are identifying far more frequently than heretofore—little as yet can be offered of a conservative character. Theoretically there should be no justification for sacrificing an apparently sound ovary or tube. If one is infected and the other not it would seem to be reasonable practice to leave the sound tube, even though a later infection should occur and a second operation be necessary. This doctrine would be beyond controversy did we actually know that the endometrium and the lining of the sound tube were uninfected. In the light of our present methods, however, we can not know, and hence we must be guided by clinical experience; this has taught us that when one tube becomes manifestly tuberculous infection of the other is almost inevitable and that in this infection the uterus is frequently first. With this pathology we now know but one safe course of action—total extirpation. The future should hold something better than this.

The technic in tubal surgery must depend largely on the character and degree of infection and the extent of destruction and deformity. In simple stricture at the isthmus, resection of the isthmus and the anastomosis of the ampulla with the proximal end of the tube near the cornu, offers good results. A cord of heavy plaited silk should be anchored in the ovary and the other end carried down through the uterine cavity to a gutta percha bobbin in the vagina to insure the mucous tract remaining patulous during the repair of the anastomosis. I have known pregnancy to occur within three months following this procedure.

When the mouth of the tube has closed by a sealing over of the processes of the fimbria the adhesions, which are usually thin and delicate, should be opened up, the cavity of the ampulla carefully syringed out with salt solution and the edges of the released fimbria folded back over the tube like a cuff and tacked there with interrupted stitches of fine catgut.

When the tube is angulated or kinked by fine dense bands these should be snipped and the tube straightened out, and if a stricture persists the dorsum of the tube should be laid open and the lumen given the usual toilet. Attempts to probe the tube are usually so fruitless that such injury as is sure to result should be avoided. Careful syringing with salt solution in a fine syringe is far safer and more effective.

In tubes which have been septic or gonorrheal and in which active cocci no longer exist, or in mild types of hydrosalpinx, the occluded tube should be opened up, cleansed and the cystic portion utilized as an ampulla. If perfectly free from infective material the dorsum of the obliterated portion of the tube may be laid open and a silk cord passed downward as in the case of anastomosis already described. Frequently these tubes will be found patulous just proximal to the barrier of the cyst wall and the incision may cease a line or two beyond it.

When the tubes are large pus sacs it has been customary to remove them and, commonly, the uterus. It remains to be seen if we shall not be able to do better than this by carefully and properly applied drainage in a series of operations. It can not be denied that so far little of merit in this direction has been recorded; yet if we were to quail at a septic tendovaginitis in the hand and wrist, as we do at conserving pus tubes in the pelvis, what a melancholy showing we should have. Possibly a little more patience, a little more plodding, a little more dogged belief in Nature's regenerative power when nicely and intelligently aided, would be rewarded here as elsewhere, in fields more difficult but not more worthy.

What has been said of preserving the tube when impaired by infection will apply with equal force to tubes changed by ectopic pregnancy. A tube should never be sacrificed if it is possible to save it or even a portion of it, for the normal one may suffer soon in like manner and both thus perish.

In every instance of conservative work on the tubes and ovaries the uterine cavity should be thoroughly curetted and cleansed, since it may be necessary to establish and maintain communication for several weeks between the abdominal cavity and vagina through the uterine canal. That this can be done with perfect safety has been repeatedly proved, but the price is strict asepsis, both as to the sterility and toilet of the tubes and after toilet of the vagina.

It will often be found necessary to shorten the uterine round ligaments to correct version and descent, and this should not be overlooked.

The results of value in a certain procedure are the end results. I know of none more positive or beneficent than in this field, provided careful judgment and attention to minute detail is faithfully observed. My hopes will be more than realized if this paper shall serve to revive a just interest in a field by no means worked out and one in which conservatism will be of much benefit to the race.

THE VALUE OF ALCOHOL IN CARBOLIC ACID POISONING.

A CLINICAL AND EXPERIMENTAL STUDY.*

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1. INTRODUCTION.

The use of alcohol as an antidote in carbolic acid poisoning was first brought before the medical profession by Dr. Seneca D. Powell of New York City. At his clinical lectures, from about 1894 on, he was in the habit of demonstrating his theory by such an experiment as washing his hands in pure carbolic acid and then in alcohol, and had even filled his mouth with the pure acid, and by quickly rinsing it with 95 per cent. pure alcohol had experienced no harmful results. From these experiments he made the statement that alcohol is an antidote to carbolic acid, and suggested its being used internally in cases of poisoning. The results of his five years experience were reported to the Medical Society of the County of New York Feb. 27, 1899, and appeared in the reports of this society.¹ He, however, had had no practical experience in poisoning cases and goes no further than to mention a case of which he had heard of a one-month-old infant, who, after swallowing the strong acid, had been given a teaspoonful of whisky, though apparently moribund, and had recovered temporarily. Furthermore, this child, having died eight days later of pneumonia, showed no signs of an eschar in the stomach.

The first mention of alcohol as an antidote from a clinical standpoint is in the report by Donald B. Fraser² of a patient who took an ounce of the pure acid in a glass of whisky, and, though unconscious for eight hours,

* From the Medical Service of the Lakeside Hospital and the Pharmacological Laboratory of Western Reserve University.

1. Powell: Medical Record, New York, 1899, vol. iv, p. 372.

2. Fraser Ibid., 1899, vol. xviii p. 741.

eventually recovered, repeated lavage with water having been resorted to. He then states that alcohol is apparently a true antidote in carbolic acid poisoning. Whether the author was familiar with Powell's work, at that time unpublished, he does not state.

The credit of first using alcohol in poisoning cases seems to be due to Dr. J. Drysdale Buchanan,³ who used lavage with two quarts of 35 per cent. alcohol for a patient who had taken one ounce of the acid.

Since these early reports there have been a number of isolated cases in the literature from which the most positive conclusions have been drawn. For example, Kelly⁴ in 1899, after reporting one successful case, states that "alcohol is undoubtedly the best antidote for all cases of carbolic acid poisoning, and, indeed, is an absolute antidote." Phelps,⁵ on the strength of Powell's work, says: "In cases of carbolic acid poisoning, if immediately after the administration of the poison, alcohol was thrown into the stomach the poisonous effects of the carbolic acid would at once be neutralized." Pinkney,⁶ after reporting a fatal case, says: "The amount of carbolic acid left in the stomach and intestines when alcohol came in contact with it was rendered harmless, and the general action of the already absorbed poison was checked immediately." Finally, Gross,⁷ in discussing the subject, refers to alcohol as a chemical antidote, and says that the reaction forms "a new phenol benzene or aromatic compound having the chemical and therapeutic properties of alcohol."

Two years ago one of us, having been favorably impressed by some of these reports and by good results obtained by the use of alcohol by himself, and other members of the staff of the Johns Hopkins Hospital, in cases of carbolic acid poisoning, introduced the use of alcohol in the emergency room of the Lakeside Hospital. Since that time thirteen patients with carbolic acid poisoning have been admitted to the hospital, for all of whom the alcohol treatment has been used.

2. CLINICAL DATA.

BY T. W. CLARKE.

