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Accidents, poisoning and SIDS

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POISONING IN CHILDHOOD

Poisoning in children may be accidental, non-accidental, and iatrogenic or, in older children, deliberate.

ACCIDENTAL CHILD POISONING

Epidemiology

Accidental poisoning is predominantly seen in children under the age of 5 years but older children may be involved if they are developmentally delayed. The peak age is between 1 and 4 years. More boys than girls take poisons accidentally. Some children die from poisoning each year, but the number of deaths has fallen over recent years, probably because of better treatment and because of the child-resistant container (CRC) regulations. There are also less tricyclic antidepressants prescribed. Though the numbers of deaths are few, many more children are admitted to hospital for treatment and observation and even more present to hospital Accident and Emergency (A & E) departments. Many of these are sent home directly because they have taken relatively nontoxic substances.

Substances taken

Children may take a variety of substances accidentally. These are conveniently divided into medicines (prescribed and nonprescribed), household products and plants. The majority of children who take poisons do not have serious symptoms. Medicines may be of low toxicity, e.g. the oral contraceptive pill or antibiotics; intermediate toxicity, which may cause symptoms in young children; or potential high toxicity. Many of the household products children take may be relatively nontoxic, but a few such as caustic soda, soldering flux and paint stripper may cause serious harm. The commonest household product that children take is white spirit and turpentine substitute. About 10% of these children have patchy chest X-ray changes. In developing countries paraffin (kerosene) poisoning is a particular problem as it is used as a cooking fuel and is often kept in open containers. These incidents are common in poor social circumstances and in summer and are probably largely related to thirst. Kerosene may cause serious aspiration pneumonitis and death.

A child may eat a poisonous plant accidentally or a group may sample a plant, such as laburnum, together. Most plants are relatively nontoxic, e.g. cotoneaster, rowan or sweet pea. However, some such as arum lily, deadly nightshade or yew can cause serious symptoms.

Etiology

Perhaps surprisingly, the availability of poisons does not appear to be a major factor in accidental child poisoning. There is evidence that family psychosocial stress and behavioral problems, such as hyperactivity, predispose towards child poisoning[1] and these family and personality findings have importance for the prevention of child poisoning.

Preventing child poisoning

Education

A campaign in Birmingham, UK, to publicize accidental child poisoning and to encourage the return of medicines concluded that 'publicity, storage and destruction of unwanted medicines have little preventive value'. A New Zealand study evaluated placing 'Mr Yuk' stickers on poisons together with a campaign to prevent child poisoning. No reduction in poisoning admissions was found. The link between acci-

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dental child poisoning and family psychosocial stress and hyperactivity make it unlikely on theoretical grounds that education will be effective. Families under stress will be unlikely to remember safety propaganda.

Child-resistant containers

CRCs were first suggested in 1959 by Dr Jay Arena in Durham, North Carolina. These containers were evaluated in a community in the US by Scherz[2] and found to be successful. They were then introduced into the US for aspirin preparations, with successful results. Following this work in the US, child-resistant closures were introduced by regulation in 1976 in the UK for junior aspirin and paracetamol preparations. This resulted in a fall in admissions of children under 5 years with salicylate poisoning.[3] In 1978 CRCs were introduced by regulation for adult aspirin and paracetamol tablets. Child-resistant containers or packaging are now a professional requirement by the Royal Pharmaceutical Society and are regulated for a number of household products (e.g. white spirit and turpentine substitute).

Other methods of preventing child poisoning

Lockable medicine cupboards have been suggested for the prevention of child poisoning. On theoretical grounds they are unlikely to be effective as parents under stress are less likely to remember to put their medicines away or lock the cabinets. Making household products unpalatable with bitter chemical agents (e.g. Bitrex – denatonium bromide)[4] is a possible preventive measure to child poisoning. Serious accidental poisoning might also be prevented by a reduction in the prescribing of toxic drugs. This has been done for barbiturates and tricyclics and might also be done for quinine and vaporizing solutions.

DELIBERATE SELF-POISONING IN OLDER CHILDREN

A number of older children take poisons deliberately. They may take medications as a response to an emotional crisis (mainly adolescent girls) or ingest excess alcohol (predominantly adolescent boys). They form one end of an age spectrum of overdose in adults.

NON-ACCIDENTAL POISONING

The recognition of non-accidental poisoning as an extended syndrome of child abuse was made in 1976. These children are deliberately poisoned by their parents and may present with bizarre or unusual symptoms rather than poisoning directly. A number of medicines or household products may be given to children including salt in feeds to babies. These cases probably form part of the syndrome of factitious illness in childhood.

TREATMENT OF CHILD POISONING

Management of accidental poisoning in childhood

General

The majority of children who present to hospital after accidental poisoning do not have serious symptoms. There are nevertheless a few children who have taken a significant poison and who are potentially ill or are ill. We should clearly like to prevent unnecessary admissions to hospital whilst maintaining safety. A way round this dilemma is to classify the substance the child has taken into one of four categories: low toxicity, uncertain toxicity, intermediate toxicity or potential high toxicity. After classification children can be either sent home, observed in the A & E department or admitted for observation or treatment. **The Poisons Information Center should be contacted if there is any doubt about the toxicity of a substance a child has taken or the treatment that is needed**. A list of some of the poisons commonly taken accidentally by children is shown in Table 6.1.

Older children who have taken poisons deliberately and cases of iatrogenic poisoning and non-accidental poisoning should all be admitted to hospital.

The parents of all children who present with accidental poisoning should be given advice regarding the storage of medicines and household products. The health visitor should be contacted in all cases, remembering that family psychosocial stress is often linked with accidental poisoning in childhood. There may be specific problems, which need medical or social help.

Emptying the stomach

It used to be standard practice to empty the stomach in cases of accidental poisoning in childhood. This has come under review and emesis using ipecacuanha pediatric mixture (ipecac) is now not used.[5] Gastric lavage was largely abandoned some years ago. It may be needed in certain cases, such as iron poisoning, for substances to be instilled into the stomach. The stomach should never be emptied in cases of hydrocarbon ingestion, such as paraffin or white spirit, or with corrosive substances such as caustic soda.

Activated charcoal

Activated charcoal is being increasingly used in the management of childhood poisoning. Activated charcoal absorbs toxic materials in the gut by offering alternative binding sites. Its routine use is limited by its poor acceptance by children. Activated charcoal may not be effective more than 1 h after ingestion.[6] Two preparations are available: Medicoal 5 g sachets and Carbomix 50 g. Activated charcoal has been used for a variety of drugs including aspirin, carbamazepine, digoxin, mefenamic acid, phenobarbital, phenytoin, quinine and theophylline. It is particularly useful in tricyclic antidepressant poisoning.

Accidental poisoning with substances of low toxicity

Children who have taken substances of low toxicity can be allowed home after assessment. Their parents should be given advice regarding storage of medicines and the general practitioner should be contacted. If there is doubt over what substance has been taken, a child should be admitted for observation.

Accidental poisoning where there is uncertainty of toxicity

If there is any doubt about the toxicity of a substance a child may have taken, the Poison Information Center should be contacted day or night:

London – Guy's Hospital Tel. 0207 407 7600 or 0207 635 9191
Edinburgh – Royal Infirmary Tel. 0131 536 1000
Cardiff – Llandough Hospital Tel. 02920 709901
Belfast – Royal Victoria Infirmary Tel. 01232 240503

Some children arrive in hospital having taken unknown tablets or household products. Often research with the pharmacist will help. If there is doubt the child should be admitted for observation.

Intermediate toxicity

Children who have taken substances of intermediate toxicity accidentally should be observed for a period in hospital (usually up to 6 h) until the practitioner can be confident that significant symptoms are not going to occur. This observation can be undertaken in many cases in an A & E department which has a section for children or for short periods in the pediatric ward or day unit. If there are adverse factors, particularly social factors, children should be admitted to hospital for longer periods.

Accidental poisoning with potentially toxic substances

Children who have taken substances of potential toxicity should be admitted to hospital for observation and treatment.

Treatment of individual poisons

Table 6.1 shows the toxicity of the substances which are most frequently taken accidentally by children under 5.

Deliberate poisoning in older children

Deliberate poisoning in older children should be treated differently from accidental poisoning in younger children. These children form one end of the age spectrum of overdose in adults and such children are more likely to take significant amounts of poison. Substances that

Table 6.1 Guide to toxicity of substances taken in accidental child poisoning

Low toxicity

Medicines
Antibiotics (except ciprofloxacin, sulfasalazine and chloramphenicol)
Antacids
Calamine
Oral contraceptives
Vitamin preparations which do not contain iron
Zinc oxide creams

Household products
Chalks and crayons
Emulsion paints and water paints
Fabric softeners
Plant food and fertilizers
Silica gel
Toothpaste
Wallpaper paste
Washing powder (except dishwasher powder)

Plants
Begonia
Cacti
Cotoneaster
Cyclamen
Honeysuckle
Mahonia
Rowan
Spider plant
Sweet pea

Intermediate toxicity

Medicines
Cough medicines (most)
Fluoride
Ibuprofen
Laxatives
Lidocaine (lignocaine) gel
Paracetamol elixir
Salbutamol

Household products
Alcohol-containing colognes, aftershaves and perfumes
Bleach
Detergents
Disinfectants (most)
Nail varnish remover
Paints (oil based)
Pyrethrins
Rat or mouse poison
Talc
Window cleaners

Plants
Berberis
Fuchsia
Holly
Pyracantha

Potentially toxic

Medicines
Benzodiazepines
Carbamazepine
Codeine-containing cough medicines
Clonidine
Digoxin
Diphenoxylate (Lomotil)
Hyoscine
Iron
Mefenamic acid (Ponstan)
Metoclopramide
Mianserin (Bolvidon)
Paracetamol tablets
Phenytoin
Quinine
Salicylates
Theophyllines
Tricyclic antidepressants (including dothiepin and amitriptyline)

Household products
Acids
Alcoholic beverages
Alkalis
Bottle-sterilizing tablets
Camphor and camphorated oil
Carbon monoxide
Cetrimide
Disk batteries
Essential oils (e.g. real turpentine, pine oil, citronella and eucalyptus)
Methylene chloride (paint stripper)
Organochloride insecticides
Organophosphorus insecticides
Paradichlorobenzene mothballs
Paraquat
Petroleum distillates (white spirit, paraffin, turpentine substitute)
Phenolic compounds
Slug pellets (metaldehyde)

Plants
Arum lily
Deadly nightshade
Laburnum
Philodendron
Yew

can be regarded as having intermediate toxicity when taken accidentally should be regarded as potentially toxic when taken deliberately.

Poisoning in older children should be recognized as a serious symptom and an indication of child and family disturbance. Children who deliberately take poisons show more disturbed family relationships than children referred for psychiatric help for other reasons. They have a high level of psychiatric symptoms, especially depression. All children who take poisons deliberately should be admitted to hospital and should be assessed by a child and adolescent psychiatrist. Many will need educational, psychological and social work help as well as psychiatric assessment.

CHRONIC POISONING

Lead poisoning

Lead is a serious poison for children. Its toxic effects are due to its combination with sulfhydryl groups of essential enzymes resulting in disturbances in carbohydrate metabolism, cell membrane transport, renal tubular absorption and other body processes. The blood level at which toxic effects become evident varies from child to child but in general major symptoms are unlikely if the whole blood lead level is less than 2.5 µmol/L (52 mcg/100 ml). It is probable that behavioral and learning difficulties may result from exposure to only moderately elevated lead levels between 1.4 and 2.9 µmol/L. Low-level fetal lead exposure at less than 1.4 µmol/L may also affect mental development. Children in the UK may be poisoned by sucking or chewing lead paint. Lead from burning batteries, lead shot for fishing and lead from old water pipes are other potential sources. Children from the Indian subcontinent may be poisoned by *surma*, the lead-containing eye make-up used even in young babies.

Clinical features

Children who are poisoned by lead are likely to present with pica (compulsive eating of substances other than food), anorexia, abdominal pain, irritability and failure to thrive. Severe lead poisoning may present

with neurological symptoms including drowsiness, convulsions and coma from lead encephalopathy. Lead poisoning may also present as progressive intellectual deterioration.

The diagnosis is made by elevated blood lead levels and anemia with hypochromia and basophilic stippling. There may also be increased bone density with transverse bands at the ends of the long bones on radiological examination.

Treatment

The source of lead should be identified and removed. Chelating agents should be used to form nontoxic lead compounds. In mild cases D-penicillamine should be used orally in two daily doses of 10 mg/kg. In severe cases sodium calcium edetate (EDTA) should be used, 40 mg/kg by i.v. infusion over 1 h twice daily for up to 5 d. Each gram of EDTA should be diluted in 100 ml normal saline. The effectiveness of EDTA can be enhanced by the deep i.m. injection of dimercaprol 2.5 mg/kg 4-hourly for 2 d, 2–4 times on the third day, then 1–2 times daily until recovery.

Mercury poisoning

This once common disorder was called 'pink disease' because of the color of the extremities or 'acrodynia' because of the accompanying pain. It was largely due to the use of mercury-containing teething powders which have now been withdrawn. There was anorexia, loss of weight and hypotonia as well as the characteristic painful red or pink extremities. A differential diagnosis of this condition is the red extremities of neglected children. Treatment of mercury poisoning is by the deep i.m. injection of dimercaprol 5 mg/kg 4-hourly for 2 d, 2.5 mg/kg twice daily for the third day and once daily for the next 10 d.

Chronic boric acid poisoning

Chronic boric acid poisoning was a major problem in the 1940s and 1950s. It was caused by ingestion of boric acid used either as a treatment for nappy rash or as a pacifier. It presented with convulsions, vomiting and diarrhea.

NOTES ON POISONING WITH INDIVIDUAL SUBSTANCES

Intermediate toxicity

Medicines

Cough medicines. Most cough medicines do not cause serious symptoms in the doses available to children. Medicines based on antihistamines may cause drowsiness and anticholinergic effects. Drowsiness will usually not need treatment but if coma occurs resuscitative measures should be used. Medicines based on codeine should be regarded as potentially toxic.

Fluoride. Fluoride has a rapid action but is seldom toxic in the quantities taken by children. Symptoms include vomiting, nausea and abdominal pain.

Ibuprofen. Ibuprofen and other nonsteroidal anti-inflammatory agents only seldom cause symptoms in children. Symptoms may include gastrointestinal irritation, kidney and liver damage. Oral fluids should be encouraged.

Laxatives. Serious symptoms after laxative ingestion are rare. If diarrhea occurs it occurs quickly. Occasionally patients may need i.v. fluids. The child should be observed for serious symptoms for a short time.

Lidocaine (lignocaine) gel. Local anesthetics such as lidocaine are toxic in overdose, causing convulsions and circulatory collapse. Significant amounts of lidocaine gel are seldom ingested accidentally by children.

Paracetamol elixir. Paracetamol elixirs such as Calpol are sweet and sickly in large doses and serious accidental poisoning is very rare. There is insufficient paracetamol in most small bottles of elixir to cause problems. Blood levels should be checked 4 h after the ingestion if more than 150 mg/kg has been taken. Treatment (see potential toxicity section later) is only needed if the serum paracetamol level is above 200 mg/L at 4 h (or in rare delayed cases in children above 50 mg/L at 12 h). In most cases children can be discharged after a period of observation.

Salbutamol. There may be peripheral vasodilatation, muscle tremors and agitation. Serious symptoms are rare although severe hypokalemia and arrhythmias have been seen.

Household products

Alcohol-containing perfumes, cologne and aftershave. Symptomatic cases are rare. Asymptomatic children can be allowed home after a short period of observation to make certain they do not become drowsy.

Bleach. Ingestion of household bleach causes fewer problems than would be expected.[7] Ipecac or lavage should not be used. Milk or antacids can be given orally. Local lesions in the mouth can be treated symptomatically and in the few cases where significant esophageal involvement is possible, endoscopy can be undertaken.

Detergents (anionic). Dishwashing liquid and shampoo are only toxic in large doses. Vomiting occurs in large doses. A period of observation may be needed.

Disinfectants. Serious cases are unusual.

Nail varnish remover (acetone). Observation for a period should be all that is needed but nausea and vomiting may occur, going on to coma if large amounts are taken.

Paints (oil based). Unless the paint has lead in it, the only problems that occur are caused by the petroleum distillate base. The stomach should not be emptied. In practice children do not seem to take significant amounts.

