> speech. Above all, do nothing that would make him regard them as abnormal or disgraceful. (p. 7)
>
> Do not label Fred a 'stutterer.' If you do, you will have an almost irresistible tendency to treat him as if he were as defective and unfortunate as the label implies. (p. 7–8)

The theory and the clinical advice that followed from the Diagnosogenic Theory attained widespread acceptance, and for decades no clinician, or anyone else it seems, would ever think of directly treating early stuttering by calling attention to it. The situation in the 1970s is portrayed here:[6]

> one of us presented a workshop on speech and language disorders to a group of early childhood teachers … Stuttering was included and the presenter used the word *stuttering* when the topic was introduced … the teacher said that they had been taught that using the "label" *stuttering* would cause a child to become a stutterer … they had also been taught that these children were experiencing "disfluencies", that they were not actually stuttering, and that the problem would worsen and they would become stutterers once they were labelled and treated as such. (p. 49)

The fall

The fate of most theories during the course of advancing knowledge in a field is for them to be wrong. They can't all be right,[11] and part of scientific development is the eventual realisation that a theory is wrong, and this is what happened with the Diagnosogenic Theory. Emerging research evidence during the 1970s strongly suggested that it was wrong. A prime example was the reports of verbal contingent stimulation of stuttering with pre-schoolers, described during Lecture One. The most famous of those was a 1972 publication[12] showing that two pre-school children reduced stuttering when attention was called to it, and a similar finding to the same effect was published some years later.[13] If the Diagnosogenic Theory was correct, then calling attention to children's stuttering would have worsened it, not improved it. The first public proclamation that the Diagnosogenic Theory was wrong was published in 1983,[14] but its influence lingered long after that.

There was much controversy and colour surrounding the theory. As mentioned during Lecture Two, Johnson conducted a dubious experiment during the 1930s that was not published[15] but which, decades later, was found to not support the theory[16] and which resulted in legal proceedings.

On the colourful side, the theory predicted that if a culture could be found with no word for stuttering or concept of what it was, there would be no stuttering in that culture. Johnson published a report in 1944[17] stating that the Bannock and Shoshone Indians of Idaho in the United States had neither any word for nor concept of stuttering, and none of them stuttered. Correspondence came to light in 1981[18] that Johnson was informed at the time that he was wrong, and that the tribes in question had 18 ways of referring to stuttering. When prefacing the fourth edition of his landmark text in 1987,[19] Oliver Bloodstein announced that the period since the previous edition had seen some "notable surprises," one being "the discovery that American Indians of the Great Plains do stutter and probably did stutter a generation ago, when they were reported not to."

The Diagnosogenic Theory provides a telling illustration not only of how a causal theory can influence clinical practices, but how that influence can go awry. A recent report shows that, decades later, when the theory is obviously wrong, some clinicians still believe it to be true. A recent study of 37 speech-language pathologists and 70 speech-language pathology students[20] reported that "more than half of the participants indicated that parents are the primary etiological factor in stuttering and the word 'stuttering' should be avoided" (p. 778).

Testing causal theory of stuttering

Introduction

At first, it might seem that the notion of what causes a disorder, or anything to happen, is a simple matter. It is well worth reading a brief introduction to the philosophy of science that deals with

causality, in the first two chapters of a landmark text.[6] Those chapters cover concepts such as necessary and sufficient conditions for something to occur, fallacies of causal reasoning, the philosopher Thomas Kuhn and scientific revolutions, pseudoscience, paradigms, laws and theories, models, and hypotheses. The discussion on pages 10 and 11 about what causes a bushfire gives some insight into the potential complexity of studying causality.

This is not to say that causality never is simple. The authors of that text mention single gene anomalies that cause human problems, such as cystic fibrosis or sickle cell anaemia. All those who have the genetic anomaly have the disorder and nobody has the disorder without the genetic anomaly. Or to say it another way, a single gene is necessary and sufficient to have the disorder.

That reference text[6] discusses four ways to evaluate a causal theory: testability, explanatory power, parsimony and heuristic value. The first two of these are now overviewed.

