Intensive care management of acute organophosphate poisoning

A 7-year experience in the western Cape

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Summary

Organophosphate poisoning (OPP) was an important reason for admission of patients to the respiratory intensive care unit (ICU) at Tygerberg Hospital, Parowvallei, CP, during the period 1979 - 1985; a marked increase in the number of cases was evident over the last 2 years. We retrospectively reviewed the medical records of 61 patients with OPP admitted to the ICU over this 7-year period. Diagnosis was based on the history, clinical manifestations of OPP, and low pseudocholinesterase levels. Suicidal ingestion was the predominant cause of OPP. Of the 61 patients, 46 (75%) were under 40 years of age. In more than 50% of cases the clinical presentation was characterised by classic signs of OPP such as increased secretions, fasciculations and small pupils. In 61% the level of consciousness was disturbed. We retrospectively classified and graded patients on a scale of 0 - 3 on the basis of the initial clinical findings, blood gas values and chest radiographs, in an attempt to facilitate identification of high-risk cases. Patients with grade 3 intoxication (attempted suicide, stupor, partial arterial oxygen pressure (Pa_{O_2}) < 10 kPa and an abnormal chest radiograph — two or more factors present) were more likely to require ventilatory support and stayed in the ICU longer than patients with grades 0 - 2 intoxication ($P < 0,05$). Patients who presented with pulmonary abnormalities (admission chest radiograph abnormal or $Pa_{O_2} < 10$ kPa) also required ventilatory support more frequently than did patients whose chest radiographs and blood gas values were normal on admission. The mortality rate was 16% and most deaths were due to respiratory complications. No correlation could be demonstrated between serum pseudocholinesterase levels and the clinical degree of intoxication.

We conclude that patients should be graded on admission to identify those at risk (grade 3), who require ICU care. Patients with early signs of respiratory involvement ($Pa_{O_2} < 10$ kPa or an abnormal chest radiograph) should also be admitted to an ICU. Atropine should be given early and in adequate doses to minimise nasopharyngeal and bronchial secretions. Preventive measures should focus on better public education, emphasising adequate safekeeping and careful use of organophosphates.

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Inadvertent or suicidal organophosphate poisoning (OPP) represents a serious problem in the western Cape. Organophosphates are considered to be more dangerous to humans than any other agricultural chemical with the exception of paraquat. They are freely available in shops and supermarkets and are used extensively in agriculture. Public awareness of the toxicity of this group of chemicals is limited, and preparations containing organophosphates are frequently stored on farms and in households in a careless and haphazard way.[1,2]

Organophosphates cause poisoning by inhibition of the enzyme cholinesterase with subsequent accumulation of acetylcholine and symptoms relating to overactive cholinergic action. The mechanism of inhibition is phosphorylation of the esteratic locus on the enzyme with the formation of a stable chemical complex which is biologically inactive.[3]

During the 7-year period 1979 - 1985, 61 patients with serious OPP were admitted to the respiratory intensive care unit (ICU) at Tygerberg Hospital, Parowvallei, CP. In 1984 and 1985 there was a marked increase in the number of cases seen — in 1985 OPP was the second most common reason for ICU admission. Correct and early diagnosis of OPP and institution of adequate treatment for serious cases, preferably in an ICU, has therefore become imperative.[4] Recognition of OPP and correct initial treatment in hospitals and by general practitioners, with early referral of serious cases, can be of vital importance in determining prognosis. Treatment of seriously ill patients in an ICU is important for many reasons, including good observation to assess the need for ventilatory support and intensive nursing care.[5] Specialised treatment such as bronchoscopy and mechanical ventilation is also more readily available. Owing to the limited availability of beds in ICUs as well as the high cost involved not all patients with OPP can be admitted, although clinical deterioration can be dramatic and unpredictable and patients may need rapid intubation and mechanical ventilation.[5,6] Clinical features which indicate the severity of poisoning and predict the need for ventilatory support must therefore be identified to facilitate early recognition of patients who should be admitted to an ICU. Such risk factors have not previously been proposed, and in this study we attempted to identify them. The clinical course of the patients admitted to our ICU, complications encountered and the mortality rate and causes of death have also been analysed.

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Patients and methods

A retrospective study was done of 61 patients with OPP admitted to the respiratory ICU during the period 1979 - 1985.

