Executive Summary
2013 Childhood Apraxia of Speech Research Symposium

Sponsored by the Childhood Apraxia of Speech Association of North America (CASANA)
February 21 – 22, 2013
Emory Conference Center
Atlanta, GA

Introduction

The Childhood Apraxia of Speech Association of North America (CASANA) is a public charity whose mission is to improve the lives of children with apraxia of speech so that each child is afforded his or her best opportunity to develop speech and communication. One goal of the association is to fund and support research development to better understand the underlying nature of CAS, its proper identification, and treatment. In February 2013, CASANA hosted the 2013 Childhood Apraxia of Speech Research Symposium to learn about and discuss "state of the art" research developments in Childhood Apraxia of Speech over the last decade since its first research symposium in 2002.

There were a number of specific objectives for the symposium. First, as one of the only funding sources for CAS specific research and the world's top advocates for children with apraxia of speech, CASANA's board of directors wanted to fully understand the current state of the art in research regarding this population. Secondly, CASANA hoped that by understanding the current state of the art in CAS research it could strategically target and leverage any research funding and support. Finally, CASANA's broader community and constituents need to be apprised of emerging findings (including how current research may impact CAS theories, assessment, and treatment), along with research needs, trends, and ideas for future research.

The symposium brought together top researchers worldwide and other invited scientists and clinicians with CASANA's board and staff, representing parents and families. During the two-day event, there were five topic area presentations and panel discussions that included an overview of current issues faced by families of children with apraxia and four distinct research topic areas, as well as a concluding summation and discussion. A full list of presenters and attendees can be found at the end of this summary. CASANA has made the four research topic area presentations and panel presentations available for viewing on its website Apraxia-KIDS.org. This Executive Summary is intended to provide an annotated overview and documentation of the main ideas and themes shared at the symposium meeting.

Current Trends in CAS from the "Street"

CHAIR: Lawrence Shriberg, Ph.D., CCC-SLP
TOPIC AREA PRESENTER: Sharon Gretz, M.Ed.
PANELIST PRESENTERS: Kathy Jakielski, Ph.D., CCC-SLP, Rebecca McCauley, Ph.D., CCC-SLP, Edythe Strand, Ph.D., CCC-SLP

Sharon Gretz discussed five broad themes that impact the daily lives and concerns of parents and caretakers of children with apraxia. In the area of assessment, the parameters regarding appropriate assessment protocols and diagnosis continue to be a high level need directly impacting families and children. Families find it necessary to go to multiple providers who provide varied responses (yes, no, or maybe) on whether or not the child in question has apraxia of speech. Co-morbid issues continue to blur boundaries for diagnosis for clinicians and often serve to confuse parents as to the primary impact on the child's communication and development.

Next, Ms. Gretz discussed the concerns and questions that families have regarding the underlying cause(s) of their children's speech problems, including those that could possibly be preventable or treatable. It is still unclear what exposures and/or risk factors may lead to CAS. While genetic research has been very exciting to date, families are uncertain if and when to have genetic testing done as well as understanding the meaning of positive findings. Within the CASANA Apraxia Research Registry (a family portal to enter case history information regarding their affected children), the following genetic "differences" have been reported: 8p23.1 duplication; 47XYY: 49, XXXY; Down Syndrome; Ehlers-Danlos Syndrome; Trisomy X (TriploX); xp11.22 to Xp11.23 duplication; 6p abnormality; balanced transfer of Chromosome 11 and 20; MecP2 mutation; 15Q26.3 duplication; 46,XY; 4p14p15.33; and Ring 22.

Families wish to seek help for their children at the earliest possible time. In the CASANA Research Registry, the mean age at which parents reported being concerned about their child's speech was 17 months, with 27% being concerned before the child's first birthday. While 95% of parents report discussing their concern with the child's primary care pediatrician, only 62% were provided with referral to a speech-language pathologist or other professional. The medical community is inconsistent in being responsive to concerns that families have and providing early help, Ms. Gretz suggested.

Treatment issues continue to dominate "life on the street" for families that have children with apraxia. These issues reach both wide and deep and include: Both private insurance and public funding inadequacies; disparate professional knowledge and implementation of appropriate treatment methods; proper guidance for treatment frequency, intensity, and "dose" along with individually tailored "ingredients" and tools within therapy sessions to assure the child's full communication profile is addressed; and increasing interest beyond the realm of speech therapy to the possibilities for neurological and/or biological treatments.

Finally, families are interested in understanding conditions that may co-occur in children diagnosed with CAS, conditions that possibly unfold overtime, and factors that may help in prognosis for fully intelligible speech, each necessitating good longitudinal studies.

In order to highlight the impact of the extent of needs that still exist, Dr. Jakielski provided an overview of the types of questions she receives from clients, parents, and speech-language pathologists in her roles as researcher and designer of clinical services. Intersecting with research activity, the needs of children, parents, & clinicians include their desire to be able to:

- Secure appropriate diagnostic & therapeutic services
- Receive appropriate diagnosis of & intervention for co-occurring disorders
- Locate CAS-specific continuing education & research
- Understand the genetic bases of CAS
- Learn how to work with older children with persistent CAS
- Understand the long-term effects of CAS

Dr. Jakielski summarized that the needs of children affected by CAS, their families, and the clinicians that serve them are varied and they are great.

Dr. Strand identified several needs she has observed in her practice. She confirmed that, as suggested by Sharon Gretz, that it is common for her to see children from all over the country who have been to multiple providers seeking diagnosis. Some clinicians continue to believe or have been told they cannot diagnose CAS because it is a "medical" issue. Additionally, clinicians may not be confident nor well trained in identification and assessment procedures for childhood motor speech disorders. Dr. Strand and Dr. McCauley have published validity data of a new assessment, the "Dynamic Assessment of Motor Speech Skill" (DEMSS).   Dr. Strand recommended that there must be continued work in the area of validating diagnostic markers; determining how behavioral markers may change with age and severity, and devising dynamic assessment tools for moderate to mild CAS and older children.

In the area of treatment, Dr. Strand shared observations that many treatment plans and progress reports she has seen do <u>not</u>:

- Mention the role of motor learning or speech movement variables
- Describe how stimuli are selected
- Mention prosody

All of the above are key features of appropriate treatment programs for CAS; yet do not appear to be included in treatment for affected children. Dr. Strand's work in the area of treatment efficacy research has yielded three published studies demonstrating efficacy in using the Dynamic Tactile and Temporal Cueing (DTTC) method for this population.   She advised that more large scale studies are needed. Overall, in the area of treatment, Dr. Strand suggested that clinicians and researchers continue to add to the efficacy of various treatment methods, compare treatment approaches and study the effects of various methods of selecting treatment stimuli, dosage effects as a function of severity, age, and

comorbidities, and the effects of a number of principles of motor learning as they relate to both age and severity.

