow was estimated by the clearance of para-aminohippurate. Each patient served as his own control and the values were not connected to body surface area. Mean blood

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pressure was measured by direct intra-arterial manometry. Arterial blood which was collected through a manifold was used for the chemical analyses. Methods and techniques employed have been described previously (1, 2). Sodium and potassium excretion and the concentration of these electrolytes in the plasma were determined using a Beckman flame photometer for analyses. All observations were made in the supine position. After suitable control studies (3 successive 10 minute collection periods) were obtained, the pendiomid was administered by continuous intravenous infusion using a concentration of one to two mg. per ml. of 5 per cent glucose in distilled water. The rate of the infusion depended on the blood pressure response. The initial blood pressure reduction was usually more marked than that observed after hemodynamic stabilization. The initial response usually was observed after 5 to 10 mg. of pendiomid was administered. After hemodynamic stabilization a "floor" in the blood pressure was obtained, beyond which it was difficult to lower the pressure. Only with a very rapid infusion was it possible to obtain an additional depression in blood pressure and this usually was limited to a reduction of 10 to 20 mm. Hg in mean pressure. After the initial induction period, the drug was usually administered at the rate of 2 to 6 mg. per minute. The maximum amount of pendiomid administered was 1,000 mg. over a four hour period. The average was 250 mg. over a three hour period. Observations on renal function were made for successive 10 minute periods during the first hour of hypotension and 2 ten minute periods were collected one hour later.

Following two hours of continuous hypotension which was maintained with pendiomid, norepinephrine was administered by continuous intravenous infusion to 6 of the 9 patients. The infusion rate of norepinephrine was adjusted so as to return the blood pressure to control levels. The infusion of pendiomid was continued at the same rate as during the period of hypotension in order to maintain the same degree of ganglionic blockade. After the blood pressure was stabilized at the control level with norepinephrine, 3 successive ten minute collection periods were obtained. Glomerular filtration rate, renal blood flow, and the excretion rates of water and electrolytes were determined similarly to the observations which were obtained during the hypotensive period.

RESULTS

All but one of the patients (No. 8, C. G.) obtained a significant reduction in arterial blood pressure. The maximum reduction in pressure for the group occurred after two hours of pendiomid infusion ($p < 0.01$), which was due to a more rapid rate of infusion at that time. The initial reduction in blood pressure frequently was associated with a sharp depression in renal blood flow and an increase in

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TABLE 1a

RENAL HEMODYNAMIC RESPONSE TO BLOOD PRESSURE REDUCTION WITH PENDIOMID

Patient	Mean Blood Pressure (mm. Hg)					Renal Blood Flow (ml. min.)[†]					Renal Vascular Resistance*					Hematocrit					Pulse Rate				
	C	D_1	D_2	D_3	D_4	C	D_1	D_2	D_3	D_4	C	D_1	D_2	D_3	D_4	C	D_1	D_2	D_3	D_4	C	D_1	D_2	D_3	D_4
1. L. B.	87	82	79	76	71	1,113	929	1,145	1,109	1,563	0.08	0.09	0.07	0.07	0.05	31	31	34	34	35	80	92	—	120	120
2. B. L.	113	73	58	57	60	797	414	452	657	596	0.14	0.18	0.13	0.09	0.10	35	35	35	35	30	88	96	80	80	82
3. T. M.	81	72	69	66	59	1,445	1,449	1,281	1,536	1,429	0.06	0.05	0.05	0.04	0.04	45	45	41	41	38	88	84	82	84	86
4. R. H.	107	84	75	80	—	1,255	875	1,277	1,248	—	0.03	0.10	0.06	0.06	—	42	40	40	40	—	64	84	84	88	88
5. P. R.	88	68	70	78	69	1,786	195	1,168	1,064	1,190	0.05	0.35	0.06	0.07	0.06	41	40	37	37	38	88	88	84	88	75
6. J. K.	84	80	79	79	69	1,095	1,112	1,038	1,089	1,013	0.08	0.07	0.08	0.07	0.07	35	35	40	38	38	92	94	100	100	102
7. G. W.	102	87	84	82	66	2,188	2,912	2,326	2,212	2,064	0.05	0.03	0.04	0.04	0.03	43	43	42	42	42	68	68	80	80	76
8. C. G.	104	103	111	103	102	895	1,106	1,173	945	712	0.12	0.09	0.09	0.11	0.14	40	49	49	45	48	63	60	96	94	94
9. A. W.	105	94	94	70	69	1,205	1,053	1,013	926	1,028	0.09	0.09	0.09	0.08	0.07	39	38	38	38	35	67	98	92	90	92
Mean	97	83	80	77	71	1,309	1,116	1,208	1,198	1,199	0.08	0.12	0.07	0.07	0.07	39	40	40	39	38	78	85	87	92	90
Per cent of control		86	82	79	73		85	92	92	92		150	88	88	88		103	103	100	97		109	112	118	115
P value $< \neq$		0.01	0.05	0.01	0.01		NS	0.30	0.30	0.30		NS	0.10	0.20	0.20		NS	NS	NS	NS		.20	.20	.10	.10

