Psychopharmacology and Applied Behavioral Analysis: Tandem Treatment of Severe Problem Behaviors in Intellectual Disability and a Case Series

Lee E. Wachtel, MD,[1,3] and Louis P. Hagopian, PhD[2,3]

[1] Department of Child and Adolescent Psychiatry, Kennedy Krieger Institute, Neurobehavioral Unit, Baltimore, Maryland, U.S.A.

[2] Department of Behavioral Psychology, Kennedy Krieger Institute, Neurobehavioral Unit, Baltimore, Maryland, U.S.A.

[3] Department of Psychiatry and Behavioral Science, Johns Hopkins University, School of Medicine, Baltimore, Maryland, U.S.A.

Abstract: Many individuals with intellectual disability will at some time in their lives engage in problem behaviors that may place them and others at risk, and reduce their opportunities for healthy psychosocial functioning. These behaviors may reach severe proportions in both intensity and frequency, necessitating intervention. Both psychiatrists and behaviorists are often approached regarding negative behaviors in intellectual disability, and each discipline offers key tools in behavioral assessment and resolution. We believe that the coordinated effort of these two disciplines affords the most comprehensive and efficacious method of assessing, understanding and treating a wide range of problem behaviors and associated psychiatric pathology in individuals with various forms of intellectual disability. This paper briefly reviews the background of problem behaviors in intellectual disability and treatment of such disturbances through separate psychiatric and applied behavioral modalities, followed by the proposed coordinated *neurobehavioral model.* A case series ensues, describing the successful application of the neurobehavioral model to the severe problem behaviors demonstrated by three individuals with intellectual disability related to autism, Cornelia de Lange syndrome and traumatic brain injury.

Introduction

Problem behavior background

The presence of problem behaviors in individuals with intellectual disability (ID) has been the subject of much concern and research over the past decades, and remains a significant challenge to many with ID, as well as those involved in their lives. Problem behavior in ID can take many forms, including self-injury, aggression, disruption, destruction, pica, elopement and inappropriate sexual behavior. Significantly, such negative behavioral expression often includes multiple topographies at any given time, and tends to persist and stabilize over years (1).

Multiple studies have shown a high prevalence of problem behaviors for people with ID in various settings. Prevalence rates in U.S. surveys of ID range from approximately 2–28% for aggression, 10–31% for self-injury and 7–30% for property destruction, with rates consistently higher with more severe degrees of mental retardation and in institutions compared with the community (2, 3). These behaviors are of significant concern due to their far-reaching physical, emotional, social, educational and economic consequences (1). Not only can self-injury and aggression cause serious physical injuries, they also lead to reduced educational, community and habilitative opportunities (1, 4). Maladaptive behaviors are further correlated with decisions to seek residential over family placement (5), and their financial cost is staggering, with annual costs in the U.S. exceeding $3 billion (1).

Understanding Problem Behaviors

Understanding the etiology of problem behaviors re-

Address for Correspondence: Lee Wachtel, Medical Director, Neurobehavioral Unit, Kennedy Krieger Institute, 707 N. Broadway St., Baltimore, MD, 21205, U.S.A. E-mail: wachtel@kennedykrieger.org

quires consideration of many factors. First, assessment for additional psychopathology in the form of concomitant or underlying mental disorders is critical, as negative behavioral expression and mental retardation are both strongly associated with an increased risk for frank psychiatric illness (2, 6, 7). Actual rates of psychiatric illness in the ID population range from 10–80%, with a suggested prevalence of 3–4 times that of the general population (2, 8). In the United States, the diagnosis of psychiatric illness in ID relies largely on the criteria set forth by the Diagnostic and Statistical Manual in its current edition, the DSM-IV-TR, as well as multiple rating scales designed specifically for patients with cognitive impairment (9–11). Even with such, there are potential pitfalls in the diagnosis of psychiatric pathology in ID, characterized by various forms of *diagnostic overshadowing* whereby psychopathology is grossly attributed to the underlying mental retardation (2, 7).

Known genetic illness may also increase the likelihood of problem behaviors and psychiatric pathology in the ID population. Indeed, there are myriad genetic syndromes with known behavioral phenotypes of which aberrant behaviors and mental illness are accepted elements (12–15).

