

was rapidly increased to twenty grains t. i. d., and this was continued for a month without any noticeable change in the condition of the ocular muscles.

This case is undoubtedly one of uncomplicated, progressive ophthalmoplegia externa, and can be classed as one of the rarer forms of ocular paralysis.

The disease as first described by von Graefe in 1868, is characterized by a progressive, and as a rule, symmetrical paralysis of all the external ocular muscles; the upper lids drooping; the eye-balls becoming more and more limited in their movements, and in the end becoming almost completely motionless.

The intra-ocular muscles, in the uncomplicated form of the disease, do not suffer; the pupils retain their normal size and reaction, and the power of accommodation is unimpaired.

In 1879 Jonathan Hutchinson brought the disease into greater prominence by reporting seventeen cases; three of these were uncomplicated, while the remaining fourteen were complicated with other nervous diseases. Since the publication of Hutchinson's paper the affection has attracted a good deal of attention, and there has been quite a number of cases reported, both uncomplicated and complicated. I find but four of the uncomplicated cases reported in this country; two by Birdsall, one by Mittendorf, and one—a congenital case—by Tilley.

The disease, as a rule, occurs in otherwise perfectly healthy individuals. The atrophic changes remain confined to the ocular muscles; and as the accommodation and vision are not interfered with, the only inconvenience the patient is apt to suffer is from the immobility of the eyes, which, of course, necessitates more frequent movement of the head, and in some cases from the high degree of ptosis which compels him to tilt the head backward, so that the pupils may be brought below the borders of the drooping lids.

A few cases have been reported in which other nervous lesions, chronic bulbar paralysis, locomotor ataxia, and progressive muscular atrophy, developed in the course of this disease; but it is certainly not usual for other muscles to become involved when the atrophic changes begin in the ocular muscles. Although the pathology of this disease has not definitely been determined by autopsy, it is considered by Förster, Mouthner, and other investigators, to be a degenerative process involving the motor nuclei of the ocular muscles, situated upon the floor of the fourth ventricle and aqueduct of Sylvius, and is thought to be identical with the change occurring in progressive bulbar paralysis, and the spinal form of progressive muscular atrophy.

The centres of the motor oculi nerves are, as a rule, the first to become affected, and the corresponding muscles of the two eyes, supplied by these nerves, are usually involved at about the same time and in an equal degree. Exceptions to this symmetrical failure of power are rarely observed. Mouthner, however, reports one case, in which the ptosis developed in the left eye twenty years before it made its appearance in the right, and neither were the other muscles of the eyes symmetrically affected. At the time of Mouthner's examination some of the muscles still retained their normal power.

The levator muscles of the lids, in the majority of cases, never entirely lose their power, even when the other muscles supplied by the third nerves are completely paralyzed.

The degree of ptosis is therefore variable; it being sometimes so slight that it is not noticed by the patient, and causes him no inconvenience. It is usually, however, of a moderate degree, and is sometimes complete.

Diplopia is not often present, but it is sometimes complained of where the disease progresses with moderate rapidity, that is in cases where the loss of power becomes almost complete within a few months. In the larger proportion of the cases recorded the progress is very slow, and diplopia is not mentioned as being present. The case which I have just reported seems to be exceptional in this respect, as, although double vision is not complained of, it is found to exist, and is made more noticeable when the pupils are completely exposed by raising the lids.

In regard to the sex and age of persons afflicted with this disease, it is much oftener met with in males than females, and in most of the cases reported, the trouble has come on before the thirtieth year. I find the reports of three congenital cases, and of three others, including the case just mentioned, in which the trouble was first noticed during the third year.

Lawford reports one case, in which the lids began to droop when the patient was about fifty years old, and I do not find any others recorded where the trouble began later than this.

In regard to cause: a history of previous syphilis is sometimes obtained, though not as frequently in these uncomplicated cases as in the cases seen in connection with other nervous diseases.

The trouble is also sometimes dated from an injury. In Lawford's case, there is a possibility that the disease was hereditary, as the patient's grandmother and two male cousins are said to have drooping lids.

