Poisoning

Poisoning in children 1: General management

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Basic principles in the management of poisoning

In this, the first of a series of five articles, we deal with basic principles in the management of poisoning in children.

A working knowledge of the management of poisoning in children is essential for all those involved in acute paediatric care. An estimated 52 000 people attended accident and emergency departments with poisoning in 1997, the majority of whom were children.[1] Table 1 shows the commonest agents involved.

POISON IDENTIFICATION

Wherever possible the constituents of the substance ingested and its dosage per kilo body weight should be identified as accurately as possible.

In younger children the substance taken is often easily identifiable but the dosage can be difficult to ascertain. Some idea of the maximum amount of substance that could have been ingested can be gathered from comparing the number of tablets, or volume of liquid remaining, with details on packaging.

Table 1 Common agents involved in poisoning[1]

Toxin	Percentage
Paracetamol	8.9
Cleaning products	5.9
Cough and cold remedies	4.8
Bleach	4.4
Plants and berries	4.3
White spirit	4.0
Oral contraceptives	3.4
Antibiotics	2.7
Benzodiazepines	2.6
Vitamins	2.2
Aspirin	2.1
Alcohol	2.9
Creams	1.6
Asthma medication	1.6
Antihistamines	1.5
Non steroidal anti-inflammatory drugs	1.4
Rodenticides	1.3
Petrochemicals	1.3
Vapour treatments	1.2
Nail care	1.2
Compound analgesics	1.2
Aftershave	1.1
Antidepressants	1.0
Iron	1.0
Washing powder	0.9
Mushrooms	0.8

Care must be taken not to overlook the involvement of other children in a poisoning incident.[2] When children share a poisonous substance, it must be assumed that each child has taken the maximum amount.

In older children, a clear history of ingestion may not be forthcoming and illicit drugs are more likely to be involved. Specific enquiry should be made into the medicines prescribed to each member of the household, both currently and in the past.

RECOGNISABLE POISON SYNDROMES

In cases where poisoning is suspected, but cannot be confirmed by clinical history, a detailed physical examination, including a full neurological assessment, is an essential part of substance identification. Table 2 shows collections of signs and their possible causes.

A number of toxins acting on the autonomic nervous system can produce a mixed clinical picture because of effects on both muscarinic and nicotinic receptors.

In most cases of poisoning, clinical suspicion is raised because symptoms do not fit a common pattern. Certain poisons can produce symptoms that mimic common diseases. A high index of suspicion is necessary in such cases (see table 3).

PREVENTING ABSORPTION

There is no place for the use of emetics.[6] The routine use of gastric lavage[7] or activated charcoal[8] is inappropriate. While the latter techniques may have a role in the early management of poisoning with a small number of specific substances, their effectiveness in these circumstances remains unproven.[7 8] Gastric lavage is contraindicated if a corrosive substance or volatile hydrocarbon has been ingested. Adequate airway protection is essential if any of these procedures is to be performed in the presence of an altered level of consciousness. Table 4 lists poisons for which activated charcoal has been proven to be ineffective.

ENHANCING EXCRETION

Active elimination techniques have a limited role in the management of poisoning. Their use should be restricted to situations where prolonged exposure to high concentrations of toxin is predictably deleterious. Examples of such situations would include haemodynamic instability despite supportive measures, intractable seizures, or organ failure.

The use of *repeated doses of activated charcoal* to remove toxins undergoing enterohepatic circulation is one of the simplest active elimination techniques.[9] Table 5 lists substances for which this technique may prove useful. The technique is not without its complications; these include bowel obstruction and perforation. Careful monitoring of bowel sounds is essential.

Forced diuresis has previously been recommended as a method of enhancing elimination of salicylates and barbiturates. The risk of fluid overload is high and this technique should be avoided.

