Review

Infantile colic: A critical appraisal of the literature from an osteopathic perspective

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Abstract

The term infantile colic though often used is poorly understood. This common affliction of early infancy, in which crying is the primary symptom, has been the subject of numerous publications over many decades. This appraisal of the literature reveals that lack of consensus on the definition of colic poses difficulties when assessing the outcome and validity of research. In addition, the numerous hypotheses, reviewed here, on the precise cause of infantile colic have resulted in various approaches to treatment and no clear strategy for management. The possibility that mechanical factors play a role in the aetiology of colic underlies the osteopathic approach to treatment which, despite anecdotal success, is poorly documented. This article is designed to appraise osteopaths with an overview of the field to better inform their practice.
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1. Introduction

Infantile colic is often described as a behavioural and non-pathological condition in early infancy with crying being the primary symptom. It is a poorly defined but common affliction of early infancy, the cause of which is still currently obscure and controversial and there is therefore a lack of an appropriate strategy for the management and treatment of this condition. This lack of a consensus on the aetiology of colic, in spite of over six decades of research, has resulted in various treatment approaches of dubious and varying merit, a trial and error approach that may compound the distress that is inevitably also suffered by the parents.

This article is based on a review of the literature identified through searching online (Medline) and at academic libraries accessible to the author. It is written in the hope that an appraisal of this field will better inform osteopathic practice. A historical overview of the subject is beyond the scope of this paper.

1.1. Definition of infantile colic

There is much debate as to what infantile colic actually is. The term colic is derived from the Greek word *kolikos*, an adjective of *kolon* meaning the large intestine. The word colic suggests that the condition is a manifestation of some type of abdominal or visceral pain, possibly intestinal cramps. This is contentious as the implication that the crying of a colicky infant is a result

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of the infant experiencing pain that is abdominal in origin is not necessarily true in all infants who cry.[1]

The most universally accepted definition of colic and one which is most used in clinical research is that proposed by Wessel et al.,[2] in a study involving 180 mothers and their infants. The inclusion criterion for this study was "paroxysms of irritability, fussing or crying lasting for a total of more than 3 h a day and occurring on more than three days in any one week" in an infant who was otherwise "healthy and well fed". The study stated that if an infant "had no such paroxysms or if the paroxysms were less than the above in total duration he was classified as contented". There was therefore an arbitrary fixing of a threshold for colic in this study, which did not include infants who were mildly fussy. There is clearly a spectrum of severity of crying and the distinction between normal crying and crying that signifies colic is unclear. A similar criterion was proposed by Illingworth.[3,4]

Variations of the above definition can be seen in the literature, such as a stipulation for crying for at least three weeks,[5] crying for at least one week,[6] or crying or fussing for at least 90 min each day for six out of seven days.[6] These different definitions may reflect different degrees of severity of colic. This lack of a standard definition in the literature poses a stumbling block when comparing the outcome and the validity of much of the research carried out in this area. In an attempt to overcome this problem, Helseth and Begnum[7] proposed a comprehensive definition of crying that incorporates the three categories of crying (intense crying, non-specific fussing and crying, feeding-related crying) detected in their study. They suggest that this proposal may provide an acceptable framework for data collection and assessment of infants in clinical practice.

It is important to emphasize that a diagnosis of colic is one of exclusion and that no pathological cause for the crying can be ascertained. Other possible causes of crying such as hunger, or pathologies such as otitis media, intussusception,[a] anal fissures and urinary tract infection need to be ruled out before infants can be considered colicky.

2. Crying

The principal symptom of colic is crying. However, all neonates and infants cry and there is a daily variation in the amount of crying and the pattern of crying between infants. Research to determine what constitutes normal

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[a] Intussusception is the invagination of part of the intestine into itself. The presentation is with a painful cry, drawing up of the knees and going pale, presumably in relation to colic. Vomiting is a common symptom and the passage of redcurrant jelly stools is frequent. An abdominal mass is usually papable.

crying has been carried out by various workers[8–10] but because of the different methods used to record crying these results have varied. These studies reinforce the point that crying is a normal developmental phenomenon and that what is termed colic may simply be the upper spectrum of a natural physiological and developmental process or behavioural response in normal infants. That is to say, colic may not necessarily be a disorder but the extreme of normal early infant behaviour.