C—Control.

D_1—First 10-minute period after pendiomid.

D_2—Average of second and third 10-minute periods after pendiomid.

D_3—1 hour after pendiomid (average of 2-10 minute periods).

D_4—2 hours after pendiomid (average of 2-10 minute periods).

†—Renal blood flow $= \frac{\text{Renal plasma flow}}{\text{1-hematocrit}}$.

*—Renal vascular resistance $= \frac{\text{Mean blood pressure}}{\text{Renal blood flow}}$.

NS—P value > 0.30—only P values of less than 0.30 are recorded.

$\neq -t = t\sqrt{\frac{n(n-1)}{S^2}}$.

renal vasoconstriction.This response was not consistent enough to be statistically significant ($p < 0.30$) for the entire group of patients. When this response occurred, it probably was due to blockade of the sympathetic ganglia to other areas of the body which was more complete than it was to the renal vessels. As a result, when the blood pressure decreased a relative renal vasoconstriction followed. However, when such a reduction in renal blood flow occurred it usually corrected itself rather rapidly and returned to or towards the control levels within the first ten to thirty minutes after the infusion of pendiomid was started.

TABLE 1b

EFFECT OF BLOOD PRESSURE REDUCTION (WITH PENDIOMID) ON GLOMERULAR FILTRATION RATE AND RENAL PLASMA FLOW

Patient	Glomerular Filtration Rate (ml./min.)					Renal Plasma Flow (ml./min.)					Filtration Fraction*				
	C	D_1	D_2	D_3	D_4	C	D_1	D_2	D_3	D_4	C	D_1	D_2	D_3	D_4
1. L. B.	112	117	123	129	137	768	641	756	732	1,016	0.15	0.18	0.16	0.18	0.13
2. B. L.	82	46	56	57	67	518	269	294	427	417	0.16	0.17	0.19	0.13	0.16
3. T. M.	120	109	110	109	120	795	797	756	906	886	0.15	0.14	0.15	0.12	0.14
4. R. H.	95	71	85	69	—	728	525	766	749	—	0.13	0.14	0.11	0.09	—
5. P. R.	214	63	117	101	139	1,054	117	736	670	738	0.20	0.54	0.16	0.15	0.19
6. J. K.	120	123	119	118	108	712	723	623	675	628	0.17	0.17	0.19	0.17	0.17
7. G. W.	220	261	202	209	194	1,247	1,660	1,349	1,283	1,197	0.18	0.16	0.15	0.16	0.16
8. C. G.	97	94	102	101	80	537	564	598	520	370	0.18	0.17	0.17	0.19	0.22
9. A. W.	146	108	133	110	76	735	653	628	574	668	0.20	0.17	0.21	0.19	0.11
Mean	134	110	116	111	115	788	661	723	726	740	0.17	0.20	0.17	0.15	0.16
Per cent of control		82	87	83	86		84	92	92	94		118	100	88	94
P value <		0.20	0.20	0.20	0.10		NS	0.20	NS	NS		NS	NS	0.20	NS

*—Filtration fraction $= \frac{\text{Glomerular filtration rate}}{\text{Renal plasma flow}}$.