Initial evaluation on admission to the general ward included clinical examination, a chest radiograph, and determination of blood gas and pseudocholinesterase values. The diagnosis of OPP was based on: (*i*) a positive history of intake or exposure; (*ii*) clinical manifestations of OPP; and (*iii*) low serum pseudocholinesterase levels. In all the patients studied the serum pseudocholinesterase values were abnormally low (< 3 KU/l; normal 3 - 8 KU/l) and one or both of the other criteria listed were also present. Poisoning by carbamates could be excluded with reasonable certainty in most cases by the history of intake, clinical assessment, e.g. a smell of organophosphates, and the clinical course of the illness, although a few patients may have ingested both carbamates and organophosphates.

Patients with suspected severe poisoning were referred for admission to the ICU. There were no set criteria for admission, and patients were individually assessed according to the degree of their symptoms and signs of poisoning. Patients admitted to the ICU had one or more of the following: (*i*) history of intake of large quantities of the poison; (*ii*) copious secretions; (*iii*) disturbed level of consciousness; (*iv*) clinical signs of hypoventilation or respiratory obstruction; and (*iv*) abnormal blood gas values (partial arterial oxygen pressure (Pa_{O_2}) ≤ 10 kPa and/or partial arterial carbon dioxide pressure (Pa_{CO_2}) ≥ 6 kPa). Patients who were not admitted to the ICU were observed for at least 72 hours in the general wards and transferred to the ICU if any signs of serious toxicity developed.

To assess the severity of poisoning patients were retrospectively graded according to the clinical features, chest radiograph and arterial blood gas values (Table I).

TABLE I. PROPOSED SYSTEM OF GRADING FOR OPP AS APPLIED TO 61 PATIENTS ADMITTED TO AN ICU*

Grade 0	**Positive history**
(7 patients)	**No signs of OPP**
Grade 1	**Secretions 1+**
(11 patients)	**Fasciculations 1+**
	Normal level of consciousness
Grade 2	**Secretions 3+**
(9 patients)	**Fasciculations 3+**
	Rhonchi, crepitations or hypotension (systolic BP ≤ 90 mmHg)
	Disturbed level of consciousness, but not stuporous
Grade 3	**Attempted suicide**
(34 patients)	**Stupor**
	Pa_{O_2} < 10 kPa
	Chest radiograph abnormal

*Two or more criteria needed for specific grade. If less than 2 use previous grade. BP = blood pressure.

Observation of patients in the ICU centered mainly on the respiratory system and included regular determination of blood gas values, chest radiographs as indicated, and blood and sputum cultures. Determination of serum electrolyte levels, renal and liver function tests and blood counts were done regularly. Criteria for pulmonary infection were at least two of the following: (*i*) raised white cell count; (*ii*) purulent bronchial secretions; (*iii*) positive Gram staining and culture of the sputum; (*iv*) positive blood culture; and (*v*) new pulmonary infiltrate not explained by any other means. Fever alone was not considered a sign of infection.

Treatment

Atropine was given by constant intravenous infusion and titrated to keep the mucous membranes dry and pulmonary secretions minimal. When relapses occurred patients received additional atropine as a bolus and the infusion was accelerated. When atropine toxicity was considered to be present the drug was withdrawn and the clinical response assessed. The infusion rate was decreased when clinical signs of OPP had been absent for 24 hours, and the drug was gradually tapered off on a trial-and-error basis under constant observation until complete withdrawal. Oximes were administered to 29 patients in doses ranging from 250 mg to 5 g over 48 hours, 23 patients being treated with obidoxime and 6 with pralidoxime. Mean doses of obidoxime and pralidoxime over 24 hours were 1,7 g and 0,8 g respectively. Obidoxime was given in relatively large doses (1 g over 8 hours intravenously) to 18 patients as part of a separate study to assess the efficacy of treatment with oximes. The time between ingestion of poison and oxime treatment varied and was unknown in the majority of cases. In 8 cases oximes had been given by general practitioners before admission. Infections were treated with appropriate antibiotics.

Indications for endotracheal intubation and mechanical ventilation were as follows: (*i*) excessive secretions not controlled by atropine and ordinary suction techniques; (*ii*) absent or weak cough reflexes; (*iii*) airway obstruction in unconscious patients; (*iv*) respiratory failure with a Pa_{O_2} of below 8,0 kPa (despite 40% oxygen given by mask) and/or a Pa_{CO_2} above 6,7 kPa; and (*v*) vital capacity < 15 ml/kg. All patients were ventilated with pressure-cycled ventilators in the intermittent mandatory ventilation mode with positive end-expiratory pressure (PEEP). The inspiratory oxygen concentration and PEEP were varied to maintain the Pa_{O_2} above 8,0 kPa and the minute volume to contain the Pa_{CO_2} between 4,0 and 5,5 kPa. In 10 cases a continuous positive airway pressure (CPAP) mask was sufficient to maintain adequate oxygenation.

