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Special Panel Session: Driving Critical Initiatives in Motor Speech

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Abstract

The following report is a summary of the Special Panel Session, entitled, “Driving Critical Initiatives in Motor Speech,” that was conducted at the Conference on Motor Speech, March 2008, in Monterey California. Don Finan (Program Chair for Speech Motor Control) and Julie Liss (Program Chair for Motor Speech Disorders) invited four distinguished scientists (Drs. Gary Weismer, Steven Barlow, Anne Smith, and John Caviness) to share, briefly, their opinions and views on selected topics. This was followed by an hour-long general discussion session with conference attendees. This report contains an introductory statement followed by the panel members' own summaries of the opinions and ideas expressed in their talks. We then summarize the major topics that were considered during the discussion session. This summary reflects the biases and opinions of the participants, and is meant to serve as a thought-piece for the readership of *JMSLP*, rather than as a scientific report.

Introduction (D. Finan and J. Liss)

The eve of the 40th anniversary of Darley, Aronson and Brown's 1969 publication of the discipline-shaping classification of motor speech disorders seems to be a suitable time to assess the current and future directions of our basic and clinical science. Our self-evaluation is especially critical at this juncture, as the playing field has changed dramatically in the past 40 years. The application of emerging technologies allows for an unprecedented opportunity to ask and answer pivotal questions regarding speech motor control and its disorders. Even the roles of the clinician and researcher in motor speech are evolving, with evidence based practice principles dictating increased and enhanced documentation of speech changes in the search for effective treatments. Indeed, the next 40 years of clinical practice and science in motor speech may bear little resemblance to the previous 40. It is with this eye toward the future that we have invited a panel of distinguished scientists to share with us their visions. The goal was to generate lively discussion to illuminate an array of critical initiatives with which to drive the field forward.

With Foreword and Discussion Summary by: Don Finan University of Colorado Boulder Julie Liss Arizona State University

Motor Speech Disorders: Growth of the Knowledge Base, 1975-2008; Needs of the Knowledge Base Over the Next 40 Years (G. Weismer)

The purpose of this presentation is to summarize the evolution of the knowledge base in motor speech disorders, consequent to research between 1975 and the present. The starting year of 1975 is chosen because this is the year of publication of *Motor Speech Disorders* (Darley, Aronson, & Brown, 1975), the textbook that has defined the parameters for research in motor speech disorders for at least three generations of scientists.

I will make summary statements for the several areas of research indicated below. A first level of summary involved a data base search from 1975 to the present, to estimate the volume of published research on dysarthria. These searches did not use the term "motor speech disorders" by design, focusing on the more specific case of "dysarthria" because of its more straightforward nature as a motor speech disorder; the searches therefore underestimate the published literature if "apraxia of speech" is understood as a motor speech disorder. Two searches were done, one of the *Linguistics and Language Behavior Abstracts* (*LLBA*) data base, the other of *PubMed* which casts a wider net and is likely to pick up any published paper in which motor speech disorders are mentioned, for whatever reason.

Table 1 shows the results of these two searches. Overall, the *LLBA* search produced 500 citations relevant to dysarthria research; the *PubMed* search produced roughly six times this number, most likely because the latter results include many citations from the medical literature in which dysarthria is mentioned as a symptom of various neurological diseases (as compared to a phenomenon under investigation). This interpretation is supported by the large number of PubMed citations returned when "Diagnosis" was included as a keyword. It is most interesting that when "stuttering" is searched on *LLBA* for the 1975-2008 time frame, three times as many citations are returned as for "dysarthria", even though dysarthria is clearly a more common disorder. The following is a brief review of selected topic related to dysarthria.

Speech movements of persons with dysarthria have been shown to be slower and less extensive than normal speech movements. Movement abnormalities of one articulator (for example, the lips) do not necessarily predict movement abnormalities of a different articulator (e.g., the tongue) within the same speaker. Finally, there is no clear evidence to date that speakers with dysarthria have frank interarticulator coordination disorders.