C—Control.
D_1—First 10-minute period after pendiomid.
D_2—Average of 2nd and 3rd periods (10-minute) after pendiomid.
D_3—1 hour after pendiomid (average of 2–10 minute periods).
D_4—2 hours after pendiomid (average of 2–10 minute periods).
NS—$P > 0.30$—see table 1a.

As the blood pressure decreased, glomerular filtration rate was also moderately reduced (mean values reduced from 82 per cent to 87 per cent of control). There was a sharp initial depression (period D_1) in some instances which quickly increased and then stabilized at a moderately reduced level. The glomerular filtration rate in a few patients showed only a moderate reduction initially which was not altered appreciably throughout the hypotensive period. As a result of the variability in response of glomerular filtration rate for the group, the changes were not statistically significant.

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TABLE 2

EFFECT OF BLOOD PRESSURE REDUCTION WITH PENDIOMID ON WATER AND ELECTROLYTE EXCRETION

Patient	Urine Volume (ml./min.)					Plasma Sodium (mEq./L.)				Plasma Potassium (mEq./L.)				Sodium Excretion (mEq./min.)					Potassium Excretion (mEq./min.)				
	C	D_1	D_2	D_3	D_4	C	D_1	D_3	D_4	C	D_2	D_3	D_4	C	D_1	D_2	D_3	D_4	C	D_1	D_2	D_3	D_4
1. L. B.	4.4	0.2	1.1	0.8	0.7	131	131	131	131	3.71	3.69	3.61	3.60	109	103	99	44	46	30	21	18	16	35
2. B. L.	11.2	4.2	1.2	0.5	0.8	148	148	152	152	4.30	4.30	4.07	4.07	206	78	24	20	26	45	24	49	70	75
3. T. M.	1.4	1.9	2.6	2.4	0.7	133	133	133	130	4.72	4.72	4.72	3.69	117	78	60	82	15	77	81	69	55	51
4. R. H.	9.4	1.8	0.7	0.5	—	143	139	139	—	3.84	3.84	4.25	—	115	72	86	67	—	24	13	29	29	—
5. P. R.	16.2	3.0	2.1	1.4	4.1	145	144	144	140	4.75	4.17	4.17	4.07	69	41	88	73	85	45	19	58	52	86
6. J. K.	14.0	15.5	13.0	0.9	0.5	144	144	144	139	4.51	4.51	4.51	3.94	89	81	77	138	94	29	32	33	40	31
7. G. W.	1.1	1.8	1.4	0.7	0.2	130	131	126	129	4.56	4.22	3.84	4.01	203	338	149	112	91	69	10	71	55	86
8. C. G.	11.1	9.9	8.7	2.7	6.0	128	128	128	128	4.72	3.84	4.02	4.12	309	335	376	253	138	23	21	19	25	19
9. A. W.	15.3	6.0	5.8	1.7	1.6	131	131	137	131	4.30	4.30	3.84	3.79	242	188	156	66	48	45	30	26	17	40
Mean	9.3	4.9	4.1	1.3	1.8	137	137	137	135	4.38	4.18	4.11	3.91	162	146	124	95	68	43	28	41	40	53
Per cent of control		53	44	14	19		100	100	99		95	94	89		90	77	59	42		65	95	93	123
P value <		0.05	0.01	0.01	0.01		NS	NS	NS		0.10	0.10	0.01		NS	0.20	0.02	0.01		0.05	NS	NS	NS

C—Control.
D_1—First 10-minute period after pendiomid.
D_2—Average of second and third 10-minute periods after pendiomid.
D_3—1 hour after pendiomid (average of 2–10 minute periods).
D_4—2 hours after pendiomid (average of 2–10 minute periods).
NS—P > 0.30—see table 1a.

