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#### Gambling in Las Vegas: The Children Win by James B. Hale, PhD, ABPdN

As EDDA Chair, I never thought I would have such a wonderful opportunity to work and think alongside some of the most accomplished scholars and practitioners in the world. With the convergence of so much talent at the EDDA symposium and roundtable in Las Vegas last month, it was an opportunity of a lifetime, offering me a chance to put names to faces, words to voices, and ideas to personalities.

As the day progressed it became apparent that AAPN President Dr. Steve Hughes' vision for EDDA was becoming a reality, and I felt honoured to be a part of it. After listening to the players involved, both during the formal symposium talks, the informal roundtable discussion, and across the table over dinner, it became clear that we had amassed an incredible pool of talent in EDDA. Dr. Hughes' vision was becoming a reality, one that could change what we do in serving children with disorders of attention for years to come.

With each talk and position offered to the packed room, one clear consensus emerged from participants. Both scholars and practitioners alike recognized how current diagnostic and treatment practices were limited when behavioural approaches alone were used to address the many neurobiological disorders that affect attention. We acknowledged that these practices could be enhanced if those affected by attention disorders, their families and loved ones, the professionals that served them, and even the general public were made more aware of the neuroscience of attention and the neuropsychology of attention disorders. We gained commitment to bring the pediatric neuropsychology and neuroscience of attention and its disorders to the forefront of public awareness and policy, but simultaneously recognized that this EDDA project was merely a small step in what could become a much larger movement accomplished by many people in many different ways.

As we gained consensus, we simultaneously recognized how daunting a task this might be. How does one change ideas and practices so firmly entrenched in society? Although we subsequently talked of ideals, we were simultaneously tempered by the pragmatics of the situation. We acknowledged that no one person could do this, not even EDDA alone could do what needed to happen – that accomplishing this feat would require effort by many people at many levels. Those with the patience, fortitude, and determination to make change a reality would help lead the way for others.

We recognized that in many ways, we had already accomplished an important first step – recognizing that change was needed for the benefit of the children with disorders of attention. We took comfort in recognizing our common ground. By spreading the

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Chair's Column

## Las Vegas 2012 EDDA Symposium

In a big room where more tables and chairs were needed to accommodate the large audience, the EDDA symposium began with an introduction by Dr. Peter Entwistle, Pearson Assessment, sponsor of the EDDA Symposium/Roundtable. Dr. Entwistle, an accomplished professional knowledgeable in measurement of attention and its disorders, highlighted how EDDA could help guide assessment and intervention practices in the years to come.

In the first talk, Dr. James B. Hale (Brad) provided the audience with an overview of the significant issues facing EDDA and the larger professional community, and then concluded with EDDA Practice Survey results. One of the first points raised in this talk was the difficulty in separating basic attention from executive control of attention. This foundation framed the following description of the frontal-subcortical circuits, and how they influence one another in modulating attention. In presenting his Balance Theory, Dr. Hale noted how different circuit patterns could lead to different types of psychopathology. Recognizing this research was still in its infancy, Dr. Hale pointed to the need for further research in

- Genetic determinants and epigenetic influences on disorder expression
- Neurotransmitter actions and interactions (excitatory and inhibitory)
- Structural determinants (grey and white matter density/volumes)
- Functional determinants(circuit hypo/hyperactivity, white matter connectivity)
- Electrical activity and intervention response (QEEG)

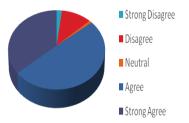
Of particular interest in this talk was the circuit interactions, and how subcortical structures (e.g., caudate, putamen, globus pallidus, thalamus, nucleus accumbens, cerebellum, locus coerelleus) influenced the dorsolateral, orbital, cingulate, oculomotor, and motor circuits. Finally, Dr. Hale argued these research efforts were not only important to consider from a basic science perspective, but results also had to lead to meaningful changes in diagnostic and treatment practices if they were to have substantive long-term impact on the field.

The EDDA Practice Survey results highlighted these issues for the audience, with Dr. Hale suggesting results provided the impetus for EDDA's continued efforts. Of the 92 EDDA Members, 72 completed the survey, with many others indicating they felt they should not respond as they were primarily researchers. Demographic data showed EDDA is an accomplished group, with 4610 professional presentations, 917 peer-reviewed journal articles, 313 scholarly chapters, and 47 published books amassed by participants. Results suggested that current practices were limited in many ways. Respondents felt that there was a greater need for neuropsychological testing of children with attention disorders, especially when determining whether a child had a primary attention problem, or attention problem secondary to another disorder. When asked how best to represent attention disorders diagnostically, a majority of respondents felt that multiple diagnostic labels were preferred over a single diagnostic label, single diagnostic labels with subtypes, or no diagnostic labels. Additionally, respondents felt that we needed to define these disabilities for clinical and research purposes based on neuropsychological - not behavioural criteria. This is in contrast with current diagnostic practices.