Brazelton's (1962) work in which 80 mothers documented their infants' crying in a diary for 12 weeks is often cited as the referenced norm for crying.[8] He reported that at two weeks of age, the median amount of crying was 1.75 h a day, at six weeks it was 2.75 h, decreasing thereafter to less than 1 h by the 12th week. Interestingly, twenty-five percent of the infants in his study cried for at least 3.5 h a day at six weeks of age, thus falling within the Wessel's[2] definition of colicky crying.

A more recent study by St. James-Roberts and Halil[10] also established a baseline standard for normal infant crying with the following characteristics:

- Crying is at its maximum in the first three months and an average of about 2 h crying per day is expected. This average halves between four and 12 months.
- Crying peaks at about six weeks, with individual variation.
- In the first three months, the crying clusters in the late afternoon and evening. The hours between 6.00 pm and midnight account for 40% of the 24 h total.

Interestingly Brazelton's study,[8] much like the study by St James-Roberts and Halil,[10] also showed a pattern of evening clustering of crying in that infants cried most between 6.00 and 11.00 pm at three weeks of age and from 3.00 pm to midnight at six weeks. Although this evening pattern of crying is commonly perceived to be a diagnostic feature clinically, these two studies have shown also that infants who are severely colicky tend to cry throughout the day, rather than just in the evenings.

2.1. Broadening the definition of colic

Wessel's definition of colic[2] was based solely on crying. However, disturbances in sleep, awake and contentment patterns, and disturbances in feeding frequently accompany colic and, therefore, have been used to further refine the definition of colic.[4,11,12]

In addition to crying, other symptoms and signs that may be associated with colic[13,14] include:

- Crying that is difficult to console
- Feeding problems including spitting, vomiting and crying with feeding

- Poor sleep
- Passing a lot of gas

Other signs apart from crying that may be reported or observable during a physical examination include:

- Drawing up of the legs
- Abdominal distension
- Hypertonicity
- Arching
- Flushing

These activities and motor behaviours are non-specific and may be observed as accompanying crying in infants in general. But there are some distinguishing features. Mothers often report that colicky infants appear hungry and that feeding does not always relieve the crying. Studies have shown too that there are two types of crying behaviour. The first type is where the infant is difficult to console and crying continues in spite of all parental efforts.[11,15] These infants fit Wessel's definition of colic, where a bout of crying typically lasts for 38 min, on average. This lack of soothability also characterizes moderate to severe colic. The other type is frequent bouts of crying that are calmed quickly in response to parental intervention, which may represent milder forms of colic.

Additionally, mothers often report that the colicky cry sounds different. The difference in the cry of a colicky infant has been shown to have a particular intensity[16] and a higher pitch than is normal.[17] White et al.[16] also found that their sample of 20 colicky infants cried twice as much as 20 non-colicky infants and they were less consolable.

2.2. Onset and duration of colic

The onset of colic has been reported to be between three days and three weeks of age[3] with a mean of 1.8 weeks.[18] Premature infants develop colic later than full-term infants. In a study on premature Dutch infants at an average of 32 weeks gestation, the crying peaked at six weeks corrected age, which is an average of 14 weeks after they were born.[19] This is consistent with Brazelton's data[8] on the natural history of infant crying.

In the majority of infants colic subsides by 16 weeks of age[3,20] with a reported mean of 13.6 weeks.[18] But some studies have also suggested that colic can last up to six months of age.[21]

3. Aetiology

Although the precise cause of colic is unclear, there are numerous hypotheses in the mainstream medical literature and they can be grouped under two broad headings:

1. physiological factors, ranging from cow's milk intolerance, immaturity of the gastrointestinal system to immaturity of the central nervous system.
2. non-physiological factors, such as difficult infant temperament, inappropriate maternal response and deficiencies in parenting.

In addition, mechanical factors may play a part and these are discussed later as osteopathic considerations in the treatment of infantile colic.