As all former reports on this treatment have been of single cases, from which, owing to the natural tendency of the profession to report their successful cases only, but slight conclusions can be drawn, it has seemed advisable to report the present series of thirteen cases more or less in full. For permission to use these cases we wish to thank Drs. Hunter Powell, John H. Lowman, Henry E. Upson and Edward F. Cushing, visiting physicians to the Lakeside Hospital.

CASE 1.—Patient admitted at 8 p. m. Dec. 14, 1903, unconscious. Had taken carbolic acid, 3ss, in whisky. There were two burns on chin. Pupils react, breathing heavily. Pulse was so rapid and weak that it could not be counted. Stomach was washed out with two quarts alcohol, 10 per cent., then milk and eggs, Oi, left in stomach. Strychnin, 1/30 gr., digitalin, 1/30 gr., given hypodermically. At 9 p. m. patient was conscious and very noisy; quieted with sodium bromid and chloral, ʒʒi, by rectum, and slept the rest of the night. Temp. 100, pulse 110, at midnight.

December 15: Some pain on swallowing. Nothing to be seen on examination of the throat and mouth. Temp. 99 to 101.5, pulse 80, resp. 20, urine dark.

December 16: Patient allowed up in wheel chair. Still

slight soreness on swallowing. Physical examination negative. Temp. normal, pulse 70 to 80, respirations 20 to 26.

December 18: Eats well, no difficulty in swallowing. Scar on chin disappearing. Discharged, well.

CASE 2.—Patient admitted 11 a. m. Jan. 17, 1904, having taken carbolic acid, ʒiss, shortly before, in glass of beer. Patient was unconscious. Pulse arrhythmic, rate 160. No burns on lips, slight erosion on cheeks. Lavage performed with alcohol, 10 per cent. The washing contained carbolic acid and much undigested food. Milk and eggs, Oi, added and left in. Artificial respiration was resorted to several times. Strychnin, 1/20 gr., and digitalin, 1/30 gr., were given twice in quick succession. Temp. 96.4, resp. 58, pulse 160. At 1:30 p. m. the pulse and respiration became most irregular. Strychnin, 1/20 gr., and digitalin, 1/30 gr., steadied both. Hot water bottles were applied and coffee, ʒviii, given *per rectum*. At 3 p. m. subcutaneous infusion of normal saline, 600 c.c., was given and repeated at 7 p. m., the last containing 7 gm. sodium sulphate. Patient vomited frequently between 4 and 5:45. Patient first showed consciousness at 7 p. m. while the infusion was being given.

January 18: Patient is doing well. Temp. 100.5 to 101, pulse 100 to 110, resp. 30 to 40. The patient looks well and seems but little the worse for her experience. Urine black, slight trace of albumin, no blood, no casts.

January 19: Last night the patient's temperature rapidly rose, and this morning was 105, resp. 70 and pulse 160. Leucocytes, 5,900. The patient is very cyanotic. Sponge baths, strychnin and oxygen ordered. Examination of chest is negative. Urine yellow, heavy trace of albumin, no blood, no casts.

January 20: The temperature has remained in the neighborhood of 105, pulse 150 to 170, resp. 60 to 65. To-day there is a definite pneumonia area over right upper lobe. Over the entire chest there are patches of tubular breathing and large mucous rales. Patient died at 10:30 a. m.

Autopsy showed an aspiration bronchopneumonia and a small, irregular, dark-colored patch in the fundus of the stomach.

CASE 3.—Patient admitted March 4, 1904, 9 p. m. Walked in, said he had drunk three glasses of whisky and then carbolic acid (doubtful quantity). Was quite conscious and rational. On left side of mouth and lower jaw were acid burns. Inside of mouth and pharynx slightly burned. Respirations normal, 28; pulse good, 100 a minute; temperature normal. Stomach was washed with 10 per cent. alcohol, and water, and sent to ward. He showed practically no constitutional symptoms; slept all night after 1:30; pulse 98 to 104, but of good quality. The next day the inside of patient's mouth was blistered. Discharged, cured.

CASE 4.—Patient admitted May 9, 1904, 12:10 a. m. Brought in in ambulance in a delirious condition. Had been drinking and had then taken carbolic acid, ʒi. Pulse fairly good quality, 100 per minute. Some burns about the lips. The stomach tube was passed and food particles removed, smelling strongly of phenol. Lavage with 10 per cent. alcohol solution and followed with water. Milk, Oi, and 4 eggs inserted and left in stomach. Patient was given sodium bromid, 25 gr., and slept until morning. Temp. 96.6, pulse 100, resp. 24. The urine the next morning was black, contained a faint trace of albumin and a few hyalin casts, sp. gr. 1038. The next morning patient appeared well; slight eschar on lips; nothing to be seen in throat; some epigastric tenderness. Patient was kept in the ward until May 15 and then discharged, well.

CASE 5.—Patient admitted 5:20 p. m. July 22, 1904. She had been drinking for five days, and at 5 p. m. of the day of admission drank 10 cents' worth of carbolic acid (ʒi), with suicidal intent. She was taken into a saloon and became unconscious. On admission she was unconscious, cyanotic, breathing heavily, a great deal of mucus protruding from the mouth, vomiting a little. Pulse rapid and small. The face, mouth and pharynx were badly burned. Temp. 98, pulse 120, resp. 30. The stomach contents showed carbolic acid. Lavage with 10 per cent. alcohol, followed by water, and milk and eggs left in place. She was unconscious after reaching the ward, but regained consciousness at 8:20 p. m. Patient dozed

3. Buchanan: *Ibid.*, 1899, vol. lvi, p. 241.

4. Kelly: *Merck's Archives*, 1899, p. 441.

5. Phelps: *New York Med. Jour.*, 1899, vol. lxi, p. 62.

6. Pinkney: *Amer. Med.*, 1901, vol. i, p. 358.

7. Gross: *Fort Wayne Med. Jour. Mag.*, 1899, p. 73.

all night, waking several times to vomit mucus. The temperature remained elevated for several days, 100 to 101 July 23, 99.5 to 100.5 July 24, 99 to 100 July 25, 26 and 27. The pulse remained at 120 until noon July 23 and then gradually came down to 100 that night, and was 80 on July 26. The respirations from 20 to 30 the first day, 20 after that. The urine was never obtained black, but contained a heavy trace of albumin and many hyalin and granular casts. The leucocyte count the day of admission was 18,400, reds 4,226,000, and hemoglobin 95 per cent. Patient discharged on July 27. The scars on her face were very sore and showed some signs of sloughing. Temperature was 99.

CASE 6.—Patient admitted 2:30 a. m. July 26, 1904. Had taken one ounce of carbolic acid in a glass of whisky. Became unconscious in five minutes, and was sent immediately to the hospital. On admission was unconscious, no sign of burns about mouth or lips; breathing stertorously; pupils small; pulse small, 120 a minute; no vomiting. Lavage with alcohol, 10 per cent., followed with water; after washing, milk and eggs and sodium sulphate, $\frac{3}{4}$ i, were put in through tube. Patient became conscious at 5 a. m. Recovery was uneventful. The urine was dark-brown, acid, sp. gr. 1014, and contained a few hyalin and granular casts. On admission temperature was 97 and rose to 100.2 at 8 a. m., then gradually came down to 99 July 28. Pulse on admission was 118, at night 100, and 70 on the night of July 27. Respirations 32 on admission, at noon 14, and after this 18 to 20. The leucocyte count the morning of admission was 19,500. Patient did well and was discharged July 28, well.