Testability of a theory

The prime source of information used to evaluate theory is experimentation, which, to cut short a long story, is observations in contrived circumstances that make them more powerful. For example, imagine a theory of stuttering that the problem is in the larynx with vocal fold function. There have been such theories, with one in particular being prominent.[21] An experiment could explore that notion, by having those who stutter speak using their vocal folds and without using their vocal folds: during lipped speech, when there (presumably) is no vocal fold function, and during standard speech, when there is vocal fold function. If the theory is correct and stuttering is a problem with vocal fold function, there should be no stuttering during lipped speech. In fact, such experiments have been done[22,23] and it is obvious that stuttering can occur during lipped speech.

The influential philosopher Carl Popper is credited with the axiom that experimentation does not prove a theory to be correct; it only fails to disprove a theory. However, experimentation can provide results that may be interpreted as disproof of a theory. A theory that constantly resists active attempts to disprove it attains increasing credibility. A theory that resists active disproof for a long period can become known as a law. For example, the theory of gravitation is often referred to as the law of gravitation. (But even gravity, it seems, is not immune from observational challenges.[24])

Explanatory power of a theory

The more that a theory can explain about its topic the more credible it is. Stuttering presents so many things that need to be explained by a theory, and a causal theory of stuttering needs to be evaluated in light of how well it explains them. The following are just some of the prominent research findings about stuttering that causal theories need to take account of in order to be credible.

Behavioural diversity

Stuttering is behaviourally diverse. Why does it have such a range of behavioural manifestations involving different types of repeated movements, fixed postures, and superfluous behaviours? Even more challenging for causal theory is that everyone who stutters does so in a different way, even though they obviously have the same disorder. They have different types and combinations of the seven stuttering behaviours described during Lecture One.

The influence of spoken language

As outlined during Lecture One, stuttering moments are not random but tend to occur more on consonants than vowels and mostly on initial sounds of words and on initial words of utterances. Those who stutter often encounter idiosyncratic difficulties with particular sounds and words. Stuttering occurs more commonly on content than function words. And a most obvious but commonly overlooked fact for any causal theory to explain is that stuttering does not occur on every syllable spoken; it presents as an intermittent problem involving stuttering moments. It is even more challenging for causal theory to explain that language is not even necessary for stuttering to occur; stuttering can occur experimentally on non-words, where lexical processing is not necessary.[25]

Epidemiology

As outlined during Lecture Two, stuttering begins during the pre-school years, but why not later in life? Why does it sometimes resolve naturally but sometimes becomes a lifelong problem? Why does it sometimes begin abruptly and sometimes gradually, and why are repeated movements often among the first signs of stuttering?

Conditions that reduce or eliminate stuttering

There are diverse fluency inducing conditions as outlined during Lecture One. Stuttering nearly always vanishes when people sing or speak in rhythm, or when they speak under chorus reading or shadowing conditions. Stuttering decreases with verbal response contingent stimulation and under conditions of altered auditory feedback and masking. How can such a range of diverse conditions reduce or eliminate stuttering?

Stuttering and wind musical instruments

Playing wind instruments has in common with speech that it involves respiratory activity combined with tongue and lip movements. There are intriguing reports, dating from the early 1950s, that some who stutter appear to do so when playing a musical wind instrument.[26,27,28,29] One of those reports[29] provided acoustic evidence of this occurring. So whatever causes stuttering is capable of affecting a non-speech activity.

Stuttering and manual tasks

Although there have been findings to the contrary, there are research findings that signs of the disorder are to be found outside the speech mechanism. Examples include delayed manual reaction times for those who stutter[30,31,32] and finger movement tasks.[33,34,35] There have been recurring reports that those who stutter do not perform as well as controls with bimanual motor sequences. One research group has found this to occur with finger tapping, key pressing, handle turning, and even peddle pushing.[36,37,38,39,40,41,42,43] Such results have been independently replicated,[44,45,46] and interest in the topic seems to persist with a further replication.[47] Compared to controls, those who stutter have been shown to have more timing asynchrony when playing piano melodies.[48] All this is even more intriguing than findings about playing wind instruments and speaking nonsense words, because such tasks have nothing at all to do with speech. There is evidence, however, that the effect is not present with pre-school children who stutter,[49] raising the suggestion that the effect is connected with the effect rather than the cause of stuttering.

