(2) On *Staphylococcus Pyogenes Aureus*.

Glycoboron Solution added	100 %
After 1½ hours	****
" 24 "	****

(3) On Anthrax Spores.

No effect, even in full strength.

E. Experiments with Carbolic Acid.

These six experiments were made parallel with those to determine the inhibitory action of styrene and sulphonaphthol on the *staphylococcus pyogenes aureus*. A saturated solution of carbolic acid was prepared and used in the same manner as the solutions in the former experiments. Temperature 37° C.

Carbolic Acid Sol.	0.8 %	1.6 %	2.4 %	4 %	5.6 %	8 %
Growth after 24 hours,	****	****	****	****	***	*
" " 5 days,	****	****	****	****	****	***

THE CLINICAL HISTORY OF ARSENICAL POISONING.¹

BY A. F. HOLT, M.D., CAMBRIDGEPORT.

ARSENIC is by far, the best known of all poisons, there being hardly a person in the community but what is familiar with its deadly properties. It can not only be had for the asking at nearly all the street corners, but it is even extensively advertised in the form of rat-poison and Paris-green for the destruction of potato-bugs. It is tasteless and odorless, and can readily be mixed with most kinds of food, both solid and liquid.

These facts, no doubt, in a large degree account for the almost daily occurrence of cases of arsenic poisoning both suicidal and homicidal. For the perpetration of both these crimes it is probably used oftener than all of the other poisons combined, and its use for such purposes is constantly increasing. In my own limited experience in the last four years, sixteen cases have occurred, nine of these were probably cases of murder, the remaining seven being suicides or attempted suicide.

The exceedingly common use of this substance for criminal purposes makes the subject of arsenical poisoning not only one of great interest to the medical jurist, but also one of which he must have a thorough and critical knowledge for a proper performance of his duties.

The first suspicions of a criminal case of poisoning with arsenic, and it is only with such that the medical examiner has to deal, are usually aroused at the bedside of the victim; and as such suspicions must often be confirmed or dispelled by the clinical history of the case alone, such a history becomes of great medico-legal importance. Such cases are, as a rule, fairly typical and well-marked, most of them following the same general course and presenting the same prominent symptoms. Unfortunately, however, some of the diseases that result from natural causes present the same general symptoms, and to distinguish between these is always difficult and often impossible.

¹ Read before the Massachusetts Medico-Legal Society, October 3, 1888.

No better evidence of this can be had than that presented by the notorious Robinson cases. Here were eight cases of criminal arsenical poisoning, seven of them occurring within a period of about five years in one family, and the other in a near relative. All of these cases were treated by physicians of large experience, most of whom are among the very best in our profession, men who, as careful and accurate observers, stand in the very front rank. Yet the real cause of the sickness and death of these victims was not even surmised until a chain of circumstances, unknown, of course, to the physicians, came to the knowledge of the associates of the Robinson family. There was no suspicion of arsenical poisoning in these cases, and nothing to excite such suspicion except the symptoms presented by the patients themselves, and these could readily and properly be accounted for by diseases of every-day occurrence.²

Reports of cases of arsenical poisoning are scattered through the medical journals that have been issued within the last forty or fifty years, and such reports comprise very largely the literature of this subject, so far as the clinical history of such poisoning is concerned. All kind of symptoms have been reported to result from an overdose of this substance.³ Some medico-legal writers have divided these cases into periods, some of one time and some of another; while others have described them as acute and chronic; and the symptoms that are described by writers on the subject represent nearly all the ills that flesh is heir to. No one has attempted to reduce them to order, and I am sure no successful effort could be made to eliminate from them a clear and concise description of the clinical history of arsenical poisoning that would apply to all, or even a majority of such cases. No such effort to eliminate from the mass of symptoms ascribed to arsenic a clear and detailed description of such poisoning will be made in this paper, for the writer has had no better opportunities of observing cases of this sort than his predecessors, and only an attempt will be made to describe such symptoms as will probably occur in such cases, and the order in which they appear, symptoms that should at least

² There has been a good deal of adverse criticism, not only on the part of the community at large, but in the public press, both as items of news and in editorials, because the physicians in attendance on these cases did not discover their real cause, or at least have their suspicions aroused. The general impression is that they were all treated, or at least most of them, by the same physicians. This, of course, was not the fact. The guilty mother was very careful not to employ the same physician twice; and, in ordering a consultant, she always sent for a different one each time. Not only this, but no more than two of the victims died in one house or one neighborhood. But even had one physician treated them all, his suspicions might not have been aroused. There never was a breath of suspicion on the part of the friends of the Robinson family, who must, of course, have known of all the deaths and the circumstances surrounding their occurrence. As the sickness of these unfortunate people was like that of diseases from other causes than arsenic, diseases, too, of common occurrence, why should the suspicions of the physicians have been aroused more than that of the friends? Doctors are not looking for a murderer behind the door of every house they enter, where there is vomiting, purging and other gastric disturbances the cause of which does not readily appear. A physician who has no more faith in humanity than this, has certainly mistaken his calling, and such an one would be unworthy of confidence or respect.