CASE 7.—Patient admitted 8:30 a. m. Sept. 14, 1904. Had taken carbolic acid, $\frac{3}{4}$ i. She was unconscious, tongue and lips slightly burned, very weak, cyanosed, breathing stertorously, pupils much contracted. Pulse barely perceptible, quite irregular, 140 a minute. Stomach tube passed and contents showed carbolic acid. Lavage with alcohol, 10 per cent., and then water, eggs, milk and sodium sulphate, $\frac{3}{4}$ i, by tube. Strychnin, $\frac{1}{30}$, and digitalin, $\frac{1}{30}$, given twice within an hour. Intravenous injection of soda sulphate, 2 per cent., 250 c.c. Patient became conscious in two hours. In the afternoon had severe vomiting spell, which was relieved by dilute hydrocyanic acid, 4 minims, and bismuth subnitrate, 10 gr. On admission temperature was 99, pulse 112 and resp. 30. Temperature went up to 100 that night, and then down to normal the following evening. The pulse remained at 110 for 24 hours, and then dropped to 80 and remained in that neighborhood, with occasional flare-ups. Respiration came down gradually from 30 to 20 in 3 days. The urine was pale, sp. gr. 1020, faint trace of albumin, and a very few hyalin casts. Patient suffered considerably from soreness of the mouth and had very little pain in the stomach. No vomiting after the first day. Discharged, well, Sept. 19.

CASE 8.—Patient admitted Feb. 28, 1905. Child, aged 19 months, swallowed an unknown quantity of 95 per cent. carbolic acid three hours before admission. Seen by a physician at the home five minutes later, who washed stomach with alcohol and water. Patient was unconscious when seen at that time. On admission was very drowsy, temperature 101.5, pulse 160. Tongue, pharynx and inside of cheeks very badly burned, white in color. On admission patient was given magnesium sulphate, $\frac{3}{4}$ i, and strychnin, gr. $\frac{1}{250}$. March 1, temp. 104, pulse 152, resp. 40. March 2, temp. 100, pulse 100, resp. 20. From then on the temperature varied from normal to 100, pulse from 100 to 150, and respirations from 25 to 30. The child did well, and on March 6, the mouth having healed, patient was discharged.

CASE 9.—Patient admitted on March 23, 1905. The history was that she apparently fainted in an elevator, and poisoning was not suspected for one and a half hours. On admission she was moribund, unconscious, cyanotic, resp. 36, pulse small and weak, 160, pupils dilated, faint trace of carbolic to breath, mucous membranes, gums and palate white. Patient was stimulated with strychnin, $\frac{1}{20}$ gr., digitalin, $\frac{1}{30}$ gr., and stomach washed with alcohol, 10 per cent., and eggs and milk and magnesium sulphate, $\frac{3}{4}$ i, left in the stomach, 500 c.c. Soda sulphate, 2 per cent. solution, given intravenously. Pa-

tient never rallied and died half an hour after reaching the ward.

CASE 10.—Patient admitted May 5, 1905, in an unconscious condition. Had taken carbolic acid, time not known. Severe burns of the cheek, corner of mouth, lips, tongue and roof of mouth. Pulse weak and rapid. Stimulated with strychnin and digitalin. Stomach washed with 10 per cent. alcohol and water. Eggs and milk, magnesium sulphate, $\frac{3}{4}$ i, and sodium sulphate, $\frac{3}{4}$ ss, inserted through tube. Temp. 100, pulse 110, resp. 20. Patient was restless after getting to ward, but later slept. Temperature during the day rose to 100.8 and pulse 120, but on the second day temperature became normal and pulse 80. The leucocytes on May 5 were 28,200. Patient had difficulty in swallowing and considerable epigastric tenderness.

On May 7 patient felt well except for the burns on the face, and insisted on going home.

CASE 11.—Patient admitted, unconscious, 9:30 p. m. June 1, 1905. Had taken carbolic acid, $\frac{3}{4}$ i, a few minutes before. Two scars, 4 cm. long, on upper lip; slightly cyanotic; pulse good. Lavage, 10 per cent. alcohol, then magnesium sulphate and eggs and milk by stomach tube. No stimulation. Consciousness returned 10:30 p. m. Next day scars marked on lip and slight on roof of mouth and tongue. Large white patch on right tonsil and posterior wall of pharynx. Larynx inflamed, abdomen rigid and sensitive in upper half. Blood examination: Leucocytes, 11,800; hemoglobin, 83 per cent.; fresh blood normal. Urine greenish, sp. gr. 1020, acid, loaded with hyalin, granular and leucocyte casts. June 4 and 5: Urine, sp. gr. 1015, neutral, no casts. June 5: Discharged, well.

CASE 12.—Patient admitted 3:30 p. m. Sept. 7, 1905. Had taken carbolic acid, $\frac{3}{4}$ i diluted with a little water, fifteen minutes before. She had not swallowed all, as the glass was knocked from her hand after two swallows.

Patient was comatose, cyanotic. Pupils at first dilated and then pinpoint. Pulse very weak. There were two scars on the lips and the entire mouth and tongue was whitened, on the chest and abdomen was very extensive burn, extending from the neck to the pubes. The treatment consisted of lavage with 10 per cent. alcohol and the introduction of yolk of eggs and sodium sulphate $\frac{3}{4}$ i. Strychnin $\frac{1}{20}$ gr. and digitalin $\frac{1}{20}$ gr. were given hypodermically. The patient regained consciousness in two hours and was quite violent for a short time, then quieting down. The next day the urine was of the characteristic brown-black color sp. gr. 1005, for three days contained a few hyaline and granular casts; after this it was normal. The temperature remained slightly elevated for five days, 99 to 101.5. For two days there was some pain in the mouth, especially on swallowing. By the fifth day the mouth had cleared up, but the skin burn was still very sore, red and blistered. The patient was discharged well on the ninth day.

CASE 13.—Patient admitted 8 a. m. Sept. 14, 1905. Had taken carbolic acid $\frac{3}{4}$ i in water fifteen minutes before admission. Semi-conscious, cyanotic on admission. Burns on lips and tongue. Lavage with alcohol 10 per cent. Magnesia sulphate $\frac{3}{4}$ i, two eggs and milk by tube. Strychnin $\frac{1}{20}$ gr., digitalin $\frac{1}{25}$ gr. hypodermically. Temperature 100, pulse 98; next day pulse and temperature normal. Discharged well September 16.

