

Changing Our Understanding of Infant Colic

COLIC IS truly a noisy phenomenon in terms of the crying that the infant does. But it is also a noisy phenomenon to study in terms of the basic phenomenology of crying that constitutes the defining behavioral feature of the syndrome. As can be appreciated in Figure 3 in the article by Clifford et al¹ in this issue of the ARCHIVES, even at 3 months of age, after most colic has resolved, there is still a substantial range of minutes per week (or per day) of crying and fussing behavior. This is also true in the sixth week of life, when crying is usually manifest at its highest levels.²⁻⁴ In addition to the range of “individual differences” in levels of crying among infants, there is no single level of crying in early infancy that is normative.⁵ Indeed, the total number of minutes of daily distress manifest an n-shaped pattern over the first 3 months or so of life: they begin to increase at about 2 weeks of age, peak during the sixth week, and then decline to under 1 h/d by 12 weeks of age. While this pattern is typical for groups of infants, individual infants may experience their maximum distress at earlier or later ages so that a single measure at a particular age may not capture the maximum for that infant (see Figure 2, Barr⁴).

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All of this has made early crying and its main clinical manifestation of colic difficult to study. Nevertheless, an increasing number of studies with a variety of designs has brought some degree of “order”⁶ to this noisy phenomenon. Among the themes emerging from these findings are that (1) the n-shaped (or “peak”) pattern of crying, long thought to be the defining characteristic of the clinical syndrome of colic, is now understood to be a likely behavioral universal of normal infant development in the sense that human infants share a propensity for increased crying that is characteristic of all groups of infants in the human species, if not of all infants⁶; (2) probably all of the other defining features of the syndrome can also be accounted for without positing any pathophysiological process or abnormality in most infants with colic^{7,8}; (3) the levels of crying variously and variably described as defining characteristics of colic syndrome^{9,10} reflect in most cases the upper end of the spectrum of early developmental crying behavior, such that the prevalence varies depending on the time of measurement and the cut-off value of daily distress used; (4) despite its name that suggests gastrointestinal tract problems, the prolonged crying episodes may well be related to indi-

vidual differences in central nervous system functioning rather than gastrointestinal tract dysfunction^{8,11-13}; and (5) the outcome for infants with colic is good, at least in low-risk populations and in the absence of significant comorbidity or stress in the infant and/or parents (see Clifford et al¹ and others¹⁴).

None of the above is inconsistent with there being some infants with significant disease or pathophysiological processes, or with abnormal cries or amounts of crying, being included in the group of infants who meet clinical criteria of colic.^{15, 16} Nor is it inconsistent with there being increased crying in mother-infant dyads in which the normal coregulatory interactions that modify the infant behavioral state have broken down.^{17,18} Furthermore, it remains possible, if still hypothetical, that the source of the disturbance could be situated in the gastrointestinal tract even in the absence of disease processes if the crying reflects visceral hypersensitivity to otherwise innocuous intestinal stimuli.¹⁹ This would be the infant analogue of the processes of “hyperalgesia” (which refers to a reduced pain threshold or a greater or longer duration of response to a painful stimulus) and “allodynia” (which is when painful or discomforting experiences are due to stimuli that do not normally produce pain or discomfort) in adults. If one follows this hypothesis, the hypersensitivity occurs when there are changes in the sensitivities of the primary afferent neurons or in secondary dorsal horn neurons.^{20,21} The fact that local interneuronal inhibitory connections in the substantia gelatinosa and the descending inhibition (“gating”) from the brainstem on dorsal horn cells are all postnatal events (in the rat and probably in humans) might account for the decrease in crying during the third month owing to this mechanism.²² In short, the new information about the normative developmental crying pattern has not solved the clinical problem of infant colic; rather, it has provided important basic information against which these other processes now need to be assessed with regard to diagnosis, therapy, and prognosis.

The article by Clifford and colleagues¹ in this issue is the latest of the still too few prospective studies of infant colic in which, importantly, there was some means of measuring infant colic when it occurred, rather than depending on parent recall.¹⁴ They report a number of important observations, including the prevalence of levels of crying consistent with colic at age 3 months (6.4%), and that 85% of cases with colic remitted by age 3 months. They also report that, at least in this relatively low-risk population, (trait) anxiety and postnatal depression are not elevated in the mothers of infants who previously had

colic. Importantly, this was assessed with control measures of anxiety and depression at 1 week post partum, a methodological strength of this study missing in most others (but see also Murray and Cooper²³).