The people and the press that have made these unjust criticisms forget that cases of poisoning with opium, strychnin, the strong acids, and, in fact, those produced by all the other poisonous substances, are almost invariably detected at once by the physician, even if he has never seen a case of poisoning. The reason of this, of course, is, the symptoms produced by most poisons are clearly defined and distinctive, while those produced by arsenic are not so, but are like those produced by many other causes.

³ Many unquestionably, even if not a large majority, of the cases of arsenical poisoning that have been reported in the medical journals were observed by men little used to habits of close observation, and less to recording what they saw, and this fact may account for some of the symptoms that have been referred to arsenic; still there can be no question but what under different circumstances of administration, and in different individuals, the results may be variable and widely different in different cases.

arouse our suspicions of arsenic poisoning where they cannot be clearly and definitely accounted for in some other way.

Arsenic is both a narcotic and irritant poison: sometimes one set of symptoms and sometimes the other, may prevail. It has a specific effect upon the mucous lining of the stomach and bowels, and this effect is produced whether the poison is swallowed or administered in some other way, as for instance, where a considerable quantity is applied to an ulcerated surface.

The time between the taking of a poisonous dose of arsenic and the occurrence of the first symptoms is variably stated as being from a few minutes to sixteen hours, depending upon the condition of the stomach. The real cause of the great difference in time in producing poisonous effects is, I believe, largely due to the sparing solubility of arsenious acid (the form in which this poison is usually taken). If, for example, the stomach is covered with mucus, or contains some mucilaginous material with but little water, or if it contained semi-solid food of any kind, the crystals of arsenic might readily become imbedded in it and remain for a considerable period before enough would be dissolved to produce poisonous effects. The usual time for the first evidences of the effect of the poison to appear is from half an hour to an hour. They are rarely delayed more than two hours, and almost never appear sooner than twenty minutes. A single case has been reported where the vomiting and pain occurred almost as soon as the poison was swallowed.

The symptoms of arsenical poisoning as given by one author are as follows: "The symptoms produced by the poison are an austere taste; fetid state of the mouth; frequent ptyalism; continual hawking; constriction of the pharynx and œsophagus; the sensation of the teeth being on edge; hiccough; nausea; anxiety; frequent sinkings; burning pain at the præcordia; inflammation of the lips, tongue, palate, throat, bronchi, and œsophagus; irritable stomach, so as not to be able to support the blandest drinks; vomiting of matter, sometimes brown, at other times bloody; black, horribly fetid stools; small, frequent, concentrated and irregular pulse, but occasionally slow and unequal; palpitations; syncope; insatiable thirst; burning heat over the whole body, or a sensation of icy coldness; difficult respiration; cold sweats; suppression of urine; scanty, red, bloody and sometimes albuminous urine; change in the countenance; a livid circle around the eyelids; swelling and itching of the body; livid spots over the surface, and occasionally a miliary eruption; prostration of strength; loss of feeling, especially in the feet and hands; delirium; convulsions, often accompanied with insupportable priapism; falling off of the hair, detachment of the cuticle, etc. In some cases there is inflammation with burning pain in the urino-genital organs. Occasionally the symptoms have a perfect resemblance to those of Asiatic cholera, in a stage of collapse." This author further says: "It is very rare to observe all these symptoms in a single individual," and, "that sometimes they are nearly all wanting, death taking place without any pain or prominent symptom."

From such a confused mass of symptoms as these, indicating disturbance in every part and every vital organ of the body, and these perhaps presented in all conceivable combinations with nothing else to arouse his suspicions of arsenic poisoning except the condition of his patient, how is the physician at the bedside

to determine that the case with which he is dealing is one of arsenic poisoning? or how is he to have his suspicions aroused in this direction? Certainly not from arranging in his mind, and keeping before him for instant use, such a confused mass of ills as are here enumerated, as such an attempt could only lead to failure, but rather, I believe, by the careful consideration of the prominent symptoms produced by arsenic, and the order in which they occur.

