Hospital Topics

Accidental poisoning in childhood: five year urban population study with 15 year analysis of fatality

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Abstract

Patterns of accidental poisoning in children are changing dramatically. A five year population study (1977-81) was undertaken in urban children from Brisbane (population 1 000 000). A total of 2098 children were poisoned during this period with only one fatality, which represents a dramatic reduction in mortality. Over the past 15 years (1968-82) 13 children have died from accidental poisoning from this population, and two were murdered with drugs. A study of secular trends has indicated that peak incidence occurred in 1979, and the rate has been falling progressively since. The current age corrected rate of poisoning is 393 per 100 000 children per year (0-5 year olds). The rank order of poisons, drugs, and chemicals causing hospital admission and death is: petroleum distillates 13%; antihistamines 9%; benzodiazepines 9%; bleach and detergents 7%; and aspirin 6%. The ratio of fatalities to ingestions requiring hospital admission was calculated to give an index of a practical danger of noxious agents to which children are currently exposed and the rank order is: cardiotoxic drugs, one fatality to 25 ingestions; tricyclic antidepressants, one to 44; sympathomimetic drugs, one to 54; caustic soda, one to 68; aspirin, one fatality to 350 ingestions.

Accidental poisoning of children leading to death has been reduced because patterns of drug prescriptions have changed, packaging of dangerous drugs has been made safer, and substances such as kerosene have been coloured blue.

Introduction

Accidental poisoning in childhood is a world wide problem, and in the past three years has been the subject of major studies in the United Kingdom,^{1 2} Europe,³⁻⁵ the United States of America,^{6 7} India,⁸ and Australasia.⁹ These investigations have shown that patterns of child poisoning are changing rapidly. It is important to document current trends, as poisoning has hitherto been a notable cause of mortality in toddlers¹⁰⁻¹² and

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a major cause of hospital admission for children in this age group.^{13 14}

In some centres there is good evidence that vigorous preventive programmes have helped considerably to reduce mortality from accidental poisoning in childhood.³¹⁴ Nevertheless, the types of pharmaceutical and chemical agents ingested accidentally by children also change with fashions in prescribing¹⁵ and with changes in safety packaging^{16 17}; and, at least for aspirin have produced a shift in the types of poisoning.18 There is some evidence that urban children differ from rural children with respect to both the incidence and severity of accidents in general¹⁹ and to accidental poisoning in particular.¹² In all reported series children between the ages of 2 and 4 years constitute over 90% of the children at risk,²⁰ but age corrected data on risk are not readily available, and sociodemographic profiles of a reported series are usually not specified. The ratio of fatalities to ingestion for important poisons ingested remain unknown.

We undertook to examine these secular trends, to examine changes in the profile of agents currently producing clinical poisoning, to provide estimates of the ratio of fatalities to ingestions, and to provide age specific data on incidence. These can be provided only by prospective studies or by retrospective surveys of defined populations without selection. We therefore describe a population study (five year study of morbidity; 15 year study of mortality) of poisonings in childhood from Brisbane, Australia.

Material and methods

Brisbane is a subtropical western city with a population of 1 401 000 at the midsurvey point (1979),²¹ of whom 115 600 (8%) were preschool children under the age of 5 years. Ninety eight per cent (1 372 980) of the population is white, and 90% (1 260 000) are of central or western European origin. Lifestyle is casual and relaxed, mean winter temperature exceeds 8°C, and toddlers spend a considerable time outdoors.

Fatalities due to poisoning were identified by a perusal of all case files of the Institute of Forensic Pathology, Brisbane, which has responsibility for the investigation of all deaths not resulting from natural causes. Case files for the years 1968-82 were complete and formed the basis of the study of fatalities. Details of age, sex, circumstances of poisoning, and the drug or chemical agent concerned were compiled from individual case records.

The City of Brisbane is served by two paediatric casualty centres (the Royal Children's Hospital, and the Mater Misericordiae Children's Hospital); referral, emergency transport, and admission are centralised to either of these hospitals, and no case of clinical importance occurs outside these two centres. Non-fatal cases were identified by the perusal of admission registers, intensive care unit registers, and hospital records for the five year period 1977-81 inclusive. Details of the type of drug or chemical ingested that had caused hospital admission were recorded in each case. In the case of accidental ingestion of poisons admission policy is conservative, and all potentially serious cases are admitted to hospital for observation.

Results

Fatalities (15 year study)—Thirteen children (six boys, seven girls) died from accidental ingestion of drugs and chemicals during the 15 year period 1968-82 inclusive. Modal age was 2 years (range 11 months-5 years). Two further children were murdered using chemicals. Table I shows the drugs and chemicals causing these fatalities. A dramatic secular trend was found, with eight fatalities in the period 1968-72, four in the period 1973-7, and only one in the period 1978-82.