To my mind, by far the most intriguing observation is not even mentioned in the abstract and may turn out to be the most important one for our future understanding of this syndrome. This is the documentation of the fact that of the 6.4% of infants who met criteria for colic at age 3 months, only about half of those met criteria for colic at 6 weeks and at 3 months of age (which they called “persistent” cases) and the other half did *not* meet criteria for colic at 6 weeks but *did* at 3 months of age (which they called “latent” cases). There are a number of reasons why this observation may be particularly prescient.

One reason is that it has been thought for some time that, in the absence of pathology, infant colic is the earliest manifestation of later “difficult” temperament. Although temperament caregiver report measures taken when the infant *has* colic always confirm one or other form of “difficultness,”^{24,25} prospective longitudinal studies do not support this hypothesis (see especially Lehtonen et al²⁶ as well as a review of this evidence by Barr and Gunnar²⁴). The question is, if infants with prior colic do not become infants with difficult temperaments later, which ones do? One possibility, counterintuitive though it may be, is that infants with difficult temperaments actually tend to be those who do *not* have colic early, but whose difficultness emerges after the early 3-month period, or following what is sometimes referred to as the “biobehavioral shift.”²⁷ Barr and Gunnar²⁴ have incorporated such a speculation in their “transient responsivity hypothesis” about infant colic. They argue not only that infants with colic are as well regulated as infants without, but that they may even be better regulated later. Although intriguing, the evidence for this is still very weak. If Clifford and colleagues decide to do a follow-up study on these infants (as they have promised), they may be able to address this speculation empirically.

A second reason is this. Mechthild Papousek and her colleagues^{17,18,28,29} have been carefully documenting for some time the interactional breakdowns that occur between constantly crying infants and their caregivers in the first year of life who present to their exceptional clinic in Munich, Germany. A proportion of their patients clearly have prior colic with crying that continues. However, they also tend to be “high risk” in the sense that there are also disturbances in feeding and/or sleeping, mild developmental delays, and organic risk factors—a group that has been dubbed the “persistent mother-infant distress” syndrome group.^{7,18,30} Because these were clinical samples in which prior colic could only be obtained by history in most of the cases, it was not clear whether these infants and families represented a “worst case” scenario for the outcome of colic syndrome, or whether they represented a distinct syndrome that would have occurred anyway, whether or not the infant had earlier colic, possibly due to different developmental factors that contribute to infants becoming temperamentally difficult *after* 4 months of age. The findings reported by Clifford et al are, to my knowledge, the first to use crying measures to document such latent emergence of increased, clinically sig-

nificant crying. This makes it likely that there are at least 2 pathways to the mother-infant distress syndrome. In light of these findings, and to respect the developmental nature of these syndromes, it is probably important to reserve the term colic for the *early* increased crying, and something else such as “caregiver-infant distress syndrome” for the later emerging clinical presentations. “Persistent caregiver-infant distress syndrome” could then be used for those who had increased crying *early and later*, after typical colic had resolved.

In their discussion, Clifford and colleagues comment that the relatively good prognosis they describe “should not justify the abandonment of research.”^{1(pswk)} Indeed, not. More research is critical even if (as I and others have argued elsewhere^{7,8,31}) the n-shaped curve of early infant crying with its large interindividual differences and accompanying prolonged episodes of unsoothable, painful-appearing distress are a manifestation of typical behavioral development, probably an inheritance from our evolutionary history—in short, even if it is completely “normal.” The increasing recognition that these very properties of early crying are the most important stimuli for the tragedy of shaken baby syndrome when caregiver tolerance is exceeded is a stunning reminder of how important a thorough understanding of this phenomenon is. If true, it seems unlikely that there will ever be a “cure” for colic. In recognition of this, the National Center for Shaken Baby Syndrome is preparing materials for a prevention campaign (available at: <http://www.DON'TSHAKE.com>). The theme is to make everyone aware of “The period of PURPLE crying,” where the letters of the word “PURPLE” each refer to one of the properties of early crying that is so frustrating to caregivers (P for peak pattern, U for unpredictability of the crying bouts, R for resistance to soothing, P for painlike facial expression, L for long crying bouts, and E for evening clustering). Hopefully this knowledge will decrease the frustration that comes from caregivers thinking they are “failing” to be a good parent when their infant cries and thus decrease the impulse to shake the child. Clifford and her colleagues are right: despite the good news in their study, there is still much more we have to learn—and to do—in relation to early crying.

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