Rectal prolapse in children

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Summary: Some of the factors thought to be responsible for rectal prolapse in children are reviewed. In the United Kingdom management has in the past been conservative. It is suggested that children should be treated at an early stage by means of an injection of phenol in almond oil, in order to reduce the discomfort of recurrent manipulative reductions of the prolapse in the child and alleviate the anxiety of the parents. In 18 cases treated during the past 3 years a single injection performed under general anaesthesia, as a day case, was successful in preventing further prolapse of the rectum.

Introduction

Over the past 30 years there have been only two meetings on rectal prolapse in children at the Royal Society of Medicine. Thirty years ago the Presidential Address to the Section of Proctology given by Muir (1955) was on the subject of rectal prolapse, with emphasis on management of the condition in the adult. This lecture included a comprehensive review of the history, with an interesting quotation from a short chapter by Ambroise Paré entitled 'Of the falling down of the Fundament' (1634, English translation):

'When the muscle called the sphincter which ingirts the Fundament is relaxed, then it comes to pass that it cannot sustain the right gut. This disease is very frequent to children by reason of the too much humidity of the belly: which falling downe upon the muscle molesteth and relaxeth it or presseth it down by an unaccustomed weight, so the muscles called Levator Ani or the lifters up of the Fundament are not sufficient to beare up any longer. A great bloudy flux gives occasion to this effect. A strong endeavour to expell hard Excrements, the Haemorrhoides, which suppressed doe over-loade the right gut but flowing relaxe it. . .'

Just over 20 years ago, in a symposium on rectal prolapse, Nixon (1962) presented his thoughts on the condition in children. Ambroise Paré's observations 350 years ago were pertinent with regarding to aetiology and the frequent occurrence in children; and since Nixon's presentation 20 years ago, very little has changed regarding either aetiology or management.

Prolapse can be either partial or complete. Opinions vary as to whether the majority of prolapses are partial or complete in children. The partial prolapse is distinguished by the rectal mucous membrane prolapsing for one half to one inch from the anal verge with characteristic radiating folds. In complete prolapse, all coats of the rectum are involved. Two inches or more protrude and the prolapse is distinguished by having circular folds of the mucosa visible. The majority of cases appear under the age of 2 years.

The following anatomical features are thought to be important in the aetiology of prolapse:
(1) vertical course of the rectum;
(2) few lateral and anterior posterior curves;
(3) flat surface of coccyx and sacrum;

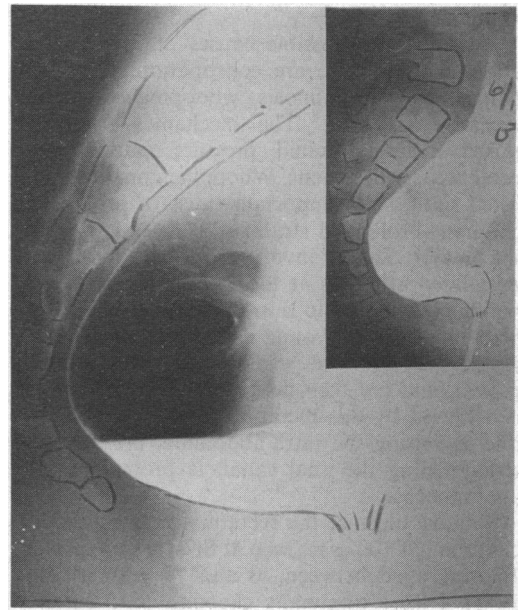

Figure 1. Contrast X-rays showing the pelvis of an adult (left) and a six-month old infant (right). Note increased curve of the adult sacrum

(4) relatively low position of rectum and other pelvic organs;
(5) great mobility of the sigmoid colon;
(6) lack of support by levator ani muscle.

Figure 1 shows the lateral view of an infant's rectum compared to the adult, demonstrating the increased curve of the sacrum and coccyx. With relaxation of puborectalis during defaecation the rectum takes a more vertical course. Excessive straining at stool would tend to prolapse the mucosa more readily than in the adult. Magnus (1967), in a post-mortem study of Houston's valves in 80 stillborn fetuses and infants, stated that Houston's valves are present in only 25% of infants under one year of age. The valves were present in 7 of 8 sigmoidoscopies carried out on children aged 13 days to 13 years; however, she did not think that post-mortem handling of the rectum had any influence on the presence or absence of the valves. We have embarked on a prospective study of barium enemas on infants and newborns, as the lack of valves and absence of anterior and posterior curves does not seem to be a prominent feature in our experience.

Predisposing causes

Predisposing causes of rectal prolapse fall into five categories: (1) raised intra-abdominal pressure; (2) fibrocystic disease; (3) neuropathic, e.g. myelomeningocele; (4) maldevelopment of the pelvis, e.g. ectopia vesicae; (5) 'iatrogenic', following pull-through operation for anorectal anomalies.

