Structure and Irregularity in the Spontaneous Behavior of Young Infants

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Persistent, irregular fluctuations in spontaneous motor activity are common in the young of many vertebrate species, but whether the irregularity is intrinsic to the dynamics of motor activation or the result of random perturbations is not known. Analysis of the second-by-second variation in the general body movement of awake human infants 1 and 3 months after birth revealed low dimensional structure in the characteristically irregular motor activity and exponential rates of divergence of initially similar states of motor activation. Results support the conclusion that irregularity is an intrinsic property of the dynamics of motor activation involving relatively few effective degrees of freedom and raise questions about the advantages or disadvantages of irregularity built into early behavioral organization.

In the human fetus, neonate, and infant, there are irregular fluctuations in spontaneous motor activity (cyclic motility [CM]) composed of general movements of the limbs, trunk, and head (Robertson, 1990). The fluctuations occur on a time scale of a few minutes or less. A fair amount is known about the early development of CM in the human. Far less is known about the mechanism responsible for the characteristic fluctuations in motor activity or their functional significance. In this report, we describe our efforts to use some of the theory and methods from experimental dynamics to understand the core properties of CM. Insight into the core properties of CM will guide attempts to understand both the mechanism and functional significance of the ubiquitous fluctuations in spontaneous motor activity.

CM emerges before midgestation in the human, perhaps as early as the 12th postmenstrual week (De Vries, Visser, & Prechtl, 1982). The properties of CM, especially its irregularity, are stable during the second half of gestation, and the substantial physiological and environmental changes at birth do not appear to induce any lasting changes in CM (Robertson, 1985, 1987). Fetal CM is influenced by maternal diabetes but is normal by the end of gestation and continues so after birth (Robertson, 1988; Robertson & Dierker, 1986). In the fetal sheep, nonlabor uterine contractions cause transient changes in CM that may reflect fetal hypoxemia (Robertson et al., 1996). Experiments with the fetal rat indicate

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that there is more than one source of CM in the motor system with different preferred frequencies (Robertson & Smotherman, 1990), and data from the fetal sheep (Robertson & Bacher, 1995) and human neonate (Robertson, 1993b) are consistent with this hypothesis. CM in the human persists beyond the neonatal period and beyond the end of what has been called the period of the fetus ex utero (2–3 months after birth) (Robertson, 1993a).

The fluctuations in spontaneous motor activity are not rhythmic or clocklike. The research on CM provides compelling evidence that irregularity is a robust property of the fluctuations under a range of physiological and environmental conditions, both normal and pathological. It is common to associate regularity with lawfulness and then to search for the underlying mechanism responsible for the regularity. Likewise, it is common to associate irregularity with unexplained variation or measurement error and then to try to isolate or reduce it. There is another possibility. The irregularity of the fluctuations in spontaneous motor activity may be an intrinsic property of the dynamics of the underlying mechanism. If so, then we must consider the functional implications of behavioral organization with built-in irregularity.

The field of dynamics, which focuses explicitly on how a system's behavior changes in time, provides a theoretical framework and set of methods with which to address this question. In particular, relatively simple systems with few degrees of freedom governed by chaotic dynamics can exhibit highly irregular behavior. The irregular fluctuations exhibited by such systems result from nonlinear interactions among the variables that define the system's state as it evolves over time, not from perturbations from outside the system.

Our first goal, therefore, was to determine whether the irregular fluctuations characteristic of CM in awake human infants might reflect the output of a dynamical system with relatively few degrees of freedom. To do this, we used techniques developed in experimental dynamics to analyze the fluctuations in spontaneous motor activity for evidence of low-dimensional structure. It is not feasible to search for high-dimensional structure because it would require much longer time series than can typically be obtained from awake infants.

Our second goal was to determine whether the irregularity in the movement time series might be an intrinsic property of the mechanism responsible for the structure. To do this, we analyzed the fluctuations in spontaneous motor activity for evidence that initially similar states of the underlying mechanism become dissimilar at exponential rates. The exponential divergence of similar states is a defining property of chaotic systems, which accounts for their irregular behavior (Fraser & Swinney, 1986; Guckenheimer, 1982).