Speech intelligibility measures of dysarthria have mainly functioned as indices of severity. Different measures of intelligibility (such as word tests, sentence tests, direct magnitude estimates) seem to reflect the same phenomenon in different ways, although the proper work to identify the benefits and problems of different measures has yet to be done. There is no evidence to date that the perceptual dimensions of Darley, Aronson, and Brown are, in fact, separable estimates of intelligibility made with any of the measures described above. To date, speech intelligibility research among speakers with dysarthria has not exploited the rich literature in speech perception that might make speech intelligibility measures more sensitive and fine grained. Early hopes for "explanatory" tests of intelligibility have not fulfilled their promise of this kind of increased sensitivity.

Speech acoustics research has shown that a number of measures reveal reliable differences between speakers with dysarthria and neurologically-normal speakers. These acoustic phenomena most often indicative of dysarthria include compression of vowel space; reduction in formant transition extents and slopes; longer-than-normal segment durations; reduction of contrast metrics (such as VOT, fricative spectra, tone types); and *suboptimal source characteristics*. Relations between the measures and underlying articulatory behavior on the one hand, and speech intelligibility on the other hand, remain murky, mysterious, and almost

certainly dependent on factors about which we understand very little (such as compensation and severity).

With respect to *variability*, it is clear that speakers defined with the same type of dysarthria, or disease type, will show wide inter-speaker variability on almost any speech production (or oromotor, nonspeech) measure made in the laboratory. Somewhat less clear for an understanding of motor speech disorders is the *meaning* of increased, normal, or decreased intra-speaker variability on these measures. Stereotypy, or the lack thereof, has not received a proper theoretical treatment as an explanatory "player" in an understanding of motor speech disorders.

Oromotor, nonverbal tasks continue to be used in studies of persons with dysarthria, under the assumption that they provide a meaningful window to speech motor control processes. It has been exceedingly difficult to demonstrate, using various laboratory approaches, the transparency or even translucency of such a window; a substantial series of papers has yet to demonstrate or suggest that we will learn much from laboratory tasks in which the speech acoustic signal is eliminated as a player in the concept of speech motor control.

Treatment research has shown that behavioral change focused at one level of the speech mechanism may "spread" to other, untreated levels, as predicted by Rosenbek and LaPointe (1978) 30 years ago. In addition, the "one-size-fits-all" treatment approach suggested by Darley et al. in 1975 has evolved to type-specific treatments, discussed at length by Duffy in his 1995 text. Treatment probe outcome measures, however, have not been adequately discussed and evaluated in the research literature.

An adequate *theory* of dysarthria has yet to be developed. We started with a theory thought to be true (that the classical symptoms of neurological diseases account for the different dysarthria types), but have accumulated some good evidence that this is not always or even mostly true. In the past several years, it has become obvious that a productive theory of dysarthria will depend on the much more developed theories of normal speech production.

In conclusion, there is a need for consortium research in which large numbers of participants can be studied at many different centers, using a unified protocol that will include multiple measures.

Emerging Technologies in Motor Speech Research and Rehabilitation (S. Barlow)

In this talk, I will focus on three emerging technologies that may have a dramatic effect on motor speech research, and particularly rehabilitation, in both the present and near future. These include 1) technology for the stimulation of the nervous system during active skilled motor tasks, 2) remote sensing and device control, and 3) field programmable gate arrays (FPGA).

Stimulation of the Nervous System during Active Skilled Motor Tasks

The role of adaptive plasticity in recovery of function following ischemic injury to the motor cortex is the subject of intense study as a therapeutic agent for upper limb motor recovery. The principles outlined below seem equally applicable to the recovery of speech motor control and swallowing following cortical injury. The basic idea in this relatively new area of study in rehabilitation and neuroscience is that functional use of the affected limb can be attained using rehabilitation techniques that focus on motor relearning and skill training, applied in combination with direct application of a physical modality to the cerebral cortex (Harvey & Nudo, 2007; Nudo, Plautz, Frost, 2001). Many studies in human and animal models utilize functional neuroimaging of stroke recovery to document expansion of cortical areas

undergoing adaptive plasticity (Johansen-Berg, 2007). Facilitating brain plasticity post-ischemic injury through direct application of patterned inputs is optimized and dependent on active skilled use of the affected structure. The delivery of patterned inputs has been achieved using repetitive transmagnetic stimulation (rTMS), transcranial direct current stimulation (tDCS), peripheral electrical stimulation (Ludlow, 2008), epidural cortical stimulation (Nudo, 2007), and patterned orocutaneous stimulation applied most recently in the human infant orofacial model (Barlow, Finan, Chu, Lee, 2008).

