Descriptive Analysis of Epileptic Seizures and Problem Behavior in Adults With Developmental Disabilities

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Abstract
We studied possible relations between seizures and problem behavior in 3 adults with developmental disabilities. Each person was observed for between 56 and 92 days to record occurrences of seizures and problem behavior. Results of our descriptive analysis indicated an association between seizures and problem behavior for each participant. For Stan, most problem behavior occurred following absence seizures. For Tom, problem behaviors only occurred before tonic–clonic seizures but showed no relation to absence seizures. For Mick, problem behaviors began before absence seizures, but no consistent relation was established between problem behavior and tonic–clonic seizures. Findings suggest that seizures and problem behaviors can be associated, but these patterns appear to be highly idiosyncratic across individuals.

When the brain is functioning normally, neurons transmit electrochemical signals in a seemingly stochastic pattern among various cortical regions. This neuronal signaling guides the complex behavior of humans, including motivational, cognitive, motoric, autonomic, sensory, and emotional processes. A seizure occurs when the neurons of the cortex fire in an atypical, synchronous pattern, initiating a cascade of biochemical and physiological events that can result in epileptic seizures (Steinlein, 2004). Epilepsy is a medical condition that is diagnosed after the occurrence of repeated seizures. According to current epidemiological studies, at least 1% of the general population in the United States is diagnosed with epilepsy and 10% have experienced a seizure at some point in their lifetime. Prevalence estimates for epilepsy vary in people with developmental disabilities, ranging from 12% to 30% for persons with comorbid disabilities (e.g., cerebral palsy) or genetic syndromes (e.g., fragile X syndrome) (Jansen, Kroll, Groothoff, & Post, 2004; Niedermeyer, 1999).

Persons experiencing seizures commonly report preictal, perictal, or postictal feelings of fear, anxiety, confusion, or déjà vu associated with the event (Biraben et al., 2001; Scicuttella & Ettinger, 2002). In addition, when a seizure occurs, a person may experience unusual auditory, visual, gustatory, or olfactory sensations. Behavioral manifestations of seizures may include simple repetitive movements, such as repeated chewing, or complex motoric tics, such as buttoning and unbuttoning clothing (Niedermeyer, 1999; Provini, Plazzi, Montagna, & Lugasidi, 2000).

Case studies in the neurological literature describe seizures preceding or following unusual behaviors. These have included strange verbalizations and behavioral anomalies (see Moore, Abou-Khalil, Fakhoury, & Matthews, 1998). There may also be an association between seizure activity and problem behavior. Caplan and Austin (2000) reported a higher incidence of behavior problems in persons with mental retardation who had seizure disorders. Leung, Ma, and McLachlan (2000) noted complex responses during seizures, such as
hitting others, engaging in picking motions with the hands, or running. Yung, Park, Cohen, and Garrison (2000) found that 31% of participants who had seizures also had problem behaviors such as inattention, hyperactivity, aggression, or oppositional behavior. Several researchers have reported a possible link between seizures and aggressive acts, describing violence before, during, or after a seizure in populations other than people with developmental disabilities (Blumer, Davies, Alexander, & Morgan, 2001; Brower & Price, 2000; Holzer & Baer, 1997; Marsh & Krauss, 2000; Shulman, 2000). Each of these findings suggests a possible relation between seizures and aggressive behavior.

Relatedly, researchers have suggested a link between epilepsy and anxiogenic or psychotic behaviors (Alper, Barry, & Balabanov, 2002; Biraben et al., 2001; Boylan, 2002; Manchanda, 2002; Scicutella & Ettinger, 2002). This has led some researchers and clinicians to suggest a link between seizures and psychiatric conditions, such as intermittent explosive disorder or impulse control disorder (Kaufman, Kugler, & Sachdeo, 2002; Van Elst, Woermann, Lemieux, Thompson, & Trimble, 2000; Woermann et al., 2000; Wylie, Glazer, Benbadis, Kotagal, & Wolgammuth, 1999).

