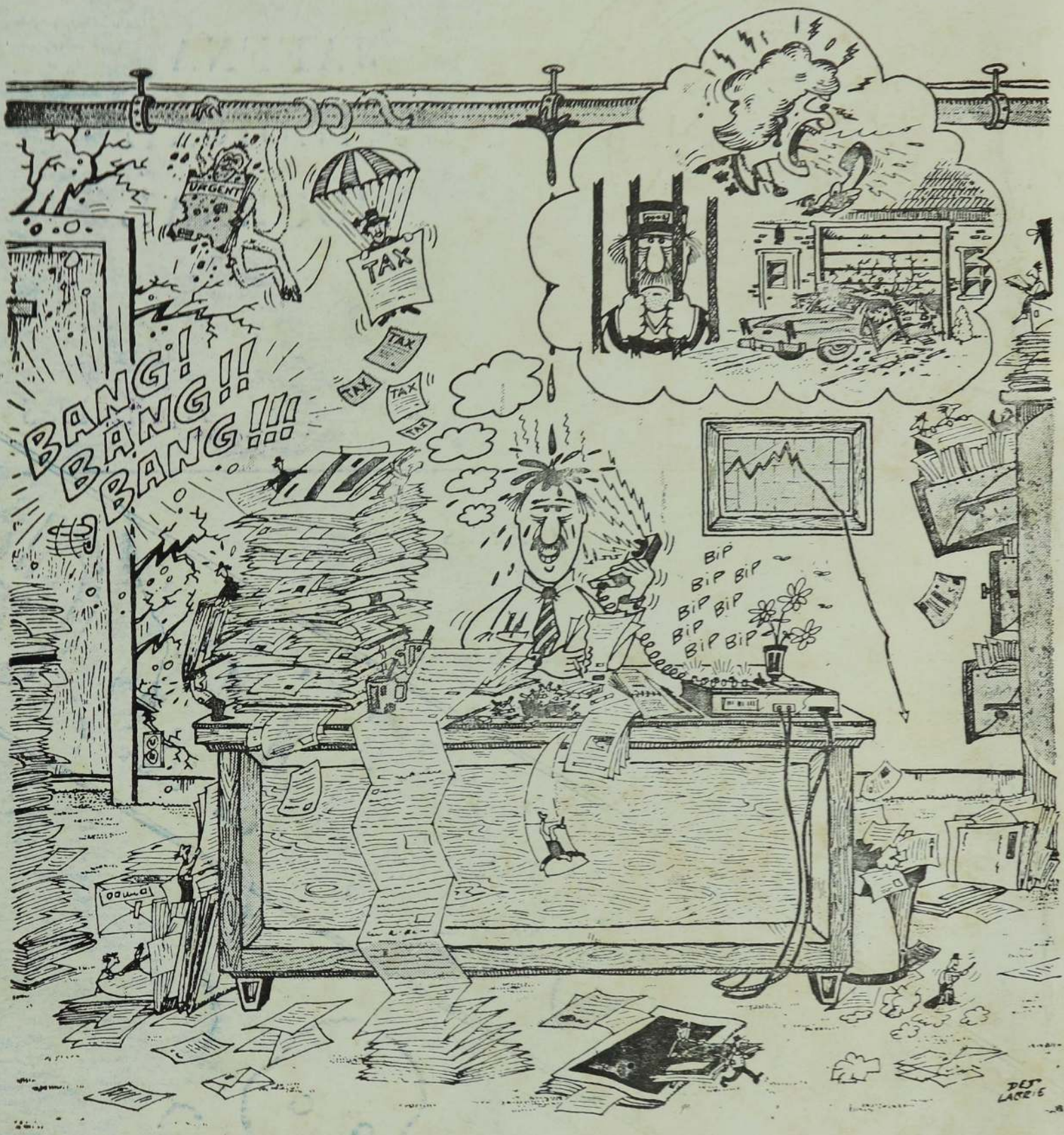
ISSN 0301—788

JAFFNA MEDICAL JOURNAL

Volume XX No. 1 April 1985

Contents

When stress leads to gut reaction...



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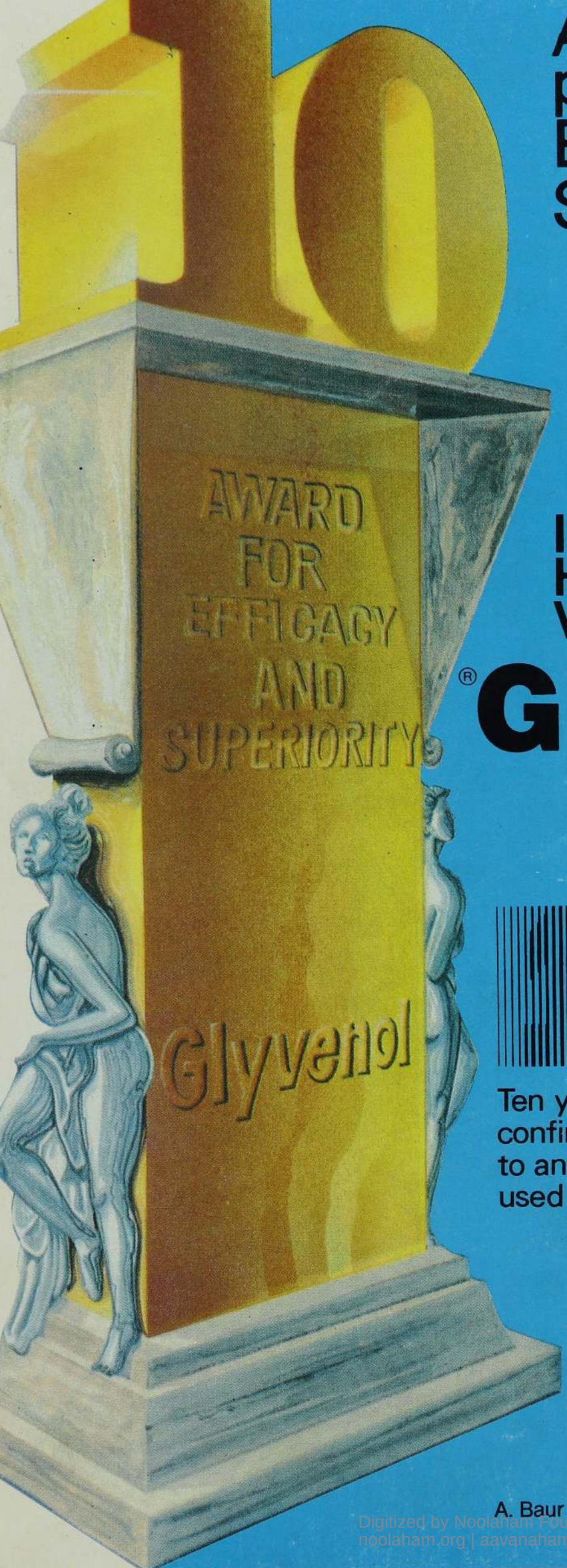

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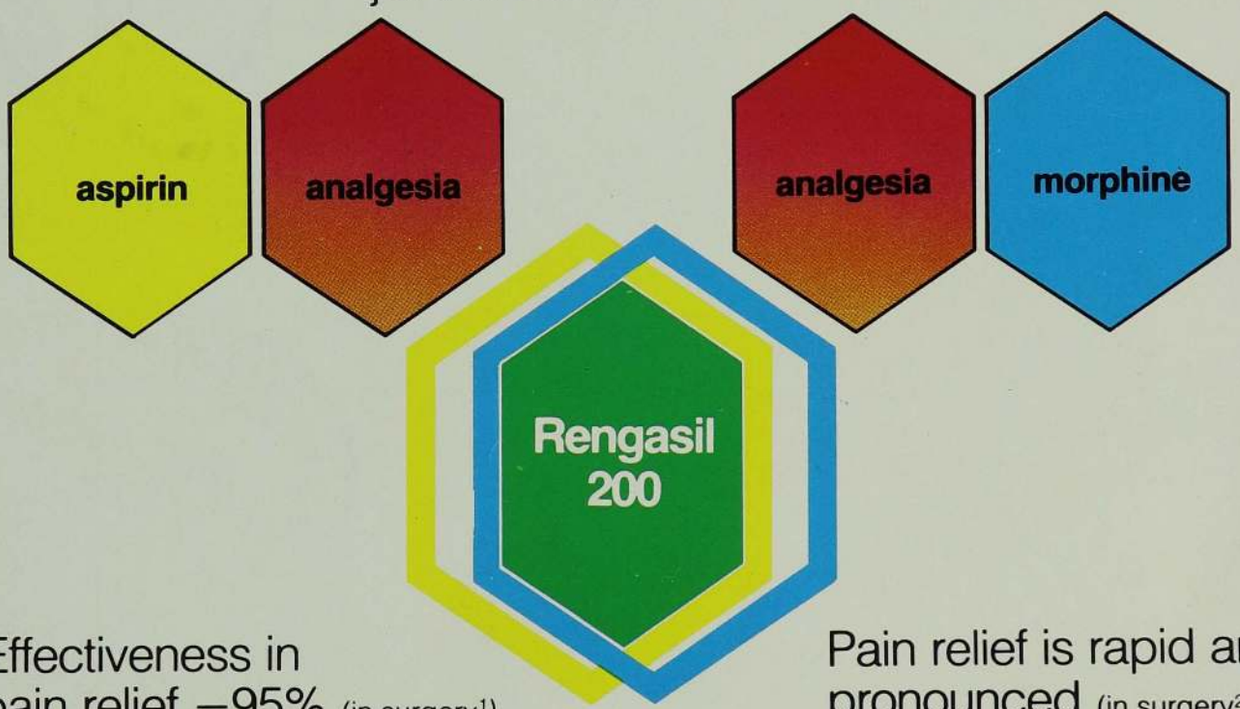
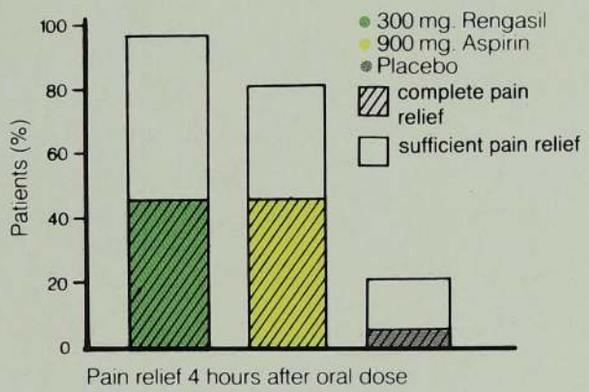
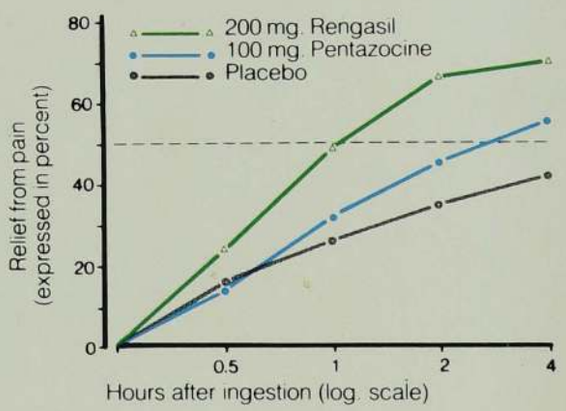


Effectiveness in pain relief –95% (in surgery[1])



Pain relief is rapid and pronounced (in surgery[2])



A sensible balance between aspirin and the narcotics

1. M. Hultin; K-J Olander: Comparison of pirprofen, acetylsalicylic acid, and placebo in post-operative pain after oral surgery

2. Sperr W.; In van der Korst, J.K. (Editor) A new antirheumatic-analgesic agent: pirprofen. Int. Symp., IXth Europ. Congr. Rheumatol; Wiesbaden 1979

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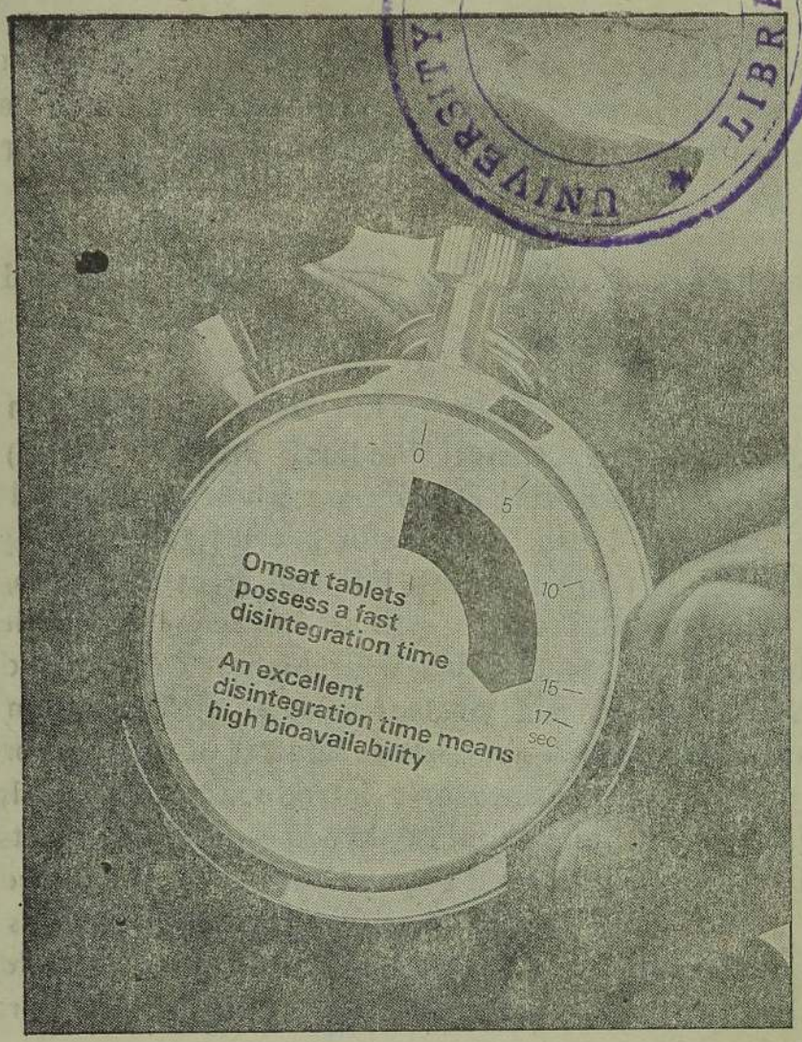

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- of the respiratory tract
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Communications regarding business matters and advertising should also be addressed to the Editor.

Manuscripts The Jaffna Medical Journal will subscribe to the policy of uniform requirements for manuscripts described in the British Medical Journal (1979) 1: 532-535 and the Lancet (1979) 1; 428-431. Intending authors are advised to consult these instructions. Two copies of manuscripts, typed on one side only of good quality white paper, with double spacing and 3 cms margins at both left and right should be submitted. Each manuscript should have the following sections in sequence:- title page (on a separate page) with authors namee and listing their highest degrees and diplomas, their positions at the time of the study, and present post if different from the above, the institution where the work was carried out and the address of the Author who will deal with correspondence and reprints; summary; introduction; materials and methods; results; discussion; references. Tables should be typed on separate sheets of paper and numbered in sequence with Roman numerals. Figures should be numbered with Arabic numerals. Both tables and figures should have accompanying legends. Photographs should be, good quality, unmounted glossy prints. All illustrations should have a label pasted on the back indicating the name of the author and the figure number.

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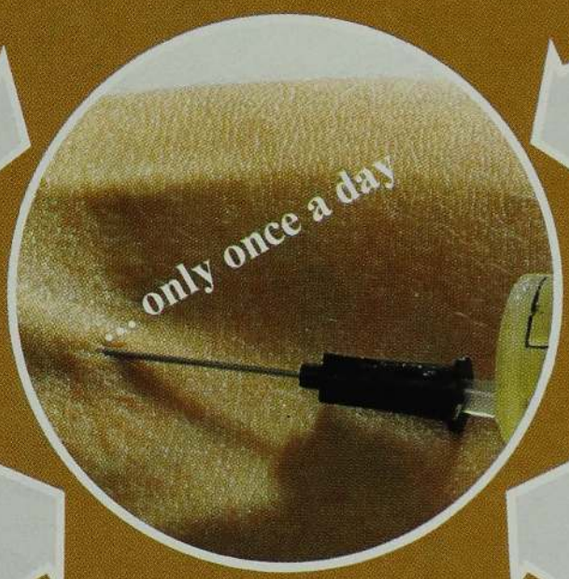

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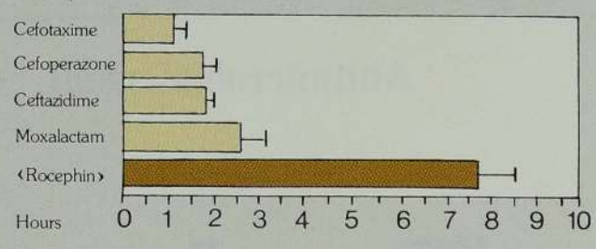

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Reference: 1. Archimbaud, J.P., and Ejazi, M.: *Lyon Méd.* 236:285-287, 1976.

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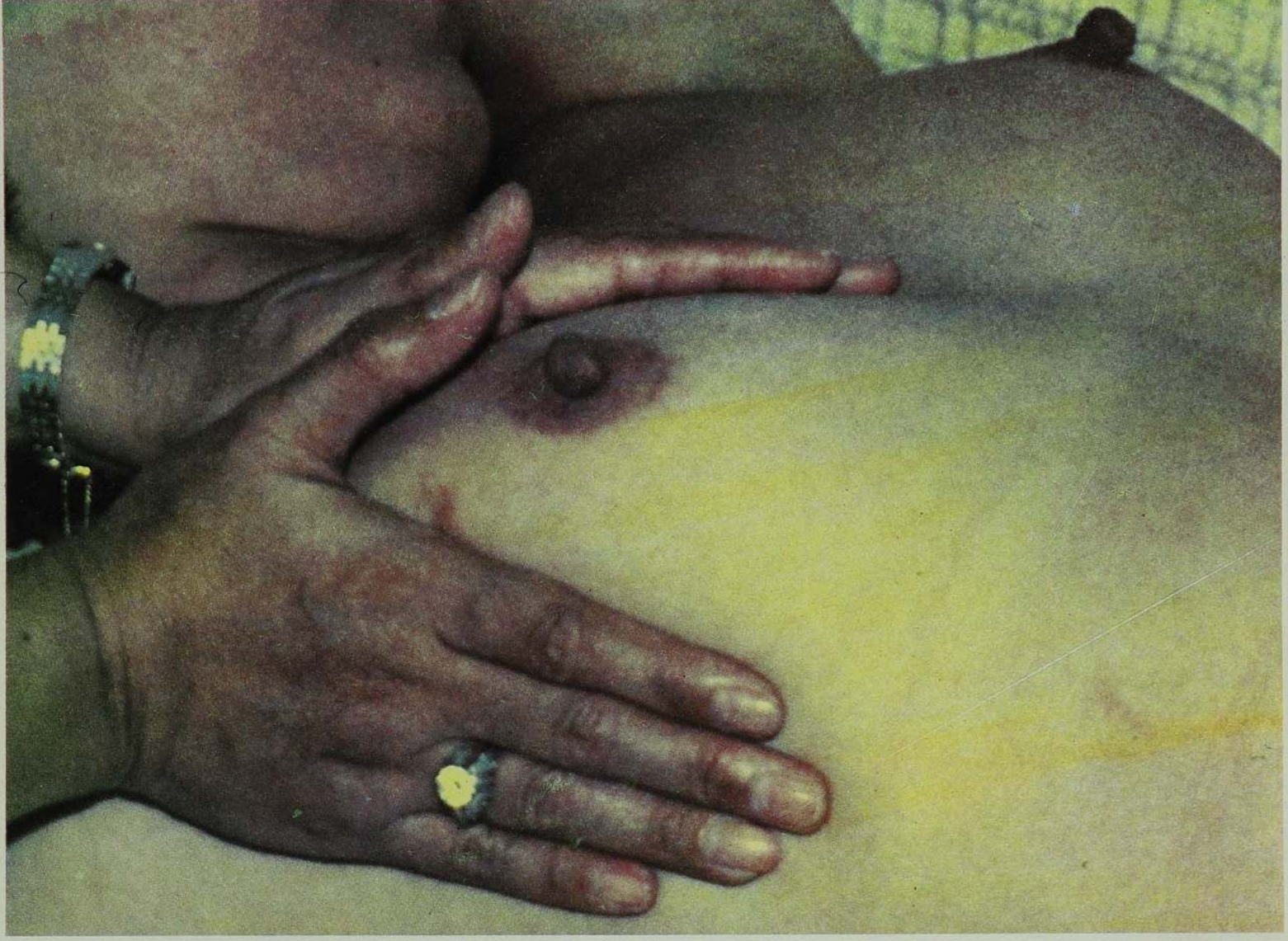

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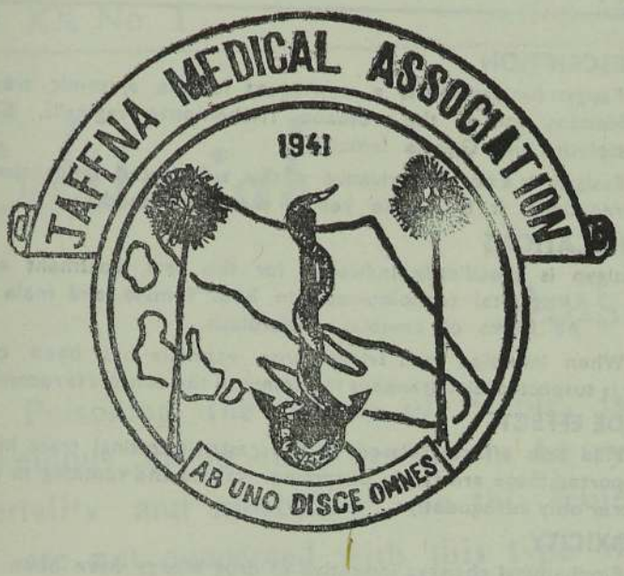

JAFFNA MEDICAL JOURNAL

Volume XX No. 1 April 1985

Contents

IZER INTRODUCES

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Simple three day dosage regime for Amebiasis, both intestinal and extraintestinal.