Pyrethrins. These insecticides are not usually a hazard if ingested or inhaled accidentally. The child should be observed for a short period.

Rat or mouse poison. The common ingredients of rat or mouse poison (warfarin or dichlorolose) are usually nontoxic in the doses taken by children. The exact type of poison should be identified using the poisons center and in most cases the child can be sent home after a short period of observation.

If large amounts of warfarin are ingested vitamin K can be used but this is not needed in most cases.

Talc. Talc is only toxic if inhaled. It may cause retching and choking due to pulmonary edema. Cases of ingestion only need a short period of observation to make certain inhalation did not occur.

Window cleaner. Most are nontoxic unless aspirated. The cleaner should be identified using the poisons center. In most cases the child can be sent home after a short period of observation.

Plants

Berberis. Very occasionally causes confusion, epistaxis or vomiting. The child should be observed for a short period.

Fuchsia. Unlikely to cause problems although potentially toxic. Observe for a short period.

Holly. Unlikely to cause problems although potentially toxic due to ilicin and theobromine.

Pyracantha. This causes nausea and vomiting but is unlikely to cause problems. The child should be observed for a short period.

Potential toxicity

Medicines

Barbiturates. May cause coma and hypotension. Cases are becoming less common, as they are less frequently prescribed. Activated charcoal can be used.

Benzodiazepines – tranquilizers and hypnotics such as diazepam (Valium) and nitrazepam (Mogadon). These can cause drowsiness and coma, but problems are unusual in accidental ingestion. In very young children respiratory depression may need treatment with artificial ventilation.

Carbamazepine. This drug has some anticholinergic activity. Paradoxically convulsions and violent reactions may occur as well as cardiac problems such as heart block. Activated charcoal is useful to adsorb carbamazepine.

Codeine-containing cough medicines. If significant amounts are taken there can be respiratory depression, for which the antagonist naloxone can be used (10 mcg/kg i.v.)

Clonidine. Clonidine can cause bradycardia, hypotension, coma and gastrointestinal upset. The use of atropine and dopamine infusion for the hypotension is controversial and supportive treatment (including assisted ventilation) may be adequate for even the most severe cases.[8]

Digoxin. Digoxin can be a serious poison in children, with only a few tablets being fatal. Activated charcoal is useful. These children should be monitored very closely, probably in an intensive care unit, with careful ECG monitoring. Beta-blockers such as propranolol should be used in severe cases, with atropine if there is heart block. The serum potassium should not be allowed to go too low or too high. Digoxin-specific antibody fragments are now available for the reversal of life-threatening overdosage (Digibind, Wellcome).

Diphenoxylate (the active constituent in Lomotil, the antidiarrheal agent). This compound has an opiate-like action, which causes prolonged respiratory depression. Treatment is with the opiate antagonist naloxone (10 mcg/kg as i.v. bolus). There may be a transient improvement followed by relapse and cases should be observed for at least 36 h and repeated doses of naloxone given as necessary.

Hyoscine. Hyoscine may cause dilated pupils, dry mouth, tachycardia and delirium due to anticholinergic effects. Observation will be all that is needed with most patients.

Iron. Iron is a potentially very serious poison, initially causing vomiting and hematemesis, but going on to acute gastric ulceration and shock. Later convulsions and cardiac arrhythmias may occur. Iron tablets may be detected by X-ray of the abdomen. Further treatment should be aimed at preventing additional absorption of the iron, by the use of the chelating agent desferrioxamine methylate, instilled into the stomach (5–10 g in 50–100 ml of liquid). Desferrioxamine should also be used parenterally (15 mg/kg/h to a maximum of 80 mg/kg) in all cases where a potentially toxic amount of iron may have been taken. The severity of a poisoning episode can be judged by the serum iron level. Levels above 16.1 mmol/L at 4 h indicate significant poisoning.

Mefenamic acid (Ponstan). This drug rarely causes problems in young children. Activated charcoal is effective. Convulsions can be treated with diazepam.

Metoclopramide (Maxolon). In overdose this drug causes extrapyramidal signs, drowsiness and vomiting. If extrapyramidal signs develop antiparkinsonian drugs such as procyclidine can be used.

Mianserin (Bolvidon). Mianserin has milder anticholinergic effects than the tricyclic antidepressants. Serious problems are uncommon. Drowsiness is the most common symptom.

Paracetamol (acetaminophen). Serious accidental ingestion of paracetamol is rare in children because the tablets are bitter and difficult to swallow and the elixir is too sweet to take in toxic quantities. Serious paracetamol poisoning may cause hepatocellular necrosis. Patients at risk of liver damage can be identified by measurement of blood levels 4 h after the ingestion. Treatment is needed if the serum paracetamol level is above 1.32 mmol/L (200 mg/L) at 4 h (or in rare delayed cases in children, 0.33 mmol/L at 12 h). Treatment is with oral methionine (at a dose of 1 g 4-hourly) for four doses. N-acetylcysteine intravenously is an alternative particularly in children who are vomiting or who present after 12–24 h when methionine is ineffective.

Phenytoin. Phenytoin ingestion may cause ataxia and nystagmus. Activated charcoal can be used.

Quinine. Quinine is a significant poison in children and has caused several deaths. It is used for night cramps. Quinidine and chloroquine are also toxic. Activated charcoal can be used.

Salicylates. Severe poisoning is now rare as aspirin preparations are no longer used for children because of the dangers of Reye's syndrome. Hyperventilation is an early sign of significant salicylate poisoning due to stimulation of the respiratory center with resultant respiratory alkalosis. There may also be a metabolic acidosis. In severe cases there is disorientation and coma.

The severity of a poisoning episode can be judged by salicylate levels. Toxicity can occur at levels above 2.2 mmol/L (300 mg/L) in children. In asymptomatic and mild cases nothing more needs to be done apart from encouraging fluid and electrolyte replacement and giving vitamin K. Activated charcoal is useful. Forced alkaline diuresis can be used in moderate to severe cases but its use is controversial and alkalinization of the urine is the important thing rather than the induction of excessive urine flow. Peritoneal dialysis can also be effective.

Theophylline. Theophylline can cause restlessness, agitation, vomiting, convulsions, coma, hypotension, hypokalemia and ventricular tachycardia. Activated charcoal can be used. Convulsions can be treated with diazepam.

Tricyclic antidepressants. Tricyclic antidepressants such as amitriptyline are serious poisons for young children. They may cause cardiac effects such as sinus tachycardia, hypotension and conduction disorders and death by their direct effect on the myocardium. There may be blurred vision and dry mouth from the anticholinergic effects. There may also be central effects of agitation, confusion, convulsions, drowsiness, coma and respiratory depression. Activated charcoal should also be used.

There is no specific antidote for tricyclic ingestion. The ECG should be monitored for cardiac arrhythmias. No treatment is indicated if there is adequate tissue perfusion and blood pressure. Metabolic acidosis should be corrected. Convulsions should be treated with diazepam. Life-threatening arrhythmias should be treated with propranolol. As tricyclics are protein bound active methods of elimination such as hemodialysis do not remove significant amounts of the drug.

Household products

Acids. Acids tend to cause inflammation and ulceration at the pylorus rather than the esophagus. This may lead to stenosis. Emesis or lavage should not be undertaken and chemical antidotes should not be given as the heat of the chemical reaction may increase injury. The extent of the injury should be assessed by endoscopy at an early stage. Steroids should be used to suppress the inflammation (prednisolone 2 mg/kg/d).

Alcoholic beverages. These may cause severe hypoglycemia. Blood alcohol levels are useful in management. Hypoglycemia should be detected by frequent blood glucose measurements, and prevented and treated by i.v. glucose infusions.

Alkalis. Alkalis such as caustic soda and dishwasher powder can cause burns to the mouth and esophageal ulceration, leading to stricture: review by esophagoscopy and treatment with steroids have improved the outlook for this condition. Emesis or lavage should not be undertaken, nor any chemical antidotes as the heat of the reaction may increase injury. The extent of the injury should be assessed by endoscopy at an early stage. Steroids should be used to suppress the inflammation (prednisolone 2 mg/kg/d).

Bottle-sterilizing tablets. Bottle-sterilizing tablets contain a bleach-like substance (sodium dichloroisocyanurate). They effervesce with water to make a sterilizing solution. If this reaction takes place in the mouth considerable damage can take place with edema and ulceration. Cases need to be monitored for their airway patency and i.v. fluids may be needed. Monitoring for esophageal involvement by endoscopy may be needed in some cases. The use of steroids is logical in severe cases.

Camphor and camphorated oil. These are dangerous poisons for children. They are absorbed quickly and because they are lipid soluble, enter the brain causing delirium, rigidity, coma and convulsions. Convulsions should be treated with diazepam.

Carbon monoxide. Hemoglobin has an affinity for carbon monoxide over 200 times greater than for oxygen. Carboxyhemoglobin will reduce the amount of hemoglobin available to carry oxygen and also hinders oxygen release. The incidence of carbon monoxide poisoning has fallen since house gas no longer contains this substance. Carbon monoxide poisoning should be treated with 100% oxygen over a period of several hours. Hyperbaric oxygen should be considered in severe cases if it is available.

Cetrimide. Cetrimide is a cationic detergent and can be caustic when concentrated. The stomach should not be emptied. If problems with ulceration occur steroids should be used. Cetrimide may also have depolarizing muscle-relaxing effects leading to breathlessness.

Disk or button batteries. Mercury cell, alkaline manganese and silver cell batteries contain a strong alkali (usually potassium hydroxide) as a main ingredient. Mercury cell batteries contain toxic amounts of mercury. Silver cell batteries generally contain less toxic ingredients than the other types. Worn batteries are less toxic than new ones.

Disk batteries can cause problems if they lodge in the gut and become corroded and release their contents. They may cause ulceration or perforation from caustic injury if lodged in the esophagus or stomach and should be removed endoscopically if they lodge there. If they go beyond the stomach they are usually passed without problem. Their progress should be monitored by abdominal X-ray. Mercury levels should be measured when appropriate. If the battery shows signs of leaking or breaking it should be removed surgically.

Essential oils. Essential or volatile oils contain mixtures of cyclic hydrocarbons, ethers, alcohols and ketones. They include turpentine, pine oil, citronella and eucalyptus as well as such things as Karvol capsules. Their toxicity varies, with real turpentine (not to be confused with turpentine substitute) being very toxic. Symptoms of essential oils include vomiting, drowsiness and convulsions. Ipecac should not be used.

Methylene chloride (paint stripper). This is a very serious poison for children. It is caustic and may cause damage to the skin, stomach, mucous membranes and pharynx. Vomiting, dizziness, confusion, toxic myocarditis and hemoglobinuria may occur. Methylene chloride is metabolized to carbon monoxide and carboxymethemoglobin concentrations may be elevated for several days. The stomach should not be emptied. Fluids should be given to dilute the methylene chloride. High-flow oxygen should be given if carboxyhemoglobin is present.

Organochloride insecticides. These include DDT, dieldrin and lindane. Symptoms include excitability, muscle twitching and convulsions. Activated charcoal is valuable.

Organophosphorus insecticides. A wide range of compounds which include malathion. They act by inhibiting cholinesterase in the blood. Symptoms include confusion, nausea, vomiting, wheezing and convulsions. If symptoms appear atropine (i.v.) 0.05 mg/kg and pralidoxime 20–60 mg/kg as required, depending on the severity of the poisoning, should be given by slow i.v. injection and repeated if needed.

Paradichlorobenzene mothballs. Most cases of accidental ingestion do not have serious symptoms. Ingestion may cause nausea and vomiting and cyanosis may develop due to methemoglobinemia. This should be treated with methylene blue.

Paraquat. Paraquat weedkiller is available in two forms: a concentrated form (Gramoxone) available only to farmers and horticulturists and a granular form (Weedol) which contains only 2.5% paraquat. Accidental ingestion of the concentrated form is rare and ingestion of the granular form rarely causes serious problems. Paraquat causes local ulceration and in severe cases a proliferative alveolitis. Treatment should be to prevent absorption by Fuller's earth or bentonite.

Petroleum distillates, e.g. kerosene, turpentine, white spirit and turpentine substitute. These substances may cause a pneumonitis from lung aspiration. Ipecac or lavage should not be used. Kerosene poisoning is a particular problem in the Third World. Rhonchi are the most common physical sign and X-ray changes are common. Treatment in mild cases is symptomatic, together with the use of prophylactic antibiotics. Corticosteroids are often used, but clear evidence of their effectiveness is lacking. Severe cases may need oxygen and intensive respiratory care.

Phenolic compounds. These include cresols, menthols, phenols and hexachlorophene. Coal tar vaporizing solution contains cresol. They may cause local corrosive damage and there may be cerebral symptoms. Activated charcoal can be used.

Slug pellets (metaldehyde). Slug pellets contain about 3% metaldehyde which is toxic in children and 4 g is said to be fatal for a child. Experience suggests that problems do not arise after accidental ingestion. The child should be observed for 4–6 h to check for serious symptoms such as flushing, salivation and convulsions. Convulsions should be treated with diazepam.

Plants

Arum lily. Causes gastrointestinal side-effects and later CNS manifestations.

Deadly nightshade (atropa). This plant has an atropine effect causing photophobia, visual disturbance, dryness of mouth, flushed skin, etc. If there are symptoms a slow i.v. injection of physostigmine can be used.

Laburnum. Causes vomiting, diarrhea and nausea. Although quite commonly taken, serious problems are very rare.[9]

Philodendron (Swiss cheese plant) and Dieffenbachier. This plant has a local caustic action due to oxalic acid and may cause a sore mouth and laryngeal edema. Steroids may be useful in severe cases.

Yew. Causes gastrointestinal side-effects and later CNS manifestations. There may be severe hypotension.

UNINTENTIONAL INJURY

This section deals with the nature of injury, sources of data, the magnitude of the problem, and the etiology and prevention of injuries. In many cases websites are listed where up-to-date information and evidence-based material can be found. As scientific publications often refer to data 2–3 years previously, information in the print media is always somewhat dated. Many, but not all, websites are constantly updated, and where these are available they have been included as preferential references.

DEFINING INJURIES

The first issue to determine is: what is an injury? Injury can be divided into physical injuries and psychological injuries. Physical injuries are due to tissue damage caused by excess energy transfer. When physical, radiation, or thermal energy absorbed by the body exceeds the innate capacity to deal with such forces, tissue damage results. Whether such injuries are recognized will depend on the extent of the damage and whether this causes sufficient pain or loss of function to come to the notice of the injured person. Many toddlers have bruise marks on their shins from bumping into objects but these are generally not considered to be 'injuries'. Generally, an 'injury' must reach a certain severity threshold before it is considered as such. This threshold varies between groups and cultures and depends to a considerable extent on the availability and ease of access to healthcare. The severity threshold for injury is often defined as one which leads to restricted activity for a given period (1 or 3 d are commonly used) or which leads to the injured person seeking medical attention. This leads to different methods of counting injuries, which to a large extent depend on available data sources. There are two main options: surveys and information abstracted from administrative registers, which are discussed later.

SOURCES OF DATA ON INJURY

There are a considerable number of these, including data on mortality, morbidity, and disability.

MORTALITY DATA

Mortality data are the most widely available with most countries collecting and reporting on the causes of death to their citizens. Publications on the causes of death are generally made available by national governments and some of these are summarized by bodies such as the World Health Organization (WHO). The WHO has an excellent site where it is possible to obtain a large amount of information on the scale of the burden of injuries.[10]

On this site it is possible to download major reports, such as the 2002 World Report on Violence and Health, and the 2004 World Report on Road Traffic Injury Prevention, and to access links to many useful sites and initiatives. Clicking on the research tools button brings one to a series of useful links, including the WHO mortality database.[11] Entering

this site allows the downloading of data from countries and also understanding of the completeness and coverage of mortality statistics around the world. Data are missing or incomplete from many middle and low income countries, making it impossible to accurately count the global total number of deaths or the exact distribution of causes of death. The WHO site has recently been amended to include a specific area for children.[12] This area includes the WHO–UNICEF document *A Global Call to Action: child and adolescent injury prevention*, published in 2006.[13]

This report estimates that a minimum of 875 000 children under 18 years of age die from injuries each year and that 95% of these occur in low- and middle-income countries. In children aged between 1 and 15 years four mechanisms of injuries appeared in the top 15 causes of death: road traffic injuries (6th), drowning (7th), fire-related burns (11th), and poisonings (15th). The WHO has followed up this report with an action plan to reduce this enormous burden on children's health with six areas identified for improvement: data and measurement, research, prevention, treatment services, capacity development and advocacy.[14]

The WHO will publish a World report on Child and Adolescent Injury Prevention in 2008.

