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Effect of Sludge Ice Cooling on Renal Function and Renal Histology in the Dog

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Summary. The effect of sludge ice surface cooling on the compensatory hypertrophied dog kidney was investigated. Renal function was measured prior to and on days 1, 3 and 7 after the cooling procedure by means of inulin clearance, PAH clearance and sodium excretion capacity during normal hydration and after volume expansion. No alteration in renal function was shown. No freezing lesions or thromboses were seen on histological examination.

Key words: Renal hypothermia, Intrarenal surgery.

INTRODUCTION

Animal experiments have shown that cooling of the normal kidney to 15 - 20°C provides good protection from ischaemia for as long as 3 h (5). Sludge ice cooling is widely used in clinical practice during intrarenal surgery, especially for staghorn calculi. However, some urologists fear that this simple type of cooling might be responsible for the decrease in renal function which is sometimes observed post-operatively, which might be due to a combination of freezing lesions and thromboses at the renal cortex and ischaemic lesions of the medulla. It has been suggested that these latter lesions are caused by inadequate cooling of the inner parts of the kidney.

The present study was designed to investigate the immediate and long term effects of sludge ice cooling on renal function in the dog before and after volume expansion and to study renal histopathology in this experimental setting.

MATERIAL AND METHODS

Experiments were performed on 11 female dogs varying in weight between 17 an 25 kg. Six weeks prior to the experiments a right nephrectomy was performed through a lumbotomy incision. During that period all dogs developed a compensatory hypertrophy of the remaining left kidney.

The day before the sludge ice cooling a control measurement of the renal function was made. All dogs were conscious during these function tests. Urine collections were obtained by a 12-French Tiemann balloon catheter introduced into the bladder.

The following renal function tests were performed before and after volume expansion: inulin clearance (as a measure of the glomerular filtration rate (GFR)), para-amino-hippuric (PAH) clearance (to assess the effective renal plasma flow (ERPF)) and the absolute and fractional sodium excretion. The extracellular volume expansion consisted of the intravenous administration of an isotonic Ringer's solution over 1 h in a volume amounting to 7.5% of the animal's body weight.

The renal function tests were repeated on days 1, 3 and 7 after the cooling procedure. The latter was performed under a pentobarbital anaesthesia. Before cooling, a silicone catheter was introduced into the abdominal aorta distal to the left renal artery. A subcutaneous tunnel was made from the left lumbotomy to the lumbar interscapular area, where the catheter was brougth out to the surface to allow easy blood sampling during the function tests. The left kidney was then cooled with sludge ice (a freezed Hartman solution) for 90 min to approximately 15°C. The intrarenal temperature was measured by a thermistor probe located 1.2 cm under the renal capsule. During the cooling procedure which

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lasted 90 m the whole vascular pedicle, as well as the ureter, were clamped.

All dogs received an intramuscular injection of 2 ml of an antibiotic solution, containing 150.000 U of procaine benzylpenicillin and 250 mg of streptomycin, for 7 days starting on the 1st day of operation.

Plasma and urine inulin and PAH concentrations were determined by the method of Führ et al. (2) for inulin and by the method of Harvey and Brothers (3) for PAH. Sodium concentrations in plasma and urine were measured by a flame photometer.

The results are expressed as mean ± standard error of the mean (Table 1).

Statistical comparison of the results was performed using Student's test for paired observations. A p value of less than 0.05 was considered to be significant.

RESULTS

In the compensatory hypertrophied dog kidney the temperature of 15°C was reached within 10 to 20 min after the onset of cooling, depending on kidney size. The rate of cooling is shown in Fig. 1.

Before completing all the renal function tests, 4 dogs died: two dogs pulled out their aortic catheter and died from bleeding, another died of acute haemorrhagic gastritis and a fourth died of an unknown cause. These four dogs had normal postcooling renal function on day 1.

Figure 2 represents the control and follow-up GFR and ERPF before and after volume expansion. Although both GFR and PAH clearance significantly increased after volume expansion on each day, no significant changes in either parameter were obtained, when compared to control values before and after volume expansion.