Files were analysed retrospectively on the patient's discharge or death. Factors predictive of ventilatory support were analysed by the chi-square test and multiple regression analysis. Duration of ICU management was analysed by means of the Mann-Whitney *U*-test. Findings were considered significant at $P < 0,05$.

Results

General findings

The number of cases of OPP necessitating admission to the ICU during 1979 - 1985 is shown in Fig. 1 and illustrates an almost linear increase in 1984/5. Of the patients 39 were male and 22 female. The mean age (± SE) was 33 ± 1,9 years (range 14 - 63 years), the majority of the patients (75%) being under 40 years of age. The group was made up of 50 coloureds, 9 whites and 2 blacks. The most important cause of OPP was attempted suicide

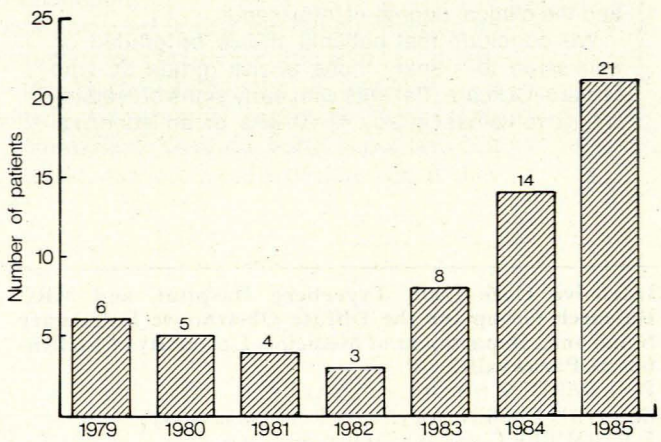

Fig. 1. Number of patients per year admitted to an ICU with OPP, 1979 - 1985.

(37 cases, or 61% of the total group). Accidental ingestion had occurred in 13 cases and occupational contamination in 8 (all in agriculture); in 3 cases the cause was unknown. Nine patients (15%) of the total group who had ingested organophosphate poison accidentally did so because it had been stored in a cooldrink or a liquor bottle.

Clinical features

The most commonly encountered symptoms and signs are shown in Table II. Increased salivation, fasciculations, small pupils and a disturbed level of consciousness were present in more than 50% of patients.

TABLE II. SIGNS AND SYMPTOMS OF ORGANOPHOSPHATE POISONING IN 61 PATIENTS AT PRESENTATION, AS RECORDED IN MEDICAL CHARTS

	No. of patients	%
General examination		
Sweating	**14**	**23**
Salivation	**37**	**61**
Respiratory system		
Tachypnoea (> 30/min)	**24**	**39**
Rhonchi or crepitations	**29**	**48**
Hypoventilation	**12**	**20**
Cardiovascular system		
Tachycardia (> 100/min)	**30**	**49**
Bradycardia (< 60/min)	**13**	**21**
Hypotension (systolic BP < 90 mmHg)	**12**	**20**
Gastro-intestinal system		
Vomiting	**23**	**38**
Diarrhoea	**13**	**21**
Central nervous system		
Disturbed level of consciousness*	**37**	**61**
Miosis	**50**	**82**
Fasciculations	**33**	**54**

*Level of consciousness was graded as follows: 0 = normal; 1 = confused, able to sit/stand (5 patients); 2 = confused, unable to sit/stand (16 patients); 3 = stuporous — no reaction to speech (16 patients).
BP = blood pressure.

Special investigations

The chest radiograph was normal on admission in 44 cases, nonspecific radiological abnormalities being noted in the other 17. These included well-circumscribed areas of opacification, diffuse opacification and in 4 cases features of pulmonary oedema. Radiological changes resolved after periods ranging from 2 days to more than 3 weeks.

In 34 patients (56%) blood gas analysis on admission (fractional inspired oxygen concentration (Fio_2) 0,21) showed a Pao_2 of below 10 kPa, with a mean value for the 61 patients of 10,8 ± 0,5 kPa (range 6,0 - 15,7 kPa). The mean $Paco_2$ was 5,1 ± 0,2 kPa (range 3,7 - 9,8 kPa) and there were 9 patients with a Pao_2 of below 8,0 kPa and a $Paco_2$ of above 6,7 kPa.