Not only does dynamics provide a way to address fundamental questions about CM, but CM in turn presents an opportunity to test the utility of dynamics for the study of behavior. Dynamical concepts provide a rich and coherent set of metaphors that could possibly lead to deeper insights into the organization of behavior in both real and developmental time (Smith & Thelen, 1993). To know whether this promise will be fulfilled, we must bring the theory into direct contact with empirical facts. Progress in this effort is impeded, in part, by the need for large amounts of high-resolution data with which to carry out some of the basic calculations. Spontaneous motor activity, recorded over many minutes, provides the kind of data needed to evaluate the utility of the theory and methods of dynamics.

Method

Participants

We studied infants at 1 and 3 months after birth because of the substantial evidence for important changes in the neural and behavioral organization of awakeness during that period (e.g., Wolff, 1984). Eleven infants (6 male, 5 female) provided sufficient amounts of data at 1 month (26-31 days) after birth, and 13 infants (6 male, 7 female) provided sufficient amounts of data at 3 months (82-89 days). All infants were healthy with no known motor or sensory problems. Birth weights were between 2.74 and 4.57 kg ($M \pm SEM = 3.68 \pm 0.15$ kg at 1 month, 3.47 ± 0.11 kg at 3 months), t(21) = 1.17, p = .26 (birth weight was not available for 1 infant at 3 months). Gestational ages at birth were between 38 and 42 postmenstrual weeks (39.6 \pm 0.3 at 1 month, 40.4 \pm 0.3 at 3 months), t(20) = 1.59, p = .13 (gestational age was not available for 2 infants at 3 months). Because the planned analyses require a large number of repeated measurements with no interruptions (Abarbanel, Brown, Sidorowich, & Tsimring, 1993), we used an infant's movement data only if the length of the longest artifact-free period exceeded 4,000 points (approximately 7 min; see Data Acquisition and Reduction section). Insufficient data were obtained from 34 infants at 1 month and 8 infants at 3 months. An additional 21 infants at 1 month and 4 infants at 3 months provided no usable data because they were fussy or sleepy at the time of study. The infants who provided insufficient data or no usable data did not differ from those who did provide usable data on sex, 49% female at 1 month, $\chi^2(1, N = 66) = 0.05, p = .84, 50\%$ female at 3 months, $\chi^2(1, N = 25) = 0.04$, p = .85; birth weight, 3.58 \pm 0.06 kg at 1 month, t(64) = 0.67, p = .51, 3.69 ± 0.13 kg at 3 months, t(20) = 1.31, p = .51.21; or gestational age at birth, 40.0 ± 0.1 postmenstrual weeks at 1 month, t(64) = 1.24, p = .22, 39.6 \pm 0.3 postmenstrual weeks at 3 months, t(19) = 1.39, p = .18. Birth weight and gestational age were not available for 2 infants who provided insufficient data or no usable data.

The study's design and procedures were approved by the University Committee on Human Subjects. Parents volunteered to participate in the study by responding to a letter given to all parents giving birth at the local hospital. Informed, written consent was obtained from all parents before data collection began.

Procedure

Infants were studied at a time of day that the parents indicated would be optimal for obtaining a maximum period of nonfussy awakeness. Sixteen

infants were studied between 8 a.m. and noon, and 4 at each age were studied between noon and 4 p.m.

The laboratory was acoustically insulated, and white noise at a constant 50-dB (A) was used to mask equipment and other sounds that might trigger motor activation or quieting. Infants sat lightly clothed in an infant car seat approximately 120 cm from a black felt curtain in front of which was mounted an interesting but static array of visual stimuli. Ambient light level was approximately 300 lux. Data acquisition continued uninterrupted until the infants became fussy or sleepy, as judged by the parents.

