**Current topic**

**Infantile colic revisited**

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More than 30 years ago, with the help of the *Index Medicus*, I reviewed the world’s published reports on evening colic. Since then the *Index Medicus* has listed about two papers a year, with no advance in our understanding, but with the coming and going of a highly effective drug, dicyclomine hydrochloride.

The early term ‘three months’ colic’ confused many, who interpreted it as meaning colic beginning at age 3 months. I prefer the term ‘evening colic’, because of its characteristic circadian rhythm of pain in the early evening. Similar attacks of pain may occur during the day in the older infant, at around 6 months of age, but this may or may not be the same condition. The term ‘infantile colic’ became popular, but is too all embracing, and in the minds of many includes troublesome crying for any reason—hunger, thirst, overheating, an itch, fatigue, allergy, boredom—but above all, crying which promptly stops when the baby is picked up and loved. Some (other than me) would add overfeeding as a cause. A sensible term is ‘paroxysmal fussing’. I am told that in India and the Punjab it was called ‘sanjhana’, and was deemed to be due to the evening gods.

**Definition**

The classic picture as described by Joe Brennemann of Chicago, Benjamin Spock, and others, was that of a well, thriving baby who in the early evening, for no apparent reason, develops paroxysms, beginning with flushing of the face, a frown, drawing up of the legs, followed in a few seconds by high pitched screaming, suddenly ending in a few minutes, and followed in a few minutes by another paroxysm. The attacks recur for up to two or three hours. In the attack the baby is unconsolable, there are loud borborygmi, and the pain is relieved by the passage of flatus from the rectum (not from the stomach), by the passage of a stool, or, it is said, by an enema. The above description is that of moderate or severe cases. In milder ones there is merely otherwise unexplained intermittent fussiness in the evenings. The attacks begin in the first week or two and cease by the age of 2, 3, or occasionally 4 months. It has been said that the onset is delayed in preterm babies.

The condition is common, but because of imprecise definition, figures for its incidence are of little value.

**Aetiology**

**Psychological factors.** A popular view, especially among psychiatrists, is that the colic is due to the mother’s psychological problems, excessive anxiety, picking the baby up unnecessarily (and boding him after a feed) and ‘misinterpretation of infant cries leading to ineffective responses’-Taubman, but Taubman’s definition of colic was inadequate. Wessel, using a satisfactory definition of colic, thought that ‘family tension’ was an important factor. Lakin, in a study of 20 mothers of babies with colic and controls without colic, found ‘poorer parent child relationships, greater intrapersonal conflict, conflict over role acceptance, concern about inadequacy as a female, poor marital adjustment, and less motherly love’. On the other hand Paradise, with his characteristic thoroughness, found no connection with emotional problems. He found that the mothers were stable, cheerful, and feminine. On the basis of clinical impression, I have never noticed characteristic features in the mothers; nor have other paediatricians. None of the psychiatrists who blame the mother for causing the colic made any attempt to explain exactly how the mother causes the infant to have pain that is obviously of intestinal origin. Neither do they seem to realise that mothers, particularly in the evening when they are tired after a hard day’s work, are likely to be upset by the baby’s obvious pain and their inability to console him. If the colic were due to maternal anxiety, one would expect it to occur more often in the case of the first born. Though there is some disagreement, there is good evidence that this is not the case. I suggest that a mother’s anxiety is not the cause but the result of the colic.

Several have suggested that the baby’s tempera-
ment is the cause, or his sensory threshold for pain. This is a reasonable suggestion, but the temperament does not necessarily cause the colic: the temperament and colic may both be due to other factors. In the many hundreds of colicky babies whom I have seen, I have failed to notice anything characteristic about their temperament. Bruce seemed to suggest that the baby with colic is a malingerer; he wrote ‘I feel sure of one thing: the infants are usually not in as much pain as they appear to be, or as their parents think they are’.

**Allergy.** It is difficult to interpret the many papers on allergy to cows’ milk as a cause of colic, because of the lack of satisfactory criteria. The Malmö workers defined colic as paroxysms of severe crying with abdominal distension and frequent sucking. I have not found that abdominal distension or frequent sucking are notable features of evening colic: radiological studies during attacks of colic did not show gaseous intestinal distension.

In one study, 60 infants were fed on either a cows’ milk or soya formula. Eleven babies were free from colic on soya, 32 were no better when given both cows’ milk and soya, but lost their symptoms when fed on hydrolysed casein. Seventeen were no better when tried on a cows’ milk free formula or soya. A third of those free from colic on the milk free diet developed colic again when challenged with milk. In a later study, 66 mothers of 66 breast fed babies who had colic were tried on a milk free diet; 35 babies lost their colic but developed it again when the mothers were challenged with cows’ milk. Ten mothers were given cows’ milk whey capsules and nine of their babies then developed colic.