A randomized, multicenter human clinical trial is currently underway to test the effectiveness of epidural cortical stimulation on upper limb motor control (Levy et al, 2008). Epidural cortical implant technology has several advantages over transcranial technologies, including (1) stable fixed position of the active electrode over the peri-lesion sensorimotor cortex, (2) more focal stimulus field, (3) avoids the tedious task of remapping the cortical 'hot spot', (4) longer trains of cortical stimulation at higher frequencies can be delivered during a single session, and (5) provides a more practical method for delivering stimulation during skill motor retraining permitting a variety of settings and environmental conditions. The combination of cortical stimulation plus motor training surpasses the effects of motor training alone (Nudo, 2007). The principal risks lie in surgical complications (~ 1 to 4%), and seizures.

Another fascinating technological approach to motor rehabilitation is known as *Robotic Assisted Motor Therapy* which involves the physical coupling of servo actuators to an affected structure to help the patient complete partially executed movement trajectories or sequences (Takahashi, Der-Yeghiaian, Rehan, Motiwala, Cramer, 2008). This therapeutic approach is largely unexplored for speech movement control systems, but is based on repetition, attention, velocity, force, position end-points, precision (variability and accuracy), timing, and virtual reality movement-space environment training simulations. Neural reorganization is mapped using clustered volume acquisition *f*MRI. Consistent with the cortical and peripheral patterned input studies reviewed above, successful robotic assisted motor therapy is optimized during active skilled use of the affected structure.

Remote Sensing and Device Control

Extending traditional laboratory-based recordings of speech acoustics and sensorimotor physiology to real life situations (i.e., home and work environments), to dysarthric populations with significant dyskinesias, or among developmental populations which are not amenable to tethered cable links to hardware recording devices will benefit from remote sensing based on BlueTooth (BT) technology. BlueTooth 2.0 is a standard radio communications protocol primarily designed for low power consumption, with powerclass-dependent ranges based on low-cost transceiver microchips with an integral antenna in each device. One of the principal advantages of this enabling technology is that line-of-sight is not required between the instrument or transducer and the host transceiver which is often embedded within a microprocessor or personal computer. This feature of BT permits the tester and patient to be located in different rooms up to 100 meters apart. The new physiological monitors also incorporate flash memory to permit extended recordings when the BT device is out of range. This is particularly useful in speech motor control applications where patient performance and motor status can be monitored away from the laboratory environment (i.e., home, work, travel). Variable channel arrays and sampling rates (up to 2048 samples/sec @ 24-bit) make the BT technology suitable for electrophysiology, dynamics, kinematics, and aerodynamics (Lee, Shin, Mun, 2006). Most devices commercially available incorporate digital channels (128 samples/sec) for event marking and pulse stimulus control.

Significant advances in data transmission rates now make it possible to remotely sense and transmit multichannel physiological data (12-16 channels) with up to 24 bits of resolution from the test subject to the data management microprocessor. A consortium known as the WiMedia

Alliance has proposed a new high-bandwidth standard for data transmission that would far exceed the current BT 2.0 standard. This will permit even higher bandwidth data streams to be sampled from the human speaker, including high-fidelity acoustic, kinematic, dynamic, and electrophysiological signals related to speech and vocalization.

Field Programmable Gate Arrays (FPGA)

A programmable logic device known as the Field Programmable Gate Array (FPGA) was invented by Xilinx Corporation (www.xilinix.com, San Jose, California) in 1984. FPGAs represent a revolutionary technology which evolved from simple glue logic chips to replace custom application-specific integrated circuits (ASICs) and processors for digital signal processing and logic control applications. FPGAs are reprogrammable silicon chips which utilize prebuilt logic blocks (64 to approx 10,000 logic cells) and software development tools or programmable routing resources. This programmability provides a revolutionary alternative to fixed or custom logic devices that typically require many months to design, test, and manufacture. FPGAs can be configured to implement custom hardware functionality without ever having to pick up a breadboard or soldering iron. One can develop digital computing tasks in software and compile an instruction set on how the components should be wired together. Thus, FPGAs are completely reconfigurable and can instantly take on a brand new "personality" when the user recompiles a different bitstream. FPGA chips are presently adopted across all industries and combine the virtues of ASICs and processor-based systems. This translates to low cost development platform technology. FPGAs enjoy hardware-timed speed and reliability, and eliminate high volumes to justify the large upfront expense of custom ASIC design.

