

LESSONS FROM A JOINT SYMPOSIUM ON PAEDIATRIC EMERGENCIES*

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A joint symposium between the Royal College of Physicians of Edinburgh and the Royal College of Paediatrics and Child Health was organised to present current trends and discuss issues in Paediatric Emergency care. The symposium was a great success, attracting 350 delegates from a mixture of Paediatric, Accident and Emergency Medicine and Primary Care backgrounds. A panel of internationally renowned speakers provided a superb selection of lectures with interactive discussion. This report aims to summarise the salient points from each of the lectures.

ACUTE ASTHMA IN CHILDREN

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Asthma remains a common chronic disease of childhood; it is the most common medical reason for children to be admitted to hospital, and such admissions have continued to rise since the 1970s. Recognition of acute severe asthma in children can be difficult. Management involves immediate high flow oxygen, nebulised β_2 agonist via oxygen-driven nebuliser and steroids. The route of bronchodilator administration is an important consideration.

In 1998 a study showed that 20% of children received bronchodilator via nebuliser only, but 60% received bronchodilator by nebuliser and spacer. The Cochrane Review¹ examined management of children with acute life threatening asthma in Accident and Emergency (A&E) departments up until September 1999. It compared bronchodilator administration via large volume spacers versus nebulisers. No significant difference in clinical outcome was shown between the two groups. Observed benefits in the large volume spacer group were that the children were in the A&E department less frequently, there were fewer side-effects and there was improved oxygenation.

Another study looked at children admitted to hospital with acute asthma and randomised them to receive high dose bronchodilators via nebuliser or alternatively via nebuliser. They were treated and discharged. At two weeks, a morbidity score was calculated over the telephone. The children who had received bronchodilator via spacer had lower morbidity scores. At one year follow-up they had lower readmission rates. The difference in the results was attributed to the fact that nurses were able to educate children about the large volume spacer devices during their stay in hospital, and this practice could then continue at home. An initial 'alternative treatment' was shown to give the patient better long-term outcomes.²

Early treatment of acute asthma with steroids (within one hour of presentation) is recommended. It is associated with lower admission rates. The maximal effect is seen four to six hours after treatment and there is no difference between oral and intravenous administration.

In childhood asthma it is vital to consider the circumstances of admission: was there an avoidable precipitating cause; did the patient/family react appropriately; was the patient complying with therapy; was medical management appropriate? Data from Melbourne suggest that there are often deficiencies in all these management aspects which can be addressed to improve future care.³

Discharge planning in hospital is also important: ideally, prior to being discharged home, children and their parents should attend an information and discussion session and be given an asthma information booklet, a tailored home management plan and access to an asthma helpline. Nurse-led home management training can significantly reduce acute asthma readmissions (25% down to 8%). Lower reported asthma morbidity was reported at follow-up.

In summary, an acute asthma attack may give hospital staff the opportunity to offer patients a key educational opportunity, and this will result in a reduction of subsequent mortality. The strategies used to treat an acute attack must be carefully planned and the opportunity used to empower the patient. Guideline implementation is important but it must be ensured that these guidelines are of benefit to the patient.

ACUTE UPPER AIRWAY OBSTRUCTION

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Upper airway obstruction (UAO) is potentially life threatening. Laminar flow is proportional to the fourth power of the radius of a tube, so in children with small airways only 1 mm of mucosal oedema will increase resistance to flow 16-fold. Upper airway obstruction presents most commonly with stridor; other signs include increased effort breathing and signs of end-organ hypoxia such as decreased level of consciousness and cyanosis (which occur late and are often pre-terminal).

A study from Guy's Hospital in London over a three and a half year period looked at 67 children with UAO referred to the paediatric intensive care unit.⁴ The commonest cause of UAO was croup (in 39 children). Other causes include sub-glottic stenosis, bacterial tracheitis, laryngomalacia and epiglottitis.

Croup is defined as acute laryngotracheitis, with most cases being due to parainfluenza virus. The child presents with a prodromal illness, low grade fever and stridor. The illness becomes worse at night and lasts three to five days. Croup is rare under four months and is rarely associated

* Held in the Royal College of Physicians on 28 September 2000

with a pyrexia of more than 38.5° C. Most children with croup are managed at home, with only ten per cent attending hospital.