Dr. Strand concluded that CASANA could leverage its influence and research dollars by:

- Focusing on efforts to bring together the research that will be discussed throughout the rest of this meeting to clinicians in a way that makes it possible and practical for them to utilize, i.e. "translational research"
- Increasing research funding for the development of better assessment tools and to further investigate treatment efficacy

Dr. McCauley focused her remarks on the gap between research and practice. She suggested that the research to practice gap needs to be addressed in a multitude of ways so that the needs of children and families, as well as professionals who will implement treatments, are met. The diffusion of innovation in diagnostics and treatment and its adoption requires consideration of the relative advantage, compatibility, and complexity of the new methodologies or procedures. Research may be needed to better understand the needs of "end users."

State of the Art in CAS Genomic Research

CHAIRPERSON: Thomas Campbell, Ph.D.
TOPIC AREA PRESENTER: Simon Fisher, Ph.D.
PANELIST PRESENTERS: Barbara Lewis, Ph.D., Beate Peter, Ph.D., Shelley Velleman, Ph.D.

Dr. Fisher began his presentation discussing why genetics is important to CAS research. Identifying specific genomic risk factors could precipitate earlier diagnosis and thus earlier treatment, drive us to develop more novel interventions, and assist us in gaining insights into the causative pathways. In the past few years there have been dramatic advances in various molecular technologies and the tools are very quickly moving from research to the clinic and public domain. Various genetic changes were overviewed including, chromosome rearrangements, duplications, and deletions. Some changes or variations are common in the population and have no observed impact, yet others can be rare and have a significant impact on function.

Insights in the state of genomics in CAS have come from rare genetic cases, with the starting point as the KE family and the discovery of the FOXP2 gene mutation. This mutation results in speech and language problems with CAS as the most significant and core feature. The affected KE family members had difficulty learning and producing sequences of complex movements underlying speech. They also had problems in spoken and written language and receptive language. Subsequent screening showed that most cases of CAS do not have the mutation of FOXP2; however there now have been a number of cases described in the literature. FOXP2 translocations and cases of FOXP2 deletions have also been reported. It will be important to figure out the profile of deficits presented by those with FOXP2 affected in some way and how it is best treated.

Dr. Fisher described other gene involvement that has been discovered through array technology called comparative genomic hybridization (array-CGH), one of the tools that moved quickly from research to clinic. This has led to identification of microdeletion syndromes that may be related to CAS. For example, microduplication of 7q11.23 may lead to a CAS. Another one is 16p11.2 microdeletion, first identified as a possible autism risk but it now appears to be more related to speech than to autism; several cases were identified when screening children with CAS. Microdeletion of 12p13.33 has also been identified as being a possible locus for CAS. The common gene, in this case, was ELKS which is now being researched for cases of mutation. Additional candidate chromosomal regions have been identified by screening children with a solid diagnosis of CAS. An unbalanced translocation of 4q;16q has been identified in three siblings. And, CAS has been found in a good portion of children with galactosemia, a type of metabolic disorder of genetic origin.

Finding genes, stated Dr. Fisher, is just the starting point of the research. The next steps are to understand the gene function. Studying the way the genes work leads to understanding the biology. There are many gaps to be filled between genes and the speech symptoms of CAS. Also deserving study are the proteins made by genes, how the genes function in cells, how the cells are involved in neural circuits and how those circuits work in the brain. It will take many years to fill in all the details. Dr. Fisher described FOXP2 as a sort of "molecular window" into the brain.

FOXP2 gets switched on to make its protein and then that protein finds target genes to bind to, in order to switch them on or off. FOXP2 adheres to certain genes and tunes down their expression much as a dimmer switch works. For example, FOXP2 sticks to another gene called CNTNAP2. When a cell has high FOXP2, the level of CNTNAP2 is diminished and this phenomenon can be seen in the developing human cortex. Dr. Fisher found other families that had typical forms of language impairment and found a section of CNTNAP2 where risk variants were correlated with reduced abilities in the families. The variants were then independently associated by other researchers with delayed language in children with autism. In a large Australian sample, Fisher also found the gene was correlated with measures of early language. More recently, they have shown CNTNAP2 is associated with dyslexia and nonword repetition and also with some cases of FOXP2.

Dr. Fisher clarified that he was describing two different types of genetic effects. The first, rare mutations such as in the case of FOXP2 in the KE family, change the proteins, which results in a speech and language disorder. A FOXP2 mutation is enough to cause the disorder. The second involves common variants that are not affecting the protein, but probably are more regulatory in nature and are associated with increased risk of disorders, such as in the case of CNTNAP2. However, FOXP2 and CNTNAP2 share a common functional pathway. Thus, Fisher described that there may be genetic links across different disorders that have common genetic underpinnings. FOXP2 mutations have been found to also influence other genes such as SRPX2/uPAR (Rolandic epilepsy with CAS), MET (autism), and DISC1 (schizophrenia).

Animal models have been used to determine what role FOXP2 plays in brain development. Mouse FOXP2 is actually very similar to human FOXP2 and similar in brain expression patterns in the

cortex, basal ganglia, thalamus and cerebellum. Dr. Fisher also described that the neural circuits where FOXP2 is expressed are sensory pathways for sensorimotor integration and motor-skill learning, which could relate to speech motor learning. FOXP2 seems to be important to neuronal outgrowth and how neurons connect to other neurons, as Dr. Fisher and colleagues have seen in mouse studies in which FOXP2 was damaged. The researchers were also able to observe that damaged FOXP2 led to significant deficits in mouse motor skill learning of sequential movements despite adequate and normal baseline motor skills.

To summarize, Dr. Fisher described FOXP2 as a regulatory gene. It, along with downstream targets, is providing researchers the ability to observe and begin to understand the neural pathways that are disrupted in speech disorders. Versions of FOXP2 are found in many species and seem related, for example, to motor learning in mice and vocal learning in birds. This gene may help to regulate plasticity of brain networks such as the cortico-basal ganglia circuitry. With the advent of new techniques in sequencing of DNA, one's genome can now be sequenced in several days for under $4,000. These new technologies and the discoveries being made with them, lead Dr. Fisher and others to surmise that in the future we are likely to discover that multiple genes in the same functional pathway may potentially lead to the disorder of CAS. Fisher asserted that next generation sequencing will have a major impact on CAS research. Currently the ability to generate large DNA sequences outstrips the capacity to understand it. However, understanding functional neurogenetic pathways will help researchers identify critical variants, which hopefully will impact diagnosis and treatment of CAS in the future.

Dr. Lewis was in agreement with Dr. Fisher in her remarks. Rare and common variants both contribute to CAS. FOXP2 has provided a wonderful model to discover rare variants and to follow them through the functional and neural pathways. The FOXP2 "story" has helped the field understand how the genes create functional neural pathways and eventually will explain the comorbidities that are so often seen. Dr. Lewis focused her presentation on several projects in her lab, one that is a rare variant and the other a common variant.

