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Paediatric intussusception

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Intussusception is one of the commonest causes of intestinal obstruction in infants and accounts for about 700 hospital admissions each year in England and Wales. Improved results of treatment have followed recent technological developments, which include ultrasonographic imaging and pneumatic reduction techniques. Most intussusceptions can be reduced successfully without the need for operation but close cooperation between surgeon and radiologist is essential. Mortality and morbidity rates from the condition have progressively declined in recent decades but avoidable deaths still occur.

Intussusception, the invagination of one portion of the intestine into an adjacent segment, occurs infrequently yet is one of the commonest abdominal emergencies in paediatric surgery. The condition has been recognized for hundreds of years but was first accurately described by John Hunter[1] in 1793 (*Figure 1*). During the 19th century, reduction by means of hydrostatic enemas, rectal insufflation of air or gas, or manipulation with a wand were all attempted[2]. The first successful operative reduction was performed by Jonathan Hutchinson[3,4] in 1871.

In 1876, Hirschsprung published the first of several papers on the reduction of intussusception by hydrostatic pressure[5], and in 1926 Hipsley reported a series of 100 patients treated with this technique[6]. One year later, several authors[7–10] independently described the use of radiologically controlled contrast enema reduction, which became the definitive non-operative method of management after its popularization by Ravitch and McCune[2,11] in 1948.

Incidence and epidemiology

Many of the epidemiological studies of intussusception were performed more than 30 years ago when its incidence varied from 1·5 to 4·3 per 1000 live births[12–15]. Recent data suggest that the figure may be higher in China[16] but is about 2·4 per 1000 live births in Sweden and New York[17,18]. In Scotland the incidence may be declining[19] but in England and Wales[20] hospital admissions remain at about 700 per year. Annual fluctuations may reflect environmental influences such as viral epidemics[21]. In temperate climes, a seasonal incidence has been reported with a peak in summer[17,22–24] but this has not been an invariable finding[2,12]. In Nigeria there is a greater proportion of older children with caecocolic intussusception and subacute or chronic presentations[25,26], and in southern Africa colocolic sigmoid intussusception is peculiarly common[27].

The condition can present at any age but only 10–25 per cent of cases occur after the age of 2 years, the peak incidence being between the fifth and ninth months of life. It may occur in the neonatal period[28,29] and even prenatally as a rare cause of intestinal atresia[30,31]. Overall, 60–70 per cent of intussusceptions arise in males[2,12,22,32], with a greater proportion of boys in older age groups[12]. Affected infants are no better nourished than normal[17] and up to 16 per cent may be malnourished[33].

Pathology

Intussusception results in compression and angulation of the mesenteric vessels of the invaginated bowel, leading to a strangulating obstruction. Necrosis begins in the outer layer of the intussusceptum at its apex and extends proximally[34]. The intussuscipiens may suffer from secondary ischaemic pressure necrosis resulting in free perforation or prolapse of the intussusceptum into the peritoneal cavity[35]. In animal studies, pathogenic bacterial translocation to the serosal surface of the bowel rapidly occurs even with viable intussusceptions[2].

The condition has been classified according to the site of the inner intussusceptum and outer intussuscipiens. More than 80 per cent involve the ileocaecal region, with the isolated ileoileal, caecocolic, colocolic and jejunojejunal varieties being progressively rarer. Ileocolic intussusception usually starts several centimetres proximal to the ileocaecal valve as a short ileoileal invagination which then advances into the colon, but some have a long ileoileal component. Approximately 80 per cent of ileocolic intussusceptions have their apex in the ascending or transverse colon[2].

Multiple small bowel intussusceptions, either antegrade or retrograde, have been observed at autopsy (agonal intussusception) and are thought to be caused by small bowel dysmotility around the time of death. Similar multiple, short-segment invaginations have occurred either spontaneously[36] or in survivors of abdominal trauma[37]. Isolated retrograde small bowel intussusception has been described[38] and both gastroduodenal and duodenogastric invaginations have been reported as complications of gastrostomy tubes[39,40].

There is abundant evidence that some acute episodes resolve spontaneously, either by reduction or by sloughing and passage

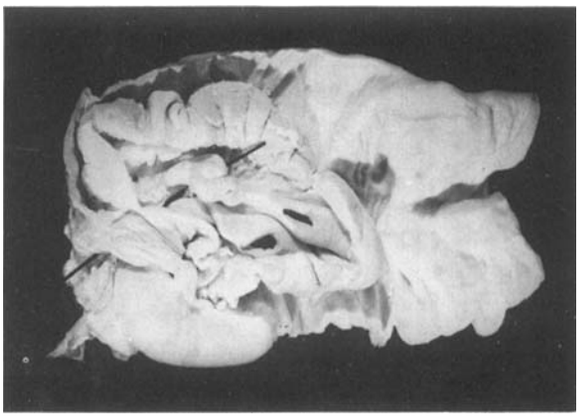

Figure 1 *John Hunter's specimen of an ileocolic intussusception from a 9-month-old boy. The invaginated terminal ileum and appendix have been fenestrated. Reproduced by kind permission of the President and Council of The Royal College of Surgeons of England*

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Table 1 *Pathological lead points in intussusception*

Structural
Meckel's diverticulum[69–72]
Ectopic gastric mucosa[73,74]
Ectopic pancreas[48]
Appendix (normal, appendicitis or mucocele) or appendiceal stump[43,61,75,76]
Enteric duplication cyst[2,77,78]
Anastomotic suture line[70,79]
Vascular/haematological
Henoch–Schönlein purpura[80]
Haemophilia[22,81]
Abdominal trauma[82]
Leukaemia[83,84]
Idiopathic thrombocytopenic purpura[85]
Haemolytic–uraemic syndrome
Neoplastic
Polyps (adenomas, hamartomas (Peutz–Jeghers, juvenile), inflammatory fibroid polyp, lymphoid)[17,71,72,86–88]
Small bowel carcinoid tumours[71,89]
Haemangioma[72,89,90]
Lymphangioma[91]
Lipoma[92]
Adenomyoma[93], mesenchymoma
Leiomyoma[94]
Lymphoma/lymphosarcoma*[95,96]
Fibrosarcoma[72]
Metastatic tumours[84]
Colonic adenocarcinoma[97]
Inflammatory
Foreign bodies, e.g. trichobezoars[98,99]
Granulomas (amoebic, eosinophilic, Crohn's)[100,101]
Ascaris lumbricoides[89,102]
Cystic fibrosis*[103]

*Particularly subacute or chronic intussusception in older children

of the gangrenous intussusceptum with union of the bowel at the neck of the intussusception[41–46]. Short, transient invaginations are occasionally found during laparotomy for other conditions and sometimes there is a past history of a suspected, spontaneously resolving episode in a patient with proven intussusception[2].

Aetiology

Primary idiopathic intussusception

The wall of the terminal ileum of infants is rich in lymphoid tissue and in approximately 90 per cent of cases there is no obvious cause other than hyperplasia of this lymphoid tissue[47]. An upper respiratory tract infection or enteritis commonly precedes development of the condition[2,48]. Adenoviruses, and to a much lesser extent the rotavirus, have been implicated in up to 50 per cent of cases[49–53]. Because many infants are immunized during the period of peak incidence of intussusception the two events may coincide, but there is no discernible relationship with polio virus immunization (M.D.S., unpublished observations). Other potential factors of aetiological importance include changes in the diet of the weaning infant, loss of passively acquired maternal immunity and the disproportionate diameters of the infant's ileum and caecum; there are no hard data on these aspects.

Lymphoid hyperplasia may be responsible for the lead point in idiopathic intussusception, but does not explain why some infants develop the condition and others do not despite exposure to similar environmental influences. The condition has not been regarded as having any hereditary basis[2] but large series have demonstrated that 1–3 per cent of children have affected siblings[12,54]. This could be explained by common exposure to an environmental precipitant such as a virus[55] but this mechanism does not account for the occurrence of the condition in sequential generations[56,57]. In some families the incidence of recurrent intussusception raises the possibility of inherited predisposing anatomical anomalies such as malfixation of the ileocaecal mesentery.