Worrying features of croup include continuous stridor at rest, use of accessory muscles at rest and decreased air entry. A decision to admit may also be based on social factors such as distance from the hospital, lack of telephone or transport. Treatment of croup includes humidification, adrenaline and steroids. Humidification *per se* has no benefits other than the reassuring presence of a parent in a warm, light environment.

Nebulised adrenaline in croup is beneficial, with few side-effects. The duration of action is approximately two hours. If a child is well with no evidence of relapse at three hours after the use of nebulised adrenaline, it may be considered for discharge home.

Steroids in croup are also beneficial. Fifteen randomised placebo controlled trials have shown the benefit of an early administration of a single dose of steroid in terms of clinical parameters, decreased hospital stay and need for rescue medication, e.g. dexamethasone given orally 0.6 mg/kg, budesonide given nebulised in a dose of 2 mg. Trials show that there is little difference in outcome between the two preparations. Choice may depend on the child's acceptability of route of administration.

Subglottic stenosis may be congenital but is more commonly iatrogenic when an endotracheal tube causes pressure necrosis of the mucosa and subsequent stenosis. Most children with mild stenosis only become symptomatic when they have an upper respiratory tract infection.

Laryngomalacia is a common cause of stridor in young babies. It is due to a floppy epiglottis with redundant aryepiglottic folds which flop into the tracheal opening on inspiration. Diagnosis is usually made on endoscopy. Stridor is often present from birth but may also develop in the first few weeks; it may be worsened by lying down or by concurrent upper respiratory infection. It is usually benign, resolving by two years of age. It may cause failure to thrive or dyspnoea and these may be indications for surgery (aryepiglottoplasty).

Foreign body aspiration is most common in children less than three years of age. Food material is the commonest cause, with peanuts perhaps being the single largest culprit. Fortunately, less than ten per cent of foreign bodies lodge in the upper airway where they cause varying degrees of stridor.

Epiglottitis is now rare. It causes supraglottic obstruction (compared to croup which is subglottic) and presents with drooling, dysphagia, dyspnoea and dysphonia. The Hib vaccine has reduced the incidence of epiglottitis markedly.

In summary, UAO is a challenge for any doctor. Most of the potential causes can be managed conservatively. When critical obstruction is present, specific diagnosis is not essential but the medical expertise required to secure the airway must be readily available.

ANAPHYLAXIS

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Anaphylaxis has an incidence of ten per 100,000 people. It is associated clinically with bronchospasm or with documented fall in blood pressure. One death from

anaphylaxis occurs every 60 days in Britain. Most allergic reactions do not result in anaphylaxis although they increase the risk of it occurring. The rate of anaphylaxis in allergic reactions is 1:5,000 in males and 1:2,000 in females.

A retrospective review of 32.4 million hospital discharge episodes revealed 2,424 coded as anaphylaxis. The incidence of anaphylaxis has increased from five to six cases per 100,000 in 1991 to ten cases per 100,000 in 1994. The major cause of this increase is food-related anaphylaxis.

In studying fatal and non-fatal food-related anaphylaxis in children, three out of six cases were due to peanuts and one occurred at home; of the non-fatal cases in children, two out of seven were due to peanuts and all seven occurred at home. The major distinguishing features between the fatal and non-fatal reactions were a history of asthma (especially if poorly controlled) and a delay in administration of epinephrine (adrenaline). Another feature was the attack taking place in an unfamiliar environment.

Anaphylaxis occurs most commonly at home. Causes are varied and include latex in 27%, insect venom in 15%, peanuts in 11%, antibiotics in 9% and shellfish in 7%. Common initial presenting signs and symptoms are dermatologic (60%), respiratory (25%), gastrointestinal (5%), neurological (4%) and cardiovascular (2%). Children with significant reactions should not be discharged before four hours have elapsed since the episode as they may develop a rebound reaction.

The requirement for the administration of epinephrine is controversial. Asthma is an independent risk factor for a severe reaction in those with food allergy so it is useful to give an adrenaline auto-injector to patients with a history of severe food allergy and asthma.