In the UK mortality statistics are collated by the General Register Office for England and Wales,[15] with separate offices and statistics for Scotland[16] and Northern Ireland.[17]

Table 6.2 has been compiled by abstracting data from a publication by the Office for National Statistics[18] showing the distribution of injury-related deaths in children and young adults in England and Wales in 2003.

Table 6.2 shows several interesting patterns. The absolute numbers of deaths increases across the age groups but it should be remembered that the denominators are different. Rates are required for comparisons of groups or areas but absolute numbers can also provide easily interpretable information.

From the table it can be seen that transport accidents are a major cause of injury-related deaths at all ages. Pedestrian fatalities are a problem for all age groups but become hugely outnumbered in older age groups by occupant fatalities when this becomes the dominant mode of transport. Falls account for few deaths in any of the three age groups and are substantially outnumbered by deaths due to drowning or as a result of exposure to smoke, fire and flames. Accidental poisoning fatalities are exceptionally rare in the younger age groups but would be expected to be much more frequent than in those in older age groups. It is difficult to conceive of situations in which adults mistakenly ingest a toxic substance. The low rate in the young age group is undoubtedly due to the effectiveness of child-resistant containers introduced some decades ago, one of the most effective of all injury prevention initiatives.

The much higher number of accidental poisoning deaths in the 15–24-year-old age group does not represent accidental ingestion of substances but ingestion of greater than planned amounts of recreational drugs and which result in 'accidental' deaths. The lack of quality control in the production of illegal recreational drugs produces variations in the strength and content of products. Whilst the risk is probably very low for each ingestion, the frequency of ingestion by large numbers of young people mean that even rare failures in quality control will result in frequent tragedies, both amongst new and habitual users.

Whilst the focus of this chapter is on unintentional or accidental injuries it should be noted that intentional self-harm is one of the commonest causes of death in those aged 15–24 and also that quite a few children and young adults are killed during acts of violence.

There are a limited number of studies providing international comparative data. One such study[19] compared death rates across European Union countries in the 1–4, 5–9 and 10–14 age groups. For both sexes injury deaths exceeded those due to infection of cancer at all age groups. Injury-related deaths accounted for a low of 27% of all deaths in girls aged 1–4 and a high of 51% of all deaths in boys aged 10–15. There was considerable variation between countries. The countries with the lowest injury-related death rates were Sweden and The Netherlands and the highest death rates occurred in eastern Europe.

Interpreting the meaning of variations in injury mortality across countries is not straightforward. Variations in death rates can be due to variations in exposure to certain factors or to the effectiveness of countermeasures. For example, warm countries with a lot of childhood exposure to open water, will inevitably have higher drowning rates than those which have much less open water or where the temperature is so cold that swimming is avoided. The excess deaths in the warm countries with a lot of open water will be due to a mixture of inadvertent exposure (falling in) and to difficulties encountered during planned exposure (swimming). The degree to which children are taught to swim and to which physical barriers and observation (lifeguards) protect children will also impact on the mortality rates. Generally speaking, mortality statistics are based on the place of usual residence and can mask holiday-related hazards. An analysis of childhood drowning deaths in the UK[20] demonstrated the much higher risk to UK children from unsupervised swimming pools whilst on foreign holidays than from pools in the UK. Similarly, mortality league tables for road traffic related injuries will be influenced by the degree of motorization within countries, as well as behavioral traits and investment in safety measures. Road traffic increases rapidly with industrialization and economic development and there is generally a marked rise in road traffic related deaths, which then plateau and decrease somewhat as safety measures and legislation are introduced and enforced.

Table 6.2 Numbers of deaths due to injury and poisoning by cause and age band to children and young adults in England and Wales in 2003 (Office for National Statistics, 2004)

	Age group		
Category	1–4	5–14	15–24
All injury and poisoning	100	172	1573
Transport accidents	18	23	221
Pedestrian	8	11	29
Motorcyclist	0	1	43
Falls	3	2	7
Drowning	11	2	6
Smoke, fire and flames	15	2	3
Poisoning (accidental)	1	0	29
Intentional self-harm	0	3	301
Assault	6	12	75

MORBIDITY DATA

There are several sources of information on nonfatal injuries, although these vary across and within countries. Sources include hospital discharge or separation data, A & E department attendance data, trauma registers, survey data and data systems operated by nonhealth bodies with a focus on high-risk events such as road traffic collisions and house fires. Unlike mortality data, information from these sources is rarely reported using methods which allow direct comparison. Guidance on methodological issues in comparing injury incidence data across countries has been produced.[21] Some of the apparent differences in morbidity rates between countries are due to differences in the supply and scope of different aspects of the health service, as described in the sections dealing with hospital admission and Emergency department data. Greater or lesser proportions of trauma care are provided as inpatient, outpatient or specialized clinics in different countries and settings.

Much of the data on hospital admissions and A & E departments in this section has been derived from the Collaboration for Accident Prevention and Injury Control (CAPIC) website.[22] CAPIC is a multidisciplinary, multi-agency voluntary body which supports injury prevention initiatives in Wales.

HOSPITAL ADMISSION DATA

Table 6.3 shows hospital admission rates for all types of injury, by 5-year age groups in Wales in 2003. It can be seen that admission rates

Table 6.3 Hospital admission rates for all types of injury by 5-year age groups in Wales – 2003

Age group	Number of events	Rate/1000 population
0–4	2187	13.7
5–9	1779	9.8
10–14	2574	13.1
15–19	3249	16.6
20–24	2955	16.3
25–29	2233	14.4
30–34	2454	12.8
35–39	2436	11.5
40–44	2143	10.4
45–49	1617	8.6
50–54	1487	7.7
55–59	1571	7.8
60–64	1288	8.0
65–69	1367	9.6
70–74	1735	13.8
75–79	2401	22.6
80–84	3249	40.1
85+	4915	84.1
All ages	41640	14.2

for children and young adults are higher than in middle age but not as high as in older persons, when falls and osteoporotic fractures lead to many admissions.

The data in this section reflect age-specific hospital admission rates for Welsh residents in 2003 derived from the CAPIC data. Around 1.3% of all children and young people (0–19 years) are admitted to hospital with an injury each year. Major causes of hospital admission with an injury in this age group are falls (n = 3493), accidental poisonings (n = 783), being struck by or against objects (n = 659), involvement in motor vehicle transport accidents (n = 541), and from noncollision pedal cycle injuries (n = 440). Injuries caused by glass (n = 242), knives and tools (n = 80), scalds (n = 276) and unintentional fires (n = 35) are less common but are often associated with substantial morbidity.

There are many factors which influence the probability that a person with an injury of moderate severity will be admitted to hospital. These include issues such as bed availability, variation in clinicians' propensity to treat fractures conservatively or with surgery, co-morbid conditions, uncertainty about intentionality, and social factors. These factors generally mean that it is difficult to use hospital admissions as a valid measure of the incidence of injuries of a given nature. Cryer and Langley[23] developed the most valid injury indicators to date for hospital admissions, based on a group of conditions with a probability of inpatient death of greater than 5.9%. This approach is useful for understanding national trends but the downside is that it includes a very small proportion of all injury-related hospitalizations. Generally speaking, there are insufficient numbers of these high-risk injuries to be able to use this technique for regional or local data and consequently it is not suitable for the targeting or monitoring of local preventative interventions. Guides to the evaluation of injury prevention initiatives can be found on the CAPIC website.[22]

EMERGENCY DEPARTMENT DATA

Emergency department data are increasingly being made available as electronic collection of health information grows. In many cases the information collected merely involves the administrative and diagnostic details needed to treat individuals whereas in other cases the information systems used also collect data on the location, antecedent factors and activities resulting in the injury occurrence. Both types of data collection are useful for those interested in prevention but clearly the latter are much more useful. An excellent example of the latter is the Victoria Emergency (Department) Minimum Dataset (VEMD) in use in Victoria, Australia and exported to many parts of the world.[24] Systems such as this have provided the basis of injury surveillance which stimulated preventative interventions and the basis of much of our knowledge on the effectiveness of interventions.

Many more children are seen and treated at Emergency departments than are admitted as inpatients. Generally speaking there are 10–20 injuries treated and discharged in Emergency departments for every admission.

In the US the on-line National Electronic Injury Surveillance System (NEISS[25]) allows queries to be run on the database. In 2004 there were an estimated 13 096 983 Emergency department attendances with an injury in the US. In Europe, EuroSafe, the European Association for Injury prevention and Safety Promotion, manage the European Commission's Injury Database and collate data from Emergency departments in participating countries.[26]

Table 6.4 shows age-specific injury attendance rates in Wales in 2004. Rates in females are about one quarter or one third lower than in males at all ages. Overall, around one in four or five children and young people attend an Emergency department with an injury each year. The importance of home safety initiatives for young children is shown by the fact that 69% of all injuries to children aged 0–4 took place in the home.

Emergency department attendances are influenced by many factors other than the presence of an injury. Many less serious injuries are self-treated or treated in primary care. Distance and access can have profound effects, with one study showing a 50% reduction in injury attendance rate over a distance of 10 miles.[27] This distance decay effect was present for all injuries with the exception of fractures. Consequently fractures are frequently used as an indicator variable. The fracture rates per 1000 populations in Wales in 2004 were 16 for the 0–4 age group, and 33, 55 and 39 in the next three 5-year age groups. These are higher than any age group other than those aged 85 and over (62/1000). A further revision of the fracture indicator has been proposed, selected radiologically verifiable fractures (SRVFs), due to changes in the clinical management of different injuries. This takes into account changes in practise relating to avoiding X-ray exposure for many head and facial injuries and the likelihood that many distal radial greenstick or buckle fractures in young children do not present to Emergency departments.

Table 6.4 Estimated numbers and rates of injury for Wales, by age band, 2004

Age	Estimated numbers of injuries	Estimated rate (per 1000 population)
0–4	24560	182
5–9	27197	156
10–14	47338	241
15–19	46395	229
20–24	39104	187
25–29	29002	157
30–34	28878	141
35–39	28611	127
40–44	26306	116
45–49	19338	96
50–54	16583	85
55–59	16190	77
60–64	11706	68
65–69	9391	63
70–74	9212	72
75–79	8942	82
80–84	9371	109
85+	12493	184
Unknown	88	
Total	410704	133

SURVEY DATA

Admission and Emergency department data are often not available and even where they are, details of how and where the injury happened may not be collected. These factors are crucial to understanding the etiology and in developing preventive strategies and interventions. In such circumstances survey data can be very helpful. The World Health Organization (WHO) has developed guidelines for those carrying out community surveys on injuries and violence.[28]

Community-based surveys are particularly useful in poorer countries where access to high-quality care is often scanty and most injuries are self-treated or attended to by traditional healers. The rationale for such surveys is to implement preventive measures as outlined in the four-stage public health approach (Fig. 6.1) recommended by the WHO.

Even in wealthy countries survey data can be useful for obtaining additional information on location of injury and antecedent factors not collected at Emergency departments and also for obtaining exposure metrics.

DISABILITY DATA

There is a lack of data on the medium and long-term consequences of injuries. The Global Burden of Disease and Injuries project[29] has produced initial estimates but these are based on quite sparse data and in many cases there were no data for specific types of injury. The likely distribution of outcomes for each injury was obtained by asking the opinion of medical experts based in hospitals. This is problematic as most hospital clinicians see and treat acutely injured patients and do not have a systematic process of measuring outcomes, particularly measures of function and disability. Prospective studies and empirical data on outcomes at fixed points in time are required to answer these questions. Another issue with many of the existing studies is that the injuries included in the study were often selected using a severity measure, such as the Abbreviated Injury Scale (AIS).[30] The AIS is designed to measure the threat to life from injuries in order to improve the evaluation of counter measures. However, it is not clear how closely threat to life and threat to disability are related. A stab wound to the heart has a high threat to life but if the patient survives the probability of any physical disability is slight. Amputation of a thumb carries a very low threat to life but a very high threat to disability. One of the best studies of post injury disability in young adults was that carried out on the 1958 British National Child Development Study cohort in 1981.[31] In that study 12 537 participants were asked about unintentional injuries and disability since age 16. Two thirds of men and one third of women reported at least one injury requiring hospital treatment. Injuries requiring hospital admission carried the highest risk of disability (9.7%) but 54% of permanent disabilities reported by men and 74% reported by women resulted from injuries treated as outpatients. Fractures accounted for one third of disabilities. Among injuries resulting in permanent disability hand injuries featured prominently (82% of disabling work injuries and 32% of disabling home injuries). The greatest gap in estimating the true burden on injuries is the lack of information on the medium- and long-term consequences of injury.

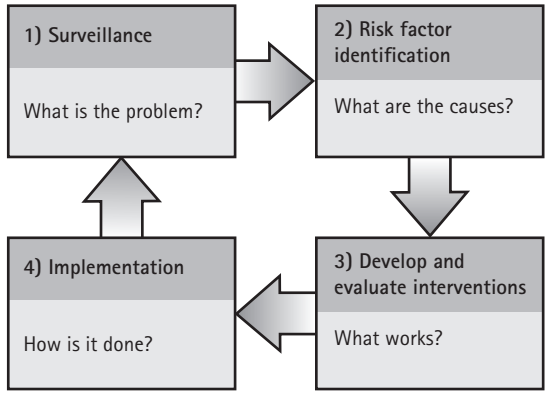

Fig. 6.1 A Public Health Perspective on injuries. (World Health Organization, p. 5 http://whqlibdoc.who.int/publications/2004/9241546484.pdf).

INEQUALITIES IN INJURY

Social inequalities in health were 'discovered' in the nineteenth century by seminal public health figures, such as Virchow in Germany, Villermé in France, and Chadwick in England. However, interest in social inequalities in health faded in the twentieth century until the publication of the Black Report in the UK in 1980.[32] This study compared death rates between the social classes. The largest of all the differences in death rates occurred in childhood injury deaths where the children from parents of social class 5 (unskilled workers) were several times more likely to die than those whose parents were from social class 1 (professionals). The ratio varied by a factor of 4 for all injury deaths to 7 for pedestrian deaths and 15 for deaths due to household fires.

Studies using individual level measures of socioeconomic position, such as social class, income or parental educational level show the greatest difference between the extremes. Social class scales tend to differ across countries and are not universally available. Usually several questions have to be answered to apply the scales, which prohibits their widespread use. In some countries, such as the US, concepts such as occupational social class are not commonly used and metrics such as family or individual income or educational level are preferred. Many studies use ecological or area-based measures to categorize groups by socioeconomic standing. These measures are usually obtained from official statistics taken during population censuses or the distribution of state-funded benefits. Area-based measures are attractive as they are easily applied once an address, postcode or zipcode are known. In many cases they are more suitable than individual level measures as many injury-prevention initiatives are implemented on an area or group basis. The strength of the relationship between injury deaths and ecological measures of socioeconomic position is attenuated due to the application of average measures to everybody within a community and the inevitable misclassification which results from applying an average score to a heterogeneous population.

The situation is not as simple when nonfatal injuries are considered. Some injuries show a marked socioeconomic gradient with higher rates in the less affluent groups or areas but the reverse occurring for other injuries. A population-based study of childhood fractures in Wales[33] demonstrated little overall variation in fracture rates between the most and least affluent groups. However, the most affluent group reported higher rates from sports participation, particularly notable for fractures related to horse riding and in-line skating, sports which require financial support. In contrast, assault-related fractures were substantially more common in the most deprived group. Clearly, some of the variations between socioeconomic groups related to differences in the presence and degree of exposure to hazardous behaviors, activities and environments. Childhood pedestrian injuries demonstrate substantial inequalities between affluent and deprived areas[34] as do childhood poisonings resulting in hospital admission[35] and burns.[36] These particular injuries show such a steep inequality gradient that they should be a main focus of effort to reduce inequalities in child health.

