TABLE I. Effect of Hypothermia upon Resistance to Bacteria Given during or after Shock of 2 Hours Duration.*

Group	Experiment	No. of dogs	Survivors No.	Survivors %
I	*E. coli* intravenously in normal dogs	10	10	100
II	2 hr shock in normothermic dogs	10	9	90
III	2 hr shock in normothermic dogs, and *E. coli* intrav. during shock or up to 24 hr after transfusion	30	0	0
IV	Hypothermia (28°C) induced prior to 2 hr shock, *E. coli* intrav. during hypotensive period—warmed to normal temp. after transfusion	8	6	75
V	Same as Group IV except *E. coli* given after body temp. was back to normal†	14	11	79

* Hemorrhagic shock—bleeding to blood pressure 30 mm Hg for 2 hr followed by transfusion of all shed blood.

† This group includes 4 experiments in which a coagulase-positive hemolytic *Staphylococcus aureus* was used. Recovery rate is about the same for this organism as for *E. coli* in normothermic and hypothermic dogs.

ficient to fully sustain their original potency.

Conclusion. The anti-bacterial defense mechanisms of the dog, which are severely injured by exposure to hemorrhagic shock of two hours duration, are protected to a considerable degree by precooling to 28°C.

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Fluorine in Urinary Tract Calculi.*† (22208)

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The pathogenesis of urolithiasis is one of the unsolved problems in modern medicine. A recent survey answered by 340 American urologists showed an average recurrence rate of 13.7%(1). It is apparent that urinary tract calculi form an important disease entity and that any new facts which may help clarify the problem of calculogenesis are worthy of attention.

The purpose of this note is to report that fluorine has been found in urinary calculi with frequency. Searching the literature failed to reveal reports of urinary calculi analyzed for fluorine. It has been reported that renal damage, albuminuria and hematuria have been found in humans and animals suffering from fluorine poisoning(7-13).

Methods. Parts of the stones from 10 cases of urolithiasis were analyzed for calcium, ammonium, magnesium, oxalates, phosphates, urates and carbonates in our laboratories. Other parts were analyzed for fluorine by the laboratories of Dr. H. Amphlett Williams of London, England, and Dr. Chester A. Snell of Foster D. Snell, Inc., N. Y. City. Determination of fluorine in such materials is a difficult, delicate and tedious laboratory procedure accomplished by different methods by each individual. The method used by Dr. Williams is that described by him in 1946(2). Dr. Chester Snell states(3) that his methods in determining fluorine content of calculi were those mentioned in the "Official Methods of Analysis

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TABLE I. Clinical and Laboratory Data.

Age	Sex	History	Location and No. of calculi	Analysis	Ca content (%)	Fluorine (parts/million) Williams	Snell
66	♂	Prostatic hypertrophy 1 yr	Recurrent vesicle (2)	Uric acid, calcium oxalate	Not done	340	
46	♀	1 yr	Ureteral (3); vesicle (2)	Calcium oxalate and phosphate	" "	790	816
80	♂	5 yr of bilateral nephrolithiasis, 1 yr vesicle calculi	Vesicle (35)	Calcium carbonate and magnesium ammonium phosphate	26.9	210	315
72	♂	2 yr prostatic hypertrophy	" (100+)	Uric acid, calcium and ammonium carbonate and phosphate (trace of magnesium)	13.6	570	850
57	♂	1 mo	Ureteral (1)	Calcium oxalate	24.9	240	520
4	♂	Congenital bilateral ureterovesicle anomalies	Bilateral ureteral and vesicle (numerous)	Magnesium ammonium phosphate	0	30	35
72	♂	Several years prostatic hypertrophy	Vesicle (numerous)	Uric acid and calcium oxalate	5.9	19	0*
62	♂	1 yr prostatic hypertrophy	Small prostatic (numerous)	Not done	26.9	730	1290
68	♂	2 yr increasing prostatic hypertrophy	Large vesicle (2)	Inner layer uric acid	0	5	0*
				Outer " " "	0	4	0
37	♂	17 yr recurrent renal calculi	Ureteral (1)	Not done (previous calculi reported as calcium oxalate)	26.3	1560	1790

* Less than 1 part/million.

