it also seems desirable to limit the dose of α-methyldopa to less than 2 grammes per day. In our clinic several drugs are used in combination to minimize the side effects of each (Doyle, 1966). If this type of régime is used doses of α-methyldopa larger than 2 grammes per day are seldom needed. Finally, it should be remembered that follow-up of the hypertensive patient does not merely involve inquiry about his well-being and taking his blood pressure, but requires considerable attention to detail. Periodic checks of cardiovascular and renal functions are essential. As well as this, one must be on the look out for the development of gout, skin rashes, depression and other side effects of hypotensive therapy. The patient on α-methyldopa therapy should be observed carefully for the development of anæmia, as hæmolysis in these patients is usually not acute in onset but mild and insidious, and therefore amenable to diagnosis before severe anæmia and complications have developed.

SUMMARY

In a series of 80 patients treated with α-methyldopa the incidence of positive reactions to the direct Coombs test was 14%. The incidence was dose-dependent, being 25% in those taking more than 750 mg. a day of the drug. There was no evidence of anæmia or hæmolysis in this group of patients. Examination of the patients with hæmolytic anæmia giving positive results to the Coombs test who were seen at the Royal Melbourne Hospital since 1961 revealed that two patients were taking α-methyldopa when their anæmia presented.

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ANTIVENENE IN THE TREATMENT OF AUSTRALIAN AND PAPUAN SNAKE BITE

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IN 1896 Calmette produced the first commercially available antivenene for the treatment of snake bite, prepared by injecting horses with a venom mixture containing 80% cobra venom. Calmette (1894 *a*, *b*, *c*; 1895) claimed that the antivenene would be effective against all forms of snake-poisoning.

This claim was disputed by Kanthack (1896) who said it was contrary to Behring's law of serum specificity, and was soon proved to be wrong by Martin (1897*a*) and Tidswell (1906) in Australia, who showed that Calmette's serum did not protect animals against Australian tiger snake, black snake, brown snake and death adder venoms. Martin (1897*b*; 1898) further proved that the subcutaneous dose of antivenene had to be 10 to 20 times the intravenous dose, and recommended that the intravenous route always be used in treating snake-bite cases. It is also of interest to note that as early as February, 1893, while others in Australia were preoccupied with the merits of strychnine as a cure for snake bite, Bancroft thought that the serum of animals immunized against snake venom "might prove useful as a remedy in snake bite".

In 1901 the first specific antivenene to be produced in quantities was prepared by Tidswell (1902, 1906), working in the laboratories of the N.S.W. Department of Health. Tidswell produced a specific tiger snake antivenene which he had prepared by cautiously immunizing a horse over a period of three years with tiger snake venom. He showed that this antivenene was ineffective against other venoms, and was uncertain as to what role it would eventually play in therapeutics.

Lamb in India (1903, 1904*a*, 1904*b*, 1905) produced specific cobra and daboia antivenenes and confirmed Tidswell's findings that antivenenes were specific in their action, effective only against the venom with which they had been prepared. Later work showed that antivenenes may have some protective action against the venoms of species of snakes other than the one used for their preparation and that their action, while being highly specific, was not entirely so (Houssay and Negrete, 1923; Vellard, 1930).

After the important early work of Martin and Tidswell on snake venoms and antivenenes in Australia, interest waned until 1927 when the late Sir Neil Hamilton Fairley (1929), after his return to Australia, interested the Director of the Walter and Eliza Hall Institute, Dr. C. H. Kellaway, in the study of Australian poisonous snakes and their venoms. Through Dr. Kellaway, Dr. F. G. Morgan, the Director of the Commonwealth Serum Laboratories, was interested in the production of antivenene, and he commenced work with the object of producing tiger snake, copperhead and death adder antivenenes in July, 1928.

Tiger snake antivenene became generally available in Australia towards the end of 1930. It was considered to be a highly effective antivenene and to give some cross protection against the venom of the Australian copperhead snake, and was subsequently recommended for use in the treatment of death adder bites, brown snake, black snake and mulga snake bites. Difficulty in producing an effective death adder antivenene was experienced for many years (Morgan, 1929; 1933), and it did not become available commercially until 1958. Specific taipan antivenene and specific Papuan black snake antivenene were produced in 1955 and 1958. In 1962, the first Australian polyvalent antivenene was prepared for use in treating snake bite in Papua and New Guinea.

