Health education interventions

In the past there has been only limited success achieved by interventions aimed at reducing the prevalence of adolescent smoking. Early interventions depended on ‘fear arousal’, a technique not now thought to be particularly useful. Subsequent interventions have focused on either changing knowledge and attitudes (based on the premise that changes in behaviour will follow) or those which focus on the development of social skills to resist pressures to smoke.

Interventions that have merely used information to try and dissuade children from smoking have not met with a great deal of success. Those that have coupled this information with attempts to change attitudes have been rather more successful, particularly if parents have been involved with the programme. Interventions that have attempted to equip the child with the skills necessary to resist smoking have also generally achieved some success, at least in the short term. However, many of the studies documented suffer from methodological weaknesses, frequently with biased or small samples. The long term effect of many interventions remains unknown. Good longitudinal surveillance remains an essential prerequisite for properly targeted health education.

The task for health education

Despite the greatest incidence in children’s smoking occurring around the age of 14 years it seems likely that, in order to deter the experimental smoker from becoming the regular smoker, interventions to reinforce negative views of smoking need to occur much earlier than this, around the time of likely experimentation.

As the smoking habits of parents, siblings, and teachers are important precursors to adolescent smoking, it is important that the health education of young people is not balkanised, and is seen as part of a broader health promotion strategy, based on the notion of health as a positive attribute, rather than always focusing on ill health and victim blaming. An effective policy to reduce adolescent smoking must promote non-smoking as the norm in the home, at school, and outside in the external environment.

Acute appendicitis in the preschool child

Acute inflammation of the vermiform appendix is probably as old as man. An Egyptian mummy of the Byzantine era exhibits adhesions in the right lower quadrant, suggestive of old appendicitis. That it may become inflamed was recognised by case reports of postmortem examinations in the 1700s and the term perityphilitis was used. Fitz was first to use the term appendicitis and recognised that removal of the inflamed appendix could result in the cure of what had previously been an almost universally fatal condition.

The three major advances in the management of acute appendicitis have been: improved fluid resuscitation, better anaesthetic techniques, and the introduction of antibiotics—more importantly those with activity against anaerobes. Despite these major advances, however, acute appendicitis remains the cause of substantial morbidity.

Overview

Acute appendicitis in the preschool child is rare and accounts for less than 5% of all paediatric admissions with
Acute appendicitis in the preschool child

that diagnosis. It appears to be slightly more common in boys than girls. It therefore presents a diagnostic challenge for both the general practitioner and the admitting surgeon. Between 26–36% of patients with perforated appendicitis had sought advice from a healthcare professional before admission and surgical intervention had not been initiated. The fundamental premise is that earlier diagnosis of acute appendicitis leads to a better clinical outcome. The greater the delay, the greater the risk of perforation. Perforation is associated with a greater morbidity. This is manifest as postoperative septic complications and a longer hospital stay with its associated psychological morbidity.

The perforation rate, however, remains high in the preschool child, approaching 90% in one series. Some workers have suggested that this is due to a thin walled appendix in the child predisposing to early perforation. Indeed in experimental studies Tsuji et al have demonstrated gangrenous changes as early as eight hours after appendiceal luminal obstruction. Others attribute the higher perforation rate to a delay in diagnosis. The reason for this delay is probably multifactorial. The ill child finds it difficult to localise pathology accurately and then communicate to the parents. Parental delay has been identified as a factor contributing to delayed diagnosis.

Presentation and diagnosis

The mainstay of diagnosis remains a detailed history and careful physical examination. The child may have non-specific symptoms, for example, irritability, restlessness, crying, etc. Associated illness, for example, upper respiratory tract infection, otitis media, and gastroenteritis are often present and this may further confound the clinical picture. This may help to explain why 11–24% of these children are admitted to an inappropriate medical service (N Williams, L Kapila, unpublished data). Vomiting is the most frequent symptom in this age group and is present in 75–81% of children. Abdominal pain is not invariably a feature of acute appendicitis but is the next most common symptom. It is more likely to be central than confined to the right iliac fossa. Shift of pain is also less likely in the preschool child. Aggravation of pain by movement and coughing is good evidence of peritoneal irritation. Anorexia, poor feeding, and loss of appetite are also good indicators of an intra-abdominal inflammatory process. Sleep disturbance may be present in over half of cases.

