PREDICTION OF NEONATAL STATE AND MATURATIONAL CHANGE USING DIMENSIONAL ANALYSIS

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ABSTRACT

PURPOSE: Nonlinear time series analysis techniques have been used to analyze physiological signals such as EEG and heart rate. In this paper we illustrate the application of dimensional analysis (DA) to assess neonatal sleep states at increasing gestational ages up to fullterm age.

METHODS: One hundred and sixteen EEG-polygraphic recordings were performed on 55 neonatal subjects between 28 and 43 weeks gestational age from which state assignments were initially scored by visual analysis. A single channel of EEG (ie. FP1-C3) was selected for dimensional analysis. Two tailed T-tests were used to test for differences in the correlation dimension (CD) between active and quiet sleep states for both preterm and fullterm neonates as a function of maturation.

RESULTS: 1) A significant difference in CD between active and quiet sleep states (p < 0.001) was noted for the fullterm infant. 2) A positive correlation between CD and increasing conceptional age was noted (p < .001). 3) DA showed an increase in the complexity for both active and quiet sleep as the preterm infant matured toward a fullterm corrected age. 4) Lower dimensionality (CD), indicative of reduced complexity, was noted for the healthy preterm cohort at corrected fullterm age when compared to the fullterm group.
CONCLUSIONS: Dimensional analysis demonstrated a positive correlation for both active and quiet sleep, as the infant matured towards corrected term age. Lower dimensionality was noted for the healthy preterm cohort at corrected fullterm age. Our findings support the concept of physiologic dysmaturity for the preterm neonate as a reflection of altered neuroplasticity of the brain as a result of the conditions of prematurity.

Keywords: neonate, EEG, sleep, nonlinear, maturation, dimensional analysis
INTRODUCTION

Traditional linear methods of signal analysis rely primarily on the frequency characteristics of the signal, often neglecting the temporal characteristics that are also encoded in the signal. Nonlinear analysis methods can make use of this additional information by interpreting physiologic behaviors as a measurement of the state of some dynamical system. Using such measurements, the time evolution or dynamics of the EEG system can be reconstructed into a multidimensional phase space. This reconstruction can then be used to reveal dynamical properties of the system that may not be apparent from classical frequency analysis.

Dimensional analysis (DA) was selected to measure the complexity of a biologic system (i.e. EEG) based on the geometrical properties of the system’s evolution in phase space (Grassberger, 1983a, b). DA has been used to study a variety of neurological states including epilepsy, Pijn et al., 1997; Pritchard et al., 1995; Lehnert et al., 2001; Babloyantz et al., 1986; Osorio et al., 2002; Lai et al., 2002; Ferri et al., 2001; Aschenbrenner-Scheibe et al., 2003) and sleep in adults (Babloyantz et al., 1985; Ackermann et al., 1994; Fel et al., 1996; Pradhan et al., 1996; Szelenberger et al., 1996; Ferri et al., 1996; Ferri et al., 2002; Pereda et al., 1998; Pereda et al., 1999; Kobayashi et al., 1999; Kobayashi et al., 2001; Shen et al., 2003) and older children (Ferri et al., 1998, Ferri et al., 2001). Our current research applied DA, using the correlation dimension, to EEG signals during neonatal sleep at increasing gestational ages up to fullterm ages.
METHODS

Patient Selection

The clinical and demographic data for 55 neonates included 29 female and 26 male infants. Institutional Review Board approved informed consents were obtained for all study subjects. Thirty-three infants, ≤ 32 weeks gestational age with a mean birthweight of 1191 gms (range: 788-1670 gms) were recruited from a neonatal population who were admitted to the Neonatal Intensive Care Unit of Magee-Women’s Hospital, Pittsburgh PA. The selection was based on the review of maternal and neonatal medical records, combined with consultation with the attending neonatologist. All infants remained clinically asymptomatic throughout the study period. None were treated for major organ system illness (i.e. respiratory distress syndrome, sepsis, intracranial hemorrhage). No infant was medically ill during the study period including encephalopathy, seizures or systemic medical illnesses. Normal cranial ultrasounds were described for all of the preterm subjects.