The Australian venomous land snakes all belong to the family Elapidæ. The lethal factor of these venoms is the

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neurotoxin which produces a peripheral neuro-muscular block (Kellaway *et alii*, 1932), causing a generalized flaccid muscle paralysis. Respiratory obstruction is produced by paralysis of the jaw and tongue, or by the accumulation of secretions in the pharynx and bronchi. The obstruction to respiration aggravates the respiratory insufficiency caused by weakness of the muscles of the chest and diaphragm. The victim dies from asphyxia. Some venoms also contain a thrombase, which produces intravascular coagulation and a secondary fibrinogenopenia. Venoms may also contain a hæmolysin which may lead to hæmoglobinuria.

Other toxic factors in the elapine venoms are of little clinical importance. After a venomous snake bite no symptoms may occur, mild, moderate or severe symptoms and signs may be produced with eventual recovery, or death may result. The outcome in untreated cases depends on the dose of venom injected relative to the size of the victim.

Because the amount of venom injected by a snake is quite variable, the clinical evaluation of the effectiveness of antivenene in the prevention of the symptoms and signs of envenomation following a venomous snake bite is very difficult. In fact, it is impossible to know in a particular case whether antivenene has been of value in preventing serious envenomation or not. Hence isolated case reports, claiming antivenene to be of value in the prevention of snake-poisoning, are of little value.

While clinically there may be doubt about the effectiveness of elapine antivenenes in preventing the serious symptoms and signs of envenomation, there should be no doubt about the effectiveness of elapine antivenenes in the treatment of established snake-poisoning, "for nothing is more dramatic in the whole range of specific therapy than the effects of intravenous antivenene in badly paralysed cases bitten by colubrines (elapine snakes)" (Fairley, 1933). The bad paralysis is dramatically cured or reversed by the antivenene. An antivenene which is effective in reversing the effects of the venom would also be effective in preventing signs and symptoms of snake-poisoning.

Cobra antivenene was shown by Arthus (1910) in experiments on rabbits to reverse, over a period of two to six hours, the paralysis of the diaphragm produced by cobra venom, and clinical experience in India (Fairley, 1933; Mackie, 1933; Ahuja and Singh, 1954) and elsewhere (Purananda, 1956), have confirmed its effectiveness in treating established cobra envenomation.

Kellaway (1932), however, was unable to reverse to any significant extent with specific antivenene the muscle paralysis produced by tiger snake venom in rabbits, and despite the availability of antivenenes in Australia for over 30 years, there is little published evidence of their effectiveness in treating human cases of snake bite. Bill (1902) was the first to use snake antivenene in Australia and claimed it was of value, although he used Calmette's antivenene and this had previously been shown to be valueless in the treatment of Australian snake-poisoning.

Tisdall and Sewell (1931) published the first report of a case of snake bite in which tiger snake antivene was used in Australia, and claimed that it had saved life. Their patient only had a partial paralysis when seen 24 hours after the bite, and the recovery rate of the paralysis following the use of the antivene was not accelerated. Lloyd (1932) treated three patients with tiger snake antivenene, and stated that it was effective in saving life in one case, even though no symptoms of snake-poisoning were evident in this patient. Morgan (1933) also said that he knew of 14 cases in which tiger snake antivenene had been used and "life had been saved" by its use in some seriously ill cases. No details of the cases were given, so it is difficult to evaluate his assertion.

Tiger snake antivenene was used by Reid and Flecker (1950) in the treatment of a patient bitten by a taipan, and was said to have contributed to the recovery of the patient, but to have been of no value in another case of taipan bite reported by Benn (1951). Lester (1957) reported the first case of a taipan bite treated with specific taipan antivenene and considered that improvement commenced after the injection of the antivenene, but no dramatic reversal of the paralysis took place. Indeed, before 1966 there was only one case of snake bite in the Australian medical literature in which a dramatic reversal of the paralysis has occurred after antivene administration. In 1959, Knyvett and Molphy reported the complete disappearance of all signs 10 hours after the use of 12,000 units of taipan antivenene in the treatment of a seriously paralysed patient bitten by an unknown snake. Previously the paralysis had progressed to a stage where artificial respiration was required despite the use of 33,000 units of tiger snake antivenene.