An analysis of the preceding cases shows several interesting features. The local effect on the mouth is greater if the acid is taken pure or in water than if taken in alcohol, all ten cases of the first class showing local eschars, whereas two of the three of the latter class showed no burns whatever. All the cases were unconscious on admission, except two who had taken the drug after drinking whisky and one who had taken it in water. This includes the cases which had taken the poison in whisky and beer. The longest period of unconsciousness occurred in the patient who had taken the acid in beer. The average period of unconsciousness of the cases having taken the drug in alcohol was three and three-quarters hours and of those in which no such history was obtained was one and three-quarters hours.

The mortality record was low, only two of the thirteen cases having died, one of these from bronchopneumonia a few days afterward, probably due to an inhalation of material during the lavage, which was performed with difficulty, and the other having been admitted moribund after having been kept for two hours outside without treatment. The treatment in all the cases consisted in a most thorough lavage with 10 per cent. alcohol and in the later few cases this was followed by lavage with plain water. In eleven of the cases milk and eggs were introduced through the tube and left in the stomach. Sodium sulphate was administered through the tube in three cases and intravenously in three cases and magnesium sulphate in the stomach in four cases. Stimulation with strychnin and digitalin was resorted to in eight cases.

CASES TREATED WITHOUT ALCOHOL.

A study of the records of the fourteen cases of carbolic acid poisoning treated at the Lakeside Hospital previous to the introduction of the use of alcohol in treatment surprised the authors by their similarity to their own results. In this series of fourteen, all of which were treated by thorough lavage, either with plain water or a solution of magnesium sulphate, there were only three deaths, two of these due to pneumonia and the third a case admitted ten hours after taking two ounces of the acid, an emetic of magnesium sulphate having been resorted to, but no lavage performed. In this early series also the cases which had taken the poison in alcoholic beverages seemed to show a greater tendency to unconsciousness than those that had taken it pure or in water. In the ten cases in which the record was made as to the manner in which it was taken, five took it with alcohol and five either pure or in water. Of the five alcoholic cases four were unconscious and one semi-conscious, while of the five that had not used alcohol two were unconscious, one semi-conscious and two conscious.

CASES COLLECTED FROM THE LITERATURE.

The following cases have been collected from literature, and though many of the reports are too meager for any conclusions to be drawn from them, they are inserted that the reader may see the various ways in which alcohol has been used in carbolic acid poisoning in recent years. It will be noticed that they are all of isolated cases.

In these cases the following abbreviations will be used:

- S. and A. Sex and age.
- P. Amount of poison and how administered.
- T. Time elapsed before treatment.
- L. Local eschars.
- Sy. Symptoms.
- Tr. Treatment.
- C. Time elapsed before consciousness returned.
- A. After history.
- R. Results.

CASE 1.—Fraser,² 1895. S. female. R. phenol, 3i, in whisky. T. 15 min. L. none. Sy. coma. Tr. atropin hypo. lavage. C. 8 hours. A. vomited 24 hours. R. well.

CASE 2.—Buchanan,³ 1899. S. and A. female, 35. P. phenol, 3i. T. short. Tr. sodium sulphate hypodermic, lavage with 2 quarts of 35 per cent. alcohol. R. well.

CASE 3.—Adams,⁴ 1899. S. and A. female, 36. Tr. alcohol 1 pint, by mouth; alcohol, 3iv, and whisky, 3iv, q. 15 min. Six doses whisky, 3ii q. h. 8 doses. R. well.

CASE 4.—Kelly, 1899. A. 21 months. P. phenol, 3i. T. 6 min. Tr. olive oil. Alcohol, 3vi. Apomorphin, 1/30 gr. Whisky, 3i, q. 10 min. Eight doses. C. 30 min. R. well.

CASE 5.—Klein,⁵ 1900. S. and A. female, 29. P. phenol,

3ii. Tr. alcohol, 3iv, by mouth q. 1/2 hours, 4 doses, then 3i q. i hour, 4 doses. Morphia, 1/4 gr. R. well.

CASE 6.—Rodman,¹⁰ 1900. S. and A. female, 60. P. phenol, 3ii, pure. T. short. L. lips and tongue. Sy. coma, cyanosis, subnormal temperature. Tr. alcohol, 3iv, into pharynx, then lavage with warm water and then with dilute alcohol. A. temperature for 2 days, 101 to 102. R. well.

CASE 7.—Pinkney,⁹ 1901. S. and A. female, 3 1/2 years. P. phenol, 3iv. T. 1/2 hour. L. lips and chin. Sy. coma, cyanosis, thready pulse. Tr. alcohol, 3iiiss. Strychnin, hypodermic, 1/30 gr. Infusion normal saline, 1 pint. C. 7 hours. A. temperature rose rapidly to 106. R. death in 12 hours.

CASE 8.—Gries,¹¹ 1901. S. and A. female, 3. Sy. coma, weak pulse, cyanosis, appeared moribund. Tr. Hypodermic of ether. Magnesium sulphate, 1 1/2 quarts, by stomach tube, then lavage with 20 per cent. whisky and 3iv left in. Atropin, 1/200 gr. Strychnin, 1/40 gr., hypodermic. C. few hours. R. well.

CASE 9.—McDonald,¹² 1902. P. phenol, 3i. T. 25 min. Sy. collapse, coma. Tr. stomach tube, alcohol, 0i left in a few minutes and siphoned off. This repeated once. Then lavage with water, whisky, 3iv, nitroglycerin, 1/100 gr., strychnin, 1/20. C. six hours. R. well.

CASE 10.—King,¹³ 1902. S. and A. female, 33. P. phenol, 3ss. 3 min. Tr. alcohol, 0ss. Then lavage with water, repeated. Alcohol, 3iii, left in. C. 4 hours. R. well.

CASE 11.—Marshall,¹⁴ 1903. S. and A. male, 74. P. phenol, 3i, and olive oil, 3iv, per rectum. T. immediate. Sy. coma, pulseless. Tr. irrigation with 2 quarts of water 1 hour and 10 minutes. Alcohol and water, aa3iv, repeated and irrigated. C. 8 hours. R. well.

CASE 12.—Atwell,¹⁵ 1903. S. and A. female, 35. P. phenol, 3ss, in water. T. 20 min. Sy. coma, cyanosis, weak pulse. Tr. atropin, 1/50 gr., hypodermic. Dilute alcohol, 3vi. Lavage with water, gallon 1. Alcohol, 95 per cent., 3vi. Lavage in 5 minutes. Magnesium sulphate, 3i, in water 0i. Stimulation. Infusion normal saline. R. well.

These two facts shown by a study of our own cases, namely, the immaterial difference in the mortality, whether the stomach was washed with alcohol or with some other liquid, and the apparently greater constitutional symptoms when the drug was taken with alcohol then when taken clear or in water, seemed to throw such grave doubts on the antidotal power of alcohol to carbolic acid when taken internally, and these doubts agreeing so well with the opinions expressed in the recent textbooks on toxicology, the authors determined before publication to undertake some experimental work in the effort to prove or disprove the antidotal action.

3. EXPERIMENTAL DATA.

BY DRs. BROWN AND CLARKE.

Acting under the direction of Dr. Torald Sollmann, a series of animal experiments were carried on in the pharmacological laboratory of the Western Reserve University, the results of which are herewith published.

