TABLE I.

Amount of Ascorbic Acid added to 100 cc. blood mg.	Ascorbic Acid found per 100 cc. blood mg.	Recovery %
0	1.68	—
1.2	2.92	105
1.8	3.33	93

hundredths. The titration should be complete within 3 minutes. This procedure allows for sufficient filtrate to permit 2 titrations. Table I indicates the degree to which added ascorbic acid may be recovered from blood.

Allowing the blood to stand at room temperature for varying periods up to 24 hours has no appreciable effect on its ascorbic acid content.

All figures on which this report is based were obtained from bloods taken in the post-absorptive state and represent observations on about 100 different individuals. The values in apparently normal individuals ranged from 1.19 to 2.66 mg. %. Those in patients suffering from a variety of chronic diseases (including diabetes mellitus, hyperthyroidism, rheumatic heart disease, arteriosclerosis, acromegaly and chronic glomerular nephritis) ranged from 1.11 to 2.88 mg. %. No correlation between the diseases investigated and the ascorbic acid values obtained is apparent, although cases of coronary sclerosis were almost uniformly grouped at the upper limit of the above range. We have also been unable to make any correlation between the total ascorbic acid content of the blood and the dietary regime.

8000 P

A Biliary Precipitate Characteristic of Cholelithiasis.

I. ARTHUR MIRSKY. (Introduced by Samuel Soskin.)

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We have been unable to find any previous report of a chemical test by which bile drawn from the gall-bladder or bile ducts of patients suffering from cholelithiasis may be distinguished from

* This work was started at the Richard Morton Koster Laboratory, Brooklyn, New York.

normal human bile. For the past 3 years we have been observing a phenomenon, as yet unexplained, which is characteristic of the bile drawn by needle and syringe from human gall bladders or bile ducts containing calculi, but which is absent in normal human bile and in dog's bile. Through the courtesy of Dr. Harry Koster of Brooklyn, N. Y., and of Dr. Ralph Bettman of Chicago, Illinois, we have now observed this reaction in over 200 cases of cholelithiasis. Our attempts to make this test on biles obtained by duodenal drainage have been unsuccessful thus far, due probably to the factors of dilution and interfering substances. We are reporting our observations at this time, in the hope of stimulating work by others which may lead to a valuable clinical test for cholelithiasis.

The characteristic phenomenon is observed when 1 cc. of N/12 sulphuric acid is added to 1 cc. of fresh bile, removed by needle and syringe at operation, or autopsy soon after death, from patients with cholelithiasis. An immediate precipitation of a pale, yellow-green, gelatinous substance occurs. This precipitate, once seen, will not be confused with the fine, particulate precipitate produced by the same procedure in biles free from calculi. The amount of the characteristic precipitate which appears is roughly proportional to the bulk of the stones found in the gall-bladder, and our predictions in this regard, based on tests of biles without preceding knowledge of the surgical pathology, have been surprisingly accurate. We have been unable to make any correlation between the occurrence of the characteristic precipitate and the concentration, viscosity, protein, or cholesterol content of the bile which we tested.

In none of the 200 cases where gall stones were demonstrated did we fail to get the characteristic precipitate. However, in several cases, clinically diagnosed as cholelithiasis and in which the bile yielded a small amount of the typical precipitate, no calculi could be found. These instances may represent failures of the test, but probably do not in view of the following considerations. In one such case, careful examination of the gall bladder after surgical removal revealed a quantity of calculous material resembling sand. In another instance clinically diagnosed as cholelithiasis and in which the bile was positive according to our test, no stones were found at operation. Nevertheless a cholecystectomy was performed and a drainage tube inserted into the common bile duct. The bile collected from the drain continued to give consistently positive results and on the fourth day post-operative, a small calculus, presumably hepatic in origin, was passed through the drainage tube. This latter case also suggests that the substance responsible for the positive reaction

originates in the liver and not the gall bladder. This is confirmed by the fact that fluids drawn from the gall bladder in 2 cases of hydrops of the gall bladder with stones in the cystic duct, both failed to yield the characteristic reaction.

Our results indicate a significant difference from the normal in the bile from cases of cholelithiasis. The alteration in the composition of the bile responsible for the precipitate probably does not originate in the gall bladder. Further work on this problem should shed light on the origin of gall stones.

It is a pleasure to acknowledge the aid and direction of Dr. Samuel Soskin.

8001 C

Peptic Ulcers Produced by Feeding Cincophen to Mammals Other than the Dog.

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Churchill and Van Wagoner[1] demonstrated that gastric and duodenal ulcers can be regularly produced in dogs by administration of cincophen. Cases of alleged cincophen poisoning in man have not been characterized by such ulcers. It appeared desirable, therefore, to determine whether this effect in the dog is a species-selective phenomenon.

Cats, rabbits and guinea pigs were given cincophen orally suspended in cotton seed oil. The dosages were calculated according to the principle used by Churchill and Van Wagoner, *i. e.*, on the basis of 22 mg. per kg. body weight which corresponds to the human dose of 7.5 grains t.i.d. for the average adult of 150 lbs. During the experiment the animals received the usual care given to the particular type of experimental animal. In each case the postmortem examination was done as soon after death as possible.

The following tables give a resumé of the results.

Results with Cats

Cat No. 1. 2 doses each 10x N.H.D.* Death on second day. A few superficial erosions in gastric mucosa up to 4 mm. in diameter.

1 Churchill and Van Wagoner, PROC. SOC. EXP. BIOL. AND MED., 1931, **28**, 581; *Arch. Path.*, 1932, **14**, 860.

* N.H.D. Normal Human Dose.