Prescribing and dispensing more than one auto-injector per patient is recommended, as in 105 anaphylactic reactions due to venom, 38 patients (35%) needed more than one injection. Epinephrine auto-injectors buy peace of mind and quality of life, but it must be ensured that regular detailed training is given to the patient and family. Patients with prescribed kits commonly forget to take them out, have out-of-date kits or incorrect dosage and forget how/when to use them.

In summary, anaphylaxis is common and increasing in frequency. In hospital, the chief causes are latex and drugs. In the community, the chief causes are foods and venom. The diagnosis must be made and the precipitating causes considered. Treatment consists of oxygen, adrenaline (in repeated doses if necessary) and fluids. Steroids and anti-histamines may be given later. The at-risk groups should receive epinephrine self-injector kits and 'medic alert' bracelets. It should be ensured that epinephrine dose and family training are reviewed regularly.

PAEDIATRIC HEAD INJURY

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Paediatric head injury remains an extremely common cause of morbidity and mortality. Prevention is vital to minimise primary and secondary cerebral damage. As in all cases of scalp trauma, it is essential to assess and manage airway, breathing and circulation prior to neurosurgical assessment. Small children may lose significant volumes of blood from scalp lacerations and may also bleed into the sub-galeal

space and become shocked. Sub-dural or extra-dural haematomas (EDH) or diffuse axonal injury are also common.

Minor head injuries are common and awareness of the potential for complications is vital. Significant head injury may occur with maintained level of consciousness, e.g. with a depressed skull fracture. External findings do not necessarily correlate with the severity of head injury.

Extra-dural haematoma presents classically in only 12%. The classical history is head injury followed by a 'lucid interval' and then by a deterioration in consciousness level, pupillary changes and hemiparesis. Two-thirds of children with EDH have a skull fracture. The presence of a skull fracture is the commonest associated predicting factor for EDH. Controversy exists in relation to imaging in head injury. The current protocol at the Royal Hospital for Sick Children of Edinburgh is set out below.

- A skull X-ray is performed on children less than one year of age when there is visible evidence of head injury or a suspicious history.
- Children with loss of consciousness for more than five minutes, amnesia, persisting headache, vomiting, lethargy, coagulopathy or bleeding diathesis are admitted for observation and/or a CT (computerised tomography) scan.
- Immediate CT is performed on those with GCS <14, focal neurological signs, seizure (focal or prolonged), signs of base of skull fracture, penetrating injury or depressed fracture.
- Plain X-ray can still be useful in depressed skull fractures and 'growing' fractures. Growing fractures are peculiar to childhood and occur when there is a linear fracture which takes in a loop of dura. Because of the CSF in the dura, the pulsatile process makes the fracture bigger. These fractures can be difficult to treat and split skin grafts may have to be applied.
- Ultrasound is useful in children with open fontanelles. It will assist with assessment of ventricular size, haematoma formation and cerebral blood flow velocity. Computerised tomography is the investigation of choice for mass lesions. Magnetic resonance imaging (MRI) is used to detect white matter lesions.
- A significant isolated head injury runs the gamut of injury from a depressed skull fracture to the presence of another fracture, a GCS <10, 'V' or less on 'AVPU' scale, seizure or neurological deficit.
- Airway management and cervical spine control are vital. High flow oxygen is given and intravenous access obtained. Breathing and circulation are assessed and managed as appropriate. Saturation, ECG monitoring and blood pressure measurement are instituted. Fitting is treated with intravenous diazepam. Appropriate X-rays are taken with supervision. Elective paralysis and ventilation are performed if indicated, with the cooperation of the ITU/anaesthetic teams; in such instances the neurosurgeons are notified. In general, children with GCS 3–10 are sedated, paralysed, intubated and ventilated. After CT they are taken to theatre or to ITU for ICP monitoring as appropriate. Children of GCS 10–12 have a CT scan and further management depends on the results along with clinical progression. Children of GCS 12–15 without a fracture are admitted for observation and CT may be performed.

The management of paediatric head injuries remains a common challenge. It aims to optimise intellectual and physical development and decrease intracranial damage with the minimum loss of life and function.