Urinary alkalinisation can be used to enhance the excretion of weakly acidic drugs. The unionised form of the drug is filtered and reabsorbed. Urinary alkalinisation increases the proportion of ionised drug in the tubule, preventing its reabsorption. Examples of substances which may undergo significantly enhanced excretion include salicylate, isoniazid,[10] phenobarbitone, and dichlorophenoxyacetic acid.[11]

Conversely, decreasing urinary pH can be used to enhance the excretion of weakly alkaline drugs. *Urinary acidification*, using ammonium chloride, has previously been used to enhance excretion of amphetamine, strychnine, quinine, quinidine, and phencyclidine.[10] The dangers of acidosis and hyperammonaemia outweigh the benefits of this technique.

Whole bowel irrigation[12] can be used to physically eliminate highly toxic substances that are not absorbed by activated charcoal and have a long gastrointestinal transit time. Treatment is based on the enteral administration of large quantities (30 ml/kg/h) of osmotically balanced polyethylene glycol electrolyte solution to induce a liquid stool. Treatment is continued until rectal effluent clears. Substances for which this technique may prove useful include iron and sustained release or enteric coated preparations.

Dialysis, haemoperfusion, and haemofiltration have all been used to actively enhance toxin excretion. While many case reports exist in the literature, the efficiency of such methods is very difficult to assess clinically. Estimates of efficacy based on blood levels before and after treatment are likely to be misleading as they cannot take into account enterohepatic circulation, hepatic metabolism, or urinary excretion.[13] It is generally accepted that extracorporeal elimination is worthwhile if it increases total body clearance by 30% or more.[14]

For dialysis to be effective, a toxin must be of low molecular weight (<500

Table 2 Recognisable poison syndromes

Poison syndrome	Associated signs	Possible toxins
Increased sympathetic nervous system activity (these features are common in disease generally)	Pyrexia Flushing Tachycardia Hypertension Pupillary constriction Sweating	Cough and decongestant preparations Amphetamines Cocaine Ecstasy Theophylline
Anticholinergic activity	Similar clinical picture to sympathomimetics Clinical differences include: Pupillary dilatation Dry mouth Hot dry skin	Tricyclic antidepressants Antiparkinsonian drugs Antihistamines Atropine and nightshade Antispasmodics Phenothiazines Mushroom poisoning (*Amanita* species) Cyclopentolate eye drops
Increased parasympathetic nervous system activity	Pupillary constriction Diarrhoea Urinary incontinence Sweating Excessive salivation Muscle weakness* Fasciculation* Paralysis*	Organophosphate insecticides Drugs for myasthenia gravis, e.g. pyridostigmine
Metabolic acidosis	Tachyopnoea Kussmaul breathing (sighing respiration)	Ethanol Carbon monoxide Antifreeze Iron Diabetic medication Tricyclic antidepressants Salicylates
Chemical pneumonitis	Cough Respiratory distress Central nervous system depression A history of vomiting following ingestion need not be a feature	White spirit Turpentine Essential oils
Acute ataxia or nystagmus		Antihistamines Alcohol Anticonvulsants (especially phenytoin and carbamazepine) Piperazine Diphenylhydantoin Barbiturates Carbon monoxide Organic solvents Bromides
Methaemoglobinaemia	Cyanosis resistant to oxygen therapy	Alanine dyes Nitrates Benzocaine Phenacetin Nitrobenzene Chlorates Sulphonamides and metoclopramide (in neonates)
Renal failure	Oliguria or anuria Haematuria Myoglobinuria	Carbon tetrachloride Ethylene glycol Methanol Mushrooms Oxalates[3]
Violent emesis		Aspirin Theophylline Corrosives Fluoride Boric acid Iron[3]

*Due to excessive cholinergic stimulation at the motor end plate.

relative molecular mass (RMM)) and highly water soluble. It must have a small volume of distribution (<2 l/kg) and bind poorly to protein. Examples include salicylate, methanol, ethylene glycol,[15] vancomycin,[16] lithium,[17] and isopropanol[18] poisoning. Dialysis is of particular value where concomitant electrolyte or acid-base disturbance exists.

