

## Aldicarb poisoning of dogs and cats in Gauteng during 2003

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### ABSTRACT

A survey of aldicarb poisoning in companion animals was conducted by posting questionnaires to all private practitioners in Gauteng Province, South Africa. The survey was designed to determine the percentage of aldicarb cases seen, treatment regimen, clinical signs observed, proposals for preventative actions and more effective treatments. Other questions included duration of treatment, survival rate, cost to clients, *post mortem* findings and reasons for poisonings.

**Key words:** aldicarb, cats, dogs, poisoning, treatment.

Verster R S, Botha C J, Naidoo V, Van Schalkwyk O L. **Aldicarb poisoning of dogs and cats in Gauteng during 2003.** *Journal of the South African Veterinary Association* (2004) 75(4): 177–181 (En.). Department of Paraclinical Sciences, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa.

In order to better gauge the incidence of aldicarb poisoning in the country, a retrospective survey was conducted in Gauteng Province, South Africa.

### MATERIALS AND METHODS

A questionnaire, designed to gain information on the incidence of aldicarb poisoning in dogs and cats, was compiled and sent to all veterinary facilities in Gauteng registered with the South African Veterinary Council. Veterinary practitioners were requested to provide an estimate of their total caseload and the related incidence of organophosphor and/or carbamate poisonings with specific focus on aldicarb poisonings. They were also asked to indicate how many suspected aldicarb cases were confirmed after submitting specimens to the Onderstepoort Veterinary Institute or other laboratories for chemical analysis.

The veterinarians were also requested to provide information on the frequency of clinical signs observed and their treatment regimen by ticking boxes marked *always*, *often*, *sometimes* or *never*. Clinical signs listed in the questionnaire included tremors, salivation, emesis, miosis, diarrhoea, urination, dyspnoea, bradycardia and seizures. They were asked to point out any other clinical signs not listed. The treatment options included administration of atropine, activated charcoal, fluid therapy, enzyme reactivators, diphenhydramine, oxygen therapy, induction of emesis or gastric lavage and no treatment, *i.e.* when euthanasia is performed. The veterinary practitioners were also asked to propose any other effective treatment.

Other specific questions focused on average duration of treatment, cost of treatment, survival rate, *post mortem* lesions, reasons for poisoning, suggested preventative measures, perceived seasonal occurrence and whether there was an increase in the incidence of poisonings.

All the data from the returned questionnaires were captured in Microsoft Access. The geographical maps were compiled using ArcInfo (ESRI, New York). Practice locations were determined by geocoding their street addresses through the web service AfriGIS ([www.afrigis.co.za](http://www.afrigis.co.za)). Density determination was done by the Kernel method (bandwidth: 5 km, cell

### INTRODUCTION

Pesticides are frequently and widely used to protect crops from various pests. Annually, 500 000 tonnes are produced in the United States of America, representing over 900 compounds, which are formulated into 25 000 registered products<sup>9</sup>.

Both the organophosphors and carbamates are important and widely available agricultural pesticides in South Africa. Following exposure in animals the carbamates react with the serine group of acetylcholinesterase to yield a carbamylation of the serine hydroxyl group<sup>20</sup>. This carbamylation is reversible and the carbamylated complex hydrolyses in time<sup>13,20</sup>. On the other hand, the organophosphor compounds phosphorylate acetylcholinesterase, but unlike the carbamates, the inhibition becomes irreversible after 24–48 hours<sup>12</sup>.

The inhibition of acetylcholinesterase promotes the accumulation of acetylcholine, which initially excites and then paralyzes transmission at muscarinic cholinergic synapses in the central nervous system, parasympathetic nerve endings and a few adrenergic nerve endings, such as sweat and salivary glands<sup>12</sup>. The nicotinic cholinergic receptors at somatic nerve endings and the ganglionic synapses of autonomic ganglia are also affected<sup>12</sup>.

Clinically, muscarinic-induced effects include hypersalivation, lacrimation, urination, diarrhoea, bradycardia, bronchoconstriction with excess bronchial secretions and miosis<sup>3,8,9</sup>. Nicotinic effects manifest as tremors, muscle stiffness, weakness and paralysis<sup>3,8,9</sup>. The muscular hypertonia, tremors and convulsions can lead to exertional rhabdomyolysis<sup>16</sup>. Mortalities are commonly attributed to respiratory failure<sup>8,9</sup>.