The prognosis, as far as any marked improvement in the ocular condition is concerned, is not good. A moderate increase in the movement of the eyes is sometimes observed, after iodide of potassium has been taken in large doses for a month or more, and there may also be some improvement in the ptosis.

Mouthner reports one case, a child eleven years old, that entirely recovered in two months, with no treatment whatever. The trouble developed rapidly in this patient; there being a complete double ptosis, three months after the first symptoms were noticed; there was also a slight facial paralysis, so that it can hardly be classed as a typical case of this disease.

ARSENIC IN THE COURTS.¹

BY F. A. HARRIS, M.D.,
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INASMUCH as arsenic is one of the best known of the deadly poisons, and as it is also one of the most easily obtained, owing to its extensive domestic use in the form of what is popularly known as "Rough on Rats," and its more extensive use in agriculture in the form of Paris green, it is not strange that by far the largest number of suicides by poison should owe their success to the ingestion of this particular one.

As white arsenic and Fowler's solution are practically free from taste or odor, and are readily administered in food or drink without liability to attract the attention of the victim at the time, it is not surprising

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

that this drug has been a favorite with those contemplating secret murder.

That its use for this purpose is constantly diminishing is wholly due to the gradually-increasing knowledge on the part of the general public that it is one of the homicidal drugs most certain of detection; and as modern chemistry has succeeded in removing much if not all of the mists and fogs which surrounded trials in past generations, the risk in the use of this poison has become so great that trials for murder with arsenic are becoming rarer year by year.

Yet, from time to time such cases do occur; and when they do, although the management of the trial must be wholly in the hands of the gentlemen of the legal profession, still they must rely largely upon the advice and assistance of the doctor. It is true, even if he be a medical examiner, his position is that of a witness only to tell what facts he knows, and such opinions as he can fortify; but he must also assist in clearing away any doubts which counsel eager in the defence of their client may seek to arouse in the minds of the jury while confining his own evidence within strict lines of impartial demonstration of scientific fact. The chain of proof in a trial for murder by arsenic will naturally include—

(1) The death and the cause of the death, namely, that it was arsenic in some form, and from nothing else; or, if from something else, directly, how far the arsenical poisoning was contributory thereto.

(2) That such death was the result of homicide, and not of suicide or accident.

(3) That the accused was the person who administered the drug,—this question involving motive, opportunity to administer, possession of the drug anterior to the crime, and other matters still more closely within the province of the lawyer.

It is with the first part of this chain of proof with which the doctor, and especially the medical examiner, has to do, and with the second part only to a limited extent. As in ordinary warfare inventions for defence have closely followed inventions for greater destruction, so in the legal warfare of murder trials, the skill of defence has been assisted by experience and the knowledge of increased means of medical proof, and to-day the closest attention to detail is necessary on the part of the physician. Formerly it would have answered every purpose to have demonstrated the presence of arsenic within the stomach. To-day such proof would be insufficient, as the presence of unabsorbed arsenic is consistent with a continuance with life. To-day the medical evidence must demonstrate not only the presence of the drug, but also, generally, that the drug has been absorbed, and also the method by which it has destroyed life, whether by the violent inflammation of the stomach and intestines coupled with the exhaustion of the violent vomiting and purging, or whether, at a later period, by the destructive effect of the absorbed drug upon the other viscera, notably the liver and kidneys, by producing fatty degeneration. A chemical examination of the vomitus and the urine voided, while of assistance in a capital trial, has not the importance that it would have in a trial for attempted murder where the victim survived, inasmuch as in such cases the proof of rejected and eliminated arsenic, combined with the history of the symptoms, would be the only proof of arsenic ingested or absorbed. But where death has occurred the pathological changes are so marked that it is not

difficult to demonstrate that an irritant poison has been at work, and the chemist is able to demonstrate what that irritant was.

