POISONING BY ACONITINE

REPORT OF TWO CASES

BY

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The history of poisoning by aconite is a long one, going far back into ancient times. This is not surprising, since the plant species from which the poison is derived, notably *Aconitum napellus* (monk's-hood) and *Aconitum ferox*, are common and widely distributed. Within modern times, however, aconite poisoning has been relatively uncommon, certainly within the United Kingdom; and poisoning with the pure alkaloid itself, aconitine, is still more rare. For this reason the symptoms are worth recording, as they occurred in two recent non-fatal cases.

Two medical students, engaged in "spring-cleaning" a privately owned collection of materia medica, found in one of the cupboards a number of small glass phials labelled as containing various vitamin preparations. These they decided to throw out, but before doing so they each took a "pinch" from certain of the phials, including one from which a detached label, inscribed "vitamin C" appeared to have fallen. Subsequent investigation showed that the white crystalline powder in this phial was in fact aconitine, in a total quantity of about 250 mg. (4 gr.), but failed to reveal how the aconitine came to be in the collection or how long it had been there.

In an attempted assessment of the amount they had had, the students demonstrated exactly how they had taken a pinch of powder between finger and thumb, put it on the palm of the other hand and then transferred it to the tongue. Although an estimate arrived at in such a manner cannot be regarded as exact, it is based on more direct evidence than is available in many cases of poisoning, and the students' statements and demonstrations were consistent and convincing. From these it appears reasonably certain that each student had a dose of between 5 and 10 mg. (1/12–1/6 gr.) of aconitine, and more nearly the latter than the former. Both agreed that there was no great difference in the quantity of powder taken, but that Mr. B. (Case 1) probably had slightly more than Mr. T. (Case 2).

The aconitine was taken at 1.20 p.m., and the following is a description of the subsequent symptoms in each case.

Case 1

Mr. B. experienced a "sharp," slightly bitter taste, and a rapidly developing sensation of warmth and tingling in his tongue, which increased in intensity and extent until his whole mouth was affected. Within 10 minutes his tongue, cheeks, and lips felt numb and "drawn," his throat felt constricted, and he had a feeling of nausea. He gargled with water and took a large drink of water to make himself vomit. In this he was successful, and vomiting recurred at intervals during the next four hours without any further efforts on his part. Within half an hour of taking the dose, tingling and numbness had appeared in his fingers, and gradually spread "all over," but became particularly noticeable and distressing in his jaws and face. The vomiting, tingling, and numbness increased to a maximum about 4 p.m.—that is, some two and a half hours after taking the poison, and at this time he was conscious also of great tiredness and actual muscular weakness, so marked that he could only lie on his bed. In spite of his tiredness, however,

he felt restless, the restlessness being attributable at least in part to a feeling that the sensory disturbances in the face and elsewhere were more distressing if he stayed still. From 4 p.m. onwards his symptoms abated gradually, and by the later part of the evening he felt quite fit and well again.

Case 2

Mr. T. experienced almost immediately a slightly bitter taste and a tingling, burning sensation in the tongue, mouth, and throat, which developed into a condition of numbness, tightness, and constriction, lasting till 2 p.m. At 2 p.m. he felt slight nausea and was salivating freely but did not vomit. Indeed, during the next hour his symptoms regressed and he felt fairly well. At 3.15 p.m., however, he began to experience a tingling sensation, which gradually pervaded all parts of the body, becoming especially marked in the face. By 5 p.m. this tingling and numbness in the face were very distressing; he had had three bouts of retching and vomiting; he felt very sick and salivated freely; he felt faint and very cold, especially in the limbs, and his voice seemed far away. His skin was cold and moist.