3.1. Physiological factors in infantile colic

3.1.1. The gastrointestinal tract: pathology and physiology

Many physiological reasons for colic are proposed in the medical literature and most relate to the gastrointestinal tract. Colic is frequently considered to be pain that is gastrointestinal in origin. This impression is reinforced by the frequent observation of wind or flatus, tense and bloated abdomen, and the intensity of crying with body posturing involving the back and limbs. But there is surprisingly little evidence that the gut is implicated directly in colic. There is certainly no association of colic with any overt gut pathology as the infant usually has good weight gain, unless the colic is extremely severe. Examination of the stool for intestinal damage such as fecal α-antitrypsin or occult blood in colicky infants has not yielded any results.[22,23]

The symptoms of gastroesophageal reflux (GER) can be confused with that of colic and the differential diagnosis is no easy task particularly when oesophageal pH monitoring is not available outside the hospital setting and is not routinely done in infants under 3 months of age. Some clinicians are of the view that not all infants with reflux have pronounced vomiting or spitting: reflux may be 'silent' with the infant crying excessively, refusing feeds or screaming soon after a feed or waking from sleep 1–2 h following a feed.[24] These symptoms of reflux oesophagitis are very similar to that of a severely colicky infant. On the other hand, studies also support the fact that oesophagitis is virtually always associated with frequent vomiting or frank regurgitation in infancy.[25,26]

Physiological differences have been reported between colicky and non-colicky infants. Lehtonen et al.[27] reported that infants with colic had hypocontractility of the gall-bladder in a study using ultrasonography. This hypocontractility was especially apparent in the evenings and during colicky crying. The causes of this finding are unclear, but disturbance of cholecystokinin (CCK) secretion or action, which normally stimulates gall-bladder contraction, may be a factor. CCK also induces postprandial sleep and feelings of satiety. Disturbance in the secretion or action of CCK may be related to the poor sleep and the apparent hunger seen in

colicky infants as well as to the finding of gall-bladder hypocontractility.

3.1.2. Intestinal motility

The symptoms of colic such as constipation and straining to stool, wind, abdominal distension and painful cry suggest disordered intestinal motility. Motilin, a gut hormone, accelerates gastric emptying and reduces intestinal transit time by increasing the motor activity of the gut. Serum levels of motilin are high in the neonatal period and decrease with increasing age. Infants with colic were found to have higher levels of cord blood motilin at six and 12 weeks of age compared to non-colicky infants and levels have also been found to be higher in neonates who later develop colic.[28,29] The finding that motilin levels are higher on day one following birth is interesting because this suggests that there is a biological predisposition to infant colic, long before the symptoms of colic develop. Gastrin and vasoactive intestinal polypeptide levels were found to be normal.

3.1.3. Malabsorption and gas

Most term infants are unable to absorb fully the lactose that is present in formula or breast milk in the first four months of life in spite of having adult levels of mucosal lactase activity.[30] Additionally it appears that some infants are deficient in the enzyme lactase needed to digest lactose, the sugar in milk. In a study by Kearney et al.,[31] milk formulas were incubated for 24 h with lactase drops and fed to 13 infants meeting Wessel's definition of colic, resulting in a mean reduction in crying time in excess of 1 h.

The undigested lactose is broken down by bacteria in the large bowel to produce gases and this may underlie the high breath hydrogen levels seen in infants[30] as well as the windy behaviour and abdominal bloating of colicky infants. It has also been observed that the quantity of incomplete lactose absorption (represented by hydrogen gas in expired air) tends to increase in the second month of life, and then decrease, in a pattern similar to the peak of crying.[32]

However in other studies where lactose intake has been reduced or lactase enzyme supplied to reduce the amount of undigested carbohydrate reaching the colon in breast and bottle fed infants, no reduction in the crying was noted.[33,34] This raises the possibility that breath hydrogen excretion may reflect fermentation of substrates other than lactose in the infant large bowel.

Apart from colonic fermentation, another source of excessive gas is swallowed air. This may result from crying itself, or from poor feeding technique and feeding position at the breast or bottle. It may also be due to inadequate winding of the infant following feeds. It may be argued that intestinal gas is the result of, and not the cause of, crying in a colicky infant. Radiographic studies attempting to define the amount of gas within the gut of colicky infants have not found significant evidence of excess gas.[35] The hypothesis that gas, whether swallowed or the result of fermentation in the gut, is a cause of infantile colic is therefore inconclusive.