EDDA Chair Dr. Hale noted research is needed in:

- 1) Genetic and epigenetic influences on disorder expression
- 2) Neurotransmitter actions and interactions
- Structural and functional determinants of attention and attention disorders

EDDA Members Prefer Defining Research Groups Using Neuropsychological Criteria\*



To support this position, Dr. Hale presented some of his own data from the data set published in Hale et al. (2011). This data was consistent with prior research in that there were weak correlations between behavioural criteria and neuropsychological measures. However, what Dr. Hale showed (see Table 1) is that the baseline neuropsychological factors were more related to medication response than informant-reported DSM-IV criteria. Consistent with these data, EDDA members preferred a neuropsychological – not behavioural – diagnosis for research purposes.

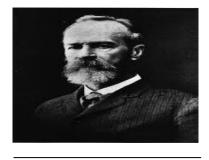
Measure	Cognitive Medication Response $r(r^2)$	Behavioural Medication Response r (r <sup>2</sup> )
DSM-IV Inattention Ratings (Parent Report)	.09 (.008)	.03 (.000)
DSM-IV Hyperactivity- Impulsivity Ratings (Parent Report	.30* (.090)	.25 (.063)
Dorsolateral-Dorsal Cingulate "Cool" Circuit Functions Factor	.44** (.194)	.33* (.109)
Orbital-Ventral Cingulate "Hot" Circuit Functions Factor	.45** (.203)	.31* (.097)

# Neuroscience Domain Presentation by Dr. Frutiger

Dr. Frutiger began her talk with noting the wide gap between social science and neuroscience research in our understanding of attention and its disorders. She noted that techniques used to build functional models are behavioural, which limits our ability to generalize basic neuroscience findings to behavioural descriptors. A major goal of the Neuroscience Domain was to develop a way to make neuroscience research clinically useful.

Can correlational/multivariate techniques permit statements of causation or identification of specific neuroanatomical mechanisms associated with various clinical constructs? If we look at our data from a different perspective, will we see something different? What are the methodological differences between experimental neuroscience research and correlational clinical research, and how does this affect conclusions drawn from each? Dr. Frutiger indicated that in order to move forward, the communication gap between neuroscience and clinical practice needs to be closed.

The deficit model assumes that frontostriatal dysfunction is linked to ADHD based on anatomical studies showing decreased volumes of the prefrontal cortex, caudate, and globus pallidus, and neuroimaging studies showing underactivation of brain areas associated with response inhibition tasks. Further, the proposal of dopaminergic dysfunction is based on the efficacy of psychostimulant medication, specifically at the level of the striatum.



"Everyone knows what attention is. It is the taking possession by the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. Focalization, concentration. of consciousness are of its essence. It implies withdrawal from some things in order to deal effectively with others, and is a condition which has a real opposite in the confused. dazed. scatterbrained state." William James, Principles of Psychology (1890)

However, limitations of making neuroscience research relevant for clinical practice include:

- Biased ascertainment many studies use adult samples, more child studies needed
- Structural and functional differences reflect a posteriori conclusions, not hypothesis driven and falsifiable research
- Studies rarely incorporate developmental and plasticity considerations
- Assuming neurotransmitter abnormalities underlie ADHD when medications affect both ADHD and typical samples in similar ways

Neuroscience models suggest that several systems may operate in parallel to direct attention to relevant stimuli, while concurrently monitoring the environment for meaningful but anticipated low probability events. We know that complex mental functions are not localized to specific brain regions, but rather that they exist as distributed systems, each of which plays different roles within the functional system. Dr. Frutiger highlighted how the principles of multiple determinism, nonadditive determinism, and reciprocal determinism limit simple explanations for complex human behaviors. Can we develop a more dimensional approach to understanding variance in the regulation of attention, impulsivity, and activity level in undiagnosed children, or children diagnosed with disorders that have overlapping phenotypic manifestations. The development of models based on neural system function and developmental principles could help lead to the development of neuropsychological tests that are both sensitive and specific to explaining the heterogeneity that exists in children diagnosed with ADHD. Test development directly linked to our current knowledge of neurobiological systems can lead to improved psychotherapeutic and cognitive intervention methods.