He had to lie down because he was so weak, and he could scarcely move his limbs. Voluntary effort to seek or call for assistance was impossible. Yet he felt restless, and again the restlessness was dictated in part at least by the feeling of apprehension induced by the numbness in his face, and elsewhere, which seemed to be worse when he lay still. Any attempt to get up, however, brought him near to fainting. He remained in this state for more than two hours, the symptoms of tingling and numbness, salivation, and sickness, coldness, and faintness, weakness, restlessness, and a feeling of dread all persisting and becoming even worse. During this period he repeatedly tried to feel his pulse, but it was undetectable. His illness reached its climax between 7 and 8 p.m., and from 8 p.m. onwards he improved gradually. About 11.30 he fell asleep. He slept soundly throughout the night and felt quite recovered the following day.

Comment

It will be recalled that Mr. B. had, if anything, a larger dose than Mr. T. From the relative severity of their illness it would appear probable that Mr. B. succeeded in getting rid of a proportion of the poison by means of his self-induced vomiting 10 minutes after the dose was taken and by the further attacks of vomiting soon afterwards. Mr. T. did not take the same precautions, nor did he have any vomiting for the first two and a half hours or so. Indeed, he was lulled into a false sense of security by the almost complete absence of symptoms between 2 and 3 p.m.

Neither student received any treatment, although they reported to the out-patient department of a hospital about 2 p.m. They were told to go home, lie down, keep warm, and return if they felt worse—sound enough advice so far as it goes, and perhaps reasonable in the circumstances at that time, although in the light of events it might well have been supplemented or replaced by more active measures and a period of observation. The students went from the hospital to their respective lodgings, where, as it happened on that particular day, they were obliged to endure in solitude what was, particularly in the case of Mr. T., a most unpleasant and alarming experience.

Identification.—The powder in the phial was submitted for chemical investigation. It was reported as being purely alkaloidal in character, and giving reactions which were consistent with its being aconitine. With the usual range of chemical tests for alkaloids it is not possible as a rule to be more specific than this when seeking to identify aconitine, and confirmation must be sought by means of physiological tests. The substance was personally sampled, perhaps slightly to excess, for not only was there a rapid development of warmth and tingling in the tongue and mouth, but this went on to numbness and tightness of the mouth and throat and tingling in the fingers, which persisted for some time. Finally a dose of 0.5 mg. (1/128 gr.) was injected

subcutaneously into a rat. After a delay of about a minute, the animal showed signs of restlessness and agitation, and started to run. Salivation, retching, and respiratory distress developed, followed by paralysis, affecting first the hind limbs and then the forelimbs. There were terminal convulsions before the animal died three minutes after being given the injection. These were the results on which the opinion was based that the material in the phial was in fact aconitine.

Discussion

The fatal dose of aconitine is variously stated as being anything from 1/60 to 1/10 gr. (1 to 6.5 mg.) or slightly more. The uncertainty on what constitutes a fatal dose is no doubt related to the infrequency of poisoning with the pure alkaloid and also to its unstable nature. Furthermore, there are several varieties of aconitine, and they are not all equally poisonous. Factors of this kind presumably explain why the results were not even more serious in the cases described, although in each case the dose was certainly in excess of that which is commonly regarded as fatal or potentially so.

Aconite (aconitine) has both a local or peripheral and a systemic or central action, and both have served as a basis for its use as a therapeutic agent. The local or peripheral effect is the more striking and characteristic—namely, tingling and numbness, experienced either on local application or in the mouth when ingested, and in the extremities after absorption. The drug was formerly used, usually in the form of the tincture, for the relief of neuralgic or rheumatic pain. The central effect of value was believed to be a slowing of the heart and a reduction in blood pressure, hence its former use in certain cases of heart disease and in various febrile conditions.

To-day aconite is very little used therapeutically, and never internally, except perhaps in homoeopathic practice. Aconite poisoning is therefore uncommon, and the occasional cases which do occur are usually due to the suicidal or accidental ingestion of a liniment containing aconite mixed with other noxious substances. Accidental death through mistaking monk's-hood root for horse-radish is less frequent to-day than it appears to have been some years ago.