Dr. Lewis described a study with a single family with three siblings, each of whom had a different communication disorder: William's syndrome, Autism, and CAS. Using complicated genetic analysis, the researchers determined that in the larger family pedigree there was extensive history of speech, language and reading disorders. They identified the region on chromosome 7 that would be expected for Williams syndrome. The sibling with autism also had an affected area on chromosome 7, but the mother and father were identical in that area. So far they have not been able to identify genetic difference in the sibling with CAS. In discussing common variants, a large study identified the genes DRD2, AVPR1a and ASPM that are all associated with receptive vocabulary, phonological memory, and decoding deficits. Lewis and colleagues also found that AVPR1 has a role in language development and that ASPM is related to speech sound production.

In conclusion, Dr. Lewis stressed that both rare and common genetic variants contribute to CAS. For example, in some families a rare variant may cause CAS and in another family a combination of several genetic risk factors may reach a threshold and result in CAS and/or other speech and language

impairments and reading disorders. Lewis emphasized the importance of carefully selecting behaviors to study in order to look for new speech sound genes that may be less rare than FOXP2. Finally, Dr. Lewis stated that it was important to look longitudinally to see which genes may be turned on or off during development. Longitudinal studies may help predict which individuals with CAS will recover versus those individuals for whom the disorder will persist.

Dr. Peter addressed several themes in Dr. Fisher's presentation: ongoing gene discoveries, simple inheritance in complex phenotypes, and additional comments on why it is important to study genomics.   Dr. Peter described that her lab works from a heterogeneous framework, in which they study one family at a time rather than groups of children from different families.   Using genome wide linkage analysis in one family, Peter and colleagues found four new regions of interest for CAS, including one area on chromosome 6. This area was not the previously noted dyslexia area. Dr. Peter could not go further with this family because it was not large enough.   She described "family 11" that has 23 people and, through several methods of analysis, has led to a candidate gene. Dr. Peter could not mention the gene because she and colleagues had not submitted a paper yet. Speaking about phenotypes, Dr. Peter described that her research began to focus on examining rapid and sequential movements, not just of the speech mechanism, but also other motor systems, as well as cognitive and linguistic sequencing. Within several of the family groups studied by Peter for finger movements, affected members had much poorer sequential skills than unaffected members of the family on both word and nonword tasks. Peter and colleagues replicated this finding in 5 other families and propose that the CAS phenotype may include an underlying deficit in sequential processing, including: speech, motor speech, hand movements, reading and spelling.

Dr. Velleman's presentation focused primarily on one genetic condition that has been associated with CAS and called 7q11.23 duplication syndrome (Dup7), a recently documented genetic disorder estimated to occur in 1 out of 7,500 live births. This autosomal dominant condition includes an extra copy (duplication) of the same genes on chromosome 7 which are deleted in Williams syndrome. Dr. Velleman further described common physical and nonspeech/language characteristics of the syndrome. Individuals with the Dup7 syndrome have characteristic facial features and hypotonia, yet lack the medical conditions often associated with William Syndrome. Thirty percent of individuals with Dup7 display social anxiety, which coincides with information from mouse models of the disorder in which affected mouse pups develop separation anxiety. Additionally, Oppositional Defiant Disorder (ODD) and Attention Deficit Hyperactivity Disorder (ADHD) are very common in individuals with Dup7.

In a sample of 43 children with 7q11.23 Duplication Syndrome, ranging in age from 4;0 to 21, Dr. Velleman and colleagues have documented a range of motor speech conditions using conservative diagnostic measures. Forty-five percent of the children in the sample had either oral apraxia or symptoms of it and 73% had either CAS or symptoms consistent with CAS.   Some of the children in the sample also had dysarthria (16%), but occurring more frequently in the children were symptoms of dysarthria (58%). Finally, phonological disorder or symptoms of it were observed in 49% of the children

in the sample. There were no children with Dup7 that had no symptoms or no history of speech disorder. Severity of speech symptoms ranged from quite mild to severe.

Dr. Velleman and colleagues went on to compare their sample group of children with Dup7 to children with Williams syndrome. They identified that toddlers with the duplication syndrome do not progress as quickly in their speech development as do toddlers with Willams syndrome and the differences between the two groups increases with time. In 5 – 7 year old children with Dup 7 or Williams syndrome, the children with Dup7 had higher general intellectual and spatial ability whereas children with Williams syndrome had significantly higher speech scores. Finally, more children with Dup7 had CAS (47%) than did those with Williams Syndrome (10%), while all children with Williams syndrome had dysarthria. Given the findings of the comparison groups, Dr. Velleman argued that there are "dosage-sensitive" genes in the 7q11.23 region that contribute to variations in speech, language, and intellectual abilities. The researchers went on to compare children within the Dup7 group that did or did not demonstrate CAS. The children with Dup7 plus CAS had more speech disability and were generally lower on various intellectual ability measures. There was a significant negative correlation with chronological age in the CAS group for verbal IQ, expressive vocabulary and spatial abilities. Dr. Velleman explained that when the research team divided the Dup7 group by conditions other than CAS and compared them, there were no correlations by age for any other group, including comparison by severity of speech disorder. In summary, Dr. Velleman stated that the presence of CAS in children with Dup7 appeared to increase their risk for greater discrepancies in performance over time. She encouraged colleagues to examine all of the traits associated with CAS to determine which of them commonly co-occur with each other, are predictive of other traits, are associated with various outcomes, and respond to different types of interventions.

STATE OF THE ART IN CAS DIAGNOSTIC MARKER RESEARCH

CHAIR: Adam Jacks, Ph.D.,
TOPIC AREA PRESENTER: Lawrence Shriberg, Ph.D.
PANELIST PRESENTERS: Karen Forrest, Ph.D., Megan Hodge, Ph.D., and Tricia McCabe, Ph.D.

Dr. Shriberg focused his presentation on the premise that CAS diagnostic markers will aid theoretical and clinical understanding of the disorder if embedded in a cognitive neuroscience and pediatric speech disorders framework that integrates genomic, neurodevelopmental and speech processing findings. Currently, he stated, there are no universally-accepted diagnostic marker findings or assessment protocols for CAS across the lifespan. The disorder continues to be over-diagnosed worldwide.

Dr. Shriberg discussed many challenges in conducting programmatic diagnostic marker research. A "gold standard" to validate classification must be identified. Further, candidate signs must be selected and organized; data methods must be operationalized; criteria for a positive marker using a reference database needs to be standardized; and data methods for acquisition, reduction, and analysis needs to be computerized as much as possible. Next, in order to arrive at diagnostic markers, Dr. Shriberg stated that a large number of diverse subjects, representing various contexts for CAS (i.e. those with complex

neurodevelopmental conditions, neurological conditions, and idiopathic cases), must be tested before evidence based statistics can be applied to a diagnostic marker that conclusively discriminates CAS from other speech problems.