THE CHARLES MCNEIL LECTURE

MANAGEMENT OF FEBRILE ILLNESS IN THE PRE-SCHOOL CHILD

Dr M. Harper, Assistant Professor of Paediatrics, Harvard University, Boston

Occult or unsuspected bacteraemia occurs when a pathogen is isolated from the blood stream of a child aged between three months to three years who has normal immunity. The child should look well on examination and have a high fever (greater than 39° C rectal).

They must have no identifiable infection requiring antimicrobial therapy with the exception of otitis media. They must have no specific identifiable viral infection. Most cases of occult bacteraemia do however include patients with non-specific viral infections such as gastroenteritis, upper respiratory infections etc.

It is possible to employ strategies to identify children at risk of occult bacteraemia and therefore to avoid the complications which may arise from it.

Pathogenesis of occult bacteraemia

Fifty per cent of children aged three months to three years have nasopharyngeal colonisation with *Streptococcus pneumoniae*. Invasion of the blood stream can lead to prompt host clearance with low risk of complications and a full recovery. In others no host clearance occurs with consequent bacteraemia and systemic sepsis. Ideally, it would be prudent to intervene and prevent nasopharyngeal colonisation from occurring in the first instance (e.g. by vaccination). If this is impossible, it would be helpful to identify the children with bacteraemia prior to the development of complications.

Incidence of invasive disease according to pathogen varies with age. In children who are less than one month of age, the predominant organism is group B *Streptococcus*. *Streptococcus pneumoniae* is the commonest pathogen between one and 23 months.

Occult bacteraemia has an incidence of about one to two per cent. The rate is relatively low in those from three to six months: they benefit from protection from maternal antibody and are less ambulatory, and so are at reduced risk of nasal colonisation.

Several studies of febrile children revealed high rates of misdiagnosis of bacteraemia. Clinical indicators that help predict which children will become bacteraemic would be very useful. The Yale observation score (YOS) quantifies clinical features such as quality of cry, reaction to parent, colour, hydration, etc. Each is graded 1, 3 or 5; 1 is normal, 5 severely impaired. The best score is 6 (normal), the worst 3. In a study of 7,000 children, 71% with positive blood cultures had the lowest (best) possible YOS, so it is a poor tool to detect bacteraemic children.⁵

Equally, no difference is shown in the incidence of a social smile in children who are bacteraemic compared to non-bacteraemic. The incidence of bacteraemia does increase the higher the temperature is. A response to antipyretic agent does not predict bacteraemia.

In a study of 411 children with fever and petechiae, 1.5% were known to have sepsis or serious invasive bacteraemia; two had *Neisseria meningitidis*, one had group B *Streptococcus* and three had sepsis with negative blood cultures. All appeared clinically unwell at presentation.⁶

Unsuspected meningococcaemia represents more of a problem. Half of all infections occur in children under two years. It accounts for 0.025% of three to 36 month old febrile well appearing children. Of all children with meningococcal disease, in 25–50% the disease was occult and the child had already been discharged. Clinical and haematological indices do not reliably identify these patients.

Meningitis is caused most commonly by *Streptococcus pneumoniae* (69%). In children with pneumococcal meningitis, 81% will have a fever for >24 hours prior to the diagnosis being made. Of these, half had visited a clinician prior to the diagnosis being made and were discharged home with an alternative diagnosis.

In the US, a new conjugate vaccine has been launched, and 37,000 children were enrolled in a trial of the vaccine versus placebo (who received meningococcal vaccine). There were 48 cases of invasive meningococcal disease and all occurred in the control group.

What strategies should be employed for young febrile children?
In a decision analysis model, a hypothetical cohort of 100,000 children was studied looking at cost versus life years saved. The strategies were no evaluation, clinical judgement based on YOS, blood culture alone, blood culture and treat while results awaited, blood count and culture if the white cell count (WCC) was elevated. Performing a full blood count and a blood culture (if the WCC was greater than 15,000) and then treating was shown to save the most life years at the least cost.

As the conjugate pneumococcal vaccine is introduced, it is likely that the rate of bacteraemia will decrease and this will change current strategies.

In a study of children with high WCCs >20,000 and fever, 40% of those with symptoms of pneumonia had a positive chest X-ray but, more surprisingly, of those with no symptoms or signs of pneumonia, 26% had signs of consolidation on their chest X-ray.⁷ Therefore, chest radiography is a useful investigation in children with fever and raised WCC, even if respiratory examination is normal.

