

The Thiamin Protocol

A Solution for a Third of the World's People who Stutter

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IMPORTANT NOTICE

This report is intended as a reference work only, not as a medical guide or a manual for self treatment. One should always seek competent medical advice before trying or even suggesting the supplements described in this report

Acknowledgements

For my wife,

Judith

and to the memory of my mother-in-law,

Jeane Stockheim

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Foreword

In this report I demonstrate that thiamin (vitamin B1) taken daily and in sufficient quantities can put stuttering into remission for almost a third of young adult males who stutter - regardless of how long they have stuttered or how severely.

It has now been five years since the original investigation and those who became fluent during that first study and continued to take the vitamin have remained fluent.

For these individuals stuttering was, and remains, a thiamin-deficiency disorder.

Chapter 1

Background and the accidental discovery

It was 1960 and I was a graduate student in speech science at Ohio State University, preparing to take an examination that, if passed, would allow me to start writing a doctoral dissertation. As part of my preparation, I decided to read the literature in speech pathology from its beginning. I was interested in learning more about the history of the profession and felt if I devoted some time each day to reading early journal articles, I would get a better understanding of the growth and direction the profession had taken since its inception some forty years earlier.

One day, during my reading, I happened upon a study that, in spite of its many flaws, seemed quite remarkable. It had to do with the effect of thiamin on the speech of children who stuttered.

Its title was “A Consideration of Thiamin Supplement in the Prevention of Stuttering in Preschool Children” and it was published in 1951 in a respected clinical journal. The title is somewhat of a misnomer because the study was not about prevention, but rather about whether thiamin could stop stuttering in children who had already begun. Also, some of the children were not preschool since their ages ranged from two to eight. Each child was given 30mg of thiamin hydrochloride, then the most common form of thiamin, or a placebo each day using a classic double-blind randomized presentation format.

Here were the findings:

“Observable speech improvement occurred in 55 percent of all cases.”

“There may have been improvement in an additional 20 percent of the cases.”

“No improvement could be claimed in 20 percent of the cases.”

“Of the two- and three-year-olds in the experiment, 80 percent were observably improved.”

“Only 50 percent of the four-year-olds were definitely improved.”

“It is doubtful that either of the two five-year-olds made much improvement.”

“Thiamin administration appeared to have no effect upon the speech pattern of the seven- and eight-year-olds.”

“In four cases where there was regression of speech fluency occurring following the cessation of thiamin therapy, there was improvement noted again after thiamin was resumed.”

“One of the cases which was reported as unimproved during thiamin therapy, showed an increase of tensions, hesitations and other stuttering symptoms after the thiamin had been stopped.”

“With only one exception, the cases that showed marked reduction of stuttering symptoms made the improvement within the first two weeks of treatment.”

I remember saying to myself that even if these findings were remotely true, they would be amazing. Of greatest significance was the author's finding was that for two- and three- year-olds, stuttering could be substantially improved for more than three quarters of them in less than two weeks! Now while it is true that most children will eventually improve to the point where they outgrow their stuttering, such improvement doesn't usually happen in two weeks. It was clear that something real was happening.

As I read the study, I imagined the pages of the journal filled, in subsequent issues, with many discussions of the results of this study. But no, nothing. It was if the study hadn't been published.

Perhaps it had to do with the extremely poor quality of its design. There were many mistakes. But still, nothing ever appeared in the journal, not even a criticism of it. I was amazed but since I had, at that time, just a passing interest in stuttering and other things to do, I simply noted this piece of information and continued my reading of the early literature in speech pathology in preparation for taking the doctoral qualifying exams in speech science.

Chapter 2

NYU Medical School and another accidental discovery

We move now to 1974, some 12 years after receiving my doctorate. During that time I had been doing basic research, moving up the academic ladder, and publishing articles that, while interesting in an abstract sense, had no clinical relevance. Then, in 1974, I decided to shift my focus to clinical issues. I reexamined the speech pathology literature and decided that the area of stuttering, for several reasons, would be fertile ground for investigation.

First, in spite of the fact that it had a huge research literature, no one seemed to have a good understanding of what was going on. And second, the enigmatic nature of the disorder intrigued me. (For example, people who stutter often stop stuttering immediately and completely when they go into an empty room and speak to themselves out loud alone).

Then there was a very excellent text written by an eminent expert in stuttering named Charles Van Riper. The text was called The Nature of Stuttering and it had well over a thousand references to research studies that had been conducted over the years on all aspects of stuttering. I decided to examine the book in great detail to see if I could use the findings to generate a model of stuttering that would account for most, if not all, of the features described by Van Riper.

Coinciding in time with this came an offer to join the faculty at New York University Medical School as a research professor. This was quite appealing but I had one proviso: In addition to doing research on the speech of people born with diverse cranio-facial anomalies (such as cleft lip and palate), I also wanted time to do research into stuttering. They agreed and I took the position.

My appointment at the Medical School was at the Institute of Reconstructive Plastic Surgery. I began to examine a whole host of speech problems arising from a variety of genetically-based malformations of the head. A previous interest in the anatomy and physiology of the speech mechanism stood me in good stead as I began to collaborate with several surgeons on the design of new operations to improve the speech of people born with cleft palate.