Another premise of Dr. Shriberg's was that CAS is a "sensorimotor" disorder, as research has documented integrated sensorimotor pathways for speech development. Additionally, he described CAS as a "multiple domain disorder", a premise supported by evidence in the FOXP2- CAS literature that has identified its impact on multiple domains and pathways, and other research reports documenting auditory-perceptual deficits, encoding and memory deficits.

Dr. Shriberg's final premise was that the diagnostic signs of CAS should be integrated with their explanatory substrates, including genomic, neurodevelopmental and speech processing accounts. To illustrate this point, he speculated on how diagnostic marker research could be integrated into a dual stream framework and a speech processes framework. A dual stream framework could represent the neural substrates for CAS. Shriberg suggested that the ventral and dorsal streams could be appropriate "cover terms" for the neurological level of speech processing. Additionally, Dr. Shriberg submitted that a six element system of speech processing could be integrated to represent speech processing substrates. The six elements for the speech processing system included representation, planning, programming, feedforward, execution, and feedback. Finally, at the third level of this integrated model, a system for signs of a diagnostic marker is needed. In preparation for reporting recent findings, Dr. Shriberg offered a four-sign marker that was being tested in his research program. The four-sign marker includes three prosodic signs (slow articulatory rate, inappropriate pauses, and inappropriate stress) and one segmental sign (inappropriate transcoding). Within the four-sign diagnostic marker, the classification criteria for CAS is that a child must present with at least 3 or all 4 of the signs. Three of the signs (slow articulatory rate, inappropriate pauses, and inappropriate stress) are derived from a conversational speech sample. The fourth sign, described as inaccurate transcoding, is obtained from a metric using results on a task of sequencing syllables into multisyllabic non-words (Syllable Repetition Task).

To further illustrate the premise that diagnostic signs of CAS should be integrated into a framework with their neurological and speech processing substrates, Dr. Shriberg proposed a possible model. He speculated that neurological pathways in the ventral stream underlie speech representation and planning processes; pathways in both ventral and dorsal streams underlie speech programming, feedforward, and feedback levels in the speech processing system; and pathways in the dorsal stream underlie speech execution. Incorporating the four signs in the Madison diagnostic marker—slow articulatory rate, inappropriate pauses, and inappropriate stress- Dr. Shriberg speculated that:

- Slow articulatory rate may reflect deficits in any or all levels of the speech processing system and relate to both ventral and dorsal streams.
- Two additional signs, inappropriate pauses and stress, may relate to deficits in the speech processing system at the level of representation, planning, programming, and/or feedback and associate with both the ventral and dorsal streams.

- And, inaccurate transcoding, the fourth sign, may associate with difficulty with representation and/or planning within the speech processing system and the ventral pathways

Presenting on recent findings, Dr. Shriberg reported on a recent study of the four-sign marker that he and colleagues conducted on data from 500 participants. Study participants included those with speech delay, CAS, adult AOS, and complex neurodevelopmental disorders. The Madison Speech Assessment Protocol (MSAP) was administered to each participant. The MSAP consists of four age-appropriate protocols that include 25 tasks, with 15 of the tasks being speech related. Out of the 15 speech-related tasks, two of them – the conversational speech sample and the nonword repetition task – were sufficient to elicit the four signs of CAS in the four-sign marker. The "gold standard" used to verify the positive CAS classification of a participant identified as such using the Madison marker was a pediatric adaptation of the Mayo Clinic criteria. Diagnostic agreement for positive CAS classification between the Madison marker and the Mayo criteria was roughly 80%. A second part to the study examined children in the group not only vetted as having CAS per the Mayo criteria, but also adding in the participants' speech or treatment histories. Focusing on this group, the sensitivity of the four-sign marker increased to 84.4%. The specificity data are from a group of children with speech delay in which only 4.5% were misclassified as having CAS using the four-sign marker, rendering a specificity of 95.5%. Very recent data from Dr. Shriberg's lab has gotten the specificity of the marker down to 1%. Additional findings highlighted by Dr. Shriberg included:

- Inappropriate pauses are most prevalent in CAS of any type (over 90%)
- Participants with complex neurodevelopmental disorders had fewer difficulties with transcoding than did individuals with CAS or those with speech delay.
- Participants with speech delay demonstrated few stress errors.
- Subtyping within the CAS classification may be possible using the four-sign marker.
- Effect sizes in various comparisons were quite significant.
- Within the group of individuals with complex neurodevelopmental disorders there are observed subtypes, i.e. some individuals demonstrating CAS and others that do not.

Dr. Shriberg concluded by proposing that CAS is a sensorimotor, multiple domain disorder in which neurodevelopmental deficits in the ventral and dorsal streams cause disruptions in speech processes. Additionally, he proposed that differences in the pattern of deficits among the diagnostic marker's four signs may help in identifying underlying genomic, neurodevelopmental and speech processing bases for CAS. Segmental markers of CAS will be more difficult to identify due to speaker differences and methodology constraints. However, performance on nonword repetition tasks may eventually make it possible to provide lifespan signs in the areas of auditory-perceptual, memory, and speech production deficits of CAS speakers. Dr. Shriberg stated that it is not possible to get conclusive behavioral markers of CAS in older speakers without proper case history documenting late onset of speech, effortful speech, and a significant lag in speech normalization. Finally, the understanding of CAS as a sensorimotor disorder with deficits in multiple domains should have implications for treatment planning.

Dr. Forrest's research has focused on comparing three-to-six-year-old children with speech sound disorders who had no prior treatment history to children with typical speech. Starting from a different perspective, Forrest and colleagues identified inconsistent consonant productions and errors as the primary classification criteria to make a diagnosis of CAS. Their data are based on an extensive probe list in which the researchers considered the percent of errors and the inconsistency of errors. Forrest and colleagues devised a metric to calculate high inconsistency and low inconsistency. Those children falling in the high inconsistency group were classified as having CAS, those in the low inconsistency group as having phonological disorder. Some of the findings reported by Dr. Forrest included that the children with CAS had reduced vowel space and voice onset time differences when compared to children in the typical speech and phonological disorder groups. Volitional oral movement did not reveal any group differences. Speech perception was challenging to measure and is being examined in various contexts. In treatment, Forrest and colleagues demonstrated that children with high inconsistencies (i.e., CAS group) made significant gains in percent consonants correct and reductions in inconsistency using the core vocabulary approach combined with stimulability training.

Dr. Forrest recommended that identifying inconsistency of segmental productions may help discriminate CAS from phonological disorder and that there are likely to be a list of comorbidities. Dr. Forrest concurred with Dr. Shriberg that the research priorities are to have the specificity and sensitivity needs defined, life span profiles, and protocols that are used across studies. Finally, Dr. Forrest suggested a gold standard based on independent variables to determine which of them may converge on a diagnostic category.