Haemoperfusion is better suited to toxins with low water solubility. Such substances must have a high affinity for the adsorbent, a fast rate of equilibrium from peripheral tissues to the blood, and a low affinity for plasma proteins.[14] Examples include carbamazepine, barbiturates, and theophylline.[11]

Haemofiltration can remove compounds with a high molecular weight (>500–40 000 RMM). It is of particular use in aminoglycoside[14] and theophylline[19] overdose. Haemofiltration may also be of benefit in iron[20] and lithium overdose.[21]

Substances not amenable to significant extracorporeal removal include benzodiazepines, tricyclic compounds,[22] phenothiazines, chlordiazepoxide, and dextropropoxyphene.

LABORATORY INVESTIGATIONS

A careful history may obviate the need for blood tests. Particular attention should be paid to safe ingestion levels.

Routine measurement of plasma paracetamol and salicylate may be considered in older children presenting with deliberate ingestion. The value of such practice has been questioned in adults.[23 24] However, Ashbourne *et al* found that 1 in 500 adult overdose patients, not suspected of having taken paracetamol, had levels above the treatment threshold.[25] In adolescents, the cost of missing serious paracetamol ingestion probably outweighs the price of the test.

Techniques exist to identify a wide variety of toxins, particularly drugs of abuse, from samples of blood and urine. These tests are expensive and rarely provide immediate results. They may have important medicolegal and social consequences, but rarely alter clinical management.[26] Samples are best obtained acutely and stored for future use as deemed necessary.

ADMISSION CRITERIA

Decisions about the need for hospitalisation of children presenting with possible poisoning are sometimes difficult. Most children will be asymptomatic and a short period of observation, in an emergency department or admission ward, is often all that is required. The nature, and quantity, of the substance consumed must clearly be taken into account. Reports are available on the speed with which symptoms develop for a variety of poisons.[27] Suggested observation times, for asymptomatic children, are based on these figures.

The circumstances surrounding a poisoning episode may have an important impact. Deliberate ingestion may signal significant psychosocial problems. An inconsistent history of poisoning in a younger child may raise the possibility of abuse or neglect.

Other factors that need consideration include family circumstances, parental confidence, and the availability of emergency care should the child deteriorate unexpectedly.

Table 3 Poisons which mimic common disease

Symptoms and signs	Possible toxin	Possible differential
Non-ketotic hypoglycaemia, collapse	Ethanol	MCAD, glycogen storage disease
Acute liver failure	Paracetamol	Idiopathic liver failure
Hyperglycaemia, ketosis, CNS depression	Acetone Theophylline[4]	Diabetic ketoacidosis
CNS depression, fits, pyrexia	Ecstasy	Febrile convulsion[5]
Hyperthermia, tachypnoea, sudden onset of symptoms	Salicylates	Pneumonia

Table 4 Poisons for which activated charcoal has been proven to be ineffective

Alcohols (e.g. ethanol, isopropanol)
Essential oils
Petrochemicals
Iron
Lithium
Bleach

Table 5 Substances where repeat doses of activated charcoal may prove useful in enhancing clearance[9]

Carbamazepine*
Barbiturates*
Dapsone*
Quinine*
Theophylline*
Salicylates‡
Amanita phalloides (death cap mushroom)‡
Slow release preparations
Digoxin† and digitoxin†
Phenylbutazone†
Phenytoin†
Sotalol†
Piroxicam†

*Experimental and clinical studies; †volunteer studies; ‡little firm evidence.

SPECIFIC ANTIDOTES

A wide range of specific antidotes exists (see table 6).

SUPPORTIVE MANAGEMENT

Initial management should focus on assessment of airway, breathing, and circulation. Depression of the central nervous system is a common symptom. This may lead to airway compromise, respiratory failure, or aspiration.

Diuresis, vomiting, and diarrhoea may all contribute to profound dehydration and shock. Aggressive fluid resuscitation, guided by invasive monitoring, may be necessary. Hypotension, unresponsive to adequate fluid replacement, requires treatment with inotropes. Dopamine and dobutamine are the agents most commonly used. The inotropic effect of glucagon has been used in the management of β blocker and tricyclic antidepressant induced hypotension.