Aldicarb ('Temik', Bayer CropScience), an oxime carbamate insecticide and nematocide, is registered in terms of the Fertilisers, Farm Feeds, Agricultural and Stock Remedies Act, 1947 (Act 36 of 1947). Aldicarb is one of the most toxic pesticides, with a rat oral LD<sub>50</sub> of 0.93 mg/kg<sup>21</sup>. Worldwide, several incidents of aldicarb poisoning have been reported<sup>6,10,11,15</sup> and malicious poisoning of dogs and cats with aldicarb occurs frequently in South Africa. The Division of Toxicology, Onderstepoort Veterinary Institute (OVI) confirmed 72 cases in 1998, 67 cases in 1999 and 72 cases in 2000; this increased to 115 in 2001 and 114 in 2002. The majority occurred in dogs and cats, but sporadic occurrences in cattle, birds, monkeys and antelope were also reported. In 2003, 97 cases of aldicarb poisoning were confirmed which included 32 dogs and 6 cats in the province of Gauteng (J P J Joubert, ARC-Onderstepoort Veterinary Institute, pers. comm., 2004). However, these official figures are not considered to be representative of the actual number of aldicarb poisonings, as numerous incidences of malicious poisoning have been reported in the media.

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Received: September 2004. Accepted: October 2004.

size: 1 km<sup>2</sup>) using the Spatial Analyst extension to ArcInfo.

## RESULTS

Of the 315 questionnaires posted, 97 were returned and 12 were not delivered. However, 14 of the completed questionnaires were submitted from outside the borders of Gauteng and were excluded. Thus, 83 out of 289 questionnaires were included, furnishing a response of 28.7 %.

Only one third (34 %) of respondents indicated the total number of all clinical cases presented to their clinics during 2003. The percentage of suspected aldicarb cases as a proportion of all cases ranged from 0.05–2.6 % for dogs and 0.09–3.33 % for cats. One clinic estimated an incidence of 6 % (3 aldicarb cases out of 50 feline patients in total), which is much higher than all the other veterinary practices (Figs 1–4). A quarter of respondents (26.5 %) sometimes submitted samples for laboratory confirmation of an aldicarb diagnosis.

Frequency of clinical signs observed as reported by the veterinary practitioners are depicted in Fig. 5.

The majority of veterinarians always observed tremors and salivation. Emesis, seizures, bradycardia, dyspnoea and miosis were also often seen. Although diarrhoea was frequently observed, excessive urination was only occasionally recorded. In addition, paresis or paralysis was also noted.

Most animals suffering from suspected poisoning were treated with atropine. Intravenous fluids and electrolyte therapy as well as activated charcoal were also used frequently. The enzyme reactivators and diphenhydramine were seldom administered (Fig. 6).

Other treatments include liver supportive therapy (thioctic acid), vitamin B complex, benzodiazepine sedatives (diazepam) and intravenous anaesthetic agents (pentobarbitone). The clinicians also suggested the use of the bronchodilator, aminophylline and intravenous colloids and loop diuretics (furosemide) to alleviate pulmonary oedema. Non-specific treatments such as anti-inflammatory agents and analgesics were also sometimes used.

Two thirds of respondents estimated a 50–75 % survival rate following treatment, which could extend from 1–7 days, with probability of survival increasing in animals when treatment is initiated as soon as possible. The average cost of treatment varied from ZAR500.00 to 1500.00 and was related to the duration of hospitalisation and range of treatments.