For individuals with intellectual disabilities, problem behavior may further emerge out of a combination of factors including deficits in communication and adaptive behavior. Large-scale epidemiological research has shown that the risks for problem behavior increase as a function of the level of mental retardation, degree of communication deficits, and placement in a low-stimulation environment (16). For an individual with a limited adaptive behavioral repertoire, getting basic needs met may be quite difficult and frustrating. As is the case with typically developing young children, frustration can induce negative emotional states as well as problem behavior (e.g., tantrums). These problem behaviors may be inadvertently reinforced when they effectively produce favorable changes in the environment — including gaining access to attention or preferred materials, or escaping non-preferred situations including academic demands. Parents' and teachers' efforts to calm a child (by redirecting him/her to toys or by removing demands), though effective in reducing problem behavior for the moment, may inadver-

tently strengthen it through the process of reinforcement (17). For an individual with limited adaptive behavior (limited communication and problem solving skills, etc.), problem behavior may become highly effective for accessing reinforcement or avoiding non-preferred situations. Indeed, for most individuals (at least two-thirds) with ID and problem behavior, problem behavior can be shown to be mediated for social variables such as attention, access to toys, or escape from demands (18).

Psychiatric Treatment

Where a distinct psychiatric illness is identified in patients with ID, it has long been accepted that psychotropic interventions should be the same as for the general population (19, 20). The American Academy of Mental Retardation has published specific medication recommendations for the major psychiatric illnesses identified by an expert consensus panel as identical to those received in surveys for psychiatric illness without MR (19).

Research in the psychopharmacology of ID and psychiatric illness has also provided support for the efficacy of various psychotropic agents, including antipsychotics, mood stabilizers, antidepressants and stimulants, for specific psychiatric diagnoses in ID (21–24). However, many of the reviews also raise questions as to the methodological stringency of various studies of psychotropic usage in ID, as well as pointing out the risk of side effects in ID, particularly with antipsychotics and the concomitant usage of multiple agents (21–24).

Psychotropic medications may also be utilized in ID for the treatment of problem behaviors alone without an additional psychiatric diagnosis of the target symptoms and/or disorder, including a complete investigation of operant or somatic causes for such. Both antipsychotic and antidepressant medications may play a role in the treatment of self-injury, as well as the opioid antagonist naltrexone (22, 25–29). In fact, aggressive behavior is the most frequent reason for initiation of medication in patients with ID, and often prompts polypharmacy. Aggressive patients have historically been most frequently prescribed typical and atypical antipsychotics. While these medications may be excessively used, they also have demonstrated clinical efficacy in reduction of

some aggressive behaviors in ID (23, 24, 26, 27). Serotonin-enhancing antidepressants have shown success as well in the treatment of aggression in patients with ID (28, 30), as have lithium, anticonvulsants and antihypertensives (31, 32).

Applied Behavioral Analysis-Based Assessment and Treatment

The assessment and treatment of problem behaviors and potential psychopathology is not, however, limited to the use of the DSM, concrete rating scales and psychotropic medications. The field of applied behavior analysis has produced an impressive body of research related to effective assessment and treatment of self-injurious behavior (SIB) (33), aggression (34) and pica (35). The hallmarks of this discipline are 1) the *application* of operant learning principles, 2) the precise measurement of observable *behavior*, and 3) the use of experimental *analysis* methodologies to identify behavior-environment relations.

Behavioral assessment of problems is designed to determine what factors are occasioning and maintaining these behaviors (functional behavioral assessment). In some cases, identification of these controlling variables is possible based on careful interview and observation of the individual. However, for others a more formal and intensive "functional analysis" is required. Functional analysis (36) is the most empirically rigorous methodology used to assess the environmental determinants of behavior, and is considered best practice in the field (19). Functional analysis involves a process of experimentally manipulating antecedent and consequent events and then precisely examining how the behavior of interest changes as a function of those manipulations. The experimental conditions are designed as analogs to situations in the natural environment, wherein potentially reinforcing consequences are provided for problem behavior. The research literature indicates that in approximately two-thirds of cases, self-injury can be shown to be maintained by socially-mediated events such as access to preferred items, access to attention, and escape from demands to name a few (37, 38). The proportion of cases with socially mediated aggression, disruptive, and other problem behavior is undoubtedly higher than for self-injury.

Though the immediate purpose of the functional analysis is to identify the variables (antecedent and consequent events) controlling problem behavior, the ultimate purpose is to guide treatment selection and development. Effective behavioral treatments for problem behavior typically rely on training individuals in alternative adaptive behavior repertoires, altering adult responses to problem behavior to minimize reinforcement for that behavior, and engineering the environment to minimize the probability of problem behavior (39, 40). Generally, with few exceptions, most behavioral interventions involve reinforcement of alternative responses concurrent with extinction for problem behavior, which is the withholding of reinforcement following problem behavior — in an attempt to weaken this response. One class of intervention, functional communication training, involves teaching the individual an appropriate communication response to access the reinforcer responsible for maintaining problem behavior (41). Once this new learning history is established, then treatment is aimed at teaching the individual to tolerate waiting or not always gaining access to the requested activity without reverting to problem behavior. Another class of interventions, noncontingent reinforcement, involves providing free access to the reinforcer responsible for maintaining problem behavior. Subsequently, access to reinforcement is gradually decreased while maintaining low levels of problem behavior (40). Other classes of interventions involve providing reinforcement for incompatible or alternative responses such as compliance in the absence of problem behavior (42). Further interventions involve the provision of stimuli and activities that have the property of competing with reinforcement maintaining problem behavior (43). For many individuals with ID, communication deficits necessitate the use of discriminative stimuli to signal transitions, activities, and which "rules" are in effect at any given time. These specialized procedures permit individuals with even the most severe intellectual disabilities with the opportunity to express their preferences and choices, thus empowering them to actively participate in the development of their behavior program.

