While cry serves as an important cue frequently used in making judgments of pain in infants, once attention has been attracted, its importance diminishes because it is not definitive. In studies of nurses judging infant pain, cues such as context, facial activity, and body movement, as well as vocalizations, were identified as "frequently useful" in making pain ratings (Fuller & Conner 1996; Pigeon et al. 1989; Shapiro 1993). In a survey of nurses, restlessness and facial activity were more frequently identified as indicators of pain in infants than cry (Howard & Thurber 1998). In a study of parents judging pain in infants receiving immunization injections, infant vocalizations were secondary to facial activity in importance as cues (Smith et al. 2002). These studies examined self-reports of cues, whereas others have explored the strength of the relationship between cues and assessed pain levels. In a study of parents viewing videotapes of infants receiving heel lances, facial activity accounted for 43% of the variance in ratings of pain sensation and 49% of the variance in ratings of pain affect, while fundamental frequency accounted for only an additional 2% of the variance (Craig et al. 1988). In another study by Hadjistavropolous et al. (1994), cry characteristics accounted for 38% of the variance in observer pain ratings, while facial activity accounted for 49% of the variance in ratings. When facial activity and cry characteristics were considered together, they accounted for 54% of the variance in pain ratings, leading to the conclusion that cry was mostly redundant to facial activity. Once attention has been attracted, therefore, caregivers rely more on directly observable cues, because they provide more specific information.

This is not to argue that the signal value of infant cry is unimportant, but rather, that it needs to be viewed in relation to infant behavior, environment, and social context. Soltis suggests that chronically abnormal acoustic characteristics of infant cry may be a precipitating factor for neglect, abuse, and infanticide in humans, and he outlines an excellent series of studies that would incorporate features of contextual factors and caregiver characteristics and reactions in order to understand mechanisms for caregiver judgments and actions. Appreciating infant well-being and survival necessitates an understanding of not only cry and infant characteristics, but also the caregiver and the relationship to the child. For example, caregivers having greater experience with infants are more accurate in differentiating between different types of cries (Craig et al. 1988; Owens 1984), and therefore are more likely to be able to identify sources of infant distress. In addition, characteristics of the caregiver, such as cognitive biases, sensitivity, knowledge of and relationship to the infant, can influence this process (Craig et al. 1996).

Caregivers unable to determine the source of an infant's distress could respond with either frustration or denial. A caregiver faced with an infant crying for unknown reasons may respond with abuse out of a sense of complete exasperation and frustration with being unable to soothe or quiet the child. Conversely, a caregiver may suspend sensitivity to an infant's crying if faced with the unacceptable reality that an infant is suffering and nothing can be done to relieve that distress (McGrath & McAlpine 1993); this could potentially result in neglect. As Soltis correctly points out, these extreme responses to abnormal infant crying likely originate in adult dispositions prior to parenthood.

Finally, although Soltis limits his discussion to early infant cries, before the age of three months, the target article invites speculation about the processes whereby maturation and experience enhance the signal functions of crying. Little is known about how crying becomes a speech act and how language concerning pain and distress emerges and comes to control caregiver behavior across infancy. Neurodevelopmental maturation of infants and social learning factors likely lead to changes in cry acoustics, and this can influence the signal functions of cry. Recent calls for an exploration of developmental changes in infant pain cry (Green et al. 2000) suggest the importance of considering developmental factors in the communicative aspects of cry.

It is now important to follow up with research to provide empirical basis for this area of study, taking into account behavioral, social, environmental, and developmental context. We are grateful to Soltis for provoking debate into the field of communication of infant distress.

Infant crying and colic: What lies beneath

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Abstract: The neural structures implicated in crying are reviewed, based on studies in animals. Brain regions involved include the anterior cingulate gyrus (a cortical structure), amygdala, thalamic tegmentum, periaqueductal gray of the midbrain, and the nucleus ambiguus of the caudal brainstem. It is hypothesized that the crying associated with colic may be a manifestation of differing developmental stages in the brain circuits involved.