INJURY PREVENTION

Given the scale of the impact of injuries on children's lives, preventing injuries should be a major goal of society. Injuries are caused by interactions between individuals and their environments and the prevention of injuries requires an understanding of these issues. Few pediatricians or child health practitioners are taught the principles of injury prevention and how these can be applied to their everyday work environment. The World Health Organization has responded to this deficiency by

producing free teaching materials suitable for different circumstances. The teaching materials are suitable for three different levels of involvement: an intensive 2-day course; a 1-week short training course; and a 6-month training session. The materials are available from the WHO website.[37]

The learning objectives of the courses include:

1. to identify the basic principles of injury prevention, control and safety promotion;
2. to diagnose problems from a multidisciplinary perspective;
3. to design, implement and evaluate injury prevention and control interventions;
4. to advocate for injury prevention in communities;
5. to practice injury prevention control and safety promotion based on universally accepted ethical principles.

One important model to understand the causal chain of events involved in injuries is that proposed by William Haddon, commonly known as the Haddon Matrix (Table 6.5).

It is obvious from the matrix that injury prevention is a multidisciplinary and multi-agency task. There is scope for many different groups to become involved, either separately or in collaboration and there are many points at which preventive initiatives can be implemented. Initiatives can broadly be aimed at changing the environment, improving enforcement of safety legislation or at changing behavior through education. These are referred to as the three 'Es' of injury prevention. Clearly, each type of injury will require a tailored approach. Preventing injuries to the under fives at home will involve different groups and approaches to preventing childhood pedestrian injuries, for example. However, for almost every issue child health practitioners can play a substantial advocacy role. This is particularly the case where childhood injuries are very common or where there is social disadvantage and large inequalities in injury incidence. Advocating for those who are not in a position to protect themselves (children) and whose parents and guardians may lack the necessary information and influence to carry out this task should be a core function of child health practitioners. There are many groups which can provide assistance, including charities such as the Child Accident Prevention Trust (CAPT[38]), Royal Society for the Prevention of Accidents (RoSPA)[39] or the European Child Safety Alliance.[40]

Among the useful products produced by the European Child Safety Alliance is an 18-country Child Safety Action Plan. This action plan should help bolster support for injury prevention within participant countries and reduce the burden of injuries. Clearly, there is scope to copy this or similar initiatives in non-participant countries or with in regions and districts within countries. Child health practitioners are well placed to lead such activities but perhaps require additional training in advocacy and public health skills.

THE EVIDENCE BASE

Knowing what to implement and how to get evidence into practice are important components of the knowledge and skills framework for injury prevention practitioners. Like all aspects of health, publications relevant to injury prevention are increasing year on year and it is impossible for any practitioner or specialist to read all the published material. Several initiatives have made life easier.

For those with a need to know about latest developments SafetyLit[41] provides a weekly electronic current awareness service. This includes titles and abstracts sorted into categories from publications in over 2600 journals. For the more generalist approach to injury prevention the first source of information on effective interventions should be through systematic reviews. There are several sources of systematic reviews pertinent to injury prevention. The greatest supply of high-quality systematic reviews is produced by the Cochrane Injuries Group[42] based at the London School of Hygiene and Tropical Medicine. Their reviews are published by the Cochrane Library. Although the highest quality reviews are published by the Cochrane Group there are many others producing systematic reviews pertinent to injury prevention. Sometimes the conclusions of different groups looking at the same topics differ. This usually occurs due to differences in the thresholds set for study inclusion. The Collaboration for Accident Prevention and Injury Control (CAPIC[22]) provides a searchable database of all systematic reviews to allow the reader to identify all the relevant reviews and decide whether to focus on the most up-to-date review or read all the reviews. Some reviews focus on specific topics, such as the effectiveness of traffic calming or smoke alarms whilst others are broader ranging and cover many topics. One of the most broadly based reviews was that carried out by Elizabeth Towner and colleagues and published in two volumes,[43] in the journal Injury Prevention. These reviews cover a very wide range of topics from traffic calming to bicycle helmets to smoke alarms and educational interventions.

It is important to realize that systematic reviews only cover part of the evidence base. They are suitable for summarizing interventions which are amenable to evaluation by experimental design over a relatively short period. However, not all interventions can be evaluated using these methods. If observational studies demonstrate an intervention to be very effective then the ethical position of equipoise is lost and RCT cannot be instituted. If one considers trauma treatment as an analogy there is no trial evidence backing the use of plaster of Paris for immobilization of fractures. The evidence is based upon the principles of anatomy, physics and common sense. Nobody would ever suggest conducting a trial of immobilization versus watchful waiting to demonstrate the effectiveness of plaster of Paris although they might run trials to demonstrate the relative effectiveness of different methods of immobilization. At the other end of the spectrum it is very difficult to evaluate the effectiveness of long-term advocacy in changing cultural views in relation to the acceptability of implementing some injury prevention measures. No single mass media campaign has demonstrated effectiveness in reducing the incidence of driving whilst under the influence of alcohol. Yet, cultural attitudes to drink driving have changed over the past 30 or so years with a substantial reduction in prevalence and associated numbers of deaths.

Table 6.5 The Haddon matrix (This model is reproduced from the WHO Teach Violence and Injury Prevention course materials with permission) (http://www.who.int/violence_injury_prevention/capacitybuilding/teach_vip/en/index.html)

	Host (person)	Agent (vehicle or product)	Physical environment	Socio-economic environment
Pre-event	Is the person pre-disposed or overexposed to risk?	Is the agent hazardous?	Is the environment hazardous? Possibility to reduce hazards?	Does the environment encourage or discourage risk-taking and hazard?
Event	Is the person able to tolerate force or energy transfer?	Does the agent provide protection?	Does the environment contribute to injury during event?	Does the environment contribute to injury during event?
Post-event	How severe is the trauma or harm?	Does the agent contribute to the trauma?	Does the environment add to the trauma after the event?	Does the environment contribute to recovery?

How much of this change would have happened without mass media campaigns?

One area of evidence which is often derided is cross-national ecological comparisons. However, much can be learned from comparing practices and programs in different settings and cultures and trying to find out why some interventions seem to work in some settings and not others. One of the most useful reports on pedestrian safety in recent years is Children's Traffic Safety: International Lessons for the UK produced by Nicola Christie and colleagues for the UK Department for Transport.[44]

There were five things which distinguished the best countries, in terms of lower death rates, from the rest:

1. they had invested very widely in speed reduction measures, including environmental modification and low speed limits;
2. such measures were widely implemented outside schools;
3. there were outside play areas, such as parks and playgrounds, in most residential areas;
4. there were national publicity campaigns aimed at child pedestrian safety conducted at least once a year; and
5. they had legislation that assumes driver responsibility for accidents involving child pedestrians in residential areas.

Thus, the evidence base for injury prevention is very broad and includes systematic reviews, randomized trials, and observational, ecological and qualitative studies. Different study designs are appropriate for different questions. A comprehensive approach is usually needed to reach the correct conclusions. Providing and maintaining a broad evidence base is essential for advocacy, implementing proven strategies and developing new interventions to reduce the burden of childhood injury. The ultimate and only important aim is to have fewer dead, injured and disabled children.

POISONOUS ANIMAL BITES AND STINGS

An overview of poisonous animal bites and stings published by the World Health Organization, stated that in excess of 100 000 deaths result worldwide each year from snakes bites, scorpion stings, spider bites, marine envenomations and anaphylactic reactions to stings.[45]

SNAKE BITES

Snakes are the most widely distributed of the reptiles, and different species, often with highly individual features, predominate in different countries. Overall, there are 2.5 million snake bites each year, with a fatality rate of between 1.5 and 3%.[45,46] Tragically, in children, the mortality from snakebite is higher[47] and may reach in excess of 10% in some areas of Papua New Guinea.[48] Environmental change in some parts of the world has resulted in changes in the pattern of snakebite – as for example in parts of Africa, where prolonged drought has reduced vegetation cover, favoring the spread of *Echis ocellatus*, which has resulted in more bites.[45] Of the nearly 3000 known varieties of snake, only a minority are of medical significance and clinicians should concern themselves primarily with the characteristics and venomous effects of the poisonous snakes in their own area and importantly, have to hand details of their local supplier of antivenom.

Adder bites (Europe)

The common viper or adder (*Vipera berus*) is found throughout Europe, with the notable exception of Ireland, and its bite seldom causes death in adults, although there are reports of deaths in young children.[49] The fully grown adder may be 50–60 cm in length and is recognized by the dark zigzag band which runs along the center of the back, although rarely the snake may appear uniformly black. It is a shy creature, normally disappearing quickly on the approach of people and is only likely to bite if disturbed unexpectedly.

Clinical features

When the amount of venom injected is small, as is usual in a defensive bite, there may be few signs or symptoms, but fear often causes transient pallor, sweating or vomiting. With moderate poisoning there may be local swelling and tender enlargement of the regional lymph nodes. This swelling may increase over 1 or 2 d to involve the whole limb but usually resolves within a few weeks. A burning pain is experienced at the place bitten. Vomiting may begin within a few minutes of the bite (often followed by diarrhea) and may continue for up to 48 h. Shock is likely with weakness, sweating, thirst, coldness, absent pulse, hypotension, drowsiness and occasionally loss of consciousness. Swelling of the face, lips and tongue may occur, ecchymoses and swelling appear and may gradually extend up the limb. Bleeding may occur from gums, wound and infection sites. Other indications of envenomation include ECG abnormalities, peripheral neutrophil leukocytosis, elevated serum creatine kinase or metabolic acidosis.[49] With recovery, discoloration slowly changes from blue to green and finally to yellow before disappearing. The child generally recovers quickly from the initial collapse but in severe poisoning there may be persistent or recurrent hypotension, acute renal failure, and pulmonary or cerebral edema.[49] Full recovery may take 1–6 weeks.

Treatment

A child who is bitten by a snake should be taken to hospital as quickly as possible and, *if it can be done without causing delay and without risk of further bites*, the reptile should be killed for identification. The bite should not be incised and the patient should be kept quiet and the limb maintained at rest by splinting, helping to retard absorption of venom. Tourniquets and compression should not be used. Both the child and parents should be reassured about the expected outcome. On admission to hospital paracetamol may be given for the pain and chlorpromazine, if required, for vomiting. In hospital, blood pressure should be monitored hourly, and bleeding time should be recorded; the white cell count (raised), serum creatine phosphokinase (may be raised), serum bicarbonate (may be low) and ECG should be determined twice daily. Tetanus toxoid should be administered if there is no record of tetanus immunization within the previous 5 years, but tetanus antitoxin is not required routinely. Broad-spectrum antibiotics are required if evidence of bacterial infection develops. Antivenom (two ampoules) should be given if there is evidence of systemic poisoning, especially hypotension, bleeding or ECG changes. It should be given diluted with isotonic saline, by slow i.v. injection or infusion over a period of 30–60 min. Adrenaline (epinephrine) and i.v. antihistamine and hydrocortisone should be ready in case of anaphylaxis.[49] If the patient does not show any clinical improvement, the treatment may be repeated.[49] If there is a delay in obtaining antivenom or if a history of allergy contraindicates its use, blood transfusion or i.v. fluids may help to combat collapse.

Tropical and subtropical snakes

Morbidity and mortality from snake bites are highest in those areas where snakes have adapted to farm, plantation and village life and live in close proximity to large human populations. Examples are the Indian cobra, krait and Russell's viper in South-East Asia, some pit vipers in Latin America, the Taipan in Papua New Guinea and *Echis* species in Africa.

Land snakes may be roughly classified according to the presence or absence of poison injecting fangs, and their position when present in the snake's mouth (Table 6.6). Sea snakes, of which there are some 50 species, have characteristic flattened tails and short front fangs.

Although the Elapidae include many highly venomous varieties, these usually retreat when approached by humans and are generally non-aggressive unless cornered or molested. The large family of Colubridae contains only a few snakes of medical significance and these are rarely the cause of bites in humans. The Viperidae, on the other hand, are broad, sluggish snakes which hold their ground and may easily be trodden on (puff adder) or touched, on or in the ground or among rocks (rattlesnake, burrowing adder, berg adder). These snakes, despite their sluggishness, strike with great rapidity and power, virtually stabbing their victim with frontally situated large fangs which are swung forward during the strike.

Table 6.6 Classification of snakes

Group of snake	Features of groups	Common examples and habitat	Principal action of venom
Viperidae	Mobile front fangs	Viperinae Puff adder, widespread in Africa Gaboon viper, central and southern Africa Berg adder, southern Africa Night adder, southern Africa Rhinoceros viper, Tropical Africa Russell's viper, Asia and Indonesia Saw-scaled viper, India, Iraq, North Africa European viper (adder), Europe, Asia, Japan Crotalinae (pit vipers) Cottonmouth moccasin south-eastern United States Copperhead, N. America Fer-de-lance, Latin America; W. Indies Bushmaster, Central and South America Jaracara, South America Rattlesnakes, Mexico, southern and western United States	Cytotoxic (tropical rattlesnakes and berg adder are mainly neurotoxic)
Elapidae	Fixed front fangs	Cobras, widespread in Africa and Asia Mambas, east, central and southern Africa	Neurotoxic (king cobra also has cytotoxic effect)
Colubridae	Back fangs	Rinkhals, southern Africa Indian krait, India, Burma, Malaya, Indonesia Tiger snake, Brown snake, Australia Taipan; Death adder, Australia and New Guinea Coral snakes, southern United States; C. America Boomslang, widespread in Africa	Hematoxic
Hydrophidae (Now included in Elapidae)	Short fixed front fangs	Various species of sea snake. Mainly in the Pacific but a few species in the Indian Ocean	Neurotoxic; myotoxic
Nonvenomous	Fangless	House snake Grass snake Boas, pythons. Widespread in tropics.	Nil

Many nonvenomous snakes are capable of inflicting bites which are liable to become infected, sometimes with exotic bacteria, such as *Arizona* species.

Snake venoms broadly correspond with the families shown in the table. They are, however, complex mixtures of toxins and enzymes and effects depend upon which of these predominate. Viper (and spitting cobra) venoms are mainly cytotoxic but bleeding diatheses are common. Elapid venoms produce neurotoxic and cytotoxic effects, as do varieties of tropical rattlesnake (Crotolinae), but again microangiopathic and intravascular hemolysis may be a feature of the bites of some species. Overall, snake venoms are complex with varying mixtures of neurotoxins, myotoxins, procoagulants, anticoagulants and nephrotoxins.[46,47,49,50] The venoms may therefore also cause hemolysis, hemorrhage and coagulation disturbances.

Colubrid (e.g. boomslang) venoms are hematoxic and anticoagulant, while hydrophiid (sea snake) venoms are mainly myotoxic. Nephrotoxic properties have also been demonstrated in puff adder, sea snake and rattlesnake venoms.

Children are particularly at risk in areas where snakes are common because of their love of outdoor pursuits, together with their curiosity and carelessness. However, it is important to realize that all bites from snakes are not caused by venomous species. In the case of bites from nonvenomous snakes, two distinct puncture marks are not seen and the bites are irregular and lacerated to a greater or lesser degree, with little local swelling or pain. Even bites by venomous species do not always cause clinically significant envenomation as the bite may be deflected by clothing or the venom stores of the snake depleted. When envenomation has occurred, symptoms tend to be more severe in children because of their smaller size relative to the volume of injected venom.

Despite popular belief, while sudden collapse and death may occur (e.g. Australian brown snake, death adder), signs of systemic envenomation in even the most poisonous snake bites seldom occur before 30 min. The earliest features are often those due to fright – shock, pallor, sweating, vomiting, weak pulse and faintness. Severe pain and swelling at the site of the bite, with rapidly spreading edema, are the first signs in most viper and spitting cobra bites. Later large bullae may form around the bite and painful lymphadenopathy may develop. Sometimes there is extensive bruising in superficial and deeper tissues. Within 5 or 6 h the whole limb may be tensely swollen. There is thus a profound local cytotoxic effect and subsequent systemic disturbance is largely due to this tissue damage rather than to circulating venom. Necrosis of superficial or deep tissues may be seen. Disseminated intravascular coagulation with resultant hemolytic anemia, hemorrhagic manifestations and hemoglobinuric nephrosis, can complicate the bite.

In heavy envenomation, especially by puff adders and Gaboon vipers, bloody saliva may be expectorated, and sudden death may follow due to circulatory collapse. Notable exceptions in this family of snakes are the hemorrhagic reactions seen in bites from saw-scaled and Russell's vipers and the neurotoxic effects occurring in tropical rattlesnake and berg adder bites.