INTRODUCTION.

Some experimental investigations of the subject have been undertaken by others, but these are rather unsatisfactory; the number of animals used by each investigator seems rather too limited to make the conclusions binding.

In 1901 Thornton¹⁶ made some experiments on a series of five dogs, giving by stomach from one to four

10. Rodman: Med. Record, N. Y., 1900, vol. lviii, p. 70.

11. Gries: Therapeutic Gazette (Detroit), 1901, vol. xxv, p. 587.

12. McDonald: Fort Wayne Med. Jour. Mag., 1902, xxii, p. 59.

13. King: New Orleans Med. and Surg. Jour., 1902, vol. liv, p. 740.

14. Marshall: Med. Record N. Y., 1903, vol. lxiii, p. 174.

15. Atwell: Virginia Med. Semi-Monthly (Richmond), 1903, vol. viii, p. 73.

16. Thornton: Progressive Med., Dec. 1901, p. 343.

8. Adams: N. Y. Med. Jour., 1899, vol. lxx, p. 780.

9. Klein: Jour. Am. Med. Assoc., 1900, xxxv, p. 1557.

drams of phenol, undiluted, or with alcohol or oil. This was left in the stomach, together with the antidote. He gave to one animal phenol mixed with 95 per cent. alcohol, and after the onset of toxic symptoms administered more alcohol. The result was death in four hours, with no signs of improvement. Another animal was given phenol mixed with oil, which gave practically the same result. In the remaining animals phenol was administered and, after the poison symptoms had become manifest, alcohol in single or repeated doses was given without any beneficial effect.

The following year Wallace¹⁷ made observations on two rabbits and two cats. The first rabbit was given the lethal dose (0.6 gm. per kilo) subcutaneously, resulting in death in one hour and ten minutes. The second rabbit was given 5 cc. of 60 per cent. alcohol subcutaneously and this followed five minutes later by the same dose of phenol as was given the other rabbit. The toxic symptoms appear after the same interval of time as in the first rabbit. Five cc. doses of the diluted alcohol were injected at intervals, without improvement of the condition. The animal died in one and one-fourth hours.

The cats were given the lethal dose by stomach, one receiving the aqueous solution and the other receiving the phenol in 60 per cent. alcohol. Alcohol was later injected subcutaneously, but without effect. Both animals died after nine hours.

Fig. 1.—Intravenous injections of 1 c.c. per kilo of 1 per cent. phenol in normal saline at A, B and C. Time marks 15 seconds.

Method of Investigation.—Our experiments may be divided into three groups:

1. Systemic antagonism (intravenous injection).
2. Antagonism in the stomach (5 per cent. phenol).
3. Efficiency of lavage (undiluted phenol).

I. SYSTEMIC ANTAGONISM.

For the purpose of determining whether alcohol can lessen or abolish the systemic effects of phenol, a 1 per cent. solution was injected intravenously into dogs, the dose of 1 c.c. per kilogram (0.01 gm. of phenol) being repeated every minute. Three solutions of phenol were employed—one in normal saline solution, the second in 10 per cent. alcohol, and the third in 20 per cent. alcohol, the results produced by these solutions being compared.

Operative Methods.—All dogs used in these experiments were first given sufficient morphin to render them quiet and then kept under complete ether anesthesia during the course of the experiment. Before coming out of the anesthetic condition they were killed.

A cannula was placed in the trachea, through which the anesthetic (ether) was afterward given, and a cannula in the carotid for blood pressure tracings. The intravenous injections were made through a cannula placed in the femoral vein and connected with a graduated burette which contained the injection solutions.

(A) *Effects of Phenol in Normal Saline.* These will be described first for the purpose of comparison.

Blood pressure, and in some cases respiratory tracings, were taken. In all the animals the intravenous injection causes a fall of blood pressure which has a tendency toward recovery, depending on the quantity injected (Fig. 1.) Th. Gies¹⁸ obtained the same results in his experiment with animals. The average fall is from 20 to 30 mm. of mercury. The fall begins almost immediately after the injection; reaches the minimum in thirty seconds and returns to about normal after sixty seconds. Each successive injection becomes less effective, with a tendency toward a slow gradual fall of blood pressure. Where the lethal dose is injected there is a rapid fall which terminates by stoppage of the heart (Fig. 2.).

Respiration is but little affected except by the mechanical interference of the convulsive movements.

Onset of Convulsive Movements.—In all the animals employed the intravenous injection causes a gradual onset of convulsive movements beginning with a tremor of the legs and jaw, which gradually increases until the contractions acquire a clonic convulsive character. The absence of a rise of blood pressure which is usually associated with convulsions is probably due to the deep anesthesia and to the rather mild type of the convulsions.

(B) *Effects of Alcoholic Solutions of Phenol.*—

Fig. 2.—Death from intravenous injection of phenol. Injection made at A.

These do not differ from those of the saline solution of phenol in either quality or quantity. This is shown by Table 1.

TABLE 1.—INTRAVENOUS INJECTION. (Dog).

Dog. No.	Solvent.	Fall of Blood Pr. after first injection.	Twitching movements after c.c. per kilo.
1	Normal saline...	30 mm.	5 c.c.
4	Normal saline...	40 mm.	5 c.c.
2	10 p. c. alcohol..	24 mm.	3 c.c.
3	20 p. c. alcohol..	40 mm.	5 c.c.

II. ANTAGONISM IN THE STOMACH.

To determine the possibility of an antagonism in the stomach, the phenol was administered by stomach tube to rabbits. The just fatal dose (0.6 gm. per kilo.) was given, as a 5 per cent. solution in water, and in 10 per cent. alcohol and in 10 per cent. glycerin. These animals were not anesthetized.

(A) *Effects of the Watery Solution.*—The first symptoms start in one to three minutes and consist in a general trembling of the body and legs. The legs grow weaker and the animal falls on its side. Convulsive movements of a clonic type then appear, varying somewhat in intensity; the legs may become extended and rigid, while in some cases the head is thrown backward. Death occurs in from twenty-six minutes to six days.

The alcoholic and glycerin solutions produced the same effect quantitatively and qualitatively. This is shown in Tables 2 and 3.

17. Wallace: N. Y. University Bul. of the Med. Sci., 1902, vol. ii, p. 58.

18. Gies: The Archiv. für Exp. Path. and Pharm., 1880, vol. xii p. 401.

TABLE 2.—DETAILS OF STOMACH EXPERIMENTS ON RABBITS.