Pet owners were advised of the following preventative measures: keep dogs

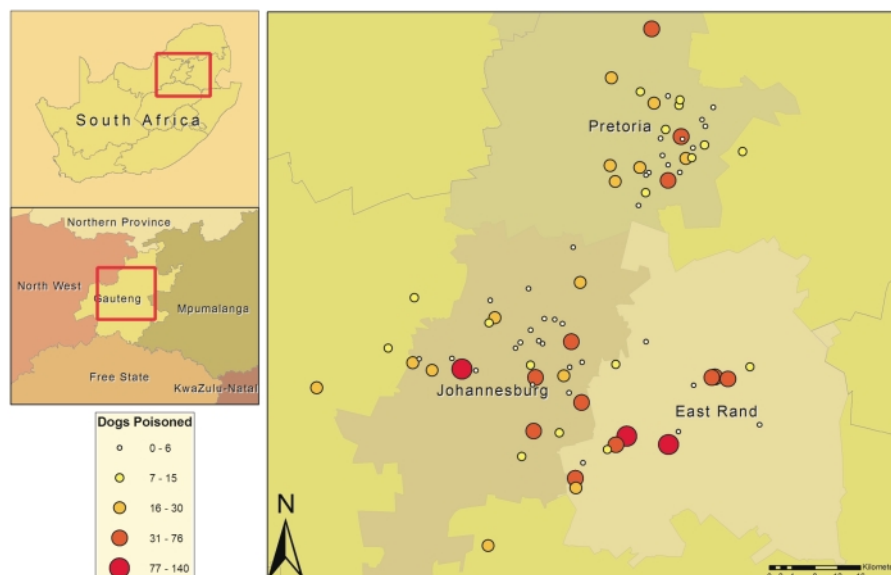


Fig. 1: Estimated number of aldicarb cases in dogs.

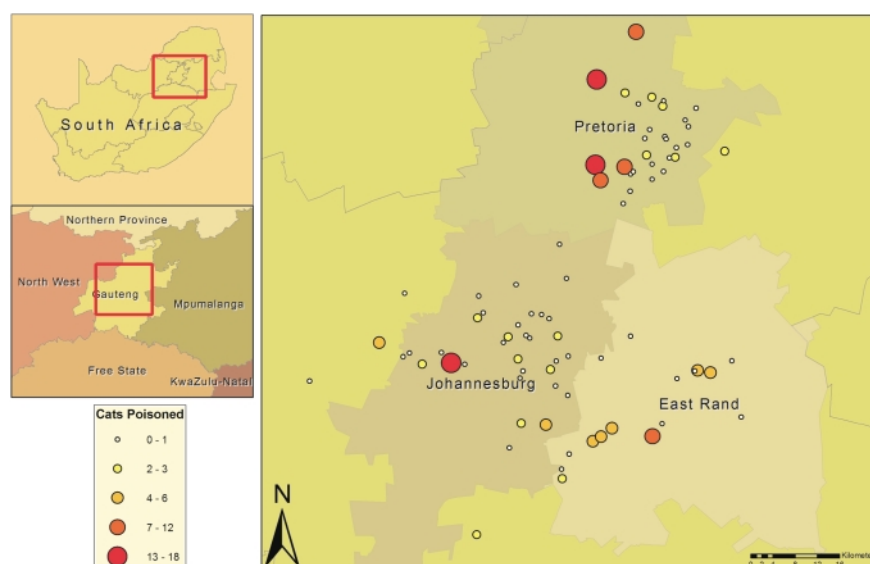


Fig. 2: Estimated number of aldicarb cases in cats.

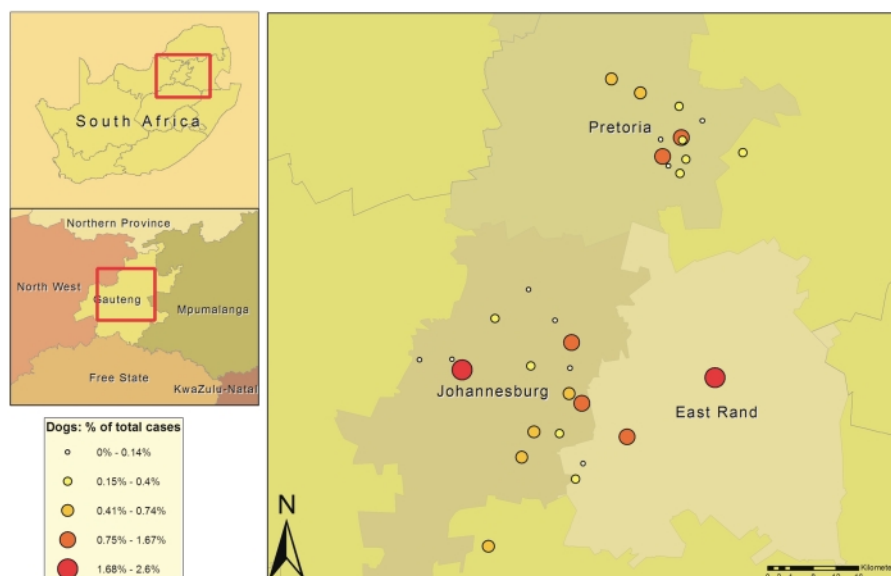


Fig. 3: Aldicarb poisoning as percentage of total caseload in dogs.

inside at night or in back yard; feed dogs at night to thwart ingestion of baits; obedience training for dogs to prevent food acceptance from strangers; check on the animals and for unfamiliar food in the garden early in the morning and do not leave vehicles and valuable items outside. The veterinarians are of the opinion that the incidence of poisoning should not be published in the local media, that an aversive substance could be included in the aldicarb granular formulation, that the sale of aldicarb to unauthorised persons should be prohibited and that the law should be enforced.