Twenty-four appropriate for gestational age fullterm infants (mean birth weight 3556 gms) (2353-4800gms) were selected from eight well-child nurseries. Careful review of the medical records as well as physical examinations were carried out to verify the healthy status of these fullterm infants. Infants in both cohorts were clinically assessed for at least two years of age and were neurodevelopmentally normal either by examination or parental report.
EEG-Sleep Recordings

Electroencephalographic/polysomnographic studies were carried out in an environmentally controlled setting in which sound, light, humidity and tactile stimulation were monitored. All infants were studied while sleeping prone or on their sides in an open bed, whichever was their usual sleeping position in the nursery. Continuous recordings began after a diaper change and feedings, at approximately 9:00 p.m., ending at approximately 9:00 a.m. the following morning. The entire 24-channel recording was digitized on a Hewlett Packard workstation, (Palo Alto, California), with the first three hours of the study simultaneously recorded on paper using a 21-channel electroencephalographic machine, (Nihon Kohden, Model 4221).

One hundred and sixteen EEG polygraphic recordings were performed. One study was performed on the fullterm infant while serial studies were performed on the preterm infant at approximately monthly intervals up to corrected term ages.

Digitized neurophysiologic data for each minute of sleep during the recording were compared with the contemporaneous minute of EEG sleep, and were visually assigned one of six sleep states according to conventional neonatal EEG-sleep criteria. (Pope, et al., 1992) for the fullterm infant (i.e. two active and two quiet sleep states as well as indeterminate and waking states). Minutes for the preterm infant sleep study were scored
either as continuous or discontinuous EEG, with the degree of discontinuity scored as
records of quiescence during each minute.

A neonatal research nurse provided clinical care for each infant during the recording
session. Sleep, feeding, behavior, diaper changes, medication administration and
technical comments (i.e. equipment malfunctions and environmental measures etc.) were
documented in the computer database. No infants were given medications during the
studies and no infants received circumcisions prior to a sleep study.

Analytic Methodologies

Dimensional analysis was selected to measure the complexity of a single channel of EEG
(i.e. FP1 to C3). The correlation dimension (D2) was selected as the DA measure with
the computational method based on the correlation integral as proposed by Grassberger
and Procaccia (1983a).

Correlation dimension is one of a family of dimensions that is commonly selected
because of its robustness to experimental conditions, and its modest computational
requirements. To compute the correlation dimension, we first computed the correlation
integral proposed by Grassberger and Procaccia (1983b):

\[
C(r) = \lim_{N \to \infty} \frac{1}{N^2} \sum_{i=1, j=1}^{N} \Theta(r - |x_i - x_j|)
\]

where \(\Theta(y)\) is the Heaviside function, that is the function that is 0 for \(y<0\) and 1 for \(y>0\).
Basically, the correlation integral sums the number of inter-point distances that are less
than a given radius $r$ and gives an estimate of the probability that the distance between a
given pair of points on the attractor is no more than $r$. For small $r$, the correlation integral
scales as a power of $r$, that is:

$$C(r) \approx r^{D_2}$$

Thus, to compute the correlation dimension ($D_2$) of the attractor, the correlation integral
is computed for several small values of $r$, and then the slope of the linear region, or
scaling range, of the $\log(C(r))/\log(r)$ curve is computed. For computational efficiency,
we have used an implementation of the Grassberger-Procaccia algorithm described in
Parker and Chua (1989) that takes advantage of the floating point representation of the
data used in the computation.

To compute the correlation dimension of a time series of EEG data, we first reconstruct
the attractor corresponding to the EEG in an appropriate phase space. While the attractor
may be contained in a small-dimensional subset of the phase space, the dynamical system
that is generating the measured time series may require many more variables for a full
description and in practice it is seldom possible to measure all relevant dynamical
variables. Fortunately, in typical situations, the measurement of a limited number of
variables is sufficient to ensure that the (topological) properties of the attractor are
preserved in a manifold that is constructed from the measurements. According to a result
from Takens (1981), by interpreting a single channel of EEG data as the projection of a
d-dimensional attractor onto a single dimension, we may reconstruct an “equivalent”
(isomorphic) copy of the attractor in an $m$-dimensional phase space ($m > 2 \cdot d$), from
sampled time series measurements of the signal $x(t)$ by the following time delay
reconstruction:

$$y(t) = (x(t), x(t-\tau), \ldots, x(t-(m-1)\tau))$$

where $\tau$ is a fixed time delay parameter, and $m$ is known as the embedding dimension of
the reconstruction. Each point $y(t)$ in the phase space corresponds to a unique point on
the system attractor.