FPGA chips are field-upgradable and do not require the time and expense common to an ASIC redesign. Reprogrammable silicon also has the same flexibility of software running on a processor-based system, but it is not limited by the number of processing cores available. FPGAs are truly parallel in nature so different processing operations do not have to compete for the same resources. Each independent processing task is assigned to a dedicated section of the chip, and can function autonomously without any influence from other logic blocks. Thus, performance of one part of the application is not affected when additional processing is added.

National Instruments reconfigurable I/O (RIO™) technology allows the user to define custom measurement hardware circuitry using reconfigurable FPGA chips and LabVIEW™ graphical development tools. The RIO core includes an FPGA chip and surrounding signal conditioning circuitry to enable LabVIEW to perform hardware synthesis. Kieweg and Barlow (2008) have developed a specialized FPGA application (coded using LabVIEW™) for servo-control of a pneumatic actuator for delivering patterned orocutaneous stimulation and multichannel 24-bit sampling of pressure, force, and displacement from the infant orofacial structures. This system includes the FPGA microprocessor, memory, power supply, signal conditioning modules (multichannel bridge amplifier module, DC-coupled power amplifier), multifunction I/O (A/D, D/A, digital control), transduction, and custom linear pneumatic actuator. An added benefit of this technology for applications in speech motor control is the reduced size and cost of electronic components which render the traditional bulky 19" rack-based system components obsolete. Moreover, FPGA-based systems will lead to a variety of portable applications for translational applications in speech and swallowing physiology from bench to bedside or clinic.

Issues for Understanding Speech Motor Development. (A. Smith)

In my presentation I consider six general issues that I believe are important for understanding speech motor development. (1) General Models of Speech Motor Control, (2) Relationship of Speech Production to Earlier Oral Motor Behaviors, (3) Oral Motor Reflexes and Speech, (4) Growth Curves for Speech Motor Development, (5) What Does Decreasing Output Variability

Signal?, and (6) Units of Production During Speech Motor Development. Below I summarize my comments on each of these topics.

At one level all motor behaviors require the recruitment of motor neurons, which in turn activate the muscle fibers they innervate. Motor neuron pools (the collections of motor neurons in the brainstem or spinal cord that innervate a muscle) are the sites of integration of inputs from many different centrally originating pathways, including cortically originating networks involved in language and motor processing, brainstem pattern generators (e.g. for sucking, chewing, breathing), and other centers. In addition, peripherally originating pathways including reflexes have strong effects upon the excitability of motor neuron pools. Infants obviously are not born with all of these circuits intact and functioning as they do in adults. Therefore, an important question is: How in the process of development do infants, toddlers, children, and adolescents emerge to have adult-like control of motor neuron pools for speaking?

At birth, the infant possesses a repertoire of motor behaviors including breathing, crying, and sucking. All of these rhythmic motor behaviors arise from central pattern generator circuitry in the brain stem. By 12 months, the basic pattern of chewing is well-established, and interestingly, the duration of the masticatory cycle is established at this age (0.7 sec) and is maintained through adulthood (Green et al. 1997). Thus we know that brainstem central pattern generators have strong and well organized control pathways out to the motor neuron pools of the lip, jaw, tongue, laryngeal, and chest wall muscles. A critical question is whether speech motor pathways operate via these pre-existing CPG circuits to control the motor neuron pools involved in early vocal behavior and later for speech; or are the CPG networks completely bypassed by cortically originating speech motor control circuits?