Le Blanc regarded the Malmö work as not statistically significant, and himself found that a soya formula without cows’ milk did not help. Evans in a double blind study of 20 breast fed babies, did not find that elimination of cows’ milk from the mother’s diet prevented the colic: he argued that the relevant factor in the mother’s diet was not cows’ milk but a variety of foodstuffs. Liebman, with a satisfactory definition of evening colic, found that lactose intolerance was not a factor; as for cows’ milk protein, he found no difference in the babies’ IgE or radioallergosobent test results (cows’ milk protein) when compared with controls.

In my view, the Malmö workers showed that allergy to cows’ milk protein or to soya could cause abdominal pain in infants: but I am not satisfied that it is a common cause of evening colic. If allergy were an important factor for evening colic, one would expect that there would be a significant incidence of allergy in the family, of other manifestations of allergy in the infant, such as eczema or asthma, or other evidence that the baby’s colic would cease when the mother avoided cows’ milk. There is much evidence against this. This does not exclude allergy, but it makes it less likely.

There is a difference of opinion as to whether infantile colic is more frequent in breast fed babies. My impression is that it is so. Others had the same opinion, and some disagreed.

**Passive smoking.** A French report suggested that some cases of colic were due to the baby inhaling tobacco smoke. It has been shown that nicotine and cotinine are found in considerable quantities in the infant’s saliva, urine, and serum when exposed to passive smoking, or when taking breast milk from a mother who smokes. These findings should be investigated, but may prove to be irrelevant.

**Other factors.** Various workers have found that infantile colic is not related to the mother’s age, parity, or social class, or to the baby’s mode of delivery, prematurity, birthweight, deficient or excessive weight gain, vomiting, diarrhoea, constipation, parity, or as much pain as they appear to be, or as their parents think they are’.

A theory and a call for research

The symptoms of evening colic, with the rhythmical paroxysms, the loud borborygmis, the relief by the passage of flatus per rectum, or by the passage of a stool or an enema, must surely indicate an intestinal origin, as Brennemann and other suggested long ago. Perhaps there is intestinal spasm. A Swedish study reported colonic hyperperistalsis and increased rectal pressure, responding to an anticholinergic drug, methyl scopolamine nitrate. The fact that another anticholinergic drug, dicyclomine, was strikingly successful in preventing evening colic, suggests clues for research. Perhaps other anticholinergic drugs, such as propantheline, would be effective. An anticholinergic drug would reduce gut motility and slow transit time.

I believe that the enigma of evening colic now calls for fundamental combined physiological and clinical research. The role of vasoactive intestinal polypeptide (VIP) and gut hormones (for example motilin, enteroglucagon, neurotensin, and opioid) deserves investigation. The VIP is thought to mediate relaxation of the gut musculature, inhibit peristaltic reflex, and cause gastric relaxation.
it has been implicated in many intestinal conditions associated with diarrhoea, such as the ganglion
euroma. The intestinal opioid peptides\(^3^4\) are also concerned with gut motility. Two constipated
adults, one of whom had been constipated from childhood, promptly responded to the specific
opioid antagonist naloxone.

The intestinal prostaglandins have a profound effect on the transport of water and electrolytes
through the intestine, and on intestinal motor function.\(^3^1\) Injection into volunteers caused
abdominal colic.\(^3^5\) Raised prostaglandin E was shown in an adult with functional intestinal
obstruction.\(^3^7\) An Israeli report linked prostaglandin with diarrhoea of different causes. Dodge in
Cardiff\(^3^9\) showed that in the benign ‘toddler’s diarrhoea’ there were raised prostaglandins,
especially PGF\(_2\) \(_\alpha\) and that the diarrhoea was inhibited by prostaglandin inhibitors, indomethacin, or aspirin.

It is of interest that Murray Davidson\(^4^0\) wrote that infantile colic predisposes to later functional
gastrointestinal problems, and Jorup\(^2^3\) wrote that 40% of infants with colic later had diarrhoea or pains of
‘spastic colon’. But in a 13 year follow up study of 30 babies with colic,\(^4^1\) only one had recurrent abdomi-
nal pain in later childhood. A report from Finland\(^4^2\) noted a previous history of infantile colic in 12 of 27
children in a clinic for toddler’s diarrhoea.

I believe that intestinal physiology may provide the clue to the enigma of infantile colic (and
incidentally to otherwise unexplained constipation in infants and perhaps to recurrent-abdominal pain
in the young school child).