Metabolic acidosis is frequently encountered. In many cases, this is mild and does not require specific therapy. In some cases, the correction of a mild metabolic acidosis may decrease toxin clearance (see Urinary Alkalinisation).

Hepatic and renal function should be monitored closely. Urine samples should be checked regularly for blood, haemoglobin, protein, glucose, and myoglobin.

Hypoglycaemia should be identified rapidly and corrected using intravenous boluses (5 ml/kg) of 10% dextrose.

Convulsions can usually be treated with benzodiazepines.

Children developing symptoms after ingestion, other than perhaps mild nausea, vomiting, or diarrhoea, require hospital admission. For most poisons, treatment is supportive.

Children with nausea and vomiting may require intravenous fluids. Treatment with antiemetic drugs is best avoided.

Table 6 Specific antidotes

Toxin	Antidote
Benzodiazepines	Flumazenil
β blockers	Adrenaline infusion, glucagon
Carbon monoxide	Oxygen
Carbon tetrachloride	N-acetylcysteine[28]
Digoxin	Digoxin antibodies[29]
Iron	Desferrioxamine
Isoniazid	Pyridoxine, sodium bicarbonate[30]
Lithium	Sodium replacement, low dose dopamine
Methaemoglobinaemia	Methylene blue
Methanol Ethylene glycol	Ethanol, alcohol dehydrogenase inhibitor (fomepizole)
Metoclopramide	Procyclidine
Opiates	Naloxone
Organophosphate insecticides	Atropine, pralidoxime
Paracetamol	N-acetylcysteine
Thyroxine	Propanolol[31]

MANAGEMENT OF ARRHYTHMIAS

A variety of common household poisons may produce arrhythmias in overdose. Arrhythmias are rarely encountered in general paediatric practise and can prove daunting.

The presence of an arrhythmia in a poisoned child does not necessarily indicate direct cardiac drug toxicity.[32] The first step in treatment is to ensure adequate resuscitation and supportive therapy. Underlining hypoxia or hypercarbia must be corrected. Abnormalities of electrolytes or acid-base balance should be addressed.

While many arrhythmias have serious consequences for children and adults with significant cardiac pathology, they may be relatively benign in healthy children.[33] Only if supportive measures prove inadequate should specific therapy, aimed at correcting an arrhythmia, be considered. Table 7 lists specific antiarrhythmic drugs, and also drugs to avoid.

MANAGEMENT OF CORROSIVE INJURY

The investigation and treatment of caustic ingestion in children is controversial. Alkalis tend to cause more damage than acids, while liquids cause more scars than powders. Products that can become trapped in the oesophagus cause the most damage, for example, batteries, Clinitest, or dishwasher tablets.

The outcome for most children following corrosive ingestion is good. Superficial oesophageal burns occur in 20% of cases, deeper burns are seen in 5%, and stricture formation in 1–3%.[44] Attempts at neutralisation of corrosives, or gastric decontamination, are best avoided. The utility of early upper gastrointestinal endoscopy in symptomatic children, followed by steroid treatment if oesophageal burns are identified, has been called into question. Early signs and symptoms do not predict the presence of oesophageal burns,[45] and Anderson *et al* found no evidence that steroid treatment improved outcome.[46]

Table 7 Specific antiarrhythmic drugs used in poisoning; drugs to avoid are also shown