Serum pseudocholinesterase levels were markedly low (< 0,5 KU/l; normal 3 - 8 KU/l) in 46 patients. The remaining 15 patients all had levels below 1,5 KU/l. Recovery to normal was unpredictable, and levels were still below 3 KU/l in 37 patients who had recovered clinically with no residual signs of poisoning for at least 72 hours and who had been discharged from hospital.

Grading

The results of our system of grading are shown in Table I. The majority of the patients (34) had grade 3 intoxication, with an even distribution of patients in the other grades.

Course and treatment

All patients were treated with a continuous intravenous infusion of atropine. The median dosage required to control the clinical signs of poisoning was 20 mg/24 h (range 10 mg - 1,3 g/24 h). No correlation could be demonstrated between the grading of a patient on admission and the atropine dosage required in the ICU ($r = 0,25$). The mean duration of treatment was 7,1 ± 0,7 days (range 2 - 23 days), with at least one episode of atropine toxicity noted in 16 cases (26%). The toxic signs and symptoms resolved on decreasing the atropine dosage. Twenty-nine patients were treated with oximes, but owing to variations in dosage, time lapse between poisoning and oxime administration and degree of intoxication, no assessment of the efficacy or side-effects of these drugs was attempted.

Ventilatory support with a CPAP mask or endotracheal intubation and PEEP was needed in 34 cases (56%) in spite of optimal oxygenation by mask, atropine therapy and intensive nursing care. Factors that were predictive of a need for ventilatory support are shown in Table III. Ventilation was required by 29 of 34 patients (85%) with a Pao_2 of below 10 kPa but by only 5 of 27 (18%) with a Pao_2 of above 10 kPa ($P < 0,05$), and by 13 of 17 patients (76%) with an abnormal chest radiograph but by only 21 of 44 (47%) with a normal radiograph ($P < 0,05$). Ventilation was required in 77% of cases of grade 3 intoxication (26 of 34 patients) but in only 30% of cases of grades 0 - 2 intoxication (8 of 27 patients) ($P < 0,05$).

TABLE III. FACTORS PREDICTIVE OF VENTILATORY SUPPORT IN 61 PATIENTS ADMITTED TO AN ICU

	Ventilation needed	No ventilation	Significance
Pao_2 < 10 kPa	**29**	**5**	***P* < 0,05**
Abnormal chest radiograph	**13**	**4**	***P* < 0,05**
Grade 3 intoxication	**26**	**8**	***P* < 0,05**
Grades 0 - 2	**8**	**19**	**NS**

The most commonly encountered complications were as follows: (*i*) respiratory infection (36 patients); (*ii*) renal dysfunction (13); and (*iii*) abnormal liver function (12). Clinical features suggestive of septicaemia secondary to respiratory infection developed in 18 patients and could have contributed to dysfunction in other organs. No abnormalities that could be ascribed directly to organophosphates were detected in haematological and clotting studies.

The 61 patients spent a median time of 8,0 days (range 2 - 30 days) in the ICU. Patients with grade 3 intoxication needed ICU care for a median of 10,5 days (range 8 - 30 days), as opposed to a median of 5,0 days (range 2 - 26 days) for the grades 0 - 2 group, a significant difference ($P < 0,05$).

Mortality rate

Of the 61 patients 10 died, giving a mortality rate of 16%. Death was due to respiratory infection with secondary septicaemia in 7 cases, to acute renal and hepatic failure in 2 and to bilateral spontaneous pneumothoraces due to infection in 1.

Discussion

OPP was an important reason for admission to the respiratory ICU at Tygerberg Hospital during the period 1979 - 1985. Only rib fractures and 'shock-lung' syndromes were more frequent causes of admission. The reason for the consistent rise in numbers of patients with OPP admitted during 1983 - 1985 (Fig. 1) is unclear. Young coloured patients under 40 years of age who had attempted suicide were the largest group.

Attempted suicide was the cause of serious OPP in 40 - 70% of cases in two studies reported in southern Africa,[4,7] and our study confirms that it is an important cause of serious poisoning. The extent of acute and chronic poisoning with organophosphates has not been determined in spite of extensive agricultural use of the agent in the western Cape. Perold and Bezuidenhout[8] reported acute intoxication in a cohort of agricultural students at a local college who had experienced windborne exposure. The widespread use in agriculture does contribute to poisoning through deficient control during spraying and especially storage of poisons. The practice of keeping these poisons in liquor and cooldrink bottles should be strongly discouraged because it can lead to accidental ingestion, as was the case in 9 of our patients with serious OPP who came from farms.