3.1.4. Gut immaturity

There is evidence that the gut mucosa is affected in some way in infants with infantile colic. Human α-lactalbumin is a protein that is not present in the blood except in pregnant and lactating women, and in infants fed on breast milk. It has been shown that intestinal permeability to macromolecules such as human α-lactalbumin is increased in infants and preterm infants, measured as serum concentration of human α-lactalbumin, and this permeability decreases gradually over the first six months of age as the gastrointestinal system matures.[36] In a controlled study by Lothe et al.[37] the absorption of human α-lactalbumin was studied by measuring the concentration of α-lactalbumin in serum after the intake of human breast milk. Breast milk was given orally to 41 colicky breast-fed infants and 23 colicky formula-fed infants and the serum samples analyzed after 30 and 60 min. The results showed that the absorption of human α-lactalbumin was found to be significantly higher in *both* breast and formula-fed colicky infants, suggesting that the gut mucosa is affected in infants with infantile colic.

There are at least four sites of entry for macro-molecules to gain access to the internal environment:

1. through the epithelial cell by endocytosis, e.g. human α-lactalbumin
2. through the intercellular tight junctions when they are broken down, e.g. in malnutrition
3. via uptake of molecules at the tips of the villi
4. through the lymphoepithelium overlying lymphoid tissue

This permeability is a reflection of the immaturity of the gut and the closure of this intestinal transport system is not completely understood. However, it is clear that the intestinal mucosa does not act as a complete barrier to luminal contents, and the continually absorbed macromolecules may gain access to local lymph nodes either via the lymphatics or directly into capillaries. It is possible, therefore, that passage of food antigens via this route and the immunological response generated as a consequence may be a factor in the development of allergy or intolerance in the young infant. It should be noted that proteins are not ideal markers for measuring gut permeability as they are not inert molecules and may be hydrolysed in the gut, but evidence of their uptake through the gut wall does lend support to the development of intolerance to some factor in the diet.

3.1.5. Allergy or intolerance to cow's milk

Wessel's study[2] was conceived as a result of the observation that there was a relationship between colic and

allergy, though the conclusion of the study was that family tension seemed the more significant factor. Clinical studies have not shown that colic is more prevalent in formula-fed babies as compared to breast-fed babies.[38] Some studies report colic in infants whose mothers consume dairy products[39] but others not.[40]

An increased number of IgE plasma cells in jejunal mucosa in eight infants with colic exposed to cow's milk has been observed.[41] But the allergic nature of colic cannot be deduced from this study alone and the conclusions extended to the colicky infant population as biopsies were taken from a sub-population of colicky infants with more severe gastrointestinal symptoms including vomiting, constipation or diarrhoea and poor weight gain. Also, biopsies were taken after the age when colic has usually resolved, i.e. beyond 4 months, and the results again may not be a true representation of most infants with colic.

Lothe et al.[42] found that cow's milk seemed to be a major cause of infantile colic in formula-fed infants. The flaw in this particular study was that neither the parents nor the investigators were blinded in the use of a hydrolysed casein formula. Therefore, parental reports of improvement in crying following the introduction of the hydrolysed protein formula may be biased. Nonetheless 23% of the highly selected sample did seem to have sensitivity to cow's milk formula. So in a minority of infants at least, cow's milk intolerance may contribute to the symptoms of infantile colic.

Another study by Lothe et al. implicating cow's milk protein as a possible source of allergen was somewhat flawed in that the criterion of 1.5 h per day of crying for the colic response to the double-blind, crossover challenge of bovine whey protein or a placebo was different from the entry criterion of 3 h per day of crying.[43] Again, the parents were not blinded to the formula switch in the earlier observation phase of the study. However this study, together with their earlier study,[39] do suggest that bovine whey proteins, and in particular β-lactoglobulin, can be a contributory allergenic factor in both formula and breast-fed infants. It has been shown that bovine β-lactoglobulin was present in the breast milk of lactating mothers.[44] It has also been shown that the concentration of β-lactoglobulin was decreased in lactating mothers on a cow's milk-free diet.[45]

In a study involving the elimination of cow's milk protein from the diet of 70 formula-fed infants with severe colic, Iacono et al. concluded that a considerable percentage of severely colicky infants also have cow's milk protein intolerance.[46] In this group of severely affected infants (duration of crying >4 h a day for five days a week), the authors have suggested that dietary management should be the first therapeutic approach.

