The Report Committee for Ashley Elizabeth Chamberlain
Certifies that this is the approved version of the following report:

Cognitive-Communication Deficits Caused by Topiramate: A Summary of Implications Relevant to SLPs

APPROVED BY
SUPERVISING COMMITTEE:

Supervisor:

Joyce L. Harris

Dena H. Granof

Cognitive-Communication Deficits Caused by Topiramate: A Summary of Implications Relevant to SLPs

by

Ashley Elizabeth Chamberlain, B.S.

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Dedication

This report is dedicated to my always encouraging and supportive family and friends.

To Dad for always reminding me to wipe my tears and keep working hard - your positive attitude keeps me going!

To Mom for allowing me to cry when Dad wouldn't and reminding us all "Life is Good."

To my sister, Jennifer, for having faith in me and reminding me that to have a little faith in myself!

To all of my wonderful friends who spent countless hours working with me and providing me with moral support throughout this entire process. Without these people, I would not have completed this work successfully!

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Abstract

Cognitive-Communication Deficits Caused by Topiramate: A Summary of Implications Relevant to SLPs

Ashley Elizabeth Chamberlain, M.A.

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Supervisor: Joyce L. Harris

This report provides an overview of the adverse effects of the antiepileptic drug topiramate. Specifically, it evaluates the negative cognitive-communication effects of topiramate on individuals with epilepsy and postulates that treating these deficits is within the scope of practice of speech-language pathologists. It begins with a discussion on epilepsy, description of seizures, and the mechanism of action for antiepileptic drugs. It then provides an overview of cognitive communication deficits caused by antiepileptic drugs, including: memory problems, impairments in attention, and executive dysfunction. The final section provides an outline of potentially beneficial treatments a speech-language pathologist may provide to patients experiencing adverse effects from topiramate and how continued research can expand this area of practice.

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Case Study

Tom is a 27-year-old male who recently presented with difficulty starting and completing tasks, and has had trouble verbalizing his thoughts. His wife indicated that while Tom was previously a verbose and animated speaker, it is now difficult to follow his stories. She also stated that he frequently fails to communicate complete thoughts. Formerly highly attentive, Tom recently failed to complete a yearlong work project, causing him to be passed over for a promotion at work. Although Tom demonstrates awareness of his distractibility, he finds that he is unable to correct his behavior. Approximately four months ago, Tom began taking the antiepileptic drug (AED) topiramate after being diagnosed with epilepsy following a gran-mal seizure at home.

While Tom has not had any additional seizures, his wife had become concerned about his recent behavior and decreased communication abilities. She believes that Tom might have experienced brain damage following his seizure. A magnetic resonance image of Tom's brain indicated there was no damage. Although his symptoms are similar to those which individuals may experience following a traumatic brain injury (TBI), Tom's physician explained that his problems with memory, attention, and executive function may be linked to usage of the AED topiramate. Because topiramate had been successful in suppressing Tom's seizures, his physician recommended that Tom learn to accept these problems as part of his new reality. Tom and his wife are pleased that his epileptic seizures are under control, but they wanted know more about the possible adverse cognitive-communication effects caused by topiramate. Additionally, they wanted to

know if it is possible to regulate the cognitive-communication deficits caused by topiramate.

PURPOSE

According to the American Speech-Language-Hearing Association Scope of Practice (2007) treatment of cognitive-communication deficits is within the scope of practice for speech-language pathologists (SLPs). Currently, SLPs treat individuals with similar impairments in attention, memory, and/or executive function caused by a TBI. While it has been shown that prescription medications, including topiramate, cause impairments in attention, memory and/or executive function (U.S. Food and Drug Administration, 2011), the ASHA Scope of Practice (2007) does not recognize these adverse effects of prescription medication as a potential etiology of cognitive communication disorders. This report provides an overview of the limited knowledge and evidence regarding the consequences of the AED topiramate on cognitive-communication function in patients with epilepsy and proposes that treating patients experiencing these adverse side effects is within the SLP's scope of practice.