While Takens’ theorem states that an attractor may be constructed with theoretically any
time delay or suitably large embedding dimension, these results apply only when an
infinite amount of noise-free infinite precision data is available. In practice, it has been
noted that the values of both the time delay and embedding dimension parameters used in
reconstructing the attractor can have a significant effect on the quality of the correlation
integral curves and their usefulness in computing the correlation dimension. Several
prescriptions exist to guide the selection of both parameters. In terms of the time window
($\tau_w = \tau \cdot (m-1)$) which spans between the first and last coordinates of each point of the
reconstructed time series. Once an appropriate time window is chosen, the optimal
selection of the time delay ($\tau$) and the embedding dimension $m$ are intertwined. The time
window, as opposed to the time delay parameter, has the most significant effect on the
computation of the correlation integral curves, and thus the correlation dimension.

Assuming the reconstruction requirements are similar for all of the EEG data, the effect
of the time window for a sample of epochs was analyzed visually, and a typical optimal
time window size of $\tau_w = 100 \pm 20$ was found. Computations for varying embedding dimensions were completed while keeping the time delay fixed. Preliminary examination of these computations indicated that fixing the time delay at $\tau = 12$ while computing the correlation integral curves for embedding dimensions 4 through 14 gave reliable results for all of the data used for the computations.

Correlation integral curves were then analyzed to estimate the correlation dimension. Firstly, each curve corresponding to a single embedding dimension was analyzed individually to identify the linear region. This was accomplished by computing the point-wise slope of each curve using a Lagrangian approximation of the derivative. From each element of the point-wise slopes, a relatively flat region was determined within which the slopes deviated from the reference slope by less than 15%. The longest such region was selected as the plateau of the slope curve and the corresponding elements of the correlation integral curve were selected as the linear region. With the linear region thus identified, linear regression was then used to compute the slope of this line, which is equal to the correlation dimension of the associated curve. If there were no plateaus spanning at least 3 points on the log – log plot) or if the regressed line had a large residual (i.e. $> 2$), then this individual correlation integral curve did not converge to a linear region.

Once the individual slopes were computed for each correlation integral curve, a similar plateau finding methods was used to determine the convergence of the individual curves a single embedding dimension for the epoch. The computed correlation dimension can
be no larger than the embedding dimension for a given attractor, computations with embedding dimensions that are too small do not reveal the correlation dimension of the attractor. For embedding dimensions that approach the true embedding dimension of the attractor, the computed correlation dimension is a good estimate of the dimension of the attractor and should remain approximately constant as the embedding dimension increases. To determine a range of embedding dimensions that converge to a single overall correlation dimension for the attractor, all embedding dimensions within one standard deviation of the median of all dimensions was selected. The overall correlation dimension of the time series was then taken to be the mean of the estimates from this range. If the standard deviation of the range of all individual estimates was greater than one, the epoch was rejected for poor convergence over the range of embedding dimensions.

Two tailed T-tests were then used to test for differences in the dimensional analysis or correlation dimension between active and quiet sleep for both the preterm and fullterm cohorts, and as a function of increasing conceptional age to the corrected fullterm age. The null hypothesis was that the means for one group were not equal to the means in the other groups ($p < 0.5$). Based on the above-mentioned methods that were used to compute the correlation dimension, Table 1 lists the rejection results based on the convergence rejection criteria described previously. Approximately 12% of the epochs were rejected for failure to converge, either because too few of the individual correlations integral curves had sufficiently long linear regions or because the individual dimensions did not appear to converge as the embedding dimensions increased. For the premature
discontinuous state (i.e. prototypical quiet sleep) the rejection rate was 20.6%, attributable to the pronounced nonstationarity within the epochs as a result of the discontinuous bursting activity. (See Table 1)

Results:

1. Correlation Dimension Compared to Sleep State

Distributions of the estimates of correlation dimension within each Age/State grouping were approximately normal. Descriptive statistics for each group are given in Table 2. Table 3 lists the results of the statistical comparisons between these two groups of patients. For the premature infant cohort, there was no significant difference in CD between active and quiet sleep (p = 0.223). For the fullterm infant, there was a significant difference in correlation dimension between active and quiet sleep (p < 0.001). In addition, there was a statistically significant difference in correlation dimension between the preterm and neonatal groups for both active and quiet sleep.