In Papua there are three highly venomous snakes—the death adder (*Acanthophis antarcticus* Shaw), the taipan (*Oxyuranus scutellatus cani* Slater), and the Papuan black snake (*Pseudechis papuanus* Peters and Doria). Specific antivenenes are available for the treatment of bites by these snakes and there is in addition a Papua and New Guinea polyvalent antivenene available.

Early experience with the antivenene therapy of snake bite at the Port Moresby General Hospital was disappointing (Campbell and Young, 1961; Campbell, 1964). This evaluation of antivenene therapy was primarily concerned with its effectiveness in preventing and treating the effects of the neurotoxins in the venoms. The effect of the antivenene on other toxic factors in the venom was not assessed, though hæmoglobinuria had been noted to cease shortly after the administration of antivenenes (Campbell and Young, 1961) and the blood was noted to clot normally in wounds 24 to 48 hours after admission.

If injected early, antivenenes were considered to be of value in preventing paralysis. When used after significant paralysis was present, the antivenene never reversed the paralysis, which often would have progressed to a fatal issue if a tracheotomy had not been performed and artificial respiration carried out. However, two patients did show rapid improvement of their only sign of paresis, ptosis, after the injection of antivenene (Campbell, 1964). (In retrospect, both patients were probably bitten by death adders.)

PRESENT STUDY

At the Port Moresby General Hospital from March, 1964 until November, 1965, 28 patients with symptoms and signs of snake-poisoning were given intravenous injections of antivenene. The relevant clinical details are summarized in Table 1. Five of these patients had also received antivenene three to 17 hours previously on an outstation. If the species of snake inflicting the bite was known with a reasonable degree of certainty, specific antivenene was injected. More commonly, polyvalent antivenene was given. The usual dose of antivenene was twice the recommended dose (16 cases). The recommended dose is calculated to neutralize the average venom yield of the snake, and only three patients received this dose. Three patients received three times the recommended dose; four, four times the recommended dose; and two, five times the recommended dose. The method of injecting the antivenene intravenously has been reported previously (Campbell, 1963). No tests for serum sensitivity were performed. The term "probable death" is used when it was thought that death would have resulted had not a tracheotomy or a tracheotomy and artificial respiration been carried out.

RESULTS

Death Adder

The only patients to have their paralysis reversed by antivenene were those bitten by death adders. New Guinea polyvalent and death adder antivenenes (doses used—two, three and five times the recommended dose) were highly effective antivenenes, at least up until seven hours after the bite, reversing within two or more hours of the injection the serious effects of death adder bites.

Taipan

In the patient with serious paralysis (two doses of polyvalent and one dose of taipan antivenene having previously been given on the outstation), the paralysis was not reversed by polyvalent antivenene (twice the recommended

TABLE 1

Summary of the Clinical Details and Response to Antivenene Therapy in 28 Cases of Snakebite

Type of Bite	Hospital Number	Sex	Approximate Age (Years)	Hours Between Bite and Antivenene	Condition when Antivenene Given	Progress after Antivenene
Death adder	27853	M.	25	4 (1 at outstation)	Early signs—no symptoms	No change[1]
	28488	M.	20	3	Extensive paralysis	Paralysis reversed
	35645	M.	35	7	Early signs and symptoms[2]	No progression
	28040	M.	35	6	Early signs and symptoms, slight paralysis	Paralysis reversed
	32029	F.	35	4·5	Extensive paralysis	Paralysis reversed
Taipan	30447	M.	40	2·5	Early signs and symptoms incoagulable blood, hæmoglobinuria	No progression
	31521	F.	30	21 (4 at outstation)	Moderate paralysis	Progress to probable death
Papuan black snake	24281	M.	30	4	Severe early signs and symptoms. No paralysis, incoagulable blood, hæmoglobinuria	Progress to slight paralysis
	26601	M.	45	45	Extensive paralysis	No change
	28643	M.	25	3	Early signs and symptoms. No paralysis	No progression
	29314	M.	8	8 (4·5 at outstation)	Extensive paralysis	Progress to probable death
	30956	M.	55	61	Extensive paralysis	Died
	26580	F.	9	1	Severe early signs and symptoms, incoagulable blood	Progress to probable death
	27130	M.	10	4	Early signs and symptoms, slight paralysis	Progress to moderate paralysis
	28868	M.	26	4·5	Early signs and symptoms, incoagulable blood, hæmolysis present	No progression
	30202	M.	25	3	Early signs and symptoms, incoagulable blood, hæmolysis present	No progression
	33122	M.	40	26 (20 at outstation)	Slight paralysis, hæmoglobinuria	No change
Unidentified	26051	F.	25	6	Slight paralysis, incoagulable blood, hæmoglobinuria	Progress to probable death
	30101	M.	30	14	Slight paralysis	Slight progression
	31439	M.	9	5	Severe early signs and symptoms, no paralysis	Progress to probable death
	31853	M.	21	3·5	Early signs and symptoms	Progress to moderate paralysis
	33198	F.	30	21	Extensive paralysis	Progress to probable death
	35182	M.	25	2	Early signs and symptoms, incoagulable blood	No progression
	29286	M.	10	24 (21 at outstation)	Extensive paralysis	Progress to probable death
	31019	M.	28	2	Doubtful early signs	No progression
	31524	F.	11	0·5	Severe early signs and symptoms, incoagulable blood	Died
	32282	F.	25	4	Early signs and symptoms	Progress to slight paralysis
	34674	M.	6	2	Early signs and symptoms, incoagulable blood	No progression