Neurobehavioral Model

Having worked with several hundred children and adults with intellectual disability and severe concomitant behavioral disturbances, it is our experience that the *tandem* usage of psychiatric assessment and applied behavioral analysis affords the best opportunity for accurate understanding of problem behaviors and appropriate diagnosis of additional psychiatric illness, which in turn maximizes the potential for effective treatment interventions. We term this data-based, principled approach the *neurobehavioral model*, and find it allows us to understand problem behaviors along a diagnostic spectrum of psychiatric and operant components.

First, we recognize that some patients exhibit problem behavior that is *primarily* due to a psychiatric disorder. For example, in the case of an individual with ID and psychosis or mania, disruptive or aggressive behavior may occur secondary to the disinhibition, disorganization of thought, or delusions that may occur as a function of the underlying psychiatric illness. Successful pharmacological treatment of the identified illness results in resolution of the behavioral disturbances, and the recommended first line intervention would therefore be psychotropic medication. Of course, additional operant functions may also exist for the patient's behavior, such as graphic descriptions of delusions or excessive behavioral displays for the purpose of attention. This superimposed behavioral layer would also necessitate behavioral intervention as the core psychiatric illness resolved pharmacologically.

At the opposite end of the spectrum we find patients for whom behavioral and psychiatric assessments reveal that problem behavior is largely environmentally caused and there is no related or concomitant psychiatric disorder other than that used to describe the problem, such as Stereotypic Movement Disorder and Disruptive Behavior Disorder. In these cases, problem behavior may be occasioned and reinforced by events mediated by others (usually teachers and parents), or by sensory stimulation that the behavior produces. An example of such would be a cognitively impaired child who engages in aggressive acts at school in order to escape academic demands, without any concomitant psychopathology such as ADHD, anxiety or a mood disorder. For cases where problem behavior is primarily under operant control, the recommended first line intervention is behavioral, although the clinician should remain vigilant for any emergent psychiatric disturbance requiring pharmacological treatment as well.

Between these two poles of the spectrum, we further recognize myriad degrees of mixed psychiatric and operant behavioral presentations. Some individuals may display problem behavior that is *both* environmentally caused *and related to*, or perhaps exacerbated by, a psychiatric disorder. For example, functional behavioral assessment results may reveal that problem behavior is occasioned by the presentation of demands, and maintained by escape from demands, although this "socially mediated" problem behavior may be exacerbated *at the same time* by inattention and hyperactivity due to ADHD. In such cases, coordinated treatment with both behavioral interventions and psychotropic medications will likely prove most fruitful.

Similarly, dual interventions are usually necessary for cases of a fourth behavioral gradation, where it becomes evident that *some* problem behaviors are environmentally caused, while there is also a *concomitant* psychiatric disorder causing *other*, separate problems requiring treatment. For example, an individual with ID may display self-injury in order to gain access to adult attention. He may concurrently have an affective disorder that does not affect the rate or intensity of self-injury, yet causes significant neurovegetative symptoms, depressed mood, crying and irritability.

These mixed classes of behaviors, representative of the gradations between primarily psychiatric and primarily operant behaviors, may indeed be quite complex, and often times the presence of two classes of problems is not evident until treatment is underway targeting one class of behavior. We would recommend applying behavioral interventions first when possible, if only for the purpose of further clarifying the related psychiatric disorder. That is, in many cases, the specific nature of the psychiatric problem may become more fully understood once behavioral treatment has been applied and operant variables controlling problem behavior have been eliminated.

As such, the neurobehavioral model affords the

cooperative usage of psychiatric and behavioral explanations for assessment and treatment of a wide range of maladaptive behavioral expression. While it is difficult to buttonhole problem behaviors in exact categories, consideration of the neurobehavioral model's spectrum can be quite informative. It is important to recognize, however, that there is potential for overlap in both understanding of problem behaviors and treatment development. A prime example of such would be the treatment of self-injury or aggression caused by the blocking of compulsive behaviors. A frequently encountered case in patients with ID and autism, the behavioral disturbance may be conceptualized in the middle of the spectrum with both a psychiatric element in the form of an obsessive-compulsive disorder, as well as a behavioral element related to the interruption response from the environment. Such a patient might be successfully treated pharmacologically with a serotonin-enhancing agent. Success may also arise from behavioral treatment involving interventions such as teaching the individual to discriminate the conditions under which the response is, and is not allowed. In such cases, either single intervention may be successful, yet both interventions may actually complement one another, working together for a synergistic treatment effect.