2. Correlation Dimension with increasing conceptional age.

We found a significant positive correlation between dimensionality and increasing conceptional age; (i.e. correlation dimension) significantly increases for active and quiet sleep as the infant matured to a fullterm age, (Table 4). The correlation between the dimension and active/quiet sleep was slightly higher in the active sleep state. There was
no significant difference found in the distributions of these two groupings. The linear least squares fit for these two groups were identical. (See Table 4)

3. Influence of Prematurity on Correlation Dimension

To determine whether prematurity had an effect on dimensionality for patients at fullterm age, epochs of sleep for newborns between 38 and 42 weeks conceptional age were divided into two groups depending on their age at birth. Approximately half of the studies were derived from infants who were born prematurely (i.e. before 35 weeks gestational age), while for the remainder they were born after 38 weeks gestation. Table 5 lists the descriptive statistics and T-test results for active and quiet sleep segments for these two populations. A statistically significant difference in dimensionality was noted between the two groups; dimensionality for the prematurely born infant (i.e. the child born less than 35 weeks gestational age) was smaller than those of the infants born at term. (See Table 5)

Since differences in dimensions based on age at birth were greater than the differences in dimensionality revealed from the active and quiet sleep analysis, we reanalyzed the differences in dimension based on state, considering each of the birth age cohorts separately. The results of this analysis given in Table 6, indicates that the dimensions for active and quiet sleep remained significantly different for the two cohorts individually, with differences remaining similar when the two populations were assessed together. (See Table 6)
Discussion

Dimensional analysis demonstrated a positive correlation for active and quiet sleep, as the infant matured to corrected term ages. Lower dimensionality (i.e. less complexity in the EEG signal) was noted for the healthy preterm cohort at a corrected fullterm age. These findings support the concept of physiologic dysmaturity as previously discussed (Scher, et al., 2003). Physiologic dysmaturity is an expression of neuroplasticity for the healthy preterm cohort when compared to a fullterm group. The concept of dysmaturity has been reported for different high-risk neonatal groups. Dysmaturity in EEG patterns (Holmes et al., 1979), for preterm infants with respiratory distress syndrome consisted of transient electrographic immaturity that resolved after resolution of their pulmonary illness (Tharp et al., 1989). Hahn et al., (1990) presented results of visually assessed EEG-records at fullterm for neonates with chronic lung disease, who expressed dysmature electrographic patterns, predicting compromised neurodevelopmental outcome at 3 years of age. Dysmature sleep patterns, defined as altered arousal numbers and sleep architecture were noted in premature neonates with prenatal substance exposure and chronic lung disease (Scher et al., 1988; Scher et al., 1992a). Children at risk for SIDS have also been described as expressing immaturity regarding sleep organization and arousal (Harper et al., 1983) using digitized spectral EEG signals. Scher et al., (1997) also described physiologic dysmaturity in terms of spectral EEG/sleep measures in an asymptomatic preterm neonatal population, known to be at high risk for SIDS compared to a fullterm group.
Environmental and biologic conditions may either accelerate or delay brain maturation, depending on which specific neuronal circuitry is affected. The infant’s altered functional expression of any brain activity reflects an adaptation to stress, to maintain homeostasis for survival (Oppenheim, 1981). This general biologic adaptive process has been more recently termed activity-dependent development, and underscores the complexities of remodeling of neuronal circuitry expressed as the developmental processes of dendritic arborization, synaptogenesis and apoptosis (Hughes et al., 1999). Earlier adjustments in expected remodeling, however, may prove maladaptive at later developmental stages (Oppenheim, 1981). An asymptomatic fullterm cohort for example, who exhibited accelerated sleep behaviors at birth may be at high risk for developmental delay during the first years of life (Freudigman et al., 1993).

Neurophysiological adaptation of preterm infants who express altered sleep behaviors compared to fullterm infants are associated with lower developmental scores during infancy and early childhood (Whitney et al., 1993; Scher et al., 1996).