One such operation is called a pharyngeal flap. In this procedure, a flap of tissue is undermined from the back throat wall and attached to the palate to close the cleft. I was assigned the task of using ultrasound to study the preoperative movements of the throat during speech to help in the determination of an optimum donor site for the flap.

One of the cleft palate patients stuttered and when the ultrasound-generating device was applied to the side of the neck, something happened as the patient spoke that was not expected. The throat seemed to constrict just before and during most stutter events. The behavior was relatively consistent and perked my attention. So I contacted the speech clinic at the medical school and over the next several months obtained five more individuals who stuttered (but with no cleft palates) and, again using ultrasound, made similar observations.

Further research revealed that the center of this activity was at the base of the throat in a structure called the larynx and, within that structure, the vocal cords. The thought occurred that these activities, since they not only coincided in time with the stutter but also often preceded it, might somehow have been provoking or triggering it.

It is well known that the vocal cords are, in part, controlled by the breathing centers of the brain. So it seemed logical that some sort of breathing maneuver performed just before speaking might be capable of exerting a positive influence on reducing the apparent effort associated with these constricting vocal cord movements. After experimenting with a variety of breathing techniques, one was settled upon that reduced the frequency of stuttering by almost 70%.

Later it was discovered that a combination of this new breathing technique coupled with a slowed first word of each sentence could further reduce the frequency of stuttering to almost 94%. I wrote a book about this combined approach and this led eventually to the establishment of The National Center For Stuttering.

The functions of The Center were to treat clients and train speech pathologists. During the 1980s and 90s over eight hundred speech therapists were trained in what was later called the Airflow Technique. In the process, over fifteen thousand individuals who stuttered were seen for treatment, making The National Center For Stuttering, at that time, the largest treatment and training facility of its kind in the world.

Chapter 3

Thiamin and stuttering

The Center, during the peak of its operation, maintained a hotline number for the general public. The hotline was in existence from 1977 until 2010. When parents called requesting guidance for young children who had recently begun to stutter, along with the usual stress-reducing recommendations, the Center recommended that the child be given 30mg of thiamin daily - provided the child was between the ages of two and four and the parent had first obtained permission from the pediatrician.

Over the years, as a result of this recommendation, a body of anecdotal information was accumulated regarding early childhood stuttering and thiamin. Here were the relevant observations: If thiamin proved to be effective, the result occurred within two weeks and was dramatic. It was as if a 'switch' had been thrown, and the stuttering, as reported in the aforementioned 1951 study, was either markedly reduced or eliminated. Almost 60 percent of the children showed the 'switch effect'. For those children, the possibility of a placebo seemed unlikely. On the other hand, if two weeks passed with no improvement, none would likely occur, regardless of the length of time the supplement was taken.

In no instance was more than 30mg of the vitamin per day recommended.

In 2002 the Center began informal explorations using larger amounts of thiamin with adults who stuttered. It was from these explorations that, several years later, a formal study was undertaken. What follows is a detailed description of the formal study (explanations of some terms for the non-

specialist are shown in parentheses). Later, we will discuss the enhancements that have subsequently been made to it to increase the percentage of those showing positive results.

The study

In this study we examined the percent syllables stuttered from two groups of presumed equivalent adult males who stuttered, one taking 300 milligrams of thiamin (vitamin B1) daily and the other, a placebo.

In all, 38 males between the ages of 21 and 37 participated. All had stuttered since early childhood. All were native speakers of American English, all had at least a high school education and none was receiving speech therapy at the time of the study.

Excluded was anyone with a medical condition that would interfere with thiamin absorption or any medication or dietary habits that could negate the effects of ingesting the vitamin.

The experiment lasted for two weeks. This time period was selected because prior clinical experience and the earlier 1951 report had indicated that positive speech effects of thiamin, if they were to occur, would take place within the first two weeks. In addition, prior clinical experience had also shown that 300mg daily of the vitamin was the level at which positive speech effects for adult males might be expected to occur.

Accordingly, all subjects were instructed to take one pill with each meal for a total of three pills daily. Each pill contained either 100mg of thiamin hydrochloride (the form of thiamin used in the earlier study) or a placebo. Subjects were assigned randomly to each of the two groups and received either the vitamin or the placebo in a classic randomized, double blind presentation format. (Double blind means that neither the subjects nor the experimenter knew whether they were taking the vitamin or the placebo until after the experiment was completed. This is to prevent experimenter bias from influencing a subject's performance and to spread the power of suggestion evenly among the subjects.)

Prior to starting the protocol, all subjects were administered a modified version of the percent syllables stuttered portion of the SSI (Stuttering Severity Instrument) test, the most commonly used instrument for quantifying stuttered speech. Samples were obtained from three speaking situations: reading, face-to-face conversation and telephone calls. The percent syllables stuttered constituted an average derived from the three speaking situations. This average, obtained before and after the experimental intervention, constituted the data for the statistical analysis. (The purpose of a statistical analysis is to determine whether the average stuttered syllables difference found before and after the experiment could have occurred simply by chance or truly represented a difference that was so great it was unlikely it could have occurred by chance. The result is usually expressed in how confident we are that the difference was truly due to the intervention being studied.)