Following a systematic review of treatments for infantile colic, Lucassen and colleagues conclude that the elimination of cow's milk protein is an effective treatment for infantile colic and suggest a one week trial of hypoallergenic formula milk.[47] Their study looked at effect sizes from seven trials; they were unable, however, to establish the effectiveness of soya milks in infantile colic and state that more research is necessary on dietary treatment.

3.2. Developmental issues

Infantile colic has been attributed to the immaturity of the central nervous system (CNS). There are frequent references in the literature to this but very little in the way of evidence to support this hypothesis. The concept of an abnormally sensitive CNS in colicky babies, whereby minimal stimuli can provoke strong responses such as crying to relieve tension, was first proposed by Eppinger and Hess in 1915.[48] Spock (1944) in turn postulated that colic is an expression of CNS immaturity.[49]

Crying during the first three months may be a by-product of the major reorganization of the human brain that takes place during this period.[50] St. James-Roberts suggests that this fundamental developmental reorganization takes place at key points in the infant's life and may be responsible for excessive crying. Significant developmental changes occur when the infant is three or four months old, a time when the pattern of crying also changes. Though why or how such developmental changes can provoke crying or result in a decrease in crying as a response is not clear.

3.3. Non-physiological factors in infantile colic

One of the underlying themes in the aetiology of infantile colic is infant behaviour and interaction with the environment. It is beyond the scope of this article to review the literature in full; however, the idea of a difficult temperament leading to a less than optimal parent–infant interaction is explored.

3.3.1. Difficult infant temperament

Temperament refers to an individual's predisposition to certain behaviours or responses. The hypothesis that infants suffering from colic have 'difficult temperaments' is often cited in the literature. Temperament refers to a behaviour pattern that appears in early life that is relatively stable, at least over the first postnatal year.[51] However, an argument against this theory is that crying eventually abates, changing for the vast majority of infants early within the first year, whereas the infant's temperament would be expected to be stable. Mothers often report in clinical practice that their colicky infants are not difficult to manage during their non-crying hours, a fact also mentioned by Medoff-Cooper,[52] who asserted

that "colic should not be equated with a difficult temperament".

Carey[53] reported a significant correlation between infants with colic and infants whose temperaments were rated as difficult by their mothers. However, the excessive crying in colicky infants may have adversely influenced the mothers' ratings in Carey's study. Objective evaluation of infant temperament by an independent observer would have strengthened the results of that study. Indeed, Barr[54] suggests that it is unclear whether there is an overlap between difficult temperament and infant colic.

3.3.2. Parental anxiety and tension

The question of whether mothers whose infants are colicky are more anxious or lacking in parental skills constitutes yet another hypothesis for infant colic. Contradicting this theory is a study by Hubbard and Ijsendoorn[55] which investigated the effect of delaying the mother's response to infant crying. They found no correlation between the mother's responses with the duration of crying. There is in fact a belief that over-stimulation of the infant may exacerbate crying, a reflection of the immaturity of the central nervous system and its sensitivity to stimuli at this early age, and parents often instinctively make a conscious decision to delay responding to their infants' cries.[56]

It is unclear if the anxiety and stress experienced by the mothers are a result of their infants' colic, or vice-versa. While, surprisingly, there is some evidence that maternal depression has no effect on either the amount of crying or on care practices,[57] a prospective study has found that irritable infant behaviour can account for postnatal depression.[58]

The hypothesis that inadequate parental care causes crying is still open to debate. Indeed, excessive crying is more likely to provoke greater efforts on the part of the parents to resolve the problem. However, a systematic review of behavioural intervention trials failed to provide clear-cut recommendations on appropriate parental approaches.[47]

4. Osteopathic considerations

Osteopaths have been treating infantile colic with anecdotal success but very little research has been done to date to assess the true efficacy of osteopathic treatment and why treatment may work. Because osteopathic treatment is directed primarily at the structure of the body, i.e. the musculoskeletal system, the improvement in colic observed following treatment may suggest a possible mechanical factor in the aetiology of colic, as discussed below. However, the non-specific effects of reassurance, attention and support provided to parents during the osteopathic consultation cannot be ruled out at this stage of knowledge.