Urinary tract infection occurs in 3.3% males and 6.5% females less than three months of age with a fever >39° C. In young febrile girls risk factors for urinary tract infection were shown to be a younger age, white race, temperature >39° C, fever for more than two days and no other source of infection on examination. Any two of these criteria led to a positive predictive value of 6.4% and a negative predictive value of 0.8%.

In summary, occult bacteraemia is hard to identify using history and examination alone. Invasive testing is recommended, but when and how much depends on the child, family and clinician. Urinalysis and WCC are useful but imprecise, each only having a sensitivity of 80%. Empirical antibiotic therapy does prevent complications, although due to small numbers this has not been conclusively demonstrated in meningitis. Use of the conjugate pneumococcal vaccine should make the routine use of laboratory testing and empirical treatment unnecessary if the rates of bacteraemia remain low.

MENINGOCOCCAL DISEASE: PRESENTATION, STABILISATION, TRANSFER AND OPTIMAL MANAGEMENT

Professor M. Levin, Professor of Paediatrics, St. Mary's Hospital, London

Meningococcal disease shows a great fluctuation in incidence and marked seasonality. In children, a peak incidence is recorded between six months and one year of age, and a second smaller peak in teenagers. In meningococcal disease the concept of a 'golden hour' is very useful; this is a period after the insult commences in which relatively simple interventions to correct the patient's disordered physiology can affect the chances of survival.

Two distinct pathophysiological processes occur. The majority of children presenting with petechial rash and coma have meningitis and the primary problem is raised intracranial pressure. A smaller number have septicaemia and septic shock.

Meningitis commonly presents with a vague short history. Lumbar puncture is a hazardous procedure in patients with raised intracranial pressure, particularly if associated with DIC. Papilloedema is a late sign and its absence does not make it safe to carry out a lumbar puncture. Similarly, CT can exclude other causes but is unreliable in excluding raised intracranial pressure. If the child is deteriorating rapidly or appears shocked, he or she should be commenced on antibiotics, given mannitol and electively ventilated.

In children who present with septicaemia, mortality rate is high. Frequently there is a short history of a petechial rash and shock (signs of end-organ perfusion) with a low WCC and rapid deterioration. Bacteria cause activation of the host's immune system with macrophage activation, release of cytokines, neutrophils and platelets. Endotoxin is a key mediator and is associated with different concentrations of cytokines. A higher mortality rate is recorded in those with higher levels of endotoxin.

In septic shock four basic events in the pathophysiology have to be addressed for successful management: increased vascular permeability, vasoconstriction and vasodilatation of vascular beds, intravascular thrombosis and myocardial dysfunction. Volume resuscitation is vital as these children have leaked their intravascular volume.

Increased vascular permeability occurs in these situations. In normal endothelium small molecules are freely filterable, larger ones are not. Negatively charged molecules such as albumen are restricted, whereas positively charged molecules tend to leak because the glycosaminoglycans on the endothelium are highly negatively charged and are antithrombotic. Release of enzymes from the neutrophils and macrophages results in leaky capillaries due to loss of the negatively charged barrier, and this initiates the increase in vascular permeability. In addition, there is a tendency to intravascular thrombosis with consequent occlusion of blood vessels and end-organ infarction. Normally, when thrombin forms in the circulation, it binds to thrombomodulin on the endothelium. This enables the plasma protein, protein C, to become activated. Protein C requires protein S as a co-factor. The combination of these changes inactivates factor 5a and 8a, and also neutralises plasminogen activating inhibitor. Patients with meningococcal disease are consequently deficient in protein C, protein S and antithrombin 3, and therefore prone to thrombosis. It was thought that if protein C was

given to these patients the risk of thrombosis would decrease. However, protein C requires thrombomodulin on the endothelium to be activated. In meningococcal disease there is virtually no thrombomodulin. Therefore, activated protein C would need to be given and this is now entering trials.