Before cooling, the mean control GFR was 2.77 ± 0.20 ml/min.kg and rose significantly after volume expansion to 4.75 ± 0.49 ml/min.kg. The GFR values on day 7 after the cooling proce-

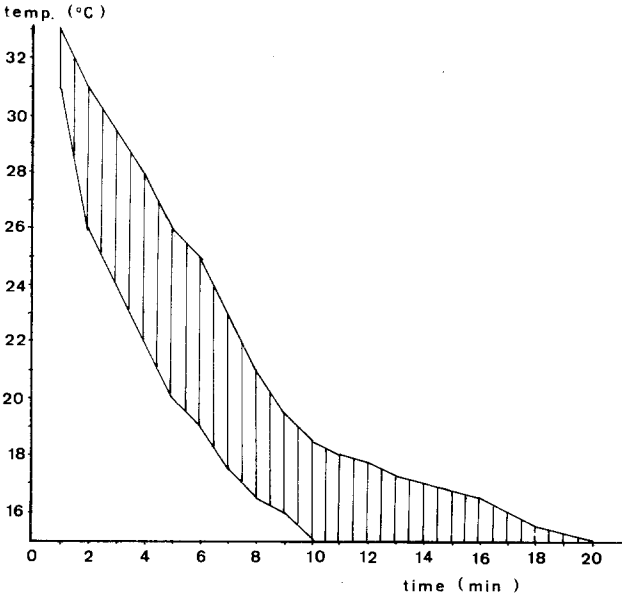

Fig. 1. Intrarenal temperatures during surface cooling (all measured values are included in the dotted area)

Table 1. Values of glomerular filtration rate (GFR), effective renal plasma flow (ERPF), absolute sodium excretion ($U_{Na}V$) and fractional sodium excretion (FE_{Na}), before and after the cooling procedure

		Control	Day 1	Day 3	Day 7
GFR (ml/min.kg)	(1)	2.77 ± 0.20	3.02 ± 0.24	2.61 ± 0.36	2.83 ± 0.25
	(2)	4.75 ± 0.49	4.57 ± 0.34	4.27 ± 0.76	4.01 ± 0.59
ERPF (ml/min.kg)	(1)	6.94 ± 0.64	7.61 ± 0.80	7.50 ± 1.03	8.63 ± 0.89
	(2)	13.47 ± 0.77	12.29 ± 1.33	9.90 ± 2.10	10.97 ± 1.57
$U_{Na}V$ (μE/min)	(1)	48.46 ± 13.65	33.03 ± 8.38	33.13 ± 8.05	48.26 ± 16.17
	(2)	1021.06 ± 165.88	721.51 ± 107.69	880.35 ± 141.26	461.18* ± 114.13
FE_{Na} (%)	(1)	0.55 ± 0.15	0.35 ± 0.07	0.50 ± 0.15	0.53 ± 0.16
	(2)	7.99 ± 1.04	5.16 ± 0.99	7.82 ± 1.55	4.11* ± 1.20
	n	11	11	9	7

(1) = Values during normal hydration
(2) = Values after volume expansion
n = Number of dogs
* = $p < 0.05$ from control value

dure were 2.83 ± 0.25 ml/min.kg and 4.01 ± 0.59 ml/min.kg respectively ($p > 0.05$ versus control).

The control pre-cooling ERPF was 6.94 ± 0.64 ml/min.kg and rose after volume expansion to 13.47 ± 0.77 ml/min.kg. After the cooling procedure, the ERPF values on day 7 were 8.63 ± 0.89 ml/min.kg and 10.97 ± 1.57 ml/min.kg respectively ($p > 0.05$ versus control values).

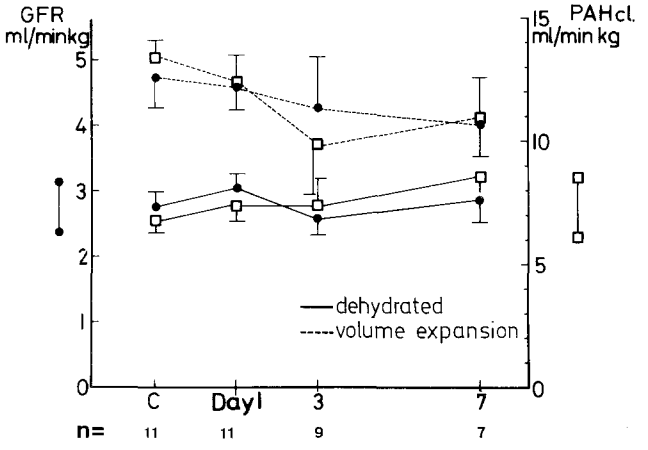

Fig. 2. Glomerular filtration rate (GFR) and para-amino-hippuric acid clearance (PAH cl) in ml/min.kg under normal hydration and after volume expansion