Data Acquisition and Reduction

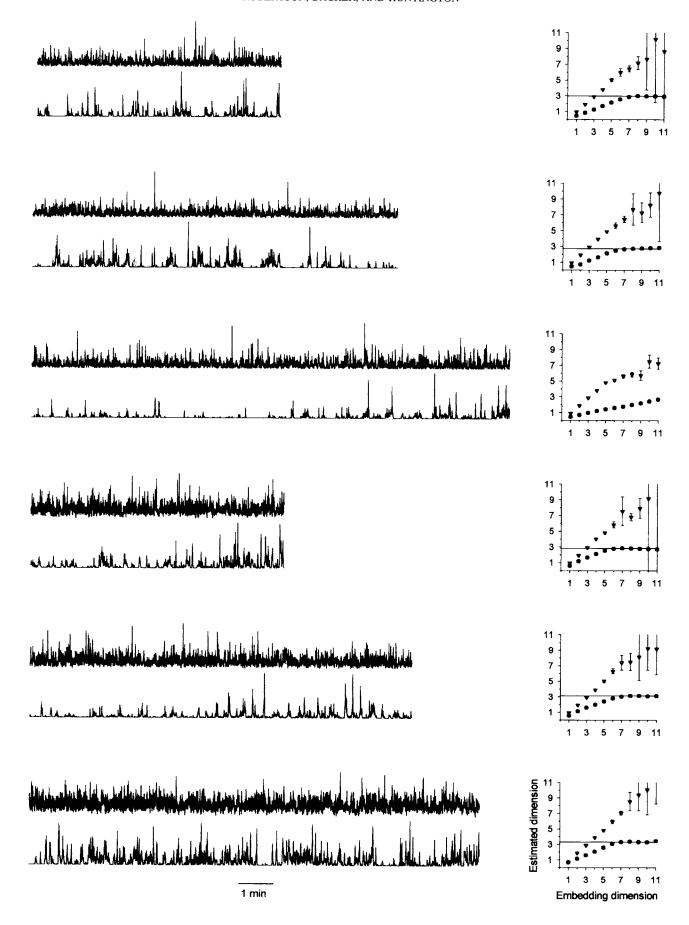
Motor activity was detected by custom-fabricated piezoelectric sensors mounted in the back and bottom of the infant seat. Sensor output was amplified (Coulbourn S75-01; Coulbourn Instruments, Allentown, PA), band-pass filtered (1-40 Hz, Coulbourn S75-34), and then digitized at 30 Hz (DT2801A; Data Translation, Marlboro, MA, or AT-MIO-16H9; National Instruments, Austin, TX). Breathing was detected by a thin elastic gauge (Plethysmograph 271; Parks, Beaverton, OR) around the abdomen. The breathing signal was used to determine thresholds for each infant that discriminated background sensor activity (mainly chest wall and abdominal movements associated with breathing) from general motor activity. Suprathreshold sensor activity was smoothed with a 1-s tapered moving window (current and past samples had relative weights $w(j) = \frac{1}{2}(1 +$ $\cos(\pi i/31)$), $i = 0, 1, \dots, 30$ to focus subsequent analyses on the secondby-second behavioral fluctuations of interest (see Figure 1) and decimated to 10 Hz to reduce the otherwise prohibitively long computation time required for the analyses.

Data Analysis

The biological variables that define the state of the system responsible for CM at any point in time (its state variables) are unknown. Therefore, the relations among these variables that describe how spontaneous motor activity changes in time (CM dynamics) cannot be studied directly (Robertson, Cohen, & Mayer-Kress, 1993). However, techniques have been devised that allow some important properties of an unknown dynamical system, such as its dimension and the rate at which similar states diverge over time, to be estimated from a series of repeated measurements made on a single output variable (not necessarily a state variable) of the system (Packard, Crutchfield, Farmer, & Shaw, 1980; Sauer, Yorke, & Casdagli, 1991; Takens, 1981). The methods take advantage of the general fact that the patterns of fluctuations in the output of a system may contain useful information about the system's underlying dynamics.

We used these methods to analyze each infant's movement time series to estimate the inherent structure (dimension) and irregularity (divergence of similar states) of CM. The first step was to construct an embedding space for each movement time series (a space in which the coordinate axes are measurements of motor activity separated by multiples of a short time delay, t). The dimension of the embedding space is the number of delayed movement points that are used to construct its coordinate axes. Each point in the embedding space thus corresponds to a sequence of points in the movement time series. In particular, the specific location of each point in the embedding space, $y(i) = [x(i), x(i + t), x(i + 2t), \dots, x(i + mt)]$, represents a specific pattern of fluctuation in the corresponding sequence of m + 1 movement points. Nearby points in the embedding space represent similar patterns of movement fluctuation occurring at different times. A trajectory of points in the embedding space, therefore, represents the evolution of the patterns of movement fluctuation over time.

An optimal time delay for constructing the embedding space maximizes the independence of the coordinate axes, which, in turn, facilitates the analysis of the trajectories that describe the evolution of patterns of movement fluctuation. We used the time delay at which the mutual information between the movement time series and itself (a nonlinear analogue of autocorrelation) reached its first minimum, which tends to



maximize the independence of the coordinate axes (Fraser & Swinney, 1986).