4.1. Mechanical factors and the aetiology of colic

It is possible that dysfunction of the musculoskeletal system may contribute to colicky behaviour in infants. Magoun[59] hypothesized that excessive or constant crying suggests involvement of the vagus nerve resulting from perinatal trauma to the infant skull. Osteopathic management of infantile colic has been based on the hypothesis that colic is possibly the result of vagal nerve irritation that is associated with tissue strain patterns in the cranial base, petrobasilar and occipitomastoid areas of the cranium. Such strain patterns at the cranial base may cause either direct compression or distortion of the nerve itself, or in the nerve supply (nervi nervosum) and blood supply (vaso nervosum) to the vagus nerve, leading to vagal irritation. Some support for this concept of autonomic imbalance as an aetiology for colic can be found in the conclusion by Jorup[60] that intestinal hyperperistalsis may be due to parasympathetic predominance. However, the occurrence of diarrhoea in all the infants in that study makes it doubtful if many of them had colic in the generally accepted sense. Wessel et al.[2] postulated, too, that the association of vomiting, diarrhoea and 'serious paroxysmal fussing' in infantile colic could arise from parasympathetic overactivity.

Eppinger and Hess[48] speculated that many disorders of the autonomic nervous system which manifested without identifiable disturbances in the target organs such as the heart, stomach, etc., were caused by a condition in the parasympathetic nervous system that they called *vagotonia*. Vagotonia was characterized by high vagal tone and hypervagal reactivity. It has been shown that infants older than six months of age who are unable to regulate sleep-states, to digest food effectively and to self-soothe in response to changing sensory stimuli have higher baseline vagal tone.[61] The authors in this study cautiously suggested that extreme difficultness in neonates may also be associated with high neonatal vagal tone.

Perinatal trauma to the musculoskeletal system can arise during delivery because of the immaturity of the neonate skeleton. The bones of the cranial vault do not yet have sutures in infancy and are able to overlap (moulding) to decrease the overall diameter of the head to allow for passage of the foetal head through the birth canal. Also, the bones of the base of the skull are composite bones in that they are made up of parts. For example the occipital bone is made up of four parts through which cranial nerves IX, X, XI and XII exit from the cranium. Distortion of the parts of the occipital bone may lead to disturbed function of these nerves.[59]

Compression of the cranium may arise from a long birth or an early and/or low engagement of the head towards the end of pregnancy. This is seen in the excessive moulding of the head following a protracted labour.

Table 1
Prospective studies of chiropractic spinal manipulation or cranial osteopathic manipulation in infantile colic

Citation	Infants: entry criteria	Intervention	Study design	Key results and conclusions
Klougart et al. (1989)	316 infants (2–16 weeks); crying 1.5 h/day on ≥5 of 7 previous days	Chiropractic spinal manipulation (evaluated after 2 weeks)	Prospective, observational, uncontrolled	'Satisfactory result in 94%' after mean of three treatments
Wiberg et al. (1999)	50 infants (2–10 weeks): colic crying 3 h/day on ≥5 of 7 previous days	Chiropractic spinal manipulation (2 weeks) or dimethicone (control)	Randomised, controlled, single blind	'Positive short-term effect' 67% reduction in hours/day of colic after manipulation
Olafsdottir et al. (2001)	100 infants (3–9 weeks); colic crying ≥3 h/day on 3 days/week over 3 weeks (Wessel's criteria)	Chiropractic spinal manipulation (3 visits in 8 days); placebo treatment = holding	Randomised, double blind, placebo controlled	No difference in symptom scores between groups; 'chiropractic manipulation no more effective than placebo'
Hayden and Mullinger (2006)	28 infants (1–12 weeks); inconsolable crying 1.5 h/day on ≥5 of 7 previous days	Cranial osteopathic manipulation (weekly for 4 weeks) or no treatment (parental support + reassurance)	Randomised, open, controlled (no treatment)	Significant decline in crying and improvement in sleeping

Compression of the cranium may also arise where forceps have been incorrectly applied or excessive traction used in a vacuum extraction (ventouse).