No seasonal occurrence was noticed with 95 % of respondents indicating that poisoning occurred throughout the year. However, there was an impression that cases of malicious poisoning increased during holiday periods.

Few respondents conducted *post mortem* examinations, but when it was performed the following macroscopic findings were noted: generalised congestion, petechial haemorrhages, lung oedema, enteritis and aldicarb granules in stomach.

Thirty-three respondents thought that there was an increase in the number of aldicarb cases, but 34 felt there was no increase during 2003. Sixteen respondents were reluctant to venture an opinion (Fig. 7).

The majority of veterinarians (80 %) indicated that they thought criminal intent was the main reason why animals were poisoned. Most veterinarians indicated that animals were presented within 1–2 hours after owners noticing symptoms, but delays can occur due to transport problems.

## DISCUSSION

The survey was conducted in Gauteng, the most populous and affluent province of South Africa, and although it may not accurately reflect the situation in the rest of the country, it is a good indicator of the occurrence of aldicarb poisoning in pets.

Veterinarians in the greater Pretoria area reported that most cases in dogs occur in the eastern suburbs and parts of Centurion. The majority of cases observed in dogs in the greater Johannesburg area occur in the central and eastern areas and on the East Rand. It appears that Roodepoort, Benoni and Boksburg are experiencing a high incidence of malicious poisoning (Figs 1, 3).

It is, however, important to note that the density distribution (Fig. 4) only reflects the density of cases as reported by the respondents. Areas with poor response rates could therefore still have had a high density of cases, although it could not be

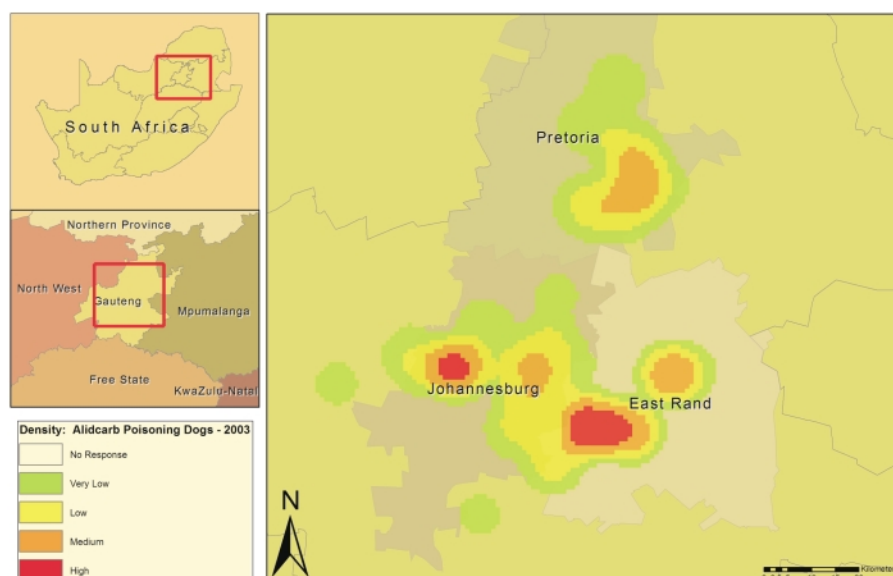


Fig. 4: Density distribution of aldicarb poisoning in dogs.