BACKGROUND

Epilepsy is a brain disorder that causes recurrent, spontaneous seizures. It is the most common condition in which chronic seizures are the primary symptom (Duvivier & Pollack Jr, 2009). Epileptic seizures are caused by an abnormal synchronization of the action potentials of numerous neurons. In a typical adult's brain, the excitatory and inhibitory systems are balanced through proper regulation of synapses. In patients with

epilepsy, synapses are not properly regulated and thus become hyper-excited. This increased excitability means that excitation and inhibition of brain activity are no longer in balance thus, seizures occur (Rogers & Cavaos, 2008). To control for epilepsy-induced seizures, AEDs are commonly prescribed. These drugs prevent both chronic and intermittent seizures caused by epilepsy. While AEDs are not a cure for epilepsy, they work to suppress seizures caused by the imbalance of neuronal activity (Engel & Pedley, 2008).

Under ideal circumstances AEDs will eliminate seizures without adverse effects. Possible negative effects may range in severity from life-threatening allergic reactions to more manageable problems, including nausea and irritability. Whether a patient will present with adverse effects depends on age, overall health status, and the type and dosage of an AED. In some cases, a higher drug dosage correlates with an increase in adverse effects. Other drugs cause adverse effects irrespective of dosage. It may be possible to minimize adverse drug reactions by decreasing the AED dosage, but at times, deficits are unavoidable and may be irreversible (Fisher, 2009). In particular, patients using AEDs may experience undesirable effects on cognitive-communication processes including disturbances in attention, memory, and executive function.

Although there is no exact formula for choosing the best seizure medicine for each patient (Fisher & Saul, 2010), both the effectiveness of eliminating seizures and the occurrence of adverse effects must be considered (Neal et al., 2008). Because topiramate is considered highly effective for seizure control in patients presenting with epilepsy, it is

frequently prescribed despite its effects on cognitive-communication abilities (Kockelmann, Elger, & Helmstaedter, 2003).

Types of Seizures and AEDs

Epileptic seizures can be classified as either primary generalized seizures or partial seizures. Primary generalized seizures are caused by a widespread imbalance of neuronal activity within the brain (i.e. involves the entire brain). Partial seizures occur when imbalance of neuronal activity is limited to a specific portion of the brain. Although seizures manifest differently, all patients with epilepsy experience seizure activity (Fisher & Saul, 2010).

Antiepileptic medications are divided into two categories: broad spectrum and narrow spectrum. Broad-spectrum AEDs are effective for treatment of both generalized and partial seizures. Narrow-spectrum AEDs are used only for treatment of partial seizures. Many adults are prescribed broad-spectrum AEDs because they work for a wide variety of seizures. An additional consideration for the administration of AEDs is the use of a monotherapy or polytherapy regimen. Monotherapy is the use of a single AED; polytherapy is the use of more two or more AEDs. Often, patients prefer monotherapy because taking one AED is easy to remember and it may minimize the potential for adverse effects (Reynolds & Shorvon, 1981).

Used as part of a monotherapy or a polytherapy regimen, topiramate has been shown to be effective in controlling both primary generalized seizures and partial seizures. Therefore, topiramate is prescribed to epileptic patients presenting with either type of seizure (Sachedo, Reife, Lim, & Pledger, 1995).

MECHANISM OF ACTION AND ADVERSE EFFECTS OF AEDS

Within the United States, it is estimated that approximately three million people currently have active epilepsy. Active epilepsy is defined as a history of epilepsy plus occurrence of at least one seizure or the use of an AED within the past five years (Fisher & Saul, 2010). Neurotransmitters are the chemicals released by neurons in the brain. There are two key neurotransmitters involved in understanding and treating seizures: gamma-aminobutyric acid (GABA) and glutamate. GABA and glutamate receptors are found on approximately 100% of neurons within the brain. Decreased brain inhibition is caused by the reduction in GABA activity combined with an increased glutamate activity. The goal of AEDs is to restore the balance of brain activity by: increasing GABA, reducing glutamate, or decreasing neuronal firing (Rogers & Cavaos, 2008).