An unfixed caecum, attached to the posterior abdominal wall by way of a mesentery, is found at autopsy in 20 per cent of infants[58] and a high mobile caecum with failure of complete intestinal rotation has been implicated in the pathogenesis of intussusception[25,59–62]. Intussusception accompanied by malrotation, with or without mid-gut volvulus, has been described by several authors[17,59,63–67]. By way of its association with malrotation, the condition has a link with duodenal stenosis, which may explain why some affected infants have failed to thrive[68]. The incidence and significance of mid-gut malrotation and its relationship to intussusception deserve further investigation, but mid-gut volvulus does not appear to have been reported as a late complication of non-operative reduction.

Secondary intussusception

In 2–12 per cent of patients the condition is secondary to a distinct pathological lead point or underlying abnormality (*Table 1*)[17,21,23,43,48,69–104]. With the exception of neonates, secondary intussusception is progressively more likely with advancing age and in those with recurrent intussusception. After the age of 2 years, a pathological lead point is found in one-third of patients[72] and in one-half of those with a recurrent condition[32], whereas under the age of 2 years secondary intussusception occurs in less than 4 per cent[72,105].

Both Meckel's diverticulum and duplication cysts can cause intussusception throughout childhood; the former is the commonest pathological lead point. Intestinal polyps and lymphomas affect older children[71,72,96]. Small bowel tumours are rare in children but most often present with intussusception[71]. Over the age of 3 years, ileal lymphoma or lymphosarcoma should be considered in all patients, especially in those with chronic ill health or an irreducible intussusception[95,96]. To our knowledge, the condition has not yet been reported in association with small bowel tumours or atypical infections in children with the acquired immune deficiency syndrome. Subacute presentation of ileocolic intussusception occurs in about 1 per cent of 9–12-year-old children with cystic fibrosis and is thought to be precipitated by abnormally viscid bowel contents[103,106].

Postoperative intussusception affects both sexes equally but accounts for only 1–2 per cent of all cases[17,22,107]. These are commonly ileoileal invaginations and 90 per cent are confined to the small bowel; this form has occurred after a wide range of procedures (*Table 2*)[76,107,108]. The operation need not directly disturb the abdominal contents, e.g. cervical lymph node biopsy[109], ventriculoatrial shunt revision[110], inguinal herniotomy or thoracic surgery[79].

Table 2 *Surgical procedures preceding postoperative intussusception in 178 patients*[17,76,79,107,108]

Procedure	No. of patients
Abdominoperineal pull-through procedures	
Hirschsprung's disease	15
Anorectal anomalies	7
Appendicectomy	17
Reduction of ileocolic intussusception*	16
Meckel's diverticulectomy	9
Nissen fundoplication	16
Wilms' tumour excision	13
Neuroblastoma excision	14
Urinary tract surgery	
Coloureterostomy or ileal conduit	4
Cutaneous ureterostomy	3
Open bladder biopsy	1
Bladder augmentation	1
Miscellaneous abdominal and extra-abdominal operations	62

*Excludes recurrence of ileocolic intussusception

Table 3 *Frequency of classical symptoms and signs in recent studies of intussusception*

Reference	Year	Place	No. of patients	Abdominal pain (%)	Vomiting (%)	Rectal bleeding* (%)	Symptom triad (%)	Abdominal mass (%)
Hutchison *et al.*[42]	1980	Glasgow	209	82	80	46	—	50
Pollet[104]	1980	Aberdeen	77	89	97	45	38	56
Raudkivi and Smith[112]	1981	New Zealand	98	49	81	63	20	61
Man *et al.*[113]	1983	South-east England	75	87	84	61	46	70
Sparnon *et al.*[23]	1984	Australia	128	81	74	56	45	56
Liu *et al.*[67]	1986	Dublin	66	89	89	50	—	74
Beasley *et al.*[32]	1987	Australia	580	84	91	63	—	79
Bruce *et al.*[17]	1987	USA	583	85	68	37	21	48
Mackay *et al.*[24]	1987	Australia	91	93	95	46	—	77
Winstanley *et al.*[114]	1987	Manchester	78	83	89	65	20	60
Wilson-Storey *et al.*[61]	1988	Edinburgh	125	88	86	56	>50	63
Mean				83	85	53	32	63

*Overt rectal bleeding or noted on rectal examination

That intussusception after operation is almost always limited to the small bowel, and that it most often follows certain types of operation, suggests that its pathogenesis is different from that of the commoner idiopathic form. A neurogenic mechanism producing intestinal dysmotility is the most likely cause and anaesthetic agents may also be important. Small bowel intussusception has been reported after the administration of vincristine, which is known to cause intestinal dysmotility[84], and after faradic stimulation of the bowel or premotor cortex of experimental animals[2,111].

Clinical features

Acute ileocolic intussusception

Classically, intussusception is said to present in a previously healthy baby who develops repeated screaming attacks accompanied by pallor and drawing up of the legs. Between episodes the infant initially appears well but later becomes restless and refuses feeds. Vomiting follows and becomes bilious. A normal stool may be passed followed by blood from the anus, either in the form of pink mucus or 'redcurrant jelly'. Progressive dehydration and strangulating intestinal obstruction are usually fatal within 5 days.

The frequency of classical signs and symptoms is age and site related (*Table 3*)[112–114]. Vomiting is the commonest feature and may be the sole presenting sign in infants, while older children tend to complain of colicky abdominal pain. Constipation may be a late feature. The intussusception prolapses through the anus in 1–2 per cent of patients[17,42,48].

The typical lesion is palpable as a tender 'sausage' in the right hypochondrium or epigastrium but a mass may be discovered at any site along the line of the colon[48,61]. The detection of a mass depends on the skill of the clinician and the degree of abdominal relaxation or distension in the infant. Rectal examination often reveals blood and rarely the apex of an intussusceptum. Late signs include abdominal distension, hypovolaemic shock and peritonism. Fever is common and hyperpyrexia indicates a poor prognosis[2,20].

Atypical presentation results in delayed diagnosis and treatment[115]. Diarrhoea occurs in 7–30 per cent of patients[17,23,24,42,48,114,115] and the erroneous diagnosis of gastroenteritis may lead to a fatal delay in treatment[2,20,115]. The apparently 'painless intussusception' occurs in 13–20 per cent of patients[23,42,116], who take twice as long to be admitted and have a greater incidence of irreducibility than those with obvious colic[116]. Some infants present with profound listlessness and apathy prompting unnecessary neurological investigations[117,118]; endogenous opiate release has been suggested as a cause[119].

Acute small bowel intussusception

Intussusceptions confined to the small bowel account for fewer than 10 per cent of paediatric cases[17,22,120]. Presentation is usually with vomiting and pain, and rectal bleeding or a palpable mass is rare. Contrast studies are often unrewarding[76,107] and the diagnosis is frequently delayed until laparotomy for intestinal obstruction is undertaken[121].

There are three situations in which small bowel intussusceptions are over-represented: in the child aged over 2 years[105,122]; in those with a pathological lead point[72]; and in the postoperative period. Two-thirds of intussusceptions following operation develop within 1 week of surgery[76,107].

Chronic intussusception

Occasionally, intussusception presents in the older child with a history lasting 2 weeks or more, when the condition is non-strangulating and incompletely obstructing[123–125]. Various sites have been recorded, most commonly ileocolic. Typically there is intermittent abdominal pain, anorexia, vomiting and weight loss, with altered bowel habit, particularly diarrhoea[105,124,125]. Rectal bleeding is rare but an abdominal mass may be intermittently palpable. Malabsorption and protein-losing enteropathy may be the dominant features[124,126]. Ultrasonography or contrast studies usually establish the diagnosis.

Although uncommon, chronic intussusception may occur in cystic fibrosis[103] or with intestinal lymphoma[95,96] (*Table 1*). Hydrostatic reduction is rarely successful and operation is usually required.

Diagnosis

A combination of clinical, ultrasonographic, radiological and operative techniques may be necessary for complete diagnosis. Plain radiography commonly shows a paucity of gas in the right iliac fossa with the intussusception visible as a soft tissue opacity in up to 50 per cent of patients (*Figure 2*). Small bowel obstruction is a late sign.