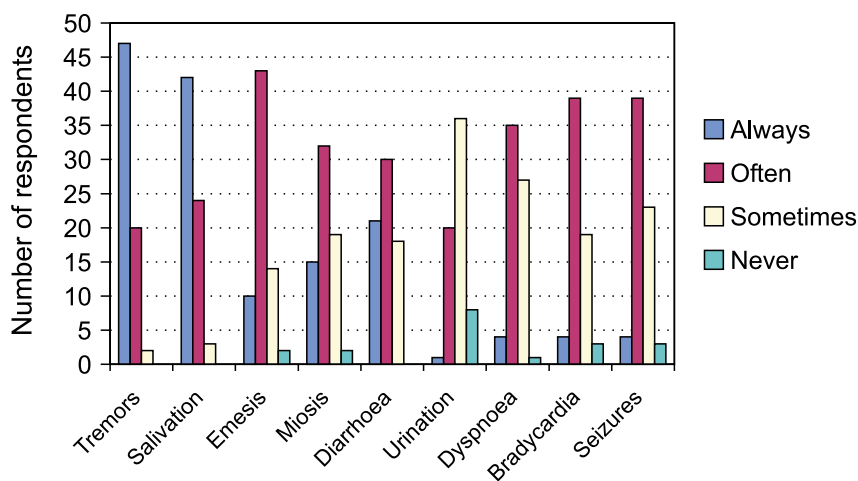


Fig. 5: Clinical signs observed by veterinarians.

accurately reflected on the map, due to the lack of data.

The incidence of poisoning in cats follows a different pattern and they are probably often unintentionally poisoned

when bait is placed out (Fig. 2).

However, the exact prevalence of aldicarb poisonings in dogs and cats will most likely never be fully known, as not all cases are presented to veterinarians.

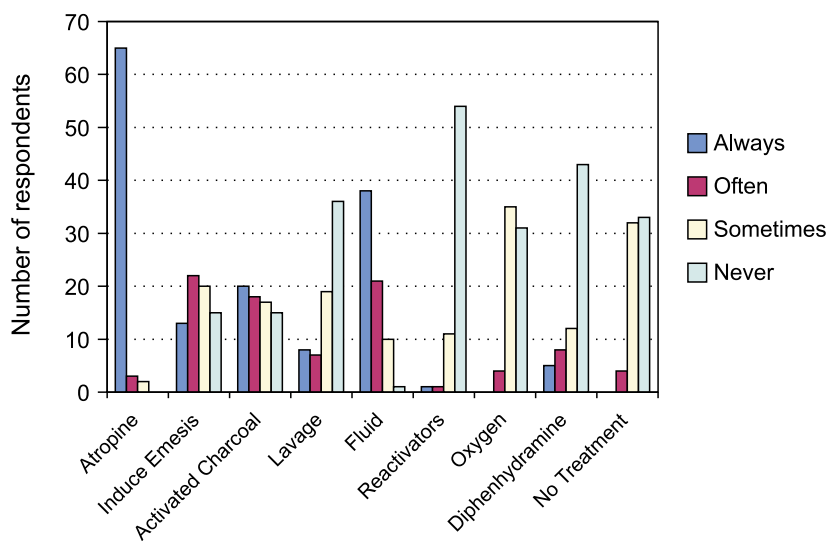


Fig. 6: Treatment regimen of veterinary practitioners.

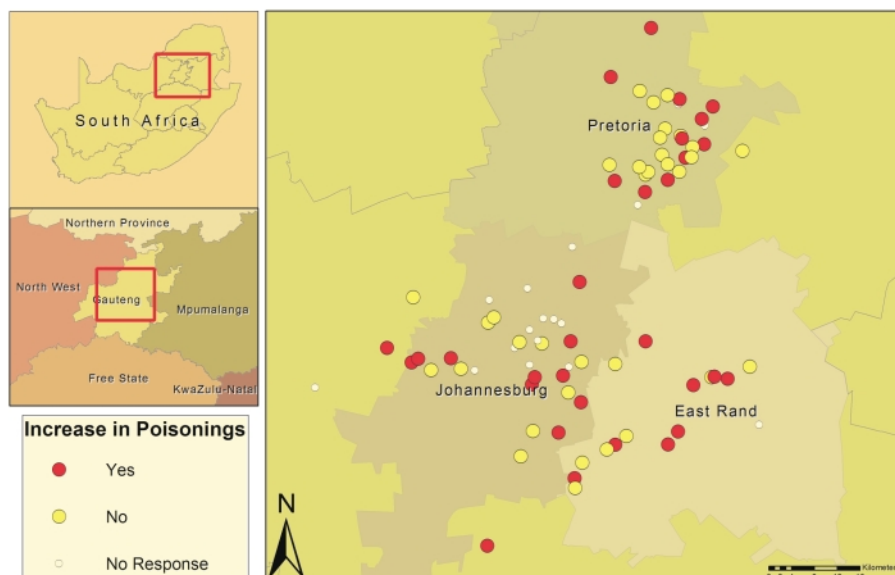


Fig. 7: Estimated increase in aldicarb cases reported by veterinarians.