The presence of diffuse intravascular thrombosis leads to coagulopathy with thrombocyclopaemia and purpura, the latter being a poor prognostic sign. Treatment of coagulopathy involves aggressive resuscitation of shock and the use of fresh frozen plasma to correct the depletion of clotting factors. Tissue plasminogen activator may be considered but there is a difficult balance between death from bleeding or clotting. In association with these blood and vascular changes, a release of cardiodepressant factors also occurs. Meticulous correction of any metabolic disturbances such as acidosis, hypoglycaemia, hypocalcaemia, hypokalaemia and hypophosphataemia should ensure no further cardiotoxicity.

Pulmonary dysfunction is also common, and there must be a recognition that children with a capillary leak will 'drown' unless electively intubated. Any child who is still shocked after 40 ml/kg colloid has been given should be electively intubated and ventilated.

Endotoxin binds to endotoxin binding protein and then interacts with the CD14 receptor on macrophages and on the endothelium. This causes the activation of tumour necrosis factor release and alteration of adhesion molecules on the endothelium. Trials investigating the effectiveness of monoclonal antibodies have been unsuccessful. However, a natural neutrophil protein called 'Bacteriocidal Permeability Increasing protein' (BPI) has been found to neutralise endotoxin. A recombinant fragment of this was produced and shown to completely block endotoxin-induced shock in adult volunteers. It has also been administered to children with meningococcal sepsis. Although it reduces amputations and improves outcome, no statistical benefit on death has been shown. Further trials of BPI to be given in A&E departments are now being planned.

The mortality of this disease is still high as pathophysiology is poorly understood. Children are frequently admitted to units unfamiliar with the disease where the patient is deemed 'too sick' for transfer, and it would therefore be prudent to centralise care for these patients.

Children with meningococcal meningitis and raised intracranial pressure also require fluid replacement, elective intubation and mannitol and neurointensive care. Children with meningococcal sepsis require aggressive volume resuscitation, inotropic support and ventilation, correction of factors that impair myocardial function and early contact with intensive care services.

PAEDIATRIC POISONING

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Accidental poisoning is most frequently encountered between one and five years of age. Less than one per cent of poisoning in children is serious. It remains a challenge to identify those at risk at an early stage. Management includes risk assessment and meticulous supportive care.

Various modalities are available to treat the child with potentially serious poisoning. Ipecac (Ipecacachuan), though once a common treatment, is no longer in use. It does not affect the morbidity or mortality in any favourable way and is not recommended. Activated charcoal remains useful for those substances which can be bound by charcoal which are ingested in the hour prior to hospital referral. Multiple dose activated charcoal (1 g/kg every four hours) is used for substances ingested that have a long half life; these include theophylline, anticonvulsants, digoxin and aspirin. Charcoal can cause a charcoal bezoar that may cause obstruction and can also cause disastrous problems if aspirated.

Gastric lavage is recommended if a potentially life-threatening poisoning has occurred during the previous one hour. Gastric lavage is contraindicated if the airway cannot be protected, also in the ingestion of petroleum distillate and corrosive. Children will need to be intubated in most cases and thus stomach washouts are infrequently indicated.

The complications of gastric lavage include aspiration pneumonia, hypoxia, mechanical injury to the gut and the induction of hypo- and hyper-natraemia. It is likely that gastric lavage will be totally abandoned in the future.

Whole bowel lavage is well tolerated in children. It is advocated for the treatment of sustained release lithium, theophylline and iron poisonings. It consists of irrigation with half a litre of polyethylene glycol per hour in the pre-school child. There is still a lack of evidence in whole gut irrigation, and it is limited to case reports at present.

Children frequently ingest household substances and most of these are non-toxic. Household products containing alcohol are concerning as they may provoke hypoxia and fits. Petroleum distillates may cause lipoid pneumonia. Dangerous substances for children include iron, paracetamol, some essential oils, theophylline, tricyclic antidepressants. With iron, serious ingestion occurs if ≥ 60 mg/kg are ingested. Initially there is vomiting, diarrhoea and mild abdominal pain; most children do not progress any further than this. Some occasionally develop coma, fits and severe metabolic acidosis in the initial phase but this is rare. The third phase occurs 12–48 hours after the second (latent) phase with cardiovascular problems, metabolic acidosis, jaundice, clotting abnormalities, hypoglycaemia and renal failure. This phase is associated with a high mortality. Phase four involves the development of gut strictures.