Rabbit No.	Weight in Gms.	Amount of Phenol Given (always 0.6 gm. per kilo.)	Diluent used.	Time Elapsed Before Appearance of Convulsive Movements.	Final Result.
1	920	11	Water.	1 m. 45 sec.	Died after 28 min.
7	770	..	Water.		Tube passed into trachea.
8	490	5.5	Water.	1 m. 30 sec.	Died after 25 min.
9	980	11.7	Water.	1 m. 30 sec.	Died after 6 days.
10	770	9.2	Water.	2 m. 10 sec.	Died after 8 hrs.
3	870	10.4	10 p. c. alc.	1 m. 50 sec.	Died after 24 hrs.
5	920	11	10 p. c. alc.	6 m. 30 sec.	Died after 3 days.
12	730	8.7	10 p. c. alc.	3 m. 15 sec.	Died after 18 min.
13	670	8	10 p. c. alc.	2 m. 30 sec.	Died after 2½ hrs.
2	830	10	10 p. c. glyc.	1 m. 40 sec.	Died after 3½ m.
4	1450	17.5	10 p. c. glyc.	2 m. 45 sec.	Survived.
6	1200	14.4	10 p. c. glyc.	3 m. 30 sec.	Survived.
11	780	9.3	10 p. c. glyc.	2 m. 10 sec.	Died after 2 days.

* It was thought some of the solution was aspirated into the trachea in this animal.

TABLE NO. 3.

TIME OF DEATH IN STOMACH EXPERIMENTS. (Rabbits.)

Phenol dissolved in

Water. 25 minutes . . . 28 minutes . . . 8 hours 6 days
 Alcohol. 18 minutes . . . 2½ hours . . . 24 hours 3 days
 Glycerin. 3½ minutes . . . 2 days Survived Survived.

Microscopical Examination of the Stomachs.—This was undertaken by Dr. W. W. Williams in rabbits 1, 2, 3, 8 and 12. The findings may be arranged, according to the severity of the lesions, as follows:

Rabbit No.	Lesion.	Medium in Which Phenol was Administered.	Time Elapsing Before Death.
1	None	Water.	28 minutes.
2	Very slight	Water.	25 minutes.
3	Fairly marked	Glycerin.	3½ minutes.
8	Very severe	Alcohol.	24 hours.
12	Very severe chronic gastritis (probably not due to the drug).	Alcohol.	18 minutes.

It is seen that the local lesions are quite as severe with the glycerin or alcoholic solution as they are with the watery solution.

From these results it is evident that the alcoholic solution is no less toxic than the aqueous solution; while

washings returned clear. One c.c. per kilo. of 95 per cent. phenol was then introduced through the tube and allowed to remain for ten minutes, when lavage was started, either with water or with 10 per cent. alcohol.

(A) *Effects of Concentrated Phenol by Stomach Without Lavage.*—This was tested on one animal. This showed an immediate fall of blood pressure amounting to 40 mm. of mercury. The pressure remains about constant after this primary fall, with a tendency toward a slight gradual fall, which continues until death. At no time during the experiment was there any evidence of improvement in the condition.

(B) *Lavage.*—In those animals in which lavage is employed, the blood pressure begins to rise after an interval of time which varies with different animals. The convulsive movements cease and the animals show all signs of recovery, the condition being judged by the blood pressure and convulsive movements.

The results are shown in Figure 3. They are identical for all the experiments, whether water or alcohol was used for lavage.

The details of the experiments are shown in Table 4.

4. THE LOCAL ANTAGONISM OF CARBOLIC ACID AND ALCOHOL, AND OTHER PHENOL—SOLVENTS.¹⁹

It will be recalled that the employment of alcohol in phenol poisoning was originally based on the local antagonism of the two drugs. This antagonism, as is well known, can be easily demonstrated, by dipping the finger in carbolic acid and then washing it at once with strong alcohol. To secure a complete removal of the phenol effects, however, it is essential that the application of the phenol should be of short duration, and followed very quickly by the alcohol. This is illustrated by the experiments of Wallace.¹⁷ For instance, when the alcohol was used immediately, it removed all the effects completely; when it was used after ten minutes it removed the superficial whitening of the skin, but not the deep effects. When it was used after thirty minutes it had almost no effect. The addition of a small quantity of alcohol to the phenol increases its local effects—an obser-

TABLE NO. 4.
EXPERIMENTS ON STOMACH OF DOGS.

Dog Number.	Weight, (Kilo.)	Solvent Used.	Time in Stomach.	Initial B'd Pressure.	Blood Pressure 10 m. After Giving.	Blood Pressure Remains at Low Level.	Blood Pressure at End of Experiment.	Convulsions Ceased.	Duration of Experiment.	Result.	REMARKS.
10	14.	(Control)		106	72	Until death..		At death..	2 hrs., 45m.	Died.....	Died.
14	4	Water...	10m	102	37	1 hr., 27 m...	102	2 hours....	4 hrs., 12m.	Recovered.	Animal killed.
12	3.6	Water...	9m	103	43	30 minutes...	96	1 hr., 20m.	2 hours....	"	Died from ether.
12	8	Alcohol...	10m	98	60	34 minutes...	60	1 hr., 40m.	4 hrs., 25m.	"	Animal killed.
12	6	Alcohol...	10m	101	30	15 minutes...	100	3 hrs., 10m.	4 hrs., 29m.	"	Died from ether.
12	3	Alcohol...	10m	89	30	Until death..	20	At death..	46 minutes.	Died.....	Tube did not enter stomach in washing.

the glycerin solution appears to be rather less fatal than the others, since two of the animals recovered completely. This might be due to slower absorption; for we are loathe to attribute it to any antidotal properties, either chemical or physiologic.

III. EFFICIENCY OF LAVAGE.

To determine the effect of lavage after the administration of concentrated phenol, and the relative value of water and diluted alcohol for this purpose, we employed dogs, anesthetized with morphin and ether, and connected for blood-pressure tracings. A stomach-tube was inserted and the stomach washed with water until the

variation which effectually refutes the theory that alcohol and carbolic acid enter into an innocuous chemical combination. The increased effect is to be explained by a more rapid absorption of the slightly diluted acid.

Wallace attributes the antagonism to the solvent action of alcohol on the phenol which would remove it much more readily from the superficial layers of the skin than would water. If the theory be correct, other phenol solvents must produce the same effect as alcohol. Wallace tested this with chloroform, ether and benzol, with positive results.

¹⁹ This chapter was kindly furnished by Prof. T. Sollmann, by whom the experiments were performed.

We made the following experiments, using somewhat different solvents:²⁰

EXPERIMENT 1.—(a) The finger tips are plunged for thirty seconds into liquefied (95 per cent.) phenol, then for five minutes into the solvents. At the end of this time the finger in water is blanched, the fingers in 50 per cent. alcohol, 50 per cent. glycerin, ether and cottonseed oil appear normal. (b) The fingers are again dipped in the carbolic acid for one minute, then for one minute in the solvents.

The blanching is very conspicuous in water; then follows the 50 per cent. glycerin, then the 50 per cent. alcohol and ether.

The sensory effects are greatest in water, ether and 50 per cent. alcohol; less in 50 per cent. glycerin and oil.

EXPERIMENT 2.—The fingers are dipped into liquid carbolic acid for one and one-half minutes, then into solvents and observed at end of (a) one minute, (b) two and three minutes.

(a) One minute in solvents: The blanching is by far the most conspicuous in water; then follows the 25 per cent. glycerin and 25 per cent. alcohol. Turpentine and cottonseed

effect is mainly due to solution and is not chemical. The solvents also tend to lessen the burning sensation, probably by removal of the phenol. They appear to be quite unable to prevent the late effects of the phenol, which may be attributed to the phenol which had already penetrated deeply when the solvent was applied.