Toxin	First line antiarrhythmic agents	Other antiarrhythmic agents	Antiarrhythmic agents to avoid
Antihistamines	Sodium bicarbonate	Magnesium sulphate	Class Ia and III[27]
Antihistamines terfenadine or astemizole	Sodium bicarbonate	Propanolol, isoprenaline, flecainide[27], magnesium sulphate	Class Ia and III
Class I antiarrhythmics, e.g. Ia quinidine and procainamide, Ib lignocaine, Ic flecainide	Sodium bicarbonate, run mildly alkalotic and keep potassium low normal[33]	Atropine, phenytoin[33]	Class I (although lignocaine can be used for arrhythmias due to Ia and Ic)[33]
Class II antiarrhythmics, i.e. β blockers	Atropine, glucagon, cardiac pacing[27]	Adrenaline, isoprenaline, dopamine, dobutamine[34]	
Class III antiarrhythmics, i.e. sotalol and amiodarone	Amiodarone—toxicity is generally low Sotalol—initial treatment as for other β blockers	Lignocaine—for sotalol,[27] magnesium sulphate[34]	Isoprenaline can result in tachyarrhythmias[27]
Class IV antiarrhythmics, i.e. calcium channel antagonists	Calcium gluconate,[33 34] cardiac pacing	Atropine, glucagon, dopamine	
Cocaine	Sodium bicarbonate[35]	Diazepam, GTN, calcium antagonists[36–38]	
Dextropropoxyphene	Naloxone,[39] sodium bicarbonate[40]	Magnesium sulphate, phenytoin, atenolol[27 41]	Class I, atropine[27]
Digoxin	Correct hyperkalaemia (avoid calcium), digoxin antibodies , cardiac pacing	Atropine, lignocaine, amiodarone, phenytoin[27]	Class Ia, Ic,[29] DC shock,[29] calcium, inotropes
Quinine	Keep potassium low normal	Phenytoin, β blockers, glucagon[27]	Class Ia[27] Lignocaine may precipitate convulsions[27]
Theophylline	Avoid treatment if at all possible—2nd line agents may cause bronchoconstriction	Propanolol,[42] esmolol, adenosine[27]—all may produce bronchoconstriction in asthmatics	Verapamil, lignocaine[42]
Tricyclic antidepressants	Sodium bicarbonate, try to avoid other agents as much as possible	Phenytoin, atenolol,[27] glucagon,[43] lignocaine for ventricular arrhythmias, magnesium sulphate	Class Ia[27]

Ensure adequate resuscitation and supportive treatment. Arrhythmias may be relatively benign in healthy children and drug therapy should be avoided wherever possible.

Children should be managed symptomatically. Particular care is needed over fluid balance and respiratory function. Drooling and dysphagia persisting beyond 12–24 hours are good predictors of oesophageal scar formation[44] and should prompt upper gastrointestinal endoscopy.

FOLLOW UP OF DELIBERATE SELF HARM

Deliberate poison ingestion is a common presentation in older children. While the precipitating factors are often minor, arguments with friends or parents being the most common, these episodes should not be regarded as trivial. Children who display self harming behaviour have a significantly increased risk of underlying psychiatric illness, particularly depression.[47] In many children self harm is a manifestation of significant difficulties in other parts of their lives. Social disadvantage and disturbed family relationships are common.[48]

Adolescents presenting with self harm should undergo psychiatric assessment. This is ideally undertaken during an in-patient stay, as outpatient attendance rates are poor.[49] This assessment can also offer adolescents, and their parents, a useful forum in which to discuss ongoing difficulties.

SEEKING FURTHER ADVICE

Specific, expert advice on all aspects of poisoning is available to medical professionals in the United Kingdom via the National Poisons Information Service (NPIS). The regional centres that make up this service have recently introduced a single national enquiry number: 0870 600 6266.

A wide range of easily accessible and highly practical advice is available through the NPIS website.[50] This free service is restricted to medical professionals. On line registration is available at http://www.spib.axl.co.uk/toxbase/.

While contact with the NPIS is an important way of keeping up to date with developments in this field, information gleaned should be disseminated to reduce the need for multiple enquires from individual hospitals.

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Oral clodronate as treatment of osteogenesis imperfecta

The benefits of treatment with intravenous pamidronate in osteogenesis imperfecta (OI) have recently been reported.[1–3] These include reduced bone resorption, increased bone density, and improved clinical outcomes as judged by apparently lower fracture rates. We would like to report a single case of OI treated by the orally administered bisphosphonate, clodronate, with good effect.

