After entering the hospital a biopsy was done and histological examination made. The patient was immediately given injections of sodium thiosulphate intravenously, 1 gm. every other day, and 15 grains (0.97 gm.) of thiosulphate three times a day after meals. The intestinal bleeding stopped at once, and under continued injections the acute dermatitis of his head, hands and feet slowly subsided, and the peculiar pigmentation with lichenoid papules scattered through it—particularly on the body and on the back of the neck—showed improvement.

Summary. Sodium thiosulphate shows no deleterious action on the trypanocidal activity of the salvarsan groups when the two drugs are given simultaneously. These results are corroborated by clinical observations of other investigators. Sodium thiosulphate plays an important part in the rate of excretion and also the clinical symptoms following an intoxication due to arsenic in (a) inorganic state, (b) pentavalent organic state, and (c) the organic trivalent arsenicals.

Clinical study showed that when the injections of thiosulphate were stopped and the arsenic output in the urine dropped down to about 0.003 to 0.004 mg., the eruption on the head, hands and feet and the peculiar lichenoid eruption on the back, chest, arms and legs regularly became worse. With the renewed administration of the thiosulphate, and when the arsenic output came up to about one decigram of arsenic for each 100 gm. of moist specimen, the eruption regularly improved.

2846

Hyperglycaemia following experimental cholecystitis.

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A series of fifteen experiments were conducted as follows: A presumably normal mongrel dog was prepared for laparotomy, by administration of morphia and ether, shaving, and cleansing of the abdominal field for operation. Under sterile conditions the abdomen was opened, the gallbladder was identified and incised.

Summary of Experiments on Pancreatitis Secondary to Cholecystitis.

Exp. No.	Duration in days.	Mg. blood sugar per 100 cc. blood		Pathological Findings
		Preoperation	Postoperation	
3	17	88	?	—
5	60	96	170 120 260 160 160 130 180	Cholecystitis Lymphadenitis of biliary, pancreatis, and coeliac nodes
6	60	100	150 140 140 165 175 150 175	Cholecystitis Liver congested Lymphadenitis Pancreas thickened
8	37	88	146 100 140 148 166	Cholecystitis Lymphadenitis Pancreas thickened
11	2	?	?	—
17	0	134	—	—
18	3	260	?	Death from infection
19	39	116	126 105 90 170 136	Cholecystitis Lymphadenitis Abscess of pancreas Capsular and interlobular infiltration; congestion; hyalinization of islands
20	35	136	120 195 192	Cholecystitis Lymphadenitis Pancreas thickened
21	1	156	?	Death from infection
24	32	240	100 164 160	—
35	6	126	92	Cholecystitis Cloudy swelling of pancreas Fatty degeneration liver
37	10	110	108	Liver congested; Cholecystitis; lymphadenitis; pancreatitis. Necrosis of acinar cells.

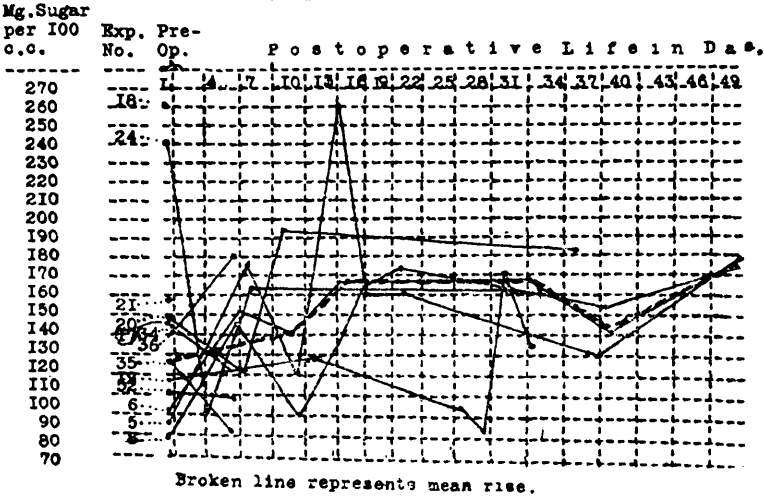
A glass bead containing faecal matter and bacteria was dropped into the bladder, the bladder was closed with suture, and left attached within the plane of closure of the abdominal wound. After such an operation the animal was placed in a recovery cage and fed water, fluids, and solid material according to the return of the dog's appetite. The diets of the animals fully recovered from operation consisted of meat scrap from the tables of the Hospital dining-room. The blood sugars were examined in each such animal before operation and at intervals of three to five days after operation. After an interval of one to sixty days, each of the experiments was terminated by dispatching the animal and by determining the pathological changes that had taken place within the dog's abdomen.

The particular blood sugar findings and pathologic changes are summarized on the accompanying chart. The fluctuations in blood sugar content in relation to the mean sugar content of the total series studied appears on comparison of the lines on the diagram below.

Glycosurias were noted in the animals examined.

It is worthy of special attention that the animals showing rises in the blood sugar after operation were animals in which pathologic changes were taking place about the gallbladder, in the lymphatics of the bile ducts, liver, and pancreas, or in the pan-

DIAGRAM REPRESENTING BLOOD SUGAR TRACINGS IN DOGS BEFORE AND AFTER PRODUCTION OF EXPERIMENTAL CHOLECYSTITIS



creas itself. Two animals with a distinct fall in the blood sugar after operation gave no evidence of lymphadenitis. The mean rise for ten observations was from 125 to 140 mg. per 100 cc. blood for the first 12 days, or, from 125 to 165 mg. for the first month after the introduction of infectious material in the gall-bladder. Experiment No. 19 with an abscess of the head of the pancreas was narcotized on the 32nd day, during a rise in sugar, to determine the possible effects of anesthesia upon the blood sugar. It was observed in this instance that the percentage fell from 170 to 135 mg. in the succeeding three days. It is felt that the rise in the blood sugar in these experimental animals has been due not to anesthesia, not to diet, not to confinement of the laboratory animals, but to morphological changes in the pancreas consequent upon infection lymphatic-borne from the gallbladder and liver.

The frequent association of diabetes or hyperglycaemia with cholecystitis, lymphadenitis and pancreatic thickening in the human patient, as met with in the surgical ward of Bellevue Hospital, has led to the suspicion that the diabetic state may be in part at least of infectious origin. The above experiments were undertaken to determine whether infection arising in the gall-bladder and spread by the lymphatics gives rise to hyperglycaemia.

2847

Differentiation of B. aerogenes and B. coli of non-fecal origin from B. coli of fecal origin.

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Koser [1, 2, 3] has demonstrated with the "colon bacilli" that *B. aerogenes* and *B. coli* isolated from soils utilize citrates as a a source of carbon and that *B. coli* of fecal origin does not use it.

[1] Koser, S. A., *Abs. Bacteriol.*, 1924, viii, 6.

[2] Koser, Stewart A., *J. Bacteriol.*, 1923, viii, 493.

[3] Koser, Stewart A., *J. Bacteriol.*, 1924, ix, 59.