To obtain a view from outside of the speech science profession, Dr. Hodge shared perspectives on classification from what developmental pediatricians are studying and learning. Rather than CAS being a condition to diagnose, others in medical professions, according to Hodge, view it as a symptom or indication of neurodevelopmental impairment. Reportedly, it is rare for brain involvement that significantly delays one area to not also influence other areas of development. Dr. Hodge described that it is likely there is a continuum that results in a wide range and severity of other associated dysfunctions in addition to the primary one and the associated impairments may mean more to long term outcomes than the primary presenting problem. Dr. Hodge highlighted the understanding that pre-linguistic vocalizations are neuromaturational markers for the whole spectrum of neurodevelopmental disorders. Thus, in response to Dr. Shriberg's presentation, Dr. Hodge proposed that, "Severe speech delay" should be added to the motor speech disorder classification label, since severe speech delay underlies CAS. Second, Dr. Hodge argued that developmental stuttering should be considered a motor speech disorder, most likely as a speech programming deficit. She then proposed that subcortical processing, in addition to cortical processing, should be included in the neural substrates being considered in CAS.

Because of the impact of pre-linguistic milestones on future development, Dr. Hodge reported that she has most recently been working on a care path model for very young children with severe speech delay. The goal is to facilitate learning responses to normal experiences as early as possible. In

the care path model discussed, response to treatment dictates a program path for those toddlers with suspected motor speech issues so that they begin receiving help early, by 24 months of age.

In response to Dr. Shriberg's presentation, Dr. McCabe urged participants to consider the "end users" perspective. She stressed that tools based on classification markers need to be clinically applicable and open sourced so they can be used as diagnostic tools accessible to clinicians. McCabe argued that markers should be examined in a life span sample so that it can be documented as to when they appear, at what level they appear, etc. Dr. McCabe also stressed the need for cross cultural, cross-linguistic validation of markers, in addition to validation across socioeconomic statuses. True markers will hold universally.

Dr. McCabe also discussed a study using data collected during recruitment for a CAS treatment study. Participants were referred from the community, including referrals from clinicians who had diagnosed CAS. Dr. McCabe and colleagues found that 32% of the referred children were misclassified as having CAS. The researchers determined that 50 polysyllabic words and a combination of syllable segregation, percentages on lexical stress matches and percent phonemes correct (PPC) plus a more complex diodocokinesis task accounted for 91% of the variability in their participant sample. Although it was a convenience sample, Dr. McCabe noted that by using these four tasks there were no misdiagnosed children.

Finally, Dr. McCabe discussed the focus of a consortium that is working toward agreement on standards for research reporting of the assessment of children with CAS. She urged researchers to include socio-demographic data in their manuscripts. Also, in order to improve reporting of classification and diagnostic information, Dr. McCabe suggested that researchers include how diagnostic markers were operationalized, assessed and measured, as well as include reliability data.

State of the Art in CAS Neuroimaging Research

CHAIRPERSON: Karen Froud, Ph.D.
TOPIC AREA PRESENTER: Angela Morgan, Ph.D.
PANELIST PRESENTERS: Reem Khamis-Dakwar, Ph.D., Barbara Lewis, Ph.D. and Jonathan Preston, Ph.D.

Dr. Morgan began with a review of the adult speech motor system within an fMRI study. One study was able to demonstrate that speakers with no speech disorders have higher level speech motor planning/programming networks. Looking toward affected populations, Dr. Morgan explained that Broca's area and other left hemisphere areas are critical as damage in these regions places adult individuals at risk of apraxia of speech. According to Dr. Morgan, right hemispheric lesions in adults would rarely, if ever, lead to an apraxia of speech.

Dr. Morgan described a study in which she and a colleague examined whether there was left hemisphere dominance in development and whether or not there was potential for compensation. They conducted a systematic review of the literature from 1997 to present of the neural bases of motor speech disorders in children and adolescents with developmental, progressive or childhood acquired

neurological conditions. The researchers were able to include only a few papers in the review. Key populations described in the included studies were cases of FOXP2 mutations, epilepsy, and syndromic or metabolic conditions. Imaging studies of the KE family have documented bilaterally reduced grey matter in some structures and increased grey matter in other structures. In epilepsy, imaging findings describe bilateral perisylvian abnormalities. Cerebellar atrophy in children with galactosemia who have CAS has been documented, according to Morgan.   There were no cases of unilateral damage reported in the reviewed studies. Even cases of right or left hemispherectomy have not resulted in long term CAS. Dr. Morgan made the speculation that the left hemispheric specialization seen in adults happens later in development.

The researchers noted that brain imaging similarities between affected areas in adult versus child speech motor control were located in the perisylvian and perirolandic cortices, the basal ganglia and cerebellum.   Dr. Morgan reported that differences between the two groups (adults versus children) included that uni-hemisphere damage in adults can lead to chronic disorder while in children bi-hemispheric damage is needed to result in severe and long-lasting effects. Sixty percent of the documented cases in the studies that were reviewed were reportedly "normal" on routine CT/MRI, yet Dr. Morgan pondered if they were actually "normal" as the findings suggested. She speculated that perhaps structural abnormalities at a sub-macroscopic level underlie CAS or perhaps CAS is associated with anomalies at a metabolic or neurotransmitter level. Dr. Morgan called for the use of quantitative imaging to examine functional anomalies, not just routine MRIs or CT scans that provide evidence of structural differences. Dr. Morgan suggested that there was strong evidence for sub-macroscopic differences within group data that would be revealed by quantitative imaging such as voxel-based morphometry, tractography, and fMRI. She explained that sample sizes of 16 to 35 cases are required to derive meaningful group data.

Dr. Morgan provided an overview of newer methods of both structural and functional neural imaging. To date the greatest body of work in imaging and CAS is with the KE family in which both structural and functional imaging was completed. She reported that currently what is known is that there are both cortical and subcortical anomalies and white matter or "language" tract differences in affected KE family members. Thus, overall in the FOXP2 context, Morgan explained that one can describe a disruption in both the structure and function of brain regions known to be involved in auditory-motor integration, speech motor learning, planning, programming, and execution of speech that results in a severe speech and language disorder, with CAS being the primary presenting impairment. FOXP2 expression in both mice and birds also aligns with neural imaging of the KE family members.

Dr. Morgan described that some further questions regarding the neural bases of FOXP2 include how different mutations would impact the neural expression and associated speech characteristics; how mutations express themselves in individuals outside of this one family, and if the types of mutations and resulting neural outcomes are prognostic for long-term impact and outcomes. Speculating further, Dr.

Morgan ended by discussing future directions, particularly related to understanding how neural imaging can:

- Provide indicators for rehabilitation or compensation
- Aid in developing treatment hypotheses based on the brain and behavior
- Facilitate documentation of pre and post treatment neural changes
- Help in treatment selection, prioritization, timing and success.