Recently, ultrasonography has emerged as a sensitive and specific diagnostic tool in the condition. On longitudinal section the mass has a tubular appearance[127–129] and in the transverse plane there is a typical target or 'doughnut' with a transonic peripheral rim, corresponding to the oedematous wall of the intussusceptum, surrounding the echogenic central area which represents its lumen (*Figure 3*). The thickness of the transonic rim may help to predict the chances of success with non-operative reduction[128,129]. The experience of the authors accords with that of Pracros *et al.*[130] who found neither false

Figure 2 *Plain abdominal radiograph demonstrating the soft tissue mass (arrowed) of an ileocolic intussusception*

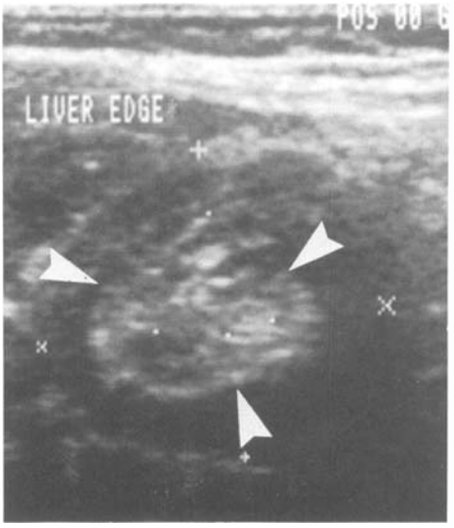

Figure 3 *Typical ultrasonographic appearance of an ileocolic intussusception demonstrating the target sign. The transonic peripheral rim of the intussusceptum is surrounded by crosses and the echogenic central lumen indicated by arrows*

positives nor negatives, although some authors have reported the occasional false-positive result[127,128]. The experience of the ultrasonographer is important and if the diagnosis remains uncertain a contrast study must be performed.

Important benefits of ultrasonography include the ability to detect pathological lead points such as Meckel's diverticulum[131], small bowel lymphoma[127] and enteric duplication cysts[78,132], which must be distinguished from fluid trapped within the invagination[133]. Enlarged mesenteric lymph nodes and free peritoneal fluid are often discovered by ultrasonography and in some patients it is possible to identify the lymphoid hyperplasia of an apparently idiopathic presentation[78]. An expert ultrasonographer may readily diagnose intussusceptions confined to the small bowel, which is a great advance because these are often impossible to detect clinically or by conventional radiology. This is of particular value in patients who have had operations and in those with Henoch–Schönlein purpura or other haematological problems, since it may obviate the need for unnecessary laparotomy in a group of particularly sick children in whom diagnosis is always difficult[44,127,134,135].

Colonoscopy and computed tomography have been used to diagnose intussusception[136,137] but the combination of ultrasonography and conventional radiology is simpler and safer in paediatric practice.

Treatment

Successful management of intussusception depends upon early diagnosis, resuscitation and prompt reduction. Inadequate fluid replacement is a major factor in 20 per cent of deaths[20]. The current preference of the authors is to use a colloid, such as 4·5 per cent human albumin solution, beginning with a bolus of 20 ml/kg in the hypovolaemic child, together with blood if necessary, and crystalloids for maintenance of blood glucose and serum electrolyte concentrations. Nasogastric aspiration and appropriate broad-spectrum antibiotics must be used, and continued in the patient undergoing operation to avoid deaths from aspiration pneumonitis and septicaemia[20].

Non-operative reduction

It is now generally accepted that non-operative reduction should be attempted in most patients with acute idiopathic intussusception[48]. Radiological reduction techniques have been criticized for their high recurrence rates, potential to overlook pathological lead points, rare perforations, incomplete success, and radiation exposure, but these risks are more than offset by the morbidity and mortality surrounding operative reduction with its longer and more expensive recovery period[17,67,113,120]. Both hydrostatic and pneumatic reduction methods are available with monitoring by fluoroscopy or ultrasonography. There is a wide variation in the application and methodology of these techniques and their results (*Tables 4* and *5*)[138–147].

Non-operative reduction should not be attempted in units where paediatric surgical facilities are unavailable and ideally the surgeon should be in attendance to monitor the infant's condition. Resuscitation should be continued during reduction; effective sedation and analgesia improve the chances of success[148]. The authors use intramuscular pethidine compound (0·06–0·08 ml/kg) but others have recommended intravenous diazepam (250–300 μg/kg)[67], papaveretum (0·2 mg/kg)[139,149] or general anaesthesia[143,144,150]. Abdominal manipulation, antispasmodics and glucagon are of dubious value[151] but thermal environmental control is important. Some authors recommend a Foley balloon catheter[48,149] but we advocate a soft rubber rectal tube held in place by firm buttock strapping. Traditional practice dictates a barium column of 1 m for a maximum of 10 min if the intussusception is static[48], but efforts at radiological reduction should be tailored to the condition of the patient and the progress of the reduction. Steady progress should encourage persistent pressure in the stable infant. Radiographic screening times should be kept to a minimum. Reduction is deemed to be satisfactory only if there is free reflux of barium into the small bowel without a residual filling defect, *together with resolution of symptoms and signs in the patient.*

Absolute contraindications to non-operative reduction are signs of peritonitis or perforation, demonstration of a pathological lead point, and the presence of profound shock (although the last group of infants can undergo successful reduction after appropriate resuscitation[2,67] despite a greater risk of iatrogenic colonic perforation[152]). Perforation of the colon during hydrostatic reduction is a rare event, occurring in up to 3 per cent of attempted reductions (*Table 4*), but if promptly recognized and treated is often followed by complete recovery[89,142,152–154]. Perforations during hydrostatic reduction have occurred in ischaemic areas of the colonic intussuscipiens or in necrotic areas in the outer wall of the reducing intussusceptum[152,153].

Relative contraindications to hydrostatic reduction include chronic and neonatal intussusceptions, when the history is in excess of 48 h[67,89,155,156], if there is evidence of

Table 4 *Recent results of hydrostatic reduction of intussusception*

Reference	Year	Place	No. of patients	Attempted reductions* (%)	Successful reductions† (%)	Perforations‡ (%)	Recurrences§ (%)
Hutchison *et al.*[42]	1980	Glasgow	209	27	58	0	—
Man *et al.*[113]	1983	South-east England	75	44	18	0	0
Sparnon *et al.*[23]	1984	Australia	128	75	50	0	17
Liu *et al.*[67]	1986	Dublin	66	90	75	0	10
Mackay *et al.*[24]	1987	Australia	91	99	78	1·1	17
Winstanley *et al.*[114]	1987	Manchester	78	69	50	0	8
Zamir *et al.*[138]	1987	Israel	61	95	50	0	—
Paes *et al.*[139]	1988	Southampton	89	80	42	1·4	17
Phelan *et al.*[140]	1988	Australia	602	60	53	0	14
Wilson-Storey *et al.*[61]	1988	Edinburgh	125	61	38	1·3	10
Skipper *et al.*[141]	1990	USA	157	89	66	0·7	21
Palder *et al.*[142]	1991	Canada	100	100	75	3·0	10

*Attempted reductions: percentage of patients in whom hydrostatic reduction was attempted. †Successful reductions: percentage of successful reductions in those patients undergoing attempted reduction (excludes recurrent intussusceptions and those in whom intussusception was found reduced at laparotomy after an initially failed reduction). The optimum figure is not 100 per cent since gangrenous bowel and pathological lead points should not be reduced. ‡Perforations: percentage of patients undergoing attempted reduction in whom perforation occurred. §Recurrences: percentage of patients undergoing successful reduction in whom there was a recurrence

Table 5 *Recent results of pneumatic reduction of intussusception*

Reference	Year	Place	No. of patients	Attempted reductions (%)	Successful reductions (%)	Perforations (%)	Recurrences (%)
Guo *et al.*[16]	1986	China	6396	—	94	0·1	—
Jinzhe *et al.*[143]	1986	China	2496	—	91	0·1	—
Tamanaha *et al.*[144]	1987	Japan	222	93	81	0	7
Stringer and Ein[62]	1990	Canada	152	>95	80	1·1	8
Todani *et al.*[145]	1990	Japan	137	93	91	0	9
Glover *et al.*[146]	1991	Australia	226	88	75	0·5	7*
Shiels *et al.*[147]	1991	USA	75	100	87	0	—
Pablot and Stringer (unpublished data)	1991	South-east England	46	87	75	2·4	5

See footnote to *Table 4*. *Estimate from Phelan *et al.*[140]

small bowel obstruction on plain radiography[67,153,156,157], and in children older than 2 years[29,152,155,158]. In isolation, none of these features is an absolute contraindication to attempted reduction but, in combination, the chances of success are considerably reduced and the incidence of complications significantly increased[152,155].