It appeared interesting to determine also whether these solvents exert any effect when the carbolic acid is left in contact with the skin. For this purpose 5 per cent. solutions of phenol were made in various solvents. These were arranged in five small beakers, so that all the fingers of the left hand could be inserted and withdrawn simultaneously. The degree of blanching, tingling and anesthesia was compared after five minutes. The tingling sensation proved to be too irregular to be of any value for comparison. As in the previous series, the nature of the solvents were unknown to the experimenter and compiler until the conclusions had been drawn.

Fig. 3.—One c.c. per kilo of 95 per cent. phenol introduced into the stomach at A, time 10:25. Began lavage at B, time, 10:35, finished lavage at C. Blood pressure at end of experiment 100, time, 2:55. Base line corresponds to 0 pressure.

oil appear normal. Burning is felt in the water, and in the glycerin and alcohol solutions, not in turpentine and oil. The water finger also shows the greatest anesthesia.

(b) Two or three minutes in solvents: The blanching is most pronounced in the water; then follows 25 per cent. glycerin, then 25 per cent. alcohol. None is seen in turpentine and oil.

(c) The finger which had been put in water and which was still very much whitened, is rinsed in 95 per cent. alcohol. The blanching disappears almost completely at once.

(d) On the following day the skin of the fingers is rough and broken, and this condition persists for several days. It is alike in all the fingers.

It may be concluded that all phenol solvents arrest the local action of the phenol, i. e., the blanching action.

This and the observation that dilution of the alcohol greatly impairs its efficiency sufficiently show that the

EXPERIMENT 3.—Comparison of 5 per cent. phenol in water (W), 10 per cent. alcohol (A), 10 per cent. glycerin (G), and cottonseed oil (O).

Experiments on two persons gave identical results:

The anesthesia was most conspicuous in W; somewhat less in A, absent in G and O.

The blanching was marked and about equal in W and A, almost absent in G, quite absent in O.

EXPERIMENT 4.—Comparison of 5 per cent. phenol in water, 25 per cent. alcohol, 25 per cent. glycerin, turpentine and cottonseed oil. The experiment was repeated on nine persons, with the following results:

Anesthesia.	Most Marked.	Less Marked.	Absent.
Water.....	cases.....	1 case.....	1 case.
Alcohol.....	2 cases.....	3 cases.....	4 cases.
Glycerin.....	None.....	1 case.....	8 cases.
Turpentine & Oil..	None.....	None.....	9 cases.

20. In all the experiments the subject was not aware of the nature of the solvent, the containers being numbered and the results tabulated by these numbers.

BLANCHING

	Most Marked.	Second Place.	Third Place.	Fourth Place.	Absent.
Water	9 cases . . .	None	None	None	None.
Alcohol	None	5 cases . . .	2 cases . . .	None	2 cases.
Glycerin	None	3 cases . . .	3 cases . . .	None	3 cases.
Turpentine	None	None	2 cases . . .	3 cases . . .	4 cases.
Oil	None	None	None	None	9 cases.

TINGLING.

	Most Marked.	Second Place.	Third Place.	Absent.
Water	1 case	None	None	8 cases
Alcohol	3 cases	None	None	6 cases
Glycerin	None	1 case	None	8 cases
Turpentine	None	None	None	9 cases
Oil	1 case	None	1 case	7 cases

These results show that the addition of all the solvents diminishes the local effects of phenol. The effect on the tingling sensation is, however, irregular. This may be attributed to the difficulty of exact observation. The effect is more pronounced on the superficial action (blanching) than on the deeper actions (anesthesia). Oil alone prevents both actions in all cases. Then come, in order of efficiency, turpentine, 25 per cent. glycerin, and 25 per cent. alcohol. Glycerin is more effective, for the same degree of dilution, than is alcohol.

In this experimental arrangement the effect of the solvents can not be explained, as in the previous series, by removal of the phenol. Nevertheless, it seems to be connected with their solvent power. It may be explained in the following manner: If a substance is introduced into a mixture of two immiscible solvents, a and b, it will be distributed between these two solvents in a ratio proportional to its solubility in a and b. If it is more soluble in a, this will contain a greater amount. This may be demonstrated by shaking a 1 per cent. solution with an equal volume of olive oil in one test tube, and with an equal volume of petroleum benzin in another. If a little ferric chlorid solution is added to both test tubes, the watery layer will not be colored in the oil tube (the phenol having passed into the better solvent), while it will be colored a deep violet in the benzin tube. The benzin being a poor solvent, almost all of the phenol has remained in the water.

In our experiment, the fluids of the tissues constitute one solvent, the fluid in which the carbolic acid is applied the other. The better the solvent power of our solvent, the less of the carbolic acid will pass into the tissues.

In the case of alcohol, and to a lesser extent with glycerin solutions, some of the solvent is doubtless absorbed into the tissues, which explains the relative inferiority of these to turpentine, and especially to oil, which are absorbed much more slowly.

These experiments contradict the rather prevalent opinion that the absorption of phenol is quickened by alcohol and perhaps also by glycerin. As far as we know, this opinion is purely theoretical. It may be true as far as the addition of small quantities of these substances are concerned, which would then act, not so much as solvents, but by increasing the fluidity of the phenol—a very important factor in absorption. It might theoretically hold also for the alimentary canal, since these solvents are absorbed there with considerable rapidity. This view is supported by the clinical experiences, although our experiments on rabbits (Series 2) can scarcely be interpreted in its favor. As concerns the skin, the experimental results constitute a positive refutation of the quicker absorption of alcoholic solutions in this situation.

The relative inferiority of alcohol to the other solvents is not sufficient to be of importance; while its greater fluidity and its miscibility with water constitute practical advantages more than sufficient to counterbalance the theoretical deficiencies. In emergencies, however, the value of the other solvents, particularly oil, should be borne in mind. It is interesting to note that the use of the latter antidote preceded that of alcohol.

GENERAL CONCLUSIONS.

From the above experiments the following conclusions may be drawn:

1. Alcohol has a local antidotal effect to carbolic-acid burns, due to its solvent action.

2. There is no evidence of chemical antagonism between alcohol and phenol.

3. There is no effect produced by alcohol on carbolic-acid poisoning after the latter has been absorbed into the system, as is shown by the intravenous experiments on dogs.

4. Alcohol and phenol placed in the stomach give no different results from phenol alone.

5. Lavage with alcohol is effective when the phenol is in the stomach, but its superiority over lavage with water is not pronounced.

From the clinical aspect it appears that alcohol has a local antagonism to carbolic acid. When the latter is taken in whisky the feeling of discomfort is less marked, and the local eschar is absent or but slightly evident. In cases of poisoning, lavage with alcohol is apparently an effective method of treatment. This is borne out by a comparison of thirteen cases here reported with a mortality of 15 per cent., with the collection made by Falck²¹ in 1868 of forty-six cases in which the acid was taken internally with thirty-five deaths, a mortality of 65 per cent. On the other hand, the fourteen cases treated at the Lakeside Hospital by lavage prior to the use of alcohol with three deaths, a mortality of 21 per cent., give results so nearly akin to the alcohol cases that it would appear that the lavage were the dominant factor rather than the alcohol. When it is considered that it was only in the early nineties that stomach washing came into general use, and even later that the fear of its use in cases of corrosive poisoning was overcome, it seems probable that the good results in carbolic-acid poisoning from the use of alcohol, during the past few years, were due to the lavage and not to any antidotal property of the alcohol.