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TABLE 3

EFFECT OF NOREPINEPHRINE (ON RENAL HEMODYNAMICS) ADMINISTERED DURING HYPOTENSION PRODUCED BY PENDIOMID

Patient	Glomerular Filtration Rate (ml./min.)				Renal Plasma Flow (ml./min.)				Mean Blood Pressure (min./Hg)				Renal Vascular Resistance				Renal Blood Flow (ml./min.)				Filtration Fraction				Hematocrit			
	C	D_4	NE_1	NE_2	C	D_4	NE_1	NE_2	C	D_4	NE_1	NE_2	C	D_4	NE_1	NE_2	C	D_4	NE_1	NE_2	C	D_4	NE_1	NE_2	C	D_4	NE_1	NE_2
1. L. B.	112	137	140	145	708	1,016	1,479	1,006	87	71	88	87	0.08	0.05	0.04	0.06	1,113	1,563	2,207	1,501	0.15	0.13	0.09	0.14	31	35	33	33
2. B. L.	82	67	89	68	518	417	516	431	113	60	114	130	0.14	0.10	0.15	0.21	797	596	748	625	0.16	0.16	0.17	0.16	35	30	31	31
3. T. M.	120	120	128	124	793	886	757	728	81	59	91	93	0.06	0.04	0.07	0.08	1,445	1,429	1,253	1,234	0.15	0.14	0.17	0.17	45	38	41	41
5. P. R.	214	139	145	151	1,054	738	1,073	862	88	59	102	107	0.05	0.06	0.06	0.08	1,786	1,190	1,759	1,413	0.20	0.19	0.14	0.18	41	38	39	39
6. J. H.	120	108	104	123	712	628	687	683	84	69	89	113	0.08	0.07	0.08	0.10	1,095	1,013	1,090	1,084	0.17	0.17	0.15	0.18	35	38	37	37
7. G. W.	220	194	254	205	1,247	1,197	1,123	1,054	102	66	112	107	0.05	0.03	0.05	0.05	2,188	2,064	2,119	1,989	0.18	0.16	0.23	0.19	43	42	47	47
Mean	145	128	144	136	849	814	939	794	93	66	99	106	0.08	0.06	0.08	0.10	1,404	1,309	1,534	1,309	0.17	0.16	0.16	0.17	38	37	38	38
Per cent of control		88	99	94		96	111	94		71	106	114		75	100	125		93	109	93		94	94	100		97	100	100
P value <		0.30	NS	NS		NS	NS	0.10		0.01	0.05	0.05		0.30	NS	0.20		NS	NS	NS		0.05	NS	NS		NS	NS	NS
P_1 value <†			0.20	0.01			0.30	NS			0.01	0.01			0.10	0.05			0.20	NS			NS	0.20			NS	NS

C—Control.
D_4—Two hours after blood pressure reduction by continuous infusion of pendiomid.
NE_1—First 10-minute period after blood pressure elevation with norepinephrine.
NE_2—Average of second and third 10-minute periods after blood pressure elevation with norepinephrine.
*—Renal vascular resistance $= \frac{\text{Mean blood pressure}}{\text{Renal blood flow}}$.
NS—P > 0.30—see table 1a.
†—P_1 = Period D_4 used for comparison with NE_1 and NE_2.

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Associated with the blood pressure reduction and depression in glomerular filtration rate, there was a significant initial ($p < 0.05$) and sustained ($p < 0.01$) depression in water excretion. The reduction in water excretion was more marked after one hour than it was during the ten to thirty minute period of hypotension and this depression usually did not increase by the two hour period. There was a parallel reduction in sodium excretion which was more marked after two hours of hypotension ($p < 0.01$) than at any other time during the study. There was a similar statistically significant ($p < 0.05$) initial reduction in potassium excretion but this returned to the control level after one hour and even exceeded the mean control value by the second hour of hypotension.