Dimension. We calculated the correlation dimension to estimate the effective number of degrees of freedom in the mechanism responsible for CM (Robertson et al., 1993). The correlation dimension is a commonly used measure that takes into account the tendency for certain states of a system to be visited more frequently than others (Farmer, Ott, & Yorke, 1983). For each infant's movement time series, embedding spaces of successively higher dimension were constructed using Takens's method as previously described. At each step, the correlation dimension of CM was estimated using Grassberger and Procaccia's (1983) method, as implemented by Schaffer, Truty, and Fulmer (1990). The dimension of the embedding space was increased and the calculations were repeated until the estimates of the correlation dimension of CM reached a plateau. A plateau occurs when the movement trajectories in the embedding space have been untangled sufficiently so that a higher dimensional embedding space reveals no new structure. The criterion we used to define a plateau was three or more successive estimates of the correlation dimension (based on calculations done in successively higher embedding dimensions) for which the minimum and maximum values differed by less than 0.2 and for which each 95% confidence interval was less than 10% of the corresponding estimate. The average of the estimates meeting this criterion was used as the overall estimate of the correlation dimension.

To determine whether dimension estimates calculated in this way might be artifacts of the smoothing or decimation procedures carried out on the time series, the same analysis was done on a shuffled version of each movement time series. In the shuffled version, the complete sequence of measurements of motor activity for an infant was reordered in a random fashion. Thus, the same data points were analyzed, but the original temporal relations among them were destroyed. The subsequent steps in processing the data (smoothing and decimation) were the same as for the unshuffled data.

Divergence. We calculated the divergence of trajectories in the embedding space to estimate the rate at which similar states of the mechanism responsible for CM became dissimilar. The divergence of trajectories is quantified by Lyapunov exponents (Gershenfeld, 1988). The largest Lyapunov exponent, L_1 , can be defined as the rate at which a small volume of points in the embedding space expands in the direction of fastest expansion (Eckmann & Ruelle, 1985; Wolf, Swift, Swinney, & Vastano, 1985). It is common to take a positive value of L_1 as an indication that the dynamics of the underlying mechanism might be chaotic (Farmer et al., 1983).

If the calculations done to estimate the correlation dimension for a given movement time series provided evidence of low-dimensional structure, we proceeded to estimate L_1 using Wolf's algorithm (Wolf et al., 1985; Wolf & Vastano, 1986) as implemented by Schaffer et al. (1990). Wolf's algorithm takes the nearest neighbor to the initial point in the embedding space and calculates the increase in separation between their subsequent trajectories during a fixed evolution time. This process is repeated until the end of the data set, and the average of the values of $\log_2 D'/D$, where D' and D are the final and initial separations of the neighboring points in each step, is used to estimate L_1 . Because short evolution times overestimate L_1 and long evolution times underestimate L_1 , we used an intermediate evolution time equal to twice the average interval at which the first minimum in mutual information occurred (18.5 s).

Because Wolf's method follows the evolution of neighboring points in the embedding space, it is important to reduce the chance that apparently near neighbors are actually false neighbors. False neighbors exist when the embedding dimension is too small. In such cases, increasing the embedding dimension reveals that the apparent neighbors are not close; rather, it is their projections in the lower dimension embedding space that are close. That is, the patterns of movement fluctuation represented by false neighbors in the embedding space appear to be similar only because the sequence of points in the movement time series used to construct the embedding space was too short. Therefore, we calculated the percentage of false near neighbors at each embedding dimension (Abarbanel et al., 1993). The embedding dimension at which the false near neighbor rate first fell below 5% was used for estimating L_1 .

As with the analyses done to estimate the correlation dimension, the calculations carried out to estimate L_1 were repeated with the shuffled data.

Results

The 24 movement time series ranged in length from 7.1 to 14.0 min $(9.9 \pm 0.7 \text{ min})$ at 1 month and 7.4 to 14.7 min $(10.9 \pm 0.7 \text{ min})$ at 3 months, t(22) = 1.05, p = .31. The corresponding numbers of data points in each time series (after decimation) ranged from 4,158 to 8,379 $(5,916 \pm 402)$ at 1 month and 4,428 to 8,844 $(6,539 \pm 428)$ at 3 months. The first minimum in the mutual information between each movement time series and itself occurred at a longer interval at 1 month $(10.7 \pm 1.3 \text{ s})$ than at 3 months $(8.0 \pm 0.6 \text{ s})$, t(22) = 2.06, p = .05. The embedding dimensions at which the false near neighbor rate first fell below 5% at 1 month (7.7 ± 0.3) and 3 months (7.2 ± 0.1) did not differ, t(20) = 1.56, p = .14. Representative time series and their shuffled versions are shown in Figure 1.