A boy, whose mother was affected with clinically diagnosed type 1 osteogenesis imperfecta, was referred to our unit aged 13½ with a recent onset of severe back pain that had required hospital admission. He appeared of normal stature with blue sclerae and was able to walk independently. He had sustained four previous limb fractures; lateral radiographs of the thoracic and lumbar spine confirmed three vertebral wedge fractures. He was 158.9 cm tall (10th centile) and weighed 49 kg (25th centile). Lumbar spine bone mineral density scanning by dual *x* ray absorpiometry (DXA, Hologic QDR-1000, Hologic, Bedford, MA) revealed a BMD of 0.398 g/cm^2 (Z score −5.22, comparing his value to the average young man). Fasting urinary hydroxyproline/creatinine ratio, an index of bone resorption, was 96.6.

With informed parental consent for "off label" usage, he was commenced on oral sodium clodronate (Bonefos, Leiras Oy, Turku, Finland) at a dose of 400 mg daily. Dietary intake of calcium and vitamin D was above the estimated average requirement and supplementation was not given. Subsequent BMD scanning after one year of therapy showed an improvement of bone density by 7.8% to 0.468 g/cm^2 with a consequent increase in the BMD Z score to −5.09.

Treatment with clodronate was continued for five years, during which time he did not sustain any new low trauma fractures. By the end of treatment, his BMD was 0.552 and his Z score had improved from −5.22 at first referral to −4.59. The fasting urinary hydroxyproline:creatinine ratio was decreased by 86% compared to baseline (13.6 *v* 96.6). Clodronate was discontinued for eight months; during this time his BMD remained stable but the Z score showed a small decline. His height at that stage lay on the 50th centile (176 cm) and his weight on the 10th (58 kg). The BMD remained considerably below the normal value and clodronate was recommenced at a dose of 800 mg daily.

Eight years after initial referral, his bone mineral density had increased by 60.6% to 0.613 g/cm^2 (Z score −4.16). To compensate for the expected increase in bone size, the bone mineral adjusted density (BMAD) was computed ($BMD_{L1–L4}$/square root of $area_{L1–L4}$) and showed an improvement of 24.6% in BMAD over the duration of therapy. Clodronate was discontinued when the patient was aged 22. He had reached a height of 177.4 cm and a weight of 60.8 kg, and the spine BMD Z score was −3.92. He had suffered no atraumatic fractures since commencing oral clodronate.

The rationale in using bisphosphonates for osteogenesis imperfecta is the inhibition of osteoclastic bone resorption leading to increased bone density and a potentially lower risk of fracture. This young man exhibited a good response to therapy with oral clodronate, suffering no adverse reactions. The increase in height of 18 cm over eight years, moving him from the 10th to 50th centile, suggests that his growth was not impaired by therapy.[4] Many studies have shown that clodronate does not impair mineralisation. We are unable to determine the contribution of remodelling of vertebral fractures to his height gain.

A limitation of this report is that pubertal status was not documented at presentation. However, the increases in lumbar spine density are in excess of the expected average rates in growing children (3–6% per year and 14–16% during puberty).[1] The increase was also observed following adjustment for bone growth by BMAD and Z scores relating the measured BMD to age matched controls. The Z score (−5.22 to −3.92) improvement during therapy was not dissimilar to that reported with pamidronate[1] in younger children.

We agree that there is increasing evidence of a role for bisphosphonate therapy as part of the multidisciplinary management of osteogenesis imperfecta. Oral clodronate in our patient appeared to elicit a similar response to that of cyclical intravenous pamidronate, suggesting that orally administered bisphosphonates may be of value in the management of this disease.

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Serum prolactin in coeliac schoolchildren

Literature published suggests that in children with coeliac disease (CD) serum prolactin concentrations are increased, and correlate with the grade of mucosal atrophy. It has been proposed that prolactin is a possible marker of disease activity.[1] Other studies, however, have failed to show this correlation in children with CD.[2]

We studied prolactin levels in children with CD, and the correlation with the severity of intestinal mucosal atrophy.