A classification based on the degree and seriousness of OPP has been proposed by Namba *et al.*[9] They categorised patients into four groups, with latent, mild, moderate and serious poisoning, on the basis of clinical findings and pseudocholinesterase levels. We did not find this classification adequate for assessing our patients or for predicting the course of their illness because many patients had fluctuating clinical features and rapid deterioration frequently took place within 1 - 2 hours of the initial period of observation. Furthermore, the clinical degree of severity of OPP did not correlate with the serum pseudocholinesterase levels. The grading proposed in this study (Table I) is based on specific clinical findings pertaining mainly to the respiratory and central nervous systems and investigations such as chest radiographs and determination of blood gas values, which are standard procedures for evaluating patients. We found a significant correlation between grade 3 intoxication and the need for assisted ventilation, as well as a significantly longer ICU stay for grade 3 intoxication than for grades 0 - 2. All patients with grade 3 OPP therefore require direct admission to an ICU, where optimal respiratory support and general treatment is available. Classification in groups 0 - 2 may be useful to identify patients with potentially severe OPP (grade 2) who could require ventilatory support. Patients with grade 2 OPP should preferably be transported to an ICU and admitted for observation; those with grades 0 - 1 OPP can be treated in a general ward for at least 72 hours, assessed regularly and transferred to an ICU if clinical deterioration occurs.

The clinical features of OPP have been well described.[3,9,10] The diagnosis can be missed initially if patients present in an unconscious or confused state with tachycardia. In contrast to the classic descriptions of bradycardia, the majority of our patients had a heart rate of more than 100/min (49% tachycardia v. 21% bradycardia). This clinical feature must be kept in mind, because tachycardia was frequently associated with serious OPP. The causes of a fast pulse rate in OPP include: (*i*) hypoxia; (*ii*) preganglionic acetylcholine accumulation in the adrenal medulla with release of adrenaline; and (*iii*) atropine treatment (given in 23 of our cases before admission). Hypotension was observed in some patients and may be due to a central action of organophosphates or to bradycardia. Central nervous system manifestations of OPP are common. A high percentage of our patients (61%) were not fully conscious, and OPP must always be considered in such cases.[6,9] Miosis, which was present in more than 80% of our patients, is a helpful clue to the diagnosis. Other neurological disturbances that have been described include restlessness, dizziness, emotional instability and disturbances of speech,[6,11] as well as more rare acute neurological abnormalities such as ophthalmoplegia, facial paralysis, bilateral pyramidal tract signs and areflexia.[12] Distal, symmetrical, predominantly motor polyneuropathy may occur 1 - 3 weeks after exposure to organophosphates.[13] Fasciculations are caused by the accumulation of acetylcholine at the motor endplate in muscle and were present in 54% of our patients; this may progress to paralysis in serious poisoning.[6] The neurological manifestations of OPP may therefore include a wide variety of abnormalities; in our study the most important and serious finding was a depressed level of consciousness. The associated problems of regurgitation of gastric contents, excessive secretions in the airways and a depressed cough reflex may lead to aspiration and respiratory complications.

Abnormalities on the chest radiograph were uncommon, but if present had serious prognostic implications. A variety of abnormalities were present in our patients, including lobar and non-lobar opacification as well as features resembling those of pulmonary oedema. The initial radiological abnormalities can be ascribed to aspiration, mucus plugs with atelectasis of lung tissue and capillary damage, possibly as a result of tissue hypoxia.[14] Aspiration of gastric contents must always be suspected; the apical segment of the right lower lobe or basal segments of the same lung are usually affected. A number of our patients had opacification in the abovementioned lung segments, probably caused by aspiration due to a depressed level of consciousness. Mechanical ventilation was required by 76% of patients with abnormal chest radiographs on admission, as opposed to 47% of patients with normal radiographs. These cases may represent a subgroup in which there is a higher risk of respiratory infection, ventilatory support is more likely to be required, and the ultimate prognosis is unfavourable.