Treatment involves an abdominal X-ray to determine the number of tablets ingested. A patient with serum iron greater than 90 mmol/l may be treated with a chelating agent. It is important to remember that serum iron does not peak until four hours after ingestion. If less than 20 mg/kg are ingested, treatment is supportive. Between 20–60 mg/kg ingestion, gastric emptying may be considered if within one hour of ingestion. Whole bowel irrigation may be of use in patients with ingestion of more than 60 mg/kg and greater than one hour since ingestion. It is especially useful if a slow release preparation has been ingested. Charcoal is of no benefit as iron does not bind to it.

Desferrioxamine has not been shown to be effective in humans but its use continues. It can cause hypotension, anaphylaxis and rashes. If renal failure develops due to its administration, the iron-desferrioxamine complex would have to be removed by haemodialysis.

Theophylline poisoning is rare but serious. Most preparations are slow release so problems develop 12–24 hours after ingestion. It is characterised by nausea, vomiting, haematemesis and diarrhoea. The patient may be hyperactive with dilated pupils, hyperreflexia, hypotonia and myoclonus; other features include arrhythmias, metabolic acidosis, hyperglycaemia and severe hypokalaemia. A theophylline assay should be obtained in these patients, hypokalaemia corrected and gastric lavage should be considered if ingestion has occurred within the previous one hour. Repeated activated charcoal is given and charcoal haemoperfusion considered if the gut is not working. Whole bowel irrigation is considered if a slow release preparation is ingested. Because H₂ blockers slow the metabolism of the drug they are contraindicated.

Tricyclic antidepressant ingestion can be fatal. Doctors often fail to recognise a potentially serious ingestion in children. It is treated with activated charcoal if within one hour of ingestion. Aggressive use of sodium bicarbonate (even in the absence of acidosis) titrating to pH 7.50–7.55 is recommended. This causes sodium channel blockade (reducing arrhythmias) and alters penetration of the tricyclic antidepressant across the blood-brain barrier. In general, meticulous supportive care is critical for a good outcome.

Essential oils are potentially neurotoxic, causing fits even in small amounts. Gastric lavage is recommended if within one hour of ingestion, as is observation of the child for at least 12 hours.

Paracetamol ingestion is common. The treatment is based on the plasma paracetamol level. The children at high risk are those with glutathione depletion, such as children with cystic fibrosis, adolescents with eating disorders and also those on enzyme-inducing agents such as anticonvulsants.

Prevention of childhood poisoning is vital. There must be adequate supervision, safe placement of medications, child safe cabinets and containers, blister packaging and education. Sources of information on poisoning are common and include Tox Base, telephone advice, books, training courses and project Isabel (St Mary's).⁸

In summary, in poisoning, prevention is the key. Most paediatric cases are not severe. Recognition of potentially life-threatening ingestions is important so that appropriate early treatment can be instituted. Aggressive meticulous supportive care is vital.

THE ACUTE ABDOMEN IN PAEDIATRICS

Professor G.G. Youngson, Consultant Paediatric Surgeon, Royal Aberdeen Children's Hospital

In the Grampian region, one in 1,000 children *per annum* will develop acute appendicitis. Each general practitioner will therefore see approximately ten children with acute appendicitis in their working career.

History is vital, yet often challenging, in children. The duration, nature and location of the pain are important. Persisting, unremitting pain for longer than six hours is a good reason for admission to hospital, as is associated bile vomiting. This implies dysmotility distal to the ampulla of Vater and is often associated with an acute surgical problem. Diarrhoea and fever are also common in children with abdominal pain. In a series of patients admitted with intussusception via A&E over half of them were initially admitted with diarrhoea to a general medical ward.