Because topiramate is a newer AED, its mechanism of action within the brain is not fully understood. First approved for seizure treatment in 1997, current research indicates that topiramate exerts its antiepileptic influence by enhancing the inhibitory function of GABA (Hung & Shih, 2011). Topiramate blocks sodium channels to reduce neuronal firing in the central nervous system, and, thus, promotes the inhibitory action of GABA (Hung & Shih, 2011). These actions restore balance to neuronal activity in the brain.

The ability of AEDs to serve as a seizure control while maintaining minimal adverse effects is a continued topic in current research. The main difference between older generation AEDs (those approved before 1990) and new AEDs is the minimization of adverse effects (Bialer & White, 2010). Adverse effects are minimized by simpler

pharmacokinetics. Pharmacokinetics is the study of the processes that affect the drug's time course within the body. The time course of a drug is related to drug absorption, distribution, metabolism and excretion. Like most drugs, the primary pharmacokinetic interaction for all AEDs involves the metabolism of the drug. Most AEDs are metabolized in the liver. Different AEDs trigger enzyme induction, enzyme inhibition, or both. Depending on the AEDs metabolic rate, the pharmacokinetic properties of the medication may be altered (Cloyd, 2000). Specifically, older AEDs, such as carbamazepine, valproate, and phenytoin, have a tendency to alter the enzyme induction and/or enzyme inhibition more severely. Enzyme induction involves an increased amount of enzyme proteins, resulting in increased metabolic rate of the drug. More rapid metabolization decreases the amount of time the AED is in a patient's body, thus possibly decreasing drug effectiveness. Enzyme inhibition can immediately cause toxicity within the body, and subsequently cause a variety of side effects (Bialer, 2005).

Unlike old AEDs, new AEDs exhibit a less severe effect on enzyme induction or enzyme inhibition. The new AEDs lamotrigine and oxcarbazepine, do not alter enzyme inhibition but do effect enzyme induction. Similar to many old AEDs, the alteration of enzyme induction can change the ability of the AED to effectively control seizures. On the other hand, topiramate alters enzyme inhibition, which may lead to toxicity that can cause greater adverse effects in the users of the drug (Bialer, 2005).

While it is important to minimize seizures, it is also necessary to ensure that patients using AEDs can sustain their activities of daily living (Mayo Clinic, 2009).

Patients' criterion for successful adjustment to living with epilepsy consists of seven components: energy, emotions, daily activities, mental activity, medication effects, seizure worry, and overall quality of life (Cramer et al., 1998). The most commonly prescribed AEDs are supposed to be those with the least likelihood of causing debilitating effects on quality of life indicators for patients with epilepsy (Fisher, R.S., 2009). However, each patient's perception of an acceptable quality of life will be different. Despite generally more favorable outcomes, adverse psychological and cognitive-communication effects still occur in many patients who take AEDs (Bialer & White, 2010).

NEGATIVE PSYCHOLOGICAL IMPACT

Negative psychological effects caused by AEDs include alterations in the patient's state of mind, including anxiety, behavioral changes, depression, irritability, and psychosis. Because of these potential effects, the FDA requires that all AED labels carry a suicide warning. Due to the current lack of large-scale studies on new AEDs, the exact incidence of negative psychological effects is unclear. In Weintraub, Buchsbaum, Resor, & Hirsch's (2007) study of 1394 adults taking new AEDs, 221 (16%) experienced negative psychological effects. This study found that previous psychiatric conditions were the strongest predictor of negative psychological effects occurring on AEDs. Nevertheless, 141 (14%) of the 1025 adults with no history of psychiatric conditions experienced negative psychological effects caused by a new AED.