The authors do not wish to be interpreted as considering that alcohol is of no value in cases of carbolic-acid poisoning. *A priori* its solvent action and a slight tendency in the experiments with phenol in the dog's stomach, to an earlier rise in blood pressure, when the stomach is washed with an alcoholic solution, than when water is used, point to its value as a means of removing the poison from the stomach. From abundant clinical evidence, both in their own experience and in literature, it would appear to them to be an aid, when properly used, in the saving of life. On the other hand, this same solvent property, and the more marked symptoms of intoxication, when the poison has been taken in whisky, seem to establish a contradiction to the alcohol being used as an antidote and left in the stomach.

The procedure recommended is immediate, abundant lavage with 10 per cent. alcohol, this to be followed by lavage with plain water, and stimulation as indicated. The point to be borne in mind is that alcohol is not effective after the carbolic acid has been absorbed and, to

21. Falck. Ref. Blyth. Poisons. Their Effects and Detection. Wood's Library, 1885, p. 146.

be of value, must be used while the poison is still in the stomach.

The other common methods of treatment of phenol poisoning are now being investigated, and the results will be communicated later.

The fact that three of the five fatal cases in the Lakeside Hospital series died of pneumonia, and one, at least, of these of a definite bronchopneumonia of the inhalation type, has impressed the authors with the necessity of care in the method of performing lavage in this class of cases. With the patient lying on his back, in a comatose condition, with the gastric contents regurgitating around the outside of the stomach tube, it is a simple matter for material to enter the larynx and set up an inflammatory process in the lung. To overcome this danger, the authors recommend, immediately after the tube is passed, to turn the patient on his side or face, with the head low, thus allowing any fluid in the pharynx to escape through the mouth. Since this method has been used, there have been but few pulmonary signs during convalescence.

For his kindness in directing the work and for valuable suggestions and criticism, the authors wish to express their sincere thanks to Dr. Torald Sollmann.

TESTS FOR ACETONE IN THE URINE.*

ALONZO ENGLEBERT TAYLOR.

SAN FRANCISCO.

The interest of the surgical section of the medical profession is just now deeply concerned with the question of acetonuria. This interest has been awakened by observations of the frequent occurrence of the urinary acetone-complex following anesthesia and associated with a toxicosis that has been fatal in a number of instances. That acetone occurs frequently following anesthesia has been long known, and that it frequently occurs following the ingestion of many narcotics is equally well known. That the toxicosis is the result of the acetone-complex, that it is, in other words, an acid intoxication, is not demonstrated. On the contrary, it is much more probable that the toxicosis and the acetone-complex are fellow results of a widespread autolytic degeneration, of a ferment intoxication that has been in some unknown way inaugurated by the anesthetic or narcotic. It is known that many poisons act, in large part, by the inauguration of such autolyses, and this observation holds as well for narcotics as for phosphorus. At present acetone is held to be always derived in the body from the fats through the intermediary stages of b-oxy-butyric and diacetic acids, but it can not be denied that it might have other derivations. Chemically, acetone might be easily derived from glycerin through the intermediary stage of di-oxy-acetone, and other reactions that are feasible from the chemical point of view will be easily recalled. For the anesthetics, a second element enters into the discussion. The chemical relations of chloroform and ether as so close to acetone that it is probable that a certain amount of acetone may be formed in the body from these substances. We have to deal, therefore, with a derivation of acetone from the fats of the body under the influence of a toxic autolysis; and a derivation of acetone of purely chemical nature from the anesthetic. Whether or not acetone occurs in the urine following every administration of chloroform is not known; in the published researches reliable meth-

ods have not been used. Certain it is that acetonuria is now being diagnosticated by unreliable tests.

For several years I have tested nearly all the urines that have come under my notice with the different tests for acetone, and have also had a large class experience with mixed urines. These experiences have taught us that the commonly used tests for acetone are quite valueless, in that they yield positive results with urines free of acetone in the practical sense. There are two really good tests for acetone, those of Stock and of Denigès. Both these tests have been repeatedly recommended for clinical use, but for some inexplicable reason they have not been employed to any extent by clinicians. The test of Stock is a particularly reliable one, because it is a test for ketones and is not given by alcohols and aldehyds. It is very simple and rapid in execution. One must use for it the distillate of the urine. This, it must be insisted on, must be the rule for all tests for acetone. In classroom work we use from 50 to 100 c.c. of urine, which is made acid by the addition of a few drops of either acetic, hydrochloric or sulphuric acids. The first 10 c.c. of distillate will contain nearly all the possible acetone. For the use of the older tests for acetone (the iodoform, the mercuric oxid and the sodium nitroprussid tests), the distillate must be subjected to a second distillation; for the Stock and Denigès tests, however, this is not necessary. About an inch of the distillate is placed in a test-tube, a drop or two of a 10 per cent. solution of hydroxylamin hydrochlorid added (shaken), then a drop or two of a 5 per cent. solution of sodium hydroxid (shaken), then a couple of drops of pyridin (shaken), then about an inch of ether (shaken), bromin water is then added, drop by drop, with mixing until the ether layer becomes yellow, and then a few drops of strong hydrogen peroxid are added; if acetone be present, the ether will turn a distinctive green-blue. The test sounds a little formidable, but when the reagents are ranged in a row of dropping bottles, it is very simple. The test is not very delicate; for example, I have never seen a positive test in a normal urine of a person on a diet of not over 100 grams of fat *per diem*, though we know that all urines contain a faint trace of acetone. The test is very elective, and for practical work the lack of a greater sensitiveness is an advantage. The test of Denigès is quite as easily carried out. About an inch of the distillate in a test-tube is mixed with an equal amount of a solution of the subsulphate of mercury (mercuric oxid 50, sulphuric acid 200, water up to 1,000) and the mixture allowed to simmer (best stoppered) for about five minutes. A white crystalline precipitation occurs on cooling, which is very distinctive in appearance. If acetone be present in excess, the test is less well given. If but a trace be present, a trace of sodium chlorid will aid the precipitation, but it must not be added in excess. This precipitate is not soluble in dilute hydrochloric acid.

These two tests agree. I have never seen the Stock test present without finding the Denigès test positive. In a few instances I have obtained faint reactions with the Denigès test when the Stock test was negative. Equally distinctive is the Bambergér test: the formation of crystals of p-nitro-phenyl-hydrazone-acetone on heating the distillate, acidulated with acetic acid, with p-nitro-phenyl-hydrazin. It is not a convenient test, however, since the crystals must be recrystallized from alcohol and their melting point determined.

It is a yearly experience in our classroom work that, with the mixed urine of the students, the iodoform reac-

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