Single dose oral treatment for Trichomoniasis.

Single dose treatment for Giardiasis.

Excellent tissue penetration.

Marked amebicidal, trichomonicidal and giardicidal activity.

Rapid relief of symptoms.

Extremely well tolerated.

Cure rates of over 95% in Amebiasis, Trichomoniasis and Giardiasis.

No need for drug combinations or multiple therapy.

PRODUCT INFORMATION

DESCRIPTION

Fasigyn (tinidazole) is a new agent for the systemic treatment of infections due to the protozoa Trichomonas vaginalis, Entamoeba histolytica and Giardia lamblia.

Fasigyn is a novel derivative of the substituted imidazole group of compounds. It is a pale yellow crystalline solid.

INDICATIONS

Fasigyn is specifically indicated for the oral treatment of
Urogenital trichomoniasis in both female and male patients.
All forms of amebiasis. Giardiasis.

When infection with Trichomonas vaginalis has been confirmed or is suspected, simultaneous treatment of the consort is recommended.

SIDE EFFECTS

Mild side effects related to the gastro-intestinal tract have been reported; these are rarely disturbing. Nausea and vomiting in particular occur only infrequently.

TOXICITY

Biochemical changes indicative of drug allergy have been reported in one patient.

As with metronidazole and other related compounds Fasigyn may produce transient leucopenia.

CONTRA-INDICATIONS

As with drugs of similar chemical structure, Fasigyn is contra indicated in patients with a history of blood dyscrasia and should be avoided in patients with organic neurological disorders. Fasigyn passes the placental barrier and is present in the breast milk when administered to nursing mothers. Thus as it is not known whether compounds of this class are harmful to the foetus or newborn, Fasigyn is contra indicated in nursing mothers and during the first trimester of pregnancy. While there is no evidence that Fasigyn is harmful during the latter stages of pregnancy, it should be used, like similar compounds, with caution and discretion.

PRECAUTIONS

As with closely related drugs, Fasigyn may produce transient leucopenia and neutropenia. Alcoholic beverages should be restricted during therapy with Fasigyn since abdominal cramps, vomiting and flushing have occurred when related compounds were taken together with alcoholic beverages. Drugs of similar chemical structure have also produced various neurological disturbances such as dizziness, vertigo, incoordination and ataxia. If during therapy with Fasigyn, abnormal neurological signs develop, therapy should be promptly discontinued.

SUPPLIES

Film coated oral tablets of 500 mg. tinidazole base.

RECOMMENDED DOSAGE FOR ADULTS

DISEASE	TABLET STRENGTH	FREQUENCY OF DOSAGE	DURATION (DAYS)
nebiasis) Amebic dysentery & all forms of Amebiasis.	500 mg.	Single daily dose of 4 tablets.	3
epatic Amebiasis) Amebic Liver abscess and other forms of Extra-intestinal amebiasis	500 mg.	Single daily dose of 4 tablets	3
ogenital chomoniasis	500 mg.	Single dose of 4 tablets.	1
ardiasis	500 mg.	Single dose of 4 tablets.	1

RECOMMENDED DOSAGE FOR CHILDREN

DISEASE	FREQUENCY OF DOSAGE	DURATION (DAYS)
Amebiasis (a) Amebic dysentery and all forms of Amebiasis.	30-60 mg/kg bodyweight once daily.	3
Hepatic Amebiasis (b) Amebic Liver abscess and other forms of extra-intestinal amebiasis.	50-60 mg/kg bodyweight once daily.	5
Urogenital Trichomoniasis	50-75 mg/kg bodyweight as a single dose.	1
Giardiasis	50-75 mg/kg bodyweight as a single dose.	1

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Editorial

Poisoning

Poisoning the minds of people, is a heinous crime that results in heavy mortality and morbidity. In this issue we are not concerned with this type of poisoning. Poisoning as discussed in this issue refers to the deleterious effects caused by the entry of noxious substances into the body. Scientific advancement has unfortunately brought in its wake destructive elements. Pesticides for greater agricultural yields and drugs for healing, have had their toll. Regretfully, it is the improper or injudicious use of scientific knowledge and tools by man himself, that has resulted in destruction (even of man by man). We propose, in this issue, to look at our experience with poisoning in our area and suggest ways and means of overcoming or minimising needless destruction.

In Jaffna, Organophospates have been responsible for most cases of poisoning, followed by drugs, plant products, petroleum produets and rodenticides in that order. (see Ganeshamoorthy. R.—first article) It is also apparent from this article, that about 2/3 the cases were of self-poisoning while 1/3 were accidental. The young were affected mostly and the mortality was about 7%. Very many of them are innocent and their being afflicted, in this so called civilised world is a serious blotch on the leaders—of the medical profession. It is time that a detailed study is made and measures to prevent poisoning instituted.

Education of the public is very important and the cooperation of many is imperative, eg, traders, pharmacists, law enforcers. Identifying the potential suicide may be a formidable task. Ganeswaran, et al (in this issue), have discussed some factors but pointed out the difficulty of reliable tests. However the parents, relatives and friends, who are known traditionally to have a knack of detecting the suicide prone should take such cases early to the psychiatrist. Extra surveillance is necessary during times of crisis but to wait for such occasions may be too late. To see that noxious snbstances are not within the reach of the potential suicide is another aspect. The avoidance of starvation of (innocent) refugees, seems to have gained importance, during recent times!

Many, if not all of the accidental poisoning cases eould be prevented. Safe storage, especially with regard to reach of children, proper handling and attention to precautionary measures while preparing (masks, gloves etc), technique of spraying (in direction of wind), attention to

presence of others in viscinity, washing of hands, clothes and having a bath need to be disseminated and perhaps supervised for some time at least. The public should also be educated on the early symptoms and the emergency measures (first aid) to be adopted. Doctors should not prescribe large amounts of drugs at the same time and symptoms of overdose must be explained. The Dangerous Drugs Act should be enforced strictly. That the public should be able to identify poisonous plants, is a proposition that is open to debate, for the advantage of avoiding accidental poisoning by proper identification may be offset by the easy recourse (identification) provided to the potential suicide. Perhaps the solution is in the Local Authorities taking steps to destory the poisonous plants, save those of medicinal value. Industrialists should observe safety precautions with regard to stages in manufacture and also disposal of waste.

A Poisons Committee consisting of members of the Health Department, Medical Associations, Local Bodies, Labour Dept, Voluntary Organisations, should start work immediately.

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HEW NEWS

U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

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(HEW NEWS-Bulletin issued by the U.S. Department of Health, Education and Welfare (Food and Drug Administration)— Ref. P 79-6 dated March 30, 1979)

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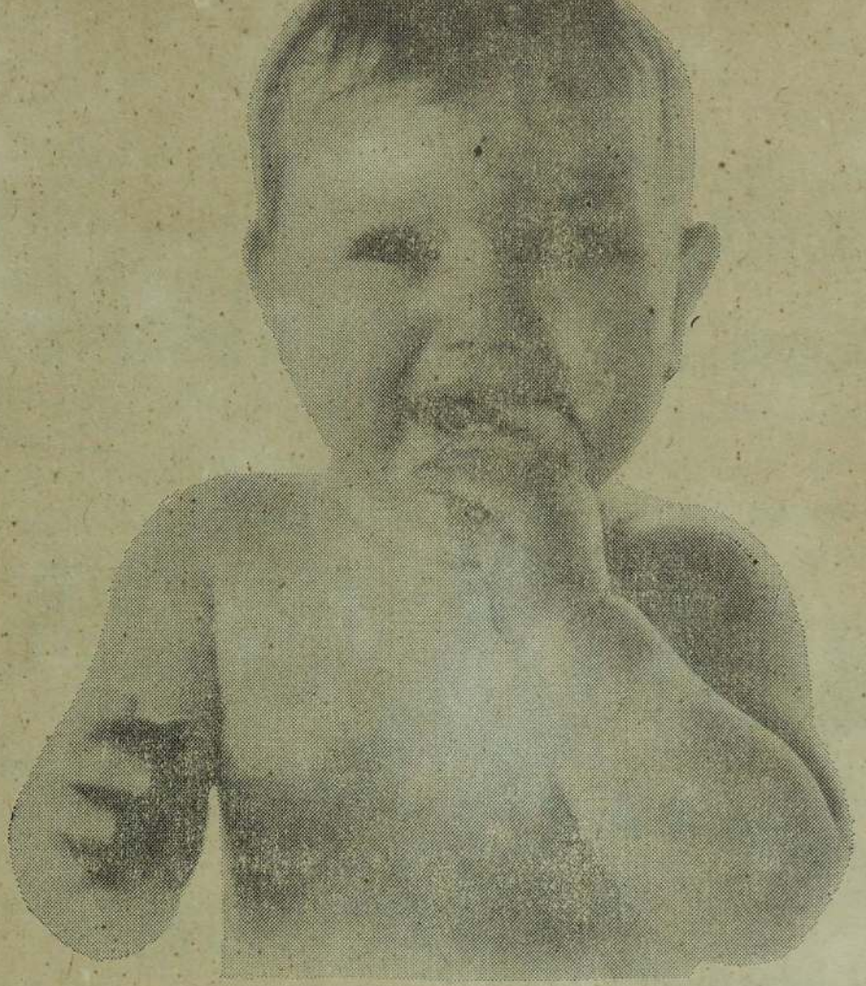

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You can confidently recommend bonjela because

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Mother simply puts the pleasant tasting gel on her fingertip and massages it into the baby's inflamed gums, not more often than every three hours.

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In a study[2] involving 48 teething children (aged 1-3 years) fast relief was shown with 83% of the children obtaining relief in 2 minutes or less and 43% showed immediate relief.

Safety	No side effects have been reported in any of the clinical trials conducted with Bonjela. It produces relief from pain, but avoids the danger of an anaesthetised mouth.
Indication	Infant teething symptoms
Packaging	Bonjela is available in 10 g tubes.
Active Constituents	Choline Salicylate 8.7% A highly soluble analgesic anti-inflammatory agent Cetalkonium chloride 0.01% A non-irritant antiseptic agent effective against gram-positive and gram-negative organisms.
Contra indications	History of salicylate sensitivity

References: – 1 Warmington, W. J. Northw. Med (Seattle), 1962, **61**, 930-932
2 Dawes, R. M., J.La. med. Soc., 1962, **114**, 85-87
3 Palmer, L. E. Ohio St. med. J., 1962, **58**, 434-435.

Further Information From :
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Spectrum of Acute Poisoning in Jaffna

— A ONE YEAR SURVEY —

R. Ganeshamoorthy, FFARCS (Eng)*

Summary :

446 patients, admitted with acute poisoning, were analysed. Male to female ratio was 2·1 : 1. Self poisoning was the cause. in 68% and accidental poisoning in 32% 73·8% of the patients were in their second and third decades of lives Agents af poisoning were pesticides in 62·3%, drugs in 16.6%, plant seeds in 8·5% and other chemicals in 6.7%. Overall mortality was 7.0%. Clinical manifestations and poor risk factors are discussed.

Jaffna, which is the provincial town of the Northern Province of Sri-Lanka, has been recently experiencing an increase in number of people resorting to self poisoning. A recent publication[1], revealed a suicide rate of 53.5 per 100,000 for Jaffna, which has a total population of 831,112. This rate of suicide, which is the highest in the world, has prompted the survey of all cases of acute poisoning admitted to General Hospital (Teaching), Jaffna, (in the year 1983). The adoption of the policy of admitting all cases of acute poisoning to the Emergency Unit, which was established in 1982, has made it easy to retrieve the records of these patients, admitted during 1983.

Patients and Method :

Records of 446 patients, who were admitted during the year 1983, with acute poisoning, were examined. Information about the sex, type of poison, mode of poisoning, time lapse before admission to hospital, clinical manifestation and outcome was analysed. Cases of envenomisation or reaction to drugs, were not included.

Results :

There were, 50,435 patients admitted to this hospital, in the year 1983 and 2131 of them were admitted first to the Emergency Unit. The number of patients admitted during the period, with acute poisoning was 446; ie 0.88% of total admissions and 20·9% of admissions to the Emergency Unit.

Age and Sex Distribution :

Age and sex distribution of these patients is shown in Table I. Male to female ratio for the series is 2.1 : 1. Patients in their second and third decades of lives, constituted 73·8%. These results are in agreement with other reports. [1], [2], [3].

Seasonal Variation :

Table II shows, that admissions were high during the first half of the year, with a peak in the month of Februry and a lull in the month of August. It also shows, that this variation is largely due to accidental poisoning with agrochemicals.

Causes of Poisoning :

Table III shows, that self poisoning was the cause in 68% and aceidental poisoning the cause in the balance 32%.

* *Consultant Anasethetist, General Hospital (Teaching), Jaffna.*

Table I

Age and sex distribution of patients

Age (years)	number of patients		
	Male	Female	Total
0 — 10	1	1	2
11 — 20	92	66	158
21 — 30	125	46	171
31 — 40	47	19	66
41 — 50	24	2	26
51 — 60	9	6	15
over 61	6	2	6
	304	142	446

Table II

Number of admissions monthly

Month	Poisoning via skin	Poisoning by ingestion	Total
January	7	27	34
February	40	32	72
March	24	38	62
April	15	35	50
May	11	35	46
June	7	32	39
July	0	21	21
August	2	13	15
September	1	24	25
October	5	26	31
November	1	18	19
December	5	27	32
	118	328	446

No case of homicidal poisoning was recorded in this study, though it had been reported in this country[4]. When agrochemicals are sprayed in the fields, these enter the body through the skin as well as through the lungs. This type of poisoning is referred to as, spray poisoning, in this locality. Out of the 143 patients with accidental poisoning, 118 were due to spray poisoning and of the twenty-five patients, who gave a history of accidental ingestion of the poison, corroborative evidence was available, in only eleven of them.

Table III

Causes of poisoning

Cause	Number of patients	
A. Accidental	143	(32 %)
while spraying 118		
by ingestion 25		
B. Self poisoning	303	(68 %)
C. Homicidal	nil	

Type of Poison:

A history of poisoning was obtained in all these prtients, either from the patient himself or from his kith or kin. The exact nature of the poison was not known in twenty-four patients; ten of them were said to have ingested some liquid and the other fourteen were said to have swallowed some tablets.

Table IV shows the type of poison and whether, it was taken accidentally or deliberately. It shows, that agrochemicals were the agents of poisoning in two-thirds of these patients. Drugs as the agent of poisoning came second and plant seeds third, just ahead of chemical agents. A complete list of names of the substances encountered is given below; the figure within brackets denoting the number of patients :

Table IV

Type of poison and its mode of exposure

Type of poison	Mode of exposure (no of patients)		
	Accidental		Self poisoning
	skin	oral	
A. Pesticides (62.3 %)			
organophosphates	117	7	135
organochlorines	—	1	5
carbamates	1	—	5
miscellaneous	—	—	7
B. Rodenticides (2.9 %)			
Run-Rat	—	—	13
C. Herbicides (0.7 %)	—	2	1
D. Unknown liquid (2.2 %)	—	—	10
E. Drugs (16.6 %)			
identified	—	7	53
unidentified	—	1	13
F. Chemical agents (6.7 %)			
alcohols	—	1	1
acids	—	—	5
petroleum distillates	—	3	11
others	—	1	8
G. Plant seeds (8.5 %)			
yellow oleander	—	—	36
castor	—	2	—
Total	118	25	303

Organophosphates : Tamarone (77), Folidol (42), Parathion (23) Runbug (17), Ekatox (13), Malathion (12), Baytex (3), Azodrin (3), Asuntol (2), Dicial (2), Sumithion (2), Chlordane (1), Mercaptophos (1), Monitor (1), Fenthion (1), and Endrex (1), Unidentified (58)

Organochlorines : DDT (5) and Gammexane (1).

Carbamates : Baygone (4) and Lannate (2)

Miscellaneous Pesticides : Thiodan (2) and Ambush (5).

Herbicides : MCPA (1), Paraquat (1) and Harcross (1).

Rodenticides : Run-Rat (13).

Drugs : Phenobarbitone (18), Phenobarbitone and another drug (4), Diazepam (11), Chlorpromazine (10), Tofranil alone or with another drug (5), Chlordiazepoxide (3), Artane (2), Aspirin (2), Franol (2), Stelazine (1), Promethazine (1) and Digoxin (1), and Unidentified (14)

Alcohols : Ethyl (1) and Methyl (1).

Acids : Sulphuric Acid (1), Harpic (11), Lysol (1), Pynol (1) and Gold Solvent (1).

Petroleum Distillates : Kerosene-Oil (1), Petrol (1), Paint thinner (1) and Turpentine (1).

Other Chemicals : Copper sulphate (3), Corrosive nitrate (1), Lithium+Chloroform+Alcohol (1), Dettol (2), Camphor+Alcohol (1) and Blue Powder (1)

Thirty-six patients had consumed alcohol along with the poisoning agent.

Clinical Features :

Ninety-eight patients (22.0%) did not show any clinical manifestation of poisoning, either on or after admission to the hospital. These patients were, however, observed for a day or two, before they were discharged.

Common manifestations in patients with pesticide poisoning were, miosis (60%), giddiness (35%), muscular twitching (31%), drowsiness (15%) and respiratory distress (14%). Dyspnoea, frothing at mouth and pulmonary oedema were considered as manifestations of respiratory distress in this analysis. Twenty-two patients (1.0%), were admitted in a state of unconsciousness and fourteen of them succumbed to the poison. Bradycardia, was seen in only two patients.

Common manifestations in patients with drug poisoning were, drowsiness (50%), unconsciousness (22.4%) and hypotension (3.4%). Sixteen patients in this group (21 6%) did not show any sign of poisoning.

Thirty-six patients were admitted after ingestion of seeds from the plant Yellow Oleander (Thevitia peruviana) known as Alari locally. This is a common dry zone plant, which is found in plenty in Jaffna. Seventeen patients (47%), remained without any manifestation and the clinical details of four patients were not available. Commonest clinical manifestations in the other fifteen patients were, vomiting (53%), varying degrees of heart block (53%), drowsiness (33%), giddiness (26.7%), unconsciousness (7%) and hypotension (7%). Two of these patients died. Number of seeds ingested varied from one to six, with a mean of three. There were two school girls in this group of plant poisoning, who ingested out of curiosity, four seeds of castor-oil plant and the only manifestation in them was mild vomiting.

Eleven patients, including two childern, were, admitted after swallowing kerosene-oil and only four of them developed lung signs. None of these patients died.