Stuttering severity is variable

Stuttering severity is notoriously variable. It is likely to vary with differing audience sizes and types,[50,51,52] generally with more stuttering as audiences become larger. Stuttering severity varies also across different everyday situations.[53] It seems that there will be more stuttering when speaking to people than when speaking alone.[52,54,55,56] The latter study showed with a group a 60% stuttering reduction when alone compared to when an experimenter was present. Experiments that have involved repeated measures of participants in the same speaking situation have shown clinically significant stuttering variability in that same situation.[57,58,59] A study of six participants over five clinic visits spanning 2 weeks [60] showed that in two cases stuttering severity was four or five times greater on some visits than others.

Statistical process control charts are a method of studying variation, and that method has been applied to stuttering.[61] The stuttering severity of 10 adults was studied during the course of their speech during a single day. Results showed that all 10 showed predictable variation around their mean severity. However, five of the participants had stuttering severity that was unpredictable, and suggestive of an "out of control system," showing severity scores more than three standard deviations from their means during the day.

Stuttering and genetics

Any causal theory of stuttering needs to be able to explain, as outlined during the previous lecture, that genetics is obviously involved with stuttering. Although details are not fully known at present, inheritance of stuttering obviously is complex, with a number of genes involved.

Brain structure and function

Also as discussed during the previous lecture, any causal theory of stuttering needs to take account of research findings connecting stuttering and problems with brain structure and function. Those findings are suggestive of a genetically determined problem with myelination of white-matter tracts.

Multifactorial models of stuttering causality

The fundamental proposition

In short, multifactorial models state that stuttering is caused by the interaction of many factors to be found in the living environments of early childhood and within children themselves. There is nothing necessarily pathological about the factors involved. They just interact uniquely for each child to be responsible for stuttering. To say it precisely, these models specify nothing as necessary or sufficient for stuttering development.

The Demands and Capacities Model

The best known multifactorial model is the Demands and Capacities Model, which, as its name implies, states that stuttering occurs when the demands for a child's fluency exceed the child's capacity to produce it. The demands on children come from the living environment, and include excessive parent language expectations, constant time pressures of living, and excessive parent demands for advanced cognitive performance. Four capacities of children are proposed: speech motor control, language development, social and emotional functioning, and cognitive development. In the words of its proponents, "there is no single etiology, but as many etiologies as there are stories of stuttering development" (p. 24).[62]

The prominence of the Demands and Capacities Model prompted an entire issue of the Journal of Fluency Disorders to be devoted to it in 2000. The model has been described at many sources,[62,63,64,65,66,67] with its first appearance in a 1987 textbook:[68]

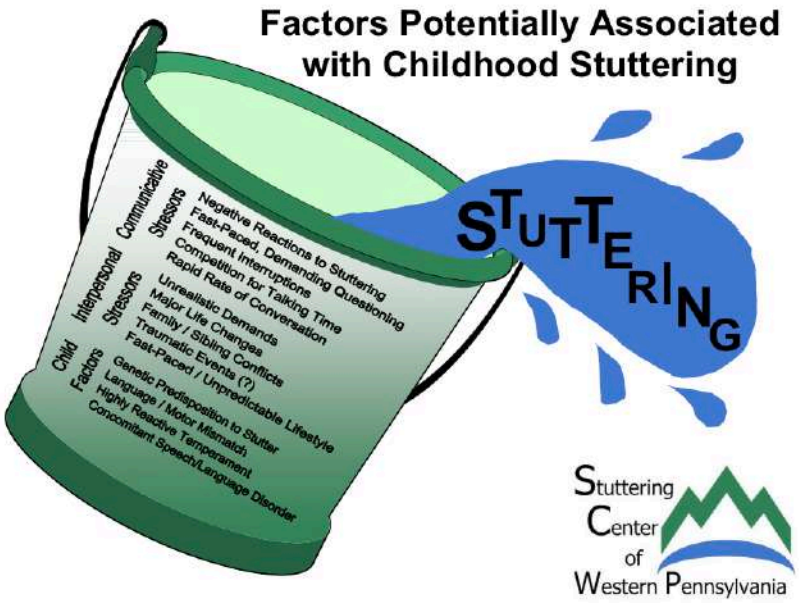
> this growing capacity to talk more easily is paralleled by increasing demands for fluent speech, demands placed on children by the people they communicate with … when the child's capacity of fluency exceeds the demands, the child will talk fluently but when the child lacks the capacity to meet demands for fluency, stuttering will occur. (p. 75)