This has been an issue of considerable debate in the literature on early vocal development. McNeilage and Davis (MacNeilage, 1998; MacNeilage and Davis, 2000) have argued strongly for the frame/content theory which posits that early vocalizations and subsequently babbling use the pre-existing masticatory CPG circuits. They propose that the masticatory oral open/close cycle is an essential building block for the emergence of speech. The open/close cycle is the “frame” onto which the “content” of speech sounds is elaborated during development. Moore, Green and colleagues (e.g., Green et al., 1997; Ruark & Moore, 1997; Moore, 2004) have argued the alternative case, which is that the neural networks controlling early vocalizations, babbling, and later speech motor behaviors are entirely independent from the pre-existing CPG circuitry. In this case cortically originating networks generate direct inputs to the motor neuron pools involved in vocalization and articulation, and presumably, the brain stem CPG circuitry is suppressed. They argue this based on evidence that the muscle activation patterns for early vocalizations are distinctive from those involved in earlier appearing CPG driven behaviors.

This debate is not resolved, and it would be important to know whether there is total separation of neural control pathways for speech and other oral motor behaviors. This issue is particularly relevant for questions about the usefulness of nonspeech, oral motor therapies to improve speech motor performance. Weismer (2006) has argued that nonspeech oral therapies are not effective, because the underlying control mechanisms and output characteristics of speech and nonspeech behaviors are so distinctive. I would note, however, that we do know that there is overlap in speech and nonspeech pathways at some levels, including the motor neuron pools, sensory pathways, and reflex and interneuronal circuitry. Any motor activity that recruits the motor neuron pools would therefore affect the status of these motor neurons and reflexes and other sensorimotor circuits.

In fact, there are extremely powerful and widely distributed reflex circuits that affect the excitability of orofacial, laryngeal, and chest wall motor neuron pools. Unfortunately the role

of these reflex pathways has been neglected for some years in speech motor control research. In contrast, accounts of the control of other motor behaviors (such as limb, eye, and neck movements) have emphasized the significant role that reflexes play in optimizing movement output. Also, the speech motor literature often contains assertions about oral motor reflexes that are incorrect. For example, it is often assumed that reflex effects are very large in the newborn, but that with development in childhood, reflex responses become smaller and less important as centrally originating pathways “take over” control of motor neuron pools. In fact, work from our laboratory and others has shown that oral motor reflexes are actually growing in amplitude during the childhood years when children are acquiring and refining their speech motor skills (e.g., Smith, Weber, Newton, and Denny, 1991). It is important to note that these are responses of jaw and lip muscles to very light, innocuous tactile stimuli such as those that would be generated by the articulators themselves during speech. The role played by these powerful cutaneous reflex pathways during speaking in developing and mature systems is not known, but the potential importance of these circuits during different phases in the human lifespan and the role they may play in a variety of speech motor disorders is worthy of further experimental attention.

Turning now to growth curves for speech motor system parameters, which often show a reduction in variability with maturation-- recent work has demonstrated an extremely protracted course of development to mature patterns of articulatory coordination (e.g., Smith & Zelaznik, 2004; Walsh & Smith, 2002). Even in late adolescence (14 to 16 years), children do not produce speech as rapidly or consistently as young adults (20 to 22 years). Adolescent girls and boys show no differences in articulatory coordination or speech rate variables, even though girls' oral motor structures mature significantly earlier. There are sex differences in speech motor performance in preschoolers. Boys lag girls in speech rate and coordinative consistency at age 4 and 5-years; but there is no sexual dimorphism in craniofacial growth patterns at this age (Voperian et al., 2005). These findings suggest that, despite the importance of craniofacial growth in terms of the control problem presented to the brain, growth patterns do not account for early sex differences in speech motor performance in girls and boys; nor do growth factors account for the protracted developmental course to adult speech motor performance. Rather we have suggested that the protracted developmental course for speech motor performance reflects the continued maturation of the cortical networks involved in language processing.

Finally as mentioned above, a feature of many of the growth curves that have been plotted for speech motor behavior is that the variability in output decreases with maturation. While variability in output in disordered speakers is often interpreted as reflecting a deficit or negative aspect of performance, greater variability in the developing motor system is an adaptive and essential feature of a system acquiring new patterns of behavior (e.g., Thelen & Smith, 1994). In fact, it is important that the speech motor system continues to have plasticity into adolescence, because of the continued maturation of many frontal lobe circuits. Thus, variability in speech motor output can be interpreted as a reflection of where the individual is in his/her lifecycle. There are also neural networks involved in speech motor control that can be changed over very short periods of time, for example, within a single experimental session (Walsh, Smith, & Weber-Fox, 2006). These changes reflect speech motor learning with practice. In summary, when we interpret variability in motor performance in different populations performing under a variety of external conditions, it is important to contextualize our interpretations to take into account the age of the speaker and the fact that there are properties of the speech motor system that reflect short-term and longer-term neural changes (see also Newell et al., 2006).