Although classification of individuals' problem behavior in these categories is initially based upon behavioral and psychiatric assessment data, the assessment process should, of course, be continuous and refined as necessary in the course of treatment. Thus, unsuccessful interventions often elucidate the determinants of problem behavior, and lead to reclassification. Treatment outcomes either confirm these hypotheses (in the case of successful outcomes) or provide additional data to refine and revise hypotheses (in the case of failed outcomes) — which in turn are tested as treatments are modified.

Case Series

The following three cases are presented to demonstrate the usage of the neurobehavioral model in the assessment and treatment of individuals with intellectual disability of various etiologies and concomitant behavioral disturbance.

1) J. was a 14-year-old female with Cornelia de Lange syndrome and Moderate Mental Retardation who was transferred to our inpatient program after a three-month psychiatric hospitalization for self-injury and aggression. Aggressions included biting, hitting, kicking and pouncing, with faces being the preferred target. Aggressions were largely random, although J. frequently warned people to stay away from her. J. had isolated herself in her room, stating that she feared hurting others, and becoming agitated when anyone approached her. Self-injury included biting and scratching her own hands to draw blood, and then smearing such on the walls. She also engaged in disruptive behaviors, including smashing computers and other small appliances onto the floor. J. reported that she heard voices, specifically "a man, a woman and Levi" who told her to hit others. Reportedly, she would also talk to Levi while alone. She was admitted on valproic acid 375mg bid with a serum level of 59 and gabapentin 400mg bid.

J. had a significant past psychiatric history, with aggression and self-injury worsening since age eight. She had been hospitalized for suicidal comments while taking paroxetine and venlafaxine two years prior to admission, and again for aggression and self-injury one year later. A fluoxetine trial had caused behavioral disinhibition. Risperidone and olanzapine led to marked extrapyramidal symptoms, and aripiprazole resulted in worsening behavioral symptoms, as well as an elevated creatinine phosphokinase level and concern for neuroleptic malignant syndrome. Clonidine had been effective for a short period of time only.

Once admitted, J. demonstrated significant irritability with whining, aggression as well as odd behaviors, including strange comments regarding "ladies and gentlemen" and conversations with Levi. J. reported that the "ladies and gentlemen" told her to hit patients and staff, and that they had threatened to kill her if she didn't obey. Interestingly, J. demonstrated no outward distress from the presence of the ladies and gentlemen, and would giggle and seem to frankly enjoy discussing them with anyone interested in listening. She was never observed responding to internal stimuli, and displayed no other signs of psychosis. Her parents ultimately shared that Levi was a young boy from J.'s school who refused a

playdate with J., causing her much disappointment. As the admission advanced, J. would immediately begin talking about the ladies and gentlemen as soon as she spotted the psychiatrist. Her other preferred topics of conversation included excrement and menstruation. While the content of her conversation was inappropriate and bizarre at the time, the form of her thoughts and conversations were quite coherent and organized. The treatment team began to suspect that these presumed psychotic symptoms and odd comments were actually a manifestation of poor social skills, and noted that J. did not demonstrate such at all when engaged in appropriate personal interactions.

Depakote was maximized, but discontinued due to lack of response in terms of aggression and irritability. Aggression persisted, and gabapentin was increased to target aggression, irritability and anxiety. Although gabapentin did confer therapeutic benefit, some symptoms persisted, and clonidine was retried as an augmentation strategy, with further reduction in anxiety and aggression. A brief trial of escitalopram was pursued to target irritability, but rapidly discontinued as J. developed severe behavioral disinhibition. J. was discharged on gabapentin 1100mg tid and clonidine 0.1mg tid with excellent overall reduction in anxiety, irritability and problem behaviors.

Behavioral assessments of verbalizations revealed that J. displayed more appropriate verbalizations and near-zero levels of bizarre verbalizations when she was engaged in conversation on topics of her choosing relative to when therapists either ignored bizarre verbalizations or when they provided her little attention. Assessments also indicated that verbal threats and problem behavior occurred to terminate or avoid close proximity to therapists. Collectively, the assessment findings supported the hypothesis that the bizarre verbalizations were a function of social/conversational skills deficits rather than psychosis, and that aggression and verbal threats were a function of social anxiety and used to maintain or avoid close proximity. These findings led to the development of an intervention that included providing J. a means to appropriately terminate or avoid close interaction by saying "no thank you" to therapists' requests, and reinforcement for voluntarily progressively sitting closer to therapists during con-

versation. Social skills training focused on teaching J. basic conversational and interaction skills. The combined behavioral/pharmacological intervention resulted in a 71% reduction in aggression relative to baseline; however, it should be noted that levels of aggression were artificially low during baseline because she did not permit staff to approach her during baseline.