Dimension

Estimates of the correlation dimension reached a plateau for 9 of 11 infants at 1 month and 13 of 13 infants at 3 months. The plateau criterion was met at a higher embedding dimension at 1 month (7.8 ± 0.3) than at 3 months (6.6 ± 0.3) , t(20) = 2.94, p = .008, but the estimated correlation dimension at 1 month (3.0 ± 0.1) and 3 months (3.1 ± 0.1) did not differ, t(20) = 0.70, p = .49. The Type II error rate was less than .05 for detecting a 15% difference in the mean correlation dimension at 1 and 3 months. In no case did correlation dimension estimates based on the shuffled data reach a plateau. Figure 1 shows these results for 3 representative infants at each age.

Divergence

Estimates of the rate of divergence of nearby trajectories were positive (95% confidence intervals did not include zero) for 7 of 9 infants at 1 month and 13 of 13 infants at 3 months. Two of the 11 infants at 1 month were excluded from these analyses because the dimension analysis described previously provided no evidence for low-dimensional structure in their movement time series. The divergence estimates exceeded those based on the shuffled data at both 1 month $(3.7 \pm 0.2 \times 10^{-2} \text{ vs. } 2.3 \pm 0.4 \times 10^{-2} \text{ bits/s})$,

Figure 1 (opposite). Representative data from 3 infants at 1 month (top three panels) and 3 infants at 3 months (bottom three panels) after birth. Left panel: Movement time series before (lower) and after (upper) shuffling the order of the points. Both shuffled and unshuffled time series have been smoothed with a 1-s tapered window (see Method section), decimated to 10 samples/s, and rescaled to the interval [0, 1]. Right panel: Correlation dimension estimates at successive embedding dimensions for the unshuffled (circles) and shuffled (triangles) movement time series. Error bars represent 95% confidence intervals. Horizontal lines indicate the overall estimate based on the individual estimates meeting the stability criterion (see Method section).

t(6) = 6.43, p < .001, and 3 months $(3.9 \pm 0.2 \times 10^{-2} \text{ vs.} 2.8 \pm 0.2 \times 10^{-2} \text{ bits/s})$, t(12) = 4.14, p = .001. The rate of divergence at 1 and 3 months did not differ, t(18) = 0.60, p = .56. The Type II error rate was less than .05 for detecting a 25% difference in the mean rate of divergence at 1 and 3 months.

Discussion

The results provide evidence for two important inferences about spontaneous motor activity in awake human infants at 1 and 3 months after birth. First, the irregular fluctuations in movement reflect an underlying neuromotor mechanism operating with relatively few degrees of freedom. Second, the tendency for similar patterns of motor activation (occurring at different times) to rapidly become dissimilar cannot be explained by random perturbations of the underlying neuromotor mechanism. Taken together, these results support the conclusion that the characteristic irregularity of the fluctuations in spontaneous motor activity may be an intrinsic property of the same mechanism that is responsible for their organization.

There was no evidence that the substantial changes in the nature of awakeness that are known to occur between 1 and 3 months are relevant to this conclusion. Neither estimates of the correlation dimension nor estimates of the rate of divergence differed between 1 and 3 months, although the analyses had acceptable statistical power to detect moderate differences if they existed. However, the finding that the mutual information function calculated from infants' movement time series took slightly longer to reach its first minimum at 1 month than at 3 months raises the possibility that one or more properties of the fluctuations in spontaneous motor activity that are not tapped by the dimension and divergence measures do change between 1 and 3 months. This study does not provide any information about the extent to which dimension and divergence might depend on infant temperament, stimulus parameters, or other unmeasured variables.