The forces of labour itself, particularly when labour has been induced by drugs, may be considerable. With each contraction of the uterus, vertical compressions are transferred to the foetus along a cephalocaudal axis. These forces are primarily absorbed in the cranial base, the craniocervical junction and the rest of the vertebral column.[62] These forces may also be dissipated into the thorax and pelvis. Additionally, compressive forces are also absorbed laterally by the pelvis, thorax, shoulders and head. Finally, rotational forces come into play when the foetal head and body moves into and through the pelvis. Acting on the head and neck, these rotational forces are absorbed by the atlantoaxial joint and, if severe, may cause distortion of the four parts of the occipital joint as well as the rest of the spine.

These forces can have different effects on the body depending on the presenting part of the foetus at birth. In a true breech, for example, the foetal pelvis initiates the passage through the birth canal and the vertical compressive forces are transmitted through the head and spine to the sacrum causing a counter-nutation[b] of the sacrum.[63] It has been postulated in the osteopathic literature that the first breath or the first cry taken by the infant on or shortly after delivery begins the normalizing of the moulding of the cranium that has resulted from the process of birth.[59,62]

Several prospective studies have lent support to the use of manual therapies for the relief of colic (Table 1). Klougart et al.,[64] Nilsson[65] and Wiberg et al.[66] reported an alleviation of the symptoms of colic as a result of chiropractic treatment of the spine. Recently, Hayden[67] reported a significant improvement in the duration of both crying and sleeping following osteopathic treatment of the head and body. A number of these studies were based on a definition for colic of crying for at least 1.5 h on five out of the seven preceding days. While this may help comparison between these studies, it is difficult to compare the outcome of these studies with those based on the more commonly used Wesssel's criteria. Furthermore, studies of base-line crying in normal infants by St. James-Roberts and Halil[10] showed that infants can be expected to cry for 2 h a day in the first three months, and Brazelton[8] showed that infants at six weeks of age cry for 2.75 h per day on average. The mean age of the infants at the beginning of the study in Klougart's study was 5.7 weeks, and in Hayden's study around six weeks. It may be argued that their criteria for colicky crying may be too close to the referenced norm.

Klougart et al.[64] used a highly select sample of infants with spinal functional disturbance. This may not be representative of the colicky infant population as a whole so that the conclusions from this study may not be applicable to all cases of infants with colic. Additionally, a control group was not used in this study.

Although the study by Wiberg et al.[66] was randomised and controlled with a blinded observer, the parents were not blinded, raising the possibility of bias in the reporting of improvement following treatment. Hayden's study,[67] a randomised comparison against an untreated control group, suffers from the same flaw in its design. Infants were not followed-up to determine if improvement was maintained in the longer term in any of these studies.

A more rigorously designed study by Olafsdottir et al.[68] concluded that chiropractic spinal manipulation over an 8-day period was no more effective than placebo

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[b] Counter-nutation of the sacrum is posterior rotation of the sacrum around a transverse axis at the level of the second sacral segment.

in the treatment of infantile colic. This was a randomised, blinded and placebo controlled clinical trial that only included infants who fulfilled Wessel's criteria[2] for colic aged between three and nine weeks and who had no signs of lactose intolerance. The limited treatment protocol (maximum of three sessions), however, may have contributed to the lack of treatment effect.[69]

5. Conclusion

There is no consensus on the clinical approach to infant colic reflecting the fact that colic is likely to arise from the interplay of physiological and non-physiological factors, as well as possible mechanical factors. It can be concluded that colic is multi-aetiological in origin and that several or all the factors mentioned may play a role in this condition. Mechanical factors contributing to colic are not adequately considered in the literature but may be of relevance in view of the anecdotal reporting of clinical success in the osteopathic management of colic and the improvement of the symptoms of colic as a result of manipulation in recent osteopathic and chiropractic studies. This needs to be investigated further with well-designed studies involving larger sample sizes, more rigorous inclusion criteria and longer term follow up.

Acknowledgements

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