Unusual Poisons: Socrates’ Curse

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Introduction
This is the first in a series of articles looking at some unusual poisons that may not be familiar to most military health personnel. This is of particular relevance, as soldiers deployed on exercises or operations in rural and remote areas may consider using local vegetation as part of their food supply to supplement normal rations.

Hypothetical case study
The paper is in the form of a hypothetical case history, but is based on symptomatology reported in other case histories.1-3 The subject was a 25 year old Reserve soldier, who had been exercising in a semi-rural area on the outskirts of Perth in Western Australia. He told a fellow soldier that he was going to make a brew from some leaves from a plant growing nearby. He prided himself on his bush food knowledge. The plant looked like parsley or the tops of carrots. He made about 200 grams of the plant into a tea, which he consumed, but complained of a somewhat unpleasant taste. He complained of feeling sick and went to lie down, where he promptly went off to sleep. He awoke about an hour later, was noted to be unable to raise himself from the bed and was breathing slowly. He quickly went back to sleep. An hour later when he was checked, he was unable to be roused, not breathing and pulseless. Attempts at resuscitation were unsuccessful.

Discussion
The unfortunate subject in this hypothetical case has died from coniine poisoning. Coniine, a plant piperidine alkaloid, comes from Spotted or Poison Hemlock (Conium maculatum). Coniine is believed to have been the poison given to Socrates in 399 BC4 and was the poison used to kill Amyas Crale in Five Little Pigs, one of Agatha Christie's Hercule Poirot mysteries.5

This ferny leafed weed grows up to 2-3 metres in height, has purple spots on leaves and stem, a small white flower and a mouse-like smell.1 All parts of the plant are poisonous and the plant is widespread in Australia, particularly in southern moister areas; Asia and North America. Poisoning is rare in Australia, but Drummer et al outlined three fatal cases in Victoria in the early 1990’s.2 Coniine is highly stable to heat and poisonings have been associated with tea made from the leaves.2,6

Coniine has its principal effects on neuromuscular transmission, acting as a non-depolarising blocker. While structurally related to nicotine, its pharmacodynamic effect is similar to curare. The alkaloid initially stimulates and then paralyses the nicotinic receptors and may produce complete neuromuscular block.1,2 It also has a narcotic-like effect, with the victim falling off to sleep before becoming unconscious and unrouseable.2 Rhabdomyolysis and acute tubular necrosis have also been described.3

Symptoms usually appear within 10 to 60 minutes and include nausea, vomiting, salivation, lethargy, narcosis, muscle pains and weakness.1,2 Signs may include tachycardia followed by bradycardia, mydriasis, hypotension, flaccid paralysis, fasciculations and shock.1,7 Death is usually caused by respiratory failure. Supportive laboratory data may include myoglobinuria, elevated muscle enzymes and raised liver function tests.2 Generally, there is no long-term renal or liver damage in survivors.8

Medical management is supportive. In cases of known ingestion, the treatment is oral activated charcoal, usually after airways protection.7 In rare cases, gastric lavage may be considered, although prior airways protection is essential. As there is no specific antidote, respiratory and renal support should be initiated rapidly.1 This should include adequate hydration and management of renal failure and rhabdomyolysis as required.

Diagnosis would generally be clinical and dependent on the described history, signs and symptoms. The differential diagnosis should include Nicotine poisoning, Golden Chain Tree (Laburnum anagyroides) poisoning; Poison Hemlock (Conium maculatum) poisoning or Curare poisoning. The generalised weakness also raises the possibility of snake envenomation and possibly organophosphate poisoning.

Nicotine poisoning would need to be excluded; however, tobacco plants are an unusual bush plant,
there was no history of ingestion of cigarettes, the nausea and vomiting would be more pronounced and muscular paralysis is generally associated with very large doses. The Golden Chain Tree (Laburnum anagyroides), which contains the alkaloid cystisine, is principally seen in Europe and North America but is occasionally seen in Perth gardens, although it is also unlikely to be in the bush. Poisoning is usually associated with the consumption of the seed, but all parts of the plant are toxic. Gastrointestinal symptoms are likely to be more pronounced. Curare is very rapid in its effect, is harmless if swallowed and its source, the Strychnos toxifera plant, would be highly unusual in the bush around Perth. A history of contact with snakes or organophosphates has not been elicited in this example and would be expected if either were playing a significant role.

So are most poisonings accidental or intentional? Despite the references to hemlock in history, mystery novels and some homeopathic texts, most cases are accidental ingestions of a plant that is sometimes mistaken for parsley, carrots, parsnip (the roots) or anise.

For the military health practitioner deployed with troops to rural areas, consideration should be given to warning personnel about using local vegetation in preparing meals, even when it may look like a food they know, and being aware that this could be a possible diagnosis when someone presents with symptoms of acute poisoning. Rapid gastric decontamination may prevent death but concurrent rhabdomyolysis and acute tubular necrosis may need to be managed.

References