Examination of the very young child in this situation needs patience above all else, otherwise valuable physical signs will be missed in a nervous, anxious child who has just been hospitalised. Tachypnoea may be noticed and can easily be mistaken to indicate a chest infection. It should be remembered that young children inspire with their diaphragms and in the presence of peritonitis they take shorter quicker breaths because of pain inhibition. The physical examination may be rendered more fruitful and informative if carried out in the mother’s lap. Initial observation will permit an overall clinical impression and allow time for detection of more subtle physical signs, for example, abdominal wall movement with respiration. Fever is present in up to 90% of cases of appendicitis. However a normal temperature does not exclude a perforated appendix. The pulse rate on admission is very likely to be affected by anxiety and so isolated readings are of little value. The importance of a sustained tachycardia as a sign of peritonitis is clear, frequently in excess of 120 beats/minute. Local tenderness is present in up to 95% of children with non-perforated appendicitis but in less than 55% of those with perforated appendicitis. Diffuse tenderness but with maximal intensity in the right iliac fossa is indicative of more advanced disease. Guarding is a more objective sign than tenderness but is not as easy to elicit because it is vital to have the child comfortable and relaxed. This may be difficult in an ill, apprehensive child. In this situation light sedation with diazepam or trimeprazine then re-examination may prove beneficial. Not only will the child be more settled, but it will give the surgeon time to assimilate the history and to observe the child at a distance for a while. Auscultation of the abdomen may be useful as absent bowel sounds increases the likelihood of intraperitoneal pathology. The preschool child is more likely to have an appendix mass at presentation. This may create a management dilemma. Conventional thinking is to treat those masses that are palpable preoperatively with intravenous fluids and antibiotics. However up to 55% of masses are only palpable under anaesthetic. Some advocate continued conservative management and would not proceed to appendicectomy if a mass was palpable under general anaesthesia. However other data suggests that early appendicectomy produces comparable if not superior results and the hospital stay is greatly reduced.

The value of rectal examination can only be determined by the assessing surgeon in individual cases. Clearly if a decision is made to operate then it has no part to play. However in doubtful cases and in cases of pelvic appendicitis when the abdominal signs are equivocal rectal examination may prove useful. The elicitation of rebound tenderness has no part to play here; it is cruel and provides no additional information that cannot be otherwise inferred.

Of all paediatric surgical admissions with abdominal pain only approximately 35% will need surgical intervention. Not all of those are obvious at initial assessment and so other ancillary methods and investigations must be employed to further distinguish those who have a surgical cause for their abdominal pain and those who do not. First and foremost there is no substitute for repeated reassessment and re-examination of the child by the same surgeon. Jones calls this ‘active observation’ if only to confirm its implementation as an active management decision and to focus attention on repeated reassessment.

Investigations

The value of haematological investigations is dubious and needs to be seen in perspective. The only value of the white cell count would seem to be to prompt observation rather than operate in a patient who has equivocal features of appendicitis together with a normal white cell count. Plain abdominal radiography for diagnosing acute appendicitis is based on the presence of one or more of the following signs: faecolith of the appendix, gas in the appendix, air-fluid levels, or dilatation of the local bowel (that is, sign of paralytic ileus). However not one of these signs is specific for acute appendicitis and may be found in patients with other causes of pain in the right iliac fossa. Ultrasound assessment lends itself particularly well to this age group because of its non-invasive nature. If the appendix can be seen on ultrasound examination this is taken to indicate the presence of acute appendicitis. If the organ cannot be seen appendicitis is excluded. Rubin and Martin reported a sensitivity of 89% and a specificity of 92% in a cohort of 110 children. In this group three were clinical false positives, however all three had negative ultrasound scans. They also managed to exclude 11 patients in whom the cause of abdominal pain was gastraeological. Clearly ultrasound has a part to play in the diagnosis and management of abdominal pain in childhood and the early reports in the literature has demonstrated its value. Its role in detecting an appendix mass/abscess should obviate the need for an examination under anaesthetic.