# **Etiology Domain Presentation by Dr. Bailey**

Before looking at the etiology of ADHD, Dr. Bailey discussed the importance of definitional issues. Given that ADHD is currently a behaviourally-defined disorder, we rely on DSM-IV criteria for determining the presence of this disorder. However, these criteria are not concerned with etiology, thus limiting our understanding of the interrelationship between the two. In order to define etiology empirically, we need to pay close attention to what we are defining (e.g., stimulus orientation, preference, divided, sustain, update/shift, etc). Differences between scientists and social critics fuel the controversy as well, when clearly brain-behaviour relationships are multi-determined. Finally, there are numerous suggested risk factors, including environmental, prenatal, biological, and multi-dimensional factors that may cause attention and/or executive problems.

Dr. Bailey reviewed a new meta-analyses conducted by Willcutt, Nigg et al. (2012), which has not been published as of press time. However, interesting findings suggest the symptom dimensions of inattention, hyperactivity, and impulsivity are valid, but that diagnostically, children vary on their symptom manifestations (or raters differ in their perception of symptom dimensions). Dr. Bailey reviewed the structural and functional determinants of different neuropsychiatric disorders associated with disruptions of attention (e.g., ADHD, anxiety, depression) for the structures most often implicated. Size/volume is not the only factor to consider, because while many frontal regions show thinner cortical tissue, others (e.g., cingulate, medial orbital) show increased cortical thickness.

Dr. Bailey then discussed gene-environment interactions, and how epigenesis is helping us understand the complexity of these relationships. Using Crosbie et al. (2008) to highlight how genetic vulnerabilities are influenced by environmental factors at each level, including proteins, cell networks, structure, physiology, and cognition, Dr. Bailey emphasized that phenotypic expression can vary tremendously. Finally, reviewing the work by Pennington and others, Dr. Bailey recognized the incredibly difficult challenge faced by geneticists when determining genetic etiology, with over 19 genes implicated for attention, and 20 genes for hyperactivity/impulsivity, with 17 of these genes showing significant relationships among symptom dimensions. Finally, in recognizing the limitations of the prefrontal explanation of ADHD, Dr. Bailey acknowledged Halperin and Schultz's argument that prefrontal dysfunction is not the cause of ADHD. Instead, they argue that prefrontal maturation actually helps children with ADHD gain control over subcortical dysregulation, and this leads to improved attention and executive function as a result. Dr. Bailey suggested further examination of quantifiable endophenotypes of ADHD, which could begin to clarify the contradictory results often reported in the literature.

### **Diagnosis Domain Presentation by Dr. Wasserman**

As neuropsychologists, Dr. Wasserman pointed out that we have a very important role to play when it comes to diagnosing attention and related disorders. However, neuropsychologists have marginalized themselves as a field by attempting to map physiologically-based processes onto behavioural diagnoses. Willing to accept behavioural diagnostic nomenclature, we essentially have limited the utility of neuropsychological assessment for differential diagnosis as a result. Many disorders share a number of similar cognitive and neuropsychological constructs, including underlying regulatory mechanisms and executive management of behaviour, so we should think of attention as a component of the executive regulatory system that works in conjunction with various other processes to produce volitional behaviour. Because there are multiple causes of behaviourallydetermined ADHD, the result is weak effect sizes and limited discriminate validity of the measures.

Reviewing several meta-analyses of attention and executive function, results suggest that:

- ADHD is associated with significant weaknesses in several key executive function domains
- The strongest, most consistent, effect sizes are obtained for neuropsychological measures of response inhibition, vigilance, working memory, and planning
- Moderate effect sizes and a lack of universality of EF deficits in ADHD suggests that they are neither necessary nor sufficient for all behaviourally-diagnosed ADHD
- EF weaknesses have been found to be significantly associated with almost all forms of emotional and behavioral difficulties including depression, obsessive-compulsive disorder, autism spectrum disorders, anxiety, and even reading disorders

So the question neuropsychologists have to ask themselves is whether there is only one pattern of executive dysfunction conforming to something that represents a subtype of individuals that we currently diagnose behaviourally as ADHD, or do the same executive skills interact with various variables to produce observable clinically relevant behaviour characteristic of various disorders. In order to move forward, Dr. Wasserman suggested researchers and practitioners alike need to identify patterns among current instruments, and construct new instruments, that can reveal different patterns of frontal-subcortical circuit dysfunction among the different disorders. If there are unique patterns of executive dysfunction that characterize particular disorders, the field will advance considerably.

