The constantly irritable infant is often referred to by primary health care providers in a somewhat offhanded way as being colicky, as if that somehow represents the final word on the subject. 'Infantile colic' is a term which is used by various health care professions to describe the persistent, often violent crying which sometimes characterizes an otherwise healthy and thriving baby. As many as 1 in 5 parents report a problem with infant irritability or crying in the first 3 months of life but less than 1 in 20 with problem crying have an organic cause (Hiscock & Jordan 2004).

Although there have been a number of theories proposed as to the etiology of colic, the literature remains fraught with difficulties in definition, methodological problems and numerous claims as to both etiology and management that are anecdotal (Hewson et al 1987). Various medical authors believe colicky behavior to be a reaction pain, probably of intestinal origin (Behrman et al 2007, Hull & Johnston 1987, Illingworth 1985). That this characteristic irritable behavior attributed to colic which is uncomplicated by other disease is a result of pain is also supported by researchers within both chiropractic and medicine (Biedermann 1992, Gutmann 1987, Kloughart et al 1989, Nilsson 1985). While the exact mechanisms for this pain remain to some extent obscure, the work of Sato (1980) and Budgell (1998) has shed considerable light on the subject, suggesting a neurophysiological mechanism that supports the clinical observations of Kloughart et al (1989) in their multicentered research trial which demonstrated a striking success rate for resolution of colicky symptoms in young infants after chiropractic management.

Infantile colic probably remains best conceptualized as the end result of a complex transaction between the infant and their environment with multiple factors potentially responsible for the crying and distress (Hewson et al 1987). However, for a subgroup of infants, early excessive crying may evolve into a more generalized ‘persistent mother-infant distress’ syndrome (Leung & Lemay 2004). Taken together, these factors open the way for wide-ranging management options for the chiropractor.

The purpose of using the term ‘uncomplicated colic’ is to clearly signify in the clinical record that there are no factors such as protein intolerance, carbohydrate intolerance, or obvious parenting errors that are complicating the colicky presentation. These complications are discussed later in this chapter.

The symptoms that are characteristic of the typical colicky infant begin abruptly: the cry is loud and more or less continuous, persisting for several hours at the same time each day, usually late in the afternoon or early in the evening; the face sometimes becomes flushed or circumoral pallor develops; the abdomen may be distended and tense with the legs drawn up; the feet are often found to be cold and the hands bunched into tightly held fists. The paroxysm may simply spontaneously terminate or relief is sometimes obvious after the passage of flatus or fecus (Barr 1998, Behrman et al 2007).

Reaching a diagnostic conclusion of uncomplicated colic is a process of elimination. The child with colicky symptoms must be carefully evaluated for any clinical signs that may suggest underlying disease. The parents/caregivers must also be carefully questioned in order to establish that there are no parenting errors, environmental factors, feeding difficulties or family history that may be contributing to the child’s irritable disposition. Only after all these factors have been taken into consideration and ruled out, can the chiropractor safely assume that the child indeed has a diagnosis of uncomplicated colic.

Table 5.1 lists some of the more common clinical conditions which may complicate the colicky presentation and their symptomatic characteristics.
Once the diagnosis has been established to a clinical certainty, there are two major aspects involved in chiropractic management. The first is to assess and precisely correct the patient's anatomical location is less predictable than we first thought and driven more by patterns of cortical dysaferentation and the associated dural tension and cerebrospinal fluid flow dynamics rather than nerve root factors.

The second aspect of care is to counsel the parents and caregivers carefully in relation to feeding practices, positive reinforcement, and the use of contingent music therapy. The correction of neurological dysaferentation, while having been shown to be effective in resolving the distressing symptoms associated with uncomplicated colicky presentations (Meadow & Smithells 1975), is not the totality of patient management as the important factors referred to above cause frustration for both the infant and mother, creating a positive feedback cycle which in turn perpetuates irritability and recurrence of the subluxation complex, presumably via the viscerosomatic, somatosomatic, and psychosomatic reflex pathways. Provision of adequate, practical counseling to the nursing mother is an essential element in the chiropractic management program (Taubman 1990).