[1] No change: no progression expected and none occurred.

[2] Signs and symptoms: headache and/or vomiting, pain in the groin, abdominal pain, spitting or vomiting blood, tender regional lymph glands.

dose) and continued to progress. No muscle paralysis developed after twice the recommended dose of taipan antivenene was given to the other patient with early symptoms and signs following a taipan bite. In view of the other evidence of serious envenomation present, antivenene probably prevented serious paralysis developing. This patient also had incoagulable blood, but a whisp of clot was present three and a half hours after the antivenene, a normal clotting time nine and a half hours after, and normal clot retraction 24 hours after the injection of the antivenene. Hæmoglobinuria ceased eight hours after the antivenene was injected.

Papuan Black Snake and Unidentified Snakes

Six patients with early symptoms and signs showed no progression of the poisoning after the antivenene. Four patients progressed to a non-fatal paralysis. In these patients the antivenene was probably of value. Six patients progressed to severe paralysis and probable death. In no patient was paralysis reversed by Papuan black snake or New Guinea polyvalent antivenene. In patients whose blood was incoagulable due to a fibrinogen deficiency, a whisp of clot was present one and a half to five and a half hours after the injection of the antivenene; a normal clotting time at seven and a half to 15 hours (observations approximately every three hours) and good clot retraction and a normal fibrinogen titre at 21·5 to 42 hours (observations every 12 hours after the first 15 hours).

REACTIONS TO ANTIVENENE

Eleven of the 28 patients had some form of reaction to the antivenene. Eight developed urticaria or an itchy skin within eight minutes or more of commencing the injection, and sometimes not until the end of the injection. Five had a shivering attack or rigor, often followed by a rise of temperature as high as 105.6° F. This reaction usually occurred within the hour after the antivenene was injected. Two patients complained that their abdominal pain was aggravated by the antivenene during the injection. One patient complained of severe pain in the arm along the course of the brachial vein as the antivenene was injected, the pain ceasing when the injection was stopped and started again when the injection was recommenced. One patient developed an increase in oral secretions and wanted to vomit; one became restless; one developed a loose cough and a wheeze; one patient developed anaphylactic shock.

No patient died as a consequence of the antivenene administration.

DISCUSSION

Death adder and Papua-New Guinea polyvalent antivenenes are highly effective antivenenes in the treatment of death adder envenomation and therefore also in the prevention of death adder snake-poisoning. It is claimed that it is never too late to inject cobra antivenene (Strover, 1955) and that even when a patient is moribund, recovery may still occur (Ahuja and Singh, 1954), but Mackie (1933) indicated that normally the patient had to have the antivenene "within a reasonable time of the bite for it to be effective", and Acton and Knowles (1915) stipulated administration before two-thirds of the anticipated time before death had elapsed. Death adder venom resembles cobra venom more closely than any other Australian venom, both in its toxic constituents and in its response to antivenene, and it is hoped that further clinical experience will indicate that it is never too late to inject death adder antivenene.

Unfortunately, this hope does not seem to hold for taipan and Papuan black snake antivenenes. After taipan and Papuan black snake bites a stage seems to be reached when the antivenenes cease to be of value in preventing death, for they do not halt the progress of the paralysis to a fatal issue. They may at this late stage possibly still act beneficially on the coagulation defect and the hæmolysis if present. It is particularly disappointing to find that, in Cases 26580, 31524 and 31439, even when the antivenene was given within one hour, half an hour and five hours of the bite, and in doses of two, three and four times the recommended dose, the antivenene did not in one case and would not in the other two cases have prevented a fatal outcome. These three patients were all children and must have received large doses of venom relative to their size.

