treated at the isolation hospital on Swinburne Island, with thirteen deaths. Two cases occurred among released passengers in New York City and state, and one member of a ship's crew. One fatal case occurred among the quarantine employees. Thirty-one cholera carriers were detected by routine examinations; these were isolated and treated as cholera patients. Four of these gave definite histories of a recent sickness with diarrhœa. During the four months of routine examination of passengers from infected ports for cholera carriers, 26,678 persons were bacteriologically examined.

Over one hundred non-cholera vibrios were isolated during the routine examination. Many of these resembled the vibrio of cholera so closely that no difference was possible, except the negative reaction with the specific serum. In the cholera cases, a fluctuating rectal temperature of a degree above or below normal persisted for some days after all clinical symptoms had subsided, and after the specific organism had ceased to be demonstrated in the stools. The cholera carriers with no subjective symptoms could be infective up to fifty-four days. The cholera cases remained infective by the presence of the comma-bacillus in the stools for one to two weeks after all symptoms had abated, exceptionally for longer periods.

CONCLUSIONS

Time and length of voyage from infected districts does not confer any special immunity against the occurrence of Asiatic cholera in this country. The well-established existence of the cholera-bacillus carrier has altered considerably the aspect of cholera prevention, for we have to deal not only with real, but also with potential agents of infection. The high efficiency of present-day quarantine procedures has rendered the possibility of spreading infection from cholera cases extremely slight. The real danger to the public is not alone the person sick with the disease, but also the person infected with the specific organism and presenting no apparent symptoms of illness. In typhoid carriers the gall-bladder is known to be the store-house of the infecting organism. That a similar condition may exist with cholera carriers is suggested by the observations of Greig,¹¹ who demonstrated by cultural methods the presence of the cholera vibrio in the gall-bladder eighty-one times out of 271 cases of fatal cholera. In the prevention of cholera, besides the effective isolation of the sick there has developed in our experience at quarantine, the necessity of extreme caution in the release of contacts or fellow passengers from detention, before a routine bacteriologic examination has proved them free from the cholera vibrio and even after such a routine procedure there remains always a possibility of the escape of a bacillus-carrier who may have been examined during a negative interval of an intermittent discharge of the infecting organism.

When sporadic cases of cholera occur in any community (the so-called cholera nests) it would seem that the bacteriologic examination of drinking-water and of food, although advisable always, is not of the same importance as a diligent search for the true source of infection, the infected, presumably healthy person, the proper means for whose detection, although involving a state in great outlays of money and time, are of necessity justified in emergencies for the proper preservation of the public health and confidence.

I am indebted to the courtesy of Dr. F. C. Clark, chief medical officer of Swinburne Island Isolation Hospital, Quarantine, N. Y., for the opportunity to review the clinical reports of cholera cases and cholera carriers.

THE "WASSERMANN-FAST TABES"

A SEROLOGIC PRECURSOR OF TABOPARESIS

D. M. KAPLAN, M.D.

Director of Laboratories, Neurological Institute
NEW YORK

The question of tabes from a diagnostic standpoint offers very few difficulties. Where clinical manifestations are few and indefinite, laboratory aids as elaborated by the latest serologic methods give ready and at times conclusive evidence of the presence of this disease. It is not my purpose to travel over well-beaten paths of diagnosis, be it clinical or serologic, but to offer to the neurologist a guide to therapy and to assist in the rendition of a prognostic opinion in this disease. To the serologist it may also be of interest to know that some forms of tabes behave differently from a serologic attitude than others. As there are different types of tabes clinically, so there are also various serologic manifestations of this disease. The clinician is thoroughly familiar with the form of disease which will ultimately present the full picture of taboparesis and is also acquainted with the type that is purely degenerative and lacks all evidence of activity in the meninges. The serologic development of the last few years in connection with diseases of the nervous system not only gave us the laboratory accompaniment of this disease, but also enabled the careful observer to point out here and there occurrences in the analysis of the serum and the fluid that are as important as the ability to diagnosticate this malady. It seems to me that neurologists are beginning to recognize the value of analyses of the cerebrospinal fluid and are gradually becoming more familiar with the utilization of laboratory data. One of the facts that I, together with Dr. L. Casamajor, pointed out in 1912, in connection with tabes, was the very favorable clinical and serologic reactions obtained with tabes of the "hyperlymphocytic" type. Our observations still hold good after a large additional number of such cases has been studied. On the other hand, we were considering the treatment of tabes that showed no cerebrospinal fluid abnormalities as rather inconsistent. Taking these things together, one can at a glance appreciate how much less informed we were in regard to the gauging of treatment before the era of "neurologic serology" as compared with the newer methods of procedure. The value of serologic data increases with their number, and it is with this in view that I have undertaken the writing of this article.

Having studied serologically over 650 cases of tabes of various types, and in some instances having observed the changes in the serum and cerebrospinal fluid on as many as twelve occasions in the same patient to note the adequacy of therapy, I had the opportunity to note the effect of various therapeutic procedures on the serology of a large proportion of cases and carried away the impression that there are distinct types of tabes that can be properly judged only after a thorough course of strenuous treatment. Although the patient may at the time of the first analysis show the serologic picture of tabes of the "hyperlymphocytic" type, the result of vigorous treatment will tend to show whether or not the serology is amenable to therapeutic effort. I have observed a number of cases in which despite the frequency, dosage and specificity of the remedy, the serum would still show a positive Wassermann reaction. A few more advanced cases would also show an unchanged positive Wassermann in the cerebrospinal fluid. I have designated these cases as "Wassermann-fast tabes" in contradistinction to those forms of