While the extant data suggests that unique executive patterns will not be sufficient in predicting the occurrence of a specific disorder such as ADHD, Dr. Wasserman suggested recognizing the executive patterns might help us understand explained variance in a meaningful way that can foster differential diagnosis of ADHD and other disorders affecting attention. Perhaps an important first step would be in examining regulation of various systems rather than just concentrating on one heterogeneous executive variable such as attention. In addition, the discriminate validity problem is exacerbated by behavioural diagnostic criteria, as this leads to neuropsychological heterogeneity within the same diagnosis. The question remains whether pinpointing certain executive pattern deficits, and developing homogeneous groups based on those neuropsychological profiles, could lead to a neuropsychological definition that reduces behavioural diagnostic heterogeneity. For that, innovative research programs are necessary to explore this potential resolution to the limited discriminate and treatment validity of our neuropsychological assessment tools.

The Diagnosis Domain asked: these bold questions:

- "Does any pattern of executive dysfunction conform to something that represents individuals with ADHD?
- Are the executive skills which underlie various disorders the same, and interact with as yet poorly understood variables to produce the observed behavioral clinical effect?
- Should we be examining regulation of systems instead of just concentrating on one heterogeneous variable such as attention?"

### Intervention/Treatment Domain Presentation by Drs. Gomes and Riccio

Given the limitations of prior research and inconsistent findings regarding etiology and diagnosis of ADHD and other attention disorders, it is not surprising that intervention effects are also attenuated by the neuropsychological heterogeneity found when behavioural diagnoses are used to define disorders. Drs. Gomes and Riccio first presented the results of their intervention survey. Results indicated that stimulants were the most common treatment (95%), but atomoxetene (63%) was also common, and a surprising 51% noted that anti-depressants were used for their patients, suggesting that mood disorders are commonly recognized as causes of attention problems. Biofeedback has been growing in popularity (18%), but the most common non-medication cognitive treatment was computerized training, with 29% reporting its use. A majority of these individuals were using CogMed (54%) or FastForward (25%). Learning Strategy Instruction (93%) and Behavioural (71%) approaches were of course common, with Parent Training (88%), Cognitive-Behavioral Therapy (55%), and Self-monitoring (48%) most frequently used. A majority (91%) reported using more than one therapeutic approach for children with attention problems.

Following this discussion, an evidence-based report of the intervention literature was offered to the audience. Although effect sizes were strong for stimulant medication (medium to high range) for children with ADHD and Fragile X, they were not as positive for children with autism and attention problems. Atomoxetene effects were also positive for ADHD in children, but not adults or autism. Other medications such as antidepressants also had strong effects, and guanfacine/clonidine were effective in reducing activity level. Unfortunately, rating scales were used to judge treatment effects, so there was little attention paid to their cognitive/neuropsychological impact. No impact of dietary modifications were noted.

Of the alternative treatments, neurofeedback (qEEG) seems to be making inroads into traditional treatments, with as many children with ADHD showing the capability to decrease theta and increase beta waves in anterior regions of the brain. However, there were several limitations of the study also addressed. Not only do metaanalyses suggest medium to strong effects on rating scale outcomes, but these were maintained well after the conclusion of treatment. Cognitive computerized training had variable impact on outcomes, with the most consistent impact on parent (as opposed to teacher) ratings. Neuropsychological outcomes were also variable.

Behavioural interventions were commonly used and showed medium to strong effects across studies. Interestingly, effect sizes were quite variable, with results suggesting:

- Behavioural interventions ranged from .75 to .79 ES for teacher ratings
- Behavioural interventions ranged from .39 to .87 ES for parent ratings
- Parent training was not as strong, with .40 average ES
- Parent training was best for young children, and those whose parents did not have ADHD
- Self-regulation techniques ES ranged from .59 to a strong 2.96.
- There wasn't sufficient evidence to support social skills or metacognitive instruction at this time (although the latter was promising)

Drs. Gomes and Riccio concluded that there was greater need to use randomized controlled trials and look at both direct (e.g., cognitive/neuropsychological) and indirect (rating scale) measures when determining treatment efficacy. To highlight this need, Hanna Kubas presented the results of our RCT work at the University of Victoria. Results suggest first that not all children behaviourally diagnosed with ADHD responded to stimulants. For children with high neuropsychological impairment and Combined Type ADHD, results were quite positive, but for the Inattentive Type and those with low impairment, results were (See Figure 1 on page 9). Even for those who were strong responders, the best dose for cognitive/neuropsychological functioning was lower than the best dose for behaviour. This could explain why children with ADHD don't get better academically, because medication titration was most often based on behaviour.