The model has been depicted graphically[69] as shown in these figures.[‡] The first shows a situation, on the left, where demands exceed capacity, and hence were stuttering occurs. The situation on the right shows a scenario when capacities exceed demands and stuttering does not occur.

[‡] Reproduced with permission: Guitar, B (2014), Stuttering: An integrated approach to its nature and treatment (4th ed), Baltimore, MD, Lippincott Williams & Wilkins.

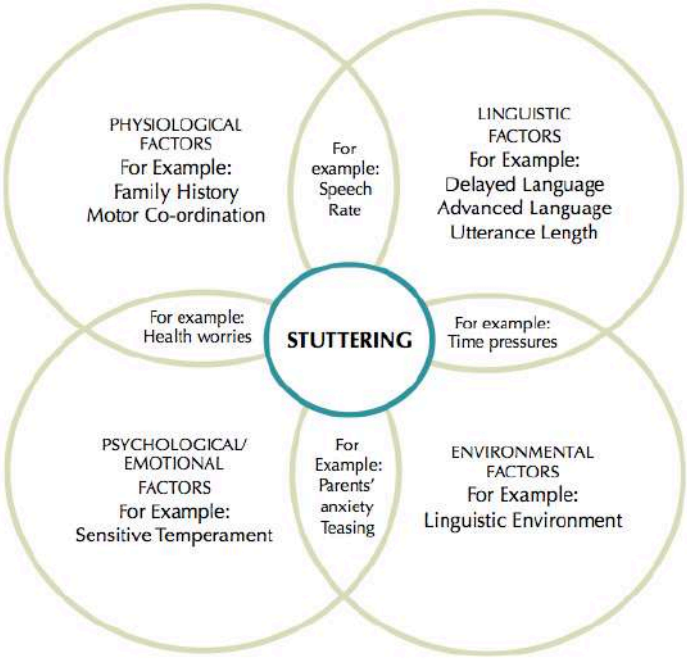
Other multifactorial models

There have been several variants of multifactorial causal models in addition to the Demands and Capacities Model. Two are used internationally as stuttering treatment models,[70,71] particularly in the United States. The next figure[‡] is a graphic from the Stuttering Center of Western Pennsylvania.[71,72] The conceptual similarity between it and the Demands and Capacities Model is apparent, as is the notion that nothing is necessary or sufficient for stuttering, as shown by the phrase "factors potentially associated with childhood stuttering."



[‡] Reproduced with permission: the Stuttering Centre of Western Pennsylvania, © 2004.

The Michael Palin Centre in London proffers another variant of multifactorial models,[73] shown in the next figure.‡ Again, the conceptual similarity with other multifactorial models is apparent. Another variant is known as the Dual Diathesis-Stressor Model,[74] which includes a temperamental proclivity component.



Testability

Although extensively popular theoretically and clinically, the Demands and Capacities Model has been criticised many times,[6,75,76,77,78,79,80] and those criticisms imply criticisms of multifactorial models in general. Those criticisms reiterate the point that multifactorial models are not testable and hence not falsifiable. This is for the simple reason that, as quoted earlier, "there is no single etiology, but as many etiologies as there are stories of stuttering development" (p. 24).[62] It is logically impossible to disprove an indefinite number of causes.