Dr. Khamis-Dakwar, in response to Dr. Morgan's presentation, discussed a need for theoretical frameworks to guide the formation of hypotheses. She explained that neuroimaging means the imaging of brain activations which can be measured either indirectly or directly. Dr. Khamis-Dakwar clarified that in addition to the imaging distinctions between structure and function raised by Dr. Morgan, there are also distinctions between metabolic and neurophysiological processes. She suggested that converging evidence from different investigative techniques will be helpful. Even though most available studies in CAS currently use fMRI, Khamis-Dakwar proposed that it could be useful to consider other methods. For example, fMRI cannot measure change as it is actually happening; however electroencephalography (EEG) is able to capture neural change in "real time". She suggested that this method may be well suited for use with children in that it does not make many demands on the child's behavior.

Dr. Froud and Dr. Khamis-Dakwar conducted a small study on children with CAS in which they demonstrated, using EEG, neurophysiological evidence of phonological involvement in the disorder. Their theoretical framework for the study was based on work done with adults with apraxia of speech in which there was an overspecification of phonological representations. The researchers hypothesized that in the developmental process, phonemic underspecifications of representations may have gone awry in some way, possibly leaving available too many options for articulation and processing which could lead to motor planning and execution problems. The researchers speculated that if it could be a problem at this level, one would need to examine the finer-grained subprocesses of what the brain is actually doing when it is trying to process speech sounds.

The event-related potential (ERP) component of the EEG signal, called "Mismatch Negativity", was used by Khamis-Dakwar to compare ERPs of children with CAS and those of children with typical speech as they listened to a large number of a syllable stimulus, mixed with occasional deviant syllable stimuli. Khamis-Dakwar explained that the brain has an automatic change-detection response and the mismatch negativity is known to peak in the EEG signal about 150 – 250 milliseconds after the deviant sound is presented. Continuous EEG recordings were made as sounds were presented through ear phones. Researchers found that the MMN was not seen in children with CAS and that there appears to be evidence that children with CAS process speech sounds differently than typically developing peers. This work added to the evidence that there is phonological involvement, not just motor speech involvement, for children with CAS.

Dr. Barbara Lewis described the various objectives in her research program of comparing neural substrates for speech motor planning and production and fine motor planning; how current clinical measures correlate with observed neurophysiological differences; and how genes influence neural development such that there are neurological processing differences in a population of children with CAS, as compared to those with speech delay and typically developing children.   In a pilot fMRI study of adolescents with a CAS history and a control group of participants with no history of speech problems, participants were asked to repeat nonwords while in the MRI scanner. Interestingly, even though the articulations of the participants with CAS were correct and not different than the participants with no history of speech problems, their underlying neural processing was different.

In another pilot study with younger children, the Syllable Repetition Task was used while participants were in the MRI scanner. Dr. Lewis and colleagues found that children encompassed in the broad category of speech sound disorder (SSD) had slightly more right lateralized neural activation as compared to children with no speech difficulty during the speech tasks. A fine motor task of tapping in accordance with auditory tones was also conducted.   Results indicated that children with SSD had lower levels of activation during the fine motor praxis task. Children with SSD had similar activation patterns of the speech motor network when compared to the control group of children with no speech problems. In the various experiments described, Dr. Lewis explained that more activation was related to poorer performance, as if children with SSD were using incredible effort for the task, whereas, typically developing children were very focused and using a very narrow area of the brain. Dr. Lewis and colleagues also observed reduced activation in the sensory motor cortex on the right side of the brain of children with speech sound disorders. In conclusion, Dr. Lewis stated that she and colleagues intended to do further studies which contrast children with speech delay, children with CAS and children with no speech difficulties.

Dr. Preston, in response to Dr. Morgan's presentation, stated that he agreed that it was prudent to focus not only on cortical regions, but also on subcortical regions and he indicated that a lot of neural activity would be subcortical during development. Dr. Preston explained that because the brain is always changing, neural imaging research is difficult in that, for example, the brain of a four-year-old child with CAS may be different than that of a 14-year-old youth with CAS. Regarding the issues of laterality, Dr. Preston speculated that there may be some right hemisphere compensation. Dr. Preston agreed that research should not focus on tissues, per se, but instead should focus on connectivity of brain regions which may be more informative for CAS. He stressed that neuroimaging cannot and will not replace behavioral assessment. Dr. Preston further asserted that basic questions regarding behavioral diagnostic questions need to be answered before neuroimaging can be most helpful and informative for CAS. Also, Dr. Preston noted that not all children with CAS will have the same neurobiological profile.

At his lab, Dr. Preston stated that they are working on the neural characterization of the speech planning and programming deficits in CAS using EEG. He discussed the temporal processing and time order of what occurs temporally in the act of picture naming. Speech planning and programming

generally occurs at 275 to 400 milliseconds after the picture is visually presented with the instruction to name the picture.   Because children with CAS struggle with challenging items that are phonetically complex or lengthy, it was Dr. Preston's expectation or hypothesis that differences would be seen in the EEG planning/programming time window in children with CAS as they produced phonologically complex words. Dr. Preston's study used ERP, which studies the EEG signal in response to a particular stimulus (onset of a picture) and provides fine grain time course information about the brain's processing. The study discussed by Dr. Preston included 10 children with persistent CAS and 10 children with no speech difficulties. Data from the study suggested that left hemisphere activity for simple and complex stimuli was somewhat similar in both typical speakers and those with persistent CAS. However, in the right hemisphere, Dr. Preston noted that while typical speakers did not demonstrate differences between simple and complex words, children with CAS show significant differences in magnitude of the signal during simple versus complex words. Thus, there was a group by condition interaction that was statistically significant in the time window that the researchers predicted it would be. Dr. Preston speculated that planning complex movements requires more neural effort from the right frontal hemisphere. Plans for future research include modifying the task to see if better group separation can be achieved for sensitivity and specificity, comparing other subtypes of speech disorder, and comparing younger participants.

State of the Art in CAS Neurocognitive-Behavioral Research

CHAIRPERSON: Barbara Davis, Ph.D.
TOPIC AREA PRESENTERS: Wolfram Ziegler, Ph.D. and Ben Maassen, Ph.D.
PANELIST PRESENTERS: Edwin Maas, Ph.D., Hayo Terband, Ph.D.

Dr. Zeigler's presentation focused on perspectives of adult apraxia of speech (AOS) versus Childhood Apraxia of Speech (CAS) or the mature versus the unfolding speech motor system. He explained that adult speech can be considered a highly over-learned skill, in which the learning extends over at least a decade. During this learning period, individuals develop domain-specific (specific to speech) and language-specific (specific to one's native language) vocal tract motor behaviors. One's articulation or vocal tract movements are language specific from the very beginning. Articulatory movements that are frequent in the language are acquired and those that do not occur often are lost. Learned articulatory movements are domain specific. An individual learns to make a movement during speech and can use it during speech, for individuals in whom the articulatory movement does not occur in their language, they are unable to make the particular movement for speech, however may be able to make the movement during nonspeech tasks. Dr. Zeigler described that as children, individuals acquire specific sequential, rhythmic combinations of gestures or "coalitions" and not single articulatory gestures. The gesture patterns follow the syllable structure of one's language and those syllables that are more frequent are more entrained in the speech motor system. Articulatory gestures and rhythmic patterns in adults are language and domain specific, highly automatic, and reflect the sequential motor patterns and rhythmic basis of one's language. Dr. Zeigler instructed that this process is reflected in the Gestural Patterning Model of adult speech.

