

CAUTION AGAINST THE USE OF NON-NEUTRALIZED (ACID)
SOLUTIONS OF SALVARSAN

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A recent experience which has been brought to my attention prompts this note of warning against the advent or inadvertent use of non-neutralized solutions of salvarsan. A physician administered to two patients, respectively, 0.4 gm. of arsenobenzol in 30 c.c. of water, *but failed to neutralize by the addition of sodium hydroxid solution*. Both patients developed alarming symptoms. The first patient complained of pain in the epigastrium, in the substernal region and in the left shoulder; later she had abdominal pains with nausea and vomiting. An irritating cough began which at the end of five days had not disappeared. The vein in which the solution had been introduced began to thrombose, and was thickened and painful for a length of 6 inches.

Immediately after the injection, the second patient developed cough and pain in the left shoulder, and the substernal and precordial regions. There was difficulty in breathing, and altogether the symptoms were alarming.

It now appears that these patients will recover, although a fatal termination might readily have taken place. It will be recalled that Fraenkel and Grouven, in the early days of the use of salvarsan, gave a patient 0.4 gm. in 15 c.c. of water; to this was added from 1 to 1.5 c.c. of a decinormal solution of sodium hydroxid. Despite this addition, the solution was still strongly acid. The patient died in three and one-half hours. The toxicity of acid solutions of salvarsan is lessened if they are given in great dilution. A year ago, a physician informed me that, acting under misapprehension of the instructions, he had administered arsenobenzol to two patients without neutralizing the solution; no reactive symptoms whatsoever developed. In this instance, however, from 100 to 150 c.c. of water were used. A few years ago, Prof. H. E. Hering of Prague called attention to the greatly increased toxicity of acid solutions of salvarsan as compared with alkaline solutions.

Dr. John A. Kolmer, Dr. George W. Râiziss and I, in a series of experiments, the publication of which is now in press, have shown that acid solutions of salvarsan administered intravenously in rats increases the toxicity from 50 to 60 per cent. This is true even in such concentration of the drug as is represented by from 0.5 to 1 per cent. solutions, the equivalent of 0.6 gm. in from 60 to 100 c.c. of water. The toxicity of acid solutions increases proportionately to the concentration of the solution. Max Joseph says that acid solutions produce a precipitate in the blood which may actually be seen in the lungs and in the right ventricle of experimental animals. The cough, which the patient referred to above developed, was doubtless due to the production of small emboli in the capillaries of the lungs.

It would seem that some physicians have confounded some of the salvarsan compounds which have been marketed under various trade names with neosalvarsan, and therefore have failed to neutralize the solution, an error which might have jeopardized the life of the patient.

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ESOPHAGEAL STRICTURE

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J. S., boy, aged 11 years, drank about an ounce of lye late in February, 1916. He was given the usual demulcent and sedative at the time, placed on a bland restricted diet, and made what appeared to be an uneventful recovery. When able to eat almost anything he wished and seemingly in excellent health he was sent to his grandmother's in another town, and I did not see him again until July. One evening in the latter part of the month his mother carried him into my office in her arms. He looked almost moribund. He was nothing but skin and bones. His mother told me that he had eaten nothing in two weeks, and that during the last few days he could get but little water down. He complained incessantly of thirst, so I offered him water; but it would go down a certain distance and then be regurgitated. I tried

to pass a small catheter bougie in order to relieve his immediate craving, but without success.

He was removed to the hospital immediately, and during preparation for operation was given water by bowel and venoclysis. Under ether the abdomen was opened through the left rectus incision, the stomach was drawn into the wound and incised, and a long rubber tube inserted. Owing to the patient's condition, it was impossible to attempt any operative procedure on the esophageal stricture, so he was returned to bed.

He improved steadily and was fed by tube: He was given liquids at first and then soft food. Finally he chewed solid food and spat in into the tube. Two weeks after the operation we began gradually dilating the stricture by means of bougies by mouth, and after a few days permitted the patient to take liquids and later soft foods in small quantities. It was not long until he took the regular hospital diet. As he swallowed it easily, the tube was removed and the fistula allowed to heal.

We kept the patient under observation two weeks longer, keeping the stricture dilated at regular intervals. I had in mind to discharge him one day when the nurse reported to me that he was unable to swallow and seemingly as much distressed as before the operation. I saw him at lunch. All food was regurgitated and a bougie could not be passed beyond the stricture area. It occurred to me at the time that we were dealing with a spasmodic condition superimposed on the true stricture. The patient was given a few drops of chloroform, and an unsuccessful attempt was made to pass a bougie; on regaining consciousness, he was still unable to swallow.

Preparations were then made to reinsert the gastric tube.

While I was in another part of the institution, the nurse in my absence insisted on his attempting to swallow some liquid, which made him vomit, and he threw up a wad of chewing gum, the size of a small marble, and about as hard. I recalled to my chagrin that, against my better judgment, I had permitted him the privilege of chewing gum a few days before. He was entirely relieved and could swallow as well as ever. He was discharged a few days later, and up until the middle of November had remained well. The hard mass of gum had been stopped at the strictured area, where it acted as a perfect ball valve.

A NEW OCULAR MUSCLE SYMPTOM IN EXOPHTHALMIC
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In the examination of a number of exophthalmic goiter patients for the purpose of determining the relative frequency of the various eye symptoms and signs, this peculiar action of the lateral eye muscles was noted in many:

After extreme lateral rotation of the eyes, either to the right or to the left, with the head fixed and with fixation of an object at this point maintained for a second or two, on attempting to follow this fixation point as it is rapidly swung into the median line, one of the eyes—it may be either—fails to follow the other in a complementary manner into proper convergence and fixation for this point when it is brought into the median plane. Either the right or the left eye makes a sudden rotation into the fixation with its fellow, but before it does so, an apparent divergent strabismus is manifest.

A muscle balance for esophoria or exophoria was taken in each case. In those in which there was an exophoria of greater or lesser degree, this apparent divergence was more marked than in those cases in which there was no exophoria. This apparent divergence was not as marked in cases in which an esophoria of any appreciable degree was present.

For example, if after extreme rotation to the right and fixation of an object in this position, the object is rather rapidly brought into the median horizontal plane, the left eye follows the object properly, but the right lags behind in the corresponding rotation into median plane fixation, but it eventually jumps, as it were, into proper convergence and fixation with the left eye.