Explanatory power

These models do not score well in terms of explanatory power. An obvious problem for them is explaining the epidemiological fact that most stuttering appears during such a narrow age range during the pre-school years. Such models would suggest that it could begin at any time during childhood family life when the factors sufficient for stuttering converge, creating a situation where demands for fluency exceed the child's capacity to produce it. Also from an epidemiological perspective, it is problematic that the models specify that a cause of stuttering is located in the living environments of early childhood. How could it be, then, that stuttering persists throughout life when that early childhood environment no longer exists? It is logically essential that "all causal factors must be operating at every moment of stuttering" (p. 226).[75] For a related reason, multifactorial models do not explain stuttering variability across time and situations throughout adult life. However, they do explain why stuttering might vary during early childhood in different speaking situations; different situations

‡ Adapted and reproduced with permission: The Michael Palin Centre, © 2014.

involve a different mix of demands and capacities. The table presents a suggested summary of the explanatory power of multifactorial models.

RESEARCH FINDING	*EXPLANATORY POWER*
Behavioural diversity	*No*
The influence of spoken language	*No*
Epidemiology	*No*
Conditions that reduce or eliminate stuttering	*No*
Stuttering and wind musical instruments	*No*
Stuttering and nonsense words	*No*
Stuttering and bimanual motor sequences	*No*
Stuttering severity is variable	*No*
Stuttering and genetics	*No*
Brain structure and function	*No*

The future of multifactorial models

Multifactorial models were first proposed two and a half decades ago. From a scientific perspective they have attracted much criticism. Such criticism is justifiable, considering that they are logically impossible to test and that their explanatory power is questionable. Rather than providing theoretical understanding of why stuttering develops during early childhood, they seem only to restate the problem; children begin to stutter because they are unable to do otherwise. Regardless, multifactorial models currently enjoy clinical popularity as a basis for techniques to control of early stuttering, and they have prompted laboratory studies exploring their clinical usefulness[81,82,83,84,85] As discussed during Lectures Six and Seven, there have been two clinical trials of such techniques.

Variants of multifactorial models seem not to have sustained much interest so far this century, although they have been described in a clinical context within two book chapters,[73,86] and they still feature as topics of presentations at international conferences about stuttering. One peer-reviewed scientific journal publication[87] restates an existing model[70] with accompanying explanation of how it is broadly consistent with some aspects of current knowledge about the disorder. Yet the publication seems to add nothing about the explanatory power of the model.

The Interhemispheric Interference Model

The fundamental proposition

It appears that the first formal proposal of this model, implicating the supplementary motor area, occurred in 1987.[88] The Interhemispheric Interference Model has two parts. The first is that the supplementary motor area of the brain is inefficient, and the second is that the system of hemispheric activation is over-reactive. These two factors are proposed as necessary and sufficient for the development of stuttering; either factor alone is not necessary. The Interhemispheric Interference Model is an extension of the now-defunct Orton-Travis Theory,[89] but departs from it by specifying that those who stutter have normal lateralisation of speech functions. The most recent iteration of the model states "an anomaly in interhemispheric relations and a deficit in the mechanisms of speech–motor control are each a necessary but not sufficient condition for stuttering" (p. 125–126).[90]

The developer of this model, William Webster, has relied on logic derived from the research findings about bimanual sequence tasks discussed earlier:[91]

> the neural systems underlying such sequential movement control overlap those involved in speech and orofacial movements. Accordingly, anomalies in sequential finger-tapping in stutterers may suggest something about the nature of the "aberrant interhemispheric relations" hypothesized by Orton and Travis. (p. 11)

This prompted the proposal that:[36]

> Although sequential response mechanisms may be lateralized normally in stutterers, the repetitive sequential finger tapping error data suggest that these mechanisms may nonetheless be unusually susceptible to interference. (p. 818)