2) S. was a 9-year-old male with Autism, Moderate-Severe Mental Retardation and Mood Disorder Not Otherwise Specified who was admitted to the Neurobehavioral Unit for multiple aggressive, self-injurious, disruptive and socially inappropriate behaviors which had been worsening over the past four years. Aggression consisted of biting, hitting, kicking, punching, scratching and slapping. Self-injury included head-banging, self-biting and scratching. Disruption ranged from crying, dropping, screaming, swiping and throwing items. S. also had significant mood lability and sleep disturbance, with total sleep averaging only 4–5 hours nightly. S. had inflicted multiple wounds upon himself, family members and school staff, had put his head through sheetrock walls, and broken multiple windows and furniture. He frequently needed to be isolated at home in a room with padded walls and plexiglass windows to minimize injury.

S.'s medical history was non-contributory and karyotype was normal. Previous medication trials had included paroxetine, valproic acid, carbamazepine, quetiapine and clonidine, all of which had either caused intolerable side effects or afforded no behavioral benefit. Admission medications included haloperidol 10mg daily and benztropine 5mg daily.

Baseline behavioral assessment was undertaken at admission while S. remained on admission dosage of haloperidol. Twenty-four hour daily assessment revealed baseline rates of aggression, self-injury and disruption averaging 417/day, 9/day and 64/day, respectively. As behaviors persisted at these elevated rates despite haloperidol administration, a decision was made to wean haloperidol. During the course of the haloperidol wean, ongoing daily psychiatric and 24-hour behavioral assessment continued, and revealed a marked cyclical pattern to both mood and overall behavioral levels, in addition to ongoing sleep

disturbance. Lithium carbonate was begun and gradually maximized to a serum level of 1.1. Significantly, addition of lithium led to a moderate decrease in aggression and other negative behaviors, and overall dampening of intensity of mood and behavioral cyclicity. However, a less intense cyclical pattern persisted, and a second mood stabilizer was added. Valproic acid was titrated to a final serum level of 85, with significant reduction in combined inappropriate behaviors and stabilization of mood. Haloperidol was retained at a dosage of 1.5mg daily to address residual aggression that persisted beyond the implementation of the following behavioral protocol.

Behavioral assessment findings revealed that problem behavior was occasioned by the presentation of academic demands and demands to transition from preferred activities, and maintained escape from these demands. The intensity and rate of problem behavior varied in a cyclical fashion, and mood lability was correlated with increased probability of problem behavior in demand and transition situations. Behavioral treatment focused on establishing alternative means to access escape via communication training, building tolerance to work and transition, reinforcement of compliance, and a time-out procedure. While pharmacological interventions resulted in overall stabilization of mood and overall level of behavior, further reductions were evident with the implementation of the behavior program. Relative to baseline levels of problem behavior (averaging nearly 300 per day), an 86% reduction was observed with the combined behavioral/pharmacological intervention in place.

3) K. was a 13-year-old male from the Middle East who was admitted due to severe aggression, disruption, self-injury and impulsivity subsequent to traumatic brain injury. K. was involved in a motor vehicle accident at age 11, with significant left frontal lobe hemorrhage. After three months of intensive care, he made an excellent physical recovery. However, he began to demonstrate indiscriminate aggression, including kicking, punching and slapping of family members and complete strangers. He also developed disruptive behaviors of throwing appliances, toys, furniture and other small objects. New-onset SIB consisted of finger and lip biting. Finally, K. developed extensive use of profanities, and inappropriate

sexualized behavior. All of his problem behaviors had caused much profound impairment at school, home and in his community, and led his Embassy to send the family for treatment in the U.S.

Prior to his accident, K. was a normally developing, healthy child who was an outstanding student and horseback rider who had never demonstrated any behavioral or psychiatric disturbance. Risperidone had led to sedation, sialorrhea, akathisia, dysarthria and "nervousness" at low dosages, with only a temporary reduction in aggression. A methlyphenidate trial had also been ineffective. Formal behavioral analysis had not taken place. K. was without any psychotropic medication at time of admission.