Management :

Management consisted in giving all patients, other than those who ingested petroleum distillates and acids, a stomach wash, basic life supporting measures and antidotes whenever appropriate. In cases of poisoning with pesticides, atropine was the main drug of choice. In a large majority of these patients, total dose of atropine used was five to fifty milligrams, but in five patients doses of atropine over 180 mgm were given during a period of fortyeight hours. One patient died

inspite of receiving 616 mgms over a period of forty eight hours. Pralidoxime (PAM), two to five grams was given to some. Atropine, was also given for Yellow oleander seed poisoning.

Time Lapse :

Table V. shows the time lapse between poisoning and hospital admission. It shows, that patients with spray poisoning sought admission after few hours, because they became aware of poisoning, only when some symptoms manifested. This is in contrast to those with self poisoning, where 25% of them sought admission within half an hour, 66% within one hour and 90% within six hours.

Table V

Time lapse between poisoning and hospital admission

Time lapse (hours)	Mode of exposure skin	ingestion	Total
0 — 1/2	1	74	75 (19 %)
1/2 — 1	4	63	67 (17 %)
1 — 2	15	68	83 (21 %)
2 — 3	19	21	40 (10 %)
3 — 4	22	13	35 (9 %)
4 — 5	16	9	25 (6 %)
5 — 6	7	7	14 (4 %)
6 — 10	19	11	30 (7,6%)
0 — 15	1	16	17 (4.3%)
5 — 20	0	5	5 (1.3%)
over 20	4	2	6 (1.5%)
unknown	8	41	49

Table VI a

Mortality

Poison	number of patients	number died	Mortality
organophosphates	259	25	9.5
yellow oleander seed	36	2	5.6 %
drugs	74	1	1.4 %
unknown	10	3	30.0 %

Table VI b

Poor risk factors in organophosphate poisoning

Factors	number of patients	number died	Mortality
drowsiness	41	7	17 %
unconsciousness	21	14	67 %
respiratory failure	21	16	76 %
repoisoning	8	5	63 %

Mortality :

Table VIa shows, that there were thirty-one deaths in this study, giving an overall mortality of 7·0%. All these deaths were due to self-poisoning. Twenty-five deaths due to pesticides included six from Tamarone, three from Folidol, two from Runbug, two from Parathion, one from Baytex. one from Endrex and eight from unidentified organophosphate. In the one death due to drug poisoning, the nature of drug was not known. Nature of the poison was not known in three other deaths,

because these patients were admitted in coma and cardio-vascular collapse, and all of them died within one hour of admission.

Table VIb shows, that in patients with organophophate poisoning, the presence of drowsiness or coma or respiratory failure on admission, or the occurrence of repoisoning carried a higher mortality.

Respiratory failure, is very often the cause of death in acute poisoning. In this study, it occurred in thirty-five patients and in twenty.nine of them, the outcome was fatal Nine patients were ventilated with an Ambu bag another ten patients werc connected to a ventilator. Adequate ventilation was not provided in sixteen patients, either due to the non availability of an Ambu bag. or due to the failure to intubate the trachea.

Concurrent medical illnesses encountered were, psychiatric disorders in twenty-one, epilepsy in eight, rheumatic heart disease in two and bronchial asthma in another two. None of these patients died.

Two patients had attempted suicide earlier. One patient is a schizophrenic, who ingested chlorpromazine and the other patient, who had ingested diazepam enrlier, had resorted to Yellow cleander seed this time. These two patients survived once again.

Discussion:

Comprehensive studies in this country, on the prevalence of acute poisoning are few. One report[3] stated, that in 1976, there were one hundred and thirty cases admitted to General Hospital, Colombo, during the month of July and August. Other reports [4,5,9], which were published in this country have dealt with only insecticide poisoning. Aaother report[1] published in 1984, dealt with suicides only. A report[2] from Nehru Hospital in India states, that three hundred and twelve patients were admitted over a period of ten years from 1970. This small number of patients was attributed to the fact, that Nehru Hospital was receiving only patients referred from other hospitals.

The admission of 446 patients to General Hospital (Teaching), Jaffna, and a high suicide rate for Jaffna, show, that the incidence of acute poisoning is high in this district. The rise in incidence of acute poisoning is a global phenomenon, and Jaffna seems to be in the lead Reports published in this country and elsewhere [1,2,3] show, that the most vulnerable are those in their second and third decades of life. This is the trend in this study too.

The cause for the high incidence of acute poisoning, has to be found among the youth and also from the conditions prevailing in Jaffna. Jaffna has been going through a period of political instability, economic depression and violence in the recent past. The insecure and uncertain state of mind, especially among the youth is a very likely potent cause for self poisoning. Another factor is the easy availability of agrochemicals in this area.

Spraying of agroehemieals in Jaffna is solely undertaken by the males and hence spray poisoning is confined to males. This has contributed to the higher ratio of males in this series. When the number of patients with spray poisoning is deducted from the total, males are still

found to predominate by 1·4 : 1. This is probably due to the fact, that men have to bear the brunt of all difficulties, in this male dominated society.

Admission of just over two-thirds of patients in the first half of the year, is largely due to the cultivation of cash crops and to the extensive use of agro-chemicals, during this period. When patients with spray poisoning are excluded, the variation in the monthly incidence of self poisoning narrows down, except in August and November. The low incidence during these months may be due to the fact, that youngsters were busy preparing for public examinations, which are held in August and December every year. Slightly higher incidence in March, is probably, due to the release of results of these public examinations.

The law of this country, requires a police investigation into all cases of self poisoning. This, probably, accounts for the absence of corroborative evidence of accidental poisoning by ingestion, in more than half these patients.

Pesticides had been the commonest poisoning agent in this study (62·3%) and out of these, organophosphates accounted for over 90%. Dicophane (DDT), was the first insecticide introduced into this country and a case of poisoning was first reported in 1954[4]. This was then then followed by an organophosphate, Folidol[5] and now Tamarone is in the lead. The absence of laws regarding sale, purchase, storage and use of these chemicals, is a leading cause for the high prevalence of self poisoning with these chemicals, which are available even in the remotest village of this district.

Poisoning with drugs comes seeond (16·6) in this study; and this was so in the Colombo study as well[3]. This is in contrast to the pattern in western countries, where drugs are the commonest agents of poisoning. In this series psychotropic and tranquilizer drugs were encountered more frequently than barbiturates. One reason for drug poisoning in this country is, that psychotropics, tranquilizers and barbiturates are generally issued in bulk to patients attending the psychiatry and the epilepsy clinics. Another reason is that these drugs can be purchsed from chemists without a prescription.

Poisoning with plant seeds, was the third commonest in this study (8·5%). There were no reports of poisoning with seeds of Yellow oleander, before 1982. The sudden surge in this method of self poisoning is attributed to the publicity given to the death of two school girls, in a local news paper[6]. The poisonous substance, in this seed, is an alkaloid similar to cardiac glycoside. Its action on the myocardium and its conducting system is responsible for the deleterious effects.

Commonest petroleum distillate ingested was kerosene oil. This is readily available, because it is used in many homes for lighting and to operate water pumps in the fields.

Diagnosis of poisoning was not a problem in this study, which is not so in other countries. In our country, there was always someone to give the history of

poisoning, whenever, the patient was unwilling or was not in a state to converse. One reason for these is that the system of extended family still prevails in this country.

The fact that 75% of patients had either minor or no manifestations of poisoning, and a large number of these patients were those of self poisoning, points to the absence of a real intention to commit suicide, but a means to escape a crisis situation, or to attract attention. This is why the term, attempted suicide or failed suicide was replaced by the term self poisoning.

Cardiac arrest occurred in thirty-one patients and in all it was fatal. Cardiac arrest may be due to either the toxic effect of the poison on the heart or due to hypoxia, secondary to respiratory failure. One has to be wary of dilated pupil in this situation because it may be due to the effect of the poison and not due to brain death[7].

Atropine and pralidoxime are still the main drugs used in the treatment of pesticide poisoning. The dose of atropine given at times, exceeded 150mg and in one patient, the dose given was 616mg. Higher doses were given in the form of an infusion. In an earlier study[8], a dose of 1182mg was given to a patient; and in this report, a plea for the manufacture of multidose or higher dose atropine ampoules was made. Atropine is still available as 0 5 to 1 0mg ampoules and breaking a large number of these in an emergency is a formidable task.

The action of pralidoxime (PAM) is to some extent, the release of enzyme cholinesterase (ChE) inhibited by organophosphates and carbamates. It is reported, that different brands of these agrochemicals inhibit ChE in different degrees and for varying periods of time[9]. It has also been observed, that PAM should be given by injection within 24 to 48 hours ; as after this period, it will not reactivate ChE.

The reappearance of symptoms and signs of organophosphate poisoning, after apparent recovery, occurred in eight patients with a fatal end in five. This phenomenon of repoisoning had been reported earlier[8]. One reason is that the action of insecticide lasts much longer than the actions of atropine and PAM. Another reason is the re-entry of poison into the blood stream from storage tissues, such as fat and muscles, and also from delayed absorption from the colon. It is said that the toxicity of the poison must be considered in the light of not only of the dose, but also of the likely time course of its effects[10].

Organophosphates were responsible for ninety percent of deaths in this study. One contributory factor may be the sale of these in a highly concentrated liquid form. The bad prognostic features of organophosphate poisoning in this series were central nervous system manifestation like drowsiness or unconsciousness and the occurrence of respiratory failure. Another study in this hospital[11] reported. that the estimation of serum pseudocholinesterase levels in these patients, may be of a predictive value in determining patients at great risk.

Time lapse between poisoning and hospital admission, or the consumption of alcohol along with the poison, or the

presence of concurrent illness had not contributed to mortality in these patients

Patients with past history of psychiatric disorder were transfered to the psychiatry unit and all the others were advised to attend the psychiatry clinic. The follow up of these patients is not included in this study. The extent of the physical harm cannot be used as a ya dstick to determine the extent of psychological illness or the need for psycho-social help[12].

Prevention of self poisoning, is a near impossible task, whereas, accidental poisoning, can be minimised if effective measures are taken. Stricter control on sale, storage and use of agrochemicals has been already suggested[1]. Safety measures to be adopted while spraying agrochemicals, too had been suggested as for back as 1962[5]. One of the protective measures suggested was wearing of protective clothing. A recent study[13] reports, that though this can minimise the risk, it cannot eliminate the risk of poisoning through the skin. The alarming inerease in Yellow oleander seed poisoning can be eliminated by destroying this plant, which is of no use to the community.

Mortality can be reduced by improving the standard of medical care. It can also be improved by developing new and more effective antidotes. An antidote for benzodiazepines has been already found[14].

This study is an attempt to know the spectrum of and to assess the magnitude of actue poisoning in Jaffna. The real picture will emerge, if further studies of this nature are continued.

" All substance are poisons, there is no such thing as a non poison. It is the amount that distinguishes a poison from remedy " — Paracelsus.

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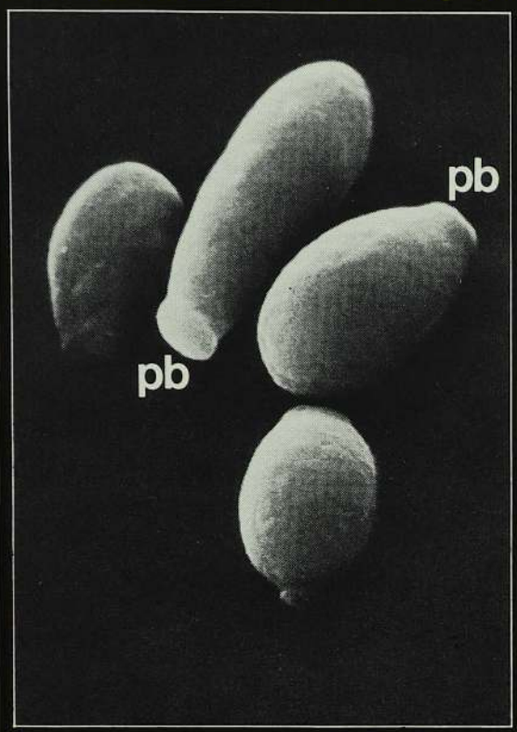
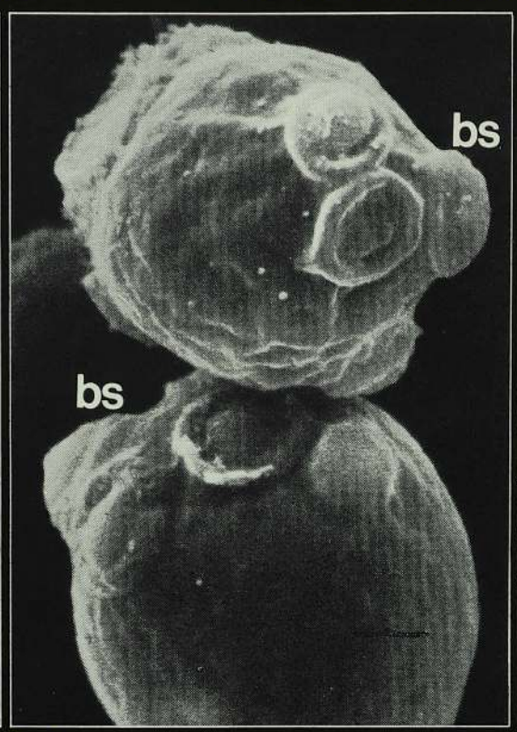
the undoing of the die-hard fungus

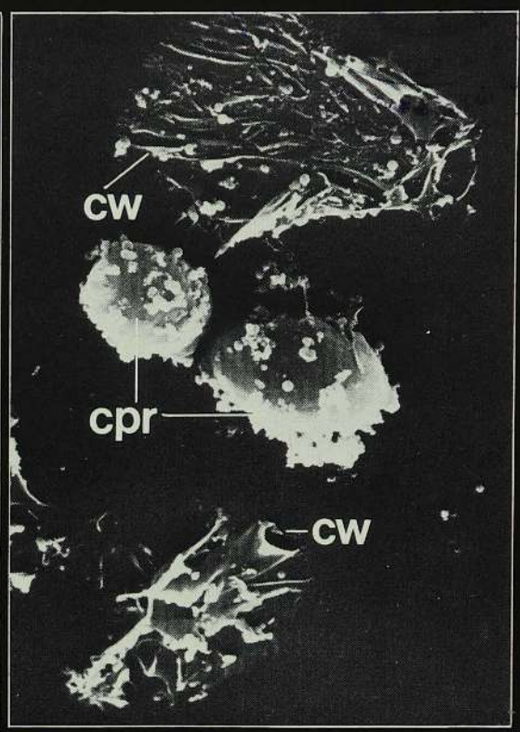
Scanning Electron Microscopic photographs of Candida albicans *before and after treatment with miconazole.*



1. Untreated cells appear as isolated yeast forms in an oval shape and with a smooth surface showing the formation of polar buds (pb).



2. Upon exposure to a fungistatic dose (10^{-7} M) of miconazole the cells tend to cluster. Budding becomes disorganized with visible budding scars (bs) that are randomly distributed over the surface of the cells.



3. Treatment with a fungicidal dose of miconazole causes the cell wall (cw) to burst. Vesicles of cytoplasm disintegrate and adhere to the surface of yet unbroken cells (cpr). (The interior of the cells is completely lytic).

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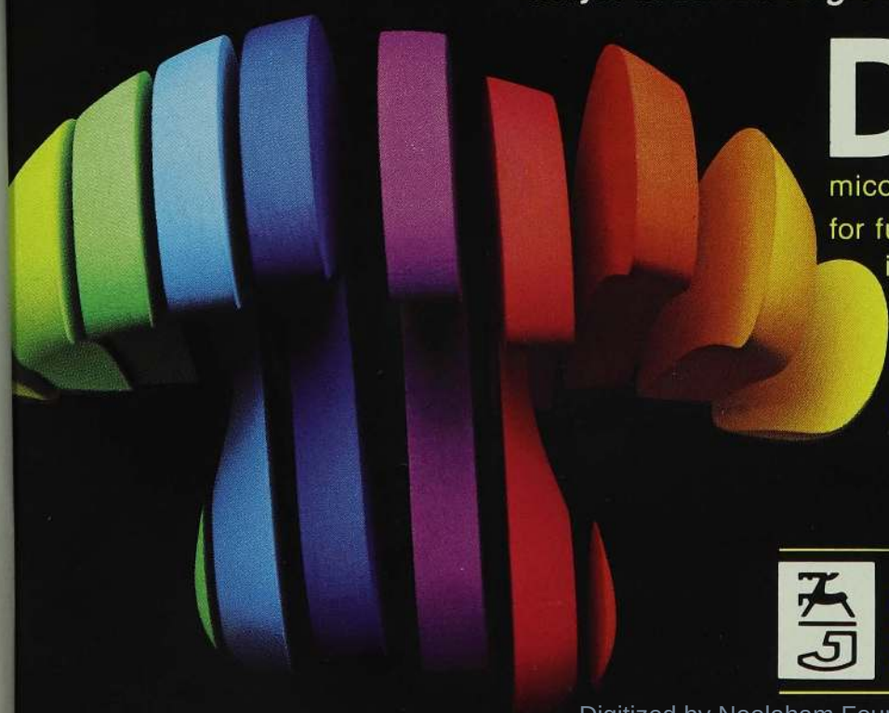

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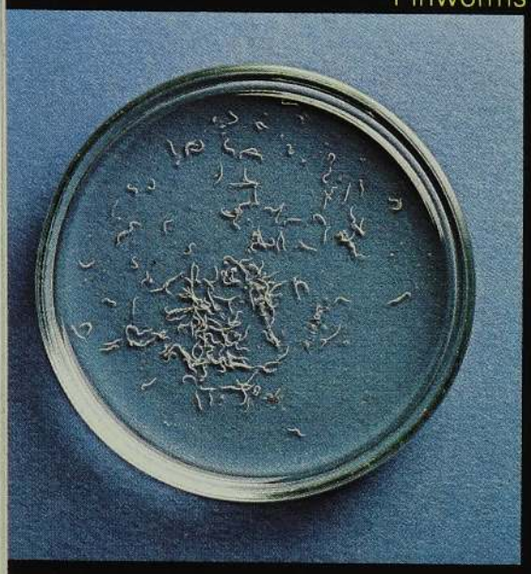
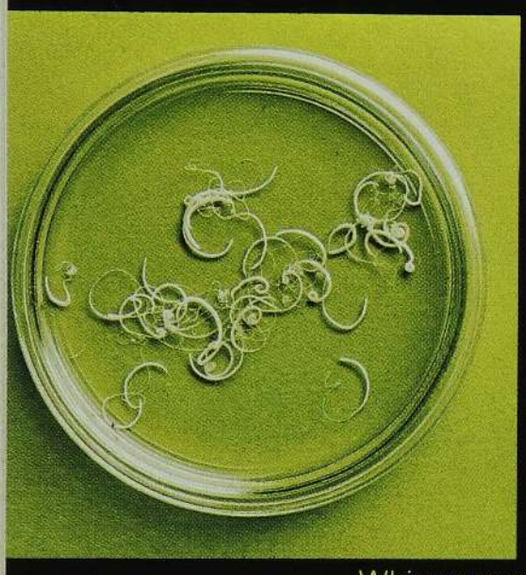


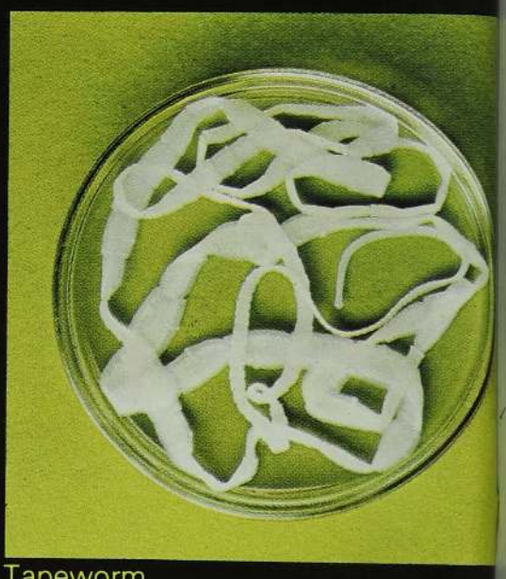
Roundworm

Hookworms



Whipworms

Threadworms



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Insecticide and Herbicide Poisoning

John. R. M. D. (Cey), M. R. C. P. (U. K.)*

The widespraad use of agricultural pesticides in the Jaffna peninsula, had made Insecticide poisoning the commonest cause of poisoning cases admitted to the Jaffna General Teaching hospital.