### Use of contingent music and differential reinforcement

It has been shown by clinical research that there is a significant behavioral aspect to children with uncomplicated colicky presentations that can be considerably varied by the use of contingent music and differential reinforcement (Larson & Allyn 1990). Parents with colicky children should be carefully counseled to turn on music when the infant is quiet and alert for a period

### Table 5.1 Clinical indicators that may imply underlying disease in irritable infant presentations

<table>
<thead>
<tr>
<th>Body system</th>
<th>Symptom/sign</th>
<th>Associated condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Integument</td>
<td>Maculopapular rash on trunk, neck, face and upper arms</td>
<td>Cow’s milk or soy protein intolerance</td>
</tr>
<tr>
<td></td>
<td>Erythematous skin on the buttocks and anogenital region</td>
<td>Congenital low pH of urine and other body fluids</td>
</tr>
<tr>
<td></td>
<td>Pustules over face and trunk with general lymphadenopathy</td>
<td>Congenital infection</td>
</tr>
<tr>
<td></td>
<td>Erythematous rash which desquamates and forms pustules in an otherwise healthy term infant</td>
<td>Congenital candidiasis</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Bloating, frequent passage of flatus, constipation and/or diarrhea</td>
<td>Cow’s milk or soy protein intolerance</td>
</tr>
<tr>
<td></td>
<td>Sudden onset of paroxysms of severe colicky pain in a previously unaffected infant which get steadily worse over a few hours</td>
<td>Intussusception or other bowel obstruction</td>
</tr>
<tr>
<td></td>
<td>Profuse, explosive diarrhea which has a sweet smell and tests positive for disaccharides</td>
<td>Lactose intolerance</td>
</tr>
<tr>
<td></td>
<td>Projectile vomiting immediately following feeding preceded by ‘golf ball’ reverse peristaltic waves seen under tangential lighting of the upper abdomen</td>
<td>Pyloric stenosis</td>
</tr>
<tr>
<td></td>
<td>Constant small volume vomiting which has a sour smell</td>
<td>Gastroesophageal reflux</td>
</tr>
<tr>
<td></td>
<td>Unchanged nappies (diapers)</td>
<td>Parenting error</td>
</tr>
<tr>
<td></td>
<td>Swallowed air</td>
<td>Nursing technique</td>
</tr>
<tr>
<td></td>
<td>Overfeeding, underfeeding, or inappropriately clothed for the prevailing temperature</td>
<td>Parenting error</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Crackles/wet sounds without obvious dyspnea</td>
<td>Cow’s milk or soy protein intolerance</td>
</tr>
<tr>
<td></td>
<td>Crackles with fever and dyspnea</td>
<td>Infection</td>
</tr>
<tr>
<td>Genitourinary</td>
<td>Fever with decreased fluids out</td>
<td>Renal infection</td>
</tr>
<tr>
<td>Head and neck</td>
<td>Hair in the child’s eye</td>
<td>Corneal irritation</td>
</tr>
<tr>
<td></td>
<td>Fever</td>
<td>Otitis</td>
</tr>
<tr>
<td></td>
<td>Nasal congestion, postnasal drip and reddened tonsils</td>
<td>Chronic catarrh</td>
</tr>
<tr>
<td></td>
<td>Head held constantly to one side</td>
<td>Congenital sternomastoid tumor</td>
</tr>
</tbody>
</table>
While intolerance to cow’s milk is typically seen in babies, the physiology of the human gut in the first year of life varies significantly from that of the older child in that the permeability is much greater. By the end of the first year of life the permeability becomes rapidly more adult-like. Because of this increased permeability, during the first year of life, proteinaceous substances can make their way across the gut wall into the bloodstream where they can produce an IgE-mediated antigen-antibody response (Plebani et al 1990, Wilson & Hamburger 1988). There is no single substance in cow’s milk that produces the allergic reaction. Casein, alpha-lactalbumin and beta-lactalbumin all show a high proportion of positive reactions (Savilahti & Kuitunen 1992) with specific IgE antibodies to these substances having been recovered from infants allergic to cow’s milk (Bjorksten et al 1983). It has been further demonstrated that children with cow’s milk intolerance have an even greater permeability of the small intestine than is the case with non-atopic children of the same age (Schrander 1992). Some cases of cow’s milk intolerance, however, cannot be shown to be IgE mediated and therefore it seems apparent that the child’s symptoms arise from a non-immune source (Foucard 1985).