The first symptom that occurs in these cases and one that all agree is usually present, is a feeling of faintness or weakness. This comes on in a half-hour or an hour after swallowing the poison, and continues until the more marked symptoms are manifest. This faintness is, I believe, from the physiological effect of the poison. The next prominent symptom, and one, too, that is almost always present, the exceptions being very rare, is vomiting. This usually occurs in from a half-hour to two hours of the ingestion of the arsenic. Occasionally this symptom is delayed; but the above rule would, I believe, include nine-tenths of these cases. The vomiting is violent and incessant, almost always continuing, if the case proves fatal, until death. The vomitus, after the stomach has been first emptied of its contents, consists of a watery secretion mixed with glairy mucus, bile and sometimes streaks of blood. This vomiting is almost sure to occur whenever anything is taken into the stomach. It is accompanied by a severe and pretty constant retching and a burning pain. The pain is referred to the region of the stomach and lower part of the chest. In one case that came under my observation, the patient complained that "she was burning up inside."

Pain in the stomach and vicinity is one of the most marked signs of arsenic poisoning, and is rarely absent: usually it is of the burning character above referred to. This continues to the end. Another pronounced symptom is dryness of the throat and mouth, and great thirst. The patient is constantly calling for water, although he knows that the drinking is sure to excite vomiting. The next prominent symptom to appear, and one that is almost always present also, is the great tenderness on pressure over the region of the stomach. This tenderness is usually very acute, the patient often crying out at the least touch. This symptom is, I believe, one of the most typical of arsenic poisoning. It does, of course, occur in other forms of gastric irritation, but generally it is not so acute, and such cases almost invariably have a very different history. This acute tenderness accompanied by vomiting, pain and thirst, coming on suddenly in the absence of any known cause, should be considered good ground for suspicion, at least of metallic poisoning.

The next prominent symptom is diarrhœa. This occurs in from one to three hours. The stools are at first yellowish and very loose, afterwards they may become watery and at times bloody. Often there is great and continuous tenesmus, with only the discharge of a little blood. This symptom of looseness of the bowels is not always present. In two cases that came under my observation, there was no discharge from the bowels; both of these terminated fatally in about eight hours. In another case, that of a girl sixteen years of age, a profuse watery diarrhœa came on soon after the vomiting, and continued up to within an hour of her death, which occurred in about six hours from the commencement of the poisoning.

The above symptoms usually continue from two to

five or six hours, when the pulse becomes quick and feeble, the skin cold and clammy, and perhaps covered with profuse perspiration; the face is pinched; in a word, the victim falls into a state of collapse, in which he may remain for some hours, death usually occurring however, in from five to ten hours after the ingestion of the poison. During the last hours of life there is often cramps (particularly in the legs and arms), low delirium, coma and sometimes convulsions. In some of these cases these symptoms continue twenty-four hours, and rarely longer, although all authors agree that a very large proportion of them terminate in nine or ten hours. In cases where the death is deferred four or five days, the vomiting continues, particularly when anything is taken into the stomach. The skin is dry and hot, and after a day or two becomes sallow and sometimes jaundiced; the eyes yellow; the tongue dry and cracked; and the lips and gums bleed readily. The pulse is small and quick; the stomach and bowels are distended and tender; the urine is diminished in quantity, and often contains a large amount of albumen; the cheeks are sunken; there is often delirium and great prostration; until at last the patient sinks into a state of collapse, as in the more acute cases, and dies.

This class of cases is much more likely to escape detection than those where death occurs in a few hours, as the effects are much less pronounced, and some of the symptoms may be absent, or so slight as to attract but little attention. Often there is little or no appearance of a fatal issue for the first two or three days, or until the fatty changes in the liver and kidneys has seriously complicated the case by bringing about a series of symptoms, the result of such changes.

In another class of cases the poison seems to act as a narcotic, the victim complaining of little or no pain or nausea, but sinks into a sound sleep, soon becomes comatose, sometimes convulsed, and in a short time dies. It is this class of cases, and those that rapidly fall into a state of collapse after the pain and vomiting appears, that are the most speedily fatal.