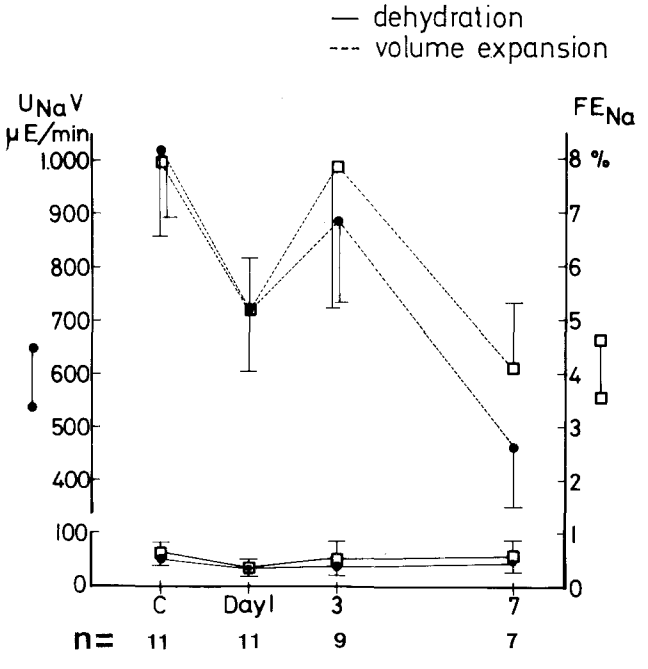

Fig. 3. Absolute sodium excretion ($U_{Na}V$ = urinary sodium concentration x urinary volume) in micro equivalents per min and fractional sodium excretion

$$(FE_{Na} = \frac{\text{absolute urinary sodium excretion/min}}{\text{GFR x plasma sodium concentration/100}})$$

The changes in absolute sodium excretion ($U_{Na}V$) and fractional sodium excretion (FE_{Na}) are shown in Fig. 3. The cooling procedure did not effect these values either before or after volume expansion. Before cooling, the absolute sodium excretion was 48.46 ± 13.65 µE/min and the FE_{Na} was 0.55 ± 0.15%. On day 7 the respective values were 48.26 ± 16.17 µE/min and 0.53 ± 0.16%.

After volume expansion both absolute and fractional excretion of sodium rose significantly and the rise was similar up to 3 days after the cooling. Absolute sodium excretion and FE_{Na} were 1021.06 ± 165.88 µE/min and 7.99 ± 1.04% prior to cooling, and 880.35 ± 141.26 µE/min and 7.82 ± 1.55% on day 3. On day 7, however, a significant decrease of the capacity for sodium excretion after volume expansion was observed with an absolute sodium excretion of 461.18 ± 114.13 µE/min and a fractional sodium excretion of 4.11 ± 1.20% ($p < 0.05$ versus control volume expansion values).

At day 7 the increase in sodium excretion after volume expansion was reduced by approximately 50%, compared to the control day.

Nine kidneys were examined histologically. With the exception of 1 kidney, which showed acute interstitial nephritis, the kidneys did not reveal any evidence of thrombosis, nor any specific lesion of the renal tissue that could be attributed to the cooling procedure. Seven kidneys showed some non-specific minor lesions of interstitial nephritis.

DISCUSSION

It has been suggested that sludge ice gives inadequate cooling of the renal medulla but at the same time provokes freezing lesions and thromboses of the renal cortex. In the present study, the long term renal function after 90 min of renal surface sludge ice cooling, measured by GFR, ERPF and sodium excretion capacity before and after a challenge with a sodium load, remained unchanged. Only after 1 week was an inability to normally excrete an acute sodium load observed. Even this finding must be interpreted with caution, since no control group, where cooling was not performed, was studied. However, this impaired sodium excretion could have been caused by some tubular dysfunction and minor histological tubular lesions were indeed observed after the third day. It could be argued that the minor histological alterations observed could have been caused by subclinical infection due to the presence of an aortic catheter. However, the only dog with a urinary tract infection at the onset of the experimental procedure, and suffering from overt acute interstitial nephritis, had no change in follow-up renal function or sodium excretion.

On light microscopy no thrombosis nor any freezing lesion of the cortex could be detected in any of the kidneys examined.

The few experimental studies available in the literature on this subject seem to confirm our results. Recently, Ackermann et al. performed a comparative study between surface cooling and perfusion cooling in pigs (1). Renal function was measured by means of serum creatinine levels and I^{131}-hippuran clearances and did not reveal any deterioration of these parameters. They concluded that application of sludge ice was the cooling technique of choice.

Surface cooling is also advocated by Luttrop et al. (4). In their study, renal function in piglets was measured by clearances of Cr^{51} ethylenediaminstetracetic acid and I^{125} hippuran. No effect was observed after surface cooling followed by 3 h ischaemia.

Based on both the present and previous studies the value of more complicated cooling perfusion techniques during renal surgery can be questioned since the simple sludge ice method affords the same results.

In conclusion, the technique of sludge ice cooling of the hypertrophied dog kidney has no significant influence on renal function. It provides sufficient protection to renal ischaemia for up to 90 min and does not provoke thrombi nor freezing lesions in the renal tissue. However, it remains to be shown whether sludge ice cooling is free from risk in the pyelonephritic kidney.

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