Because of its rapid and rather striking local effects, aconite is not an ideal homicidal poison, and it has rarely been used as such in this country. It has been suggested, however, that because of the effects on the heart, coupled with peripheral effects experienced in the arms and hands, sickness, and upper abdominal discomfort, and an early fatal outcome, cases of homicidal aconite poisoning might well be overlooked and regarded as deaths due to coronary artery disease.

In an Irish case in the first half of last century (*Regina v. McConkey*, 1841), powdered aconite root (monk's-hood) was mixed with pepper and sprinkled over the greens which formed part of the victim's dinner. Dr. Pritchard (1864-5) gave tincture of aconite mixed with Battley's solution to at least one of his two victims, but essentially the deaths were due to antimony poisoning. The outstanding homicidal case is undoubtedly that of Lamson (1881-2), also a medical man of sorts, who administered the alkaloid aconitine in a capsule (or a cake) to his invalid brother-in-law.

In the Lamson case aconitine was detected in the vomit, in the urine, and in an extract from the stomach, stomach contents, liver, spleen, and one kidney. Identification was by means of alkaloidal reactions, by the local effects produced on the tongue, and by the rapidly fatal effects on mice. Since then various colour and crystal tests have been described in attempts to render the chemical identification of aconitine more positive, but there is no general agreement on the specificity of any such tests. It has been suggested that most reliance can be placed on the effects of aconitine on the frog's heart, which appear in a characteristic sequence—namely, initial slowing followed by sudden acceleration, then incoordination, and a final slowing and arrest.

Greater certainty may be possible with the more modern methods of chromatography and spectrophotometry. Curry and Powell (1954) have described a method for the detection of alkaloids on a micro-chemical scale by means of paper chromatography which would appear to be applicable in the case of aconitine. Similarly, Kaye (1954) has described a characteristic spectrophotometric absorption curve for aconitine. Since these methods are applicable to very small quantities of material they would, if proved to be sufficiently specific, provide an extremely valuable means of identification, and might indeed relegate the older forms of testing to a role which was largely confirmatory. It is certain, however, that the characteristic and striking local effects of aconite will always retain their highly diagnostic significance.

REFERENCES

- Curry, A. S., and Powell, H. (1954). *Nature (Lond.)*, 173, 1143.
Kaye, S. (1954). *Handbook of Emergency Toxicology*. Springfield, Illinois.

Medical Memoranda

A Case of Nephrogenic Diabetes Insipidus

Nephrogenic diabetes insipidus is a heredo-familial disease characterized by polydipsia and polyuria from infancy. It is a sex-linked condition affecting males usually and transmitted by females, who act as heterozygous carriers. The condition is resistant to treatment with "pitressin," and appears to be due to an inherent kidney defect which results in failure of tubular reabsorption of water. Renal function in all other respects is unaffected. The following is a report on a case occurring in a youth of 18. This case is of interest because some of the female members of his family, besides acting as carriers, appear to have suffered from the full-blown clinical syndrome.

CASE REPORT

The patient complained that he had had to drink large quantities of water and passed large amounts of "white urine" for as long as he could remember. He found it necessary to carry supplies of water with him on train journeys or when he visited the cinema. Failure to relieve his thirst resulted in drowsiness, headache, and general malaise. He passed urine every half to one hour during the day and four to six times during the night. His daily intake and output was between 12 and 16 litres of water.

He was one of 10 children who were all alive except one brother who died at birth.

He had five living brothers and three sisters. His mother, eldest brother, a maternal aunt, a maternal uncle, a female cousin, maternal grandmother, a maternal great-uncle, and maternal great-grandmother all suffered from this syndrome from birth. The family tree showing the incidence of the disease in the two sexes was compiled from information given by the patient and his mother. The latter stated that she found it necessary to take 6 pints (3.4 litres) of water to bed with her at night and drank much more during the day.

The general appearance of the patient was normal and there were no abnormal physical signs in any system.