Elapid bites generally cause much less local reaction, but have profound systemic effects which are predominantly neurotoxic. Spitting cobras are however a different entity – the bites may cause significant local tissue necrosis[51] and in a review of bites from the Australian copperhead, one third showed significant local effects.[52] The first symptoms of neurotoxic snake bite are usually ptosis, rapidly followed by strabismus, slurred speech and dysphagia, with drooling saliva. There is confusion and hypersensitivity to tactile stimuli. If untreated, the victim may be completely paralyzed within minutes and respiratory paralysis may result in death within 15 h. In survivors there are no neurological sequelae. In the case of mamba bites, the first systemic symptoms are combined with a sensation of tightness and pain across the chest.

Violent abdominal pain sometimes occurs after krait and coral snake bites. Local blistering may be seen after envenomation by some cobras and a burning sensation at the site is often described. Local pain after snake bites is, however, extremely variable. Hematuria and hemoglobinuria may occur as may hemorrhage or menorrhagia.

Some elapids, such as the spitting cobra and the rinkhals of southern Africa are able to eject venom with considerable force and accuracy at the victim's head. Should this enter the eyes, a severe keratoconjunctivitis can result, which may lead to blindness if not adequately irrigated and treated.

The family Colubridae covers many snakes, including the Montpellier snakes, the red-necked keelbacks, the yamakagashis, the herald snake and the vine snake[49] but the boomslang is the only venomous snake in this large family which is a significant hazard to humans. Fortunately bites from this species are rare, usually occurring in those handling or working with snakes. There is little or no local reaction apart from mild burning pain but severe headache about 1 h after the bite is a regular and unexplained phenomenon. Colubrid venoms contain mainly fibrinolysins and hemorrhagins, which result in severe, systemic defibrination and generalized hemorrhage, widespread cutaneous bruising especially at sites of trauma, and free bleeding from fang punctures. There may be massive intestinal, urinary tract or intracranial hemorrhage leading to death. As mentioned earlier, the venom of certain vipers and also some elapids (such as the Papuan black snake) produces similar hemorrhagic effects.

Sea snakes do not attack in water if unmolested. Most bites result from handling the snake when caught in fishing nets. The bite shows no local reaction and minimal pain[53] and in most sea snake bites no envenomation occurs.[54] Initial symptoms, due to a myotoxin, are seen after a latent period of 30–120 min. There is muscular pain, stiffness and trismus followed by ptosis and progressive weakness which may threaten respiratory function. Rhabdomyolysis, myoglobinuria, renal tubular necrosis and acute renal failure may ensue.

Hadley et al,[55] in southern Africa, found thromboelastography to be a good predictor of severity in snake bite in children. They felt that, although it did not supersede clinical observation in the management of snake bite in children, it did allow stratification into high- and low-risk categories.

Treatment

The administration of antivenom, generally intravenously, is an effective treatment for a case of envenomation by a poisonous snake, but for many snake species, no antivenom may be available. The use of antivenom, especially polyvalent antivenom, carries a substantial risk of anaphylaxis and serum sickness because of its foreign protein content, but there is no doubt that the benefits of antivenom treatment far outweigh the risks.[45,51] Its administration in every case of snake bite is, however, dangerous, wasteful and unnecessary. As not all snake bites are due to poisonous snakes, it is obviously of paramount importance, as an initial step, to identify the snake accurately, or at least to decide clinically into which group it falls. Much can be deduced from the color and shape of the snake, manner and circumstances of striking, the situation of the bite and presence or absence of fang marks. For instance, the puff adder, responsible for 95% of poisonous snake bites in Africa, is encountered on paths or in grassy terrain, and almost always strikes at the feet or ankles. On the other hand, cobras often attack when they are surprised near outbuildings or chicken runs. They rear up prior to striking and bites are frequently inflicted above the knees or even on the trunk or upper limbs.

As many victims of snake bite are not within easy access of clinic or hospital, it is most important to lay down firm and easily understood guidelines which can be applied by a layman on the spot:

1. *Symptoms* developing within the first half hour of a bite are almost always due to fear and its effects – and not to envenomation. The patient should be calmed and reassured, and encouraged to lie down quietly so as not to disseminate the poison by restless body movements. The affected limb should be kept horizontal at this stage and gently splinted to avoid movement. A mild analgesic (paracetamol) should be given.
2. *Bites* from nonvenomous snakes should be thoroughly cleansed with a dilute antiseptic solution, any loose teeth removed, and a light dressing applied. Tetanus toxoid should be administered within 24 h to all cases of snake bite if there is no documented evidence that the child has received anti-tetanus immunization within the past 5 years, and broad-spectrum antibiotics should be given at the first indication of possible infection.
3. *Signs* of significant envenomation generally develop from half to 2 h after the bite. It is imperative to get the patient to a hospital, or a trained person to the patient, as soon as possible. Signs may be local in the case of viper bites, with swelling and pain, or systemic in the case of bites from snakes with neurotoxic and hematoxic venom, with varying manifestations including vomiting, abdominal pain and neurotoxic symptoms, as noted earlier.[51] They call for urgent administration of antivenom given intravenously. If this is not possible, the antivenom should be administered intramuscularly (see later). Sprivulis and Jelinek[51] advise that multiple doses of antivenom may be required. Premedication with parenteral antihistamine and low-dose subcutaneous adrenaline (epinephrine) (0.003–0.007 mg/kg) prior to administration of antivenom is advisable in sensitive individuals to prevent anaphylaxis. Sprivulis and Jelinek[51] recommend that a short course of oral steroids may reduce the incidence of serum sickness, particularly in children and in patients receiving polyvalent or multiple doses of monovalent antivenom.
4. *Tourniquets:* In many countries the use of tourniquets is generally discouraged and under no circumstances should any form of bandaging be applied in the case of bites by snakes with cytotoxic venom as the local effects and subsequent complications are aggravated by compression and such procedures waste transport time. However, where highly venomous snakes with pronounced systemic effects are incriminated (e.g. neurotoxic cobras, mambas, crotalids and sea snakes), and especially if there are already signs of systemic toxicity, it is recommended that a pressure bandage be applied to the affected limb to prevent further absorption. Australian experience with elapid bites has much to recommend it – replacing the previously recommended tourniquet for the 'pressure-immobilization method.' Thus it has been found that for this group of snakes, rather than using a tourniquet, venom movement, which occurs largely via the lymphatics, can be effectively delayed for long periods by the application of a firm crepe bandage to the length of the bitten limb combined with immobilization by a splint. The bandage is firmly applied to the bitten area and then continued to the distal end of the limb and then wound tightly back to the groin or armpit.[50] It is worth noting here that this 'pressure-immobilization' method may be used for all elapid snake bites and some other types of bites or stings such as those from bees, wasps, or ants in sensitive subjects, funnel-web spider bites and bites of the blue-ringed octopus and the cone shell.[51,56,57,58] It can also be used for sea snake bites.[53] However, exceptions when the method should *not* be applied include ant, bee and wasp stings in normal, nonsensitive subjects, red-back spider bites and jellyfish stings[56] and, as stated earlier, after viper or spitting cobra bites. Where pressure immobilization is impractical (e.g. a bite on the body), then infiltration around the bite site with diluted adrenaline (epinephrine) is suggested by Sprivulis and Jelinek.[51]
5. *Suction:* suction apparatus is available in many commercial snake bite kits but is probably of doubtful value. Suction by mouth entails a definite risk of absorption of venom through the oral or intestinal mucous membranes. In general, suction is not recommended for snake bite.[45]
6. *Incision:* Incisions over fang marks as a first aid measure is not recommended.
7. *Other local measures*, such as freezing, injection of antivenom, EDTA or other agents into the bite and application of permanganate crystals to incised wounds, have no place in the management of snake bites, and can only serve to aggravate tissue damage.

Antivenom

Some 30 centers in different countries manufacture antivenom appropriate to local snake species. Polyvalent antisera generally cover most bites encountered, but in the case of certain snakes (e.g. boomslang) monospecific antisera are required. Antivenom is preferably given intravenously. In the past it has sometimes been suggested that a small intradermal test dose of antivenom be given. However, prevailing opinion is that, to quote Eddelston et al:[59] 'There is no point giving a test dose of antivenom as it poorly predicts the individuals who will have an anaphylactoid response.' Adrenaline (epinephrine) should always be at hand to combat possible anaphylaxis. However, adrenaline may induce serious cardiovascular effects, especially in adults. It is advisable to give a corticosteroid, such as hydrocortisone, prior to antivenom as this tends to modify serum reactions as well as having anti-inflammatory and antihypotensive effects. Corticosteroids will not, however, prevent anaphylaxis. In a recent Cochrane Review[60] based upon one trial, the authors concluded that 'routine prophylactic adrenaline for polyvalent antivenom known to have high adverse event rates seems sensible' but they believed that 'antihistamine appears to be of no obvious benefit in preventing acute reactions from antivenoms.'

Once a decision to administer antivenom has been reached, it must be given in adequate dosage.[61] Pearn[62] has commented that as many as 10–12 ampoules may be required to neutralize the pro-coagulant components of bites by the Australian Brown Snake. It must be emphasized that the dosage of antivenom for children is the same as that for adults.[45] Antivenom is best given in an i.v. drip, diluted in two to four times its volume of normal saline over a period of half an hour. A recommended initial i.v. dose is 50 ml of the polyvalent antivenom and the volume of diluent can be adjusted. This should be repeated every 4 h if clinical response is not satisfactory and if necessary up to 200 ml should be given within the first 24 h. The doses of the different antivenoms differ quite substantially. When large doses are used, steroids should be continued to modify possible serum sickness reactions. Anaphylactoid reactions generally respond quickly to prompt adrenaline (epinephrine) injection. If a doctor is not available, the usual dose of antivenom that can be tolerated intramuscularly is about 20 ml.

In the case of ophthalmia due to a spitting cobra, the affected eye should be well washed with water or other bland fluid. Instillation of dilute antivenom is not recommended (G. Muller, personal communication).

ELISA kits (CSL Diagnostics, Australia), suitable for field use, are available in some countries such as Australia for venom detection and species identification in snake bite washings, blood or urine.[51,61] Thus in Australia, it is recommended that snake bite wounds are *not* washed as part of initial first aid technique.[51]

Further supportive care

Unless it has been shown with the passage of some hours that the bite is trivial all cases of poisonous snake bite require admission to hospital.

If severe bulbar or respiratory paralysis has developed, airway suction, oxygen and assisted respiration are indicated.

Management of acute renal failure should be anticipated by adequate i.v. replacement, careful monitoring of input and output, urinalysis and measurement of plasma urea and electrolytes and tests for myoglobinuria (including rhabdomyolysis).

Hemorrhagic manifestations require careful appraisal with a coagulation profile as hemorrhagins (Russell's viper), fibrin degradation products (boomslang) or intravascular clotting (puff adder) can be responsible. Antivenom administration is the only treatment modality which is effective. Other supportive measures include: i.v. vitamin K, blood transfusion, fresh plasma or fibrinogen, low molecular weight dextran, or alpha-aminocaproic acid may be required according to circumstances. The use of heparin is not recommended.[49]

In the event of severe local swelling, the limb should be kept slightly elevated on a pillow. Bullae should not be burst as this increases the likelihood of infection. Skin or fascial release to ease jeopardized circulation, debridement of necrotic tissue, and subsequent grafting or amputation are not uncommonly required, especially in untreated puff adder bites.

The prime cause of death from snake bites is delay in getting the patient to a medical facility. Sutherland[63] in Australia and Gold et al[47] in the USA have emphasized that snake bite deaths are more common in children and the elderly and result from:

1. victims not being observed for an adequate period – suspected snake bite victims should be observed for at least 12–24 h after the bite;
2. antivenom being withheld despite clear indication of systemic envenomation;
3. giving the wrong antivenom – often more than once;
4. giving too little of the correct antivenom or not giving more antivenom if signs and symptoms reoccur.

Pearn[62] has pointed out that a chronic elapid envenomation syndrome may develop in patients who have not been given antivenom for whatever reason, after a snake bite. This may involve both local and systemic effects including lassitude, weakness, loss of appetite and nausea.

Prevention

To develop a snake bite prevention strategy for a particular locality, health care workers need to understand the epidemiology of snake bites for their particular area along the lines discussed for Papua New Guinea by Williams and Winkel.[48] However, general guidelines include:

1. Establish where your closest medical facility and local poisons information center are and have their telephone numbers readily at hand.
2. Treat all snakes with respect.
3. Endeavor to know the snakes in your area.
4. Watch out in summer months, particularly after first rains. Be careful when walking at night; use a torch.
5. Wear boots and leggings or at least shoes and socks when walking in the bush (80% of human snake bites are on the legs and 55% on the ankle and foot).
6. Avoid thickly bushed country, long grass, etc.
7. Do not panic and run away when confronted by a snake; movement will attract attention whereas the snake is likely to move off if you keep still.
8. Keep an appropriate anti-snake bite kit with you.

It has also been found important in Australia to emphasize the need to believe a child who claims to have been bitten by a snake.[64]

Detailed coverage of dealing with snake bites can be found in Brent et al[65] and the publications of Warrell.[49,66,67]

INSECTS, SPIDERS, TICKS, BEETLES, SCORPIONS, CENTIPEDES AND CATERPILLARS

Hymenoptera stings

All stinging insects, such as bees, wasps, hornets and ants, are included in the order Hymenoptera. Bee venom contains many toxic fractions, the most important being mellitin, which alters capillary permeability, causes local pain, hemolyzes red cells, and lowers blood pressure. The venom also contains antigenic components which are capable of invoking an allergic hypersensitivity response in a significant proportion of the population if subjected to a subsequent challenge. Cross-antigenicity may occur between wasp and bee stings and even on occasions, the rare stings by bumblebees.

In Australia, bee stings and bee sting allergy continue to be a major cause of venom-related mortality[68] and serious clinical problems are becoming more common owing to severe allergy to jumper ant (*Myrmecia pilosula*)[69] and bull ant (*Myrmecia pyriformis*) bites and stings.

The management of insect stings has been well reviewed by Reisman.[70] In general terms, uncomplicated stings require no treatment, apart from mild analgesics. Bee sting barbs should be carefully removed with a flat blade, taking care not to express further venom, which will happen if the sting is grasped with forceps. It has been claimed that meat tenderizer, available in most homes, applied in a dilute solution (a quarter teaspoon mixed with 1 teaspoon of water), rubbed into the sting denatures the protein and relieves all pain within seconds. In sensitive individuals, however, even a single sting may result in acute anaphylactic shock with urticaria, hypotension, tachycardia and sweating, glottic

edema, or bronchospasm. Prompt treatment is vital. Adrenaline (epinephrine) is indicated. In the case of laryngeal edema, hydrocortisone should be injected intravenously. Tracheostomy may be life saving in the event of severe edema of the glottis.

Skin tests to detect hypersensitivity to Hymenoptera stings are unreliable. However, children who are known to react in a hypersensitive manner should undergo desensitization with a carefully planned immunization schedule, using venom immunotherapy (VIT) – a point reiterated in a recent analysis of bee sting mortality in Australia.[68] Hymenoptera antigen immunotherapy may become more reliable with the development and use of pure venom immunization and phospholipase A, when and if it becomes available. However, children appear to exhibit considerably less frequent severe side-effects to stings than adults, which has led Valentine et al[71] to believe that immunotherapy is unnecessary for most children allergic to insect stings.

Multiple bee stings may induce a life-threatening toxic syndrome due to the cumulative effect of the toxins (short chain peptides, vasoactive amines and antigens). Bee venoms also possess hemolytic properties and multiple stings, usually in excess of 100, may result in significant hemolysis with acute anemia and subsequent renal failure, rhabdomyolysis and hepatic, respiratory and cardiac dysfunction. Cases of massive bee stings should be admitted to hospital and carefully observed for early signs of these complications, where prompt treatment can be instituted and renal failure minimized by ensuring a high urine output. Biphasic renal failure has been known to occur with early renal failure due to hemolysis and a second episode of azotemia about 10 d later, corresponding with a depressed serum complement C3 level and nephritic changes on renal biopsy – a phenomenon probably representing a serum sickness reaction caused by a large volume of foreign protein.

Studies in Australia have shown that 'Stingose' (an aqueous solution of 20% aluminum sulfate and 1.1% surfactant) is an effective, wide-acting first aid treatment to counteract the venoms of insects, marine invertebrates and plants when applied topically soon after the bite or sting.