The Insecticides being marketed fall into two principal chemical groups, namely, (a) Organophosphates (b) Chlorinated hydrocarbons. Paraquat (Gramoxone) is the commonly marketed herbicide. Organophosphates, however account for majority of patients admitted for poisoning–about 60%–Ganeshamoorthy. R. in this issue. There are numerous organophosphates available in Srilanka and comprehensive list should always be available in an Accident and Emergency Unit for easy perusal.

Organophosphates cause poisoning by ingestion, inhalation of the spray, absorption throuh skin or extremely rarely as it occured in one of our cases, following deliberate intramuscular injeetion Absorption through the skin represents the greatest hazard during spraying these chemieals and is the commonest route of Accidental poisoning (about 90%—Ganeshamoorthy. R.). Ingestion of these agents is quite often a suicidal attempt–about 50% of cases having used this agent. However accidental poisoning also occurs, in this way (ingestion) by consumption of food contaminated with these chemicals, by eating without washing the hands after haadling of these chemicals and by children mistaking them for a beverage.

Pharmacology

The fundamental pharmocological action of Organophosphates is that of Anticholinesterases. They inhibit Cholinesterases and thereby ease the accumulation of Acetyl choline. This action is similar to the clinically used anticholinesterases such as Neostigmine. Howevar, while clinically used anticholinesteraes have a seIf–limiting action ("reversible"), organophosphates cause irreversible inhibition of cholinesterases. Thus in organophosphates poisoning, acetyl choline accumulates in excessive amounts at somatic and parasympathetic nerve terminals. Thls accumulation is responsible for all the symtoms and signs observed.

Clinical Features:

The features of poisoning depend entirely on three factors; (a) the dose of the poison (b) route of entry and (c) time lapse in seeking medical attention.

The parasympathetic stimulation results in:

(a) Gastrointestinal features - nausea, increased salivation, vomiting, abdominal colics, diarrhoea, tenesmus, involuntary defaecation and hepatitis.

(b) Respiratory features — rhinitis, lacrimation, frothing, crepitations, rhonchi and respiratory failure.

(c) Central Nervous system features—miosis, blurred vision, muscle twitching, muscular paralysis, depression of the respiratory centre and coma.

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presently, Department of Clinical Sciences, University of Papua, New Guinea.

Respiratory failure, neuromuscular paralysis, increased tracheobronchial secretions and bronchospasm are the dangerous features that cause death.

Certain organophosphates, eg. Tamarone, produee predominantly a motor peripheral neuropathy affecting the hands and feet. These features appear 4 to 6 weeks after initial poisoning. Similarly respiratory arrest could be delayed for up to a week and come on very suddenly after apparent "recovery". This is particularly seen after ingestion of "Baytex". Such patients should be monitored carefully in an Intensive Therapy Unit, where facilities for assisted ventilation is available.

Management :

(a) Contaminated clothing should be removed and areas exposed to the chemicals, thoroughly washed with soap and water.

(b) A patent airway should be maintained. Intubation, tracheostomy or assisted ventilation may be necessary.

(c) Stomach wash is done if patient is conscious and has arrived within 6 hours of ingestion.

(d) Drugs — Atropine antagonises the parasympathetic action as well as the central effects of the poison. 2 mgm is administered intravenously and may be all that is necessary in mild cases. If symptoms are not relieved and if the pupil size (which is a good indication of the degree of poisoning) continues to be small or pin point, further doses of atropine are administered every 5 to 10 minutes. Some clinicians administer even 10 mgms at 5 to 10 minutes intervals. If after 10 mgm atropine, the pupils remain pin point, it is our practice to start an infusion of 25 to 50 mgms in 5% Dextrose. The drip rate is adjusted depending on the degree of poisoning. With the infusion, additional doses of atropine may be necessary evry 15 min. The total dose of atropine necessary may range from 4 mgm to 1000 mgm. Our last patient who survived severe poisoning needed 1200 mgm. The duration of atropine therapy may extend up to 3 to 4 days and could be discontinued when pupil size is normal.

Pralidoxime ("PAM"), releases cholinesterase from its combination with organophosphate and the released enzyme inactivates acetylcholine. PAM also prevents further combination of organophosphate with fresh cholinesterase. PAM should be administered as early as possible preferably together with atropine. However atropine should not be delayed until PAM is available. The dose of PAM is 1 G. diluted in 25 ml of water and given intravenously. Further doses of 1 G may be repeated every 4 to 6 hours, depending on the necessity, as assessed by muscle twiching.

Diuretics—Frusemide 20 to 40 mgm intravenously helps in the rapid relief of pulmonary oedema and may have to be repeated.

Until recently, the severity of organophosphate poisoning has been assessed entirely on clinical grounds. However work done recently at the Department of Pharmacology, University of Jaffna[1], has suggested that estimation of pseudocholinesterase levels in the serum would be of value in assessing severity and prognosis. It was found that pseudocholinesterase levels are low and take longer to reach the normal range in severe poising.

Chlorinated hydrocarbons

Some examples are Carbontetrachloride, Tetrachlorethyline, D. D. T. Aldrin.

They are used as solvents and dry cleaning and degreasing agents. Some are used as insecticides. Poisoning may result from inhalation in confined spaces or by ingestion.

The effects are predominantly on the Central Nervous System producing excitement, tremors and convulsions. In the later stages drowsinese, ataxia and coma result. The liver and kidneys are also affected,

Management consists of removal from the environment and adoption of usual measures. Liver and kidney function should be monitored. N-acetyl cysteine should be given especially for carbon tetrachloride poisoning.

Paraquat

Though organophosphate is the common poisoning agent in Jaffna peninsula, poisoning with paraquat is not uncommon. It is commonly available as Gramoxone or Weedol and is used as a weedicide. It could be absorbed through the skin but this form of poisoning is rare and most cases are of self poisoning.

Paraquat causes multiorgan involvment. The main pathological features are ulceration of the mouth, gastric erosion and bleeding, renal tubular necrosis and liver necrosis.

Clinical course depends on the amount of poison taken. If the dose is greater than 6 G, patient presents with nausea. vomiting, abdominal pain, becomes breathless and hypotensive and dies within 12 to 24 hours of ingestion, before developing mouth ulceration and hepatic damage. If the dose is 3 to 6 G, gastric irritation is the main feature initially. 72 Hrs later biochemical evidence of hepatocellular and renal damage becomes apparent. 5 to 7 days later, patient becomes tachypnoeic' hypoxic and dies. When the dose is 1·5 to 3 G, the initial symptoms are mild like nausea, vomiting. After about 10 days, patient becomes breathless, jaundiced with renal failure as well Radiological evidcnce of fibrosis of the lung develops gradually. In early admissions, urine screening test and plasma concentration are useful in predicting the clinical course and prognosis. Urine screening test consists of the addition of sodium dithienal to 5 ml of urine made alkaline with sodium bicarbonate. A blue colour results, if sufficient paraquat had been ingested

Management consists of stomach wash in early admissions and administration of Fuller's earth. There is no specific antidote and the usual supportive measures are adopted. Forced diuresis and peritoneal dialysis need evaluation. Liver failure is managed as usual.

When pulmonary fibrosis develops, the prognosis is grave, Oxygen administration is avoided in the treatment, as it aggravates the course of the disease.

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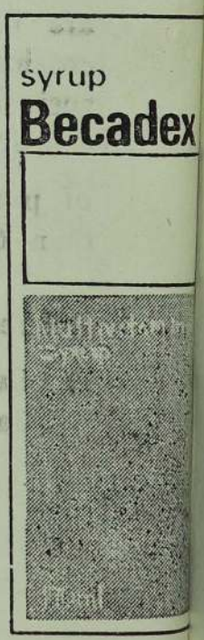

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Plant Poisoning in Sri Lanka

Saravanapavananthan T.*

The literature on poisonous plants in Sri Lanka is sparse. This article is not a complete treatise on the subject, but a documentation of poisoning by some common plants.

Chopra in 1965[1], defined a plant as poisonous, when the whole or a part thereof under all or certain conditions and in a manner and in amount likely to be taken or brought into contact with an organism exerts harmful effects or causes death either immediately or by reason ol cumulative action, due to the presence of known or unknown chemical substances in it and not by mechanical action.

The poison in a plant is very variable and is very often an alkaloid as in the case of hyosine. It may also be a glycoside as is cerberine in cerbera manghus, a resin, essential or fixed oil, toxic proteins or an unidentified substance.

Some of the poisonous plants are shown in table 1.

Manihot utilissma (Cassava, "Manioc", "Tapioca". Fig. 9. The red variety was used extensively in Sri Lanka as a food crop during the second world war when rice was a scarcity and several deaths occurred following the ingestion of manioc. Extensive research was carried out in Sri Lanka by Charavanapavan 1944[2], Joachim and Pandithasekara 1944[3], regarding the toxicity and how it could be attenuated. It was found that poisoning was due to hydrocyanic acid or prussic acid present in the roots and leaves as a cyanogenetic glycoside, linamarin which is present in two forms the autolysable aad hydrolysable forms. They also contain the enzyme linase which releases the autolysable or free prussic acid. This occurs after injury to the roots or when the roots are left exposed to the atmosphere for some time after lifting and during boiling or cooking at temperatures below 72°C, as the enzyme is destroyed at temperature above this. Some varieties remain hard or bitter after cooking when they have a high content of the hydrolysable or bound HCN and these are more dangerous.

The peel has a very high content–110 mg/100g while the flesh has about 16 mg/100 g when fresh. The young leaves have a greater content than older leaves and fresh leaves have about 78mg/100g. Cutting, washing and boiling in open vessels at temperature below 72°C reduces the content to a marked extent. Tapioca powder containing up to 5mg/100g is allowed as a food.

Signs and symptoms – may come within 2—3 hours and consist mainly of diarrhoea, vomiting and fainting. Death can occur in less than 12 hours. Pohath Kehelpanala 1907 has reported several cases of poisoning—most of them being pathetic. In one instance the parents had gone on a pilgrimage and returned the next day to find their three children dead, after having partaken of a meal

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of manioc for dinner. Resuscitative measures could be adopted if the patient is brought to hospital early in the course of the disease. Since cyanide has an affinity for iron in the ferric state it reacts with cytochrome enzymes thus inhibiting cellular respiration. Haemoglobin remains in the unreduced state giving the characteristic cherry red colour to blood at post mortem.

Treatment consists of specific antidotes and symptomatic treatment. 10ml of a 3% of solution of Sodium nitrite is given intravenously in order to oxidise haemoglobin to Methaemoglobin. This provides the ferric iron to combine with the cyanide thus releasing the cytochrome enzymes. Sodium thio–cyanate 50ml of a 20% of solution is also given intravenously to accelerate the conversion of cyanide to thio-cyanate which is non-toxic and excreted easily. Cobalt edetate 300mg in 20ml of glucose given intravenously slowly over 3–4 minutes is the more modern treatment and it produces dramatic effects. Anaphylaxis and cardiac arrythmias may occur if given too rapidly.

Chronic cassava poisoning may lead to nephropathy and goitre formation.

Table I

Common Poisonons Plants of Srilanka

Botanical Name	*Family*
1. Manihot utilissma	Euphorbiaceae
2. Modecca palmata or Adenia palmata	Passifloraceae
3. Thevetia peruviana Cerbera manghas Nerium oleander Nerium oderum Plumeria acumininata	Apocyanaceae
4. Gloriosa superba	Lilliaceae
5. Strychnos nux vomica	Loganiaceae
6. Ricinus communis	Euphorbiaceae
7. Abrus precatorius	Leguminosae
8. Datura fastuosa	Solanaceae
9. Mushrooms (edible)	

Local Irritants

1. Calotropis gigantica Calotropis procera	Asclepiadeae
2. Euphorbia antiguorum Euphorbia tortilis Euphorbia tirucalli	Euphorbiaceae
3. Pedilanthus tithymaloides	

Modecca palmata or Adenia palmata which is a climber belonging to the passifloraceae family is another plant which exhibits the effects of cyanide poisoning. The fruit is very similar to Passion fruit and children ingest them accidently leading to fatal or non-fatal cases of poisoning. They may be taken with suicidal intent. Munasinghe et al 1971[4], described 2 cases. One was a 14 years old child who had eaten 2 fruits. He was treated in hospital and survived although he developed the complications of myocarditis and a retinopathy which were temporary. The other, a child 7 years old succumbed to the poison and at post mortem, signs of severe gastro enteritis due to the toxalbumin were seen.

Balasubramaniam & Bibile (1966)[5] analysed the fruits and found the amount of free hydrocyanic acid to be low. Ingestion of 2 fruits can lead to poisoning. Two distinct poisons are responsible for the symptoms — toxalbumin for gastro enteritis and cyanogenetic glycoside for the vomiting, dizziness. and other

Fig. 1 Thevetia Peruviana.

Eig. 2. Gloriosa Superba.

Fig. 3 Cerbera manghas. — Fruit,

Fig. 4. Datura fastuosa—Fruit,

anoxic symptoms. The late symptoms consist of myocarditis, hepatitis and retinopathy which are all transient.

Thevetia or "Manchal Alari" Fig. 1 is a very common plant especially in the North. Ever since the fact that the fruits and kernel are poisonous was published in the news papers, many cases of poisoning due to Thevetia occurred. Thevetia is a tree, the flowers of which are flamboyant and yellow in colour and used in temples. The leaves are long and linear and spirally arranged. The fruits are fleshy and about the size of a small lime. It is elliptic in horizontal section and black when ripe. Fruits are present almost all the year round.

All parts of the plant abound in a milky juice which is highly poisonous. The bark has been used as a purgative and emetic and the seeds as an abortifacient.

The kernel of the seeds are ingested with suicidal and homicidal intent and also accidentally by children. The kernel of one fruit in the case of a child and 10 or more in the case of adults may prove to be fatal. Cattle grazing on grass under these trees are known to have died.

They produce numbness and burning of the mouth, vomiting and diarrhoea and depression of the heart as shown by ST sagging, resembling that produced by digitalis. The poisonous substance is a glucoside, Thevetin. Treatment is empirical-anti-arrythmics and atropine being used depending on the rhythm disturbance seen with a cardiac monitor. Successful recovery occurs although some end fatally.

Sreeharan & Puthrasingam (1984)[6] spotlighted the increase in the incidence of poisoning with Thevetia in recent years in the Northern Peninsula. It was commoner in young females & the predominant cardiovascular manifestations ranged from sinus bradycardia with sino atrial block, first and second degree heart blocks, junctional rhythms, atrial and ventricular ectopics and ventricular fibrillation. The mortality in the series was 10%.

Ipomea carnea An interesting case was reported by Dr. Shivendra Singhai and Dr. R. S. Naik, 1984[7] where a person who drank the tea which was brought to him covered by the leaf of Ipomea carnea died. This plant is grown in hedges and has a funnel shaped purple flower. It is known as Seemai Kiluvai in Jaffna. Animal experiments were done by the authors and they identfied two toxins, one of which was a Cardiotoxin.

Cerbera manghas or "Natchukkai" or 'Kadal ma" (Fig 3) As is commonly termed it is seen only in particular districts in our country. The word cerbera is derived from cerberus the 3 headed dog that guarded the entrance of Hades and whose bite was poisonous. It is a huge tree like the mango and its leaves are oblong like the temple tree. The fruits are fibrous and about the size of an orange. The kernel contains the glycoside cerberine, odollum and thevetin which are cardictoxic (Kumudesan 1985). It is usually taken minsed with sugar or jaggery as the kernel is very bitter. It is a common poison in South India and much work has been done on this subject in India.

The symptoms are similar to those of Thevitia, vomiting comes on within half to two hours and progresses to bradycardia and hypotension. Symptomatic management with Atropine, and

Ephedrine make recovery possible in some cases but some ended fatally (unpublished data—Jega Pasupati).

The kernel of two fruits, if not vomited was sufficient to cause death. It is no doubt posible that management under more sophisticated conditions with cardiac monitoring and electrolyte assessment would produce more fruitful results. Vijayaragavan 1985, has shown that hyperkalaemia can occur in some instances. Other members of the Apocyanaceae group also contain cardiotoxins.

Gloriosa superba (Fig. 2) tubers are ingested by some with a suicidal intent. Colchicine is the main alkaloid found (Dunuwille et al 1968)[8], 10g of tu ber contain about 6mg colchicine and the lethal dose may be about 60mg for an adult. It also contains Gloriosine. The clinical presentation is one of gastro enteritis leading to shock. Alopecia, depilation and polyneuropathy occur. The last results probably from damage to the schwann cells as recovery is fairly rapid and complete. It also has a depressant action on the marrow.

Angunawela and Fernando 1971[9], reported a case where a teenager had ingested about 30g of the tuber containing about 18mg of Colchicine. She developed vomiting and a profuse bloody diarrhoea, an ascending paralysis and alopecia. Recovery was complete by four weeks,

Strychnos Nux vomica: Fig. 7 Poisoning due to ingestion of seeds of Strychnos Nux Vomica has not been reported in Sri Lanka but the tree is commonly seen. The seeds are flat and round having a peculiar concavo convex shape and about 1" in diameter. Strychnine is present not only in the seeds but also the root, bark leaves and wood. Each seed has about 30 grains of powder and contains a lethal dose of strychnine. The symptoms consist of restlessness,, nervousness, muscular twitching and a feeling of rigidity in the neck. With large doses symptoms develop in 15 – 30 minutes with opisthotonus, rhisus, and respiratory muscle spasm leading to asphyxia and death. It is used as an aphrodisiac in some parts of India.

Castor seeds ("Cinthamanakku") and Abrus precatorius seeds ("Kundumani") Fig. 13 produce similar symptoms although they belong to widely differing families the Euphorbiaceae and Leguminosae respectively. Poisoning with these have not been reported in Sri Lanka although they are common.

Castor is grown for commercial purposes as the oil is used as a purgative while the Abrus or rosary bean is grown for ornamental purposes. Ingestion of only one castor bean or rosary bean has caused fatal poisoning when the bean was throughly chewed. The toxic substances are ricin and abrin respectively and the seeds cause no harm if swallowed whole. They cause agglutination and haemolysis of red cells when used in extreme dilution. The patient presents with diarrhoea, vomiting, circulatory collapse, retinal haemorrhages, haematuria and convulsion. Inhalation of the dust leads to allergic phenomenon.

Datura Fastuosa, Figs. 4, 6, Poisoining is occasionaly seen at the fruit though thorny is attractive and children may eat the seeds accidentally. Teenagers knowing the danger may use it for suicidal purposes as they have ready access to the small shrubs.

Fig. 5, Ricinus communis,

Fig. 6. Datura fastuosa.

Fig. 7. Strychnos nux vomica.

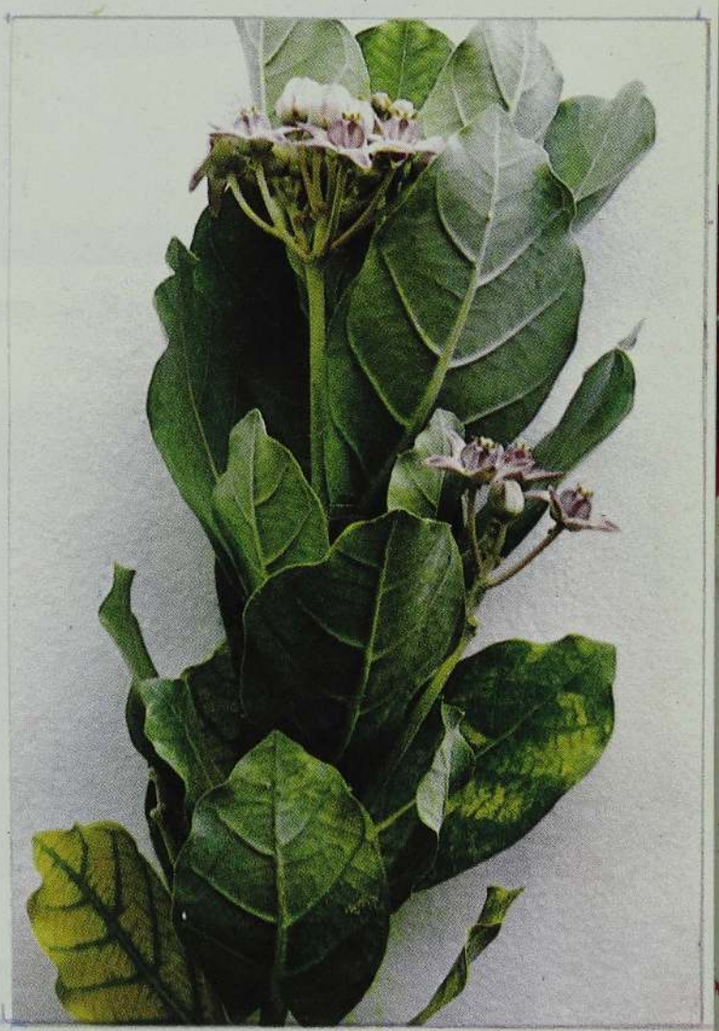

Fig. 8. Calotropis gigantica.