Testability

The model is certainly consistent with many brain imaging findings of unusual hemispheric speech processing with those who stutter. A recent review[207] overviews that literature. Yet a large-scale study of the planum temporale[†] was not consistent with the model.[92] It refuted earlier findings[93,94] of differences in symmetry between stuttering and control participants for that anatomical structure. It has been argued[6] that the model is difficult to refute experimentally because neither of its two brain components are operationally defined: the inefficient supplementary motor area and the over-reactive process of hemispheric activation. However, the model developers reported that it was verified with an experiment[88] where stuttering and control participants performed a finger-tapping task with a concurrent task using the other hand. The stuttering participants had more interference from the concurrent task than the controls.

Another experiment, though, caused a problem for the model by showing that the same result occurred with a bimanual writing task: writing with both hands concurrently.[95] The results were consistent with a cognitive problem rather than a physical problem with concurrent left and right handed activity. Webster described the problem in a later publication:

> it is unlikely that the interference with sequencing mechanisms in stutterers is strictly an interhemispheric phenomenon as was suggested by the studies of bimanual co-ordination … it unlikely that the origin of that interference is limited to callosal influences.[97] (p. 12) [†]

The model received its most sophisticated experimental test using a combination of finger tapping and bimanual crank turning tasks, and two judgement tasks involving the left and right visual fields.[90] The experiment was designed to determine whether the model could explain natural recovery in terms of speech motor control maturation, specifically in the supplementary motor area. Participants were those who had recovered from stuttering, those with persistent stuttering, and controls. Consistent with previous findings, recovered stuttering participants and controls performed better than stuttering participants with the bimanual tasks. However, the stuttering and recovered stuttering participants performed equally poorly on the visual tasks, suggesting that the latter group retained residual interhemispheric problems.

Explanatory power

The model certainly can explain the manual sequencing anomalies that have been found in those who stutter. However, its explanatory power is weakened by not only the experiment with bimanual writing tasks described earlier, but by problems with it incorporating other research findings about writing tasks.[96,97] Additionally, explaining the influence of spoken language and stuttering variability seems problematic. That aside, the model does have considerable explanatory power. It is able to explain at least some of early epidemiology with the natural recovery study.[90] It might also explain the

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[†] The planum temporale is an anatomical structure associated with language function, and it typically is asymmetrical between the two hemispheres.

[†] Collosal refers to the corpus collosum, which is a large white matter fibre structure connecting the two hemispheres of the brain.

narrow range of stuttering onset in terms of early language development exposing the underlying hemispheric problem at that developmental stage. It can explain the fluency enhancing conditions because they could simplify speech motor activity to compensate for a problem with interhemispheric speech processing. Stuttering with wind instruments, nonsense words and bimanual tasks can be explained by the model, because it does not specify that the brain problems are speech specific.

RESEARCH FINDING	*EXPLANATORY POWER*
Behavioural diversity	*No*
The influence of spoken language	*No*
Epidemiology	*Yes*
Conditions that reduce or eliminate stuttering	*Yes*
Stuttering and wind musical instruments	*Yes*
Stuttering and nonsense words	*Yes*
Stuttering and bimanual motor sequences	*Yes*
Stuttering severity is variable	*No*
Stuttering and genetics	*Yes*
Brain structure and function	*Yes*

The future of the Interhemispheric Interference Model

Webster acknowledges in several publications that the broad notion underlying his theory has a long history. It dates back to the early years of the last century and Lee Edward Travis[†] who proposed the Orton-Travis theory of cerebral dominance,[89] the origins of which are apparent in a 1925 report about dyslexia.[98] In 1978 Travis recounted tests of the Orton-Travis theory that were presented in a 1931 textbook,[99] long before the arrival of scientific journals in the discipline:[100]

> When I published the cerebral dominance theory of stuttering in 1931, I presented in its support three laboratory findings: (1) reductions of the patellar tendox reflex latency, (2) reductions in the amplitude of tremors from extended right forefingers, and (3) profound alterations in the alternating phasic movements (opening and closing) of both hands, all during tonic stuttering blocks … (p. 278)