While intolerance to cow’s milk is typically seen in babies where there is a positive family history of atopic disease (Foucard 1985, Wilson & Hamburger 1988), it is not necessarily always the case. Certainly, a negative family history does not automatically eliminate a diagnosis of cow’s milk intolerance.

### Clinical presentation


### Diagnosis

In order to make a presumptive clinical diagnosis of cow’s milk allergy, a ‘triad’ of symptoms, which must include the gastrointestinal, respiratory, and integumentary systems, must be present. The majority of children who end up with a confirmed diagnosis of cow’s milk allergy will present initially with this typical ‘triad’ of symptoms, namely gastrointestinal disturbance, skin rash and respiratory ‘wet sounds’. While a small number present atypically, it is probably safe to say that all children with cow’s milk allergy will at least have gastrointestinal symptoms and one or more of the other symptoms shown in Table 5.2.

Once the possibility of cow’s milk allergy is considered, it is wise to arrange appropriate allergy tests and an evaluation of total serum IgE. When found in combination, a positive radioallergosorbent test (RAST) to cow’s milk and an elevated total serum IgE is as reliable as laboratory investigations get in assisting the clinician to confirm a diagnosis of cow’s milk allergy. Certainly, a combination of typical symptomatic pattern and positive laboratory results will confirm the diagnosis in a good percentage of cases.

### Pathophysiology

The physiology of the human gut in the first year of life varies significantly from that of the older child in that the permeability is much greater. By the end of the first year of life the permeability becomes rapidly more adult-like. Because of this increased permeability, during the first year of life, proteinaceous substances can make their way across the gut wall into the bloodstream where they can produce an IgE-mediated antigen-antibody response (Plebani et al 1990, Wilson & Hamburger 1988). There is no single substance in cow’s milk that produces the allergic reaction. Casein, alpha-lactalbumin and beta-lactalbumin all show a high proportion of positive reactions (Savilahti & Kuitunen 1992) with specific IgE antibodies to these substances having been recovered from infants allergic to cow’s milk (Bjorksten et al 1983). It has been further demonstrated that children with cow’s milk intolerance have an even greater permeability of the small intestine than is the case with non-atopic children of the same age (Schrander et al 1992). Some cases of cow’s milk intolerance, however, cannot be shown to be IgE mediated and therefore it seems apparent that the child’s symptoms arise from a non-immune source (Foucard 1985).

While intolerance to cow’s milk is typically seen in babies where there is a positive family history of atopic disease (Foucard 1985, Wilson & Hamburger 1988), it is not necessarily always the case. Certainly, a negative family history does not automatically eliminate a diagnosis of cow’s milk intolerance.