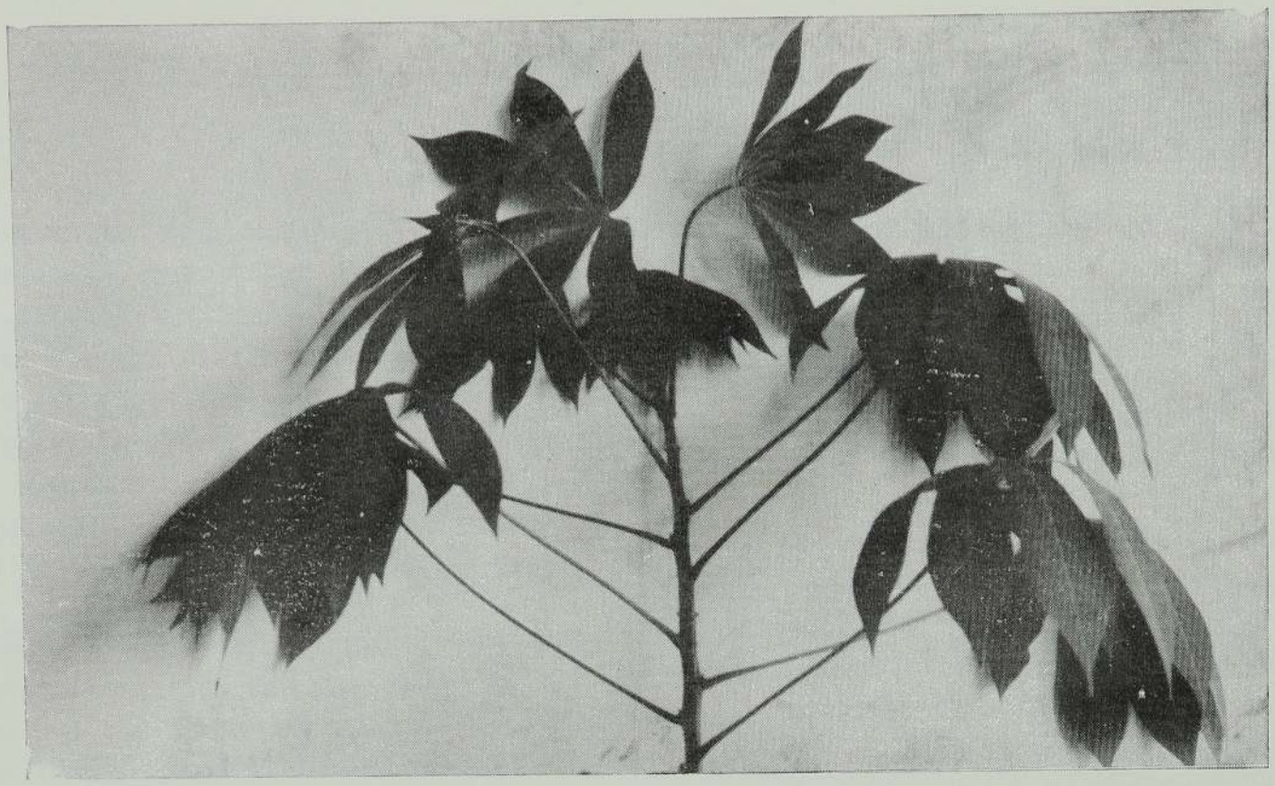

Fig. 9. Manihot utilissma.

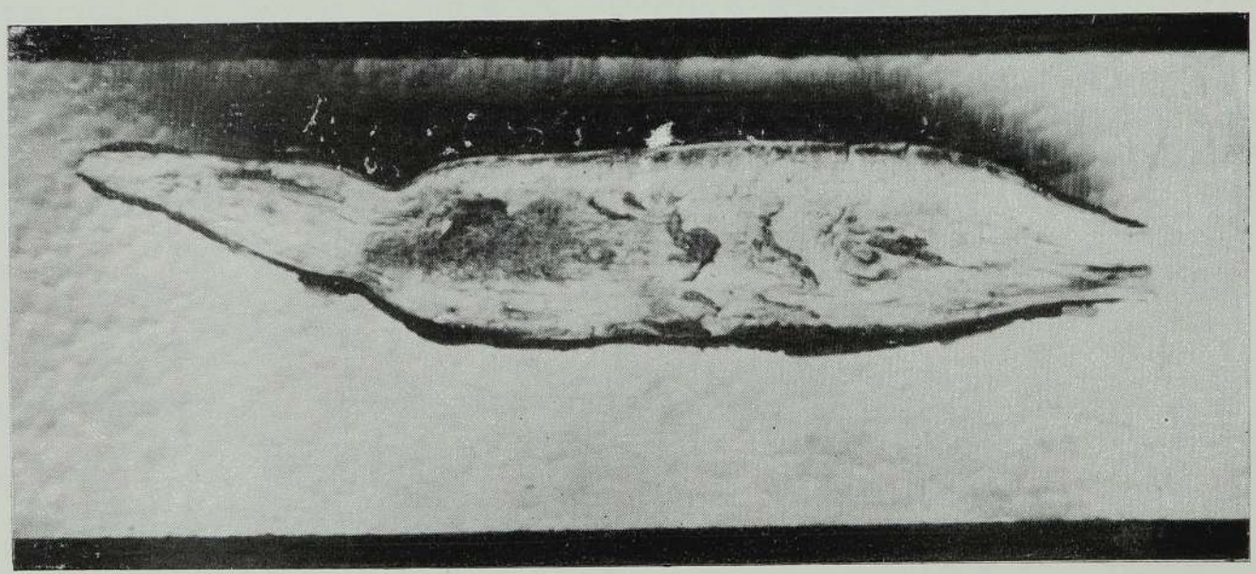

Fig. 10. Manihot utilissma — Root (damaged)

The signs and symptoms are due to hyoscine overdosage—anticholinergic action and is well illustrated by the dictum "hot as a hare, blind as a bat, dry as a bone, red as beet, and mad as a hen". They may develop convulsions and coma later. The antidote is physostigmine I·V and stomach wash is effective as emptying is delayed with antisholinergic drugs.

Mushrooms both the poisonous and non poisonous varieties are seen in Sri Lanka but the habit of consuming them is fast dying out so that cases of Amanita phylloides poisoning are rather rare.

These produce symptoms of muscarnic action leading to severe diarrhoea vomiting, bradycardia and increase in salivation. Treatment is symptomatic. Anticholinergic may be useful in the early stages and later thiotic acid and glucose I. V may be useful.

Many plants abound in a milky juice which is poisonous or irritant. eg. Calotropis, Euphorbia tirukalli (Fig. 15) etc.,. Recently Lim and Engkik Scepadimo 1984[10], have reported a case where a child sustained a corneal erosion while playing. The milk of the plant Pediladthus tithymaloides poit (Fig. 14) came in contact with the eye when he fell down. Subsequent animal experiments proved that the milk from the stem was capable of producing a corneal erosion, in the rabbit eye. However the lesion healed without any residual visual defect. Though this cannot be considered as plant poisoning according to Chopra, it is worth while remembering, as this plant is commonly grown in many gardens.

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Fig. 12. Nerium oleander

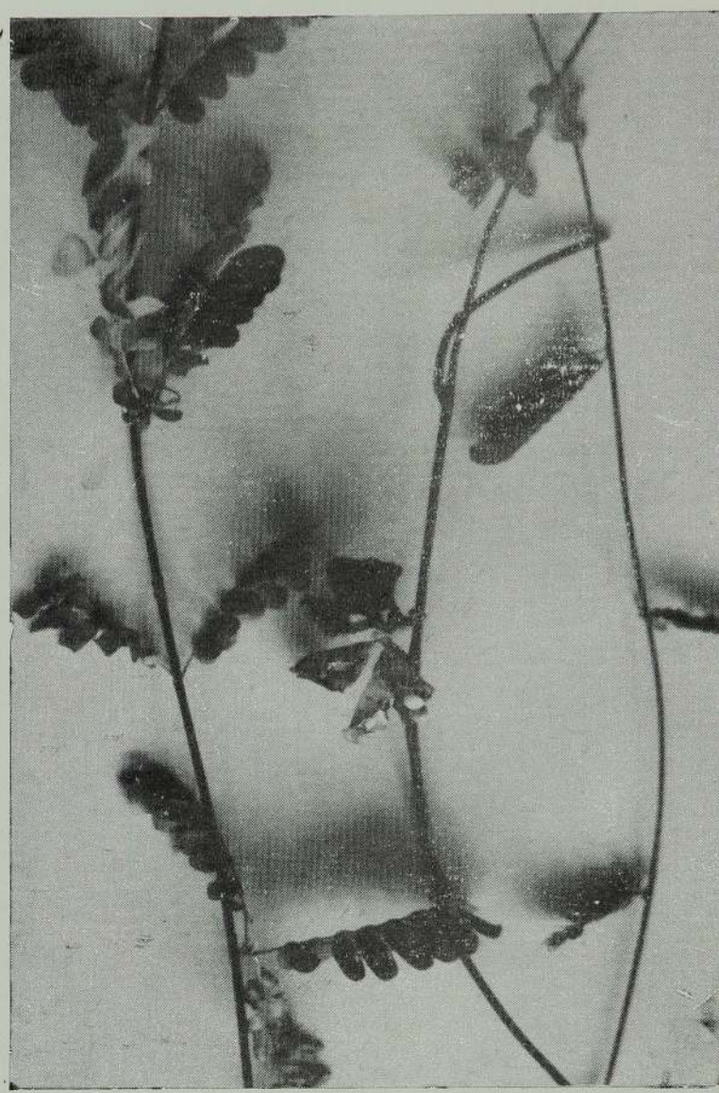

Fig. 13. Abrus precatorius.

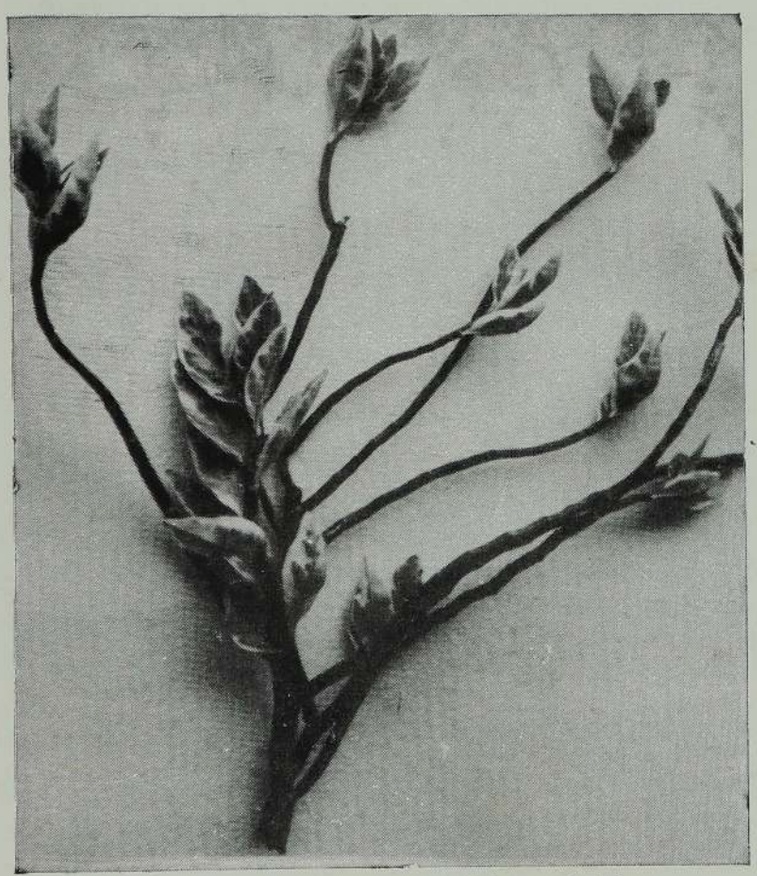

Fig. 14. Pedilanthus tithymaloides.

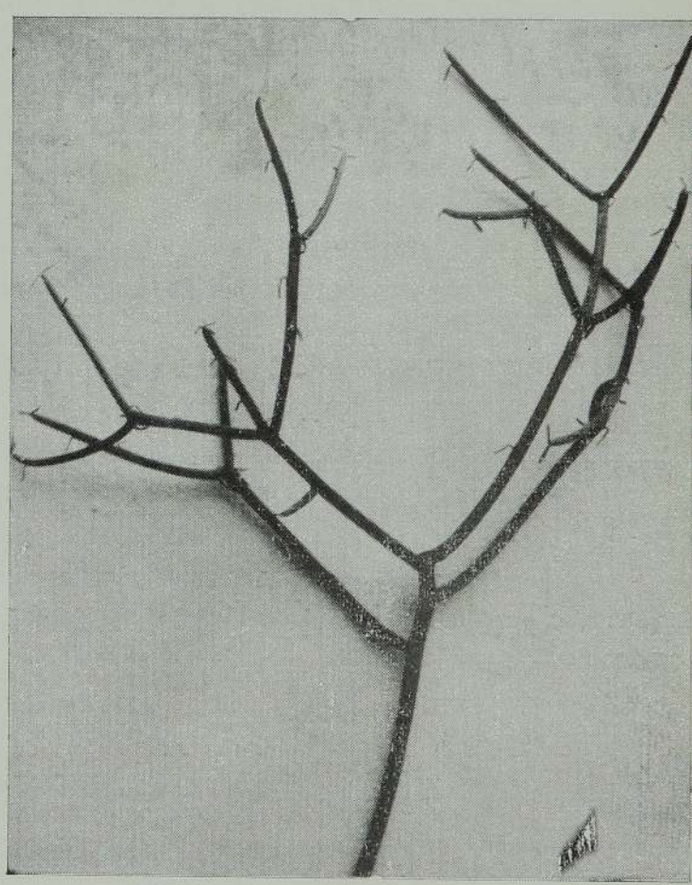

Fig. 15. Euphorbia tirucalli

Snake Bite Admissions To Base Hospital, Mannar.

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Summary

Seventy Six cases of snake bite were seen in our Unit over a period of five months from October 1983. Most of the patients sought treatment within 24 hours of being bitten. Only 3 patients presented 4 to 9 days after the bite, having taken treatment from native physicians. The types of snakes identified and the clinical features are recorded. There were no deaths in our series.

Introduction

Several reports of snake bites in Sri Lanka have appeared over the years. No specific mention appears to have been made of snake bites in Mannar district. A retrospective study was, therefore made of the cases admitted to Base Hospital, Mannar for the period mentioned.

Materials and Methods

The Records of cases admitted to the only medical unit, for the period of five months from October '83 were examined. The species involved and the clinical features were noted.

Results

Table I shows that only 20 snakes were identified. Identification was by us, when the snake was brought to the hospital or by patients or their relatives. Majority identified were vipers (10) and cobras (7). No krait was identified but this does not necessarily mean that there were no bites by Kraits.

Table I
Snakes Identified

Type of Snake	Number
Viper	10
Cobra	07
Sea Snake	03
Total	20

Table II shows the clinical features noted in our study. Vasculotoxic effects were seen in 39 cases. Combination of vasculotoxic and neurotoxic effects were seen in 8 cases. 17 patients had neurotoxic effects, such as ptosis, respiratory paresis, glossopharyngeal palsy, ophthalmoplegia, either singly or in combination.

Respiratory paresis was monitored by counting the respiratory rate, ability to blow off a candle, and ability to count numbers in one breath. None of the cases required assisted ventilation. Myalgia was observed in all the sea snake bites but not muscle stiffnes or

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weakness. 80% of the patients had local swelling. It was usually of rapid onset and marked. None of them had local necrosis. No patient was complicated by renal failure. Vomiting was present in 10 cases. Leucocytosis of 12,000 to 15,000 per c. mm was seen in 63 cases– as a nonspecific but early manifestation of systemic poisoning.

Table II
Clinical Features

Clinical features	Number of cases
Prolonged Clotting time	39
Haematuria	08
Glossophare	03
Ptosis with normal clotting time	03
Ptosis with prolonged clotting time	07
Respiratory paresis	01
Ophthalmoplegia with normal clotting time	02
Ophthalmoplegia with prolonged clotting time	01
Diminished foetal movements	01
Myalgia	03
Vomiting	10
Haemoptysis	01
Local Swelling	60
Oliguria	00
Anuria	00
Necrosis	00

Discussion

The incidence of snake bites for the Island is quoted as 61,000.[1] The number of snake bite admisions to hospitals in 1980 varied, with Colombo recording the highest (1759), followed by Jaffna (418), Vavuniya (381) and Batticaloa (68).[2] It is not clear whether the figure for Vavuniya includes that of Mannar. For five months we had 76 cases, which works out to about 180 for a year—not allowing for variation in seasonal agricultural activity.

Only 20 snakes were identified in our series. It is significant that not a single Krait was identified. De Silva. A[3] found from many published and unpublished sources, that the Incidence of bite, according to species was; Krait, 8 6%, Cobra, 12%, Russel's Viper, 17·5%, Merrem's Viper 27.1% and Others 29% with Unspecified 5.8%. Nonidentification of Kraits in our series was probably due to a large number of snakes responsible being not identified. and not due to the poverty of the species. As in other series[4] most of the victims were cultivators. The three cases of bites by Sea Snakes occured in fishermen. Sea Snake bites are reported to be uncommon in Srilanka.[5] Karunaratna and Panabokke[6] reported one case of sea snake bite in a fisherman.

The Clinical features and Management, among other aspects have been extensively dealt with in the Ceylon Medical Journal.[7] The features noted by us were similar to that of other series. Even though Cobras are mainly neurotoxic and Vipers mainly vasculotoxic, the effects occuring either alone or in combination is not unusual in some cases.[3,9] and this was the pattern seen in our series too. Vasculotoxic effects were seen in 39 cases and combination of vasculotoxic and neurotoxic effects in 8 cases. Myalgia was noted in all the seasnake bites but muscle stiffness and muscle weakness reported[10], was not observed.

An interesting feature was seen in a female patient who was 8 months pregnant.

About 4 to 5 hours after the snake bite. the foetal movements ceased but returned 48 Hrs after the administration of the anti-venom. This raised the possibiiity of venom crossing the placenta and causing some neurotoxic effect on the foetus, even though the dose of venom was insufficient to cause, similar effects on the mother.

The death rate from snake bites is reported[11] as 5.7 per 100,000 population in 1979. The number of deaths in Anuradhapura for 1982 was 36[12]. There were no deaths in our series. This could be due firstly to our patients having come to hospital soon after the bite. It is reported[13] that 72% of the deaths occured within 12 Hrs and most of them were treated by traditional physicians before coming to hospital. Secondly our series may not include all the cases of snake bites in Mannar district, though it represents most of them. On an average we used about 40 ml of antivenom, some requiring only 20 ml and others, 60 ml. Using minimum amount of antivenom is possible when the patient is carefully monitored in hospital.

Finally, it is worth noting, that in Nigeria. for every five cases treated in a University Unit, the native healer was treating several hundreds of cases only a mile away from the hospital. In Mannar District. during the period mentioned, only 16 cases are reported to have sought treatment from the native healer This shows the positive attitude of the patients (in this district) towards "Western" treatment.

Acknowledgements

We thank the D. M. O. Mannar for permission to publish.

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Bacterial toxins causing food borne poisoning

Nageswaran. A*

The major recognised toxins causing food borne poisoning are produced by staphylococcus aureus. clostridium botulinum, clostridium welchii, and bacillus cereus.

Staphylococcus aureus

Staphylococcus aureus strains that produce enterotoxin are the ones that cause severe upper gastrointestinal symptoms, very soon after ingestion. Five immunologically distinct types of entero toxins (A, B, C, D & E) are recorded. The most common source of these organisms is the human carriers who harbour these organisms in their nasopharynx. The other sources are milk and dairy products which are contaminated from infected udder. Certain foods seem to favour the growth of these organisms and production of enterotoxin, once contaminated. These are mainly pork. salted meat. cream filled cakes and canned beef. The dose of entero toxin needed to cause gastrointestinal symptoms differs between the 5 immunological types. The symptoms caused by this enterotoxin are mainly upper gastrointestinal causing profuse vomiting, nausea and abdominal carmps. These symptoms are often followed by diarrhoea. In majority of patients the symptoms subside within 24—48 hrs. In the extremes of age complications from severe dehydration are not uncommon. The management of these patients is mainly correction of fluid and electrolyte loss.