On the neurobehavioral unit, K. demonstrated aggressive, self-injurious and disruptive behaviors from the first day. During the first week, his hourly rates for aggression, self-injury and disruption were 3.67/hr, 0.29/hr and 0.64/hr, respectively. He also exhibited an ongoing lack of behavioral inhibition associated with his problem behaviors, as well as difficulties with attention, memory, motivation and executive functioning. His presentation was wholly consistent with frontal lobe injury, or executive dyscontrol syndrome. Fortunately, K. did not demonstrate any symptoms of affective, anxious or psychotic disturbance as sequelae of his brain injury. These parameters were tracked through daily behavioral data collection along with his negative behaviors. Functional behavioral assessment findings indicated that his aggressive and disruptive behaviors were maintained in part by access to adult attention and to escape demands. In order to address the attention-maintained component of K.'s aggression and disruption, functional communication training was taught to show K. how to appropriately ask for adult attention by saying "excuse me." A "wait" card was added to signal times when attention was not immediately available, and a graduated training procedure (delay to reinforcement fading) was undertaken to teach K. to tolerate waiting as long as 9.5 minutes before receiving adult attention — while maintaining low levels of problem behavior. In order to address problem behavior maintained by escape from demands, an intervention that involved using tokens for compliance with demands was introduced. Once he earned 15 tokens, he was then able to

exchange his tokens for a break from work. This treatment intervention result in behavioral reduction with compliance increased to nearly 100%. A chair time-out was later added to his demands treatment package to further decrease behaviors.

Psychotropic medications were not initiated while these behavioral interventions were being developed. Indeed, it appeared that K. had an excellent response to the treatment protocols. However, he did persist in having residual episodes of aggression and self-injury. At this juncture an amantadine hydrochloride trial was pursued to address such as well as his underlying attentional and executive functioning deficits related to the traumatic brain injury. The addition of amantadine, which was titrated up to a dosage of 50mg bid, ultimately led to a final reduction in combined inappropriate behaviors (aggression, self-injury, disruption) of 84% from baseline.

Discussion

The above cases demonstrate the efficacy of the coordinated efforts of psychiatry and behavioral analysis in the diagnostic clarification and treatment of severe problem behaviors in three children with different forms of developmental disability. The first goal in working with each child was to develop an accurate understanding of the nature of the problem behaviors. Psychiatric and behavioral assessment performed together over the course of several weeks allowed for an in-depth exploration of both environmental and psychiatric potential causes for the presenting behaviors.

While these three children were all assessed on an inpatient basis with data collected 24 hours daily, it is important to remember that these individuals presented with *severe* behavioral disturbance. The same techniques can certainly be used in a less intensive fashion for patients whose behaviors are likewise less extreme. The critical element to this approach is the collaboration between disciplines and an understanding of the import of detecting and appropriately targeting either operant factors and/or psychiatric pathology.

J. is an example of a patient with mixed and interrelated psychiatric and operant-based behavior. Her problem behavior was related to social anxiety and exacerbated by irritability. In this case, behavioral intervention targeting social skills deficits and graduated exposure therapy targeting social anxiety may not have been as effective if the pharmacological interventions were not in place for anxiety and irritability. Likewise, the pharmacological interventions may not have been sufficient to address the skills deficits that were contributing to the anxiety.

S. is another example of a mixed psychiatric and operant case in the middle of the neurobehavioral spectrum. Behavioral data collection on levels of problem behavior, mood, and sleep led to the discovery of a frank cyclical pattern, with responded classically to the serial addition of two mood stabilizers. Such biological variability likely would not have been adequately managed behaviorally, but its eradication allowed for subsequent focus on concomitant operant functions.

K. exemplifies a case with distinct, yet contemporaneous, psychiatric and operant components. That is, there was a separate, underlying biological condition, namely executive dyscontrol syndrome due to known traumatic frontal lobe injury — and a clear operant component to his problem behavior. Detection of the former led to the initiation of amantadine hydrochloride, a drug with proven efficacy in this population (44). However, superimposed on the executive dyscontrol syndrome, S. had additional operant functions to some of his behaviors, namely access to attention and escape from demands. Such behaviors would not have been expected to respond to medication alone, but certainly would be amenable to behavioral intervention.

Conclusion

Comprehensive assessment of problem behaviors in ID is not a novel suggestion. The American Academy of Mental Retardation recommends such for all people with ID and behavioral disturbance (19), and experts in the field of dual diagnosis urge the combined usage of behavioral and psychiatric assessment in diagnosis and treatment of problem behaviors, as well as further research in this area (45).

We believe that our neurobehavioral model expands upon such, and can be successfully used in the assessment and treatment of many topographies of problem behaviors in ID. Implementation of this

model by mental health professionals in ID should offer individuals with problem behaviors significant potential for overcoming their behaviors and pursuing healthy and fulfilling lives.

References

1. Paclawskyj TR, Kurtz PF, O'Connor JT. Functional assessment of problem behaviors in adults with mental retardation. Behav Modif 2004;28:649–667.