COGNITIVE-COMMUNICATION DEFICITS

Cognitive-communication deficits describe any communication deficit that results from a barrier in cognitive performance (Aldenkamp, Taylor, & Baker, 2008). Commonly occurring deficits include impairments in attention, memory, and executive function. These impairments may be caused by the seizures, the etiology of the seizures, or the drugs used to control seizures. It is not possible to entirely control for all of these factors, and in many patients a combination of causes may lead to cognitive-communication impairments (Aldenkamp, Taylor, & Baker, 2008). AEDs produce global changes in the brain through the reduction of neuronal firings or through the alteration of the levels of glutamate or GABA. These global changes in the brain may cause cognitive-communication deficits, and all patients must be informed of the increased risk for these deficits while taking AEDs (Ortinski & Meador, 2004). Although the development of new AEDs has correlated with a decline in the report of debilitating cognitive-communication effects, cognitive-communication deficits have not been eliminated. According to Arif et al.'s (2008) long-term study of nearly 2000 patients with epilepsy, all new drugs produce negative cognitive-communication effects in some patients. In fact, 12.8% of patients changed the dosage or discontinued use of their AED because of negative cognitive-communication effects (Arif et al., 2008).

Cognitive-Communication Deficits of Topiramate

Used as part of a monotherapy or polytherapy regimen, topiramate is currently one of the most commonly prescribed and effective broad-spectrum AEDs. Because it is a newer AED, there are a limited number of studies on the cognitive-communication deficits caused by topiramate. Despite the lack of in-depth research, some evidence exists to support the notion that cognitive-communication deficits occur in individuals taking topiramate (U.S. Food and Drug Administration, 2011).

Vingerhoets (2006) reviewed 16 studies on brain damage caused by seizures that indicated that recurrent seizures, such as those caused by uncontrolled epilepsy, are associated with widespread impact on brain structure and function. The review stated that it was unclear if this impact could be considered brain damage, but the effects on the brain have been associated with a decline in cognitive status. Vingerhoets (2006) also reviewed six studies on the cognitive status of patients who have seizures. Five of six studies showed mild cognitive decline was caused by changes in brain structure and function. These studies further indicated that patients with chronic epilepsy have impaired performances in memory, language, and executive function. It is unclear if these impairments are associated with the severity (defined by duration and number) of uncontrolled seizures. Because the relationship between frequency and severity of seizures and cognitive decline is rarely addressed by research, this factor may be a significant confounding variable in studies on cognitive-communication deficits caused by topiramate (Vingerhoets, 2006).

In the United States, topiramate is available from Ortho-McNeil Neurologics under the brand name Topamax. The most recent information from Ortho-McNeil Neurologics about prescribing Topamax contains a description of a controlled trial that evaluated patients' adverse reactions to the drug. According to the label placed on Topamax, 21% of patients using a topiramate as a monotherapy at the recommended dosage of 400mg/day, discontinued use due to adverse side effects. Of patients who discontinued the use of topiramate, at least 4% cited executive dysfunction or memory difficulties as the primary reason for discontinuation of the drug. Twenty-six percent of adult patients receiving the daily recommended dosage of topiramate, experienced one or more cognitive-communication deficit in the areas of attention, memory, or executive function. Therefore, approximately 22% of patients continued to use topiramate while experiencing cognitive-communication deficits. Although information on the incidence of adverse effects for all patients who use topiramate is not available, the label advises prescribers to use these numbers as an estimate of the potential effects the drug may have on the patient's cognitive-communication status (U.S. Food and Drug Administration, 2011).

A number of small clinical trials have investigated the number of topiramate users who experience cognitive-communication deficits as a side effect of the medication. Gomer et al.'s (2007) study of 21 patients using topiramate indicated that topiramate might impair frontal lobe functions, despite initial patient reports of minimal cognitive-communication consequences. Frontal lobe functions include the ability to recognize

consequences, detect similarities and differences, and inhibit inappropriate social behaviors (Gomer et al., 2007). Kockelmann et al. (2003), found that all 20 participants in their study showed significant improvement in frontal lobe function after discontinuation of topiramate. They also noted that patients frequently under reported cognitive-communication impairments while taking topiramate (Kockelmann et al., 2003). In Arif et al.'s (2008) long-term study of negative cognitive-communication effects as a result of AED usage, topiramate had the most extensive deficit profile of the fourteen AEDs investigated. The authors found that 21.5% of the 130 patients taking topiramate experienced intolerable cognitive-communication effects that lead to the discontinuation of the drug (Arif et al., 2008).