However, to date, no empirical link between epileptic events and bouts of problem behavior has been documented for people with developmental disabilities, leaving treatment recommendations for these individuals to be based on “clinical lore” or epidemiological findings. For persons with developmental disabilities who have seizure disorders, establishing a link between problem behavior and epileptic events presents a unique challenge. Limited communication may prevent attempts to predict the onset of seizure activity or to describe unusual sensations associated with seizures. Therefore, the most preferable means of establishing whether an association exists is to conduct in situ observations of persons with developmental disabilities who engage in problem behavior and experience seizures to demonstrate “proof of concept.” In the current investigation, we documented seizures and problem behavior for three adult males with developmental disabilities in their home, work, and/or community settings. Our goal was to conduct a descriptive behavioral analysis to assess whether a link between seizures and problem behavior exists at the individual level using extended, direct-observation methods.

Method

Participants and Settings

Participants were 3 adult males with developmental disabilities who had previously received behavior-analytic services, including functional behavioral assessments and behavior intervention plans, from the first author. Inclusion criteria were (a) diagnosis of mental retardation, (b) diagnosis of seizure disorder with breakthrough seizures occurring at least once per week despite anticonvulsant medication use, (c) problem behaviors reported during the previous month, and (d) at the time of the study, a behavior intervention plan based on a functional behavioral assessment implemented and demonstrated to reduce socially reinforced problem behavior to low- or near-zero levels. Seven other people with developmental disabilities were evaluated for participation in the study, but they did not meet one, or more, of the inclusion criteria.

Stan was a 29-year-old male with severe mental retardation and a psychiatric diagnosis of impulse control disorder, not otherwise specified (NOS). Neurological reports of his seizure disorder indicated that he experienced absence seizures. Throughout the study, he was prescribed sodium valproate (Depakote®) 500 mg twice a day, topiramate (Topamax®, 25 mg 1.5 tabs at bedtime, diazepam (Valium®) 5 mg twice a day, and propranolol (Inderal®) 40 mg three times a day. Stan spoke using simple sentences and phrases. He was referred for the study because of unusual behaviors observed by support persons, including sudden changes in affect, staring blankly, and rapid onset of physical aggression.

Tom was a 27-year-old male with moderate mental retardation and cerebral palsy. He had a psychiatric diagnosis of impulse control disorder, NOS. Tom’s seizures were classified as absence seizures or separate tonic–clonic seizures. Current medications were phenytoin (Dilantin®) 130 mg a.m., 100 mg noon, 100 mg 6 p.m., 130 mg at bedtime; lamotrigine (Lamictal®) 200 mg 6 a.m., 200 mg noon, 300 mg 6 p.m.; topiramate (Topamax®) 300 mg twice a day; clorazepate (Tranxene®) 7.5 mg twice a day; and triamterene (HCTZ®) 25 mg once a day. Tom used simple sentences to communicate. With regard to receptive language, he required repeated prompts to follow a request or would respond by asking, “What?” Tom was referred to the study due to sudden changes in behavior, including aggression,
throwing objects, verbal threats, and noncompliance that occurred prior to observed seizure activity.

Mick was a 46-year-old male with moderate mental retardation. His psychiatric diagnoses were a mental disorder due to encephalopathy secondary to seizure disorder and impulse control disorder, NOS. Mick’s seizure activity was diagnosed as absence seizures or separate tonic–clonic seizures. Medications were lithium carbonate (Lithium\textsuperscript{®} 300 mg a.m., 600 mg at bedtime; ziprasidone (Geodon\textsuperscript{®} 80 mg twice a day; sertraline (Zoloft\textsuperscript{®} 50 mg in the morning; buspirone HCL (Buspar\textsuperscript{®})15 mg twice a day; lamotrigine (Lamictal\textsuperscript{®} 200 mg twice a day; propranolol (Inderal\textsuperscript{®}) 10 mg three times a day; and triamterene (HCTZ\textsuperscript{®}) 25 mg once a day. He communicated using simple sentences, usually with idiosyncratic themes (e.g., favorite television shows, country music, or preferred soft drinks). Support persons referred Mick to the study due to erratic behavior associated with seizure activity.

