

AN ARGYLL ROBERTSON PUPIL BECOMING NORMAL
AFTER MERCURY AND SALVARSAN

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H. W., aged 32, married eight years, whose child, aged 3, and wife show no evidence of syphilitic lesions, had gonorrhea years ago, but does not know of ever having had a chancre or initial lesion.

I saw him July 30, 1912. He consulted his physician then on account of difficulty in walking and cramps in the calves of his legs. Examination at that time revealed complete loss of patellar reflexes; Argyll Robertson pupil in both eyes (no reaction to light); cramps in the calves; gastric and intestinal crises; marked Romberg sign; testicular reflex increased so that stroking the skin on the inside of thigh caused the testicle to be drawn up into the canal; muscle sense disturbed; gait markedly affected; Wassermann reaction positive.

The treatment consisted of mercurial inunctions and potassium iodid until August 20. He was then given 0.6 gm. of salvarsan in the right gluteal region. A marked induration at the site of the injection has persisted up to the present (December 20). November 4 he was given an intragluteal injection of salvarsan, 0.7 gm., on the left side. This was followed by an induration which remains now, but is not so marked as that following the first injection.

December 9: Examination shows: patellar reflex unchanged; Argyll Robertson pupil gone (both eyes show an immediate response to light). The same condition exists now (December 20). The Romberg sign is not nearly so marked as before treatment. While formerly the patient had to steady himself, especially in washing his face, he now experiences very little difficulty, and the gait is much improved. There has been a gain in weight of 5 pounds. He feels better than he has in years and is surer of his movements.

The restoration of the pupillary reflex I take as justification for reporting in detail a case of tabes dorsalis, as this is the first time I have seen or read of this happening after the administration of mercury or salvarsan.

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SPORADIC CEREBROSPINAL MENINGITIS; RECOVERY

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Nov. 3, 1912, I saw in consultation with Dr. F. E. Hill of this city Paul W., aged 12. The patient was comatose and had a high temperature; the neck and back muscles were stiff, and Kernig's sign was well marked. The pupils were dilated and the eyes staring. Fearing cerebrospinal meningitis I made a spinal puncture within an hour and withdrew 50 c.c. of milky spinal fluid, and, without withdrawing the exploring needle, I injected into the canal 30 c.c. of Flexner's serum.

As soon as my son, Dr. E. S. Green, could reach our laboratory he made a careful microscopic examination and found the fluid loaded with the *Diplococcus intracellularis meningitidis*.

In twenty-four hours I made a second puncture and again allowed 50 c.c. of spinal fluid to flow out and again injected 30 c.c. of Flexner's serum. Again Dr. E. S. Green made careful slides and found that the diplococcus had partially disappeared and we could see macroscopically that the spleen fluid was not so thick or cloudy, and that leukocytes were very numerous. The muscular contractions were not so firm, and the boy, while still unconscious, was easier to handle and would voluntarily change his position, which he could not do before on account of opisthotonos.

Thirty-six hours later I made the third and last puncture, again withdrawing 50 c.c. of fluid and injecting 30 c.c. of serum, making in all 90 c.c. of serum. On this occasion the boy was held for thirty minutes by his father in an incline of 45 degrees head downward, in order that he might receive some benefit from gravity.

At the third puncture the spinal fluid was clear and seemed normal both to the eye and under the microscope. On this day the boy had two severe convulsions and on the next morning awoke, asked for food, inquired what was the matter, and promptly recovered.

I believe this case valuable because every step was verified by the microscope, and the rapidity of the disappearance of the meningococcus was carefully noted. The series of slides (three of each puncture) was preserved.

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THE EMETIN TREATMENT OF AMEBIC DYSENTERY

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For a number of years English¹ physicians working in India have maintained that ipecac is the best remedy for amebic dysentery, and within the past few years the American profession has largely come to agree with them.² In March, 1911, Vedder,³ working in Manila, published his findings that a fluid extract of ipecac would kill amebas in cultures in dilutions as high as 1:200,000. The next year Rogers⁴ in Calcutta found that emetin hydrochlorid killed amebas in stools in dilutions of 1:100,000 and began the use of this salt hypodermically in cases of amebic dysentery. His brilliant results led me to try this therapy, and as I have seen so far no confirmation of his work, I submit the following cases:

CASE 1.—White man, aged 51, farmer, contracted amebic dysentery in Alabama in October, 1912. I saw the case in consultation with Dr. L. W. Hunter, Dec. 20, 1912. At that time the man was in bed, having about six stools daily, with mucus and tenesmus, but no gross blood. There was irregular evening fever, at times reaching 103. Blood examination: hemoglobin 65 per cent., white count 6,000; differential count showed polynuclears 73 per cent., large mononuclears 5 per cent., small mononuclears 16 per cent., eosinophils 6 per cent. Feces were liquid, containing mucus, considerable blood microscopically, and many amebas engorged with red blood-cells. Physical examination showed considerable tenderness over the colon from the cecum to the splenic flexure.

December 21: Admitted to the hospital and given 0.8 grain of emetin hydrochlorid hypodermically. Patient had four stools before admission.

December 22: No nausea from emetin; two stools since yesterday; examination of a fresh stool failed to show amebas; blood and mucus still present; patient given 1.4 grains of emetin.

December 23: Slight nausea last night; two stools, without either blood or amebas; mucus still present; patient given emetin, 1.4 grains.

December 24: One normal stool to-day, the first in five weeks; no blood, mucus, or amebas; no tenesmus; tenderness over the colon improving rapidly; temperature subnormal since the first dose of emetin. The patient was dismissed, December 30, apparently well.

CASE 2.—Male negro, aged 22, referred to me Sept. 7, 1912, by Dr. J. Q. Myers, contracted dysentery in the spring of 1910. He improved the following winter, but in the summer of 1911 had dysentery off and on, and had it steadily during the summer of 1912, with as many as thirteen bloody, mucous, painful stools a day. Hemoglobin 80 per cent.; differential count showed polynuclears 74 per cent., small mononuclears 15 per cent., large mononuclears 3 per cent., eosinophils 8 per cent. Feces showed blood, mucus and amebas containing red blood-cells. Physical examination was negative except for tenderness over descending colon, which was thickened. Patient was given 50-grain doses of ipecac by mouth and delayed vomiting and purging resulted, but the amebas remained. After several weeks another course of salol-coated ipecac pills was instituted, but the amebas did not disappear.

November 2: Given 0.6 grain emetin hydrobromid hypodermically, and after three days the dose had been increased to 1.3 grains. The salt was dissolved in 3 c.c. of water per grain,

1. Manson, Sir P.: Tropical Diseases, William Wood & Co. Rogers, L.: Fevers in the Tropics, Oxford Medical Publications.

2. Brem, W. V.: Am. Jour. Med. Sc., 1910, cxi, 669. Simon, S. K.: New Orleans Med. and Surg. Jour., 1912, lxxv, No. 5, etc.

3. Vedder, E. B.: Bull. Manila Med. Soc., March, 1911.

4. Rogers, L.: Brit. Med. Jour., June 22 and Aug. 24, 1912; Therap. Gaz., 1912, xxxvi, 837.