MEMORY

Memory assessment in patients taking topiramate has been primarily limited to short-term and working memory capabilities. Short-term memory occurs after an initial exposure to an idea or sensation, but before the information is encoded for storage in long-term memory. Short-term memory has a limited capacity, usually five to nine items, and is integral for new learning. Short-term memory and attention are related, and deficits in attention to task can cause short-term memory problems (Cowan, Nugent, Elliott, Ponomarev, & Saults, 1999). To assess short-term memory deficiencies, researchers used the forward digit-span task to test immediate recall of a random sequence of numbers (Lee et al., 2003). In Lee et al.'s (2003) study of 38 patients with epilepsy, who were taking topiramate, patients performed poorly on the digit-span task. Short-term

memory deficits were further evidenced by difficulty on verbal and nonverbal tasks based on the *Memory Assessment Scale (MAS)*. The MAS uses list learning, immediate and delayed word recall, word recognition, and visual reproduction to assess short-term memory. The MAS was then re-administered to four patients following the discontinuation of topiramate use. All of these patients saw improvement in short-term memory functioning after termination of drug use (Lee et al., 2003).

In another assessment of 15 patients using topiramate as a primary AED, all patients were found to have memory impairments based on their performance on the *Verbal Learning and Memory Test*. (Fritz, Glogau, Hoffman, Rademacher, Elger & Helmstaedter, 2005). This word-learning task requires five trials in which participants encode 15 words for immediate recall, recall after distraction, and recognition after a thirty-minute delay. Study participants demonstrated poor recall on all tasks. This study concluded that formal testing of memory should be administered to all patients taking topiramate, even without patient complaint of memory deficits. Fritz et al. (2003) found that memory problems occurred at a high rate, therefore, all patients should be tested. Specifically, short-term and working memory abilities can be adequately assessed through screening of phonemic word fluency and use of the forward digit-span task (Fritz et al., 2003).

ATTENTION

Because attention impairments were commonly seen in patients taking old AEDS, researchers have been eager to determine if new AEDs, like topiramate, may eliminate

this common negative effect. Similar to testing short-term memory, assessment of attention impairments often relies on the digit-span task. Because this task is used to assess both memory and attention, it has been postulated that patients' memory impairments may be caused by attention problems (Lee, Jung, Suh, Kwon, & Park, 2006).

One alternative test for assessing attention is the *Trail Making Test* (TMT). The TMT requires subjects to complete two tasks: (1) rapidly order 25 circles numerically (1 to 2, 2 to 3, etc.) and (2) connect circles in an ascending pattern alternating between numbers and letters (1 to A, A to 2, 2 to B, etc.) (Burton & Harden, 1997). This test was used to assess attention in 20 participants in Kockelmann et al.'s (2003) study. Before testing, only five participants reported complaints of attention impairments. Despite contrary self-report, performance on the TMT indicated that all 20 participants had attention impairments. A similar study by Gomer et al. (2007) replicated these findings with 21 patients with epilepsy, who all also experienced attention impairments. These patients all showed significant improvement in attention following withdrawal of topiramate (Gomer et al., 2007). Both authors explained that relatives and other individuals close to the patients were more likely to recognize the negative attention impairments than patients themselves (Kockelmann et al., 2003).

EXECUTIVE FUNCTION

Lack of self-awareness of impairments in memory and attention may be linked to concurrent impairments in executive function (Bivona et al., 2008). Executive function

encompasses the cognitive processes that require the ability of linking previous experiences with current events. In typically functioning adults, executive function allows an individual to plan, initiate, change and complete daily activities (Miyake, Friedman, Emerson, Witzki, & Howeter, 2000). Patients taking topiramate frequently score poorly on tests of executive functioning.