Motivation for the use of quantitative analysis of EEG ultimately is to reveal signal attributes that are not apparent from visual inspection. Traditional linear methods of signal analysis rely primarily on the frequency characteristics of the signal, often neglecting time-related characteristics that are also encoded in the signal. More recently there has been significant interest in applying methods from nonlinear systems theory to analyze EEG signals. Nonlinear analysis makes use of this additional information by viewing signals such as the EEG as the measurement of the state for some dynamical system. Using this single state measurement, the dynamics or time evolution of an EEG
system can be reconstructed in a multi-dimensional phase space. This reconstruction can then be used to reveal dynamical properties of the system not apparent from classical frequency analysis.

Dimensional analysis has been used to study a variety of neurologic states including epilepsy and sleep for adults (Babloyantz et al., 1985; Ackermann et al., 1994; Fel et al., 1996; Pradhan et al., 1996; Szelenberger et al., 1996; Ferri et al., 1996; Ferri et al., 2002; Pereda et al., 1998; Pereda et al., 1999; Kobayashi et al., 1999; Kobayashi et al., 2001; Shen et al., 2003; Aschenbrenner-Scheibe et al., 2003) and older children (Ferri et al., 1998, Ferri et al., 2001). Studies involving adult sleep have demonstrated that EEG signals have higher dimensionality during waking and REM sleep than during nonREM sleep. Previous published studies have not explored neonatal sleep and this was the motivation for this particular study. It was therefore provocative to explore the neonatal population, comparing prematurely born and fullterm cohorts to assess if differences in dimensionality exist during earlier stages of brain maturation in a healthy population. Medically at risk groups of neonates may show greater degrees of change. Multiple researchers have described reduced gray and white matter volumes at older ages for different groups of preterm neonates (Huppi et al., 1996; Ajayi-Obe et al., 2000; Peterson, et al., 2000; Issacs et al., 2000; Cooke et al., 1999). Our findings are provocative becasue lower dimensionality at fullterm gestational ages implies that our group of healthy preterm neonates already express altered neuroplasticity in terms of lower EEG complexity, which may result in lower gray and white matter volumes.
Reduced dimensionality for specific groups of neonates may help predict later structural changes in terms of brain volumes, as a result of altered remodeling of neuronal circuitry.

Several shortcomings of the study are recognized. Since preterm infants were studied who were less than 33 weeks gestational age, we did not address sleep behaviors of preterm infants who were closer to a corrected term age. Older gestational ages may express less physiologic dysmaturity on EEG-sleep studies. Secondly, choices for our EEG-sleep measures were based on previous studies using healthy preterm infants. Postnatal medical illness may alter brain function and maturation, affecting the specific spectral measures studied. Thirdly, alternative physiologic measurements of state may more accurately reflect differences in dimensionality between cohorts than those selected for this study. Fourthly, dimensionality may vary as a function of brain regions, given we studied only one brain region for this report.

In conclusion, dimensionality analysis identified physiologic dysmaturity for asymptomatic preterm infants who expressed altered brain function and maturation compared to a fullterm group. Future studies will investigate dimensional analysis for larger neonatal cohorts at successively older corrected ages beyond the newborn period in the context of medical illnesses of varying severity, to detect deviations from expected maturational trends.

ACKNOWLEDGEMENTS
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REFERENCES


Table 1: Rejection Results

<table>
<thead>
<tr>
<th></th>
<th>Total N</th>
<th>N too few with linear regions</th>
<th>%</th>
<th>N with large std dev</th>
<th>%</th>
<th>N rejected</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quiet Premature</td>
<td>107</td>
<td>15</td>
<td>14.0%</td>
<td>7</td>
<td>6.5%</td>
<td>22</td>
<td>20.6%</td>
</tr>
<tr>
<td>Active Premature</td>
<td>156</td>
<td>8</td>
<td>5.1%</td>
<td>2</td>
<td>1.3%</td>
<td>10</td>
<td>6.4%</td>
</tr>
<tr>
<td>Quiet Full term</td>
<td>376</td>
<td>18</td>
<td>4.8%</td>
<td>23</td>
<td>6.1%</td>
<td>41</td>
<td>10.9%</td>
</tr>
<tr>
<td>Active Full term</td>
<td>510</td>
<td>33</td>
<td>6.5%</td>
<td>29</td>
<td>5.7%</td>
<td>62</td>
<td>12.2%</td>
</tr>
<tr>
<td>Totals</td>
<td>1149</td>
<td>74</td>
<td>6.4%</td>
<td>61</td>
<td>5.3%</td>
<td>135</td>
<td>11.7%</td>
</tr>
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</table>