#### Las Vegas 2012 EDDA Roundtable

After a lunch break, the afternoon Roundtable session began with an inspiring speech by American Academy of Pediatric Neuropsychology President Dr. Steve Hughes. Dr. Hughes recognized the great need for examining disorders of attention from a neuropsychological perspective, noting the problems with sample heterogeneity and attenuated treatment effects that result from behavioural diagnoses. He argued that multiple changes in teaching, research, and service practices were needed to more effectively identify and treat children with attention disorders.

This motivating talk was followed by Dr. Margaret Semrud-Clikeman's (EDDA Co-Chair) discussion of the sticky issue of comorbidity in ADHD and other disorders of attention. Comorbidity not only makes differential diagnosis of ADHD and other disorders difficult, it also speaks to the need for more careful and thorough diagnostic practices, something that requires more clinical attention and careful case management. Dr. Semrud-Clikeman reported on how understanding the neuropsychological bases of these disorders at the etiological, neurological, cognitive, and symptom levels, based on Dr. Pennington's model, could yield insight into the various causes of comorbid disorders such as learning disabilities, conduct disorders, and anxiety/depression. She then provided the audience with an interesting case study that highlighted why this careful multidimensional diagnostic approach is needed to meet the needs of a child with attention problems.

#### Future Directions Domain Presentation and Discussion by Dr. Toplak

The roundtable was led by Dr. Toplak as the representative of the Future Directions Domain. Dr. Toplak noted the urgency of the issues facing the profession, including growing debate among practitioners, scientists, and even the general public about ADHD and disorders of attention. Dr. Toplak used Dr. Sonuga-Barke's examination of heterogeneity in symptoms of behaviourally-diagnosed children with ADHD as a foundation to explore the difficult issues EDDA was meant to address. She pointed out how executive dysfunction was never uniform in the studies conducted to date, with estimates ranging from 33% to 79%, noting that differences might be related to how executive function is defined, and the number of executive measure impairments found for affected children. For instance, Tannock (2011) reported over 83% of children with ADHD had one or more executive deficits, but the number dropped to 48% for children who had 3 or more. This is in contrast to 49% and 6% of controls respectively.

This executive function discriminate validity issue is further complicated by the reality that *most* childhood psychopathologies also have executive deficits, so it is no wonder that many policy makers do not advocate neuropsychological assessment for identification of ADHD and other disorders of attention. Sonuga-Barke (2011) examined the three areas most thought to represent ADHD, inhibitory control (dorsal circuit system), delay sensitivity (ventral circuit system), and temporal processing (cerebral-cerebellar circuit). Dr. Toplak then discussed the proposed changes in the DSM-V, including the introduction of an Inattentive Presentation (hyperactive-impulsive symptoms < 2). Finally, Dr. Toplak asked a poignant question in her examination of the factor structure of ADHD symptoms, executive functions, and even intelligence: Is it best to "clump" or "split" when trying to understand and serve children with ADHD and other disorders of attention.

With this foundation of issues in place, the entire EDDA group moved to the front of the room for the roundtable question and answer period. What followed was a lively debate about how neuropsychology needed to have a "place at the table" with other stakeholders involved in policy and practice for children with ADHD and other disorders of attention.

#### Las Vegas 2012 EDDA Roundtable

The discussion took a developmental focus early on. How can we draw conclusions about developing children, and the effects of medication on the developing child, when we know so little about changes in brain function in clinical populations such as ADHD? This led to the panel talking about epigenesis, and how this environment-individual interaction needed exploration on multiple levels. The panel discussed the need for endophenotypes of ADHD, and also to determine the various environmental relationships (e.g., SES) and individual differences (e.g., sex) that influence either the development or manifestation of ADHD.

Concerns were raised that rating scales were being presented in the popular press by some scientists as having "ecological" validity when in actuality they are merely summative judgements using indirect informant reports. The circularity issue was also discussed, that behaviour ratings were determined to be better diagnostically in part because these summative judgements were first used to diagnose ADHD (e.g., DSM-IV criteria) and subsequently determine the presence of the disorder (e.g., rating scales). Dr. Hale mentioned what if we diagnosed ADHD and other disorders of attention based on neuropsychological test results, and then looked to see if they better determined ADHD than rating scales? He argued that then behaviour ratings would be poor indicators of ADHD and other disorders of attention. Clearly, the panel agreed there was a strong need for further development of neuropsychological and behavioural instruments that have adequate sensitivity, specificity, ecological, and treatment validity.