We used samples from a serum bank obtained during a mass screening for CD in Sardinian schoolchildren, using both antiendomysial antibodies and antigliadin antibodies as screening tests, as previously described.[3] The sample included 20 children with CD on a gluten containing diet (16 girls, 4 boys, mean age 12.9 years, range 11.5–14.4 years) and 40 sex and age matched normal children (32 girls, 8 boys, mean age 13.0 years, range 11.2–14.8 years). All subjects were euthyroid. Prolactin was assayed in duplicate using a commercial immunoradiometric method; results were analysed by analysis of covariance.

Data are expressed as mean (SE). Prolactin levels were 4.62 (2.1) ng/ml in patients with CD and 5.90 (2.6) ng/ml in controls (no statistically significant difference). No correlation was found between prolactin concentrations and the degree of intestinal damage (Marsh criteria).

Our study did not confirm the increased prolactin concentrations in children with CD reported by Reifen and colleagues.[1] Our population differed somewhat in that there was a higher mean age (12.9 *v* 11.3 years), a narrower age range (11.5–14.4 *v* 5–18 years), and a different girl:boy ratio (4:1 *v* 1:1). Furthermore, our study included three potential coeliacs (subjects with antiendomysium antibodies positivity but normal intestinal biopsy[4]) and 11 asymptomatic coeliac children. The hypothesis that the normal prolactin values observed in our study may be due at least in part to the different clinical characteristics of the population studied is plausible, but its validation requires a specifically designed study.

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BOOK REVIEWS

A Clinical Guide to Inherited Metabolic Diseases, 2nd edn

Edited by JTR Clarke UK: Cambridge University Press, 2002, £29.95, pp 306. ISBN 0521890764

Dr Clarke's enthusiasm and erudition are evident on every page of this book, which is handily sized, and, wonder of wonders, costs only £30.

Most of the chapters are written with a clinically based approach, and the chapters on basic principles in understanding inherited metabolic disease, neonatal screening, hypoglycaemia, metabolic acidosis, storage diseases, and dysmorphism will be read with a sensation of increasing revelation by just about any paediatrician, and those with a secure background in biochemistry and metabolic disease will pick up many nuggets of wisdom.

Why then, do I simply not recommend every paediatrician who sometimes deals with metabolic problems—and there must be few of us who do not—to rush out and buy a copy before such a gem either goes out of print or rises in price? My caveat is that this book's clinical approach coupled with its encyclopaedic coverage of some topics means that several chapters leave the non-expert mentally breathless, and this effect is made worse by the absence of the structural formulae of the molecules named; this may be of little moment to those with these formulae at their fingertips, but that excludes rather a lot of us. By contrast the metabolic sections of "Nelson" and "Forfar and Arniel" clearly benefit from their inclusion.

So, for those on a unit dealing with many patients with inborn errors of metabolism, this book is invaluable, but general and trainee paediatricians who think they can read this book from start to finish and become initiated into the mysteries of metabolic disease are likely to be overwhelmed.

If you can peruse this paperback at your local medical bookshop, do so; you may find it an answer to prayer, and anyway, at just under £30 it won't bankrupt anyone.

R A F Bell

Eating Problems in Children: Information for Parents.

Edited by C Fox, C Joughin. London: Gaskell, 2002, £10.00, pp 621. ISBN 1901242862

How commonly do we encounter the following scenario?

A desperately anxious mother at last convinces her GP that she needs to see a paediatrician because her normally growing toddler is eating nothing. The paediatrician wonders why his time is being wasted, and "reassures" the mother that there is nothing to worry about. Needless to say the anxiety persists with, no doubt, damaging consequences. As a profession, we handle these cases poorly. With 30% of preschool children suffering from mild to moderate eating problems, we need a better way to address these issues.

The Royal College of Psychiatrists has produced this small book for parents that should prove helpful, not only to parents but also to paediatricians and other health professionals. It provides information about the epidemiology of eating problems, and gives a useful classification, categorising eating difficulties into nine types, including persistence of eating inappropriate texture of food for age, food refusal, restrictive eating and selective eating. This allows the parent or professional to come to a more specific "diagnosis", and also a sense of the anticipated course these difficulties are likely to take. In particular, it provides clear pointers for those conditions that are indicative of significant emotional or psychiatric conditions.