Blood gas values on admission were abnormal in the majority of our patients; the PaO_2 was below 10 kPa in 34 cases, and almost 86% of the patients in this group required ventilation. Few studies of blood gas abnormalities in OPP are available and our findings indicate that a depressed blood oxygen level may be an early indicator of serious pulmonary involvement. The hypoxia is caused by a combination of factors, including: (*i*) bronchoconstriction; (*ii*) excessive secretions in the airways; (*iii*) pulmonary oedema; and (*iv*) hypoventilation as a result of muscular weakness or depression of the respiratory centre.[15] Regular performance of additional tests of pulmonary function such as the vital capacity and forced vital capacity in 1 second may enable earlier diagnosis of lung or respiratory muscle involvement in OPP.

In our study of 61 cases of serious OPP, involvement of the respiratory system determined morbidity and mortality. Retrospective analysis correlated an abnormal chest radiograph, a PaO_2 of below 10 kPa and grade 3 intoxication with morbidity (respiratory complications and need for mechanical ventilation); if any of the above factors are present in a patient with OPP, admission to an ICU is merited. There were 10 deaths associated with OPP, a mortality rate of 16%. Untreated patients usually die as a result of hypoventilation and excessive secretions in the airways;[16,17] in our patients respiratory infections complicated by septicaemia and multi-organ failure were the major causes of death. Respiratory infections were the result of excessive bronchial secretions, often associated with aspiration of nasopharyngeal secretions or gastric contents. It is important for medical practitioners to assess respiratory function carefully in any patient with OPP admitted to a peripheral hospital, and to consider early referral to a hospital with facilities for bronchoscopy and mechanical ventilation.

Serum pseudocholinesterase levels were measured in all our patients and were not of value in predicting the seriousness of OPP. Values were low in all cases and significantly low ($< 0,5$ KU/l) in more than 75%; low levels can be a sensitive indicator of OPP when the diagnosis is uncertain. Despite this, plasma levels of the enzyme show little correlation with tissue levels and red cell cholinesterase levels may be a better index of clinical poisoning.[18-20] Red cell cholinesterase levels may further be of value during recovery from OPP, and a value of 30% of normal may correlate with clinical recovery and little danger of relapse.[4] This investigation was done in only 1 of our patients and may be of future value in treatment and follow-up.

Treatment with atropine blocks the excessive cholinergic effects of organophosphates, but has no effect on the motor endplate and therefore on muscular weakness. We used continuous infusion and found that dosages needed varied widely, some patients needing as much as 1 000 mg over 24 hours or as little as 10 mg. The rate of administration was assessed by the volume of secretions, which is regarded as the most sensitive indicator of adequate atropinisation.[6,21] The pulse rate was also monitored and atropine administration was increased if it fell below 60/min. In patients with tachycardia due to OPP the rate of atropine administration must be judged on control of secretions. We tapered off atropine administration gradually as the patients' clinical condition improved, the duration of treatment ranging from 2 days to 3 weeks. Oral atropine is well absorbed and may be of value when intravenous administration is no longer needed.[4] The treatment of OPP with 'reactivators' is controversial. The drugs in use are the oximes, which form a compound with the organophosphate that is bound to the cholinesterase enzyme. The organic phosphate is split off and hydrolised with reactivation of the enzyme.[4,22] The oximes are only effective against certain of the organophosphates,[3,6] such as parathion, and must be given as soon as possible before ageing of the enzyme takes place. When ageing takes place an alkyl group is split off the organophosphate, making it resistant to the oximes. Cases have been described in South Africa and elsewhere of dramatic clinical improvement after treatment of OPP with oximes,[23,24] and treatment may be beneficial if the patient presents within 8 hours of being poisoned.

The treatment of OPP by general practitioners in small towns is of great importance, because they often see the patient shortly after poisoning. Maintenance of an open airway and treatment with adequate atropine at an early stage are essential. Dosages of atropine must be such that secretions are kept to a minimum, because this may help to prevent the later respiratory complications of OPP. Practical measures such as washing the patient and gastric lavage before transportation to a bigger centre are also essential and can minimise the effects of poisoning. Indications for immediate referral would include any change in the level of consciousness or signs of pulmonary involvement such as tachypnoea, cyanosis, decreased ventilation of some lung segments, many wet sounds on auscultation, and an abnormal chest radiograph.

In conclusion, OPP is a serious health problem in South Africa and efforts should be directed towards preventive measures. This can be accomplished by creating a greater awareness among the public of the dangers associated with this particular group of insecticides, and by stressing the extremely unpleasant effects of ingesting the poison. Treatment of the condition presents a challenge in general and hospital practice, as well as to intensive care physicians.

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