When the blood pressure was increased to control levels with norepinephrine, the glomerular filtration rate also increased approximately to the control levels in all but one instance (Patient No. 5, P. R.). If the renal blood flow was depressed during the hypotensive period, it also increased when norepinehrine was infused. This occurred despite the continuous infusion of pendiomid. When administered to normotensive subjects, norepinephrine is actually a vasoconstrictor (2). Therefore, this response of the hypotensive subject to norepinephrine indicates that the reduction on glomerular filtration rate during the infusion of pendiomid is a result of the hypotension rather than being related to a specific renal vascular effect of pendiomid.

As the blood pressure and the glomerular filtration rate increased with norepinephrine, there was only a slight increase in water ex-

TABLE 4

EFFECT OF BLOOD PRESSURE ELEVATION WITH NOREPINEPHRINE FOLLOWING PREVIOUS REDUCTION WITH PENDIOMID ON ELECTROLYTE AND WATER EXCRETION

Patient	Urine Volume (ml./min.)				Plasma Sodium (mEq./L.)				Plasma Potassium (mEq./L.)				Sodium Excretion (mEq./min.)				Potassium Excretion (mEq./min.)			
	C	D_4	NE_1	NE_2	C	D_4	NE_1	NE_2	C	D_4	NE_1	NE_2	C	D_4	NE_1	NE_2	C	D_4	NE_1	NE_2
1. L. B.	4.4	0.7	0.8	0.8	131	131	131	131	3.71	3.60	3.40	3.40	109	46	93	118	30	35	49	41
2. B. L.	11.2	0.8	1.0	1.0	148	152	147	147	4.30	4.07	3.87	3.87	206	26	51	98	45	75	96	72
3. T. M.	1.4	0.7	1.0	0.8	133	130	131	131	4.72	3.39	3.43	3.43	117	15	68	66	77	51	87	64
5. P. R.	16.2	4.1	2.6	8.0	145	140	141	141	4.75	4.07	4.35	4.35	66	85	105	125	45	86	95	102
6. J. K.	14.0	0.5	0.4	0.6	144	139	137	137	4.51	3.94	4.00	4.00	89	94	68	94	29	31	42	45
7. G. W.	1.1	0.2	0.8	0.8	130	129	128	128	4.56	4.01	3.89	3.89	203	91	119	119	69	86	65	59
Mean	8.1	1.2	1.1	2.0	139	137	136	136	4.43	3.90	3.82	3.82	132	60	84	103	49	61	72	64
Per Cent of Control		15	14	25		99	98	98		88	86	86		45	64	78		124	147	131
P value <		0.05	0.05	0.05		0.30	0.05	0.05		0.01	0.01	0.01		0.10	0.20	0.05		0.30	0.05	0.30
P_t value†			NS	0.30			NS	NS			0.20	0.20			0.10	0.02			0.20	NS

C—Control.
D_4—Two hours after blood pressure reduction by continuous infusion of pendiomid.
NE_1—First 10-minute period after blood pressure elevation with norepinephrine.
NE_2—Average of second and third 10-minute periods after blood pressure elevation with norepinephrine.
NS—P > 0.30—see table 1a.
† See table 3 for key.

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cretion from 1.2 to 2 cc. per minute ($p < 0.30$) but a greater and significant increase in sodium excretion ($p < 0.02$). Potassium excretion which already had returned to control levels after two hours of hypotension was not altered significantly when norepinephrine was infused.

DISCUSSION

These observations indicate that during the immediate hypotensive period following the infusion of pendiomid there is probably a relative differential renal vasoconstriction in the kidney which rapidly, but only partially, compensates. Although there was some depression in the average renal blood flow for the entire group of patients studied, this was not enough to produce renal damage from ischemia. In fact, the degree of depression in renal blood flow was not as great as that which has been observed when normotensive subjects change from the supine to the upright position (3, 4).

As with the reduction in renal blood flow, the reduction in glomerular filtration rate was of little consequence except that apparently it led to a retention of sodium and water. This should be noted when one contemplates administering large volumes of fluid during surgical procedures employing controlled hypotension since it is conceivable that pulmonary edema and acute congestive heart failure are more likely to be encountered under these circumstances.