Table 2: Statistics for dimensions of Premature/Full Term, Active/Quiet Sleep

<table>
<thead>
<tr>
<th></th>
<th>mean</th>
<th>min</th>
<th>max</th>
<th>std dev</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature-Quiet</td>
<td>4.364</td>
<td>3.264</td>
<td>5.669</td>
<td>0.482</td>
<td>85</td>
</tr>
<tr>
<td>Premature-Active</td>
<td>4.452</td>
<td>3.705</td>
<td>5.759</td>
<td>0.441</td>
<td>146</td>
</tr>
<tr>
<td>Full Term-Quiet</td>
<td>4.866</td>
<td>3.852</td>
<td>6.31</td>
<td>0.489</td>
<td>335</td>
</tr>
<tr>
<td>Full Term-Active</td>
<td>5.078</td>
<td>3.759</td>
<td>6.649</td>
<td>0.438</td>
<td>448</td>
</tr>
</tbody>
</table>

Table 3: T-test statistics of differences in dimension between Premature/Full Term, Active/Quiet groupings.

<table>
<thead>
<tr>
<th></th>
<th>mean A</th>
<th>mean B</th>
<th>mean A – mean B</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature, Active vs. Quiet</td>
<td>4.452</td>
<td>4.364</td>
<td>-0.088</td>
<td>0.223</td>
</tr>
<tr>
<td>Full Term, Active vs. Quiet</td>
<td>5.078</td>
<td>4.866</td>
<td>0.212</td>
<td>&lt;0.001</td>
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<tr>
<td>Full Term Active, M vs. LVI</td>
<td>5.068</td>
<td>5.098</td>
<td>-0.030</td>
<td>0.503</td>
</tr>
<tr>
<td>Full Term Quiet, HVS vs. TA</td>
<td>4.955</td>
<td>4.861</td>
<td>0.093</td>
<td>0.489</td>
</tr>
<tr>
<td>Active, Full Term vs. Premature</td>
<td>5.078</td>
<td>4.452</td>
<td>0.626</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Quiet, Full Term vs. Premature</td>
<td>4.866</td>
<td>4.364</td>
<td>0.503</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
Table 4: Correlations between conceptional age and dimension in Active/Quiet Sleep

<table>
<thead>
<tr>
<th></th>
<th>Correlation Coefficient ρ</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active – Premature</td>
<td>0.515</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Active – Neonate</td>
<td>-0.079</td>
<td>0.094</td>
</tr>
<tr>
<td>Active – Overall</td>
<td>0.485</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Quiet – Premature</td>
<td>0.465</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Quiet – Neonate</td>
<td>-0.108</td>
<td>0.055</td>
</tr>
<tr>
<td>Quiet – Overall</td>
<td>0.313</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Table 5: Dimensionality for Premature Patients

<table>
<thead>
<tr>
<th></th>
<th>mean born premature</th>
<th>std dev</th>
<th>mean born full term</th>
<th>std dev</th>
<th>diff</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quiet</td>
<td>4.734</td>
<td>0.437</td>
<td>5.037</td>
<td>0.518</td>
<td>0.303</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Active</td>
<td>4.971</td>
<td>0.395</td>
<td>5.194</td>
<td>0.452</td>
<td>0.224</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Table 6: Differences in dimensionality at term age between active/quiet sleep.

<table>
<thead>
<tr>
<th></th>
<th>mean active</th>
<th>std dev</th>
<th>mean quiet</th>
<th>std dev</th>
<th>diff</th>
<th>p value</th>
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</thead>
<tbody>
<tr>
<td>Premature</td>
<td>4.971</td>
<td>0.395</td>
<td>4.734</td>
<td>0.437</td>
<td>0.236</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Full Term</td>
<td>5.194</td>
<td>0.452</td>
<td>5.05</td>
<td>0.498</td>
<td>0.145</td>
<td>0.005</td>
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