Even though the antivenenes may not have prevented a fatal outcome in these patients and others in whom the paralysis continued to progress, it is still possible that the antivenene may yet serve some useful purpose in treatment. For apart from its beneficial effect on the blood, if the antivenene can prevent complete diaphragmatic paralysis and thus eliminate the need for the artificial ventilation of the patient for periods of up to one week or more, it has performed a most useful function. It is impossible to know whether the absence of serious paralysis of the diaphragm in some cases is due to the antivenene administered or due to the smaller dose of venom injected. In some cases it would appear probable that the antivenene has limited the extent of the paralysis.

It is possible that more massive doses of antivenene may further limit or even reverse the paralysis produced by the venom of the taipan and Papuan black snake, for up to 600 ml. of cobra antivenene may be required to reverse the paralysis produced by cobra venom (C. Purana nanda, 1967, personal communication). If this was to happen, evidence of slight improvement in the paralysis of at least one of the many cases of taipan and Papuan black snake bites now treated with smaller doses of these antivenenes should have occurred. The clinical response to antivenene of patients envenomated by the taipan and the Papuan black snakes differs from that of those injected with death adder venom. This may be due to relatively less potent taipan and Papuan black snake antivenenes or, more probably, to a basic difference in the response of the Australian thrombase-containing elapine venoms to antivenene (Campbell, 1966).

Furthermore, in experiments on animals injected with tiger snake venom (a thrombase-containing venom) Kellaway (1932) was unable to demonstrate any dramatic reversal of the paralysis with doses of antivenene 16 to 50 times the in-vitro neutralizing dose. However, the antivenene was injected 24 hours after the venom. On the contrary, J. J. Graydon (personal communication, 1966) states that, in experiments conducted at the Commonwealth Serum Laboratories over 30 years ago on moribund rats poisoned by tiger snake venom and copperhead venom (which does not contain thrombase), the paralysis was reversed with tiger snake antivenene.

The fibrinogenopenia produced by the thrombase in the venom is not usually of clinical importance unless some wound is present which then continues to bleed. This bleeding does complicate the management of a tracheotomy. In the Australian literature, there is one case of fatal cerebral hæmorrhage attributed to snake bite (Foxton, 1914). The occurrence of black tarry stools 48 hours after a serious snake bite has also been reported (Mueller, 1893), and small hæmatemeses are not uncommon, so that there are potential hazards from the fibrinogenopenia other than bleeding wounds. After the antivenene, the clotting defect starts to improve within five hours, and the blood clots normally within 48 hours of injecting the antivenene. It is not known if this is an accelerated rate of recovery compared to the natural recovery rate after the injection of venom, but it probably is.

When antivenene is given, the hæmoglobinuria ceases within four to eight hours. Again, the natural course of this toxic effect of the venom has not been observed in the absence of antivenene administration, but the antivenene is probably of value in neutralizing the hæmolysin in the venom.

In a previous paper (Campbell, 1964), it was reported that 21 out of 39 patients receiving intravenous injections of antivenene for snake bite had some form of immediate reaction to the serum. Eleven developed urticaria or an itchy skin; 12, a fever; seven, a rigor; five, a cough; one, an increase in oral secretions; and three more severe reactions, including one case of anaphylactic shock, occurred. Thus, out of a total of 67 patients receiving intravenous administrations of antivenene at the Port Moresby General Hospital over a five-year period, 48% have developed a reaction of one kind or another to the serum; 28% have developed urticaria or pruritus, and 3% potentially fatal anaphylaxis. Some of the reactions were transient, so unless the patient is observed closely during and after the injection of the antivenene, the reaction will not be recorded. I had previously (Campbell, 1964) attributed the heavy albuminuria seen in some cases of snake bite to the antivenene administered. This is wrong—it is more probably due to the effects of either taipan or Papuan black snake venom. The incidence of delayed serum reactions is not known, but at least two patients developed serum sickness.