In five cases of which I have had intimate knowledge, they have all died in less than nine hours from the inception of the poisoning. In three other cases of poisoning with Paris-green, used for the destruction of potato-bugs, recovery took place, although in one case more than a dessert-spoonful was swallowed; the explanation of this escape probably being, that the stuff called Paris-green used by the farmers, is made up largely of adulterations. The other ten cases that have come under my observation, including the Robinson cases, have lived from five days to six weeks from the time evidences of poisoning were first observed. These deaths were all homicides, and in eight of them the poison was administered by one hand. It is certainly very extraordinary that all of these cases should so widely differ, so far as the deaths are concerned, from the usual rule in cases of arsenical poisoning. In some of these, undoubtedly, the dose was repeated, and perhaps more than once. To me there is no more remarkable fact connected with this notorious series of crimes (the Robinson cases) than this one. How a woman, ignorant of the effects of arsenic except to know that it was a poison, could so graduate the dose in eight successive cases as to delay the fatal issue from six days to six weeks, is beyond comprehension. It was undoubtedly this anomalous result that enabled her to conceal her crime so successfully. One of the

deaths was certified as caused by pneumonia, one by typhoid fever, one by meningitis, one by bowel disease, and one by kidney disease.⁴ Had the clinical history of one of these cases been the usual one of arsenical poisoning, with a fatal issue, it is more than probable that the real cause of death would have been detected. The causes of death in arsenic poisoning are the physiological effect of the drug, shock, and irritation of the intestinal canal and other organs. It may be from one of these alone, or from two, or from all of them combined. Cases of death that occurred from this cause within one hour of the poisoning, or even those that occur later, where no irritant effects have been observed, are from the physiological effect of arsenic; those occurring later, and within eight or nine hours are usually from the physiological effect and shock; while those that occur in three or four days are from irritation and the consequent inflammation, perhaps combined with physiological effects on the liver and kidneys. The diseases that may simulate arsenic poisoning are cholera morbus, colic with vomiting and gastric irritation from other causes. The points of distinction between these and arsenic poisoning, so far as their prominent symptoms are concerned are not well-marked; still, when the whole history of a case of either of these ailments is carefully compared with one of arsenic poisoning following the usual course, there usually are points of sufficient difference to enable one to make a differential diagnosis with a tolerable degree of certainty. For instance, the pain of colic or cholera morbus is usually paroxysmal and griping, or hard; while the pain of arsenic is burning and continuous. There is not often tenderness in either of the former, while this is a well-marked symptom of the latter. The vomiting of arsenical poisoning almost always is the first serious symptom; and this (and the retching) is continuous, or is repeated at short intervals; while that of cholera morbus and colic, if it occurs at all in the latter, comes on later, the pain almost invariably preceding it.

Thirst is a prominent symptom in arsenic poisoning while it is often absent in cholera morbus and colic. The diarrhoea of arsenic comes on after the other symptoms are well established; in the diseases we are considering, it is often the first sign of disturbance. Cholera morbus and colic usually respond readily to remedies, the pain and vomiting ceasing; and health is rapidly restored. And they are almost never fatal, except in the old and feeble; while, in arsenic poisoning, the case goes on from bad to worse, in spite of treatment, to a fatal issue. Therefore, the severity of symptoms is another of the cardinal points in differential diagnosis. Cases of gastric irritation of severe type, may, under certain circumstances simulate arsenic poisoning very closely; but fortunately such severe irritations are of rare occurrence, and usually they can be traced to some well-marked and definite cause. Cases where such well-marked cause does not appear should at once arouse suspicion of metallic poisoning. In such cases, where the vomiting continues without apparent cause, the settleings of vomitus should be examined for crystals of arsenic, and sent to

⁴ It perhaps should be further stated that in all of these cases the guilty mother selected a time to administer the poison when they were sick. For example, the sister had been suffering nearly a week with pneumonia before Mrs. Robinson saw her; the husband, whose case was supposed to be one of typhoid fever, came home from Boston suffering so severely with pain, nausea and vomiting that a physician was at once sent for; while Willie, the last victim, was under medical treatment from the result of a fall.

a chemist for analysis. This matter, however, belongs to the medico-legal considerations of arsenic poisoning rather than to its clinical history.

In cases of arsenic poisoning where death does not occur for some days, the prominent symptoms, as has already been said, are usually less well-marked: consequently the diagnosis is more difficult. Such cases can only be determined by a careful consideration of their histories and surroundings, examination of the vomitus for crystals of arsenic, and chemical analysis of the urine, as this poison is largely eliminated by the kidneys.

Chronic arsenical poisoning occurs occasionally from the repeated administration of small doses, although there is little likelihood of this being attempted for purposes of crime, except possibly by one thoroughly familiar with its action. These cases may present all the symptoms of the acute form of poisoning, although usually in a mild degree; and to these may be added extreme nervousness, trembling, paralysis (particularly of the hands or feet), tingling in the fingers and toes, a vesicular eruption on the body or limbs, suffusion of the eyes, ulceration of the mouth, jaundice, strangury, etc. The patient becomes emaciated, prostrated, and finally dies from exhaustion, or disease of the kidneys. One of the Robinson cases was of this kind, the patient, a girl twenty-two years of age, living six weeks after the first symptoms of poisoning showed themselves. In this case there was paralysis of both feet, and the urine contained albumen and casts.