### Table 5.2 Common symptoms associated with cow’s milk allergy and intolerance in infants

<table>
<thead>
<tr>
<th>Body system</th>
<th>Symptom/sign</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal</td>
<td>Bloating, frequent passage of flatus and intractable crying/distress with pulling up of the legs Chronic diarrhea, constipation or an alternating pattern of both</td>
</tr>
<tr>
<td>Integument</td>
<td>Maculopapular rash which may occur anywhere on the body, but is found most commonly on the face, neck, trunk, buttocks and upper arms. Sometimes it is only seen in body fossae and within the enclosed areas of skin folds Eczema</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Crackles/wet sounds without obvious dyspnea Wheezing and rhinitis</td>
</tr>
<tr>
<td>Neurological</td>
<td>Disturbed sleep pattern with frequent waking, crying at night, short sleep cycles and long non-rapid eye movement stage 1 (NREM1) sleep</td>
</tr>
</tbody>
</table>
It should be borne in mind when performing laboratory tests, however, that a second type of reaction to cow’s milk which is really a non-immune intolerance will return negative laboratory results while the symptomatic picture remains strongly evidential of the diagnosis. Under these circumstances, a clinical diagnosis should be made and the patient managed accordingly. A successful clinical outcome is all the diagnostic confirmation that is needed.

**Management**

Chiropractic management of the infant who reacts to cow’s milk protein is the same for IgE-mediated and non-immune intolerances. In both cases, diagnostic confirmation is dependent upon clinical outcome. The management of the breastfed infant is simple. The mother is counseled to avoid all food substances containing dairy products and to ensure a fluid intake of approximately 1.5–2 liters per day for the trial period of 15 days. The baby should be evaluated for evidence of neurological dysafferentation twice a week and correction made when necessary. Recurrent subluxation patterns are common among babies who react to cow’s milk protein, presumably owing to activation of the viscerosomatic reflex by the offending protein. If the neurological dysafferentation is left uncorrected, the child’s symptoms may persist, confirming the clinical outcome of the dairy-free trial period. If the child is clinically improved after the 15-day trial period and the correction of the neurological dysafferentation is stable, the mother should be counseled in relation to adopting a dairy-free diet and to ensure adequate supplementation for the duration of her breastfeeding, since human milk offers the best protection for the cow’s milk reactive infant (Arato et al 1996, Gruskay 1982).

Artificially fed infants are somewhat more difficult to manage. Most artificially fed infants who have cow’s milk intolerance or allergy will require feeding with a fully hydrolyzed formula, making a 14–21 day trial on such a formula the ideal approach to diagnostic confirmation. However, in some jurisdictions, Government health authorities require an initial trial on a soy-based formula prior to the trial on a fully hydrolyzed formula in order to permit the writing of a formal prescription for the hydrolyzed formula. A significant percentage of children who are allergic or intolerant to cow’s milk protein can tolerate soy protein without developing atopic symptoms in the short term (Nadasdi 1992). There are, however, a percentage of children who have a cross-reactivity to soy in whom symptomatic relief will be apparent within the 14–21 day trial, only to relapse again within a few days. Relief comes as the offending cow’s milk proteins are eliminated from the diet and exacerbation occurs as a new antigen–antibody response specific to the soy protein begins. It needs to be pointed out that prolonged use of soy infant formula is not a perfect solution; the known problems with such an approach are more fully discussed in Chapter 19.

If soy cross reactivity is identified, the child should then be trialed on a hydrolyzed formula such as Nutramigen or Pre-gestimil. Hydrolyzed formulae consist of dried glucose syrup, casein hydrolysate, a variety of vegetable oils, and a range of vitamins, minerals and amino acids. Cross-reactivity of hydrolyzed formulae with cow’s milk protein has been demonstrated (Ragano et al 1993) but the incidence is very low (Host & Samuelsson 1988, Rugo et al 1992). The major problem encountered with hydrolyzed formulae is rejection by the infant. Cross-reactive infants generally have a high incidence of chronic subluxation recurrence and therefore need to be assessed on a very regular basis during the period of dietary manipulation. As an aside, casein hydrolysate formulae are much to be preferred for their hypoallergenic properties than formulae with whey hydrolysate, which demonstrates a greater cross-reactivity with cow’s milk (Host & Samuelsson 1988, Rugo et al 1992).