Dr. Zeigler stressed that intensive motor practice changes the neural network in functional and structural ways, from synaptic to structural changes. He explained the role of the basal ganglia in speech -motor learning. The basal ganglia is instrumental in procedural learning, leading to routinized motor sequential behaviors. Additionally, the organization of the striatum (part of the basal ganglia system) supports sensory-motor learning. Children with specific prenatal or perinatal damage to the striatum may demonstrate severely impaired speech or no speech; although this condition is different than the speech impairment expected in adult acquired basal ganglia dysarthrias. Dr. Zeigler surmised that the basal ganglia most likely play a crucial role in speech development. In song birds, damage to these same areas resulted in impaired vocal learning in the animals. While the striatum plays a role in the speech learning, Dr. Zeigler explained that the left anterior perisylvian region of the left hemisphere acts as the host to mature speech motor plans in adults. Lesions to this area of the left hemisphere in adults result in apraxia of speech in which adults lose previously acquired speech motor plans. Dr. Ziegler speculated that CAS, occurring in the developing system, prevents or obstructs the formation of speech motor plans, possibly due to some dysfunction of the striatum. Dr. Ziegler ended his presentation stating that the destruction of the adult mature speech motor planning system and a disturbance in the emerging development of speech motor plans in childhood could lead to similar error patterns, thus explaining the use of the term "apraxia of speech" to describe both conditions.

Dr. Maassen's presentation began with a reiteration that there is no validated list of diagnostic markers at the behavioral level to identify CAS, although much progress has been made as evidenced by earlier presentations. He argued that the lack of diagnostic markers impacted on criteria for subject selection. A model of causation that incorporates etiology, neurobiology, cognitive, and behavioral symptoms is required, according to Maassen. He stated that a complex disorder like CAS cannot be defined at just one of the levels. Dr. Maassen proposed that a psycholinguistic model is the type of model that could be used to understand CAS. One suitable characteristic of the psycholinguistic model is that it can account for a series of organized hierarchical, cascading events for complex motor skills such as speech.   While there is an appropriate model such as the psycholinguistic model, Maassen explained that there is a tendency to interpret discrete parts of it as modular and to attribute responsibility for a function like motor planning to only one part. Dr. Maassen suggested that the motor planning component of speech production actually starts at the level of word form retrieval. Symptoms proposed as diagnostic for CAS are also symptoms that overlap with speech delay and other speech sound disorders, and yet are important parts in a hierarchically organized schema. Dr. Maassen argued that a modular conception of the psycholinguistic model does not do justice to the interaction of the whole speech production process.

Dr. Maassen described four aspects of phonological encoding which are relevant to CAS, including: quality of retrieved word forms, selection and sequencing of phonemes, prosody and syllabification.   In syllable production tasks, children with CAS demonstrated a stronger coarticulation effect between and within syllables and were more variable. In several studies on speech gestures, children with CAS reportedly had difficulty with inter-articulatory coordination. In research conducted by Dr. Maassen and colleagues that investigated gesture articulation, children with CAS had more

tongue tip variability compared to children with speech sound disorders or those with typically developing speech while lower lip and jaw variability was equal among the groups. Thus, in the children with CAS there was atypical development of speech gestures.

Dr. Maassen argued that CAS must necessarily be different than AOS because of the developmental trajectory. For example, Maassen described that if articulation is faulty in an adult, it is presumed that other aspects of speech processing may remain intact. However, when poor speech production occurs in children, they will develop weaker auditory processing, phonology, and word form lexicon. Maassen and colleagues modeled this process using the neural computational model called "DIVA" (Directions Into Velocities of Articulators). Maassen described two important phases in the DIVA model. First, there is a babbling phase for the acquisition of the perceptual-motor characteristics of the vocal tract and systemic mapping of the various movements and their auditory, tactile and proprioceptive consequences. During the babbling phase the child learns how the oral motor structures work to produce movements and experience the resulting auditory consequences of the movements. The second phase is an imitation learning phase, or "inverse mapping" phase, in which the child knows the desired sound and can map it to movement gestures.   The researchers used two hypotheses and used computer simulations to test them using the model and then compared the results to known developmental data. The two conditions in the experiments were somatosensory information degradation alone and somatosensory plus auditory feedback degradation. Results of these experiments indicated that motor difficulties with intact auditory feedback resulted in problems at the phonological level. Motor plus auditory feedback difficulties led to problems at the phonetic level, yet, the quality of the speech between the two experimental conditions was quite similar and included decreased intelligibility, increased coarticulation, groping-type behavior, and token-to-token variability. Dr. Maassen shared future research plans and directions using the DIVA model for simulation. He highlighted research goals that included:

- Test specificity of results by manipulating other parameters within the DIVA simulations
- Further track the phonological development and word form representations as the result of the deviant perceptual-motor differences

Dr. Maassen concluded by stating that one should not only look at specific symptoms of CAS but also at the unspecific symptoms because they could form part of the explanation of the underlying deficit. Speech development is characterized by associations, not disassociations, and this can obscure the underlying core deficits. Diagnosis should be based on longitudinal assessment with multiple diagnoses possible. And finally, treatment should focus on speech gestures and syllables rather than phonemes.

Dr. Maas began his comments noting that there appeared to be consensus through various speakers that detailed models are needed to evaluate processes at various levels and to guide assessment. He maintained the importance of diagnostic marker research to eliminate the "circularity" problem. Dr. Maas stated that starting with a model may help researchers make predictions of patterns and help transition into process-based assessment procedures. He added that it was refreshing to hear several speakers refer to the time-course of speech and the acknowledgement that speech has a temporal

dimension. Dr. Maas proposed that data collection should receive careful attention and be specified in at least three ways: elicitation technique (i.e., naming, repetition), materials (i.e., nonwords, clusters) and measures (i.e., speech rate, intelligibility, etc.).   He described that his research is attempting to discover how phonology can be reliable assessed in children who have speech problems. One way to do so, Dr. Maas suggested, is by using mispronunciation detection tasks. The child does not have to say anything during such tasks, but they have to access the phonology and make a judgment upon seeing a picture and hearing a sound. Priming tasks are also being employed, using reaction time to observe what phonological representations in the child may look like when the child is using them in a speech task. Dr. Maas also mentioned his work to verify predictions generated from the DIVA simulation work of Dr. Terband and Dr. Maassen because their simulations did generate novel predictions that can now be tested.