Most authors have found little correlation between the length of the invagination and the success of its reduction[18,120,159], although the latter is less likely once the apex has reached the rectosigmoid region[159]. Infants under 3 months of age are reported as having a reduced rate of reduction and a higher complication rate[155,158], but this is not a universal finding[156]. In neonates the relatively high frequency of pathological lead points[29] and irreducibility[160], together with diagnostic difficulties such as confusion with necrotizing enterocolitis[161], indicate a low threshold for laparotomy.

Success rates with hydrostatic reduction[18] have varied from 20 to 80 per cent (*Table 4*) and reflect local referral patterns, selection criteria, and the skill and determination of the radiologist. Gangrenous intussusceptions and those confined to the small bowel or with a pathological lead point are unlikely to be reduced[43,70–72,142,154,162,163] but some of the ileoileocolic form can be reduced, albeit with difficulty[142,152]. Successful hydrostatic reduction does not exclude a lead point[157]. The radiographs of older children must be carefully examined after reduction to exclude a filling defect, especially in the terminal ileum[163]. Ultrasonographic findings are of particular help in identifying the rare instances of a reduced pathological lead point.

Following the publication of several enormous series from China[16,143], there has been renewed interest in the technique of pneumatic reduction, a procedure successfully employed in the 19th century[2,164] and reintroduced in combination with manometric and fluoroscopic control[165] in 1959. This provides a cleaner, cheaper and more rapid method of reduction involving less distress and exposure of the patient to radiation[62,146]. As yet there are no controlled trials comparing the relative merits of air, carbon dioxide or oxygen in pneumatic reduction. With this technique, the chosen gas is carefully pumped into the rectum up to a pressure of 80 mmHg. With experience, the pressure may be increased to a maximum[62,146,147] of 120 mmHg. A low gas flow and pressure should be used at first because at higher rates some intussusceptions are reduced within seconds. The procedure is monitored by fluoroscopic control (*Figure 4*) and most invaginations[143] are reduced within 5 min.

Colonic perforation is rare[16,146] (*Table 5*) but in this circumstance free intraperitoneal air is said to be less hazardous than barium[62,147]. None the less, a pressure release device and a wide-bore needle must be available to deal with tension pneumoperitoneum. As with hydrostatic reduction, pathological lead points and ischaemic bowel are generally irreducible although there is one reported instance of a late ischaemic ileal stricture[146]. Studies employing historical controls have

indicated that pneumatic techniques are more effective than hydrostatic methods of reduction, possibly because gas more readily decreases the frictional forces between the intussusceptum and the intussuscipiens[62,140,146].

Pitfalls in treatment by barium or gas enemas have been described, relating mostly to the uncertainty of complete reduction because of overlapping loops of barium-laden colon[48], difficulty in distinguishing a persistent filling defect due to oedema from that caused by a pathological lead point or an incompletely reduced intussusception[166,167], and problems in the identification of free air reflux in the presence of gaseous small bowel dilatation[62]. In isolation, free reflux of barium or gas into the distal ileum does not completely exclude the possibility of a residual ileocolic intussusception[166,168] or an ileoileal component[138,142], so observation over the ensuing 24 h is mandatory. Persistence of clinical signs or a residual filling defect on postevacuation radiography is an indication for laparotomy, albeit through a Lanz gridiron incision.

In the stable patient, some authors have recommended a second attempt at radiological reduction[16,67]. This appears to be safe and justifiable in the stable, resuscitated infant during a period of up to 2 h after the first attempt[16,67], particularly when the intussusception has been reduced to the caecum. We are against later attempts at non-operative reduction[149] because of the rapidity of clinical deterioration in some infants, and recommend early laparotomy.

There have been some recent reports of successful ultrasonographic monitoring of hydrostatic[162,169] or pneumatic[145] reduction procedures in an effort to eliminate exposure to ionizing radiation. Successful reduction in 95 per cent of 377 patients, with no deaths and with only one colonic perforation, was reported in one series[162].

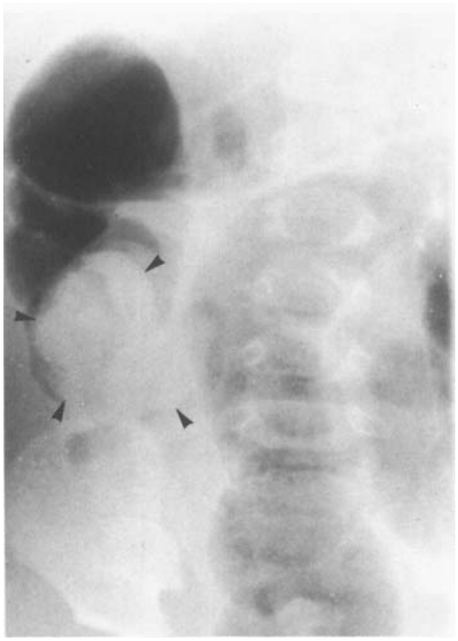

Figure 4 *Radiograph following an air contrast enema, outlining an ileocolic intussusception during reduction. The intussusceptum (arrowed) has been reduced to the caecum*

Operative reduction

Operative reduction is necessary for those in whom radiological techniques are contraindicated or have failed, when a pathological lead point is suspected, or in the case of multiple recurrences.

Various incisions have been employed but a right transverse muscle cutting incision, either just above or below the umbilicus, is associated with fewer complications than a vertical incision[170]. The intussusception is gently 'milked' back by compression of its apex. Sustained pressure to reduce the oedema of the intussusceptum may help and packs should be carefully placed to minimize peritoneal contamination from toxic fluid escaping from within the invagination. The reduced bowel should be allowed time to recover perfusion. Both the intussusceptum and the intussuscipiens must be carefully inspected for areas of doubtful viability. Seromuscular tears of the intussuscipiens with viable margins may be oversewn. Irreducible intussusceptions or those complicated by infarction or a pathological lead point require resection and primary end-to-end anastomosis[171]. Resection rates vary widely (*Table 6*) and partly reflect the experience of the surgeon and delay in presentation[17,112]. With modern antibiotics there are few indications for stoma formation.

If the caecum appears healthy, many surgeons perform an appendicectomy (either by excision or inversion), purportedly to encourage fixation of the caecum[124,172], to avoid future confusion as to whether or not appendicectomy was performed[170] and to prevent appendicitis. With increasing use of the appendix as a conduit in reconstructive surgery and the declining mortality rate from appendicitis, this procedure has to be questioned[173]. The recurrence rate after simple operative reduction is very low (*Table 6*) and most surgeons do not make any attempt to fix the caecum or terminal ileum, there being scant evidence to support such procedures. In the presence of associated intestinal malrotation, Ladd's procedure, which achieves a more stable mid-gut mesentery, should be considered[60]. In some cases a reperfusion injury[174] appears to complicate the operative treatment of ischaemic intussusception, and the protective role of free radical scavengers, such as mannitol, deserves investigation.