Children who have been shown to be cow’s milk allergic or intolerant should ideally be kept permanently on a dairy-free diet. While this strategy has not been demonstrated to completely prevent later development of atopic disease (Bishop et al 1990), there is persuasive evidence suggesting that dietary avoidance of cow’s milk protein lessens the incidence of such problems (Schrander et al 1992, Strobel 1992).

**Carbohydrate intolerance**

Gastrointestinal intolerance to sugars is thought to affect most of the adults in the world (Bayless et al 1975) to a greater or lesser extent and is also commonly encountered in infants and older children.

**Pathophysiology**

The principal carbohydrate in human milk is the disaccharide lactose, which is hydrolyzed in the small intestine by lactase phlorizin hydrolase, otherwise known as lactase (Buller et al 1991). The absence of lactase permits the passage of undigested lactose into the large intestine and is associated with the well-known syndrome referred to as lactose intolerance.

**Clinical presentation**

The most common presentation of lactose intolerance in infancy almost invariably follows a bout of acute gastroenteritis which the gastrointestinal mucosa has become damaged. This condition is self-limiting and will improve over time, but it is worth treating in order to gain symptomatic relief.

In older children the symptoms of lactose intolerance usually develop gradually beginning several years after birth and presenting with abdominal cramps, bloating, chronic diarrhea, and excessive passage of flatus related to the ingestion of dairy products (Mitchell et al 1975). While lactose is by far the most common carbohydrate to cause the above symptom pattern, some children will experience similar symptoms of abdominal distress and severe diarrhea due to sucrose isomaltase deficiency. This condition is very uncommon and need only be considered in the event that management implemented for lactose intolerance fails.
Diagnosis

The diagnosis of lactose intolerance is a clinical one dependent on the recognition of the symptom complex described above and does not normally require any laboratory investigations. However, simple tests which can be carried out on the liquid portion of the diarrheal stool may be performed if there is doubt about the diagnosis. Immediately after collection, the liquid stool specimen is mixed with two parts water, and 15 ml of the resultant fluid is tested with two Clinistix tablets for the presence of reducing sugars, while another drop is applied to the glucose tester on a Clinistix strip. Glucose at 0.5% or less should be considered normal (Behrman et al 2007). In addition, it is worthwhile to test the pH, which will be 5.5 or less in lactose-intolerant individuals.

Management

In breastfed infants, neurological dysaferentation should be corrected and the mother strongly encouraged to continue nursing. Weaning to a lactose-free formula should only be considered in the most severe, protracted cases where there is demonstrable weight loss. In artificially fed infants, the formula should be changed to one that is lactose free.

Older children in whom lactose intolerance has made a gradual appearance should be placed on a dairy-free diet, provided with adequate nutritional supplementation, and have their patterns of neurological dysaferentation corrected and monitored at regular intervals. Symptomatic resolution will come quickly and permanently provided there are no ‘binges’ on dairy products in which as little as 8 ounces of milk (half a glass) may produce a flat blood sugar curve, bloating, cramps, loose stools, or diarrhea (Mitchell et al 1975).

Gastroesophageal reflux

Gastroesophageal reflux (GER) is a common occurrence during infancy with most children having a small degree of reflux that is of little clinical consequence, usually requiring only minimal intervention in the event it causes harm (Catto-Smith 1998, Glassman et al 1995). This phenomenon is due to the fact that the intra-abdominal segment of the esophagus is virtually nonexistent at birth (Hull & Johnston 1987) and therefore no effective reflux barrier exists. This barrier is created over the first few months of life as the intra-abdominal esophageal segment lengths. It is important to recognize that the natural history of GER in infants differs significantly from reflux in adults (Catto-Smith 1998). In children, reflux-induced injury can result from acid exposure, nutrient loss, or respiratory complications.

GER has become a rather ‘fashionable’ diagnosis in recent years in infants that are irritable, with the emergence of the totally unfounded notion of ‘silent reflux’ in which it is hypothesized that acid reflux into the distal esophagus occurs without the visible evidence of frequent and excessive small volume vomitus. This notion lacks any scientific evidence at all.