Chemical analysis also adds substantially to the cost to the client, therefore only some veterinarians occasionally submit samples to confirm their diagnosis. In addition, malicious poisonings of dogs and cats are also not reported to the police.

The cost of treatment is expensive with no guarantee of a successful outcome. Current treatments seem to be reasonably successful, but hospitalisation with intensive therapy is often necessary. Some owners might request euthanasia for financial considerations or to prevent further suffering.

As can be expected, the majority of veterinarians administer atropine. When poisoning induced by an acetylcholinesterase inhibitor is suspected, the parenteral administration of atropine at 0.1–0.2 mg/kg<sup>9,14</sup> in dogs and cats is indicated. Atropine, a muscarinic antagonist, has no effect on nicotinic receptors and will not counteract muscle tremors, weakness or paralysis<sup>3,9</sup>. Diphenhydramine dosed at 1–5 mg/kg *per os* every 6–8 hours may be useful to reverse the nicotinic effects<sup>4,5,9</sup>. Gauteng veterinarians only seldom use diphenhydramine.

The use of enzyme reactivators in carbamate poisoning is controversial as the acetylcholinesterase inhibition is reversible and the enzyme reactivates spontaneously<sup>9,13,17</sup>. On the other hand, the enzyme reactivators are useful in organophosphor poisoning, but only if administered before ‘aging’ occurs, thus within 24 hours or preferably within the first 12–18 hours<sup>17</sup>.

Drugs such as phenothiazine tranquilizers, benzodiazepine sedatives, and aminoglycoside, clindamycin and lincomycin antibiotics<sup>3,9,17</sup> are contraindicated as they have neuromuscular blocking

properties or compete for esterase enzymes. Central nervous system depressants such as the barbiturates and morphine must also be avoided, due to their respiratory depression tendencies<sup>9</sup>. Aminophylline should preferably not be administered as it has analeptic properties, which could exacerbate central nervous system stimulation<sup>19</sup>.

Decontamination procedures such as induction of emesis and/or gastric lavage, in conjunction with adsorbents such as activated charcoal, can be effective to limit gastro-intestinal absorption. Activated charcoal, 1–4 g/kg<sup>1,2,7</sup> dosed orally, is an inert and safe compound. A saline cathartic should be administered after half-an-hour as activated charcoal becomes stationary in the gastro-intestinal tract and slowly releases the adsorbed toxin<sup>2</sup>.

Pets that have died from suspected poisoning should be considered hazardous and every attempt should be made to ensure the correct disposal of the carcass. The remains should not be collected by municipal services and disposed of on municipal dumping sites, as there are always carrion-eating birds. Incineration is probably the best method of disposal of a poisoned animal.

To prevent intoxication, the manufacturer of ‘Temik’ has included several safety measures in this aldicarb formulation (P Fourie & R Jones, Bayer CropScience, pers. comm., 2004). The ‘Temik’ granules, which contain 15 % aldicarb, are sieved to remove dust and lumps, thus limiting the inhalation risk. The granules are coated with a flow agent to assist with agricultural application and to decrease skin contact, thus preventing percutaneous absorption. ‘Temik’ granules also contain an outer layer of ‘Bitrex’

(denatonium benzoate), a strong, bitter agent to discourage ingestion<sup>18</sup>.

The product is registered under Act 36 of 1947, as a restricted use pesticide. Under this Act, only a qualified dealer, registered as an Aldicarb Pest Control Officer, may sell products containing aldicarb. This dealer must have completed suitable and thorough training. Farmers may only purchase ‘Temik’ after completing similar training courses and examinations. All storage facilities, including farm stores, must be inspected and comply with set standards. In addition, each ‘Temik’ container is identified by a unique serial number allowing the details of supply from site of manufacture to final user to be traced.

Despite all the legal requirements and control measures, some ‘Temik’ is still obtained illegally and used for unlawful purposes. Criminal intent is the most important reason provided by the majority of veterinary practitioners for cases of aldicarb poisoning.

The illegal possession of aldicarb will continue to be a major cause of concern, but the economic benefits gained by the registered usage of ‘Temik’ on potatoes, citrus and other crops, are such that ‘Temik’ is recognised as essential for agricultural production. The solution is thus, not to withdraw an excellent pesticide from the market, but to keep it away from unauthorised persons by enforcing the law.