2. Borthwick-Duffy S. Epidemiology and prevalence of psychopathology in people with mental retardation. J Consult Clin Psychol 1994;62:17–27.

3. Eyman RK, Borthwick-Duffy SA, Miller C. Trends in maladaptive behavior of mentally retarded persons placed in community and institutional settings. Am J Ment Defic 1981;85:473–477.

4. Borthwick-Duffy SA, Lane KL, Widaman KF. Measuring problem behaviors in children with mental retardation. Res Dev Disabil 1997;18:415–433.

5. McIntyre LL, Blacher J, Baker BL. Behaviour/mental health problems in young adults with intellectual disability: The impact on families. J Intell Disabil Res 2002;46:239–249.

6. Matson JL, Sevin JA. Theories of dual diagnosis in mental retardation. J Consult Clin Psychol 1994;62:6–16.

7. MacLean WE. Overview. In Matson JL, Barrett RP, editors. Psychopathology of the mentally retarded, Second Edition. Massachussetts: Allyn and Bacon, 1993.

8. Rutter M, Tizard J, Yule W, Graham P, Whitmore K. Research report: Isle of Wight Studies, 1964–1974. Psychol Med 1976;6:313–332.

9. American Psychiatric Association. Diagnostic Criteria from the DSM-IV-TR. Washington, DC, 2000.

10. Matson JL, Kazdin AE, Senatore V. Psychometric properties of the psychopathology instrument for mentally retarded adults. Appl Res Ment Retard 1984;5:81–89.

11. Reiss S, Valenti-Hein D. Development of a psychopathology rating scale for children with mental retardation. J Consult Clin Psychol 1994;62:28–33.

12. Harris JC. Developmental neuropsychiatry, Volume II: Assessment, diagnosis and treatment of developmental disorders. New York: Oxford University, 1998.

13. Schroeder SR, Oster-Granite ML, Berkson G, Bodfish JW, Breese GR, Cataldo MF, Cook EH, Crnic LS, DeLeon I, Fisher W, Harris JC, Horner RH, Iwata B, Jinnah HA, King BH, Lauder JM, Lewis MH, Newell K, Nyhan WL, Rojahn J, Sackett GP, Sandman C, Symons F, Tessel RE, Thompson T, Wong DF. Self-injurious behavior: Gene-brain-behavior relationships. Ment Retard Dev Disabil Res Rev 2001;17:3–12.

14. Wong DF, Harris JC, Naidu K, Yokoi F, Marenco S, Dannals RF, Ravert HT, Yaster M, Evans A, Rousset O, Bryan RN, Gjedde A, Kuhar MJ, Breese GR. Dopamine transporters are markedly reduced in Lesch-Nyhan disease *in vivo*. Proc Natl Acad Sci 1996;93:5539–5543.

15. MacIntyre DJ, Blackwood DHR, Porteous DJ, Pickard BS, Muir WJ. Chromosomal abnormalities and mental illness. Mol Psych 2003;8:275–287.

16. McClintock K, Hall S, Oliver C. Risk markers associated with challenging behaviours in people with intellectual disabilities: A meta-analytic study. J Intell Disabil Res 2003;47:405–416.

17. Sloman KN, Vollmer TR, Cotnoir NM, Borrero CSW, Borrero JC, Smaha AL, St Peter CC. Descriptive analyses of caregiver reprimands. J Appl Behav Anal 2005; 38:373–383.

18. Iwata BA, Pace GM, Dorsey MF, Zarcone JR, Volmmer TR, Smith RG. The functions of self-injurious behavior; an experimental-epidemiological analysis. J Appl Behav Anal 1994;27:215–240.

19. American Association of Mental Retardation. Treatment of psychiatric and behavioral problems in mental retardation 2000;105:165–188.

20. Baumeister AA, Sevin JA. Pharmacologic control of aberrant behavior in the mentally retarded: Toward a more rational approach. Neurosci and Biobehav Rev 1989;14: 253–262.

21. Matson JL, Bamburg JW, Mayville EA, Pinkston J, Bielecki J, Kuhn DE, Smalls Y, Logan JR. Psychopharmacology and mental retardation: A 10-year review (1990–1999). Res Dev Disabil 2000;21: 263–296.

22. Aman MG, Collier-Crespin A, Lindsay RL. Pharmacotherapy of disorders in mental retardation. Eur Child Adolesc Psych 2000;9:I98-I107.

23. Friedlander R, Lazar S, Klancnik J. Atypical antipsychotic use in treating adolescents and young adults with developmental disabilities. Can J Psych 2001;46:741–745.

24. LaMalfa G, Lassi S, Bertelli M, Castellani A. Reviewing the use of antipsychotic drugs in people with intellectual disability. Hum Psychopharm 2006;21:73–89.