**Measurement**

**Observer training.** Support persons received training on recording seizures and problem behavior from the first author to ensure that measurement was consistent across observers. The first author discussed operational definitions of problem behavior and seizures with all observers. Descriptions of seizures were available either in the participants’ medical records from neurology consults or based on documentation from previous seizure records. Observers also viewed a video with examples of various types of seizures to help them detect occurrences of seizure activity and on-site training in the use of the measurement system. The first author then shadowed the support personnel several hours per day for 1 to 4 weeks to record and provide feedback to the observers regarding the occurrence and nonoccurrence of the events of interest. Training continued until all support personnel for each participant had accurately recorded behavior problems and seizures for a minimum of 2 weeks. Definitions were then reviewed with support personnel on a weekly basis.

**Problem behavior.** For Stan, problem behavior included slapping others or scratching others’ arms. Tom exhibited aggression in the form of hitting others, kicking others, verbal aggression, noncompliance, or throwing items at others. For Mick, problem behavior was defined as hitting others, kicking others, throwing items at others, pulling others’ hair, verbal aggression, or biting others. During the study, support persons documented problem behavior on a datasheet that included information about antecedents, behaviors, and consequences. Occurrences/nonoccurrences were recorded using an event-by-time paper-and-pencil system. The recording system required the observer to record the specific event that occurred from a menu of options and the time the event occurred. If no events occurred, the time of observation was recorded as no occurrence by the observer. Data for Stan were collected during his time at a sheltered workshop (9:00 a.m. to 3:00 p.m., Monday through Friday); observations for Tom and Mick occurred 24 hours per day, 7 days per week.

**Seizures.** Seizures for Stan were staring spells and cessation of ongoing activities. He also rubbed his head and made repetitive chewing motions. He was unresponsive to others for approximately 5 to 10 seconds. Seizures for Tom were separated into two major topographies. In the first type (absence seizure), Tom abruptly stopped ongoing activities, stared blankly, and was unresponsive for 5 to 10 seconds. In the second type of seizure (tonic–clonic), Tom fell to one side, showed repetitive hand or arm movements (e.g., picking at his shirt or jerking movements), chewing motions, rapid eye movements left to right with blinking, and facial tics. Tonic–clonic seizures lasted up to 6 minutes. After the seizure, Tom was drowsy and had slurred speech. Seizures for Mick were documented as absence or tonic–clonic. Absence seizures include staring spells and unresponsiveness for a few seconds, at times with repetitive facial movements (e.g., picking at his shirt or jerking movements), chewing motions, twitching of the lips. Tonic–clonic seizures included falling to the ground with stiffness in the limbs and trunk, followed by rapid body-shaking lasting up to 5 minutes. Following tonic–clonic seizures, Mick was drowsy and exhibited slurred speech and irregular gait.

For all participants, a Seizure Record Form was used to document specific information about observed seizure activity. The form used an event-by-time pencil-and-paper system to record the date/time, participant, observer(s), location, type of seizure, duration of seizure, physiological and behavioral states, precipitating factors, and duration of recovery. For recording seizures, a form separate from, but similar to, the observation record for problem behavior was used.
Interobserver Agreement

Support staff and the first author collected interobserver agreement data on the occurrence and nonoccurrence of seizures and problem behavior. For each participant, the recording of seizures and/or problem behaviors was completed independently by observers. Interobserver agreement checks for Stan, Tom, and Mick were conducted for 27%, 24%, and 25% of days, respectively. An agreement was defined as both observers recording the occurrence of the same event within 5 minutes of each other. This time window was used because of the extended length of the observations and variances in time calibration between observers. We calculated interobserver agreement by dividing the number of agreements by the number of agreements plus disagreements and multiplying by 100%. Percentage of interobserver agreement for occurrence and nonoccurrence of seizures and problem behaviors was 100%.