Clinical presentation

In true GER the gastrointestinal signs and symptoms are related directly to the exposure of the distal esophageal epithelium to refluxed gastric contents (Behrman et al 2007). The majority of children with GER have delayed gastric emptying, and vomiting may be forceful because of pylorospasm (Behrman et al 2007). In the vast majority of children, excessive vomiting occurs within the first week of life (Behrman et al 2007, Smith 1980). Vomiting due to GER can occur at any time, unlike normal possetting in which infants will bring up a little milk as they expel swallowed air after feeding.

Respiratory systems are common in children with GER, usually owing to aspiration of refluxed material. Symptoms include chronic cough, wheezing, and recurrent pneumonia. To be safe, GER should always be considered as a likely etiology of recurrent respiratory symptoms in the first 2 years of life and appropriate diagnostic evaluation carried out.

In children with GER, growth and weight gain are usually adversely affected. This has been demonstrated in about two-thirds of all cases with confirmed GER (Behrman et al 2007).

Diagnosis

In mild cases, diagnosis is made on history and clinical assessment alone. Episodic vomiting beginning in the first 1–2 weeks of life should underpin a diagnosis of mild GER in infancy. Careful monitoring of the child’s response to management will quickly confirm the diagnosis. In more severe cases, it is wise to refer the child to a pediatric specialist for esophagoscopy or barium esophagography under fluoroscopic control. Structures, recurrent reflux and ragged mucosal outline suggestive of esophagitis are readily seen with barium esophagography; however, esophagoscopy with biopsy is the preferred technique for demonstrating esophagitis.

Management

In mild cases the infant should be neurologically managed and monitored for symptomatic change over a period of 2 weeks. In the event that the cause of the reflux symptoms is functional the child will show rapid improvement related directly to the sustainability of the correction of patterns of neurological dysaferentation. In cases in which improvement is not sustained and the dysaferentation patterns are recurrent, concurrent treatment with an antacid that does not contain aluminum hydroxide is recommended along with the formula thickening in bottle fed infants.

In more intractable cases, it is appropriate to refer the affected child to the family general practitioner for the prescription of one of the upper gastrointestinal motility enhancing agents which are generally available around the world today.

In cases involving retarded growth factors, respiratory symptoms or excessive intractable vomiting which fails to respond to conservative care, referral to a pediatric specialist should be made in order to arrange for appropriate clinical investigation and possible surgical intervention (Smith 1980).
Infantile hypertrophic pyloric stenosis

Infantile hypertrophic pyloric stenosis (IHPS) is the most common condition affecting infants that requires surgical intervention (St Peter & Ostlie 2008). The etiology of IHPS is yet to be fully elucidated. Since the 1990s, a sharp decline in IHPS has been reported in various countries. A possible correlation has been suggested between the ‘Back to Sleep’ campaign and the falling incidence of IHPS as its decline paralleled that of sudden infant death syndrome (SIDS). Recent research from Scotland, however, has shown that the beginning of the decline in the incidence of IHPS predated by some 2 years that of SIDS (which did follow the ‘Back to Sleep’ campaign), making a direct correlation unlikely (Sommerfield et al. 2008).

Infants with pyloric stenosis generally present with vomiting beginning as early as the end of the first week of life and as late as the fifth month (Behrman et al. 2007) with the usual time of onset being around the third week. The vomiting may not, at first, be projectile but will progress to that in time. The development of pyloric stenosis is a progressive and dynamic process. The rate of hypertrophy to the point of meeting diagnostic criteria is unknown and there are no data published in the literature regarding the role of repeat ultrasound in patients with persistent symptoms (Keckler et al. 2008). The usual pattern of vomiting is a large volume, forceful vomit following the appearance of an obvious peristaltic wave moving across the upper abdomen. The peristaltic wave is often referred to as ‘golf ball peristalsis’ as it has the appearance of a moving golf ball located within the abdomen. This wave phenomenon is best visualized by looking tangentially across the abdomen. An olive-shaped mass is also usually palpable midway between the umbilicus and the costal margin, just inferior to the liver border. This mass will normally be more readily palpable immediately following an episode of vomiting.