The renal hemodynamic response to pendiomid is quite similar to the response to hexamethonium. However, the renal effects with either one of these agents are not as marked as following the administration of arfonad. Only after the administration of arfonad is renal blood flow depressed enough to be statistically significant ($p < 0.01$). Apparently a mean blood pressure of 60 to 70 mm. Hg is approximately a critical level. When the blood pressure is depressed below 60 mm. Hg, as it is during the administration of arfonad, renal blood flow and glomerular filtration rate are significantly depressed.

Urine volume and sodium excretion were depressed initially and remained depressed during the infusion of all 3 of the ganglionic blocking agents. Potassium excretion was also depressed initially but returned to or towards the control levels despite a maintained reduction in blood pressure. The differences which were observed between arfonad and the other ganglionic blocking agents probably reflect a greater blood pressure reduction with the former agent rather than any specificity (ganglionic blockade or renal effects) of the drugs. This is supported by the fact that these renal effects are returned to or toward control values by returning the blood pressure to normal with an infusion of norepinephrine. If norepinephrine is administered to normotensive individuals and the blood pressure raised to hypertensive levels the opposite response is observed. Renal blood flow is depressed and glomerular filtration rate also tends to decrease.

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TABLE 5

COMPARISON OF RENAL HEMODYNAMICS RESPONSES TO PENDIOMID, HEXAMETHONIUM AND ARFONAD

	Mean Blood Pressure (mm. Hg)			Glomerular Filtration Rate (ml. min.)			Renal Blood Flow (ml./min.)			Renal Vascular Resistance			Urine Volume (ml. min.)			Sodium Excretion (mEq. min.)			Potassium Excretion (mEq./min.)			Number of Patient Studied
	C	H	2H	C	H	2H	C	H	2H	C	H	2H	C	H	2H	C	H	2H	C	H	2H	
Pendiomid	97	83	71	134	110	115	1,309	1,116	1,199	0.08	0.12	0.07	9.3	4.9	1.8	162	146	68	43	28	53	9
P Value*	—	0.01	0.01	—	0.20	0.10	—	NS	0.30	—	NS	0.20	—	0.05	0.01	—	NS	0.01	—	0.05	NS	
Arfonad	98	81	57	121	96	64	1,143	774	504	0.09	0.10	0.15	3.9	3.2	0.7	161	115	48	56	49	53	9
P Value*	—	0.02	0.01	—	0.02	0.01	—	0.02	.01	—	NS	0.30	—	NS	0.01	—	0.10	0.01	—	.05	NS	
Hexamethonium	98	68	64	113	92	93	1,108	874	1,007	0.10	0.11	0.07	3.1	1.4	0.6	105	25	21	62	40	71	9
P Value*	—	0.01	0.01	—	0.01	0.02	—	0.20	0.30	—	NS	0.10	—	0.20	0.05	—	0.01	0.01	—	0.01	NS	

C—Control.
H—Initial period of hypotension.
2H—Observations after hypotension for 2 hours or more.
*—P value recorded only when less than 0.30. When greater than this it is recorded as NS.
NS—P > 0.30.

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SUMMARY AND CONCLUSIONS

1. The renal hemodynamic response to blood pressure reduction with pendiomid infusion in normotensive surgical patients has been studied and the results compared to similar observations made on patients who received arfonad or hexamethonium. There was a slight reduction in glomerular filtration rate which was more marked during the infusion of arfonad than after pendiomid and hexamethonium. This merely reflects a greater reduction in blood pressure during the infusion of arfonad. These responses were reversed when the blood pressure was increased to control levels with norepinephrine.

2. Observations on water and electrolyte excretion made concurrently with the renal hemodynamic studies indicated an initial and sustained depression in water and sodium excretion which exceeded the degree of depression on glomerular filtration during the hypotensive period. Potassium excretion was also depressed initially but this increased to control levels despite a maintained hypotension and a depression of water and sodium excretion.

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