Bacillus cereus

This gram(+)ve spore forming rod secretes two distinct types of toxins, a diarrhoeogenic enterotoxin and an emetic enterotoxin The first type of toxin causes, mainly profuse watery diarrhoea and abdominal cramps, and this syndrome had been known to be produced by B. cereus since 1906. Many vehicles had been implicated in this type of food poisoning. Spores of these organisms can survive extreme temperatures and when allowed to cool very slowly can germinate, multiply and produce toxins. The syndrome caused by the second type of toxin which is characterised by severe vomiting and abdominal cramps with mild diarrhoea in few patients was first noted in 1974. This syndrome has so far been described involving only fried rice obtained mainly from Chinese restaurants where it is the practice to keep large amounts of boiled rice unrefrigerated (to avoid clumping) and then to flash fry the fried rice just before serving. During this process B. cereus multiplies and produces a heat-stable toxin in the boiled rice which escapes denaturation during the flash frying. The most likely source of B. cereus in all these instances was the uncooked rice and the practiee of slightly warming up, already cooked and kept rice, could eertainly cause many outbreaks in Sri Lanka. The symptoms, which occur within 1—6 hrs of ingestion of the food, disappear in most instances within 8—10 hrs and as such most patients would not end up in hospitals.

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Food borne botulism

Food borne botulism is caused by ingestion of food contaminated with preformed toxin of Cl botulinum. This illness had been recognised since the late 18th century and was first linked with eating contaminated sausage and hence described as sausage poisoning. Later epidemics were linked to home canned foods. The clinical syndrome differs from other food borne poisonings in that the features are predominantly neurological, appearing usually after 18—36 hrs of ingestion of the offending food. The neurological features result from toxin induced blockade at the peripheral cholinergic neuromuscular junctions. Initially dry mouth blurring of vision and diplopia are noticed, followed by (in more severe cases) flaccid paralysis and cranial nerve palsies. Respiratory muscle paralysis necessitates artificial respiration at this stage. Mental status is normal through out the illness. Gastro intestinal symptoms are not caused by the botulinum toxin and if these symptoms are present they are due to other bacteria and their toxins concomitantly found in the improperly preserved food. The rapidity of progression of symptoms in botulism differentiates it from other neurological illnesses. But in some, this can be slowly progressive and hence the diagnosis could be missed, especially so if the cases appear in isolation. But when several people who shared a meal fall ill with ocular and pharyngeal paralysis, the diagnosis is always botulism. The treatment of botulism is essentially supportive with maintenance of breathing by tracheostomy and artificial respiration. Although botulinum antitoxin is given to these patiants, its value is doubtful.

Clostridium Welchii causes two distinct types of gastro intestinal syndromes through its toxins. The commoner syndrome is caused by an enterotoxin produced by clostridium welchii type A resulting in watery diarrhoea with severe, abdominal cramps begining 8–24 hrs. after eating contaminated food. Vomiting is usually absent. Any form of meat left for a length of time before consumption can be the vehicle of this toxin. The second syndrome is caused by an enterotoxin of cl. welchii type C which results in a necrotising form of enteritis (enteritis necroticans). This toxin too seems to be produced in meat left for a long time before consumption and attack rates seem to be higher in the extreme age greups. The clinical illness starts on the day after the meal, with abdominal pain, vomiting and diarrhoea often containing blood. These symptoms may be followed by very rapid deterioration in the patient's condition and early death. Some patients develop clinical features of intestinal obstruction due to characteristic patchy swelling and necrosis of small intestine. Majority of patients respond to medical treatment with nasogastric suction, IV fluids and antibiotic therapy with Penicillin and Chloramphenicol. Patients who fail to respond to the above treatment and those who develop necrosis and complications such as perforation, peritonitis and intestinal obstruction would need surgical removal of affected intestine.

The symptom complex of acute onset vomiting, abdominal pain and diarrhoea could occur due to the effect of preformed toxins or due to infections by bacteria or virus. Short iucubation period, absent fever, and absent pus

cells in stools would favour toxin type food poisoning. Tbe management is essentially same for both infective and toxin type food poisoning, this being correction of fluid and electrolyte balance. This can be achieved by oral electrolyte solutions when diarrhoca is mild to moderate and the patient not severely dehydrated, or by intravenous fluid therapy using 0.9% NaCl. Anti emeties could be used when vomiting is troublesome, but antidiarrhoeal agents are generally not advocated as they are found to be ineffective and at times harmful. Antibiotics have no place in the treatment of both toxin and infective type of food poisoning except when there is evidence of bacteraemia in the latter type.

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Acute Poisoning — A Quick Look*

General Principles Of Treatment.

General supportive measures directed towards maintaining the vital functions of the patient are the keystone to successful treatment. These should never be postponed to await accurate identification of the causative agent. The scope for specific antidotes is limited. Mention is made where applicable.

Respiration,

Ensuring adequate tissue oxidation, above all of the brain, is of first priority. A clear airway should be established at the outset. If spontaneous respiratory movements prove inadequate, artificial ventilation should be applied. Where the respiratory depression is due to Morphine and related drugs, naloxone is the specific antagonist given intravenously in a dose of 0.4 to 0.8 mgm. The action of this drug is extremely rapid, and the dose should be repeated if obvious improvement has not taken place within three minutes. Thereafter, because the duration of effect from naloxone is relatively short by comparison with that of of the opiate overdose, successive doses of this antidote may have to be administered as indicated by the condition of the patient. Other non-specific respiratory stimulants like nikethamide and picrotoxin are best avoided.

Cardiovascular System.

Shock should be promptly corrected. A head-down position often produces a satisfactory response. Otherwise the blood volume should be augmented by infusing colloids intravenously. If these measures are found inadequate, it is better, then to administer a beta-adrenergic drug, e. g. isoprenaline by continuous intravenous infusion for its central cardiac action, or dopamine or dobutamine. The intraven ous administration of an alpha-adrenergic agent is seldom of avail, for maximum peripheral vaso-constriction has usually already occurred as a physiologic response.

Convulsions.

Suppression is usually achieved by injections of diazepam intravenously, in doses of 5–10mgm, which can be repeated. If the convulsions persist, paraldehyde 5–10ml may be given intramuscularly or chlormethiazole edisylate by infusion. Under these circumstances respiratory depression may be aggravated and more ventilatory assistance may have to be provided.

Body Temperature.

A dangerous fall in body temperature can occur with overdosage of drugs that are central depressants, more so when

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* *From notes prepared by Dr. R. Goulding, Director of the Poisons Unit. Guy's Hospital, London and published in Wellcome Medical Diary, 1981, With section on General Principles of Treatment abridged and some inclusions (marked +). The kind permission, of Dr. Goulding and Wellcome Foundation Ltd. to publish in this journal is acknowledged.*

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the patient has lain comatose for some hours, before being discovered. With this possibility in mind, the rectal temperature should always be checked and, if it is below normal, careful and slow warming should be carried out in severe cases. Corticosteroids should be given as well.

Fluid and Electrolyte Balance

Poisoned patients in whom resuscitative management has been delayed may well be dehydrated. This may be intensified if vomiting and/or diarrhoea has taken place. Not only must the fluid state of the body be restored, but the electrolyte status, too, if need be, according to the biochemical measurements. A check should be maintained on renal function. Forced diuresis is worthwhile only with a few drugs in overdose. eg. salicylates, barbitone and phenobarbitone (vide infra).

Pain

May arise if strong acids or caustics have been swallowed or irritant vapours inhaled. Morphine or related drugs may be given, bearing in mind that, if respiratory depression exists, it may be intensified.

Removal of the Poison

This may be contrived before absorption has taken place as by emesis or gastric lavage. Sometimes vomiting may have arisen spontaneously It not, a child who has ingested a dangerous overdose within the last few hours may be induced to do so, provided that consciousness is not impaired and the laryngeal reflex is intact, by being given an oral dose of 10–15 ml of Ipecacuanha Paediatric Emetic Draught (B. P. C.), followed by half a glass of water to drink. If this does not succeed within 15 to 20 minutes the dose may be repeated. Apomorphine should be avoided and so should coppersulphate, salt-and-water, etc. as emetics. In adults and in all comatose patients–the latter first being protected against bronchial aspiration by the insertion of a cuffed endo-traeheal tube gastric lavage should be considered if ingestion of poison has taken place within four hours (or up to possibly 12 hours with salicylates and tricyclic antidepressants) and if demonstrably effective vomiting has not come about spontaneously. Aspiration should be followed by lavage which should be discontinued at once if blood appears in the effluent. Aspiration and lavage are contraindicated if caustics and corrosives have been swallowed to such extent that oesophageal and/or gastric erosion and perforation are likely and also if paraffin (kerosene), white spirits and products containing these materials as vehicles or solvents have been ingested. For then the dangers of aspiration pneumonia may be intensified.

Suppression of absorption by prompt administration of activated charcoal may also be considered. but not when more than an hour or two have elapsed since ingestion.

Once the poison has left the alimentary canal and gained access to the tissues, the possibilities for inactivating it; or for accelerating its elimination, are very limited. Chelation may be feasible, as with desferrioxamine for iron salts, or renal excretion may be promoted effectively only with salicylates, phenobarbitone and barbitone, for which forced alkaline diuresis is a worthwhile procedure. Most of the drugs undergo metabolic inactivation in the body rather than excretion in the

unchanged state. Their half-life is, therefore, not shortened by an augmented urinary flow.

Nursing.

It will be apparent from what has already been written that comprehensive, conservative care is the basis of management for all poisoned patients and the most important single element in this respect is skilled and unremitting nursing attention. For severe cases the resources of an intensive care unit are imperative.

Social and psychiatric aspeets.

Poisoning in children is accidental. Toxic substances should be kept out of the reach of children. Any home which has been the scene ef child poisoning merits a visit from the health visitor. By contrast, adult poisoning is commonly self contrived. Such people are surely the victims of mental distrbance and shouid always be seen as soon as possible after the event by a psychiatrist and or social worker.

SPECIFIC POISONS: SYMPTOMS AND TREATMENT.

Poisons and symptoms	Treatment
ACETONE Amyl Acetate Nausea, vomiting, headache, excitement, fatigue, stupor, and coma.	Gastric lavage, Maintain high fluid out put. Sodium lactate infusion. General Supportive measures.
ACIDS, MINERAL Battery Acid, Hydrochloric Acid, Nitric Acid, Soldering fluid, Spirits of Salts, Sulphuric Acid. If swallowed, corrosions of mouth and stomach, with pharyngeal and epigastric pain and vomiting, Possibly intestinal perforation and peritonitis, Collapse.	Withhold gastric lavage, but give fluids orally to dilute, preferably of a demulcent nature eg, milk. Do not attempt to neutralise by giving alkalis by mouth. General supportive measures, Analgesics. Surgery for perforation.
ALCOHOL, ETHYL Surgical Spirits Burning of mouth and throat, vomiting, ataxia, dysarthria, depression of respiration. loss of consciousness.	Gastric lavage. Maintain high fluid output. Active supportive measures including maintenance of ventilation.
ALCOHOL, METHYL Burning of mouth and throat, Progressive loss of consciousness with abdominal pain. Acidosis. Depression of respiration. Blindness.	Gastric lavage. Liberal fluid intake. Supportive measures, especially for respiration. Correct acidosis. Ethyl alcohol intravenously to inhibit metabolism of methyl alcohol to formaldehyde.

AIKALIS Ammonia, Caustic Potash, Caustic Soda, Hartshorn. Lye. Burning and corrosion of mouth and throat Epigastric pain and vomiting. Dysarthria. Pallor and collapse.	Withhold gastric lavage. Fluids orally to dilute Give milk, white of egg olive oil or other demulcents. Do not attempt to neutralise by giving dilute acids by mouth. General supportive measures.
AMPHETAMINES+ Euphoria, excitement, insomnia, tremor, dilated pupils, dry mouth, disorientation, hallucination, stupor. Increased pulse rate and blood pressure, circumoral pallor, malar flush, brisk reflexes.	Gastric lavage. General supportive measures. Chlorpromazine and anticonvulsants intravenously.
ANTIDEPRESSANTS, TRICYCLICS, Amytryptiline, Desipramines, Imipramine, Nortryptiline, etc. Progressive loss of consciousness, increased tendon reflexes, dilated pupils, respiratory depression, cardiac dysrhythmias, convulsions and mydriasis.	Gastric lavage and maintain respiration. Control convulsions. General supportive measures. Correct acidosis anti-arrythmic drugs only if cardiac output and circulation in jeopardy
ANTIHISTAMINES Lethargy. drowsiness, muscular incoordination, confusion, convulsions, stupor	Gastric lavage, control of convulsions and general supportive measures.
ARSENIC COMPOUNDS Arsenical Weedkillers, Organic Arsenicals, some Sheep–Dips, some Vermin Killers. With inorganic, more than the organic compounds, epigastric pain, vomiting. diarthoea, muscle pains, collapse.	Gastric lavage and energetic supportive measures. Dimercaprol by injection. Restoration of fluid and electrolyte balance.
ASPIRIN see Salicylates.	
ATROPINE. see Belladona alkaloids.	
BARBITURATES Progressive loss of consciousness, weakness and respiratory depression or arrest. Hypothermia.	Gestric lavage and general supportive measures. Forced alkaline diuresis, only for barbitone and phenobarbitone in severe cases and haemoperfusion if short or medium acting barbiturates are involved.

BELLADONA ALKALOIDS Dry skin, mouth and throat. Thirst peripheral vasodilatation. Raised temperature, dilated pupils, tachycardia, delirium and excitement.	If swallowed, gastric lavage or emesis. Neostigmine by repeated injection until symptoms are abated. Oxygen and artificial respiration may be required. Parenteral fluids. Diazepam by injection.
CAMPHOR Camphorated oil Nausea, vomiting, dizziness, delirium, abdominal colic, dyspnoea, convulsions and anuria.	Gastric lavage followed by magnesium hydroxide by mouth. Control convulsions by diazepam, paraldehyde, etc. Maintain respiration. Haemodialysis if, renal failure supervenes.
CARBOLIC ACID. See Phenol.	
CARBON MONOXIDE Dizziness, confusion, tinnitus, stupor, coma. Respiratory depression, sometimes convulsions. No cyanosis.	Urgently maintain respiration together with oxygen at high concentration. For cerebral oedema, corticosteroids or mannitol intravenously.
CARBON TETRACHLORIDE Tetrachloromethane Vomiting, diarrhoea, abdominal colic, headache, confusion, stupor, respiratory depression. Hepatic, renal and cerebellar damage.	Gastric lavage followed by sodium sulphate. Avoid alcohol, adrenaline and oils by mouth. General supportive measures. Haemo-dialysis for renal failure. Treatment of hepatic failure.
CHLORAL HYDRATE Also Chloral, Betaine, Chloralformamide, Chlorbutol, Chlorhexadol. Dichloralpenzone. C. N. S depression with weakneas, loss of consciousness and depressed respiration.	Gastric lavage and customary supportive measures, with particular attention to maintaining respiration.
CHLORATES Bromates, Home-perm Heutralisers. Vomiting. diarrhoea, abdominal pain, haemolysis, eyanosis, convulsions and renal damage.	Gastric lavage followed by sodiun sulphate General supporrive measures and promotion of urinary excretion with haemodialysis if renal failure spervcnes.

COCAINE+ Excitement, pale skin. dilated pupils, convulsons, ventricular fibrillation.	Sedation and control of convulsious with Diazepam intravenously. Maintain respiration
COPPER SA1TS Blue Stone. Blue Vitriol, Verdigris Metallic taste in mouth, vomiting, abdominal pain. purging. rapid pulse, dehydration and eollapse.	Gastric lavavage followed by demulcents. Vigorous supportive measures to restore hydration, electrolyte balance and cardiovascular function. Penicillamine if there is evidence of systemic copper overload.
CYANIDES Hydrocyanic Acid, Prussic Aeid. Rapid onset of loss of consciousness, cardiovascular collapse. NO cyanosis.	If, any symptome, give inhalations of amyl nitrite at once, followed urgently by the specific antidote, viz. intravenous cobalt edetate or intravenous sodium nitritc and sodium thiosulphate.
DDT—See Organochlorine insecticides.	
DIGITALIS Nausea, vomitin", diarrhoea. Mental confusion, delirium, Fall of biood pressure.	Gastric lavage. ECG monitoring. For hypokalaemia give potassium. For hyperkalaemia give glucose-and-insulin. For tachycardia and dysrythmias give lignocaine and / or propranolol. For bradycardia give atropine. Foreed diuresis contraindicated, but haemoperfusion may avail in serious cases. Also, digoxin-specific, Fabamtibody fragments, if available.
DINITROPHENOL DERIVATIVES DNOC, Dinex, Dinitrocresol, Dinoprop, Dinosam, Dinoseb. Fatigue, insomnia, thirst, excessive sweating, pyrexia, hyperpnoea and dehydration. Collapse—often with yellowish coloration of skin and mucous membranes.	Absolute rest. If swallowed, gastric lavage. Cooling measures and general support with rehydration. Check severity and diagnosis by blood analysis.
ERGOT ALKALOIDS Excitement, weakness, pyrexia, vomiting, tremors, convulsiona. Peripheral vasoconstriction and abdominal colic. Shivering and parasthesia. Bradycardia and hypotension. Respiratory depression, stupor and coma.	Gastric lavage, Amyl nitrite inhalations. General supportive measures and sedation.

ETHYLENE GLYCOL Antifreeze fluids. Vomiting, tachypnoea, cyanosis, pulmonary oedema, stupor, coma. Marked acidosis, oxaluria, kidney damage and anuria.	Gastric lavage, Maintain respiration and give calcium gluconate, intravenously (checking serum calcium levels). To inhibit dangerous metabolism of glycol give ethanol intravenously 100 to 150 mgm per kg body-weight per hour. Promote diuresis and, if renal failure supervenes, haemo-dialysis.
FLUORIDES Sodium fluoride, Stannous fluoride. "Burning" sensation in mouth and throat, Dysphagia, thirst. salivation, vomiting. diarrhoea. muscle cramps, weakness, sweating, respiratory and cardiac failure.	Gastric lavage with lime water. Cal gluconate 10G in 250ml by mouth and then 10ml of 10% soln intravenously. General supportive measures.
FORMIC ACID Kettle descaler "Burning" and ulceration of the mouth, and throat, corrosion of glottis, oesophagus and stomach, Collapse. Sometimes gut perforation.	Delicate gastric lavage with lime water followed by demulcents. Relief of pain General supportive measures. If perforation, surgery.
IRON PREPARATION Nausea, vomiting, diarrhoea, melaena, collapse and encephalopathy.	Gastric lavage, followed by desferrioxamine 5G by mouth and then desferrioxamine parenteraly, monitoring serum iron levels. General supportive measures.
LEAD Soluble lead salts on ingestion : Metallic taste, nausea, vomiting, diarrhoea, black stools, oliguria. collapse and coma.	Gastric lavage and general supportive measures. Relieve pain. Remove excess systemic lead by sodium calcium edetate, or penicillamine.
LITHIUM CARBONATE + Thirst, nausea, vomiting, diarrhoea, frequency of micturition, drowsiness, tremor, hyperflexia and jactitation,	Gastric lavage. General supportive measures. Promote fluid intake but avoid diureties. Anticonvulsants.
LYSERGIDE LSD Excitement, disorientation, hyperactivity and hallucinations.	Sedate with injections of chlorpromazine. General protection and support,

MERCURY Soluble mercurial salts : vomiting and diarrhoea with collapse. Organic mercurials can cause brain damage, ataxia, blindness and renal damage.	Administer white-of-egg (orally) to form mercury albuminate and then perform gastric lavage. Dimercaprol by injeetion. General supportive measures. Correct fluid and electrolyte balance and relieve pain.
METALDEHYDE Metafuel ; Slug bait Usually delayed. then salivation, nausea, vomiting, abdominal pain, flushing of face, somnolence and fever. Muscular rigidity, tonic convulsions and coma. Liver and Kidney damage.	Gastric lavage followed by demulcents. General Supportive measures and control of convulsions. In preconvulsive stage, sodium lactate.
MONOAMINE OXIDASE INHIBITORS Drowsiness, depressed respiration, changes in muscle tone and blood pressure, coma. Acute reaction can occur in those receiving therapeutic doses, if they are given pethidine, sympathomimetic amines, alcohol, cheese. ete leading to acute hypertension and possibly subarachnoid bleeding.	Fnr single overdose apply general supportive measures. For acute hypertensive reaction, give phentolamine, or chlopromazine, or tolazoline-
MORPHINE AND OTHER OPIATES Progressive loss of consciousness with muscular flaccidity, depressed respiration "pinpoint" pupils, feeble pulse and fall in blood pressure.	If swallowedg gastric lavage. Oxygen and respiratory support. Naloxone 0.4 to 0.8 mgm, repeated intravenously until action of morphine etc. no longer persists.
ORGANO - CHLORINE INSECTICIDES Aldrin, D. D. T, Lindane, Chlordane, etc. Apprehension, excitement, tremors, convulsions.	If swallowed gastric lavage. Control convulsions (diazepam). General supportive measures.
ORGANOPHOSPHOROUS PESTICIDES Malathion, Parathion, etc. Anorexia, nausea, vomiting, abdominal colic, cold sweating. salivation, ataxia, anxiety, muscle twitching, diarrhoea, urinary retention, bronchoconstriction and pulmonary congestion and respiratory failure.	Complete rest. Ensure clear airway and maintain respiration. Give atropine 2 mgm by injection and repeat at 5 to 10 minute intervals until full atropinisation. Within 12 hours of onset of symptoms, pralidoxime, 1 G in 5 ml by injection, repeated as required.