Gomer et al. (2007) used phonemic verbal fluency as a measure of executive function. Before beginning topiramate, 21 patients were given one minute to write down all the words they could think of starting with the same in letter. This task was repeated after patients were on a maintenance dosage of topiramate. Comparison of results showed that nearly 48% of patients did at least one standard deviation worse on the second test. This indicates that topiramate caused a decline in executive function as measured by phonemic verbal fluency (Gomer et al., 2007). Kockelmann et al. (2003) found similar results in their study of 20 patients. The combination of poor results on tests of memory, attention, verbal fluency, and other neuropsychological measures clearly indicated that topiramate interfered with executive function.

Implications for SLPs

Current research has recommended that SLPs acknowledge the role that medications can have on cognitive-communication abilities. According to Youse (2008), SLPs play an important role in monitoring the effects of medication on patients' cognitive-communication abilities. Although topiramate is a highly effective seizure suppressant, studies have shown that it causes negative cognitive-communication effects in patients with epilepsy (Tatum et al., 2001). Since topiramate is a successful form of seizure treatment, doctors frequently recommend continued use of the medication, despite significant impairments in memory, attention, and executive function. Suppressing seizures is considered the primary goal for doctors, because if seizures persist, the brain can be further damaged (Tatum et al., 2001). Because it is clear that topiramate negatively impacts communication, SLPs must be enlisted as a resource to improve cognitive-communication abilities.

Patients using topiramate should consider speech-language therapy for treatment of attention, memory, and executive function impairments. SLPs work with other patients with communication disorders that follow patterns similar to those impairments caused by topiramate (Rohling, Faust, Beverly, & Demakis, 2009). Currently, there is a lack of research supporting the use of speech-language therapy for cognitive-communication deficits caused by topiramate or any other prescription drugs. Therefore, SLPs working with patients using topiramate should rely on cognitive-communication treatment

strategies currently implemented with patients who have sustained a TBI. Existing cognitive-communication treatments include group treatment, treatment as needed, and community based treatment.

GROUP TREATMENT

Rath, Simon, Langenbahn, Sherr, & Diller (2003) found that a cognitive-linguistic group treatment focusing on problem-solving deficits was an effective treatment for executive functioning, and increased self-appraisal skills. Sixty patients, who were at least 1-year post-traumatic brain injury, participated in this study. Participants were also required to have minimum, basic communication skills, but "basic skills" were not defined. All patients were required to be between the ages of 20-65 and have English reading abilities at approximately a ninth-grade level or greater. Additionally, all patients had documented impairments in social functioning, but were ineligible if they had a history of a psychological condition.

The 60 participants were divided into two groups: 27 patients were assigned to the problem-solving group (5 participants in this group did not complete the study) and 19 patients participated in conventional group therapy (9 patients from this group did not complete treatment). Pre-treatment, post-treatment, and 6 months following the study, participants in both groups completed assessments of (1) cognitive skills (including attention and memory, (2) psychosocial functioning, and (3) problem solving (including executive functions) (Rath et al., 2003).

Results indicated that patients in the group treatment program focusing on problem-solving deficits had significant improvement in executive function, as measured by the *Wisconsin Card Sorting Test* (WCST). Members of both the innovative treatment group and the conventional therapy group saw improvements in memory, evidenced by their performances on the *Wechsler Memory Scale – Third Edition* (WMS III). No significant improvements in attention were found in either group. Memory improvements by both groups were maintained through the 6-month follow-up assessment, although generalization of treatment was difficult to assess. Additionally, the innovative treatment group had fewer dropouts, which the authors attributed to the use of engaging, structured materials during therapy. Compared to the conventional group therapy, innovative treatment group therapy treated problem-solving capabilities in addition to global communication needs (Rath et al., 2003).

Treatment as Needed

Paniak, Toller-Lobe, Durand, & Nagy (1998) described the use of a single treatment session as an adequate intervention for patients with mild TBI (mTBI). One hundred and nineteen adults who were less than three weeks post mTBI were recruited from two hospital emergency rooms. Participants voluntarily participated in the study but were excluded if they met one or more of the following criteria: history of psychiatric disorder, previous TBI or mTBI within the past year, central nervous system disorder, inability to read English fluently, diagnosis of mental retardation, and/or current pregnancy (Paniak et al., 1998).