Dr. Maas noted Dr. Zeigler's idea that perhaps there should be attention in CAS research to the expert motor learning literature. Dr. Maas' treatment research with random and blocked practice had mixed results suggesting that there may be different sources of underlying impairments in children with CAS. Dr. Maas asserted that there is not enough evidence yet regarding how principles of motor learning should be incorporated into speech therapy for children with CAS.   In one of his own studies, Dr. Maas studied feedback frequency. In this study, he found an advantage to reducing feedback, yet not all children demonstrated the same response. Dr. Maas concluded by stating that theoretical models will allow researchers to test the specific underlying impairments while also providing more information that could inform treatment plans.

Dr. Terband began his comments recognizing the advances in neurocognitive behavioral research that have provided methods and techniques to isolate speech processes and representations. He reiterated comments of other speakers by stating that a disorder in one part of the system is likely to spread out to other levels. Dr. Terband asserted that there is no one to one relationship between CAS symptoms and underlying deficits. The diagnostic instruments currently in use are focused only on the behavioral level. Dr. Terband suggested that a process oriented assessment and treatment approach is better suited to the issues arising in CAS. He explained that in a process oriented approach, methods and techniques can be more individually tailored so that evaluation and treatment can follow the evolution of the disorder. In practice, this approach could be implemented through objective measurements of speech undertaken during systematically varied tasks and conditions. For example, all relevant factors could be mapped for each child based on their particular underlying problem. Dr. Terband argued that given children with CAS will likely be provided with treatment for one or more years, it may be prudent to spend several days in assessment properly identifying underlying a child's particular deficits.

Dr. Terband, through a Dutch collaboration, described current work on designing and implementing process-oriented diagnostics and treatment programs. Terband and colleagues are developing a computerized articulation instrument which will be interactive and administer 5 tasks: picture naming, word representation, both word and nonword repetition and diadochokinesis. The instrument in

development records the sound signal and assists in processing data after assessment. Systematic manipulation of speaking conditions; yes/no auditory feedback judgments, practice and learning effects are also being added to the system. Dr. Terband stated that the group also intends to add acoustic measurements such as speech sound quality, coarticulation and variability measures.

According to Dr. Terband, process-oriented diagnostics can be helpful in addressing the gap between research and practice and holds a number of advantages. For example, it can provide leads for treatment goals, allows for individually-tailored therapy that addresses the unique underlying deficits experienced by a child and it can improve evaluation of the evolving nature of CAS.

Concluding Remarks & Summation

PRESENTER: Susan Rvachew, Ph.D.

Dr. Susan Rvachew concluded the symposium with reflections, comments, and questions for further consideration. She organized her comments along the two applied themes of diagnosis and treatment. The major points included:

- Can clinical training programs properly prepare the speech-language pathologist for the sheer level of complexity, which will be required of them, in order to integrate characteristics or diagnostic signs of CAS with an understanding of their underlying genomic, neurodevelopmental, and speech processing substrates as discussed at this meeting? An understanding of these issues will ultimately be important in the diagnostic process.
- Longitudinal studies are needed in order to clarify how genomic and environmental risk or protective factors interact with one another and can be used to explain different developmental trajectories for affected children.
- There is a need to understand if and how current treatment methods specifically target the acquisition of speech motor planning. We need to better understand the features of treatments that target "planning", as contrasted to treatments that target phonological knowledge or the execution of newly learned speech motor plans.
- We will need to know more about the best context for speech practice and that, given the basic research that was presented throughout the meeting, includes an understanding of how to maximize the child's access to both auditory and somatosensory feedback.

List of Symposium Attendees

Below is a complete list of participants in the 2013 Childhood Apraxia of Speech Research Symposium:

Deryk Beal, Ph.D., R-SLP, CCC-SLP, University of Alberta
Thomas Campbell, Ph.D., CCC-SLP, BC-NCD, University of Texas Dallas, Callier Center
Sue Caspari, M.A., CCC-SLP, CASANA Professional Advisory Board
Barbara Davis, Ph.D., University of Texas Austin
Margaret Fish, M.S., CCC-SLP, CASANA Professional Advisory Board

Simon Fisher, Ph.D., Max Planck Institute for Psycholinguistics, Donders Institute
Karen Forrest, Ph.D., Indiana University
Sue Freiburger, M.S.P.H. – Secretary, CASANA Board of Directors
Karen Froud, Ph.D., Columbia University
Sharon Gretz, M.Ed., Executive Director & Founder, CASANA
David Hammer, M.A., CCC-SLP, CASANA Professional Advisory Board
Debra Hayden, M.A., CCC-SLP, SL-P, Reg. CASLPO, CASANA Professional Advisory Board
Kathy Hennessy – CASANA, Director of Education
Megan Hodge, Ph.D., R.SLP, CCC-SLP, University of Alberta
Jenya Iuzzini , Ph.D., CCC-SLP, MGH Institute of Health Professions
Adam Jacks, Ph.D., CCC-SLP, University of North Carolina at Chapel Hill
Kathy Jakielski, Ph.D., CCC-SLP, Augustana College
Nancy Kaufman, M.A., CCC-SLP, CASANA Professional Advisory Board
Reem Khamis Dakwar, Ph.D., Adelphi University
Barbara Lewis, Ph.D., Case Western Reserve University
Jeanne Lippert – Member, CASANA Board of Directors
Ben Maassen, Ph.D., University Medical Center, University of Groningen
Edwin Maas, Ph.D., University of Arizona
Rebecca McCauley, Ph.D., CCC-SLP, Ohio State University
Tricia McCabe, Ph.D., University of Sydney
Amy Meredith, Ph.D., CCC-SLP, CASANA Professional Advisory Board
Angela Morgan, Ph.D., University of Melbourne
Megan Overby, Ph.D., Duquesne University
Beate Peter, Ph.D., CCC-SLP, University of Washington
Nancy Potter, Ph.D., Washington State University
Jonathan Preston, Ph.D., CCC-SLP, Haskins Laboratories, Yale University
Erin Redle, Ph.D., CCC-SLP, Cincinnati Children's Hospital Medical Center
Margaret Rogers, Ph.D., CCC-SLP, American Speech-Language Hearing Association
Susan Rvachew, Ph.D., University of Montreal
Lawrence Shriberg, Ph.D., CCC-SLP, University of Wisconsin – Madison
Edythe Strand, Ph.D., CCC-SLP, BC-NCD, Mayo Clinic
Liz Stang – CASANA, Administrative and Community Outreach Assistant
Ruth Stoeckel, Ph.D., CCC-SLP, CASANA Professional Advisory Board
Hayo Terband, Ph.D., University Medical Center Groningen
Mary Sturm, MD – President, CASANA Board of Directors
Jennifer Vannest, Ph.D., Cincinnati Children's Hospital Medical Center
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Wolfram Ziegler, Ph.D., City Hospital Munich, University of Munich