Survivors (five year study)—A total of 2098 children were admitted to hospital in the five year period 1977-81 inclusive because of accidental poisoning from a variety of causes (table II). During this period there was only one fatality caused by accidental poisoning. Thus there is less than a 0.1% chance of dying as a result of accidental poisoning in childhood. Table III shows the number of poisonings, by calendar year, corrected for demographic trends and expressed as age specific rates (for 0-4 year olds) for children at risk. An increasing

> TABLE I—Agents causing fatalities in accidental poisonings of children in Brisbane, 1968-82

Agent	No of cases
Tricyclic antidepressants Digoxin, quinine	32
Caustic soda Orciprenaline, theophylline	2 2 2
Chloroquine Fluoride Aspirin	1
Oleander fruit	1
Total	13

TABLE 11—No of hospital admissions and fatalities for all forms of childhood poisoning in the city of Brisbane, 1977-81 inclusive

No	
2024	Accidental ingestions:
2023	Survivors
1	Fatalities
12	Spider bites (survivors)
rs) 33	Wasp, bee stings (survivors)
27	Snake bites (survivors)
als 2	Snake bites (survivors) Murder by drugs, chemicals
	the and the area of the area o

TABLE 111—Rates of hospital admissions for accidental poisoning in children in Brisbane, 1977-81, showing sex, secular trends, and age corrected rates of incidence (0-4 years)

Year	Total cases	% Boys	Rate per 100 000 aged under 5
1977	329	61	322
1978	455	52	437
1979	491	55	463
1980	411	61	380
1981	411	55	368
Total	2097	58	393

trend up to 1979 was observed, which appears to have been falling progressively since. Of those poisoned, 58% were boys, but the sex incidence shows no secular trend. Table IV lists the rank order of chemicals, drugs, and poisons accidentally ingested by urban children which led to hospital admission. Drugs remain the biggest problem accounting for over one third of all serious accidental ingestions. There were 25 ingestions of plants (2% of all cases) and eight of oleander, a well recognised hazard in all subtropical and tropical countries.²²

Taking into account the deaths that have occurred, the drugs and chemical agents causing them, and the number of admissions of non-fatal cases resulting from these agents, it is possible to generate a ratio of fatalities to ingestions to give a rank order of the potential danger of agents currently responsible for accidental poisonings (table V). -

TABLE IV—Rank order of 20 most common agents accidentally ingested by children (0-12 years), resulting in hospital admission in Brisbane, 1977-81

Agent	No	%	Agent	No	%
Petroleum distillates	255	13	Tricyclic antidepressants	45	2
Antihistamines	186	9	Phenytoin	40	2
Benzodiazepines (diazepam,			Iron tablets	36	2
etc)	182	9	Sympathomimetics	36	2
Bleach, detergents	142	7	Rodent killer	28	ī
Aspirin, paracetamol	117	6	Fluoride tablets	28	ĩ
Camphor; camph oil;			Cosmetics (including		-
mothballs	117	6	perfumes)	28	1
Barbiturates	97	5	Pet care chemicals (dog		-
Caustic soda	69	3	wash; flea powder)	20	1
Painting chemicals		-	Digoxin	16	ĩ
(turpentine, paint stripper)	61	3	Swimming pool chemicals	12	î
Noxious plants	49	2	Other	457	23

TABLE V—Ratio of fatalities to ingestions of poisonous agents in children aged 0-4 years

Agent	Rates of fatality to ingestion
Digoxin, quinine Tricyclic antidepressants	1 to 25
Tricyclic antidepressants	1 to 44
Sympathomimetics	1 to 54
Caustic soda	1 to 68
Aspirin	1 to 350
Petroleum distillates	1 to > 750

Discussion

If epidemiological and other variables are identified and documented during dramatic secular trends in child accidents much can be learnt about causes and prevention.

In all studies the modal ages for poisoning fatalities are the second and third years of life,¹² ¹³ and this pattern is not showing any secular trend. We think, as do others,⁵ that child directed educational campaigns, though popular in some areas,²³ ²⁴ have little place in prevention. Toddlers ingest everything from dog repellant to caustic soda out of curiosity. In clinical practice a noxious taste or burning sensation in the mouth produces one of two reflex actions—either a spitting out or a reflex swallow, often with dire results.