25. Masi G. Pharmacotherapy of pervasive developmental disorders in children and adolescents. CNS Drugs 2004; 18: 1031–1052.

26. Janowsky DS, Barnhill LJ, Shetty M, Davis JM. Minimally effective doses of conventional antipsychotic medications used to treat aggression, self-injurious and destructive behaviors in mentally retarded adults. J Clin Psychopharmacol 2005;25:19–25.

27. McDougle CJ, Scahill L, Aman MG, McCracken JT, Tierney E, Davies M, Arnold LE, Posey DJ, Martin A, Ghuman JK, Shah B, Chuang SZ, Swiezy NB, Gonzalez NM, Hollway J, Koenig K, McGough JJ, Ritz L, Vitiello B. Risperidone for the core symptoms of autism: Results from the study by the autism network of the research units on pediatric psychopharmacology. Am J Psychiatry 2005;162:1142–1148.

28. Janowsky DA, Shetty M, Barnhill J, Elamir B, Davis JM. Serotonergic antidepressant effects on aggressive, self-injurious and destructive/disruptive behaviors in intellectually disabled adults: A retrospective, open-label, naturalistic trial. Intl J Neuropsychopharm 2005;8:37–48.

29. Symons FJ, Thompson A, Rodriguez MC. Self-injurious behavior & the efficacy of naltrexone treatment: A quantitative synthesis. Ment Retard Dev Disabil Res Rev 2004;10:193–200.

30. Buitelaar JK, Willemsen-Swinkels SHN. Medication treatment in subjects with autistic spectrum disorders. Eur Child Adolesc Psych 2000;9:I85-I97.

31. Kowatch RA, Bucci JP. Child and adolescent psychopharmacology: Mood stabilizers and anticonvulsants. Pediatr Clin Nort Am 1998;45:1173–1186.

32. Ruedrich SL, Grush L, Wilson J. Beta adrenergic blocking medications for aggressive or self-injurious mentally retarded persons. Am J Ment Retard 1990;95:110–119.

33. Kahng S, Iwata BA, Lewin A. Behavioral treatment of self-injury, 1964–2000. Am J Ment Retard 2002;107:212–221.

34. DeLeon IG, Fisher WW, Herman KM, Crosland KC. Assessment of a response bias for aggression over functionally equivalent appropriate behavior. J Appl Behav Anal 2000;33:73–77.

35. Hagopian LP, Adelinis JD. Response blocking with and without redirection for the treatment of pica. J Appl Behav Anal 2001;34:527–530.

36. Iwata BA, Dorsey MF, Slifer KJ, Bauman KE, Richman GS. Toward a functional analysis of self-injury. J Appl Behav Anal 1994;27:197–209.

37. Iwata BA, Pace GM, Dorsey Mf, Zarcone Jr, Volmmer TR, Smith RG. The functions of self-injurious behaviors; an experiemental-epidemiological analysis. J Appl Behav Anal 1994;27:215–240.

38. Hanley GP, Iwata BA, McCord BE. Functional analysis of problem behavior: A review. J Appl Behav Anal 2003;36:147–185.

39. Fisher WW, Piazza CC, Bowman LG, Hagopian LP, Owens JC, Slevin I. A comparison of two approaches for identifying reinforcers for persons with severe and profound disabilities. J Appl Behav 1992;25:491–498.

40. Hagopian LP, Crockett JL, VanStone M, De Leon IG, Bowman LG. Effects of noncontingent reinforcement on problem behavior and stimulus engagement: The role of satiation, extinction and alternative reinforcement. J Appl Behav Anal 2000;33:433–450.

41. Hagopian LP, Fisher WW, Thibault-Sullivan M, Acquisto J, LeBlanc LA. Effectiveness of functional communication training with and without extinction and punishment: A summary of 21 inpatient cases. J Appl Behav Anal 1998;31:211–235.

42. Kodak T, Miltenberger RG, Romaniuk C. The effects of differential negative reinforcement of other behavior and noncontingent escape on compliance. J Appl Behav Anal 2003;36:379–382.

43. Shore BA, Iwata BA, DeLeon IG, Kahng S, Smith RG. An analysis of reinforcer substitutability using object manipulation and self-injury as competing responses. J Appl Behav Anal 1997;30:21–40.

44. Kraus MF, Maki PM. Effect of amantadine hydrochloride on symptoms of frontal lobe dysfunction in brain injury: Case studies and review. J Neuropsychiatry Clin Neurosci 1997;9:222–230.

45. Tuinier S, Verhoeven WMA. Psychiatry and mental retardation: Towards a behavioural pharmacological concept. J Intellect Disabil Res 1993;37:16–25.