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 products3. Both the organophosphorus 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 group3. This carbamylation is reversible and the carbamylated complex hydrolyses in time12,20. On the other hand, the organophosphorus compounds phosphorylate acetylcholinesterase, but unlike the carbamates, the inhibition becomes irreversible after 24–48 hours12.

The inhibition of acetylcholinesterase promotes the accumulation of acetylecholine, which initially excites and then paralyses transmission at muscarinic cholinergic synapses in the central nervous system, parasympathetic nerve endings and a few adrenergic nerve endings, such as sweat and salivary glands13. The nicotinic cholinergic receptors at somatic and autonomic ganglia are also affected12.

Clinically, muscarinice-induced effects include hypersalivation, lacrimation, urination, diarrhoea, bradycardia, bronchoconstriction with excess bronchial secretions and miosis3,8,9. Nicotinic effects manifest as tremors, muscle stiffness, weakness and paralysis3,8. The muscular hypertonia, tremors and convulsions can lead to exertional rhabdomyolysis14. Mortalities are commonly attributed to respiratory failure15,16. 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 LD₅₀ of 0.93 mg/kg17. Worldwide, several incidents of aldicarb poisoning have been reported18,19,20 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 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.

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, miosis, diarrhea, 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). Density determination was done by the Kernel method (bandwidth: 5 km, cell
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 (thiocolnic 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 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.

![Density distribution of aldicarb poisoning in dogs.](image)

![Clinical signs observed by veterinarians.](image)

![Treatment regimen of veterinary practitioners.](image)
The use of enzyme reactivators in carbamate poisoning is controversial as the acetylcholinesterase inhibition is reversible and the enzyme reactivates spontaneously. On the other hand, the enzyme reactivators are useful in organophosphorus poisoning, but only if administered before ‘aging’ occurs, thus preventing pernicious absorption. ‘Temik’ granules also contain an outer layer of ‘Bitrex’ (denatonium benzoate), a strong, bitter agent to discourage ingestion.

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 an 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.

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Book review (continued) — Cattle Plague – A History

Information on susceptibility of wild animal species, specific effects on African game, the role of African game in the epidemiology or rinderpest, and the relation between rinderpest and tsetse flies are dealt with in detail and contains valuable reference material for persons interested in those aspects of rinderpest.

The book quite rightly gives a lot of prominence to the economic and social effects the disease had in Europe, Asia and Africa. In South Africa, the great African rinderpest panzoootic that appeared South of the Zambezi river in 1896, has been described as a national disaster that had profound social effects on the citizens of the former Transvaal who experienced the biggest losses. The wide and diverse ramifications of the disease in Europe and Africa where it was much more epidemic in nature compared to its enzootic appearance in Asia, are put in historical context by the author. The historical facts of the global rinderpest eradication campaign is adequately addressed in the book, from the role that wars and governmental mismanagement played in the resurgence of rinderpest after the 1970s, to current approaches to eradicate the only remaining rinderpest virus, namely lineage 2 virus in the pastoral ecosystem of southern Somali and northern Kenya.

Although several publications dealing with aspects of rinderpest have become available during the last few decades, this book must be rated as the most comprehensive historical review of the disease. The book covers all aspects of rinderpest, including the causative organism (and speculation about the origin of the 1889 outbreak), the clinical disease, the epidemiology, the control of the disease and its socio-economic implications. It can be recommended to anyone who wishes to obtain information relating to any aspect of this disease that is recognised as the most lethal viral disease of cattle known to mankind. Even as a source for general reading, it should provide many hours of interesting reading.

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