Statistical Analysis

Data were analyzed using the Yule’s $Q$ statistic, which is a method of computing a correlation between events. To compute Yule’s $Q$ scores, we arranged data for seizures and problem behavior occurrences in a $2 \times 2$ table, based on a yes–yes, yes–no, no–yes, or no–no designation (Bakeman & Gottman, 1997). For example, if a problem behavior occurred and a seizure occurred, this would constitute a yes–yes designation, whereas if there was no seizure but problem behavior occurred, this would be a no–yes designation. For individuals with tonic–clonic versus absences seizures, we computed separate $2 \times 2$ tables for each seizure type and problem behavior. Yule’s $Q$ scores range from values of $-1.0$ to $1.0$, with $-1.0$ representing an inverse correlation and $1.0$ representing a high positive correlation between events. A score of $0.0$ represents no correlation between the events. An additional statistical method, a Yate’s correction for continuity, was applied to control for values when the expected cell frequencies were small (Howell, 1997). To conduct a Yate’s correction, one must add a value of $0.5$ to the total number in each cell before computing the Yule’s $Q$ statistic. For example, there were no cases when Stan had an absence seizure without engaging in problem behavior; thus, the expected value of the cell would become $0.5$ with the Yate’s correction applied. Finally, to test the significance of each finding, we computed $z$ scores for each $2 \times 2$ table.

Results

Figure 1 shows seizures and problem behavior for Stan. Problem behavior was recorded following a seizure for 13 out of 16 days. The sequence of events for the 13 seizures that coincided with problem behavior included a brief cessation of activity, staring blankly, repetitive motions with his hands (e.g., rubbing his head or face) and aggression toward others. The mean time between the
onset of a seizure and occurrence of aggression was less than 1 minute. The three incidents of problem behavior that did not precede seizure activity included antecedents associated with environmental causes of his problem behavior (i.e., negative peer interaction). These environmental antecedents were consistent with previous functional behavioral assessment results. A Yule’s Q analysis yielded a value of 0.994, a high positive correlation between absence seizures and problem behavior, $z = 5.882$, $p < .05$.

Figure 2 shows seizures and problem behavior for Tom. He had two distinct types of seizures, absence and tonic–clonic. We observed absence seizures on 12 days and tonic–clonic seizures on 5 days. Less than 1 minute prior to all 5 tonic–clonic seizures, Tom stopped an ongoing activity, refused to participate with requests, threatened to hit support persons, and attempted to hit or throw items at them, and then seized. There were no documented environmental antecedents to these events. In contrast, no problem behavior was reported prior to the 12 absence seizures. There were 3 incidents of problem behavior not followed by seizures, but the events were associated with environmental factors. Two incidents involved refusal to participate with a request, and one incident involved requesting a preferred item and being denied access to it. Reported antecedents to problem behavior were consistent with functional behavioral assessment results, indicating escape from nonpreferred situations or access to preferred items. A Yule’s Q analysis revealed a score of 0.122 for absence seizures and problem behavior (obtained z score was nonsignificant). For tonic–clonic seizures and problem behavior, there was a strong positive correlation of 0.993, $z = 7.08$, $p < .05$.

Figure 3 shows seizures and problem behavior for Mick. On 3 days, we recorded a tonic–clonic seizure and on 11 days we observed absence seizures. For 10 out of 11 absence seizures, Mick engaged in problem behavior, including verbal and physical aggression toward others, immediately prior to the seizure. The mean time between the occurrence of hitting and seizure onset was 2.2 minutes. There was 1 problem behavior documented prior to a tonic–clonic seizure. There were 2 problem behaviors associated with either denying requests for preferred items or refusal to comply with support persons’ requests. These findings were consistent with previous functional behavioral assessments. A Yule’s Q analysis revealed a score of 0.473 related to tonic–clonic seizures and problem behavior, showing a moderately positive association; however, there were only 3 observed instances of tonic–clonic seizures. Because the marginal sum is less than 5, the interpretation of this result is limited by the Yule’s Q statistic, and no z score was computed. There was a strong positive correlation of 0.984 for absence seizures and problem behavior, $z = 5.49$, $p < .05$.