The main results have implications for subsequent efforts to understand the mechanism responsible for CM. First, low-dimensional structure indicates that the potentially large number of degrees of freedom in the motor system are reduced to just a few during CM. The reduction in degrees of freedom is likely to be the result of both the coupling among the neural elements generating CM and the biomechanical constraints on movement imposed by the infant's body mass and structure. From a theoretical perspective, dimension estimates place a lower bound on the number of variables needed to model the dynamics of a system (Farmer et al., 1983). In the case of CM, low-dimension estimates leave open the possibility that a tractable dynamical model might be constructed in which the key variables have biological meaning. Detailed investigation of such a model might then yield further insights into the organization of spontaneous motor activity.

Second, the divergence results indicate that the characteristic irregularity of CM cannot be attributed simply to random perturbations of the system that generates the fluctuations in motor activity. Rather, at least some of the irregularity appears to be intrinsic to CM dynamics. Evidence from the fetal rat indicates that the mechanism responsible for CM is distributed rather than localized in the neuromotor system (Robertson & Smotherman, 1990). A distributed mechanism can also explain results obtained from human infants in which CM was disrupted by sudden auditory stimulation (Robertson, 1993b). This raises the possibility that

the intrinsic irregularity of CM may arise, in part, from interactions among the distributed sources of activity.

Because CM is ubiquitous in the human fetus and infant (Robertson, 1990, 1993a), exists in quantitatively similar form in the rat fetus and neonate (Bacher, Robertson, & Smotherman, 2000; Smotherman, Robinson, & Robertson, 1988) and in the sheep fetus (Robertson & Bacher, 1995; Robertson et al., 1996), and appears to exist in at least qualitatively similar form in a variety of other vertebrates (Corner, 1977), it is possible that understanding the mechanism of CM will yield some general insights into how complex variability is produced in immature behavioral systems. Specifically, low-dimensional dynamics with intrinsic irregularity may be a common basis for the persistent but ever-changing baseline of motor activity from which coordinated, adaptive behavior arises during development.

The conclusion that at least some of the irregularity of CM is intrinsic also has implications for how we think about the functional significance, if any, of the fluctuations in spontaneous motor activity. To the extent that CM is a robust property of neurobehavioral organization from the earliest stages of development, it is likely to be an important factor in interactions with the environment. Indeed, evidence indicates that responsiveness to a biologically significant stimulus is modulated by CM in the fetal and neonatal rat (Bacher et al., 2000; MacLennan, Smotherman, & Robertson, 1998; Reilly, Robertson, MacLennan, & Smotherman, 1997). In the human infant, recent results suggest that fluctuations in spontaneous motor activity may regulate visual attention. Specifically, the disengagement of overt attention appears to be coupled to the rates of change in motor activity (Robertson, Bacher, & Huntington, in press).

What might be the costs or benefits of irregularity built into the mechanism controlling spontaneous motor activity? The potential costs seem clear. Given the coupling between motor activity and attention in young infants, irregular fluctuations in movement might result in relatively unpredictable disruption of the perceptual and cognitive processes that depend on attention. Alternatively, there is evidence that randomness can have advantages in search behavior (Hoffmann, 1983; Farnsworth & Beecham, 1999). Some have speculated about the possible benefits of chaotic dynamics in this regard (Mobus & Fisher, 1999). The present results do not indicate whether built-in irregularity is good or bad for infants, but they do raise the possibility that attentional shifts that are driven in part by the irregular fluctuations of CM might increase the efficiency with which infants explore the visual environment in the first few months after birth.

There has been interest among some developmental scientists in the possibility that dynamical systems theory might provide a coherent set of concepts and methods that could be useful in understanding the nature of behavior and development. One way to evaluate the utility of dynamics in these domains is to bring theory into contact with data from a specific behavioral or developmental system and discover whether it leads to any new insights into the nature of the system. We have attempted to do this with CM. The existence of low-dimensional structure in the irregular fluctuations of spontaneous motor activity was not discovered using more traditional methods of analysis. Neither was the fact that at least some of the irregularity in the fluctuations of movement is intrinsic to the dynamics of motor activation and not simply the result of random perturbations. This is not to say that these conclusions about CM could not have been reached by other means. However,

by the criterion of new insights, the theory and methods developed to understand dynamical systems in other fields have been useful in the study of CM. In any case, other behavioral and developmental systems as well as other criteria of usefulness (e.g., Newell & Molenaar, 1998; Smith & Thelen, 1993; Thelen, Schoner, Scheier, & Smith, in press; van der Maas, 1995; van Gelder, 1998) must be examined to judge the general utility of the theory and methods in our field.

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