**Treatment**

In my review\(^1^1\) I listed the numerous treatments advocated in the published reports. Later,\(^4^3\) I
described a controlled trial of the anticholinergic drug, dicyclomine hydrochloride, indicating that it
was strikingly successful in preventing evening colic. This has since been confirmed in Australia\(^4^4\) and the
USA.\(^4^5\) Provided that the child had the classic picture of evening colic, as described, I have
virtually never known the drug fail to provide relief—and the experience included many scores of
infants. I am unable to understand the experience of one doctor\(^4^6\) who found it ineffective—but he did
not name the criteria for his diagnoses.

Most regrettably, the drug has been withdrawn after reports of possible untoward reactions. In one
report\(^4^7\) two children apparently had an immediate hypersensitivity reaction with respiratory difficulty.
Two letters following this report described two possibly similar cases. I could not interpret their
importance because of the absence of the necessary details. According to one of these letters,\(^4^8\) the child
had received a mixture of dimethicone and dicyclomine, and the writer chose to attribute the symp-
toms to the dicyclomine. The manufacturers told me that the Committee on Safety of Medicine had
received 15 to 20 reports of similar symptoms, and the report of a ‘cot death’ of an ill triplet. It is not
possible to assess those reports without knowledge of the children’s condition before the drug was
given, its dosage, and the time interval between the administration and the onset of the symptoms.

Two writers\(^2\)\(^4^5\)\(^4^9\) described an episode of rapid breathing before an attack of colic. I have no
experience of this sequence, but did not specifically ask about such symptoms. In another paper\(^4^9\)
infantile colic was linked to a ‘near miss sudden infant death’.

I know of no drug that is always free of side effects. Millions of doses of dicyclomine have been
given for over 30 years without trouble. I feel sure that the drug was grossly over prescribed, for it
seemed to be regarded as a panacea for crying, whatever the cause. Mothers are exhausted and
worried by a baby’s unconsolable crying—a symp-
tom that may lead to non-accidental injury; and infants suffer severe discomfort. We can now no
longer prevent it as the only effective drug known has been withdrawn—presumably because of the
fear of litigation. But the fear of litigation is a poor reason for causing human suffering.

A widely used drug for infantile colic is dimethicone, consisting of silica, sold under several trade
names (for example Asilone, Dentinox colic drops), sometimes combined with dicyclomine, aluminium
hydroxide, or magnesium oxide. All the oral prepar-
ations listed in MIMS carry the warning that it is
either not recommended at all for children, or not
for those under 1 month of age. It has been used to
relieve abdominal distension in adults\(^5^0\)\(^5^1\)—it was
thought to accelerate the transit of intestinal gas, but
had no effect on the volume of gas recovered, or the
number of flatus passages. In another report,
quoted by Martindale,\(^5^2\) it was found to be of no
value for gastrointestinal flatulence. In a double
blind crossover study of 27 cases of infantile colic\(^5^3\) it
was ineffective in relieving the symptoms. I find it
difficult to understand how a suspension containing
15 to 30 mg of an absorbable preparation of silica in
about 350 cm of intestine would prevent or relieve
the pain which one presumes is caused by intestinal
spasm or functional obstruction.

Innumerable other drugs continue to be used for
infantile colic. O’Donovan and Bradstock\(^4^4\) tried
four drug regimens—homatropine with phenobarbi-
tone and alcohol, phenobarbitone and alcohol,
alcohol alone, or a placebo; all the first three,
probably because of the alcohol, were better than the placebo. Some doctors prescribe gripe water: perhaps it achieves something because of its alcohol content. A patient of mine had been given regular doses of whisky for his colic. In developing countries I have seen scars of multiple abdominal incisions to let the pain escape. A paediatrician found that his own children responded only to a rocking cradle or a ride in his car.

I agree with O’Donovan and Bradstock, that ‘if we knew what we were treating, perhaps the therapy would be more successful. When the majority of patients improve no matter what is done for them, the uncritical therapist will conclude that his treatment is effective’.

Conclusion

The time has come for basic physiological research into infantile colic; but an essential preliminary is a precise clinical diagnosis; as precise as it can be in the absence of any laboratory tests to confirm it. Cases should satisfy the criteria suggested, but exclude (in the first place) similar cases in older infants, because other factors, such as allergy, may be more likely then and so might confuse the picture. The method of feeding, breast or formula, and the age of the child, should be recorded as it is a self limiting condition, resolving by about 3 months of age. Infantile colic may well prove to have a multifactorial causation, like so many other medical conditions, with the end result evening colic; but what we then want to know is what is the nature of the final common pathway that culminates in colic.

I will conclude by paraphrasing the final comment by Prensky in his discussion of the unfounded scare concerning the dangers of pertussis vaccine ‘This 50 year old controversy, punctuated by limited clinical studies and testimonials, will remain a monument to the ineffective nature of anecdotal reports’.

References


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