Soltis has provided a useful overview of infant crying, a topic not heretofore covered in a *BBS* target article. His main thesis is that crying, including the excessive crying associated with colic, is an adaptation by which an infant signals its need and vigor to caregivers. The acoustic details of crying, basic elements of which are regulated by vagal mechanisms, matter in terms of their signal value and play a role in the quality of parental care. From an evolutionary perspective, crying in human infants perpetuates the ancient function of signaling distress or discomfort and increasing proximity to, and caregiving behavior from, parents and other caregivers.

This commentary will address several points that deserve further elaboration, drawing from comparative studies and neuroscience.

Neural circuits mediating crying and their maturation have been identified in animal studies. In some nonhuman primates, infant cry sounds continue to be produced into adulthood, and serve to reunite separated individuals from their mate or social group. These so-called *isolation calls*, or *separation cries* are acoustically similar to the "cry sounds" produced by infants of these species during periods of maternal separation (such as accidental falling off from the mother while being carried, or, when older and more independent, when frightened or hungry) (cf. Newman 1985). Because the functions and acoustic details of the cry sounds of most nonhuman primates (and many other mammals) are so similar across species (including humans), I have suggested that the mammalian infant cry and the mechanisms regulating its production have had a conservative evolutionary history. Our understanding of these regulatory mechanisms is still rather primitive, but some details are beginning to emerge. While it is true that brain stem motor neurons of the vagus nerve innervating the larynx undoubtedly have an important role in regulating basic acoustic features of infant cries, they do not operate in the absence of higher-order brain stem and cerebral control.

Early studies in lightly anesthetized cats showed that stimulating the cut surface of the midbrain produced meows and other vocalizations (Kelly et al. 1946). More recently, Buchwald et al. (1988) stimulated the hypothalamus in freely moving cats and produced meows similar to the cat's natural isolation calls. Disruption of normal vocal patterns by selective lesions is a good indicator of the importance of a brain region in a given behavior. Lesions of the thalamic tegmentum in adult squirrel monkeys produced disruptions of the normal acoustic patterning of isolation peeps (the species-specific isolation call) without altering the tendency to make these calls in the appropriate context (Newman & MacLean 1982). Bilateral lesions in and adjacent to the midbrain periaqueductal gray resulted in selective mutism, in that lesioned animals failed thereafter to make isolation peeps, but continued to make other vocalizations, such as shrieks when caught (Newman &

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MacLean 1982). One cortical area in particular, the anterior cingulate gyrus, has been implicated in cry production in several studies. In a species of CF-FM bat (a species that produces long whistle-like notes when searching for prey), stimulating the midline limbic cortex (comparable to the cingulate gyrus of primates) produced CF whistles, calls that are structurally similar to infant isolation calls (Gooler & O'Neill 1988). In macaques, Robinson (1967) found that stimulating the midline cortex below the genu of the corpus callosum resulted in "coo" calls (this species' isolation call, but also used in other social contexts). Jürgens and Ploog (1970) elicited tonal vocalizations, including the isolation peep, by stimulating in the corresponding region of the squirrel monkey brain. Sutton et al. (1981) found that ablating the peri-genual anterior cingulate gyrus in macaques eliminated production of coos in macaques trained to make coos under operant conditions. MacLean and Newman (1988) likewise found that lesions of the peri-genual region of the anterior cingulate cortex in adult squirrel monkeys eliminated production of the isolation peep in a more natural setting not involving operant conditioning. Few studies have examined the brain substrates mediating isolation call production in infants, but Newman and Bachevalier (1997) reported that bilateral ablation of the amygdala in macaque neonates resulted in production of fewer isolation coos, and coos with less inflected frequency peaks when tested between 6 months and 1 year of age. Interestingly, bilateral ablations of area TE in the inferior temporal cortex, considered to be a higher visual processing region, led to animals that were more vocal and, at least in males, made more noisy calls when separated in the 6-month to one-year age period.