Table 6 *Recent results of operative treatment of intussusception*

Reference	Year	Place	No. of patients undergoing laparotomy	Resection rate* (%)	Postoperative recurrence rate (%)	Perioperative mortality rate (%)
Hutchison *et al.*[42]	1980	Glasgow	163	17	—	1·8
Pollet[104]	1980	Aberdeen	70	6·5	2·9	0
Man *et al.*[113]	1983	South-east England	69	9	2·9	0
Sparnon *et al.*[23]	1984	Australia	84	9	2·4	1·6
Liu *et al.*[67]	1986	Dublin	25	4·5	4·0	0
Mackay *et al.*[24]	1987	Australia	21	3·3	0	0
Winstanley *et al.*[114]	1987	Manchester	51	14	2	2
Zamir *et al.*[138]	1987	Israel	32	11·5	0	0
Wilson-Storey *et al.*[61]	1988	Edinburgh	94	8	1·1	0
Skipper *et al.*[141]	1990	USA	64	19	3·1	1·6

*Resection rate expressed as a percentage of patients presenting with intussusception

Even though intussusception following operation may affect multiple sites, surgical reduction is curative and pathological lead points and recurrences very rare[79].

Mortality, morbidity and recurrence

In the 19th century intussusception was usually fatal, but after the popularization of radiologically controlled contrast enema reduction the mortality rate declined to under 10 per cent and is now less than 1 per cent overall[20]. Avoidable factors still occur in 60 per cent of deaths[20]. Morbidity and mortality rates are higher when there is delay in presentation or treatment[42,175], typically in older children and in small bowel intussusceptions[2,20,76,176].

Adhesive intestinal obstruction features as a late complication in 3–6 per cent of patients undergoing operative reduction[17,61,67,104,113,120] but has not been reported after hydrostatic reduction.

Recurrent intussusception occurs in about 4 per cent of patients[22,32,177]. After radiological or operative reduction, the respective recurrence rates are about 10 per cent and 1–4 per cent[22,23,32,67,89,113,172]. Recurrence may be detected within days of hydrostatic reduction[32] but rarely so soon after operative reduction. Most recurrences develop within 1–36 months after reduction, with a peak incidence[32,177] during the eighth month. With all recurrent intussusceptions an underlying abnormality must be considered, but most first recurrences are not due to a pathological lead point and can be successfully managed by hydrostatic reduction[32,172].

In patients with multiple recurrences, structural anomalies in the ileocaecal region are common. Focal lymphoid hyperplasia of the ileum may be present[32,87,178] and, very frequently, an excessively mobile caecum attached to the posterior abdominal wall by a mesentery. Second recurrences should be managed by operative reduction but further invaginations are best treated by limited ileocaecal resection with end-to-end anastomosis[172]. Attempts have been made to stabilize the ileocaecal region by various plication procedures, such as suturing the terminal ileum to the ascending colon or using the base of the caecum as a temporary caecostomy[32,178–180], but the limited experience of the authors accords with that of Kyle *et al.*[177] who found that in some patients these procedures may even be harmful.

Improvements in the management of childhood intussusception are likely to be gained from wider awareness of its varied presentations, attention to detail in resuscitation, and prompt referral of the patient to a centre with combined paediatric radiological, anaesthetic, surgical and nursing expertise.

References

1. Hunter J. On introsusception. *Transactions of The Society for Improvement of Medical and Chirurgical Knowledge*. 1793; **1**: 103–18.
2. Ravitch MM. *Intussusception in Infants and Children*. Springfield, Illinois: Charles C Thomas, 1959.
3. Hutchinson J. A successful case of abdominal section for intussusception. *Proceedings of the Royal Medical and Chirurgical Society* 1873; **7**: 195–8.
4. Swain V. Sir Jonathan Hutchinson 1828–1913: his role in the history of intussusception. *J Pediatr Surg* 1980; **15**: 221–3.
5. Hirschsprung H. Et tilfaelde af subakut tarminvagination. *Hospitals-Tidende* 1876; **3**: 321–7.
6. Hipsley PL. Intussusception and its treatment by hydrostatic pressure: based on an analysis of one hundred consecutive cases so treated. *Med J Aust* 1926; **2**: 201–6.
7. Olsson G, Pallin G. Uber das Bild der akuten Darminvagination mit Hilfe von Kontrastlavements. *Acta Chir Scand* 1927; **61**: 371–83.
8. Pouliquen M, de la Marnierre. Indication du lavement bismuthe dans certaines formes d'invaginations intestinales. *Bulletin et Mémoirs Société Naturelle de Chirurgie* 1927; **53**: 1016–21.
9. Retan GM. Nonoperative treatment of intussusception. *Am J Dis Child* 1927; **33**: 765–70.
10. Stephens VR. Ileocaecal intussusception in infants with special reference to fluoroscopic findings. *Surg Gynecol Obstet* 1927; **45**: 698–700.
11. Ravitch MM, McCune RM Jr. Reduction of intussusception by barium enema: a clinical and experimental study. *Ann Surg* 1948; **128**: 904–17.
12. MacMahon B. Data on the etiology of acute intussusception in childhood. *Am J Hum Genet* 1955; **7**: 430–7.
13. Court D, Knox G. Incidence of intussusception in Newcastle children. *BMJ* 1959; **ii**: 408–9.
14. Smith IM. Incidence of intussusception and congenital hypertrophic pyloric stenosis in Edinburgh children. *BMJ* 1960; **i**: 551–2.
15. Steyn J, Kyle J. Epidemiology of acute intussusception. *BMJ* 1961; **i**: 1730–2.
16. Guo J, Ma X, Zhou Q. Results of air pressure enema reduction of intussusception: 6396 cases in 13 years. *J Pediatr Surg* 1986; **21**: 1201–3.
17. Bruce J, Huh YS, Cooney DR, Karp MP, Allen JE, Jewett TC. Intussusception: evolution of current management. *J Pediatr Gastroenterol Nutr* 1987; **6**: 663–74.
18. Cartensen H, Ryden CI, Nettelblad SC. Management of intussusception in childhood. *Lancet* 1985; **ii**: 953–4.
19. Pollet JE, Hems G. The decline in incidence of acute intussusception in childhood in north-east Scotland. *J Epidemiol Community Health* 1980; **34**: 42–4.
20. Stringer MD, Pledger HG, Drake DP. Childhood deaths from intussusception in England and Wales 1984–1989. *BMJ* 1992; **304**: 737–9.
21. Strang R. Intussusception in infancy and childhood. *Br J Surg* 1959; **46**: 484–95.
22. Ein SH, Stephens CA. Intussusception: 354 cases in 10 years. *J Pediatr Surg* 1971; **6**: 16–27.
23. Sparnon AL, Little KET, Morris LL. Intussusception in childhood: a review of 139 cases. *Aust N Z J Surg* 1984; **54**: 353–6.
24. Mackay AJ, MacKellar A, Sprague P. Intussusception in children: a review of 91 cases. *Aust N Z J Surg* 1987; **57**: 15–17.
25. Elebute EA, Adesola AO. Intussusception in western Nigeria. *Br J Surg* 1964; **51**: 440–4.
26. Momoh JT, Lawrie JH. Tropical paediatric intussusception – is it a different disease entity? *Ann Trop Paediatr* 1981; **1**: 237–40.
27. Mayell MJ. Intussusception in infancy and childhood in southern Africa. *Arch Dis Child* 1972; **47**: 20–5.
28. Rachelson MH, Jernigan JP, Jackson WF. Intussusception in the newborn infant. *J Pediatr* 1955; **47**: 87–94.
29. Yoo RP, Touloukian RJ. Intussusception in the newborn: a unique clinical entity. *J Pediatr Surg* 1974; **9**: 495–8.
30. Parkkulainen KV. Intrauterine intussusception as a cause of intestinal atresia. *Surgery* 1958; **44**: 1106–11.
31. Pavri DR, Marshall DG, Armstrong RF, Gorodzinsky FP. Intrauterine intussusception: case report and literature review. *Can J Surg* 1983; **26**: 376–8.
32. Beasley SW, Auldist AW, Stokes KB. Recurrent intussusception: barium or surgery? *Aust N Z J Surg* 1987; **57**: 11–14.
33. Janik JS, Cranford J, Ein SH. The well nourished infant with intussusception. *Am J Dis Child* 1981; **135**: 600–2.
34. Ravitch MM, McCune RM Jr. Reduction of intussusception by hydrostatic pressure: an experimental study. *Bull Johns Hopkins Hosp* 1948; **82**: 550–68.
35. Mitra SK, Rao PLNG, Bhattacharyya NC, Pathak IC. Rupture of intussuscipiens. *J Pediatr Surg* 1982; **17**: 300–1.
36. Gill WG. Multiple intussusceptions in a child. *Br J Surg* 1938; **25**: 707.
37. Nixon JR. Multiple 'agonal' intussusception of the small bowel. *J Irish Med Assoc* 1974; **67**: 134–5.
38. Mitra A, Roth RN. Retrograde ileo-ileal intussusception. *Postgrad Med J* 1984; **60**: 75.
39. Oswald MP, Graviss ER, Danis RK, Silberstein MJ, Cradock TV. Duodenogastric intussusception causing gastric outlet obstruction. *J Pediatr Surg* 1982; **17**: 82–3.
40. Galea MH, Mayell MJ. Gastroduodenal mucosal intussusception causing gastric outlet obstruction: a complication of gastrostomy tubes. *J Pediatr Surg* 1988; **23**: 980–1.
41. Treves F. The treatment of intussusception. *Proceedings of the Medical Society of London* 1885; **8**: 83–94.
42. Hutchison IF, Olayiwola B, Young DG. Intussusception in infancy and childhood. *Br J Surg* 1980; **67**: 209–12.
43. Eklof OA, Johanson L, Lohr G. Childhood intussusception: hydrostatic reducibility and incidence of leading points in