Giving clear indications to the parent as to when to worry is helpful, as it is likely to encourage a sense of proportion to the anxiety accompanying the more common eating difficulties. The book goes on to provide specific and sensible advice about the practical management for each of the different types of eating difficulty.

At the end of the day, one is left with the finding that for most parents, not surprisingly, reassurance is what is required. I felt, however, that this book could help us proffer the advice in a more substantive form than we do at present, and can give us an approach that is likely to help diffuse the anxiety which contributes to the perpetuation of stressful mealtimes. I suspect the book will prove to be of most value to health visitors, but selected reading could be of use to the paediatrician too.

This book is therefore of value for a problem that presents so frequently to the general paediatrician, but I must admit to some reservations. It could have been better written, and in particular was rather unnecessarily repetitive. It certainly would have benefitted from paediatric review—I wondered who or what a community practitioner was, and gulped when I saw growth hormone mentioned in the section of treatment for restrictive eating! It was rather more concerning that children with disabilities got an occasional mention, implying that they merited the same sort of approach. It surely would have been better to emphasise that they require a different sort of understanding and input. But, despite these concerns, the book should prove useful as it provides a systematic approach to the child with eating difficulties, and some clear, sensible practical advice to guide the parent in handling the problem.

M Rudolf

Childhood Headache

Edited by I Abu-Arafeh. UK: Cambridge University Press, 2002, £45, pp184. ISBN 1 898 68326 3

Headaches in children are a common problem—70% of school children have headaches at least once a year, with 25% suffering from recurrent headaches. This book is part of the Clinics in Developmental Medicine series, and provides a comprehensive overview of the subject. The book is divided into clear chapters, which makes it easy to dip into. It includes interesting sections on pain perception in children and neonates, as well as a good epidemiology section. Throughout the book there are summary tables of recently published studies. In the later chapters there are case histories, including parental descriptions, which break up the occasionally slightly long winded text. There is an extensive list of references at the end of each chapter.

I found the chapters on migraine enlightening, especially the theories on pathophysiology of migraine. The diagnostic criteria for migraine are easy to read and clear. There is an excellent overview on the psychological treatment of headaches, regardless of diagnostic type. Again, the evidence is summarised in clear tables. There is a practical section on managing abdominal migraine. Causes of headaches are divided into separate chapters for specific and rare causes, which was helpful when I used the text when on call.

The final chapter talks about setting up a headache clinic, including a discussion on diagnostic tests. There is a headache questionnaire for parents, which I would find very helpful. There is also advice on the role of the multidisciplinary team in management.

This book would be a valuable addition to a general paediatric department, both in outpatients and for reference when on call.

A Morjaria

CORRECTIONS

In the article by Nixon *et al* (*Arch Dis Child* 2002;**87**:306–11), Dr Claire Wainwright should have been included as an author. Dr Wainwright's contribution was the establishment of the methodology and early patient recruitment and testing. Dr Wainwright moved from The Royal Children's Hospital at the end of 1997, and was funded by The Royal

Children's Hospital Foundation, Brisbane and the Cystic Fibrosis Research Inc, Queensland. The authors apologise for the omission.

The authors of the letter "Childhood SARS in Singapore" in the August issue (*Arch Dis Child* 2003;**88**:742) were written incorrectly. The authors names should be P Van Bever, C P P Hia, S C Quek.

In the acknowledgements for the leading article by Duke *et al* (*Arch Dis Child* 2003;**88**:563–5), Dr Diana Silimperi should have been acknowledged as part of the Paediatric Quality Care Group. The authors apologise for the error.

An error occurred in the paper by Riordan M, Rylance G, Berry K in the November issue. (Poisoning in children 1: General management. *Arch Dis Child* 2002;**87**:393–6). In table 2, pupillary constriction associated with signs of increased sympathetic nervous system activity should read as mild pupillary dilation. Anticholinergic agents are likely to produce a more marked dilation. The authors apologise for the error.