Physical examination in children can also be challenging. Good communication is vital as well as an ability to distract the child while the examination is taking place. The diagnostic process includes a thorough history and examination. Rectal examination may be performed by the operating surgeon. In some cases ultrasound can be helpful, and in North America CT is in common use. White cell count and CRP assay may be obtained. Active observation of the child is a very important diagnostic tool. In 100 children admitted with abdominal pain, 30 will require to go to theatre that day. Of these, 25 will have acute appendicitis, two will have a normal appendix and three will have other surgical pathology. Of the 70 who undergo active observation, on first re-examination five will go to theatre and are shown to be suffering from acute appendicitis. On second re-examination, one will go to theatre and have acute appendicitis, 30 will have non-specific abdominal pain and 34 will have a medical cause for their pain. In children less than five years of age the appendix is often perforated. Inflammation in pelvic and retro-ileal appendices can be difficult to diagnose.

Other surgical causes of acute abdominal pain include intussusception, testicular torsion and volvulus. Volvulus may present with pain, bile vomiting and a relatively soft, flat abdomen. The child may die unless urgent surgery is performed.

Medical causes of the acute abdomen are as follows: diabetic ketoacidosis, *Helicobacter pylori* infection, gastroenteritis, pyelitis and coeliac disease. Gastroenteritis may be caused by *E. coli* 0157, *Cryptosporidium* and *Campylobacter*. Many surgical causes of acute abdominal pain can also present with diarrhoea such as intussusception. Pneumonia can also cause referred abdominal pain and chest radiography can be very helpful. Children's tumours, such as a primary hepatocellular tumour, may present with abdominal pain.

'Non-specific abdominal pain' (NSAP) is common. It is defined as a 'self-limiting condition with no short or long term sequelae'. In a group of patients with NSAP contacted ten years after admission, over half had had no further bouts of pain and a quarter had recurrent NSAP.

In summary, abdominal pain in children is common. The challenge is to use common sense and skill to diagnose effectively and treat appropriately.

TRAINING FOR PAEDIATRIC EMERGENCIES

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Most acutely ill children will undergo primary resuscitation, then a subsequent transfer to definitive paediatric care. Basic care at an early level will greatly affect outcome and for this to be provided, adequate training for paediatric resuscitation is required. In the 1980s Oakley⁹ showed that junior doctors were poor at paediatric resuscitation and this was further confirmed by Buss in 1993.¹⁰ Resident admitting paediatricians were surveyed. Disconcertingly, 50% were unable to give correct adrenaline doses in cardiac arrest or amounts of transfusion fluid.

In 1992/3 Paediatric Advanced Life Support (PALS) and Advanced Paediatric Life Support (APLS) were developed. The study above was repeated in those attending these courses and the answers to every question

were significantly better. Those who had done a training course did significantly better than those who had not. The Paediatric Life Support course is run by the Advanced Paediatric Life Support group. It lasts one day, is locally based and is multi-disciplinary. It involves recognition, basic life support, vascular access and defibrillation. The PALS course is a two day course and was designed by the American Heart Association and the American Academy of Paediatrics. It is based on physiological management such as the recognition of respiratory failure and shock and is recommended for doctors and health care professionals. The Advanced Paediatric Life Support course is a three day course run by the Advanced Life Support group. It examines specific diseases and also contains detailed trauma management, and is primarily aimed at doctors. These courses have improved knowledge and outcomes, and a newborn resuscitation course is currently being piloted.

The European Resuscitation Council (ERC) is currently trying to standardise courses throughout Europe. The ERC has adopted the APLS course which will have to be modified to create a standardised European version.

The important features of a 'good' course should be sought out and acted upon. A good course should be of high quality, high volume and effective. It should be multi-disciplinary but at a level appropriate to the provider's role and experience. Repetition should be avoided and re-certification should be allowed. Teaching should include recognition, basic life support, oxygenation, bag-valve-mask ventilation, volume replacement, defibrillation and specific disease management. Teaching strategies should rely on active learning. Scenarios and group discussions should be used, although this does not allow for a high volume of participants. Simulators can be very realistic and, although expensive and labour-intensive, will provide high quality, low volume work. Computer-based scenarios are being developed. These may be useful for revision and re-certification.

The traditional course is labour-intensive but effective and is likely to continue. It may be modified so that it becomes a modular course. This would involve effective use of instructor time and allow progression of the candidate to the appropriate level. It would avoid repetition and thus is educationally sound.

In conclusion, the courses available are very good but face significant challenges. They have been shown to improve knowledge and outcome. It is likely that a modular course will be developed to meet future requirements.

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