As has been already said, there are no distinctive symptoms of arsenic poisoning, and one can only be certain he is dealing with such a case when the poison is found in the vomitus or urine, or there is other proof that it has been administered. The physician should be reasonably alert in trying to determine the causes of cases that come under his observation where there is vomiting, purging, pain, etc.; but it is no part of his duty to suppose that all such cases are the result of metallic poisoning.

LATENCY OF ATAXIC SYMPTOMS IN CASES OF OPTIC ATROPHY.¹

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It has been long noted that optic atrophy, when present in locomotor ataxia, appears generally among the earlier symptoms. Indeed, given a case of optic atrophy without recognizable cause, the chances are altogether in favor of characteristic pains, bladder irregularities, or other symptoms, appearing later, which, even if not marked, will enable us to place the case under this category.

Charcot² emphasizes the fact that most women coming to the hospital with amaurosis develop sooner or later, in the majority of cases, ataxic symptoms, perhaps ten, perhaps fifteen years later. In one case cited, the blindness was followed in ten years by shooting pains and girdle pains, these symptoms remaining stationary for ten years more, when appeared symptoms of muscular incoördination.

Gowers³ mentions cases of his own in which optic

atrophy preceded ataxia by sixteen and twenty years, and quotes a case reported by Buzzard,⁴ in which atrophy existed fifteen years, associated only with lightning pains and loss of knee-jerk.

In his recent text-book, Gowers⁵ has stated that atrophy of the optic nerve so universally appears early in the disease, that, after the ataxic gait is fairly established, it is of rare occurrence. Conversely, he states that when optic atrophy has become developed, it is common for the other symptoms of locomotor ataxia to remain in abeyance. To use his own words, "In a large number of cases, the ataxia never comes on, the spinal malady becoming stationary when the nerve suffers." If this is true, its bearing on the question of prognosis is of considerable import. It is certainly something to be able to assure a patient, at any stage of this disease, of his probable, or even possible, immunity from any of its distressing symptoms. In the matter of diagnosis, also, the subject is worthy of consideration.

I have looked through the records of sixty-six consecutive cases of locomotor ataxia, — fifty-two seen in the Neurological department of the Massachusetts General Hospital, and fourteen in private practice, — in the hopes of throwing additional light on this branch of the subject. The result is as follows: Out of the sixty-six cases, the diagnosis optic atrophy was found in fourteen; and six more had decided loss of eyesight. In these six there was no description of the optic nerve given in the notes, which prevents their being classed with certainty under the cases of atrophy, although undoubtedly some, and perhaps all, of them belong there. Out of the fourteen cases of optic atrophy, the degree of incoördination was mentioned in eleven. In three of these eleven, there was no ataxia; in five, it was slight; in three only was there marked ataxia. In the eight cases in which the ataxia was slight or wanting, the duration of the disease was respectively, two, three, four, seven, eight, ten, twelve and twelve years. In nine of these fourteen cases, the knee-jerk was noticed. It was present in one or both legs, in four out of the nine; and in one other case, where the loss of eyesight was so considerable as to leave little doubt that optic atrophy was present, the knee-jerks were normal. In all of the cases the disease was well advanced. These figures, while far too incomplete to be regarded as statistical, are still of some interest, sufficiently so to encourage further investigation.

The noticeable features are the large proportion of cases in which the knee-jerk was retained in one or both sides, and the large proportion in which ataxia was either absent or very slight. With regard to the ataxia, this has been already considered by neurologists as a symptom not necessary to the diagnosis, hence the tendency to restore the name "tabes dorsalis" in preference to locomotor ataxia. The point to which I would call attention is that to which Gowers has practically alluded, namely, that it is the cases in which optic atrophy is present in which we may expect to find the absence or indefinite postponement of ataxia. Again, with regard to the preservation of the knee-jerk, the loss of knee-jerk in locomotor ataxia is, as a rule, one of the earliest and most constant symptoms, a fact which lends considerable significance to its preservation in four out of nine (probably five out of ten)

¹ Read before the Boston Society for Medical Improvement, April 8, 1889.

² *Leçons sur les Maladies du Système Nerveux*, 1880, t. II, p. 41.

³ *Gowers' Medical Ophthalmoscopy*, p. 164.

⁴ *Brain*, 1878, No. 2, p. 168.

⁵ *Diseases of the Nervous System*, 1888, p. 293.