Perhaps the most intriguing issue facing those of us who study speech motor development is the question of how the units of language are translated into movement --the language/motor

interface. We have addressed this question in two recent papers (Smith & Goffman, 2004; Smith, 2006). Our proposal is that there is no single, privileged unit of production and that multiple levels of language units, from the phrase to the segmental level, are used in the generation of motor commands that drive the motor neuron pools involved in speech. During development, we hypothesize that the child acquires multi-unit layers of mappings between language and motor systems. This issue will be very exciting to explore in future work as we try increase our understanding of how the developing nervous system generates the central and peripheral neural circuitry that produces the rapid and elegantly controlled spatiotemporal patterns of human speech.

A Clinical Neuroscientist's Point of View (J. Caviness)

It can be easily argued that medicine is on the verge of dramatic advances in the treatment and cure of a variety of neurological diseases. Further, this changing landscape will affect the science and practice of those who study and treat motor speech disorders. In developing critical initiatives in motor speech, it is crucial to be proactive relative to advances in medicine, even partnering in these discoveries and trials. In this talk, I describe some of the progress that has been made and how this progress may induce changes for those in the area of motor speech.

Last century brought a revolution of the symptomatic treatment of neurological disease, owing to the great strides that were made in the understanding of symptom pathophysiology. Prominent examples include the striatal dopamine deficiency of Parkinson's disease (PD) and the cortical acetylcholine deficiency of Alzheimer's disease (AD). In PD, dopamine replenishment--either via levodopa that is metabolized to dopamine by nigro-striatal synaptic terminals, or via direct stimulation of striatal dopamine receptors with dopamine agonists--causes a partial normalization of the balance between excitatory and inhibitory basal ganglia motor circuitry. This discovery resulted in unprecedented symptomatic relief for people with idiopathic PD. Although less clinically stunning, symptomatic treatment for AD emerged with the discovery that acetylcholinesterase inhibitors prevent the metabolic breakdown of acetylcholine in the synaptic cleft. The resulting increase in stimulation of cortical acetylcholine receptors was associated with modest memory improvements in some patients.

Despite the clinical significance of symptomatic treatments and their impact on quality of life for so many patients, symptomatic treatments fall short of true success. In even the best cases, relief from symptoms is only partial. And as disease progresses, these therapies become less effective and are associated with an increased number of side-effects that offset their value. Thus, despite their importance in providing benefit to people with neurodegenerative disease, they are no match for disease progression.

However, new hope and a new therapeutic revolution emerged from a government sponsored concentration of research efforts in the 1990s (the “Decade of the Brain”). Profoundly important discoveries were made in the genetic and molecular pathophysiology of neurodegenerative disease. These discoveries offered the possibility for a new form of neuroprotective treatment that would target these underlying mechanisms of the disease, and slow, or even halt, disease progression. There are a number of pathophysiological events that are presently being scrutinized as possible targets for neuroprotective treatment. These include mitochondria dysfunction and oxidative stress, excitotoxicity, inflammation, apoptosis, and protein aggregation. Interestingly, these pathological mechanisms are common to most of the major neurological diseases. Thus, it is possible that one or a few neuroprotective agents could be developed that would slow or halt disease progression in a number of different diseases.

Let us consider the example of PD in this therapeutic revolution. Presently, pharmaceutical companies are applying a dual-pronged approach to drug development. Major efforts are invested both in the improvement of symptomatic therapies directed at alleviating symptom

impact, as well as in the development of neuroprotective agents that target the underlying neurodegenerative processes. Yet, it is the development of the neuroprotective treatments that would have the greatest impact on disability associated with PD. Consider that even a modest slowing of disease progression on a yearly basis would have the effect of compounding interest, dramatically lessening disease severity over a multi-year period. Moreover, the longer a patient stays in the milder stages of Parkinson's disease, the longer levodopa symptomatic therapy is likely to be optimally effective. Also, behavioral and compensatory strategies for managing dysarthria will be effective longer, should dysarthria ever emerge at all.