The administration of antivenene is never to be undertaken lightly. In a hospital, antivenene should only be injected in a room where all equipment to deal with possible serum reactions is on hand and ready for use. Antivenene should not be injected directly into the vein when facilities for inserting an intravenous infusion of fluid are available. Cutaneous tests of serum sensitivity have little practical value, but it is suggested by Trinca (1963) that prior injection of adrenaline and an antihistaminic might reduce the incidence of reactions, but this would not necessarily prevent anaphylaxis occurring.

Once symptoms of envenomation appear, the need for antivenene administration is urgent if paralysis is to be prevented. Even when reactions occur during the injection of the antivenene, after they have been treated, the injection of the serum is to be cautiously continued. If there are no symptoms of snake-poisoning evident, and a reaction occurs to antivenene, the need for the antivenene should be critically reassessed.

Even though there is a long delay between the time of the bite from an Australian venomous snake and the time when the patient is first examined, antivenene in large doses (at least four times and up to 10 times or more the recommended dose) should still be administered. The hope that the paralysis may be reversed may be a vain one, but the patient should not be denied that faint hope. Furthermore, whereas it was formerly thought that extension of the paralysis after 30 hours from the time of the bite was unlikely, extension of the paralysis has since been observed to occur up to 39 and 61 hours after the bite in two patients, and antivenene may help limit this late extension of the paralysis and may still possibly counteract the effects of the thrombase, hæmolysin and other toxic factors in the venom.

SUMMARY

1. Twenty-eight patients with established snake-envenomation were treated with antivenene at the Port Moresby General Hospital over a period of 21 months. The results of the antivenene therapy and side reactions to it are summarized.

2. Severe paralysis produced by death adder venom was rapidly reversed with antivenene. In its response to antivenene, death adder venom appears to differ from taipan and Papuan black snake venoms.

3. If injected within a few hours of the bite of a taipan or Papuan black snake, antivenene is considered in most cases to prevent the development of, or limit the extent of, the muscle paralysis produced by the neurotoxins of the venoms, and to counteract the effects of the hæmolysin and thrombase in the venoms.

4. When injected some time after the bite of a taipan or Papuan black snake, antivenene, in doses up to four

times the recommended dose, would not have prevented death but, in some cases, may have limited the extent of the muscle paralysis which developed.

5. Approximately 39% of patients receiving antivenene had some reaction to it, and 3% developed potentially fatal anaphylactic shock.

6. It is recommended that in the case of bites from Australian snakes with thrombase in their venom (taipan, tiger, brown, common black and Papuan black snakes), if paralysis is present, at least four to 10 times the recommended dose of specific antivenene should be injected intravenously, in the hope that the paralysis may be reversed by the antivenene.

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Apple, potato, spinach, tomato. All are plants used in the daily cuisine of many nations. All are plants that contain deadly poisons. None, however, readily comes to mind as a potential killer. The fact is that most laymen, asked to cite examples of poisonous flora, would name the plants whose sinister reputation has come down through history or literature. Anyone who has even a nodding acquaintance with Greek history recalls that Socrates met his death through the offices of a potion of hemlock. From Shakespeare to Charles Addams, scores of ill-fated characters have been dispatched by a nip of nightshade, a dose of dogbane, a touch of toadstool...

While the formidable effects of poisonous plants cannot be overstated, instances of poisoning to humans usually result from isolated accidents, such as children being attracted to interesting looking shoots, mushrooms, or flowers. Although . . . many fruits and vegetables found at the supermarket or in the garden contain poisons that could cause serious injury or death, there are two inherent protective controls: poison in food plants is generally found in parts of the plants we do not usually eat. When the poison exists in the edible portion, abnormal quantities must be consumed before the toxic effects are felt. Thus, while potato plants contain solanine, which is extremely toxic, it is concentrated in the berries, not the tubers that we eat. Tomato vines also contain solanine. Spinach could conceivably block the absorption of calcium sufficient to meet our minimum needs, but it would have to be eaten in very large amounts in an otherwise low-calcium diet.

Man's normal food preferences, then, actually act as a safeguard. It is only when he deviates from the norm that plant poisons are liable to affect him. An apple a day, for instance, may keep the doctor away, but in the case of one man, who considered the seeds a delicacy, it brought the undertaker. Every time he ate an apple he saved the seeds, and when he had collected a capful, he ate them at one sitting. He died shortly afterward; his treat contained a high concentration of prussic acid.—"Please Don't Eat the Mandragona", *The Sciences*, 1967, 6 : 10 (April).