Amongst the possible causes of raised intra-abdominal pressure are constipation, diarrhoea, polyps, worms, phimosis, whooping cough and excessive vomiting. The mechanics by which raised intra-abdominal pressure causes rectal prolapse is self evident. Whooping cough, though listed sixth, is an important cause of prolapse in children. Prolonged straining at stool is possibly the major factor involved in causation, and misguided attempts at toilet training in the very young contributed to this. Some parents insist on placing a baby as young as 6 weeks of age on a potty, leaving it to strain until some stool is passed, and are then delighted when defaecation is achieved by this method. The result, however, due to raising the intra-abdominal pressure and straightening the anal canal, is prolapse of the anal mucosa.

Solitary ulcer of the rectum is not common in children. Of 102 cases seen at St Mark's Hospital, 15 were aged between 10 and 19 years (Rutter 1975). I have personally encountered 5 children with solitary ulcers of the rectum: the youngest was aged 10 but gave a 5-year history of passing blood and mucus per rectum. In adults, solitary

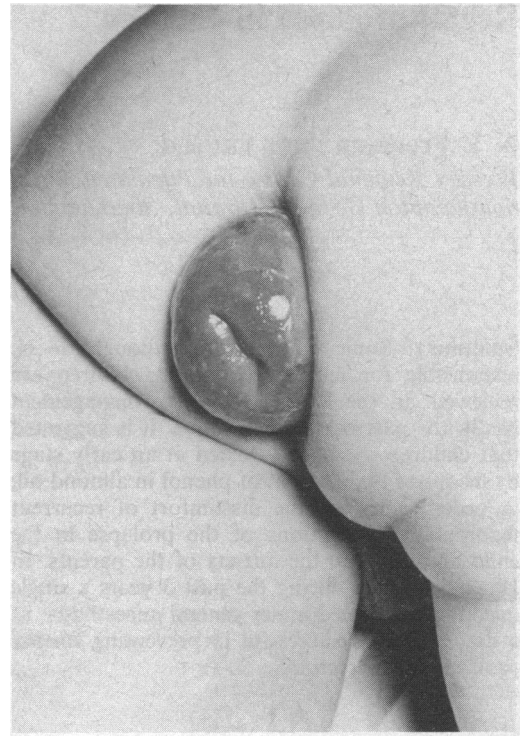

Figure 2. Complete procidentia seen with severe spina bifida

ulcer of the rectum is stated to be due to straining and internal prolapse of the anterior mucosa of the rectum, and pinching by the puborectalis (Rutter 1975). The solitary ulcer is situated on the anterior wall of the rectum within 3–5 cm of the anal verge in 60% of cases. This area appears to be the apex of the prolapse during straining. In the larger series reviewed, men and women were affected in equal numbers. The solitary ulcer of the rectum is characterized by the loss of lamina propria with infiltration of this by muscle from the submucosal layer. The aetiology is uncertain. Digital trauma has been mentioned as a possible cause but very few patients admit to this. In adults with marked descent of the perineum it is thought that excessive straining pulls on the pudendal nerves as they leave the pudendal canal, adding neurological damage of the sphincter, which leads to further relaxation of the anus (Henry 1980). This is the most likely cause of the patulous anus although other causes must be borne in mind. Homosexual activity has been suspected but not confirmed in the majority of cases, even when the patients were carefully questioned.

Nelson's 'Textbook of Paediatrics' states that 'cystic fibrosis is by far the most common cause

of prolapse of the rectum in infancy and childhood . . . the pathogenesis is not clear but relief follows pancreatic replacement therapy'. In a review of 386 cases of fibrocystic disease, prolapse was noted 85 times (Kulczycki & Schwachman 1958). In my own practice only 2 cases of fibrocystic disease have been encountered, and a personal postal survey of Wessex paediatricians in 1983 confirms that rectal prolapse is uncommonly associated with fibrocystic disease, occurring in 10% of cases. Malnutrition, lack of the supporting tissues around levator ani and the ischiorectal fossa may be factors of importance favouring prolapse in fibrocystic disease but the aetiology is not clear.

The neuropathic causes with complete prolapse, as seen in myelomeningoceles, are rare. The paralysis of the levator ani muscle with raised intra-abdominal pressure leads to procidentia and prolapse (Figure 2).

In ectopia vesicae due to wide separation of the symphysis pubis and the puborectalis muscle, a wide hiatus is created which predisposes to prolapse of the pelvic organs and rectum. I have referred to the full-thickness prolapses which occur following rectal pull-through operations for high anorectal anomalies as 'iatrogenic'. Some of the anatomical features listed above may be contributory factors. Sacral agenesis is often associated with high anorectal anomalies and the missing sacral segments shorten and flatten the curve of the sacrum. The non-development of the myotones embryologically result in poor development of the levator ani muscle itself. Furthermore, during surgical repair the 'rectum' may be pulled through with very little anterior curvature when puborectalis is not carefully identified. There is no attempt at fixing the colon within puborectalis when the pull-through operation is done. The complete prolapse which results is highly unsatisfactory both cosmetically and functionally. It causes marked distress to parents and frequently requires secondary surgery.