Discussion

This is the first empirical study demonstrating a close temporal relation between epileptic events and problem behavior for people with developmental disabilities. For all participants, problem behavior occurred pre- or postictally (i.e., within 2 minutes of a seizure). For Stan, problem behavior occurred following absence seizures. Problem behaviors for Tom preceded tonic–clonic seizures but were unrelated to absence seizures. For Mick, problem behavior occurred before absence seizures, but was not clearly related to tonic–clonic seizures. Our findings suggest that epilepsy and problem behavior can be associated in adults with developmental disabilities.

The results have important implications for understanding gene–brain–behavior relations in problem behavior. All 3 participants had undergone extensive and repeated functional behavioral assessments to identify environmentally based variables that maintained problem behavior. A set of environmental events were identified for each participant as related to problem behavior, interventions were implemented based on these events, and problem behaviors reduced in relation to these events. However, each individual continued to have problem behaviors that were not linked to identifiable environmental events. In such cases, it appears that problem behavior consistent with the pattern just described might be related to epileptic events. However, before such a hypothesis can be considered, researchers need to conduct an extensive analysis of environmental causes of problem behavior. Without a thorough functional behavioral assessment, the conclusion that epilepsy is associated with problem behavior may be inappropriate and have the unwanted effect of increasing prescriptions of antiepileptic drugs to control behavior problem through unknown and, perhaps, undesirable mechanisms (e.g., sedation).

Future researchers will need to identify the proportion of individuals with developmental dis-
Figure 2. Occurrences of problem behavior (closed circles), absence seizures (asterisks), and tonic–clonic seizures (open bars) for Tom. Five instances of problem behavior were observed to co-occur with a tonic–clonic seizure, but no problem behaviors were associated with absence seizures.

Figure 3. Occurrences of problem behavior (closed circles), absence seizures (asterisks), and tonic–clonic seizures (open bars) for Mick. On 10 of 11 instances of problem behavior, an absence seizure also occurred. Problem behavior was also observed to occur prior to one of three tonic–clonic seizures.
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Strategies for readily identifying individuals who may be at-risk for seizure-related problem behavior. The inclusion criteria for the current investigation was designed to identify people who were likely to display a relation between problem behavior and seizures. Therefore, individuals with developmental disabilities with partially controlled epilepsy (i.e., breakthrough seizures) and intermittent behavior problems may be more likely to show a similar association. Another consistent characteristic among participants was the psychiatric diagnosis of an unspecified impulse control disorder. This also suggests a diagnostic indicator (i.e., impulse control disorder, NOS) for individuals who might also be at-risk for behavior problems associated with seizure activity. An additional psychiatric diagnosis that might be appropriate for investigation is intermittent explosive disorder because of the diagnostic criterion of unpredictable behavioral outbursts without a clear relation to environmental events.

One possible intervention implication is that pre- and postictal problem behavior may not be under operant control. That is, if these behaviors are induced by atypical neurological activity, the problem behaviors may not be under the control of environmental reinforcement contingencies. Indeed, such events may not be able to be brought under environmental control because of their relation to seizure activity (cf. Cataldo, Russo, & Freeman, 1979). Although speculative, it is currently unclear whether such problem behaviors can be brought under the control of reinforcement contingencies and related behavioral processes typically used as interventions. It is possible that treatment of the underlying epilepsy may be the primary focus of intervention in such cases.

A critical qualification of our findings is the correlational nature of the data. Because the observational analysis was descriptive, we only noted a close temporal relation between problem behavior and symptoms associated with seizures. A next step for this line of research is to link electroencephalographic (EEG) recording of brain activity with behavioral observations. By simultaneously recording brain-wave activity in real time along with problem behavior, researchers can find a closer link between epileptic events and the occurrence of various behaviors. In addition, the use of EEG could define sites of epileptogenesis and generalization as well as specific seizure types. Such data may help establish a more direct relation between particular types of seizures and specific patterns of behavior problems.

In conclusion, we demonstrated a temporal association between seizures and problem behavior for 3 men with developmental disabilities. Previously, the neurological literature has included case histories of behavioral and psychiatric disturbances concomitant with seizures, but those findings have relied on case notes, interviews, and interpretative histories. Our findings are the first direct observation data to document patterns of occurrence at the individual level in persons with developmental disabilities.

References


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