Australian experience parallels that in the United Kingdom, where poisonings with certain drugs remain uncommon, but are associated with high mortality. Cardiotoxic drugs and tricyclic antidepressants are the most lethal at present (table V), but prescribing fashions obviously influence the rank order of drugs which become a threat to children. Thus in the 1950s both Ryan in Queensland¹⁰ and Craig and Frazer in Scotland¹¹ noted the comparative rarity of strychnine as a poison causing admission to hospital at that time; but drug preparations containing strychnine headed the list of drugs responsible for deaths of children in the United Kingdom in the years leading up to 1950 and were second (after kerosene or paraffin) as a cause of deaths from poisoning in Queensland. Neither strychnine nor paraffin (kerosene), however, has caused a death in Brisbane for at least 15 years. Strychnine has disappeared from the obstetric pharmacopoeia in common use; and kerosene (paraffin) is covered by legislation to colour it blue as a warning of toxicity. In 1975, a study in Cardiff showed that 25% of children admitted for ingesting poisons had swallowed aspirin,²⁵ but the rates of both fatality and morbidity for poisoning with aspirin have since declined very considerably.

Though some controversy still surrounds the apparent success of safety packaging for aspirin and paracetamol,¹ there is now incontrovertible logic to extend this to tricyclic antidepressants and cardiac drugs (see table I). In Australia and in the United Kingdom these two groups are now the biggest killers of toddlers who die from accidental ingestions. In the United States it has been estimated that child resistant packaging has prevented 200 000 accidental ingestions since 1973,⁷ and British experience is also encouraging.²⁶ This measure, together with a ban on the sale of caustic soda as a do it yourself drain cleaner, would halve accidental poisoning fatalities in toddlers on current evidence. The crusade against childhood fatalities from poisoning is one of the success stories of modern preventive medicine. At a time when some types of childhood accidents are increasing, deaths from accidental poisoning have shown one of the most heartening falls. Nine children in urban Queensland died in the five year period 1957-61 (33 in the whole state¹²), but mortality in the period 1977-81 had fallen to one child in urban Queensland with a total of only five children in the whole state (population 2.5 million). This fall has been achieved despite an increase in population from 1.36 million in 1955 to 2.17 million in 1978.

These figures, taken in demographic perspective, indicate that children living in rural areas are relatively at greater risk. Attempts to quantify this risk, and to examine the particular poisoning profiles of rural children have yet to be reported. There is some anecdotal evidence that although the rate of fatalities from accidents in general has been decreasing in recent years,²⁸ this decrease has been achieved particularly in more sophisticated urban centres.

The intentional poisoning of children is a well recognised form of child abuse²⁹ and with the fall in the rate of serious accidental poisoning is assuming greater importance. The fact that two of the 15 children in this study who died from the ingestion of toxic substances were murdered by their parents illustrates this point.

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Would the consumption of the mushrooms Psilocybe mexicana and P semilanciata in moderate (but frequent) amounts be likely to harm adults, children, or household pets?

In Britain Psilocybe semilanceata is the most commonly occurring species of hallucinogenic (magic) mushrooms and contains the hallucinogenic compounds psilocin and psilocybon, indolalkylamines, similar to lysergide (LSD) but only 1% as potent. P mexicana grows in Central America where its hallucinogenic properties are well known and is closely related to P cubensis, which is the most commonly imported species sold as dried magic mushrooms in the United Kingdom. In a recent review of 318 inquiries made to the National Poisons Information Service concerning P semilanceata ingestion a variety of signs and symptoms were found by the physician in charge (table). Most symptoms had resolved within 12 hours, though prolonged illness occurred in 26 cases (8%), 21 of whom experienced flashback phenomena of some form for up to four months' after ingestion. The number of mushrooms eaten was known in 177 cases (55%) and although usually stated as an approximation ranged from $\frac{1}{2}-1\frac{1}{2}$ mushrooms to 2-3 pounds (1 kg). No correlation between dose and effect could be found though it would suggest a quickly acquired tolerance that accompanies regular or continued use and that biological material can contain varying concentrations of active comPrincipal signs and symptoms in patients with suspected psilocybe semilanceata ingestion, 1978-81

	No (%) of patients
Hallucinations:	
Visual Auditory Tactile	113 (36)
Disturbed behaviour } Altered perception }	25 (8)
Aggression Uncontrollable	5 (2) 2 (0·6)
Nausea, vomiting, and/or abdominal pain	57 (17)
Dysuria Dilated pupils	1 (0·3) 70 (22)
Tachycardia (>100/min)	41 (13)

pounds. On the evidence listed above, *P semilanceata* and *P mexicana* are likely to be harmful to adults and children and their use is not to be recommended. Similarly, household pets may be badly affected, though no data are available.—VIRGINIA S G MURRAY, industrial toxicologist, London.

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