Secondary surgery can be directed along the lines of trimming the excess colon or attempting to reconstruct a skin-lined anal canal. To overcome the problem I have designed an operation whereby the prolapsed colon is dissected back into the perianal tissue and a full-thickness skin graft, using the patient's own foreskin, is inserted into the perianal space to create a skin-lined anal canal (Freeman 1984).

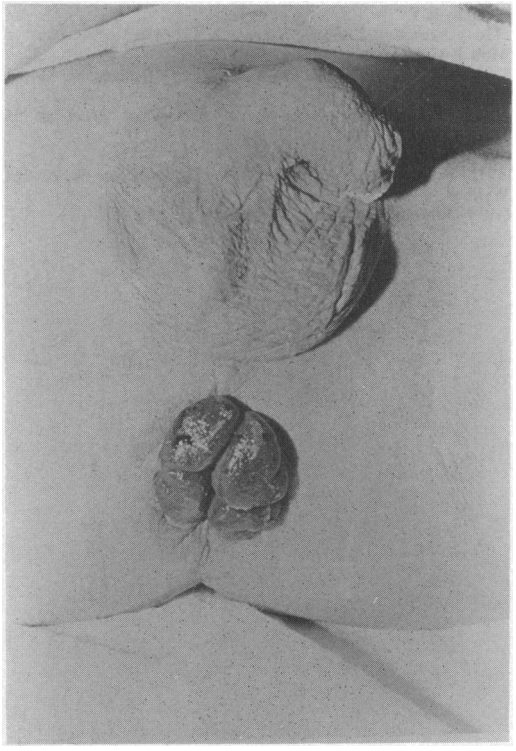

Figure 3. Preoperative appearance of 'iatrogenic' complete rectal prolapse

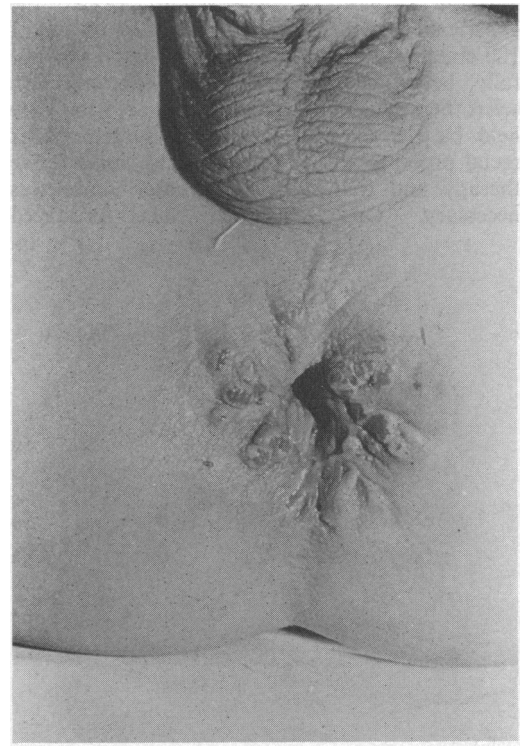

Figure 4. The anus following a 'foreskin anoplasty'. Complete take of the skin graft for 1–2 cm within the anal canal is well shown

The results in 6 cases treated in this manner have been very satisfactory (Figures 3 and 4). Two of the patients have virtually normal control and are attending normal schools, without soiling, except on the very rare occasions when they have liquid stools.

Treatment

In some cases of prolapse there is spontaneous cure. Suggested treatment methods are manual replacement; strapping of buttocks; altering the position of defaecation on the potty, i.e. raising the potty on books, using an adult-type seat, or trying to get the child to defaecate whilst lying on the side; injection of the prolapse with 30% saline, 3% quinine, 1% sodium morrhuate or 5% phenol; multiple linear thermocauterizations to the mucosa; excision of redundant mucosa; Tierch operation; transabdominal sigmoidopexy. The first three are the usual methods recommended by most British surgeons.

Fifty years ago Fraser (1930), working at the Belfast Hospital for Sick Children, stated that 'one can not attend a children's hospital without being struck by the large number of children who suffer from rectal prolapse'. He reported 50 cases successfully treated by a submucosal injection of absolute alcohol. In 1979 Wylie reported the experience at the Adelaide Children's Hospital of 100 children with rectal prolapse treated successfully between 1963 and 1979 with injection sclerotherapy. Prior to this I had shared the view held by the majority of British surgeons that rectal prolapse always responded to conservative therapy and that no surgical interference was necessary. On reflection, this produced unnecessary and prolonged anxiety for both patients and parents, as sometimes the prolapse continued for a year or 18 months. My policy now is to inject any prolapse which has been present for more than a few weeks.

Over the past 3 years I have treated 18 cases, about the same frequency as that of Wylie in Adelaide, but not of Fraser 50 years ago. The procedure was carried out under general anaesthesia, as a day case. Phenol 5 cm^3 in almond oil was injected submucosally about 2 cm above the anal valves in four sites. There were no complications, all cases responded to a single injection, and the parents were delighted. The response to submucosal injection rather than extrarectal injection confirms the majority view that rectal prolapse in children is more likely to be partial than complete.

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