The medical witness must be prepared to demonstrate clearly the pathological changes. No jury would be warranted in finding a verdict of guilty in a case where, although chemistry might show a small amount of arsenic in the system, there was still no evidence that such arsenic had produced any deleterious effects upon the organism. This is of especial importance when the liability to error in attributing minute amounts of arsenic after Marsh's test to the presence of such drug in the body at time of death is considered. A still graver error is liable to result unless the utmost accuracy is observed in weighing the amount of organs delivered to the chemist, and also the amount retained for examination in case of accident to the first portions; for an error of little moment of itself, when multiplied for the purpose of forming an estimate of the whole amount in the body, would have a possible fatal effect on the life of the accused.

This was notably exemplified in the *cause célèbre* at New Haven, where two young men were put on trial for the murder by arsenic of one Jennie Cramer. Here, though *a priori*, from the place where the body was found, and from all the pathological conditions, the case was one clearly of death from drowning, as was later demonstrated to the satisfaction of the jury, the Government endeavored to establish the theory of homicidal arsenic poisoning.

In the report of the post-mortem examination there was absolutely no proof of any deleterious effects of the drug upon the organism; on the contrary, the condition of stomach and bowels showed the absence of either vomiting or purging, and no fatty degeneration of any organs, and no inflammatory action in the stomach. The chemist who analyzed portions of the organs and of the muscles, found, by Marsh's test, minute quantities of arsenic.

It is, of course, a question if there was any arsenic in the body during life; but when it was put in evidence that he had *about* one-half of the liver and *about* so much of other organs, and the minute fractions of a grain of arsenic in these *abouts* were multiplied, as if they were exact amounts, by the weight of the body, to produce an estimate of a little less than two grains in the whole body, a very dangerous plan was adopted, when human life was at stake.

This, however, belongs rather to the province of the chemical expert than the pathologist. It ought not to be a matter of great difficulty to demonstrate the changes produced by an irritant poison, and to undergo the ordeal of a cross-examination, when the poison has been the sole cause of death. When disease is present, or organic changes in various organs, it may not be so easy to make clear to a jury just how much the arsenic had to do with the death, especially if the quantity is small, and the exhaustion from the irritant action is the chief factor. Each case must be considered and treated of itself; and that evidence will have the greatest weight with the jury which does not claim too much, or assume infallibility. The present state of medical expert testimony in the courts would undoubtedly ensure a number of witnesses to controvert any extreme position, no matter how fully convinced the examining physician might be of the soundness of his theories.

In all trials where the results of chemical analysis show only a small amount of arsenic, the theory of accidental admixture after death will be urged, especially in those long buried. To-day not much weight, if any at all, is given to claims that the body absorbed arsenic from the soil in which it lay. The physician who makes the autopsy has only to demonstrate the caution he observed to prevent admixture from anything on his table or instruments, or the use of unclean dishes or jars, and leave to the chemist the demonstration of the impossibility of any artificial accidental admixture while the organs were in his charge. One thing is absolutely conclusive. No pathological change can occur after death. If the pathological changes of arsenic poisoning are to be observed in the body, it is entirely clear that the arsenic was ingested during life.

Most of the embalming fluids, so-called, contain arsenic; and it is often the case that the body has been injected before the cause of death is suspected. This need give the medical examiner no trouble; for the utter worthlessness of any pathological or chemical examination, as far as the demonstration of arsenic poisoning is concerned, is so clear that no prosecuting officer would proceed at all in the case, if the result depended on such examination. There were some such cases involved in the late trial of Mrs. Robinson; but they were not regarded as factors, either by the lawyers or physicians.

The question of accident, suicide, or homicide, is one to be determined by collateral evidence, and really to be decided by the jury. The form in which the drug was ingested, the quantity and time and place, social surroundings and mental conditions, may all have weight in arriving at a just conclusion; and the physician can be rather of service to counsel before trial in aiding him to a proper theory, than in actual testimony upon the stand.

The other questions occurring in arsenic trials are wholly within the domain of law, and so not within the scope of a paper before this Society.

REPORT ON RECENT PROGRESS IN SURGERY.