From all accounts, those were ground breaking research methods for the field.[101] In a 1978 publication,[100] Travis outlined how the theory was able to explain a series of research findings in the 1960s and 1970s. In 1986, just before his death, Travis asserted "the stutterer differs significantly from the normal speaker only in his neuro-anatomical organization for speaking" (p. 119).[102]

That historical background could prompt speculation about the future of this idea about stuttering causality. It appears to be an intrinsic part of thought and research in the discipline about the nature and cause of stuttering. It might be interpreted as an encouraging sign that, for more than 80 years now, the Interhemispheric Interference Model has resisted definitive experimental disproof and its future is auspicious. In fact, as discussed during the previous lecture, there is now evidence of anomalies in the corpus callosum—the white matter structure connecting brain hemispheres—among

[†] Lee Edward Travis is credited as the originator of the speech-language pathology discipline at the University of Iowa, before Wendell Johnson arrived there.

children who stutter. The current iteration of the model has had, overall, encouraging empirical verification and reasonable explanatory power. That might be interpreted as a sign that such a model, in some future iteration, may eventually be accepted as a causal explanation for the disorder.

So far, there has been little experimental interest in this theoretical explanation for stuttering apart from its developers. However, a recent report[103] used magnetoencephalography to study lateralisation of brain function with stuttering and control pre-schoolers during picture naming. No differences were reported for stuttering and control groups. Arguably, however, this result is not interpretable without evidence using similar research methods for older age groups, showing that they do have a lateralisation difference. It is the case, though, that magnetoencephalography has found interhemispheric differences with adults who stutter using other methods.[104,105,106]

The Covert Repair Hypothesis

The fundamental proposition

Drawing on Levelt's model

This theory draws on Levelt's well known model of speech production,[107,108] and another model of phonological coding.[109] Levelt's model, in short, comprises three linear processes. The first is the selection of a lexical concept to be spoken. The second is selection of a word in abstract form (lemma) and its grammatical encoding. Finally, a "mental syllabry" is accessed[110] and the word becomes a set of syllables ready for articulation.

Phonological coding errors

The central proposition with the Covert Repair Hypothesis is that those who stutter have phonological coding errors in this process of preparation for articulation, and that stuttering moments are covert attempts by the speaker to correct those errors before speech execution of the faulty plan.[111,112] Those who stutter have more errors than those who do not, and consequently they need to correct those errors more. Those corrections occur before the articulatory sequence occurs, and this leads to repeated movements and fixed postures during speech.

A continuum

The hypothesis does not state that there is anything qualitatively different between those who stutter and those who don't, merely that the former group have slower phonological coding and have more errors in the phonetic plan and hence need to make more corrections. In effect, the hypothesis proposes that stuttering and normal disfluency are on either ends of a continuum. As mentioned during Lecture One, this is known as the Continuity Hypothesis.[113]

Testability

Confirmations

The developers of the Covert Repair Hypothesis, the Dutch researchers Postma and Kolk, have presented support for the hypothesis using research methods involving speech errors of stuttering participants.[114,115] Other researchers have provided supportive data for the hypothesis,[116] reporting that 5 and 6 year old stuttering children had significantly lower "phonological memory" than control children, as measured with a non-word repetition task. According to a meta-analysis[117] of nine studies dealing with nonword repetition, that effect seems to be robust. Those same researchers provided further supportive data,[118] reporting that a group of stuttering children with a mean age 5 years 7 months had inferior performance to control children on sound blending and elision tasks. (Elision is removing a phoneme from one word to create a new word.) Other researchers have reported that 11 year old children who stutter are slower with a phoneme monitoring task,[119] and less accurate than controls with non-word repetition and phoneme elision tasks.[120] That research group reported that adults who stuttered were slower than controls in repeating non-word phoneme sequences.[121] With adults, researchers[122,123] have reported that a stuttering group showed unusual responses to phoneme monitoring tasks compared to a control group.