PARACETAMOL+ After symptomless interval, nausea, vomiting, right hypochondrial pain, enlarged tender liver, hapatic failure.	Gastric lavage, 5% dextrose infusion (not saline). Check blood levels within 8–10 hours of ingestion give specific antidote, e. g. methionine or N-acetylcysteine.
PARAFFIN Burning sensation is mouth and throat. Choking and coughing. Risk of bronchial aspiration and bronchopneumonia. (Not intrinsically toxic)	Avoid gastric lavage and emesis. Check lungs radiologically and treat bronchopneumonia.
PARAQUAT It swallowed bitter taste in mouth with some inflammation of pharyngeal, oesophageal and gastric mucosa. After an interval of some days rapidly progressive proliferative bronchiolitis, with myocardial and renal damage. Large, acute doses give rice to multi-organ failure.	Urgent gastric lavage, followed by mixture of Fuller's earth and magnesium sulphate by mouth. Maintain high fluid output. Early haemodialysis, or haemoperfusion, may be to advantage. In severe cases, treatment is rarely successful.
PETHIDINE — See Morphine.	
PETROL — See Paraffin.	
PHENOL Carbolic aeid, Creosote, Cresol. Lysol Phenolic disinfectants, etc. Burning of lips, mouth and throat, pharyngeal and epigastric pain, pallor, shock, coloration of urinc. Gastric perforation. mny occur.	Discourage vomiting. Very cautious gastric lavage, followed by demulcents. General supportive measures. Surgery, if perforation occurs.
PHENOTHIAZINES + Drowsiness, slurring of speech, stupor with hypothermia, hypotension, hypotonia. Tachycardia and arrythmia.	Gastric lavage, general supportive measures. Oxygen, infusion of β - Adrenergic blocking agents for arrythmia.

QUININE SALTS Cinchona; Quinidine salts Headache, tinnitus, dizziness, deafness, ataxia, nausea, vomiting and cardiac arrythmias. Visual disturbances and possibly blindness	Gastric lavage and followed by sodium sulphate and liberal fluid intake. Promote diuresis. Maintain respiration.
SALICYLATES Nausea, vomiting, tinnitus, ataxia, hyperpnoea, stupor, respiratory depression. Electrolyte imbalance.	Gastric lavage and forced alkaline diuresis. Correction of electrolyte imbalance. In severe cases, haemoperfusion or haemodialysis.
SILVER SALTS. Photographic developers Blackening of oral mucosa. Pain in mouth and throat. Vomiting, diarrhoea, collapse, anuria.	Give liberal 1% soln of sodium chloride by mouth to precipitate silver. Remove by gastric lavage. Promote fluid input and output. General supportive measures.
STRYCHNINE Nux vomica Rapid onset of exaggerated reflexes and tetanuslike extensor spasms of arms and legs, opisthotonus and risus sardonicus. Full retention of consciousness.	Absolute quiet, with protection from external stimuli Sedation or general anaesthesia, to control convulsions. Then gastric lavage with 2 % tannic acid. Maintain respiration. General supportive measures.
TURPENTINE OIL (not turpentine substitute) Nausea, vomiting, colic, diarrhoea, excitement, delirium, dysuria, haematuria, albuminuria, convulsions, respiratory depression, bronchopneumonia and coma.	Gastric lavage. Control of convulsions General supportive measures.

V. K

Selfpoisoning — a short review with special reference to Jaffna

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Selfpoisoning is a world wide problem which imposes an unbearable burden on the resources of many national health services. Kessel [1] (1965) estimated that more than one in every thousand adult population of Edinburgh was admitted for selfpoisoning every year. Since then reports indicate a further increase in developed countries (Wexler el al 1978[2]). Dissanayake and De Silva[3] (1974) reported that 857 patients were admitted to General Hospital Colombo for selfpoisoning during 1972. Ganesvaran et al [4] (1983) analysed 174 case records of selfpoisoned surviors admitted to General Hospital, Jaffna for the year 1982 The total number of admissions for self poisoning to the General Hospital, Jaffna for the year 1984 is 299.

Nature of the poison varies not only from country to country but also from time to time in the same country. Kessel [1] (1965) showed that 90 % of patients admitted for selfpoisoning used drugs. McClure [5] (1984) confirmed that suicide by solid or liquid poisons continued to be the most common method during 1975 - 1980, though an increase in the use of motor vehicle exhaust fames for self-poisoning was observed in males. Coal gas which was the chief non drug poison in 1960 ceased to be a problem with the detoxication of domestic gas. With the rapid expansion of the National Health Service in the united Kingdom, selfpoisoning by drugs replaced non drug poisons such as corrosive agents.

In Sri Lanka, corrosive agents were replaced in the sixties by another group of non drug poisons, namely agrochemicals and insecticides of organophosphorus. Ganeswaran et al [4] (1983) showed that they formed 58 % of all cases of parasuicide by selfpoisoning in Jaffna. Other non drug poisons like kerosene oil amounted to 20 %. Seeds of Thebetia peruviana which causes digoxin like toxicity emerged as a popular poison in 1984 and formed 18% of a total of 299 admissions for self poisoning. Agrochemicals still remain the most popular agent of selfpoisoning. It is interesting to note that increasing use of agrochemicals for selfpoisoning has been reported in Fiji [6] (Haynes 1984).

It is of utmost importance to identify amongst the parasuicides, those who would ultimately kill themselves. Many approaches have been made, and one, is to estimate the intention of the act in the current episode by indirect means. Scales designed by Beck et al[7] (1974) and Pierce[8] (1981) pay direct attention to the circumstances surrounding the self injury as these can be confirmed objectively. Intention scale by Pierce has three components, namely circumstances, self report,

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and assessed medical risk. The circumstances relate to self isolation of patient before the act, timing of the act so that interference is least likely, precautions against discovery, acting to gain help during or after the act, final act of anticipation of death (eg. writing of last will), and suicide note. Self report includes patients statement of lethality, stated intent, premeditation, and reaction to the act. (whether glad or unhappy about recovery). The risk is assessed as predictable outcome in terms of lethality of the act and availability of medical treatment. A point scale from 0 — 2 is available for each item.

A behaviour of a patient is a balance or compromise between self destructive and self preservative wishes A strong wish to die may obscure selfpreservative wishes or a balance of the two wishes may result in an ambivalent attempt. A large proportion carry out the act in the belief that they were comparatively safe, disclosing what they have done either verbally or nonverbally (eg. vomiting in front of relatives) in good time to ensure their safety. Their motive was something other than suicide.

Suicide is strongly linked to affective disorder Beck [9, 10] (1963, 1967) Melges and Bowlby[11] (1969) showed hopelessness to be a core characteristic of depression. Hopelessness has been implicated in suicide. Beck[12] et al (1974) constructed a scale to measure hopelessness. They showed that depressed patients have unrealistically negative attitude towards the future and seriousness of suicidal intent is more highly correlated with negative expectancies than with depression. It is claimed that statistical association between suicidal intent and depression is an artifact resulting from a joint attachment to a third variable, namely hopelessness (Minkoff et al[13] (1973). It would be useful to clinically assess the feeling of hopelessness in all cases of selfpoisoning.

Suicidal risk is a composite measure which includes suicidal intent as reflected in individual's overt behaviour and self report with indices such as age, sex, social class, marital status, presence of physical or mental illness and other correlates of suicide. Based on the data available, attempts have been made to assess suicidal risk in patient population. The success of these predictions have come under careful examination.

Alex D Pokorny [14] (1983) made a prospective study to identify persons who would subseqnently commit or attempt suicide. The sample consisted of 4800 consecutive admissions of a Veterans administration hospital in U. S. A. They were examined and rated on a wide range of instruments and measures including most of those previously reported as predictive of suicide. It was found that each trial missed many cases of suicide ie. sensitivity of the test was low and also the trials had many false positives - ie. specificity was also low to be workable in individual cases. The suicide rate for Porkorny's sample was 286/100,000/yr. This rate is apparently too low for the sensitivity and specificity of the available tests to have a high percentage of prediction. Further, suicidal impulse varies from time to time and a single test at a point of time is unlikely to have high levels of prediction. However in practice the psychiatrist is involved in predictions during a much shorter period at times of suicidal crisis. This work of the Psychiatrist as pointed

out by Porkorny cannot be subject to research or investigation as it would not be ethical to withhold treatment or steps to ensure safety in the plea of conducting a research study. Suicide prevented at such close quarters will never be known with certainity.

In Jaffna, selfpoisoning has been the most popular method of suicide. In a total of 115 suicides 99 subjects (86%) died of selfpoisoning in 1982 88 of them (76%) used agrochemicals — (Ganesvaran et al [15] 1984) This raises the question of prevention by stricter laws controlling the sale and distribution of these substances. Stengel (1964) [16] argued that if one method is eliminated the fall in rate would be temporary and other methods would take its place. Burvill's [17] (1980) finding in Australia and McClure's[15] (1984) observation in U. K. recently of an increase in the use of other gases by males seems to support this view. Kreitman [18] (1976) attributes the fall of suicide rate since 1960 in U. K. to the increased use of natural gas. In Sri Lanka a stricter control of the sale and distribution of dangerous agrochemicals is recommended.

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Homicidal and Accidental Poisoning +

Saravanapavananthan N.*

Poisoning is a small man's weapon not only because small men are not given to violence, but also because they often suffer from a sense of inferiority. Further the prolonged action of the poison gives them a sense of power. They can sit back like gods and see it work. This may be true of Crippen, Armstrong, Pritchard, Radford and the Seddons.

Of the all the murderers, poisoners are the worst. Poisoning cases are the most difficult cases to tackle. Usually the poisoner plans elaborately and studies the properties of the poison before he administers it to his victim, which in some cases are done over a period of time. It is comforting to note that they rarely succeed in their attempts. Many of the poisons produce symptoms, after an interval giving enough time for the criminal to cover his track. Sometimes the symptoms resemble natural illness, which makes diagnosis, still more difficult. "A perfect poison" is one that is tasteless and odourless and is easily soluble in water. Very few poisons possess all these properties. Due to the great advances made by Toxicological Chemistry, the criminal poisoners have little hope of escaping detection today.

Table 1

Case	Relationship to the Accused	Homicidal Agent Identified at Postmortem Examination
Rex. V. Crippon	Wife	Hyoscine
Rex. V. Armstrong	Wife	Arsenic
Rex. V. Seddon	Miss Barrow (friend)	Arsenic
Radford	Wife	Arsenic
Rex. V. Greenwood	Wife	Arsenic
Rex. V. Maybrick	Husband	Arsenic
Rex. V. Wilson	2nd Husband	Phosphorus
	3rd Husband	Phosphorus
Reg. V. Merrifield	Mrs Ricketts (friend)	Phosphorus
Reg. V. Barlow	Wife	Insulin
Reg. V. Chapman	1st wife	Antimony
	2nd wife	Antimony
	3rd wife	Antimony
Rex. V. Pritchard	Wife	Antimony

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+ *Section on Homicidal Poisoning appeared in Jaffna Medical Journal, 1980, 15, 2, 44 — 49.*

The incidence of homicidal poisoning was high during the latter part of the last century and early part of this century, but the number has appreciably decreased due to the combined effect of the restrictions exercised by the law enforcing authorities in the acquisition of drugs and poisons on the one hand and to the advancement in the techniques used in the detection of poisons and drugs in the human tissues on the other. In most cases the victim is usually the spouse and this is indicated in Table (I).

Table (II) contains the list of common homicidal agents.

Table II

POISON
Arsenic
Antimony
Thallium
Cyanide
Phosphorus
Barbiturate
Chloroform
Hyoscine
Strychnine
Insulin

Arsenic:

Despite the severe restrictions placed on the acquisition of arsenic and the ease with which it can be detected in human tissues, it still enjoys a prominent place as a homicidal agent. Notable trials for murder by arsenic poisoning are those of Mary Blandy, Mandeleine Smith, Mrs. Maybrick, the Seddons, Armstrong and Greenwood.

Arsenic is a gastro-intestinal irritant; vomiting is almost invariable, but seldom delayed for more than twelve hours, and is associated with a sense of constriction in the throat. Abdominal pain and diarrhoea occur within five to twentyfour hours and the stools resemble 'rice water'. Some, complain of muscle cramps specially in the calves. In fatal cases, the patient rapidly goes into a state of shock and peripheral vascular collapse. Pigmentation of the skin, hyperkeratosis, peripheral neuritis, mental instability and delirium are features of chronic arsenic poisoning. Lethal dose is two to five grains. Arsenious oxide, being tasteless and odourless, can be administered without arousing the suspicion of the intended victim. Arsenious oxide is sparingly soluble in cold water and hence Mandeleine Smith used hot chocolate to dissolve the arsenic. Porridge or gruel is also a good vehicle as it can hold considerable amount of poison without exciting suspicion. Potassium arsenite is another compound of arsenic which is commonly used as a homicidal agent. Mrs. Maybriek and the Seddons used potassium arsenite from fly papers.

Simpson[1] in 1949 reported a case of arsenical poisoning that occurred under the very nose of the doctors and the staff and the poisoner nearly got away with it. Gordon Radford, a laboratory technician poisoned his ailing wife with arsenic, while she was a patient in a sanatorium. He carefully intoduced some arsenic in pies which he sent to his wife. When Mrs. Radford became sick after eating one of the pies, she remarked to one of her friends who was regularly visiting her, about her husband's extraordinary kindness since of late. She however requested her to get one of the pies examined for poison. Her friend sent the pies to the Medical Superintendent with a covering letter. The letter went into the secretary's "in-tray" and the parcel was placed on the Medical

Superintendent's table. It was a Saturday after-noon and when he saw a nice pie, thinking it was from a friend, took it home for lunch. Before he could finish eating it, he became sick. He carefully retained the remaining part of the pie and on Monday morning when he read the letter sent by Mrs. Radford, he realised why it had been sent to him. The pie was analysed and found to contain 3 grains of potassium arsenite. Mrs. Radford died 3 days after eating the pie.

Sectional analysis of her hair revealed that she had been systematically poisoned over a period of three months. Radford when he realised that he was being suspected, poisoned himself by taking prussic acid.

Phosphorus :

Phosphorus is not an ideal homicidal agent. It has an unpleasent odour and a disagreeable taste, but these however do not prevent it from being used as a homicidal agent. It produces liver damage and mimics infective hepatitis of viral origin. Poisoners use various vehicles to mask its taste and odour. In the Marifield case it was believed that the vehicle was rum and sugar or black-currant jam. In Wilson's case[2] Mary Elizabeth Wilson was found guilty of the murder of her second husband, Oliver James Leonard and her third husband George Lorence Wilson by administering yellow phosporus in the form of beetle poison in cough mixture.

Thallium has many but fortunately not all the properties of a "perfect poison". Salts of thallium are easily soluble in water and are odourless, tasteless and colourless. The symptoms occur after an interval of 12 hours giving enough time for the criminal to cover his tracks. When a patient presents with polyneuritis and alopecia, one should think of thallium poisoning but unfortunately these symptoms may be delayed as long as 10 days.

Antimony:

If not for the notable cases. Rex. v. Chapman and Rex. v. Pritchard the amount of space devoted to antimony poisoning in forensic testbooks would be very small indeed. Chronic rather than acute poisoning is common as it is usually administered in small doses at frequent intervals rather than in one large dose. The clinical picture superficially resembles natural illness. The outstanding features of antimony poisoning are nausea, vomiting, thirst, diarrhoea, and muscular cramps. The case of Chapman[3] is worth mentioning as it is historic and instructive. Even the most skilled medical personnel of Guys Hospital at that time were deceived. In July, 1902 a woman named Maud Marsh was admitted to Guy's Hospital with a history of recurring bouts of abdominal pain and diarrhoea. A diagnosis of tuberculous peritonitis was made and she was treated for almost a month in Guy's Hospital. She remained well for about two months after which, symptoms recurred and after 12 days of treatment by a local doctor, she died. A private autopsy was performed and antimony was identified in the material submitted for analysis.

Sir Thomas Stevenson repeated the analysis on the orders of the coroner and antimony was isolated in all parts submitted for analysis. Maud Marsh was living with a person named Chapman prior to her death. Investigations revealed that Chapman had purchased an ounce of tartar emetic five years back at Hastings. Fur-

ther investigations revealed that sometime after this purchase. his first wife Isabella Spinks had died and 2 years later his second wife Bessie Taylor met the same fate. The cause of death in the case of Isabella was put down to tuberculosis while Bessie Taylor was reported to have died of intestinal obstruction after the brief illness of nausea, vomiting and abdominal pain. Large amounts of antimony were found in each of the two bodies. Chapman was found guilty and executed.

Cyanide:

In cyanide poisoning death comes on with dramatic suddenness. Muscular twitching, vomiting and frothing at the mouth may precede unconsciousness. Cyanide is a rare choice for homicide. A doctor[4] once administered a lethal dose of prussic acid to his 5 years old daughter and then took his own life. A research chemist[5] murdered his mother who was suffering from eancer by giving her potassium cyanide and thereafter he too ended his life by taking same. In Malaya according to Gimlette[6] potassium cyanide was used with honey for the purpose of committing murder. The under surface of the knife used for cutting a water melon was smeared with the poison and it was so arranged that the intended victim got the lower portion smeared with the poison while the assailant ate the upper half.

Strychnine :

Strychnine is rarely prescribed but its use for poisoning continues. It has been described as the bitterest substance known, in spite of which it has been used as an instrument of murder. There are many ways of masking the taste of strychnine. It is possible to administer strychnine in alcohol without arousing the suspicion of the victim. It can be introduced into foods which have bitter taste, It can be given as pills Strychnine stimulates the higher centres and produces a picture clinically indistinguishable from that of tetanus In the famous case of Rex. V. Palmer [7] strychnine pills were given to Cook by Palmer. Thereafter Palmer persuaded 80 year old Dr. Bamford to issue a medical certificate for "apoplexy" but the intervention of Cook's father resulted in an inquest and autopsy. The autopsy was performed by a local doctor and an undergraduate. Palmer who was allowed to be present at the autopsy deliberately jostled the operator at the very moment he was dissecting the stomach causing thereby the spilling of some of its contents. In spite of all these flaws in the medico - legal procedures, Palmer was found guilty by the Jury.

Hypoglycaemic agents.

Symptoms of hyper insulinism begin when the blood sugar falls to the region of 50-60 mg/100ml. Initial symptoms of hypoglycaemia are hunger, fainting, pallor, headache and numbness of face and extremities. Further lowering of the blood sugar produces dysarthria, diplopia, muscular twitching, convulsions, coma and death. Fortunately, murder with hypoglycaemic agents is rare. The first recorded case of homicidal poisoning by insulin was that of Regina. v. Barlow. Details of this case were reported by Birkinshaw at-al in 1958 [8]. Barlow a state registered nurse married the deceased woman in 1956. In May, 1957 he lost his job when his wife was also expecting a baby. On the 3rd of May, 1957, she was found dead in the bath. When the doctor arrived at 11 - 30 p. m. he saw the deceased lying on her right in the bath.