The application of ice packs to insect stings (and platypus stings in Australia) will help to relieve local pain.[72]

Spider bites

Only a small fraction of the several hundred genera of spiders contain poisonous species and Alexander[73] has discussed these in detail. Although spiders belonging to the genera *Latrodectus* and *Loxosceles* can be dangerous to humans, most spider bites cause only minor clinical effects.[74] The topic is well covered in Brent et al.[65]

Latrodectism

Latrodectus mactans (the black widow spider of the USA), *L. indistinctus* (the button spider of South Africa), and *L. geometricus* (the brown widow spider – a cosmopolitan species) are the commonest species of the genus being widely distributed throughout the warmer areas of the world. In Australia, *L. hasseltii* (red-back spider) and in New Zealand, *L. katipo* (katipo spider) are found.[58,73]

Of the various species and subspecies of the genus *Latrodectus*, only the females are hazardous to humans. They have black or dark velvety globular bodies about 15 mm in length with orange–red markings often in the shape of an hourglass on the ventral surface of the abdomen. While they can be found under garden rocks, they tend to spin their webs in dark places, such as buildings (and especially outbuildings), garage doors, garden furniture and post boxes. Another favorite place is under lavatory seats – hence the number of bites which occur on the buttocks or genitalia. *Latrodectus* venoms (alpha-latrotoxin) possess neurotoxic properties, causing the release of peripheral neurotransmitters. Following a bite from *Latrodectus*, there is a very variable local reaction. Signs of systemic envenomation occur between 20 and 200 min later. There is often a regional lymphadenopathy within 30 min of the bite, followed by severe muscular pains involving the limbs and trunk with tightness around the chest and abdominal rigidity which may mimic an acute abdomen. Hyperreflexia is often present. Death is rare, even in untreated cases, but when it occurs, is usually due to respiratory failure. Pressure immobilization should *not* be used as the venom is slow acting and pressure will tend to increase the pain. A cold compress can reduce pain at the site of the bite. Treatment is aimed at relieving muscular spasm. It is worth noting, however, that only about 25% of people bitten progress to systemic envenomation, depending on the species of *Latrodectus* involved.[58] Calcium gluconate 10% (5–10 ml by slow i.v. injection) is effective in temporarily depressing the excited neuromuscular junctions. However, specific *Latrodectus* antivenom is the only treatment modality which will relieve pain and is available in most endemic areas.[61] The antivenom should be given intravenously (5 ml) and if necessary repeated. If the victim shows only a mild local reaction to the bite and no systemic effects are detectable after 24 h then antivenom should not be given.[63] The possibility of adverse serum reactions although uncommon should be borne in mind and adrenaline (epinephrine) and corticosteroids should always be at hand.

The effects of untreated Redback spider bites can persist for weeks to months – a picture similar to that seen in chronic fatigue syndrome.[70]

Loxoscelism

The genus *Loxosceles* (the violin spiders) includes many long-legged spiders occurring throughout Latin America as well as in focal areas elsewhere. The venom is cytotoxic and a bite is accompanied by severe local pain and bullous skin lesions, rapidly followed by marked edema, which may progress to necrosis. It may also induce systemic effects including hemolysis, coagulopathy and sepsis. Treatment is aimed at controlling the local reaction. Parenteral antihistamines have been shown to decrease both the pain and the swelling.

Funnel web and related mouse spiders

In Australia, the Sydney funnel web spider (*Atrax robustus*), and various species of the genus *Hadronyche* are the cause of serious spider bites each year. These are large aggressive spiders which rear up before attacking when disturbed and in this group of spiders, it is the male which is the more dangerous. They have a complex venom (robustoxin, an excitatory neurotoxin), and with multiple bites being the rule, considerable pain and panic ensue. In cases where systemic envenomation develops, it may occur within about 10 min of the bite, with dry mouth, circumoral paresthesia, salivation, nausea and vomiting. Muscular fasciculation is regarded by Raven and Churchill[75] as the unique characteristic of funnel web bites. Pulmonary edema and loss of consciousness may occur, and death can result from cardiac arrest.

In management of bites from this spider, atropine and diazepam are said to help.[73] Encouraging results have been obtained in the development of a funnel web spider venom antagonist.

It is claimed that the bite of several species of spider, such as the white-tailed spider *Lampona cylindrata* in Australia can cause chronic nonhealing skin ulcers,[58,76] but definitive evidence is lacking.[74] This condition is sometimes termed 'necrotic arachnidism' or 'necrotic araneism' although Warrell[74] is not happy with this terminology and feels it is misleading. Other spiders in Australia which are sometimes claimed to be dangerous, are the wolf spider (*Lycosa* spp.) and the mouse spider (*Missulena occatoria*).[58]

Tick bites: tick paralysis

Ticks are the vectors of a number of human diseases in both tropical and temperate regions. These include the tick bite fevers, certain arbovirus diseases and Lyme disease due to *Borrelia burgdorferi*.[77] However, as well as this role in the transmission of infectious diseases, dealt with elsewhere, tick bites may cause itching, irritation, can become secondarily infected and in some species, may cause paralysis.

Engorging ticks should be encouraged to detach themselves, by applying a lubricant such as liquid paraffin, before gently extracting them. They should never be hastily pulled off, as the tick's mouthparts may be retained in the skin. This may subsequently give rise to a granuloma composed of a dense dermal granulomatous reaction associated with overlying pseudoepitheliomatous hyperplasia, which on occasions may be so marked in biopsy material that it can lead the unwary pathologist to

an erroneous diagnosis of squamous carcinoma. On other occasions, the bite may lead to ulceration which is slow to heal. It remains covered by a necrotic, black eschar which takes many days to separate or the lesion may persist for months as a granuloma under the skin.[62]

Although rare, deaths from anaphylaxis following a tick bite are recorded.[62]

Certain ticks of the genera *Ixodes, Dermacentor, Haemaphysalis, Rhipicephalus* and *Hyalomma* produce a neurotoxin in their saliva which may cause 'tick paralysis'. The condition is commoner in children than in adults and particularly tends to afflict girls, probably because their longer hair hides the tick engorging on the scalp or neck, often with little or no local discomfort. A period of irritability starts 5–7 d after the tick has started feeding. This is followed by ascending symmetrical flaccid paralysis. Initially there is difficulty in walking and standing.[73] Within a day or two, paralysis spreads up from the legs to involve the trunk, arms and neck. Bulbar involvement causes dysphagia, slurring of speech and may result in death from respiratory failure. A local paralysis of the face, for example, may result when the tick is attached to the eardrum. Sensory changes are minimal although there may be paresthesia in the paralyzed limbs. The cerebrospinal fluid remains normal. Mortality may be significant, although death is uncommon if the engorging tick is removed.[73] Tick paralysis should be considered in the differential diagnosis of Guillain–Barré syndrome and it can mimic poliomyelitis.[73] Rapid and complete recovery usually attends the removal of the offending tick, although sometimes neuroparalysis may become transiently worse after removal of the tick. In Australia a canine tick antivenom is available[61] and has been used in children with promising results.

Beetles

Two large families of beetles, found in many parts of the world, produce urticating toxins. These are the Staphylinidae (rove beetles) and the Meloidae (blister beetles).

In Africa, and parts of Asia, America and Europe, rove beetle dermatitis due to the genus *Paederus* poses a difficult problem. When the beetles are brushed off the skin, or crushed, an irritant toxic principle, paederin, is released. This may cause blistering 1–2 d later. The blisters vesicate in 2–8 d and have a tendency to spread as a result of the release of fluid. Thereafter, the lesions flatten and dry out with subsequent peeling. On occasions the blistering is accompanied by systemic symptoms such as headache, fever, myalgia and arthralgia. A severe conjunctivitis, commonly known as 'Nairobi eye' results if paederin comes into contact with the eyes.

Several genera of Meloidae, including *Lytta vesicatoris*, the 'Spanish fly', produce a vesicant, cantharidin, which is falsely credited with aphrodisiac properties, but which is toxic if ingested or absorbed through the skin.

In the treatment of dermatitis due to rove or blister beetles topical application of compresses, such as magnesium sulfate is beneficial, and eye lesions should be bathed with isotonic saline.[73] Additionally the skin should be thoroughly washed with soap and water as should all contaminated clothing.

The condition termed 'Christmas eye' in Australia is as yet of undetermined origin. It may prove to be due to an orthopteran of some type, but a blister beetle seems more likely.

Scorpion stings

Scorpions have a single caudally placed sting with which they can inject venom. Some varieties, such as the genera *Parabuthus* (Southern Africa), *Androctonus* (North Africa; Turkey), *Centruroides* (Arizona; Texas) are dangerous especially to young children. Scorpion venoms are neurotoxins and their overall effect is hyperactivity of peripheral nerves, both somatic and autonomic. Alexander[73] notes that scorpion stings are more serious in children than in adults and that most deaths occur in children.

Serious stings are characterized by severe burning pain and local swelling at the site of the sting, hyperesthesia and myalgia in the affected limb, quickly followed by generalized weakness, muscle spasm, excessive salivation and rhinorrhea, excitement, coma, convulsions (especially in small children) and respiratory failure – often fatal.[73] Pancreatitis is a reported complication in severe cases of scorpionism. Less venomous varieties or stings in older subjects may result in only local pain and swelling, sometimes with lymphangitis.

Treatment

In many countries, it has been noted that scorpion stings in children can have a high mortality rate unless adequate symptomatic and specific therapy are given – especially respiratory support. Prompt application of a crepe bandage will slow the spread of venom but there may be problems in applying this to children because of the pain. Ice packs applied to the sting site are seldom effective and the most effective method of pain relief is infiltration of the sting area with local anesthetic (Muller, personal communication). Scorpion antivenom is available and this, together with respiratory support, can be life saving in cases of severe envenomation; 10 ml should be given intravenously, as soon as possible, keeping adrenaline (epinephrine) and corticosteroids on hand in case of anaphylaxis. When antivenom is not available, general supportive treatment is needed and subcutaneous atropine and i.v. calcium gluconate have been reported to be effective. *The need for assisted respiration must be anticipated.* In milder stings injection of a local anesthetic into the site is often the only treatment needed. The topic is covered by Brent et al.[65]

Centipedes

Centipedes are segmented arthropods with one pair of legs per segment. The first pair of legs are modified to form toxognaths with strong claws on which open the ducts from the venom glands. Bites from centipedes can cause considerable pain and swelling, and sometimes local ulceration and spreading lymphangitis result.

Caterpillars (lepidopterism/erucism)

Lepidopterism refers to a disease caused by contact with adult or larval butterflies and moths, while erucism is an illness caused by the larval and pupal stages of these insects.[78] The fine, spiny, venom-containing hairs of some species of caterpillar induce a very painful urticarial or vesicular dermatitis. This may be acquired by actual contact with the larva or from hairs blown in the wind. Where caterpillars are very prolific, severe symptoms may occasionally be induced in children, with extensive rashes, fever, vomiting and even paralysis. Hairs are best removed by applying adhesive tape to the site. Immersion in a very hot bath is soothing, and analgesics may be required. There is little evidence for the development of an allergic response from repeated exposure. Severe clinical effects including mild to severe shock and coma can follow contact with the stinging hairs from caterpillars of the genus *Megalopyge* in the southern USA and parts of Latin America.[73]

Calamine lotion applied to caterpillar hair stings helps alleviate the pain and removal of stinging hairs with sellotape stripping is suggested as a first aid measure.[73]

STINGS FROM VENOMOUS MARINE ANIMALS

Stings from venomous marine animals can be a major health problem in many parts of the world, including Australia, Papua New Guinea, South-East Asia, Asia and the Far East, the Mediterranean, North and Latin America, the Caribbean and even Russia.[79] As might be expected, the problem is greatest in tropical waters and its magnitude can be gauged by figures from Australia where, over the summer months of 1990–1991, more than 18 000 people were treated for marine stings[80] while in the USA, in Chesapeake Bay alone, about 500 000 jellyfish envenomations occur each year.[81]

Although mostly seasonal, the problem of marine stings in the waters of northern Australia is so significant that it often precludes swimming during the summer months, except in areas protected by special nets – termed 'stinger enclosures'.

Overall, the most common cause of jellyfish stings are coelenterates of the genus *Physalia*, the common blue bottle or Portuguese

man-of-war. Stings from these animals occur in the water or on the beach and, especially with the short-tentacled species, can be painful for up to an hour or so, but are not usually life threatening.[72] Stings from the multiple-tentacled species, however, can be severe and rare fatalities are on record.[72,80]

The so-called 'nematocyst dermatitis' from the blue bottle jellyfish is often exacerbated by the tentacles sticking to the skin, with subsequent discharge of the nematocysts causing ongoing pain and discomfort.

Treatment of blue bottle stings consists of covering the affected area with a plastic bag containing ice to alleviate the pain.[72] The use of heat to reduce blue bottle sting pain is more controversial, needing further research but is not really practical in the first aid situation.[82]

By contrast, stings due to the seawasp or box jellyfish (*Chironex* spp.) are excruciatingly painful and are associated with a significant mortality. Thus stings by this species in northern Australia have resulted in over 300 cases between 1984 and 1994, with in excess of 80 deaths being recorded from these stings in the twentieth century in Australia – often only minutes after the sting has occurred in the water.[76,83]

The bodies of people stung by this jellyfish are often covered with long red wheals which, if the victim survives, can result in permanent scarring.[72] Reactions to the sting may, however, in some cases, be delayed for some time.[80] The toxin of this species has three modes of action – it damages the skin, attacks the red blood cells and, most importantly, can affect the heart and respiration.[76,83]

The treatment for *Chironex* stings is the immediate application of vinegar by pouring it liberally over the affected area to prevent further nematocyst discharge. Irrigation with fresh water is contraindicated as it will cause further nematocyst discharge. In severe cases, CPR can be life saving[83] and the use of a specific *Chironex fleckeri* antivenom produced by the Commonwealth Serum Laboratories in Melbourne, Australia is beneficial[5,61] but may need to be given within minutes and possibly at high doses to be effective.[82] The recommended dosage should not be reduced for children.[80] Fenner[82] has suggested that the use of antivenom with magnesium might be worth considering if the antivenom alone fails.

It is worth reiterating that vinegar is essential to inhibit the discharge of further nematocysts of *Chironex* but will not reduce pain and, paradoxically, may initially increase pain. Compression-immobilization bandaging, once thought to be helpful,[53,84] is now not recommended.[82]

In Australia, a third type of jellyfish sting is the so-called 'Irukandji sting syndrome' caused by a minute carybdeid jellyfish (*Carukia barnesi*) and a number of other species;[82] this sting can result in severe joint, low back, and trunk pains; muscle cramps; anxiety; headache; shivering; sweating; hypotension and cardiac involvement.[53,76,82,83,85] Stings usually occur in the water in the afternoon and the jellyfish, being so small, is often not seen.

In Irukandji syndrome, initial treatment with vinegar should be used to prevent further envenomation and this should be followed by treatment for the pain using ice packs.[82] The use of 20 mg i.v. furosemide (frusemide) proved beneficial in one case.[80] Immediate transfer to hospital for intensive care treatment is advisable. The use of sublingual nitrate sprays, topical analgesia (topical Fentanyl) and i.v. magnesium sulphate are all discussed by Fenner.[82]

Other jellyfish known to cause stinging of humans include the large carybdeids ('firejellies') in Tomoya or Morbakka stings; the 'hairjelly' or 'sea blubber', *Cyanea*, and *Pelagia noctiluca*, the 'mauve blubber'.[72,83]

There are, of course, a large number of other potentially dangerous marine animals, including stingrays, the blue ringed octopus, sea urchins, starfish, stonefish and lion fish amongst others.[79] The management of such a diverse range of envenomations is obviously beyond the scope of this chapter, but the topic has been well reviewed by Williamson et al,[79] Fenner,[82] Pearn,[83] Auerbach[86] and is covered in detail in Brent et al.[65]

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SUDDEN INFANT DEATH SYNDROME

Sudden unexpected infant deaths have been recognized since antiquity, but it was not until post-neonatal mortality rates substantially fell in the Western world during the early part of the twentieth century that greater attention was paid to the phenomenon of unexpected and unexplained deaths in apparently healthy infants.

