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## **PERCEPTION OF PAIN ASSOCIATED WITH 1080 POISONING**

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### **Introduction**

With increasing attention being paid to animal welfare and the environment, methods of controlling pest animals are being more closely examined. Some people are totally opposed to the use of poisons, and are lobbying for the total banning of sodium fluoroacetate (compound 1080) in Australia. Whilst this body of environmentalists has been generally convinced that 1080 is not an indiscriminate poison and that it is not environmentally dangerous, it is more difficult to convince them that its actions are humane. Users of 1080 need to be more informed on the humane aspects so that they may respond to these allegations. An attempt is made here to elucidate the relationship between 1080 poisoning and associated pain by reviewing relevant literature.

### **Action of 1080**

Fluoroacetate is converted in mitochondria to erythrofluorocitrate which inhibits the enzymes (aconitase and succinic dehydrogenase) responsible for catalysing citrate and succinate metabolism, which blocks the Krebs cycle. Energy reserves are depleted and cellular function impaired, resulting in gross disorders of organs and organ systems (Kun 1982). In some animals, especially herbivores, the major effect of fluoroacetate is in the cardiac system, whereas in others, especially carnivores the central nervous system is the major organ affected.

### **Effect on the cardiac system**

Symptoms of poisoning where the heart is involved include progressive lethargy and ataxia. A small proportion of rabbits develop a sudden convulsive tonic-clonic seizure, often with a squeal (Chenoweth and Gilman 1946, Chenoweth 1949, Meldrum *et al.* 1957.) Death follows from ventricular fibrillation. McIlroy (1982) reported 1080 poisoning in Bennett's wallabies (*Macropus rufogriseus*) commonly involved convulsions and kicking and running movements while lying on the ground. The convulsions and squealing are seen by some to be signs of pain.

Stress is placed on the heart through the action of fluoroacetate in reducing cellular metabolism. This leads to poor cardiac performance and ventricular fibrillation (Chenoweth and Gilman 1947). Ventricular fibrillation results in an immediate cessation of blood flow to all organs including the brain, producing cerebral anoxia. Consciousness is lost in 10 seconds after cerebral anoxia, but is reversible if anoxia does not exceed 2 minutes. If the heart spontaneously reverts to normal and blood flow to the brain resumes, consciousness is regained and the animal soon becomes ambulatory. With anoxia comes loss of electrical activity within the cortex, followed 2 to 5 seconds later

with clonic muscular convulsions (Sugar and Gerard 1938). The squeals occur during the convulsions (Meldrum *et al.* 1957), that is, while the animal is unconscious. Nelson (cited by Batcheler 1978) reported that squeals and convulsions in rabbits did not unduly disturb nearby rabbits. Adjacent rabbits did not seem to associate this activity with fear or pain. Convulsions seen in fluoroacetate poisoning in rabbits and wallabies, and squeals in rabbits, are thought to occur when the animal is unconscious, and feels no pain.

### **Effect on the central nervous system**

The Universities Federation of Animal Welfare (1989) describes the signs of pain in dogs as appearing quiet and less alert. In severe pain they will lie still or adopt an abnormal position to minimise discomfort. Spontaneous barking is unlikely. In dogs, 1080 poisoning is seen as the sudden appearance of hyperexcitability and abrupt bouts of barking followed by alternating convulsions and running movements. The dog may recover and act normally, but relapses into convulsions. Barking and panting persist during clonic phase of the convulsive period. Breathing becomes rapid but laboured, and death is the result of repeated and prolonged convulsions of the respiratory centre (Chenoweth and Gilman 1946, Chenoweth 1949). These symptoms of poisoning are extremely disturbing to the observer, and are readily interpreted as expressions of pain (Nichols *et al.* 1949).

It is pertinent to relate the symptoms of fluoroacetate poisoning in dogs with similar conditions in humans. Fluoroacetate poisoning in humans involves stimulation of the central nervous system, with clinical signs of anxiety, agitation, nausea and generalised tonic-clonic convulsions. No pain was reported in patients by Gadjusek and Luther (1950) or Reigart *et al.* (1975). Williams (1948) became poisoned while mixing 1080 powder. He reported tingling sensations around the mouth and nasal passages, extending to the arms and legs. There was spasmodic contractions of the voluntary muscles prior to unconsciousness after 2.5 hours. He had no recollection of pain at this time.

The early signs of barking and aimless running have been explained by several authors to depict a state of unawareness of the dog's predicament or surroundings or human presence, suggestive of hallucinations (Chenoweth 1949, Peters 1973, Batcheler 1978). Chenoweth and St. John (1947) demonstrated the electroencephalographs of dogs dosed with 1080 displayed cerebral dysrhythmias identical to those found in *grand mal* and *petit mal* epileptic seizures. *Grand mal* seizures in man are associated with loss of consciousness, whereas in a *petit mal* seizure the patient "loses contact with the environment". This is similar to the state of unawareness ascribed to 1080 poisoned dogs.

Fluoroacetate poisoning has also been likened to hyperinsulinism. In each case the cause is a depletion of energy in cells. Hyperinsulinism leads to mental disorientation, convulsions and loss of consciousness (Tortora and Anagnostakos 1984). Kun (1982) considered that, as in all types of convulsive seizures, loss of consciousness occurs, and death from 1080 poisoning is not unusually troublesome. Chenoweth and Gilman (1946) reported that extreme central nervous system stimulation is shown even while the dog is anaesthetised, indicating that convulsions may occur during unconsciousness. There are enough similarities between fluoroacetate poisoning, epilepsy and hyperinsulinism in

humans to conclude the symptoms of central nervous system stimulation caused by fluoroacetate poisoning in dogs are not associated with pain.

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