The water had been drained and the body was warm and wet. According to Barlow she took off her pyjama and went for a bath at about 10-00 p. m. and in the meantime he had dozed off. When he woke up at 11-20 p. m. he found her lying face downwards in the bath. He pulled the plug, drained the bath and gave her artificial respiration. Surprisingly the sleeves of Barlow's pyjama were not wet while the pyjama of the deceased was soaked in sweat. This aroused the suspicions of the police. Post-mortem confirmed that death was due to drowning. As no poison could be detected in the internal organs of the deceased, the body was re-examined on May 8th and four puncture marks were detected on the buttocks. Barlow suggested- from the witness box that his wife might have injected insulin herself. It is not easy for one to inject into both the buttocks. The layout of the house was such that no third person could have entered the house at that time without the knowledge of Barlow. Barlow had access to insulin and he had knowledge about its ill effects. When he gave instructions to his colleagues he had commented that insulin is the best drug available to commit a perfect murder. What precisely happened is known only to Barlow. The probabilities are that after she was rendered unconscious by injecting insulin she was carried into the bath and the scene was so arranged to simulate accidental drowning. Barlow was found guilty of murder and sentenced to life imprisonment.

Barbiturates

Toxic symptoms may occur following a single dose. Death occurs due to depression or respiratory and circulatory functions. Barbiturates are used widely in the treatment of anxiety states, sleeplessness and other psychiatric conditions. The possibility of committing murder by administering barbiturates must always be kept in mind. In the Armstrong case[9], Terrance Armstrong aged 6 months, son of a naval berth attendant died suddenly. The doctor in attendance could not give a death certificate and an autopsy was ordered. At the autopsy there was nothing to account for the death but a red "berry" was discovered near the right tonsil and red "berries" were found in the stomach. The stomach contents were analysed and 1/3 grain of seconal was extracted. Futher 1/50th grain of seconal was extracted from the vomit stained pillows. These "berries" were actually parts of seconal capsule. A verdict of death due to seconal poisoning was returned. As no other evidence was forthcoming John and Janet Armstrong, the father and mother of the child remained free for about an year. During this period the Armstrongs quarelled and drifted. Janet Armstrong applied for separation and maintenance but her application was disallowed, and she walked out of the courts in tears. Inspector Gates who investigated Terrance Armstrong's death was present in Court at that time and asked Janet Armstrong whether she had anything to tell the police. She hesiated for a moment and then volunteered a statement whice resulted in the conviction of John Armstrong.

Other Agents.

Aconite

Aconite is one of the most deadly poisons known. It produces a tingling sensation, numbness of the tongue and mouth followed by constriction of the throat and difficulty in swallowing. The

case of Dr. Lamson[10] is the only known instance of homicidal use of aconite in England.

Atropine.

Atropine is rarely used as a homicidal agent even by persons who have aecess to the drug. Withaus[11] cited a case where a nurse is said to have added belladona to milk drunk by the senior surgeon of her hospital. She was acquitted owing to inadequate evidence to connect her with its administration.

Hyoscine.

Hyoscine hydrobromide was used by Dr. Crippen[12] to kill his wife. Dr. Crippen, like other poisoners, Palmer, Armstrong and Smethhurst was a very small man, was known to be gentle and well mannered He was dominated and tyranized by his wife He endured her domination for years. When his wife, who was tired of him and knew about his intimacy with Ethel-le-Neve threatened to leave him, there was a crisis in the family. Dangerous thoughts passed through Crippen's mind before he ordered five grains of Hyoscine hydrobromidc for the murder of his wife. He lied foolishly about her disappearence and he was eaught when he attempted to escape the law by ocean liner to America with his girl friend. Willcox isolated 2/5th of a grain of hyoscine hydrobromide from the viscera about 6 months after burial, Thanks to Sir. Bernard Spilsbury whose evidence regarding the operation scar on apiece of skin from the lower part of abdomen went a long way to establish identity.

Chloroform

Chloroform if used inadvertently can cause death due to ventrieular fibrillation. Adelaide Bartlett[13] was charged for the murder of her husband by pouring liquid chloroform down his throat while he was asleep. She was acquitted owing to insufficient evidence to connect her with the erime.

In conclusion most of the poisons used for homicidal purposes produce symptoms similar to those of many known natural diseases where even thc most skilled medical personnel are apt to be misled, One should therefore look for poisoning especially when the diagnosis of a natural disease is in doubt.

ACCIDENTAL POISONING

Accidental poisoning is common, particularly among pre-school children and the elderly. The substances responsible for poisoning in children are iron, lead, barbiturates, salicylates, atropine, disinfectants and others such as moth balls and kerosene oil found in many house holds.

The exploratory toddlers are often attracted by the sugar coated iron tablets prescribed for an adult member and may chew these mistaking them for sweets. Initially symptoms may be related to gastro intestinal irritation, lasting about 6 hours followed by a latent period lasting about 24 hours. Symptoms may relapse due to the development of acidosis or the toxic effect of free iron in the circulation. Death may occur in either the 1st or 3rd stage.

Children are particularly susceptible to lead. Accidental poisoning in children has resulted from chewing lead toys, crayons and from biting cots painted with lead paints. The initial symptoms are non specific. Pallor of the skin is striking. Disturbances of behaviour, irritability,

restlessness and convulsions which precede encephalopathy are usually seen in the majority of cases.

Eye drops and belladona liniment left within the reach of small children has caused poisoning in a number of cases.

Children often like to experiment with tastes and to put things inside their mouths. Meclean et al[14] reported a case where a child aged 18 months ate Roach paste on potato set out under the refrigerater in the kitchen.

Poisoning with asprin may occur from accidental consumption of the tablets, but it may also occur from the excessive administration by the parents to children who are suffering from some illness. Children are more likely to die from Salicylate poisoning than are adults. Accidental poisoning is often preventable and parents should store drugs in safe places, out of the reach of children. In some countries asprin and paracetamol are sold or supplied in child resistant containers containing not more than 24 tablets. Child proof containers with special caps are now available.

Accidental poisoning due to kerosene oil is common in children especially among those living in rural areas, when it is drunk by mistake for water. The increase in the incidence is probably due to the increased use of kerosene cookers and lamps in these areas. Its pink colour and the tendency to store it in lemonade and jam bottles may attract children. Kerosene is a narcotic and may cause listlessness, drowsiness, somnolence and in severe cases loss of conciousness and convulsions. The main action of kerosene is upon the respiratory tract.

Naphthalene balls are used for the purpose of preventing damage to cloth by moths. The principal danger of this lies with infants. Poisoning occurs either by inhalation, absorption through the skin or ingestion. Valaes and Fessas [15] in 1963 reported an outbreak where 21 infants developed acute haemelytic anaemia as a consequence of contact with blankets or diapers treated with naphthalene.

Many common plants are poisonous; and children are particularly vulnerable. They may be attracted by the berries of certain plants like: Datura alba (Hyoscine), and Thevitia Peruviana (Thevitin). The clinical features of Datura include suppression of saliva, tachycardia, flushing of the face, pyrexia, delirium and disturbances of vision. Thevitia produces symptoms similar to digoxin poisoning.

The effects of old age with its attendant infirmity or bodily disease are probably responsible for the increase in the incidence of accidental poisoning among the elderly. Sometimes more than one factor eg: poor vision, deafness, no sense of smell may contribute to an accident particularly when poisons are stored in ordinary inadequately labelled bottles. A man aged 73 mistook a bottle of ammonia for his medicine and was fatally poisoned. Similarly a bottle of Sulphuric acid may be mistaken for glycerine. A man who is deaf and has no sense of smell may not notice a leaking gas tap and others who suffer from cerebral athero sclerosis may forget to close a tap.

Sometimes poisoning may occur while being under the influence of drink or drugs. Recently a case came under my notice where a person who was under the influence of alcohol drank a diluted

solution of Parathion, placed in a cup mistaking it for tea. Happily he was saved by the timely arrival of his daughter.

Outbreaks of poisoning have occurred from time to time as a result of accidental contamination of food or drink with poisonous substances. Sweets adulterated with arsenious oxide in 1858 caused poisoning in seventy persons of whom seventeen died. Increased use of lead pipes to supply water has resulted in outbreaks of lead poisoning Alcohol prepared or stored in vessels with a lead glaze has also resulted in lead poisoning. Drinking water may be contaminated by arsenic when placed in empty tins of weed killer. Thirteen women engaged in the making of bricks in South Africa were poisoned when they quenched their thirst from one of these tins Similarly food or drink prepared in vessels plated with cadmium has resulted in poisoning. In 1973 an outbreak of paraquat poisoning occurred in the Colombo district following consumption of dhal contaminated by paraquat during shipment

Another group of accidents arise out of well intended but careless treatment. These therapeutic accidents arise either by over dosage, errors in administration or illegible prescribing. An illegible prescription led to the supply of Potassium chlorate in error for potassium chloride. The patient after taking it developed haemolytic anaemia. A woman, who was under treatment for mental confusion died following an injection of 5 ml of insulin instead of 5 units. The nurse in this instance misheard the instructions and administered 5ml. Over dosage with insulin may occur in diabetics; symptoms of hypoglycaemia devlop when the blood sugar falls to about 50-60 mg/100 ml

Accidental poisoning is a hazard in certain industrial processes. The increase in production and utilization of lead during the past two hundred years has led to chronic poisoning in those engaged in coach building, ship breaking and manufacture of lead paints.

Industrial processes continue to be the principal source of mercury poisoning. Formerly manufacture of felt hats exposed the workers to serious risk. The tremor which developed in the workers of this industry was known as 'hatters shakes' and the mental change caused by chronic poisoning led to the popular expression 'mad as a hatter' Mercury poisoning continues to be seen in those engaged in the manufacture of barometer, thermometers, radio valves etc.

Arsine poisoning is encountered only in industrry. This is known to cause massive haemolysis and sudden death.

Cyanides are reputed to be lightening killers. Hydrogen cyanide gas is used to fumigate ships and green houses. Pockets of gas may remain in ships which had been inadequately ventilated. Therefore ships should be properly ventilated before re-entry to avoid fatalities.

Accidental leakage of toxic substances from factories can be dangerous to the public. The recent Bhopal tragedy is a classic example of this menace.

With the increase in use of insecticides in agriculture and control of household pests there is a notable increase in the incidence of accidental poisoning among farmers. Disregard for normal precautionary measures appears to be the most common cause of poisoning Spraying insecticides without wearing masks and

against the wind has caused poisoning in many instances. Accidental intoxication following skin contamination has been reported. Consuming grains and vegetables that have been treated with insecticides has also resulted in poisoning. Vethanayagam [16] (1962) treated 51 cases of insecticides poisoning in the General Hospital, Jaffna during the period 1st January 1959 to 30th June 1960. The poisoning was due to eating, chewing and smoking with unwashed hands during or after spraying and also to inhalation of the substance during spraying.

Accidental poisoning particularly in children is preventable. Parents should be persuaded to store drugs in safe places in properly labelled containers out of the reach of children.

The existing regulations regarding the sale, supply and prescribing of drugs and poisons should be strictly enforced.

Measures for the prevention of poisoning in industries include proper technological design; through maintenance of operating equipment and effective monitoring and control of the work environment. Workers should receive periodic medical examinations to detect physical or emotional responses to the occupational environment.

The toxcity of many pesticides is such that their careless use by the public is bound to result in many people being affected, sometimes fatally. It is therefore very necessary that the public should have access to only slightly hazardous pesticides. The sale of pesticides having moderate or high hazard should be limited to these who are trained and licenced. The labels should carry certain essential information regarding the directions for use, the precautions to be taken during use, the protective equipment that should be worn, the symptoms of poisoning and first-aid treatment for suspected poisoning. Mixing is the most hazardous phase in the use of pesticides as the workers are exposed to a very high concentration of the pesticide during this process. Therefore it is essential that special precautions should be taken during the mixing and application of pesticides like, wearing masks and personal protective equipment designed to prevent inhalation and skin contamination.

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Accidental Heroin Poisoning in an Eight Month Old Baby

R. Ganeshamoorthy, F.F.A.R.C.S. (Eng) *

A female child, eight months old and weighing eight kilogrammes was admitted to The Teaching General Hospital, Jaffna at 8-00 p m one day in the early part of this year (1985). The mother stated, that the child, who was playing as usual at 5-00 p. m, became drowsy around 6-00 p. m. She also said, that she noticed a black sticky substance in one hand of the child. The father stated, that he suspected this substance to be a form of heroin and rushed the child to a private nursing home, where he was advised to take the child to The Teaching General Hospital.

On admission, the child was drowsy, not cyanosed, had warm extremities, facial twitching, constricted pupils, a pulse rate of 140 per minute, a respiratory rate of 24 per minute and crepitations in the lungs. There had been no vomiting at all. The child was given a stomach wash immediately and a five percent dextrose infusion was started. Nalorphine I mg, ampicillin & cloxacillin were given intravenously and the child was transferred to the intensive care unit. One hour later, the child became alert and cried. The pupils were found to be of normal size, the heart rate was 180 per minute and the systolic blood pressure was 60 mm Hg. Three hours later, the child once again, became drowsy with constricted pupils, her heart rate rose to 192 per minute and there was lot of secretions in her pharynx. Administration of another I mg of nalorphine intravenously resulted in improvement in the clinical state. The child became alert, cried well and accepted feeds, the following morning. Investigations done on the second day, showed a haemoglobin of 9.5 gm %, a total white cell count of 12,600 per cu mm and a differential count of 20 % neutrophils, 73 % lymphocytes, 5 % eosinophils and 2 % monocytes. The child was discharged from the hospital on the third day.

Diagnosis of heroin poisoning was made from the specific history given by the parents, failing which the child would have been submitted to invasive investigations and undue delay in the adminstration of appropriate therapy.

Discussion :

Heroin is diamorphine or diacetyl morphine[1]. It is an opioid; acts as an agonist, interacts with stereo-specific and saturable receptors in the brain and other tissues. Miosis, which was seen in this child is pathognomonic of heroin intake. Heroin is a potent depressor of cough reflex and this may have been the cause of retention of secretions in the lunge of this child. Heroin is largely excreted in the urine as free and conjugated morphine. Naloxone is the drug of choice in the treatment of heroin poisoning. The child was treated with nalorphine

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because naloxone was not available at rhat time. The fact that the action of nalorphine lasts for one to four hours, explains the manifestation of repoisoning in this child, four hours after the first dose. One has to give nalorphine carefully because of its agonistic action.

Heroin is said to be available in Jaffna, as a whitish or brownish powder. The colour turns black, when it is stored in polythene containers in the ground. This explains the blackish sticky substance in the hand of the child.

Though 40% of the poisoning in children under five years is due to drugs[2], there had not been a single case report of an accidental heroin poisoning in an eight monthe old child, published in the medical literature, that is available in this country.

This case illustrates, that heroin is available in this area and uuless drastic measures are taken, this area too will be saddled with the problem of heroin addiction.

Acknowledgement:

I thank Dr. S. Vamadevan, Senior Lecturer in Paediatrics of the University of Jaffna and the Director of Teaching General Hospital, Jaffna, for granting permission to publish.

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News and Notes

The University has graciously consented to allot a portion of the land opposite the hospital, to the J. M. A for the furtherance of Medical Education of the students and doctors. A building estimated to cost Ten Lakhs of Rupees is being planned. Donations from wellwishers are welcome and should be forwarded to the Treasurer, J. M. A.

The Post Graduate course for M. D, is well attended.

A Paediatric Update was held on 27 - 10 - 85 (sponsored by Messrs. Darley Butler and Co. Ltd). It was well appreciated, particularly by the General Practitioners, who have requested more sessions.

The third edition of "Health Hints" is out, and copies are being sold in the wards.

The incubation period (four years) is over and symptoms are begining to appear of a new BLOCK in the EXISTING hospital. "I shall build a NEW HOSPITAL during my tenure of office", appears to be a distant cry!. Incidentally, the first "National" Film produced in Ceylon was "Broken Promise".

The following have been successful at the respective examinations conducted by the P. G. I. M.;

Dr. N. Sivarajah	—	M. D (Community Medicine)
Dr. D. J. Somasundaram	—	M. D (Psychiatry)
Dr. S. Sriskandan	—	M. S (Obst & Gyn)
Dr. K Ranjithayalan	—	M. D (Medicine)
Dr. S. Vasanthakumar	—	M. D (Medicine) Part I.
Dr. K. Ambalavanar	—	M. S Part I.

The Annual Sessions 85/86, and V. T. Pasupati Momorial Lecture will be held on 16th, 17th and 18th of May '86. Papers (15 mins duration) should be submitted (to Academic Committee, J. M. J) before the 15th April '86 and Script of Memorial lecture before the 31st of March '86.

The ethnic disturbances have affected the Membership, the Hospital, and the Patients badly. Some idea may be obtained from the following:

(a) The following members have left the Island and the Services in the respective fields have been affected.

Dr. V. Sivagnanavel, Orthopaedic Surgeon,
Dr. V. Kunanandan, Neurosurgeon,
Dr. R. John, Physician,
Dr. S. Yoganathan, Dermatologist,
Dr. C. Gnananandan, E. N. T. Surgeon,
Dr. S. Nirmalanandan, Eye Surgeon
Dr. M. Vetpillai, Senior Lecturer in Surgery
Dr. S. Subramaniam, J. M. O.

Many Junior doctors also have ieft. Whereas the Cadre of M. 00 for the O. P. D is 23, there are only 8 M. 00 new.

(b) The number of admissions has decreased eonsiderably (not because of better health but because of transport facilities, fear of free movement, etc). Whereas in 1982 there were 54, 358 admissions, in 1983, 1984 and up to Oct !85, there were 50, 435, 43 276 and 36, 591 admissions respectively.

The number admitted in rhe Night durlng 1982, 1983, 1984 and up to Oct '85 were, 9750, 7502, 5160, and 2914 respectively. Many must be dying at

home or getting admitted in the morning, in a worser condition, (night admis ion usually being serious cases).

(c) Malnutritiou and Undernutrition cases have shown an increase : Figures in preparation.

d) Heart ailments have registered an increase : Figures in preparation.

(e) Psychiatric ailments (mainly depressions, grief reactions) have shown an increase—from 725 clinic consultation in 1982 to 983, 884, and 796 in '83, '84 and up to Nov. '85, respeetively.

(f) The Surgical Section has suffered very badly indeed. Non-availability of Anaesthetic gases (transported from Colombo), drugs, dressings, shortage, of staff in Orthopaedics, Neurosurgery, E. N. T, Eye surgery, increased trauma work, poor and at times even non-availability of transport services, have contributed in no small measure. Many Routine operation lists had to be cancelled and some worked out under local anaesthesia.

Year 1984

	Jan	Feb	Mar	Apr	May	June	July	Aug	Sep	Oct	Nov	Dec
Routine Opn Lists Scheduled	54	48	48	60	48	48	60	48	48	60	48	60
—do— cancelled	01	06	04	26	03	01	03	05	01	04	16	49

Year 1985

Routine Opn Lists Scheduled	48	48	48	60	48	48	60	48	60	48	48	
—do— cancelled	24	14	03	09	10	11	02	04	13	06	04	

Worked out under local anaesthesia : 11, 7, 2, 3 in March, April, May and July '84 respectively and 3, 4, 9, 17, 3, 6 in February, March, May, June, September and October '85 respectively.

Trauma from Road Traffic Accidents and Private feuds showed a decline but Gun Shot Injuries alone increased the work load. The figures for those brought ALIVE to OUR hospital are as follows: 106 males and 7 females and children in 1984 and 143 males and 30 females and children in 1985.

If this trend continues, Health for All by 2000 A, D, will be a fantasy.

———

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Indication: Arterial hypertension.
Contraindications: Uncompensated heart-failure, second and third degree AV block, marked bradycardia, obstructive respiratory tract diseases (e.g. bronchial asthma, spastic bronchitis), cor pulmonale, allergic rhinitis, glottic oedema, cardiogenic shock, sulphonamide-hypersensitivity, hepatic coma, therapy-resistant hypokalaemia and renal failure with anuria.
Side-effects: In every hypotensive therapy, individual instances of dizziness, palpitation, outbreaks of sweating or tiredness can occur. In occasional cases, gastro-intestinal complaints such as nausea and vomiting can occur.
Particularly at the beginning of therapy, a rapid lowering of blood pressure can lead to a deterioration in one's ability to concentrate (drivers!)
Dosage: Initial treatment: 1 tablet per day. If the antihypertensive effect is not sufficient, the dose can be increased after 14 days to 2 tablets daily, which can be taken as a single daily dose at breakfast time once good control is established.

Our representative in Sri Lanka:
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Your partner in diagnosis and therapy

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