Because the potential payoff for neuroprotective treatments is so high, shouldn't the majority of resources be placed in their research and development? In my opinion, it is difficult to give up the traditional notion that symptoms need to be studied. It is commonly stated in medicine that we need to understand symptoms before we can know how to treat them. However, if we can limit disease progression, the significance of symptoms is greatly reduced, and perhaps negligible. Put in terms of motor speech, why devote resources to studying dysarthria in PD, when the support could go instead toward preventing the progression of PD to the point of compromised intelligibility? In a climate of limited resources for funding, motor speech research will be affected by such prioritization.

In my opinion, there is a large role to be played by the motor speech scientific community in the study of, and transition to, neuroprotective therapies. The ultimate benefit of neuroprotective treatment is realized when it can be administered at the earliest possible moment. Because of the highly complex nature of speech, subtle speech changes often are the harbinger of neurological disease onset, in the absence of any other clinically detectable symptoms. There is a high premium placed on the identification of such biomarkers, whose presence reliably detects early—even preclinical—disease status. In fact, speech is inherently a good biomarker, because of its long history of being objectively measured and evaluated as an indicator of normal biological processes, pathogenic processes, or responses to therapeutic interventions. If we can identify an aspect of speech that offers sufficient levels of sensitivity and specificity (at least 80%), this would be an incredibly valuable and non-invasive biomarker for the variety of neurodegenerative diseases in which dysarthria is a component.

I'll end with one comment on the use of animal models in the treatment of neurological disease because, in my view, the tide is turning with the focus on neurodevelopmental treatments. Historically, the goal of developing animal models is to achieve phenotypic behavior correlations with the human disease counterpart (e.g., tremor and bradykinesia for PD), even if the lesions or conditions to produce these behaviors did not coincide with known human pathophysiology. These animal models have been useful; however, there is a trend to produce animals that have the basic molecular defect of human disease pathogenesis (e.g. transgenic mice), but that may have little or no resemblance to the human disease clinical syndrome. It is this latter type that holds most promise for understanding basic molecular mechanisms that will yield treatments for slowing or preventing disease, and for modeling the basic molecular pathogenesis.

Discussion Summary (D. Finan, J. Liss)

Following these four presentations, audience members and the invited panelists participated in a discussion session. Several topics generated debate. We have attempted to summarize these topics below, as an additional index of the “pulse” of the field.

1. It was suggested that we should question our conception of units, patterns, and coordination in the study of speech motor control. Do we assume an underlying pattern, and if so, what are the constituents of that pattern? What are the units, or better yet, are there valid units at all? What, then, is coordination?

2. Emerging information on the neurobiology of exercise may have the potential for revolutionizing rehabilitation strategies. How can we capitalize on this?
3. Is neural plasticity an epiphenomenon such that every time we change a behavior we get some cortical change? To what extent should we be driven by neural explanations? Should we be wary of believing that other fields have things "figured out" when we do not?
4. What is the role of speech *meaning* in the modeling of speech motor control?
5. Acoustic analysis should be a major player in explaining the mechanism of the sound production for the purposes of speech communication. Ideas about coordination will fall out in terms of kinematics.

The purpose of this report was to present the opinions of four influential scientists in our field on selected topics relevant to speech motor control and motor speech disorders. Although the topics explored here are certainly not comprehensive in terms of the field of motor speech, it is hoped that these opinions and the resulting discussion topics provide the reader with a glimpse of the future of research into speech motor control and motor speech disorders.

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Table 1

Results of database searches to discover the volume of published literature on dysarthria since 1975. The first column shows the keyword used in the search ("+" indicates a Boolean operation, limited to "Dysarthria +" pairs), the second column results from a search of the LLBA data base, the third column results from the PubMed search. The Boolean combinations do not sum to the "Dysarthria" totals because of either overlap or absence of the "+" terms from citations with the overarching term keyword "Dysarthria".

Keyword	LLBA	PubMed
Dysarthria	524	3112
+ *Movement*	40	330
+ *Intelligibility*	152	196
+ *Acoustics*	37	120
+ *Variability*	40	59
+ *Diagnosis*	61	2353
+ *Treatment*	105	1278