Over the course of two years, the patients were randomly assigned to a single treatment session or treatment-as-needed. Individuals in the single treatment session group were educated about the common constellation of impairments following an mTBI and how to cope with those problems. In the treatment-as-needed group, patients were given the same information as the single session group. Additionally, they had neuropsychological and personality assessments and were told they could return for further treatment as desired. Pre-treatment and at three to four months post-treatment, three outcome questionnaires, the *Problem Checklist* (PCL), the *Community Integration Questionnaire (CIG)* and the *Short Form-36 Health Survey* (SF-36), were administered. On all of these tests, factors related to the patient's cognitive, social, and vocational functioning were addressed (Paniak et al., 1998).

Results indicated that for patients with mTBI, brief education and reassurance was just as effective as more intensive intervention. In both intervention groups, patients who reported executive dysfunction following injury had improved three to four months later, according to self-report (Paniak et al., 1998).

COMMUNITY BASED TREATMENT

Hibbard, Cantor, Charatz, Rosenthal, Ashman, Gunderson…Gartner (2002) stated that peer support may enhance the quality of life and coping abilities of patients with TBI and their families. Twenty individuals, 11 patients with TBI and 9 family members, participated in this study. Participants had completed a TBI Mentoring Partnership Program (TBI-MPP) in which they received one-year of guidance from mentors. Each

mentor was an individual with TBI or a family member of a person with TBI. Mentors were chosen based on successful adjustment to challenges caused by TBI as evidenced through interviews with local professionals (Hibbard et al., 2002).

Results indicated that individuals with TBI and their family members felt that the TBI-MPP had some impact on their ability to cope with the consequences of TBI and on their overall quality of life as evidenced by self-report on the *Delighted-Terrible Scale*. The *Delighted-Terrible Scale* assesses quality of life through the single question, "How have you felt about the overall quality of your life in the past month?" According to a multiple-choice survey created by the authors, a majority of participants with TBI (73%) indicated that they saw little improvement in their communicative interactions. They concluded that while peer mentorship appeared to be effective in improving attitudes and outlook of individuals with TBI and their families, the areas of impact do not extend to improvements in cognitive-communication abilities (Hibbard et al., 2002).

CONCLUSION

This report describes the cognitive-communication deficits caused by the AED topiramate. Existing research does not stress the importance of understanding these impairments prior to the administration of an AED to a patient with epilepsy. Currently, patients taking topiramate are expected to tolerate the cognitive-communication impairments caused by the drug without the help of trained cognitive-communication specialists. Furthermore, studies show that many patients with epilepsy using topiramate do not self-report cognitive-communication problems. This indicates that the incidence of

cognitive impairments is likely much higher than currently reported. Arif et al. (2008) reported that, in a study of 1694 adults with epilepsy, the average rate of cognitive side effects from AEDs was 12.8%. In addition, topiramate was found "significantly more intolerable" than most other AEDs investigated. Research showed that patients using topiramate experience cognitive-communication deficits specifically in the areas of attention, memory, and executive function. Such deficits are similar to the common constellation of communication deficits that are often seen in patients who have had a TBI. Despite the similarity in deficits, the cognitive-communication problems of patients who are taking topiramate to control epilepsy are not currently treated by SLPs.

Additional research is needed to determine the efficacy of speech-language therapy in treating adverse cognitive-communication effects caused by prescription medications. Currently, SLPs must use the existing research on cognitive-communication deficits in patients with TBI as a guideline for therapy for patients who are experiencing communication deficits secondary to taking topiramate. SLPs should use strategies that are typically implemented in therapy with clients who have had a TBI to improve memory, attention, and executive function. Some current therapy techniques for patients who have sustained a TBI include group therapy, therapy-as-needed, and community based therapy. Because it is within the scope of practice for SLPs to treat cognitive-communication disorders, treatment of patients taking AEDs should be considered under the scope of practice. However, additional evidence on the efficacy of treatment for this population is needed before SLPs can provide the highest level of evidence-based

practice to their patients who are experiencing cognitive-communication deficits secondary to taking AEDs.

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