IHPS has been linked to inappropriate neuromuscular action, poor breastfeeding performance, and maternal stress (Behrman et al. 2007). The striking success that some children receive from chiropractic bears testimony to an etiology of subluxation complex-induced neuromuscular incoordination. The gastrointestinal tract receives sympathetic supply from T8–L1 and, with the exception of the distal colon, parasym pathetic supply via the Xth cranial nerve. Neurological dysafferentation involving the upper cervical complex may affect the function of the Xth cranial nerve, possibly owing to the anatomical proximity of the inferior vagal ganglion (Lawrence 1991), the effect of increased dural tension and altered cerebrospinal fluid pressure gradients. The combination of these factors may potentially affect the function of the gastrointestinal tract. In addition, it has been reported by Wiles (1990) that palpatory evidence of reduced motion in the vertebral segments between T7 and T12 has been found in association with generalized gastrointestinal disease. The diagnosis is made clinically and there is usually no need for the diagnostic imaging procedures to be performed.

Care must be taken when making management decisions in relation to children with pyloric stenosis. As the vomiting continues, a progressive loss of fluid, hydrogen, and chloride ions occurs which will, if not corrected, lead to hypochloremic metabolic alkalosis. Serum potassium levels usually remain unaffected, but in severe cases a total body potassium deficit may occur. Where no evidence of dehydration or physiological stress from biochemical deficiency can be demonstrated, chiropractic management can be safely instigated. The care program should include careful neurological assessment with correction of any dysaafferentation and maternal counseling in relation to feeding procedures and stress reduction. If the likelihood is that the mother is probably not going to be able to significantly change her circumstances to reduce stress levels, a referral after subluxation correction to a hospital for both mother and child is appropriate. In the hospital environment, the mother will receive the help she needs to cope with the demands of her baby and may be able to get some much-needed rest if sleep deprivation has been an issue. Stabilization of the neurological dysaafferentation may ultimately depend on correcting breastfeeding errors and reducing immediate stress levels (Homewood 1981).

Intussusception

Intussusception is defined as ‘invagination of bowel into an adjacent lower segment’ (Hull & Johnston 1987). While not a particularly common condition in terms of incidence, it does represent the most frequent cause of bowel obstruction in the first 2 years of life. The typical symptom pattern is characterized by episodic, uncontrollable crying, inactivity, irritability and an unwillingness to be handled, sudden onset of pallor which becomes persistent, vomiting, and occasionally rectal bleeding which may be identified in digital examination of the rectum.

When a child presents with this confluence of symptoms along with a tender palpable mass in the abdomen which is usually located in the right upper quadrant or close to the left side of the umbilicus, pediatric referral should be made without performing any investigations. Intussusception is a clinical diagnosis. Children presenting to chiropractors with symptoms and signs consistent with this condition should be referred for
urgent surgical care as bowel viability can be quickly lost (Hull & Johnston 1987).

Intussusception in a baby that has not been seen previously may be more readily recognizable than is the case with an irritable baby that is being given ongoing chiropractic care, particularly if the symptomatic response is favorable. The latter may be present in such a way that both mother and chiropractor are led into believing that the baby is simply undergoing another paroxysm of irritability caused by a recurrence of the neurological dysafferentation. The critical decision-making problem in this case is linked to the fact that the viscerosomatic reflex responsible for neurological dysafferentation with 'colic' may well be activated in an identical manner by the intussusception. To avoid making an incorrect clinical decision, the abdomen of the irritable baby should be carefully examined at each review consultation.

References


The irritable baby