Sudden infant death syndrome (SIDS) was proposed in 1969 as a descriptive term for those infant deaths that were unexpected and remained unexplained after thorough investigation. In 1994 a more precise definition of sudden infant death syndrome was proposed.[87]

The sudden death of an infant, which is unexplained after review of the clinical history, examination of the circumstances of death, and postmortem examination

In 1892 Templeman,[88] and in 1904 Willcox[89] noted the excess of unexpected infant deaths in the poorest families, and agreed that the majority of such deaths were due to accidental overlaying whilst bed-sharing, recommending that parents be encouraged to use cribs for their babies to sleep in. One hundred years later the American Academy of Pediatrics made a similar recommendation, though the evidence for this has been the subject of wide debate.[90–92]

EPIDEMIOLOGY OF UNEXPECTED DEATHS

Diagnosis of SIDS and subsequent decline in the SIDS rate

The diagnosis of SIDS is unique in that it is not a cause of death but rather a diagnosis of exclusion, arrived at only after thorough investigation. Only when recognized causes of infant death have been excluded can the death be labeled SIDS and there are valid concerns that such labeling could attribute too much homogeneity to what might be disparate causes of death.[93,94]

From the 1950s to the late 1980s the number of unexpected and unexplained deaths in the UK was probably between 1400 and 2000 per year, giving a rate of around 2/1000 live births.[95] In the late 1980s epidemiological evidence from several different countries[96–101] suggested that SIDS could be related to infants sleeping in the prone position. In 1991 the 'Back to Sleep' campaign was initiated in the UK to encourage parents to avoid placing their infants on their front and the SIDS rate fell from a peak of 2.3 deaths/1000 live births in 1988 to 0.7 per 1000 live births in 1994 (Fig. 6.2). Similar dramatic reductions have since been observed in many other countries following such an intervention campaign. The possibility that other modifiable risk factors might be amenable to further interventions in this mysterious group of conditions has led to multiple epidemiological studies of the residual deaths. Further identification of other unsafe infant care practices, particularly within the sleep environment have led to additional amendments and revision of the initial campaign message and probably helped to reduce the rate further over the last 10 years to 0.4 per 1000 live births. This equates to the prevention of over 10 000 infant deaths in England and Wales since the campaign was first launched, and more than 100 000 worldwide.

The fall in numbers of deaths has been accompanied by several major changes in the epidemiologic characteristics of SIDS,[102] most notably an increased proportion of the deaths occurring in deprived families and whilst bed-sharing. Some pathologists are reluctant to use the label 'SIDS', preferring to use the term 'unascertained', when parents have consumed alcohol or illegal drugs or the circumstances of death raise the unproven possibility of overlying.[103] Such practices emphasize the importance of a detailed multiprofessional review in establishing the final allocated 'cause' of unexpected infant deaths (see later).

Epidemiologic features of SIDS

Prior to the 'Back to Sleep' campaign in 1991, many epidemiologic characteristics of SIDS had been described. Unexpected, unexplained deaths of infants occurred in all cultures but the incidence varied widely. There were relatively fewer SIDS deaths in several Asian cultures but more deaths amongst certain indigenous populations such as Maoris, Australian Aborigines and Native Americans. The incidence in the UK

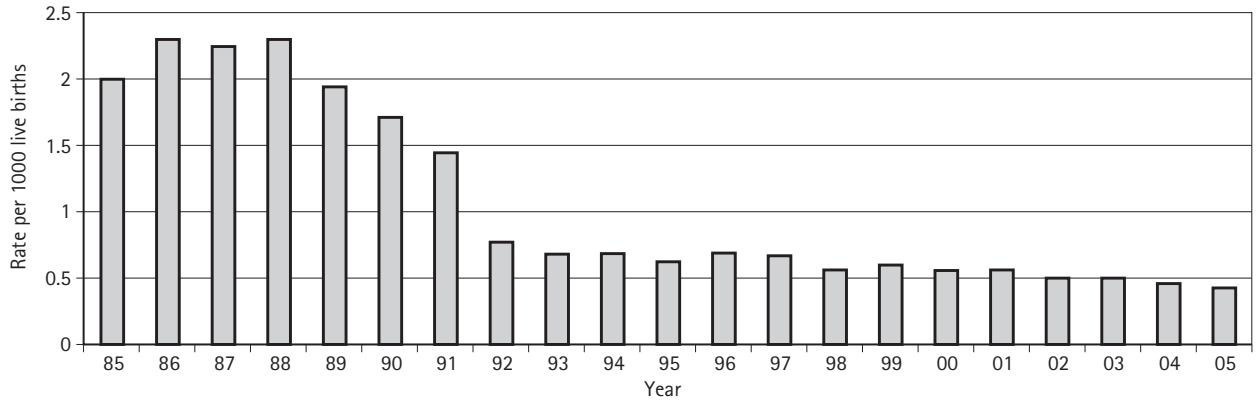

Fig. 6.2 SIDS rate in England and Wales 1985–2005 (Office for National Statistics and the Foundation for the Study of Infant Deaths).

was lower than in the white populations of New Zealand and Australia but higher than in the Nordic countries. The majority of deaths occurred within the first 6 months of life, with a peak around the third and fourth month. Many of the deaths occurred during the night-time sleep periods, and although some studies noted an excess of deaths at weekends this has not been consistent.[104–108] More deaths occurred in males and in winter months. SIDS occurred in all social strata but was more common in the socioeconomically deprived groups, particularly if parents smoked. Many of the SIDS infants had lower birth weight, shorter gestation and more perinatal problems. There was a strong correlation with young maternal age and higher parity and the risk increased with multiple births, single motherhood or a poor obstetric history.

Many of the risk factors associated with SIDS are also closely associated with other infant deaths; only the characteristic age distribution and high prevalence of tobacco exposure distinguished SIDS infants from infants who died suddenly and unexpectedly from identifiable causes.[109] Deaths from congenital malformations decrease steadily from early age whilst deaths from respiratory or infectious diseases remain relatively constant over the first year of life.[110] Overall, infant mortality rates are highest in the first month after birth, when infants are at their most vulnerable.[111,112] However few SIDS deaths occur in the first month, with a peak occurring at 3–4 months and a steady decline thereafter. Whilst smoking is most prevalent amongst mothers in the more disadvantaged socioeconomic groups, the incidence of smoking is higher amongst the mothers of SIDS infants than matched controls in all social groupings.[108,113]

It is perhaps in the infant sleeping environment that the epidemiological study of SIDS has had the most success. Prone sleeping was actively encouraged in some Western countries in the 1960s and 1970s[114] to improve infant posture and skeletal growth,[115] prevent flattening of the skull[116,117] and avoid the perceived risk of aspiration in the supine position.[118] This was also a time when neonatal intensive care units were expanding and apparent benefits of using the prone position were found amongst pre-term infants including less apnea, better gastric emptying, better oxygenation, and more effective ribcage and abdominal coupling with decreased work of breathing.[119–122] What was best in the early neonatal period for the relatively small number of pre-term infants however was not necessarily beneficial for the rest of the infant population or beyond the immediate neonatal period. Historical references to infant sleeping position in art and early medical texts suggest that very few if any infants were placed prone to sleep before the twentieth century.[123]

In the 1980s a number of population-based studies identified the prone sleeping position and heavy wrapping and/or warm environments as major risk factors associated with SIDS.[101,124–126] Gilbert found that the combination of viral infection and heavy wrapping was associated with a high relative risk[124] whilst in a study from Tasmania Ponsonby[125] found the risk from prone position was potentiated by overnight heating, swaddling, recent infection and mattress type. Williams confirmed these findings in a study from New Zealand and found a small additive effect if the mother smoked.[127]

'BACK TO SLEEP' CAMPAIGNS

Although positioning and wrapping were not sufficient to fully explain the death they could be linked to some causal chain of events and intervention campaigns to advise parents against these practices were instigated in many countries from 1990 onwards. In all countries in which risk reduction campaigns were conducted, a fall in infant prone sleeping was followed by a fall in SIDS rate. Also publicized in some campaigns was the potential risk of heavy wrapping. Studies of control infants in Avon before and after the 'Back to Sleep' campaign[124,128] showed that the thermal resistance (tog value) of bedding and clothing with which normal infants were usually covered fell by almost half after the 'back to Sleep' campaign, and the winter peaks of SIDS deaths have almost disappeared in the UK.

EPIDEMIOLOGIC CHARACTERISTICS SINCE THE FALL IN NUMBERS OF SIDS

Distal factors

A longitudinal study conducted in Avon from 1984 to 2003[102] showed that amongst SIDS families the proportion from the most disadvantaged socioeconomic groups rose from 47% to 75%. This change in the socioeconomic distribution of SIDS families was accompanied by an increased proportion of single mothers, younger mothers, mothers who smoke and lower birth weight infants. The prevalence of maternal smoking during pregnancy amongst SIDS mothers (80–90%) was twice the level expected amongst control mothers with similarly deprived socioeconomic backgrounds,[113] lending support to the hypothesis that infant exposure to tobacco smoke is some part of a causal mechanism. There is a clear increase in risk of SIDS with increasing levels of exposure to tobacco smoke, both in utero and after birth[129–131] and a recent review by Mitchell and Milerad suggests this risk has grown despite advice against smoking in almost all risk reduction campaigns.[132] In recent studies over one third of SIDS victims were preterm, compared to a UK population prevalence of 5% for preterm delivery. For such infants the effects of other risk factors in combination with the increased risk from prematurity leads to very high risk (e.g. for preterm or low birthweight infants put down on the side: OR = 9.13 [95% CI 4.93–16.90], and for those put down prone: OR = 62.8 [95% CI 12.06–327]).[133]

The previously recognized increase in risk of SIDS with increasing birth order may be changing. The longitudinal study from Avon suggests that SIDS is now most common amongst first-born infants.[102] Several studies have now shown no evidence that immunization is associated with an increased risk of SIDS, and some evidence that the risk may be reduced.[134]

Proximal factors

New evidence on risks within the infant sleeping environment has changed some of the advice now given to parents.

Risk of positioning infants on the side to sleep

Before the 'Back to Sleep' campaign few studies had looked at the use of the side sleeping position and the findings were inconclusive;[126,135–137] the side position with the lower arm extended to avoid infants rolling on their front was suggested as a safe alternative to supine sleeping. More recent studies[128,138–142] show the side sleeping position carries a significant risk, partly because the position is unstable, and some infants who roll from side to prone have difficulty extricating themselves from this position. Certain infants with abnormalities of the upper airway (e.g. Pierre Robin syndrome) may experience airway obstruction if placed supine, and some may benefit from side or occasionally prone positioning for sleep, but many can be safely placed supine. Whilst gastro-esophageal reflux is slightly reduced in the prone position compared to supine, the increased risk of SIDS means that this position should not be used to treat reflux unless this is causing severe symptoms (e.g. growth failure or recurrent aspiration) that have not responded to alternative treatments. Apart from these uncommon conditions, in most countries the only recommended sleeping position for infants is supine. In some countries use of the side position may have increased despite knowledge of its potential risks,[143,144] many parents and health care professionals citing either outdated SIDS guidelines or fear of aspiration, cyanosis or apnea when the infant is placed supine.[145–148] These concerns are not supported by findings from either pathology or epidemiology. A review of 196 infant deaths in South Australia found evidence of aspiration of gastric contents into the airways and alveoli of three infants, all of whom were found face down in the prone position.[149] Similar findings linking aspiration with the prone rather than the supine position have been found in the UK,[150] whilst a large cohort study of over 8000 surviving UK infants showed no association between the prevalence of vomiting and infant sleeping position.[151] A recent study from New Zealand[152] has linked the increase in sleeping supine with nonsynostotic plagiocephaly, recommending that parents should vary the infant head position when putting them down to sleep and to give their infants 5 min of supervised 'tummy time' each day. This may also help reduce the risk of 'unaccustomed prone' position, when infants roll into or fall asleep in this position for the first time. In studies in Australia, the US and the UK, supine sleeping was not linked to apnea or cyanosis and no demonstrable increase in symptoms or illness amongst supine sleeping infants was found.[151,153,154]

Studies in the UK[155] and in Canada[156] have shown minor differences in gross motor skills in early infancy with infants who usually sleep supine, showing slightly slower developmental progress than those who sleep prone. In the UK study these differences had disappeared by age 18 months, but in the smaller Canadian study the differences, though small were still present at 15 months.

Both the minor developmental disadvantages and the risk of positional plagiocephaly from supine sleep position can be largely prevented by the use of periods of 'tummy time' – placing infants prone for regular periods when awake and supervised.[157]

Risk of soft sleeping surfaces

Soft surfaces for infant sleep have been associated with an increased risk of SIDS[108,158,159] and there is some evidence that this risk is even higher in combination with established risk factors such as the prone sleeping position[160,161] and heavy wrapping or warm environment.[162] Pillows, cushions and bean bags have been used not just as a sleep surface but also as a prop to maintain the body position of a sleeping infant or provide easier access to bottle feeding. This practice presents the additional risk, even to supine sleeping infants, of such objects potentially covering the external airways.[163] This includes the adult size V-shaped pillows used to facilitate breastfeeding.[164] The current advice is to sleep infants on a firm mattress and away from soft objects.

Risk of bedding covering the infant

It is not uncommon for SIDS infants to be discovered dead with bedclothes covering the head and face, indeed 'accidental mechanical suffocation' was, prior to the introduction of the term 'sudden infant death syndrome', a term commonly used to describe these deaths, despite a lack of any evidence of suffocation or asphyxia.[165–169] Uncontrolled observations from early studies[170–173] that around one fifth of SIDS infants were found with bedding covering the face or head were attributed to the agonal struggle just prior to death. Subsequent findings of reduced arousability during the sleep of SIDS infants,[174] observations of undisturbed bedding[175] and lack of such a struggle during recordings of several SIDS infants who died whilst on a monitor[176] do not support the idea that head covering is just a consequence of the terminal event. Whilst postmortem examination cannot distinguish between the possible mechanisms of airway obstruction, re-breathing or thermal stress, over 20% of SIDS victims are found with bedding over the head,[102,140,142,161,170,177–180] ten times more than the incidence amongst live age-matched controls and highly significant even after adjusting for other risk factors. Studies have linked head covering to loose bedding, infant movement down under the covers and the use of duvets or quilts.[102,108,181–183] In 1997 a 'Feet to Foot' campaign, was launched in England and Wales by the Foundation for the Study of Infant Death to encourage parents to tuck the bedding in firmly, avoid using duvets or pillows and place the feet of the infant at the foot of the cot. This approach was subsequently endorsed by the American Academy of Pediatrics.[90,184]

Risk associated with unobserved sleep

Despite a complete absence of supporting evidence, many childcare 'experts' in the 1950s to 1990s recommended that infants should sleep in a room separately from parents.[185,186] Throughout history most human infants slept in a consistently rich sensory environment with close and continual contact between mothers and babies and the solitary sleep experience of Western societies was a recent development.[187] Reports from New Zealand and the UK showed that the risk of SIDS was lower if infants shared a bedroom with parents,[188–190] and further analysis of the UK data suggests that parental supervision for day-time sleeps is equally important.[191] Parental presence during infant sleep does not guarantee the infant would be constantly observed, nor, indeed that parental intervention would prevent death from occurring. However having the sleeping infant nearby during the day may alert parents to circumstances such as young infants rolling from the side to the prone position or bedclothes covering the infant head or face.

Risk associated with bed-sharing

Unexpected infant deaths can occur in any sleep environment. Recent case–control studies show that up to half of the deaths occur whilst infants share a sleep surface ('co-sleep') with an adult,[142,161,192,193] a marked rise from studies in the 1980s. This proportional rise in co-sleeping SIDS deaths has led some authorities, including the American Academy of Pediatrics[90] to recommend against bed-sharing.

Longitudinal data from Avon over the last 20 years shows that although the *proportion* of bed-sharing deaths rose from an average of 16% of all SIDS deaths prior to the 'Back to Sleep' campaign to 34% after the campaign, the *number* of bed-sharing SIDS deaths fell (by 50%), but this is less marked than the 80% fall in deaths occurring in the cot.[102] More worrying is the rise in both prevalence and number of SIDS infants found after sleeping with a parent on a sofa, which carries a markedly increased risk.[128,142,190] At least some of these deaths occurred when mothers inadvertently fell asleep whilst feeding on a sofa during the night.