Is the excessive crying associated with colic a manifestation of developmental delay or disregulation? As the brief review above indicates, several brain regions extending the entire length of the neural axis (cortex to brain stem motor neurons) can influence crying in terms of the tendency to cry and the acoustic details of the cry sounds. Given the complex interactions likely to occur between these structures, it seems possible, should the maturation of neural circuits not proceed at the same rate for all of the relevant structures, that variability in infant crying behavior might ensue. Circuits underlying responsiveness and tendency to cry that mature at a different rate than circuits underlying cry acoustics could lead either to bouts of poorly regulated crying or cry sounds that tend toward outliers in their acoustic details. Evidence for both of these is presented in Soltis' target article. To test this hypothesis, it would be necessary to evaluate the developmental status of specific brain circuits associated with crying in subjects for which detailed information on their crying behavior was also available. Given the lack of feasible methods for studying the maturation of specific brain circuits in human infants, continued findings from animal studies will be important for future investigations aimed at testing this hypothesis.

NOTE

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Infant vocalizations: Contrasts between crying and laughter

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Abstract: Crying and laughter are innate, preverbal, species-typical vocalizations that have similarities and differences which are mutually illuminating.

Crying, as noted by Soltis, is a potent, early appearing, species-typical human vocal signal that is uttered in a similar way by members of all cultures and vocal communities. The same can be said for laughing. In the following comparative treatment, I contrast similarities and differences between crying and laughing to better characterize each utterance and illuminate the general issue of innate vocal signaling. Unless otherwise noted, themes concerning crying are taken from the target article by Soltis; those about laughter are from Provine (2000).

Crying and laughing both develop very early in life; crying is already present at birth, while laughing emerges about four months later. Crying is often performed by solitary infants, in contrast to laughter, which is seldom produced outside of social settings. These differing degrees of sociality are consistent with their purpose; crying is the solicitation of attention by a caregiver, who may not be present, while laughter is a play vocalization engaging an already present person. Laughter is an important form of prespeech communication that contributes to bonding between infant and mother. The infant responds to maternal touching and tickling with laughter and smiling, acts reinforcing further maternal touching until it becomes excessive, when the infant begins to fuss and cry. Infant laughter and associated smiling are powerful, reinforcing stimuli that encourage the mother to maintain the behavior that evoked it. In contrast, infant crying is a highly aversive stimulus to the mother, who works to terminate it. Although crying and laughing differ greatly in function and social context, they nicely complement each other and contribute to infant survival and development.

Regarding the issue of "honesty of signaling" in crying, Soltis should consider the constraints against dishonesty. Species-typical vocalizations such as crying and laughing are under minimal conscious control and are poor candidates for behavioral shaping. The best demonstration of the lack of conscious control over crying and laughing is to attempt to cry or laugh on command. About half of the people I have asked to laugh comment that they cannot do so on command, and those who attempt to laugh produce obviously false simulations. Although similar data are not available for crying, I expect the same result. Recognition of the difficulty in voluntarily producing such expressive acts as crying and laughing was a motive for the development of the technique of method acting – actors try to produce a convincing emotional expression by imagining themselves in a situation that evokes that emotion. The limited voluntary control that we have over crying and laughing indicates that we should be reluctant to explain these acts in terms of rational choice. We do not "decide" to cry or laugh.

Although there are neurological constraints on the "honesty" of crying and laughing, atypical expressions of these utterances are leading examples of abnormal and inappropriate affect, significant symptoms of psychopathology and neuropathology. Frequent comorbidity of crying and laughter suggests related brain mechanisms for the two acts. Consider, for example, the emotional lability sometimes observed in amyotropic lateral sclerosis (ALS) and other instances of so-called pseudobulbar palsy. The patient may bawl uncontrollably in response to minor sadness or laugh uncontrollably in response to the joy of seeing an old friend. (Some patients may laugh until they cry, but the converse is not observed.) Epileptic seizures may drive either laughter or crying by producing the emotionless expressive act or secondarily by producing the associated emotional state.

Although not mentioned by Soltis, preliminary evidence suggests that infant crying is contagious (Simner 1971). The crying of one infant triggers crying in its neighbors. Contagion provides an amplifier effect, spreading a vocal signal from individual to individual in a behavioral chain reaction. The contagiousness of laughter is better known, and is the basis for the notorious laugh tracks on television situation comedies. However, you can still get laughs if you throw away the joke and just keep the laugh track – laughter is sufficient to trigger laughs and smiles in an audience. The age of onset of contagious laughter is unknown. Contagious signaling is an effective but neglected means of synchronizing the biological state and behavior of individuals in groups.

Crying is an undeniably powerful stimulus that can evoke re-

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