The discussion then turned to what constructs to consider when framing discussions about attention and executive function. The panel was in agreement that the time has come to start to move beyond the "g" approach to executive function, and instead understand that different executive functions are related to different frontal-striatal and frontal-cerebellar circuits. As we move beyond the "g" level of executive function, we begin to see that different disorders have different neuropsychological deficits, and this can be an important advance for differential diagnosis and targeted interventions for children with ADHD and other disorders affecting attention.

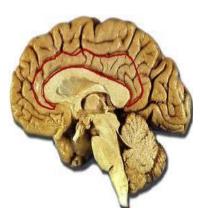
The discussion turned to intervention and treatment of ADHD. While the panel acknowledged the need for empirically-based approaches to intervention, with medication and behavioural interventions most effective, they also realized that sample heterogeneity might limit our current understanding of what it means to be "effective" and could explain why MTA's results were equivocal over time. For example, the idea that treatments might be more effective for one child with ADHD and not another was noted, and led to a discussion of how heterogeneity undermined large group treatment effects. The notion was put forth that if empirical endophenotypes are established, that differential treatment response could then be determined. In addition, the panel made a strong statement that the field of pediatric neuropsychology must evolve to address treatment more effectively, that our days as being only diagnosticians were limited. In other words, effective neuropsychological practice not only required good diagnostic skills, but good treatment ones as well.

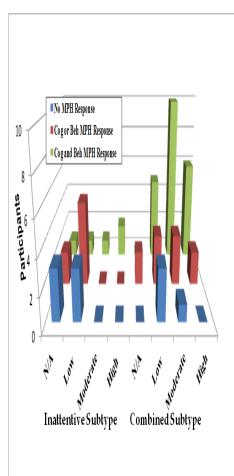
Finally, the roundtable concluded with future clinical and research needs. The panel concluded that while ADHD was one of the most researched disorders, there was substantial need for further empirical evidence and public advocacy efforts to establish the validity of a neuropsychological approach to understanding and serving children with attention problems. We need to think creatively about instruments and research paradigms if we are going to develop assessment techniques that have greater sensitivity, specificity, ecological, and treatment validity. One of the things not considered is time in assessment. We often take these executive "snapshots" of children during neuropsychological evaluations, but in reality executive functions work over time, and evolve with time. As a result, repeated measurement and longitudinal research designs are needed to further our understanding of ADHD and other disorders of attention.

# Las Vegas 2012 EDDA Roundtable

In addition, many in the audience and panel members themselves argued that research is needed at the neuroscience, etiology, diagnosis, and intervention/treatment levels where disorders of attention are defined based on neuropsychological functioning first, and then see how this maps onto behavioural

symptoms/ratings. Although this may be the most difficult and even controversial research need, the panel and audience agreed that if we are to move forward in understanding ADHD and other disorders of attention, we should take a neuropsychological approach for defining these neuropsychological disorders.





# Chair's Column (Continued from page 1)

#### Gambling in Las Vegas: The Children Win by James B. Hale

EDDA message among the leadership and audience, we opened the proverbial research, teaching, and service doors, and left Las Vegas feeling energized that we could all do our small piece to bring about change in the field and larger community.

As you read through this issue of *InFocus*, I hope you will recognize that this is just the first step, and realize that you are an important player in the effort. Each and every member of EDDA, and others who value what EDDA represents, can help by working in their offices and communities, and through Domain co-leaders, to "spread the word" about why the neurobiology of attention and its disorders is important for everyone to consider in our day-to-day professional activities.

The positions taken during the Las Vegas event were not meant to be definitive; they merely set the stage for future growth and development in the evolution of our field. It is appropriate that we should take this gamble for the sake of children with ADHD and other disorders that affect attention.

As we each do our piece, we will foster new ways of thinking, new research programmes, and new practice methods that will serve children with disorders of attention in the years to come. Change will not be quick or easy, but it can be accomplished if we each do a piece of the puzzle, and contribute our slice to the larger EDDA pie. Then we will see that our wager paid off – children with attention problems will be better served.

Finally, Peg and I want to express our sincere gratitude for all those who have contributed to EDDA, particularly Domain Co-leaders, and finally the American Academy of Pediatric Neuropsychology for the opportunity to participate in this effort. We could not have done it without each and every one of you. Thank you, and we look forward to working with you in the future.