different age groups. *Pediatr Radiol* 1980; **10**: 83–6.
44. Hu SC, Feeney MS, McNicholas M, O'Halpin D, Fitzgerald RJ. Ultrasonography to diagnose and exclude intussusception in Henoch–Schönlein purpura. *Arch Dis Child* 1991; **66**: 1065–7.
45. Goldman L, Elman R. Spontaneous reduction of acute intussusception in children. *Am J Surg* 1940; **49**: 259–61.
46. Robb WAT, Souter W. Spontaneous sloughing and healing of intussusception: historical review and report of a case. *Br J Surg* 1962; **49**: 542–6.
47. Perrin WS, Lindsay EC. Intussusception, a monograph based on four hundred cases. *Br J Surg* 1921; **9**: 46–51.
48. Ravitch MM. Intussusception. In: Welch KJ, Randolph JG, Ravitch MM, O'Neill JA, Rowe MI, eds. *Pediatric Surgery*. 4th ed. Chicago: Year Book Medical, 1986: 868–82.
49. Bell TM, Steyn JH. Viruses in lymph nodes of children with mesenteric adenitis and intussusception. *BMJ* 1962; **ii**: 700–2.
50. Gardner PS, Knox EG, Court SDM, Green CA. Virus infection and intussusception in childhood. *BMJ* 1962; **ii**: 697–700.
51. Ross JG, Potter CW, Zachary RB. Adenovirus infection in association with intussusception in infancy. *Lancet* 1962; **ii**: 221–3.
52. Konno T, Suzuki H, Kutsuzawa T *et al.* Human rotavirus infection in infants and young children with intussusception. *J Med Virol* 1978; **2**: 265–9.
53. Nicolas JC, Ingrand D, Fortier B, Bricout F. A one-year virological survey of acute intussusception in childhood. *J Med Virol* 1982; **9**: 267–71.
54. Close HG. Acute intussusception in children: statistical analysis of 363 cases at Guy's Hospital. *Guy's Hospital Reports* 1931; **81**: 436–43.
55. Thomas GG, Zachary RB. Intussusception in twins. *Pediatrics* 1976; **58**: 754–6.
56. Thomas MP, McKay DG. Idiopathic intussusception occurring in father and son, with a post-operative intussusception in the son. *Aust Paediatr J* 1979; **15**: 281–2.
57. Stringer MD, Holmes SJK. Familial intussusception. *J Pediatr Surg* (in press).
58. Waugh GE. The morbid consequences of a mobile ascending colon, with a record of operations. *Br J Surg* 1919–1920; **7**: 343–83.
59. Filston HC, Kirks DR. Malrotation – the ubiquitous anomaly. *J Pediatr Surg* 1981; **16**: 614–20.
60. Brereton RJ, Taylor B, Hall CM. Intussusception and intestinal malrotation in infants: Waugh's syndrome. *Br J Surg* 1986; **73**: 55–7.
61. Wilson-Storey D, MacKinlay GA, Prescott S, Hendry GMA. Intussusception: a surgical condition. *J R Coll Surg Edinb* 1988; **33**: 270–3.
62. Stringer DA, Ein SH. Pneumatic reduction: advantages, risks and indications. *Pediatr Radiol* 1990; **20**: 475–7.
63. Waugh GE. Referred penile pain in intussusception with notes of three cases. *Lancet* 1911; **i**: 1492–4.
64. Van Meurs DP. Ileocolic intussusception associated with non-rotation of the midgut, congenital heart disease, and congenital hydrocele. *Br J Surg* 1946; **34**: 91–3.
65. Ornstein MH, Lund RJ. Simultaneous occurrence of malrotation volvulus and intussusception in an infant. *Br J Surg* 1981; **38**: 440–1.
66. Burke TE, Fitzgerald RJ. Intussusception, volvulus and malrotation. *Aust N Z J Surg* 1985; **55**: 73–4.
67. Liu KW, MacCarthy J, Guiney EJ, Fitzgerald RJ. Intussusception – current trends in management. *Arch Dis Child* 1986; **61**: 75–7.
68. Ward HC, Brereton RJ. Intussusception and duodenal stenosis: an infrequent but expected association. *Eur J Pediatr Surg* (in press).
69. Harkins HN. Intussusception due to invaginated Meckel's diverticulum. *Ann Surg* 1933; **98**: 1070–95.
70. Ein SH. Leading points in childhood intussusception. *J Pediatr Surg* 1976; **11**: 209–11.
71. Puri P, Guiney EJ. Small bowel tumours causing intussusception in childhood. *Br J Surg* 1985; **72**: 493–4.
72. Ong NT, Beasley SW. The leadpoint in intussusception. *J Pediatr Surg* 1990; **25**: 640–3.
73. Doberneck RC, Deane WM, Antoine JE. Ectopic gastric mucosa in the ileum: a cause of intussusception. *J Pediatr Surg* 1976; **11**: 99–100.
74. Lorenzi C, Marshall DG. Jejunal intussusception: a case report and review. *J Pediatr Surg* 1988; **23**: 374.
75. Forshall I. Intussusception of the vermiform appendix, with a report of seven cases in children. *Br J Surg* 1953; **40**: 305–12.
76. Mollitt DL, Ballantine TVN, Grosfeld JL. Postoperative intussusception in infancy and childhood: analysis of 119 cases. *Surgery* 1979; **86**: 402–8.
77. Stone DN, Kangarloo H, Graviss ER, Danis RK, Silberstein MJ. Jejunal intussusception in children. *Pediatr Radiol* 1980; **9**: 65–8.
78. Stringer MD, Pablot SM, Capps SNJ. Sonographic detection of the lead point in intussusception. *Arch Dis Child* 1992; **67**: 529–30.
79. West KW, Stephens B, Rescorla FJ, Vane DW, Grosfeld JL. Postoperative intussusception: experience with 36 cases in children. *Surgery* 1988; **104**: 781–5.
80. Martinez-Frontanilla LA, Haase GM, Ernster JA, Bailey WC. Surgical complications in Henoch–Schönlein purpura. *J Pediatr Surg* 1984; **19**: 434–7.
81. Fripp RR, Karabus CD. Intussusception in haemophilia. *S Afr Med J* 1977; **52**: 617–18.
82. Badertscher VA. Traumatic triple intussusception of the ileum in a child. *JAMA* 1939; **112**: 422–3.
83. Thompson JH, Posel MM. A case of intussusception in acute lymphatic leukaemia. *Lancet* 1930; i: 1180.
84. Dudgeon DL, Hays DM. Intussusception complicating the treatment of malignancy in childhood. *Arch Surg* 1972; **105**: 52–6.
85. Gupta HL, Malik TK. Intussusception in idiopathic thrombocytopenic purpura. *J Indian Med Assoc* 1976; **67**: 145–7.
86. Howell J, Pringle K, Kirschner B, Burrington JD. Peutz–Jeghers polyps causing colocolic intussusception in infancy. *J Pediatr Surg* 1981; **16**: 82–4.
87. Atwell JD, Burge D, Wright D. Nodular lymphoid hyperplasia of the intestinal tract in infancy and childhood. *J Pediatr Surg* 1985; **20**: 25–9.
88. Nkanza NK, King M, Hutt MSR. Intussusception due to inflammatory fibroid polyps of the ileum: a report of 12 cases from Africa. *Br J Surg* 1980; **67**: 271–4.
89. West KW, Stephens B, Vane DW, Grosfeld JL. Intussusception: current management in infants and children. *Surgery* 1987; **102**: 704–10.
90. Browne AF, Katz S, Miser J, Boles ET Jr. Blue rubber bleb nevi as a cause of intussusception. *J Pediatr Surg* 1983; **18**: 7–9.
91. Lam A, Ternberg JL. Cystic lymphangioma of the cecum with ileocecal intussusception: case report. *Am Surg* 1975; **41**: 648–9.
92. Fisher W, Clarke JH. Intussusception of the descending colon and volvulus of transverse colon. *Rocky Mountain Medical Journal* 1964; **61**: 44–6.
93. Gal R, Rath-Wolfson L, Ginzburg M, Kessler E. Adenomyomas of the small intestine. *Histopathology* 1991; **18**: 369–71.
94. Edna TH. Colo-rectal intussusception due to a leiomyoma. *Acta Chir Scand* 1978; **144**: 409–10.
95. Wayne ER, Campbell JB, Kosloske AM, Burrington JD. Intussusception in the older child – suspect lymphosarcoma. *J Pediatr Surg* 1976; **11**: 789–94.
96. Ein SH, Stephens CA, Shandling B, Filler RM. Intussusception due to lymphoma. *J Pediatr Surg* 1986; **21**: 786–8.
97. Tehrani MA. Recurrent intussusception due to a primary colloid carcinoma of the large bowel in a child. *Postgrad Med J* 1976; **52**: 242–5.
98. Buchta RM. Intussusception in a 14 year old boy secondary to an intramural fecolith. *Am J Dis Child* 1978; **132**: 1140–1.
99. Rees M. Intussusception caused by multiple trichobezoars: a surgical trap for the unwary. *Br J Surg* 1984; **71**: 721.
100. Patra SB, Giri DD, Shuklagn GN, Mehta HS. Amoebic granuloma – an unusual cause of caeco-colic intussusception. *Postgrad Med J* 1984; **60**: 168–70.
101. Persoff MM, Arterburn JG. Eosinophilic granuloma causing intussusception in a three year old child. *Am J Surg* 1972; **124**: 676–8.
102. Kariholu PL, Nehru SK, Misger MS, Rashid PA. Ascarid multiple enteric intussusception. *J Indian Med Assoc* 1981; **76**: 14–15.
103. Holsclaw DS, Rocmans C, Shwachman H. Intussusception in patients with cystic fibrosis. *Pediatrics* 1971; **48**: 51–8.
104. Pollet JE. Intussusception: a study of its surgical management. *Br J Surg* 1980; **67**: 213–15.
105. Turner D, Rickwood AMK, Brereton RJ. Intussusception in older children. *Arch Dis Child* 1980; **55**: 544–6.
106. Holmes M, Murphy V, Taylor M, Denham B. Intussusception in cystic fibrosis. *Arch Dis Child* 1991; **66**: 726–7.
107. Ein SH, Ferguson JM. Intussusception – the forgotten postoperative obstruction. *Arch Dis Child* 1982; **57**: 788–90.
108. Olcay I, Zorludemir U. Idiopathic postoperative intussusception. *Z Kinderchir* 1989; **44**: 86–7.