## ACKNOWLEDGEMENTS

The financial contributions received from Bayer CropScience and the Faculty of Veterinary Science are gratefully acknowledged. The authors thank all the Gauteng private veterinarians who completed and returned the questionnaire.

## REFERENCES

1. Beasley V R, Dorman D C 1990 Management of toxicoses. *The Veterinary Clinics of North America: Small Animal Practice* 20: 307–337
2. Buck W B, Bratich P M 1986 Activated charcoal: preventing unnecessary death by poisoning. *Veterinary Medicine: Food Animal Practice* 81: 73–77
3. Campbell A, Chapman M 2000 *Handbook of poisoning in dogs and cats*. Blackwell Science, Oxford: 102–105
4. Clemmons R M, Meyer D J, Sundlof S F, Rappaport J J, Fossler M E, Hubbell J, Dorsey-Lee M R 1984 Correction of organophosphate-induced neuromuscular blockade by diphenhydramine. *American Journal of Veterinary Research* 45: 2167–2169
5. Cordoba D, Cadavid S, Angulo D, Ramos I 1983 Organophosphate poisoning: modifications in acid base equilibrium and use of sodium bicarbonate as an aid in the treatment of toxicity in dogs. *Veterinary and Human Toxicology* 25: 1–3
6. De Bosschere H, Baert K, Ducatelle P 1999

- Aldicarb intoxications in dogs and cats: A retrospective study. *Vlaams diegeneeskundig Tijdschrift* 68: 148–152
7. Dorman D C 1995 Emergency treatment of toxicoses. In Bonagura J D, Kirk R W (eds) *Kirk's current veterinary therapy* (12th edn). W B Saunders, Philadelphia: 211–217
  8. Ecobichon D J 2001 Toxic effects of pesticides. In Klaasen C D (ed.) *Casarett and Doull's toxicology: the basic science of poisons* (6th edn). McGraw-Hill, New York: 763–810
  9. Fikes F D 1990 Organophosphorous and carbamate insecticides. *Veterinary Clinics of North America: Small Animal Practice* 20: 353–367
  10. Frazier K, Hullinger G, Hines M, Liggett A, Sangster L 1999 162 cases of aldicarb intoxication in Georgia domestic animals from 1988–1998. *Veterinary and Human Toxicology* 41: 233–235
  11. Grendon J, Frost F, Baum L 1994 Chronic health effects among sheep and humans surviving an aldicarb poisoning incident. *Veterinary and Human Toxicology* 36: 218–223
  12. Haddad L M 1983 The organophosphate insecticides. In Haddad L M, Winchester J F *Clinical management of poisoning and drug overdose*. W B Saunders, Philadelphia: 704–710
  13. Haddad L M 1983 The carbamate, organochlorine and botanical insecticides; insect repellents. In Haddad L M, Winchester J F *Clinical management of poisoning and drug overdose*. W B Saunders, Philadelphia: 711–712
  14. Hansen S R 1995 Management of organophosphate and carbamate toxicoses. In Bonagura J D, Kirk R W (eds) *Kirk's current veterinary therapy* (12th edn). W B Saunders, Philadelphia: 245–248
  15. Kerr L, Pringle J K, Rohrbach B W, Edwards W C, Offut J E 1991 Aldicarb toxicosis in a dairy herd. *Journal of the American Veterinary Medical Association* 198: 1636–1639
  16. McEntee K, Poncelet L, Clercx C, Henroteaux M 1994 Acute polymyopathy after carbamate poisoning in a dog. *Veterinary Record* 135: 88–90
  17. Meerdink G L 1989 Organophosphorus and carbamate insecticide poisoning in large animals. *The Veterinary Clinics of North America: Food animal Practice* 5: 375–389
  18. Mullins M E, Horowitz B Z 2004 Was it necessary to add bitrex (denatonium benzoate) to automotive products? *Veterinary and Human Toxicology* 46: 150–152
  19. Osweiler G D 1996 *Toxicology*. Williams & Wilkens, Philadelphia. 307–308
  20. Stevens J T, Breckenridge C B 2001 Crop protection chemicals. In Hayes A W (ed.) *Principles and methods of toxicology* (4th edn). Taylor & Francis, Philadelphia: 583–591
  21. Tomlin C 1994 *The pesticide manual* (10th edn). Crop Protection Publications, Farnham: 25