Barium enema examination has been reported in adults...
Conclusion

Clearly the way ahead in reducing the morbidity from acute appendicitis in the preschool child is in early diagnosis. This theoretically will reduce the perforation rate and so reduce the septic complication rate and the hospital stay. It is important to appreciate that delays occur both in the community and once the child is in hospital. A greater awareness among healthcare professionals and among parents who may then seek medical advice sooner appear to be the initial steps. However once the child is in hospital every effort should be made to ensure prompt and accurate diagnosis and then the institution of appropriate treatment. The childhood mortality rate in the UK is presently less than 0.00 per 1000 live births. The decade 1976-85 witnessed 105 childhood deaths from acute appendicitis, of whom 29 were under 5 years of age and five were neonates.18 Thus although the preschool child accounts for less than 5% of admission in this cohort, they comprise >30% of the mortality. The last century has witnessed the decline in mortality from acute appendicitis from invariable to very rare indeed. Perhaps the next few decades will see a corresponding fall in the morbidity in this especially high risk group, the preschool child.

N WILLIAMS

Department of Surgery,
Leicester Royal Infirmary,
Leicester LE1 5WW

Screening for neuroblastoma

Neuroblastoma is the most common extracranial solid tumour of childhood with an incidence of 6–10 cases per million children (under 15 years) each year.1 The median age at diagnosis is 2 years and few children are diagnosed after 5 years of age. The tumour originates in the adrenal gland in half of the cases and elsewhere in the abdomen in a further 20%. The remaining tumours arise in sympathetic ganglia in the thorax, pelvis, or neck. Children presenting with localised, resectable (stage 1) tumours can be treated by surgical excision with a high expectation of cure (95% five year survival).2 Unfortunately, symptoms from the disease are non-specific (anorexia, malaise, limb pain, etc.) such that over 50% of clinically presenting children will already have advanced metastatic (stage 3 or 4) disease.3 For these children prognosis is very poor (25% five year survival)3 despite such intensive and expensive treatment as delayed primary surgery, chemotherapy, radiotherapy, and high dose chemotherapy with bone marrow rescue. In addition, neuroblastoma has not shown the dramatic improvement in prognosis over the past 10–15 years seen with other childhood malignancies such as acute lymphoblastic leukaemia, Hodgkin’s disease, and Wilms’ tumour.4 The aim of screening is to detect disease preclinically so that children can be treated when younger and with lower stage disease. As children diagnosed under 1 year of age with localised neuroblastoma are known to have a much better prognosis than older cases,5 screening for neuroblastoma is attractive and might be expected to reduce the morbidity and mortality. How, then, can we screen for neuroblastoma?

Children with neuroblastomas have long been known to excrete catecholamine metabolites in their urine.6 When La Brosse presented a simple test for 4-hydroxy-3-methoxy mandelic acid (VMA) using urine spotted onto filter paper mass screening for neuroblastoma became a possibility.7 Sawada et al were the first to begin mass screening in 1972, testing 42 636 children aged 3 years in Kyoto, Japan, and finding one child with stage 2 disease.8 Mass screening has advanced rapidly in Japan since that time: Nagoya joined the programme in 1977 and Osaka in 1980 and by 1985 a nationwide programme for mass screening was introduced. The age at which children are screened has been reduced to 6 months and the qualitative VMA spot test has been superseded by quantitative measurement of both VMA and homovanillic acid (HVA) by high performance liquid chromatography (HPLC). All 3 month old infants are examined under the Child Health Survey Programme and at this time parents are given a screening kit. When the infant is aged 6 months a urine sample is taken and the filter paper is mailed to the local screening centre. Children with values above mean ±2.5 SD for VMA and/or HVA are examined physically, by chest and abdominal radiography, and by abdominal ultrasound scan. By the end of 1988, a total of 4 018 630 children had been screened (78.9% of all infants) and 342 cases of neuroblastoma had been found, an incidence of 1/11 750.

Dr Sawada and his colleagues have presented extensive data regarding the outcome of children detected by the screening programme.7–9 Seventy-eight percent of cases were detected between 7 and 9 months of age, and 95% of children were asymptomatic. By the end of August 1989, fully 97% of these children were still alive and only three children had died from progressive disease. There has also been a shift away from advanced stage disease at diagnosis...