TABLE II. Fluorine Data from the Literature (for Comparison).

Material	Fluoride content, parts/million	Source
A. Human		
1. Normal teeth and bone	10–30	4†
2. " tissues	.8	4
3. a) 24 hr urine collection	.3–.5*	4
b) Normal urine	.4	5
4. Kidney (normal)	.78	5
5. " (in fatal fluorine poisoning)	11.6	5
6. Liver (normal)	.6	5
7. " (in fatal fluorine poisoning)	12.2	5
B. Exp. tissues from poisonings of dogs		
1. Acute fluorine poisoning	14–16	4
2. Chronic " "	2–5	4
C. Water		
1. N.Y.C. water supply	.1	14
2. Artificially fluoridated water	1.0	
3. Sea water	3.0	3
4. Naturally fluoridated water (very high content)	10 (Aztec, Ariz.)	6

* mg. † Ref.

of the Association of Official Agricultural Chemists," 7th edition. It is because of the difficulty of obtaining close correlation in results of the fluorine determinations that portions of all calculi, if available, were sent to both Dr. Williams and to Dr. Snell. Numerical differences reflect the difficulty in obtaining an accurate quantitative result in this analysis. It is also possible that fluorine content may differ at various parts of a calculus. This point will need further elucidation.

Table I gives a summary of clinical and laboratory data of 10 cases of urinary tract calculi studied.

Table II presents data from the literature, which show that values found in calculi greatly exceed those hitherto reported in biological materials including those obtained in experimental poisoning. The highest fluorine content in tissues reported in any of the papers is in the neighborhood of 30 parts per million; while in the calculi analyzed, fluorine contents up to 1500 to 1700 parts per million were found.

From the data on specimens in which calcium content could be determined, it will be noted that when calcium content is high, fluorine values are also high. In the two calculi

in which no calcium was found, fluorine content is low.

One factor that all these patients had in common was urography. Almost all patients with calculi have had either intravenous or retrograde urography usually performed with one type or another of radio-opaque medium. In order to eliminate the iodine-containing urographic dyes as the source of the fluorine found in the calculi, samples of 3 of these materials were analyzed and found to contain no fluorine.

Summary. Fluorine has been found in high concentration in 8 out of the 10 urinary tract calculi analyzed.

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Lactic Acid Response to Epinephrine in Experimental Liver Disease.* (22209)

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Normal and partially hepatectomized rats were studied to determine whether differences in the utilization of lactic acid following the injection of epinephrine could be correlated with liver mass. Significant differences were observed and additional studies were carried out on the effect of carbon tetrachloride poisoning and bile duct ligation on the ability of rat liver to handle similar lactate loads.

Method. Sprague-Dawley albino male rats weighing 180-210 g were used. All animals were kept in a constant temperature room on a uniform diet. Animals received a subcutaneous injection of 0.02 mg/100 g body wt., of a freshly diluted solution of epinephrine with added glutathione (Adrin, Sharp & Dohme). Tail vein samples were used for lactic acid determinations. Partial hepatectomy was performed by a technic in which 70% of the liver mass was removed. Carbon tetrachloride poisoning was accomplished by the intraperitoneal injection of 0.10 cc of CCl_4/100 g body wt., a modification of a recently described technic(1).

TABLE I. Effect of Epinephrine on Blood Lactic Acid in Normal and Partially Hepatectomized Rats.

Hr after epinephrine	Normal rats	Part.-hep. rats
Resting, before epinephrine	17 ± .25*(57)	16.5± 1.3 (17)†
1	41 ± 1.7 (36)	46 ± 3.3 (16)
2	21 ± .3 (28)	37 ± 2.3 (17)
3	17 ± 2.3 (25)	36 ± 1.4 (11)
4	—	27 ± 2.3 (11)
5	—	20 ± 1.5 (13)

* S. E. † No. rats.

Results. Resting levels of blood lactic acid were the same in the normal and partially hepatectomized animals (Table I). Both

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