Bed-sharing is perceived to be and is treated as a risk factor in the field of SIDS epidemiology and when considered in this rudimentary way there is ample evidence to advise against such a practice. On closer inspection however there are several things to be considered. Adjusting for potential confounders specifically associated with the adult co-sleeping environment such as recent alcohol consumption, sleep deprivation, overcrowded conditions and adult-sized duvets renders bed-sharing nonsignificant as a risk factor suggesting it is not bed-sharing itself but the particular circumstances in which bed-sharing occurs that puts an infant at risk.[190] An intriguing aspect of this debate is that in certain Asian cultures where particular forms of mother–infant co-sleeping (sleeping on futons) is common such as Japan[194] and Hong Kong[195] the cot death

rates are very low; corresponding to findings in the Bangladeshi[196] and other Asian[197] communities in the UK and the Pacific Island communities in New Zealand.[198] Another aspect is that of generalization: the majority of bed-sharing SIDS mothers smoke whilst the majority of bed-sharing mothers in the population do not. The magnitude of any increase in risk for nonsmoking breast-feeding mothers who are bed-sharing on a firm flat surface, and who have not taken alcohol or other drugs, is unclear, but certainly small.[142,190,199–201] There is also the wider debate beyond the field of SIDS in terms of the potential advantages associated with bed-sharing. Before the last century and in most nonWesternized cultures today the normative practice is for the mother to share a sleep surface with the infant.[202] Postulated, but largely unproven potential physiological benefits of close contact between infants and care-givers include improved cardiorespiratory stability and oxygenation, fewer crying episodes, better thermo-regulation, an increased prevalence and duration of breast-feeding, and enhanced milk production.[203,204]

It is becoming clear from recent studies that bed-sharing both for infants and mothers results in complex interactions which are completely different to isolated sleeping and which need to be understood in detail before applying simplistic labels such as 'safe' or 'unsafe'.[205–207] The unusual level of criticism and hostility generated by the recent Policy Statement by the American Academy of Pediatrics against bed-sharing[90–92] is a testament to the current polarized debate. Current advice in the UK does not advise against bed-sharing but describes particular circumstances when bed-sharing should be avoided. Co-sleeping with an infant on a sofa should always be avoided.

Apparent protective effect of infant pacifier use

The current debate on bed-sharing holds many parallels with the debate on dummy use (pacifiers). Several studies have examined the prevalence of infant dummy use and shown a reduced risk for SIDS.[142,161,193,208–213] Actively encouraging dummy use, like the advice on bed-sharing, has been met with criticism mainly concerning the potential adverse effects on breast-feeding. The evidence of a significant association is not in dispute but whether this association is causal in itself is still being debated.[91,214,215] The mechanism by which a pacifier might reduce the risk of SIDS, or by its absence increase the risk, is unknown, but several mechanisms have been postulated. These include avoidance of the prone sleeping position, protection of the oropharyngeal airway, reduction of gastroesophageal reflux through non-nutrient sucking[209] or lowering the arousal threshold.[216–218] These mechanisms however assume the presence of a pacifier in the infant's mouth but the evidence suggests pacifiers generally fall out within 30 minutes of the infant falling asleep[219] whilst many of the night-time deaths are thought to occur much later during the sleep.[191] Alternatively dummy use may be a marker for some protective factor that has eluded measurement. The physiology not only of infant dummy use, but also non-use amongst routine users and infant thumb sucking, which leads to identical physiological effects but is inhibited by pacifier use[220] deserves further investigation.

Before recommending the use of pacifiers the potential disadvantages must be considered. There appears to be a clear relationship between frequent or continuous pacifier use and a reduction in breastfeeding,[221–224] and a significantly higher risk of otitis media and oral yeast infection.[225–228] Other potential disadvantages include accidents (airway obstruction[229]), strangulation by cords tied to the dummy,[230] eye injuries[231] and dental malocclusion.[232] The current advice in the UK is no longer to discourage the use of dummies but falls short of recommending them as a preventive measure against SIDS.

INFANT PHYSIOLOGY AND PATHOPHYSIOLOGY OF UNEXPECTED DEATH

Whilst the final sequence of events leading to death is not known for the great majority of unexpected infant deaths, and there is no reason to presume that there is a single mechanism involved, a number of studies have been published of unexpected infant deaths that have occurred whilst the infant was undergoing physiological recordings.[176,233] These recordings have shown a range of physiological events leading up to the final collapse and death, but in some infants there was an initial period in which there was normal respiratory activity but a relative tachycardia. In several infants the final event was one of profound bradycardia, with respiratory activity continuing until a late stage. In many of these recordings, despite the carers having been alerted to the bradycardia by audible alarms, and having attempted resuscitation, this was not successful. This sequence of events is more suggestive of a cardiovascular rather than a respiratory event as the primary trigger for the final collapse. One possible physiological explanation for such a pattern might be a catastrophic fall in blood pressure as a consequence of sudden peripheral vasodilatation, e.g. in response to toxins or as a consequence of heat stress.[234]

Several population-based case–control studies have shown that infants who died unexpectedly were more heavily wrapped and more likely to be sleeping in warm rooms than age and community-matched controls.[108,101,201] The increased risk of SIDS from heavy wrapping was greatest for the older infants (more than 3 months of age), and was especially high for those infants with evidence of an acute viral upper respiratory tract infection.[124] In a study of the metabolic response to acute viral upper respiratory tract infection, younger infants (less than 3 months of age) commonly showed a fall in metabolic rate with infection, whilst those over 3 months usually showed an increase, commonly accompanied by fever.[235] The metabolic rate of infants during sleep rises over the first few months after birth, such that by 3 months of age healthy infants excrete up to 50% more heat per unit surface area than in the first week after birth.[236,237] Thus infants over 3 months of age might be more at risk from heavy wrapping that compromised their ability to lose heat, particularly at the time of an acute minor viral infection.

In a population-based observational study of infant thermal care at home most mothers accurately achieved conditions of predicted thermal neutrality for their infants, but younger mothers, those who smoked, and those who did not breast-feed were more likely to wrap their infants more heavily.[238] In a prospective longitudinal laboratory study of mothers and infants sharing a room or sharing a bed for overnight sleep, despite a much warmer microenvironment, infants thermoregulated more effectively, with a slightly greater diurnal fall in rectal temperature when bed-sharing with their mother than when sleeping in a cot adjacent to the mother's bed[207].

The development of the diurnal fall in core temperature occurs at ages between approximately 3 and 4 months, occurring earlier in girls and breastfed infants than in boys or bottle fed infants.[239]

Blackwell and Morris have each shown the potential importance of toxigenic Staphylococci as contributory agents to circulatory collapse and sudden death in infancy.[240,241] Toxin production by such Staphylococci increases with increasing environmental temperature and is minimal below 37 °C.[240] SIDS victims have increased nasopharyngeal colonization with staphylococci compared to healthy age and community-matched controls.[234] In the prone position, or with head covering (particularly in the presence of potential re-breathing), nasopharyngeal temperature is likely to rise above the normal value of 32 °C, with resultant increase in toxin production by any toxigenic staphylococci present on the mucosal surface.[242] Transmucosal absorption of toxin might thus lead to circulatory collapse and death without the need for invasive infection to occur.

Elevated levels of interleukin 6 (IL-6) in the cerebrospinal fluid of SIDS victims compared to age-matched controls dying of known causes raised the possibility of a vigorous pro-inflammatory response being part of the pathophysiology of SIDS.[243]

Drucker has recently shown that common polymorphisms, leading to high levels of pro-inflammatory cytokines (e.g. IL-6, VEGF) or low levels of anti-inflammatory cytokines (e.g. IL-10) are associated with increased risk for unexpected deaths in infants.[242] A high pro-inflammatory response to infection, with vigorous sympathetic activity including peripheral vasoconstriction and pyrexia might indirectly lead to further toxin production in the nasopharynx.

The relationship between the pro-inflammatory cytokine IL-1β and the risk of SIDS is complex, and Moscovis et al[244] have shown potentially important ethnic differences in the patterns of gene polymorphisms. In both Aboriginal Australian and Bangladeshi infants a particular polymorphism (TT) is found, which is uncommon in infants of European origin. This polymorphism is associated with a marked increase in IL-1β production, and increased pro-inflammatory responses on exposure to tobacco smoke. This may partially explain the major difference between Aboriginal Australian infants with high maternal smoking rates and high SIDS rate, and Bangladeshi infants, who are genetically similar with regard to IL-1β, but have very low rates of maternal smoking and very low SIDS rates.

The potential interaction between genetic and environmental factors is further exemplified by the anti-inflammatory cytokine IL-10, production of which is markedly decreased by exposure to tobacco smoke.[244]

Associations have also been described between the risk of SIDS and polymorphisms of genes involved in the development of the autonomic nervous system,[245] various cardiac channelopathies,[246] and the serotonergic system in the brainstem.[247] This latter group is of particular interest in the light of recent histological evidence of abnormalities of serotonergic neurons in the brainstem of SIDS victims.[248]

'TRIPLE RISK' HYPOTHESES AND PROSPECTS FOR PREVENTION OF SIDS

There is considerable evidence that SIDS represents a possible consequence of a wide range of genetic/developmental/environmental interactions.

The 'triple risk' hypothesis – which envisages SIDS occurring as a result of a final insult (one which is not usually fatal on its own) that affects a baby with an intrinsic vulnerability (arising from genetic or early developmental factors), at a potentially vulnerable stage of physiological development (e.g. immunological, respiratory, cardiovascular, thermal), has been proposed in various forms by a number of authors over the past 15 years.[249]

The recent developments in our knowledge of environmental, immunological, genetic and physiological factors in infants, and recognition of the changes in all these systems that occur during the first few months after birth as outlined earlier strongly support a 'triple risk' model of causation for most unexpected infant deaths, including some for which a partial or even a complete 'explanation' can be identified on thorough investigation.

This approach to understanding the pathophysiological processes that may contribute to unexpected infant deaths holds great promise for targeted interventions to further reduce the number of such deaths.

INVESTIGATION AND CLASSIFICATION OF UNEXPECTED INFANT DEATHS

The process of investigation after any unexpected infant death should seek to collect as much information as possible about factors that may have contributed to the death, in order to help understand (and in future possibly prevent) such deaths. It is essential however that the investigation is conducted with both thoroughness and sensitivity, bearing in mind that whilst the great majority of such deaths are natural tragedies it is important to identify those instances in which neglect or abuse may have caused or contributed to the death.[250,251]

The precise nature of the investigation and composition of the investigating team will vary according to the requirements of the relevant state or national legislation. In England, regulations introduced under recent legislation have defined these requirements, which will be mandatory from 2008.[252] These regulations were based upon the conclusions of the Working Party chaired by Baroness Helena Kennedy, which involved wide consultation and contributions from pediatricians, pathologists, police, coroners, social services, Government, the Judiciary and representatives of parents' organization.[251] An overview of the requirements of these regulations is given below as an example of the needs for thorough and integrated investigations.

KENNEDY PROTOCOL FOR INVESTIGATION OF UNEXPECTED INFANT DEATHS[250,251]

The protocol involves emergency first responders, clinical staff, police, pathologists, coroners, social services and other agencies working together and sharing information to minimize duplication and maximize available information to help identify contributory or causal factors. The initial investigation must include a careful and detailed medical, social and environmental history, with a thorough review of the circumstances of death, including visiting and carefully examining the scene of death. This home visit with the parents or carers should ideally be conducted jointly by a pediatrician and a child protection police officer whose combined expertise in infant physiology and development and in forensic examination respectively maximize the potential to recognize both natural and unnatural contributory factors. The pathologist (who must have appropriate pediatric training) should conduct a thorough postmortem examination to an evidence-based protocol,[108,251,253] and should be provided with as full an account as possible of the history, clinical examination of the infant and scene examination before commencing the procedure. At all stages of the investigation all agencies must continue to share information and, except in those rare instances in which criminal prosecution might be compromised by so doing, the parents must be kept fully informed. Meeting the needs of parents for care and support must be central to the process. Finally, when all investigations are completed – usually 2–4 months after the death, a multi-agency case review meeting should be convened – usually in the primary care setting. The aim of this meeting is to ensure that all professionals share information; review, and if possible come to conclusions about the cause of or contributory factors to the death; agree who is to inform the parents of the results of the investigations (usually the pediatrician plus a member of the primary health care team); and produce a report for the coroner to inform and facilitate the Inquest.

Thus the 'cause' of death as finally certified through the coroners' system reflects the full breadth of professional expertise in understanding both natural and possible unnatural contributory or causal factors.

The careful review of potentially contributory factors allows unexpected infant deaths to be separated into those for which no significant contributory factors were identified, those in which one or more factors were found that may have contributed to the death but do not in themselves give a complete explanation, and those for which a complete and sufficient explanation was found. Several classifications of unexpected infant deaths using such approaches have been published, and allow studies to distinguish varying degrees of contribution from environmental, infectious, physiological or genetically determined factors in different infants.[250,254] Table 6.7 shows the Avon Clinicopathological Classification, which is based upon this approach, and has been widely adopted in the UK.[102,108,250,251]

Most epidemiological background factors associated with unexpected but explained infant deaths (i.e. those deaths classified as III in Figure 6.3, e.g. previously unrecognized overwhelming infection) are very similar in character to those factors found amongst SIDS victims (i.e. those deaths classified as I to IIB in Figure 6.3).[109] Indeed there is some evidence that improved investigation has led to an increase in the proportion of deaths that are explained, in particular deaths due to metabolic disorders.[102,108,250] Thus it is important that similar investigation should be applied to all such deaths, and any studies of unexpected infant deaths should include all sudden unexpected deaths in infancy (SUDI), and not be restricted to those classified (either by arbitrary assignment at the beginning of the investigative process, or at the end of a full investigation) as SIDS.

Although there has been some reluctance by professionals to fully engage in such a process, on the grounds that it is demanding of both time and energy, and may not be sustainable, recent studies have shown that with minimal additional resources such an approach can be implemented and sustained over many years.[102] Certainly the savings – both financial and emotional – from avoidance of inappropriate criminal

Table 6.7 The Avon Clinicopathological Classification of Sudden Unexpected Infant Deaths[164,165]*

Classification	0	I A	I B	II A	II B	III
Contributory or potentially 'causal' factors	Information not collected	Information collected but no factors identified	Factor present but not likely to have contributed to ill health or to death	Factor present, and may have contributed to ill health, or possibly to death	Factor present and certainly contributed to ill health, and probably contributed to the death	Factor present, and provides a complete and sufficient cause of death
History: (1)						
Death-scene examination (2)						
Pathology (3)						
Other (specify)						
Other evidence of neglect or abuse?						
Overall classification (4)						

The grid is completed at the multidisciplinary case discussion meeting (usually held 8–12 weeks after the death). An entry must be made on the line of each heading line, and a score (0–III) accorded to each line as agreed by all professionals present. The overall score is generally equal to the highest score within the grid. A score of III equates to a complete and sufficient cause of death. Scores of I–IIB meet the definition of SIDS. 1, To include a detailed history of events leading up to the death, together with medical, social and family history, plus explicit review of any evidence suggesting past neglect or abuse of this child or other children in the family. 2, results of detailed review of the scene of death by the pediatrician and police child protection officer in the light of the history given by parents or carers. 3, pathological investigations to a standardized protocol, including gross pathology, histology, microbiology, toxicology, radiology, clinical chemistry, and any relevant metabolic investigations, including frozen section of liver stained for fat. 4, this will generally equal the highest individual classification listed above.

charges, together with the recognition of genuine child protection issues, whilst providing appropriate support and care to families – warrant the adoption of a robust and thorough but sensitive investigation after all unexpected infant deaths.

Bereaved parents and their organizations have spoken strongly in favor of such an approach, in which the needs of parents for help, support, information and explanations, whilst avoiding blame or unwarranted suspicion, are explicitly integrated into the practice of all professionals involved in the process.

CURRENT RECOMMENDATIONS

The scientific rigor with which data is gathered is not easily applied to the dissemination of the results and formulating advice can be a subjective exercise of weighing up the available evidence and constrained by attempts to simplify the message. The debate on the safety, advantages and disadvantages of infant care practices must be informed not just by epidemiological evidence from one narrow field but from many disciplines from different fields if it is to become more than the exchange of mere opinion. The advantages of getting the advice right are evident in the dramatic fall in SIDS deaths after advice against the prone sleeping position, but it should be remembered that adoption of the prone position was initially largely a consequence of medical advice.

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