109. McGovern JB, Gross RE. Intussusception as a postoperative complication. *Surgery* 1968; **63**: 507–13.
110. Franken EA Jr, King H. Postoperative intussusception in children. *Am J Roentgenol Radium Ther Nucl Med* 1972; **116**: 584–6.
111. Watts JW, Fulton JF. Intussusception – relation of cerebral cortex to intestinal motility in monkey. *N Engl J Med* 1934; **210**: 883–6.
112. Raudkivi PJ, Smith HLM. Intussusception: analysis of 98 cases. *Br J Surg* 1981; **68**: 645–8.
113. Man DWK, Heath AL, Eckstein HB. Intussusception in infancy and childhood. A 13 year review of 75 patients. *Z Kinderchir* 1983; **38**: 383–6.
114. Winstanley JHR, Doig CM, Brydon H. Intussusception: the case for barium reduction. *J R Coll Surg Edinb* 1987; **32**: 285–7.
115. Beasley SW, Auldist AW, Stokes KB. The diagnostically difficult intussusception: its characteristics and consequences. *Pediatr Surg Int* 1988; **3**: 135–8.
116. Ein SH, Stephens CA, Minor A. The painless intussusception. *J Pediatr Surg* 1976; **11**: 563–4.
117. Braun P, Germann-Nicod I. Altered consciousness as a precocious manifestation of intussusception in infants. *Z Kinderchir* 1981; **33**: 307–9.
118. Hickey RW, Sodhi SK, Johnson WR. Two children with lethargy and intussusception. *Ann Emerg Med* 1990; **19**: 390–2.
119. Tenenbein M, Wiseman NE. Early coma in intussusception: endogenous opioid induced? *Pediatr Emerg Care* 1987; **3**: 22–3.
120. Gierup J, Jorulf H, Livaditis A. Management of intussusception in infants and children: a survey based on 288 consecutive cases. *Pediatrics* 1972; **50**: 535–45.
121. Doyle JC. Ileo-ileal intussusceptions in infants. *Br J Clin Pract* 1964; **18**: 617–19.
122. Reijnen JAM, Joosten HJM, Festen C. Intussusception in children 5–15 years of age. *Br J Surg* 1987; **74**: 692–3.
123. Rafinesque FG. *Etude sur les invaginations intestinales chronique*. Paris: JB Bailliere, 1879.
124. Rees BI, Lari J. Chronic intussusception in children. *Br J Surg* 1976; **63**: 33–5.
125. Reijnen JAM, Festen C, Joosten HJM. Chronic intussusception in children. *Br J Surg* 1989; **76**: 815–16.
126. Page AC, Price JF, Salisbury JF, Howard ER, Karani J. Chronic intussusception. *Arch Dis Child* 1989; **64**: 134–5.
127. Dinkel E, Dittrich M, Pistor G, Weitzel D, Greinacher I. Sonographic diagnosis of intussusception in childhood. *Z Kinderchir* 1983; **38**: 220–3.
128. Swischuk LE, Hayden CK, Boulden T. Intussusception: indications for ultrasonography and an explanation of the doughnut and pseudokidney signs. *Pediatr Radiol* 1985; **15**: 388–91.
129. Lee HC, Yeh HJ, Leu YJ. Intussusception: the sonographic diagnosis and its clinical value. *J Pediatr Gastroenterol Nutr* 1989; **8**: 343–7.
130. Pracros JP, Tran-Minh VA, Wright C. Ultrasound in diagnosis of intussusception. *Lancet* 1985; **ii**: 733–4.
131. Itagaki A, Uchida M, Ueki K, Kajii T. Double targets sign in ultrasonic diagnosis of intussuscepted Meckel's diverticulum. *Pediatr Radiol* 1991; **21**: 148–9.
132. Pandher D, Sauerbrei EE. Neonatal ileocolic intussusception with enterogenous cyst: ultrasonic diagnosis. *J Can Assoc Radiol* 1983; **34**: 328–30.
133. Kenney IJ. Ultrasound in intussusception: a false cystic lead point. *Pediatr Radiol* 1990; **20**: 348.
134. Beek FJA, Rovekamp MH, Bax NMA, Donckerwolcke RAM, Feldberg MAM, Kramer PPG. Ultrasound findings in postoperative jejunojejunal intussusception. *Pediatr Radiol* 1990; **20**: 601.
135. Martinez-Frontanilla LA, Silverman L, Meagher DP. Intussusception in Henoch–Schönlein purpura: diagnosis with ultrasound. *J Pediatr Surg* 1988; **23**: 375–6.
136. Maurer G, Kratochvil P, Brandstatter G. The value of colonoscopy and ultrasonography in the diagnosis of chronic intussusception in childhood. *Endoscopy* 1986; **18**: 29–30.
137. Curcio CM, Feinstein RS, Humphrey RL *et al.* Computed tomography of entero-enteric intussusception. *J Comput Assist Tomogr* 1982; **6**: 969–74.
138. Zamir O, Mogle P, Lernau OZ, Branski D, Nissan S. The efficiency and limitations of barium enema in the treatment of intussusception in infancy and childhood. *Pediatr Surg Int* 1987; **2**: 105–7.
139. Paes RA, Hyde I, Griffiths DM. The management of intussusception. *Br J Radiol* 1988; **61**: 187–9.
140. Phelan E, De Campo JF, Malecky G. Comparison of oxygen and barium reduction of intussusception. *Am J Radiol* 1988; **150**: 1349–52.
141. Skipper RP, Boeckman CR, Klein RL. Childhood intussusception. *Surg Gynecol Obstet* 1990; **171**: 151–3.
142. Palder SB, Ein SH, Stringer DA, Alton D. Intussusception: barium or air? *J Pediatr Surg* 1991; **26**: 271–5.
143. Jinzhe Z, Yenxia W, Linchi W. Rectal inflation reduction of intussusception in infants. *J Pediatr Surg* 1986; **21**: 30–2.
144. Tamanaha K, Wimbish K, Talwalkar YB, Ashimine K. Air reduction of intussusception in infants and children. *J Pediatr* 1987; **111**: 733–6.
145. Todani T, Sato Y, Watanabe Y, Toki A, Uemura S, Urushihara N. Air reduction for intussusception in infancy and childhood: ultrasonographic diagnosis and management without X-ray exposure. *Z Kinderchir* 1990; **45**: 222–6.
146. Glover JM, Beasley SW, Phelan E. Intussusception: effectiveness of gas enema. *Pediatr Surg Int* 1991; **6**: 195–7.
147. Shiels WE, Maves CK, Hedlund GL, Kirks DR. Air enema for diagnosis and reduction of intussusception: clinical experience and pressure correlates. *Radiology* 1991; **181**: 169–72.
148. Touloukian RJ, O'Connell JB, Markowitz RI, Rosenfield N, Seashore JH, Ablow RC. Analgesic premedication in the management of ileocolic intussusception. *Pediatrics* 1987; **79**: 432–4.
149. Parkes K, Chapman S. Pneumatic reduction of ileocolic intussusception in children. *Radiography* 1988; **54**: 147–50.
150. Collins DL, Pinckney LE, Miller KE *et al.* Hydrostatic reduction of ileocolic intussusception: a second attempt in the operating room with general anesthesia. *J Pediatr* 1989; **115**: 204–7.
151. Franken EA Jr, Smith WL, Chernish SM, Campbell JB, Fletcher BD, Goldman HS. The use of glucagon in hydrostatic reduction of intussusception: a double-blind study of 30 patients. *Radiology* 1983; **146**: 687–9.
152. Ein SH, Mercer S, Humphry A, MacDonald P. Colon perforation during attempted barium enema reduction of intussusception. *J Pediatr Surg* 1981; **16**: 313–15.
153. Douglas BS. Perforation of the bowel during barium enema reduction of intussusception in infants. *Aust Paediatr J* 1969; **5**: 116–21.
154. Barr LL, Stansberry SD, Swischuk LE. Significance of age, duration, obstruction and the dissection sign in intussusception. *Pediatr Radiol* 1990; **20**: 454–6.
155. Bettenay F, Beasley SW, De Campo JF, Auldist AW. Intussusception: can the clinician predict the outcome? *Aust N Z J Surg* 1988; **58**: 899–902.
156. Stephenson CA, Seibert JJ, Strain JD, Glasier CM, Leithiser RE, Iqbal V. Intussusception: clinical and radiographic factors influencing reducibility. *Pediatr Radiol* 1989; **20**: 57–60.
157. Beasley SW, De Campo JF. Radiological evidence of bowel obstruction in intussusception. *Pediatr Surg Int* 1987; **2**: 291–3.
158. Jennings C, Kelleher J. Intussusception: influence of age on reducibility. *Pediatr Radiol* 1984; **14**: 292–4.
159. Ong NT, Beasley SW. Progression of intussusception. *J Pediatr Surg* 1990; **25**: 644–6.
160. Patriquin HB, Afshani E, Effman E *et al.* Neonatal intussusception. Report of 12 cases. *Radiology* 1977; **125**: 463–6.
161. Smith VS, Giacoia GP. Intussusception associated with necrotizing enterocolitis. *Clin Pediatr (Phila)* 1984; **23**: 43–5.
162. Wang GD, Liu SJ. Enema reduction of intussusception by hydrostatic pressure under ultrasound guidance: a report of 377 cases. *J Pediatr Surg* 1988; **23**: 814–18.
163. Ein SH, Shandling B, Reilly BJ, Stringer DA. Hydrostatic reduction of intussusceptions caused by lead points. *J Pediatr Surg* 1986; **21**: 883–6.
164. Mitchell S. Intussusception in children. *Lancet* 1837–1838; **i**: 904.
165. Fiorito ES, Recalde Cuestas LA. Diagnosis and treatment of acute intestinal intussusception with controlled insufflation of air. *Pediatrics* 1959; **24**: 241–4.
166. Hedlund GL, Johnson JF, Strife JL. Ileocolic intussusception: extensive reflux of air preceding pneumatic reduction. *Radiology* 1990; **174**: 187–9.
167. Pokorny WJ, Wagner ML, Harberg FJ. Lateral wall cecal filling defects following successful hydrostatic reduction of cecal colic intussusceptions. *J Pediatr Surg* 1980; **15**: 156–9.
168. Fitch SJ, Magill HL, Benator RM, Parvey LS, Hixson SD. Pseudoreduction of intussusception: is ileal reflux the end point? *Gastrointest Radiol* 1985; **10**: 181–3.
169. Bolia A. Case report: diagnosis and hydrostatic reduction of an