BY H. L. BURRELL, M.D., AND H. W. CUSHING, M.D.

TETANUS.

VON EISELSBERG,¹ in Vienna, has had an opportunity to investigate six cases of tetanus clinically and experimentally. Four ended fatally. In four cases fragments of skin, wound secretion, and blood were investigated, and used for inoculation experiments. In two cases splinters were removed from the wounds. In four of the cases the wound had been fouled with earth, when received. V. Eiselsberg's results corresponds with those of most other writers, except that he did not succeed in finding tetanus bacilli in the blood. Inoculation of animals with wound secretion, particles of skin, and wood splinters removed from patients subsequently affected with tetanus, were successful. In one instance tetanus followed inoculation with fragments of such a wood splinter which had been removed from a fatal case of tetanus two and one-fourth years before. With reference to prophylaxis, v. Eiselsberg recommends that even the smallest wounds which have been inoculated with particles of earth or foreign

bodies should be most carefully disinfected, and all foreign substances removed at once; that all bandage material—soiled dressings, etc., that have been in contact with a tetanus patient—should be burned; that such patients should be isolated; and that an especial set of instruments and apparatus should be used for their treatment.

Of interest in this connection are the investigations of Dr. Joh. Raum, in Warsaw,² who has made a series of experiments by inoculating rabbits with earth. Earth from Göttingen which had been carefully shut up in a closed glass vessel for three and one half years, as well as earth from different parts of Warsaw, produced tetanus after an incubation period of from two and one-half to four days. An investigation of the cream-like mass at the point of inoculation invariably demonstrated the presence of the "Nikolaier bacillus."

Wiedermann,³ of Stuttgart, was successful in his inoculation experiments with fragments of a splinter of wood taken from a fatal case of tetanus, and with the pus from the same patient although he did not find the bacilli in the latter.

Guelpa,⁴ also, after an extended investigation, has confirmed the statements already made by Nikolair, Rosenbach, Brieger, and others. He concludes that tetanus is an infectious disease; that it is not, contrary to the opinion of Verneuil, a disease of equine origin, notwithstanding horses show the greatest susceptibility to it; that the tetanic symptoms are the result of the "toxine" produced by the bacilli, which increase in number during the first outbreak of symptoms only at the seat of inoculation. In general, the dissemination of the bacilli throughout the body is rare, and only occurs, if at all, in the later stages of the disease. That the indications for treatment are: (1) the extermination of the bacillus colony by active surgical procedure; (2) the elimination of the "toxine" from the system by cathartics, diuretics and diaphoretics; (3) to prevent the excitation of the nervous system by large doses of chloral.

OPERATIVE TREATMENT OF HERNIA OF THE BRAIN AND SPINAL CORD.

Hildebrand⁵ reports three cases of operative interference in Koenig's clinic, in cases where congenital defects existed in the cranial vault, with protrusion of its contents. One was a meningocele-cerebratis; another, an encephalocele, the sac containing fluid and brain tissue; the third, a meningo-cephalocele. All ended fatally. Also five cases of spina bifida, with three deaths from suppuration and meningitis, or unknown causes. Hildebrand recommends the following rules in operating:

In all cases of cerebral and spinal hernia, when the contents of the sac are fluid, incise, evacuate its contents and remove it. If the contents be brain substance, it should be reduced, if possible, and held so with a truss. If irreducible, the contents should be excised. Ligation of the sac base, without opening it, is only to be done with meningoceles containing only small amounts of cerebrum. Escape of cerebro-spinal fluid is avoided by immediate introduction of the finger through the incision in the sac, and occlusion with its tip of the communication between it and the interior.

¹ Ztschr. f. Hyg., 1889, v. iii, p. 509.

² Ztschr. f. Hyg., 1889, v. iii, p. 522.

³ Bull. de Théor., 1888, livii, p. 508.

⁴ Ztschr. f. Chir., 1888, Bd. xxviii, heft, 45.

¹ Wien. Klin. Wchnschr., 1888, i, 10-13.