11. Greig: *Lancet*, November, 1912, II, p. 1423.

the disease that, as shown by the Wassermann test, yield readily to therapeutic efforts. When one studies the serology of general paresis one is struck with the large percentage of positive Wassermann reactions obtained on the serum and spinal fluid of this disease. When the subject of percentage of positive results is studied critically, particularly the observations that give 100 per cent. positive reactions even in general paresis, one is fully justified in asking the question: Is it possible that the cases reported were never treated with the remedies commonly used in such cases? Still further: If they were treated, some even with great thoroughness, is it possible that the Wassermann reaction remained unchanged and gave 100 per cent. of positive results? These questions are rarely asked and the results are accepted unchallenged. There should be no 100 per cent. of positive Wassermann reactions, and the experience of the majority bears out my contention. The reason why the percentage of positive Wassermann reactions in the serum from tabetics ranges between 55 and 70 is that the treatment of many of these cases was, from the point of view of serology, apparently successful. The low percentage of positive results also speak for the amenability of the serology of this disease to therapy, for it is self-evident that the easier it is to influence a disease with proper therapeutic measures, the fewer positive results are obtained with the Wassermann test. The number of cases of tabes that give positive Wassermann reactions are composed chiefly of patients who were not treated recently, or were treated insufficiently or belong to the

TABLE 2.—RESULT OF TEST IN TABOPARESIS, CASE 2*

Treatment	Serology Performed	Serum W. R.	Cerebrospinal Fluid Analysis		
			W. R.	Glob.	Lym-phoc.
Ilg. Inunct. 30 doses 4 gm.	3 weeks after therapy.	+	+	+	53
6 weeks later, 21 injections Ilg. sulleyl.	4 weeks after therapy.	+	+	—	53
8 months later, 600 0.6 in vein	4 weeks after	+	+	—	50
1 week later, 600 0.6 in vein	3 weeks after	+	+	—	41

* Reduction of Fehling's solution normal throughout.

type of cases of "Wassermann-fast tabes." The reason why general paresis gives the greatest percentage of positive Wassermann reactions is due chiefly to the fact that very few positive reactions in this disease become negative after treatment, in other words, general paresis typifies the Wassermann fast neurologic disorder of syphilitic origin.

The negative reactions obtained with general paresis are composed chiefly of those of the early cases which frequently give a negative Wassermann reaction in the serum and a positive result in the cerebrospinal fluid. It is very proper to apply to the serologic analogy of the "Wassermann-fast tabes" and its relation to general paresis the dictum of Charles Darwin: *Natura non fecit Saltus*, which is borne out by the clinical course of this type of locomotor ataxia. In the twelve cases of the "Wassermann-fast" type observed by me, nine patients developed full-fledged taboparesis and three died shortly after the parietic signs manifested themselves. To cite a few cases:

CASE 1.—Mr. H. S., aged 44, married; no children; lues eighteen years ago; shooting pains; girdle crises; Argyll Robertson pupils; Romberg phenomenon; no mental symptoms.

Serology before treatment, positive Wassermann reaction in serum and fluid; excess of globulin; 60 lymphocytes per cubic millimeter; prompt reduction of Fehling's solution.

CASE 2.—Mr. Sp., aged 52, married; wife had four miscarriages. Lues at 20; markedly ataxic; girdle crises; Argyll Robertson pupils. Serology before treatment, positive Wassermann reaction in serum, same in fluid; slight excess of globulin; 36 lymphocytes per cubic millimeter; normal reduction of Fehling's solution.

TABLE 2.—RESULT OF TEST IN TABOPARESIS, CASE 2*

Treatment	Serology Performed	Serum W. R.	Cerebrospinal Fluid Analysis		
			W. R.	Glob.	Lym-phoc.
0.6 salvarsan intraven.	2 weeks after therapy	+	+	+	33
1 week later the same	3 days after therapy	+	+	+	26
10 days later 0.14	One week after therapy	+	—	—	21
1 day later 0.14	3 days after therapy	+	—	—	21
1 week after 0.14 intraven.	One week after therapy	+	—	—	16

* Reduction of Fehling's solution normal throughout.

REPORT OF CASES

The treatment given these patients by no means represents the most active specific therapy that could be employed. The reason for this was the discouragingly rapid decline in the general health of the patients. The first one of the two died two months after the last treatment. The second patient developed taboparesis one year after the last treatment and died in a parietic decline.

These cases of tabes represent the absolute "Wassermann-fast" variety of the disease. It is sometimes, however, possible to obtain a weakly positive result after very vigorous treatment, and on very rare occasions even a complete negative Wassermann. The latter, however, does not remain negative for a long time but may return to positive a week or two after the last negative analysis. These are only relatively fast tabetics whose Wassermann reaction sooner or later will reassume its absolute fast nature, initiating in the course of time the clinical signs of taboparesis. Time will tell whether salvarsan or any other as yet undiscovered remedy used in large and frequently repeated doses will ever tend to ward off the approach of an impending taboparesis in a tabetic; at present it seems that frequently repeated small doses of neosalvarsan produce very encouraging changes in the psychic manifestations of the disease. In my opinion a patient with tabes who presents an absolute or even a relative "Wassermann-fast" tendency should be given the benefit of this treatment with small doses (0.3 gm. of neosalvarsan) every third or fourth day. I do not think that the Wassermann will be influenced at all, or at most an absolute fast condition could be made a relatively fast one. In fully developed general paresis one encounters the absolute "Wassermann-fast" condition more often than in any other syphilitic disease of the nervous system, and as the serology of a tabetic inclines toward assuming the "Wassermann-fast" tendency, it is my opinion that the clinician should be on the lookout for clinical evidences of an approaching taboparesis.

30 Beekman Place.

Physiology and Dietetics.—"The teaching of physiology on the subject of diet must be tested carefully by the light of clinical experience."—David L. Edsall, in *Clin. Jour.*