intussusception under ultrasound guidance. *Clin Radiol* 1985; **36**: 655–7.
170. Thomas DFM. The management of childhood intussusception in a district hospital. *Br J Surg* 1980; **67**: 33–5.
171. Dennis C. Resection and primary anastomosis in the treatment of gangrenous or nonreducible intussusceptions in children. *Ann Surg* 1947; **126**: 788–96.
172. Ein SH. Recurrent intussusception in children. *J Pediatr Surg* 1975; **10**: 751–4.
173. Wheeler RA, Malone PS. Use of the appendix in reconstructive surgery: a case against incidental appendicectomy. *Br J Surg* 1991; **78**: 1283–5.
174. Welbourn CRB, Goldman G, Paterson IS, Valeri CR, Shepro D, Hechtman HB. Pathophysiology of ischaemia reperfusion injury: central role of the neutrophil. *Br J Surg* 1991; **78**: 651–5.
175. Dennison WM, Shaker M. Intussusception in infancy and childhood. *Br J Surg* 1970; **57**: 679–84.
176. Cooke DC, Lewis EC. A 30-year survey of acute intussusception in childhood. *Lancet* 1960; **ii**: 1359–61.
177. Kyle J, Steyn JH, Bell TM, Mercer S. Acute recurrent intussusception. *Surg Gynecol Obstet* 1963; **117**: 323–6.
178. Sarason EL, Prior JT, Prowda RL. Recurrent intussusception associated with hypertrophy of Peyer's patches. *N Engl J Med* 1955; **253**: 905–8.
179. Mastin EV. Multiple attacks of acute intussusception. *Am J Surg* 1942; **58**: 300–1.
180. Burrington JD. Surgical technique for the prevention of recurrent intussusception in childhood. *Surg Gynecol Obstet* 1980; **150**: 572–3.

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