In all instances, judgments of stutter events were made independently from video recordings by two clinicians with a total of 47 years working with individuals who stutter. Video presentation was considered essential since, for some of the subjects, a number of the stutter events were silent struggles.

Results

Inter- and intra-judge reliability correlation coefficients (a statistical test) for repeated judgments were greater than 94%, indicating that the two raters agreed well with themselves and each other.

The average stuttered syllables for the two groups prior to the start of the study were not significantly different (meaning that both randomly selected groups probably came from the same population). The average stuttered syllables for the control group (the ones taking the placebo) pre- and post-trial were also not significantly different. However, the average stuttered syllables for the experimental group (the ones taking the vitamin) pre- and post-trial were significantly different ($p < .01$). (This means that the difference before and after taking the vitamin was so great that it could have occurred by chance less than once in a hundred repeats of the experiment.)

A closer examination of the data revealed that the 'switch effect' was present for 6 of the 19 subjects receiving the supplement. Pre- supplement, the average percent syllables for this group of six had been 9.1%. Post-supplement, it was less

than 1%, with five of the six subjects showing no stuttering whatsoever. Deleting the data for the 'switch' group, and comparing the pre- and post-supplement averages for the remaining 13 subjects yielded no significant difference.

Discussion

Within the limits of this study, one may conclude that for slightly less than a third of young adult males who stutter between the ages of 21 and 37, 300mg of thiamin, taken daily, in the absence of precluding medical conditions, may be of significant value in either substantially reducing or eliminating their stuttering. This finding, if substantiated by others, would seem to have profound implications for the approximately 1% of the world's population that stutters.

A cohort follow-up study

The six subjects showing the 'switch effect' have now been followed for five years. They form a group called *a cohort*. Cohorts are groups of subjects that share a common attribute – in this instance, a positive effect for Thiamin. The cohort is followed over a period of time. A cohort study was considered crucial to establishing the validity of the original finding because it can provide long-term evidence that either supports or refutes the original finding.

While the randomized controlled trial in the original study is considered a superior way of doing science, a prospective co-

hort study, even with all its uncontrolled variables, is still a powerful way of reinforcing the validity of the findings of the original study - providing, of course, the cohort continues to share the attribute that was used to establish it in the first place.

There is another reason a cohort study is important. The results and conclusion derived in the original study were based on a very small number of subjects. While some may object to this, saying that a small number can easily result in an unrecognized sampling error, the positive outcome of a cohort study can be used to demonstrate that the finding and conclusion derived from the original study are probably correct.

Also, bear in mind that many statistical tests have been developed for the express purpose of examining data from small numbers of experimental subjects. These tests, called small score statistics, have been shown to yield valid results. Of course, the trade-off for using small numbers of subjects is that the difference between the experimental conditions has to be much greater to reach statistical significance.

The cohort follow-up was conducted by telephone at approximate two-week intervals, now over a five-year period. The original six subjects reported that as long as they continued to take the vitamin there was no return of their stuttering. Some indicated that when they failed to take the vitamin (usually because they had run out), their stuttering would return (usually after a few days). In all instances, however, the stuttering was

reversed with the return to taking the vitamin.

There is one additional piece of evidence that lends credence to this finding - even though it is anecdotal. There is a blog on the internet called: *stuttersense* (at <http://blogspot.com>) that is largely devoted to the Airflow Technique that has, at my suggestion, started the discussion of thiamin and stuttering. Since the discussion began, the blog conducted two surveys among its readers who elected to try thiamin and found that among 74 respondents who indicated they had taken the specified amount of thiamin (300mg per day), 29 (38%) reported 'dramatic improvements' in their speech.

Chapter 4

More about thiamin

The recommended dietary allowance for adult males for thiamin is 1.5mg per day. The positive effects reported in the experiment required an amount 200 times greater. One might logically raise the issue of toxicity. Fortunately, the issue has been addressed repeatedly and the answer provided by virtually all experts in such matters is that in the amounts taken orally, there have been virtually no adverse reactions recorded. Thiamin is one of the water-soluble vitamins and the common belief is that any excess is simply urinated out. The Food and Nutrition Board of the National Academy of Sciences has indicated that there is no upper limit of toxicity for this vitamin. The Food and Drug Administration lists thiamin hydrochloride among its GRAS (Generally Recognized As Safe) supplements. A number of studies, investigating a variety of disorders, have used as much as 9000 milligrams (9 grams) per day for prolonged periods of time (months), with no negative consequences. Various international organizations have generally agreed that vitamin B1, when taken orally, in amounts up to and including 500mg per day poses no threat to the health of the general public – although, when pressed, they admit that this number is arbitrary and could just as well have been higher.

However, as with all supplements, it is good policy to always check first with one's doctor. One may be taking medications that interact adversely or there may be an unsuspected allergy resulting in an unwanted reaction. Fortunately, both are relatively rare. It is more likely that the medications will interfere with the absorption of Thiamin rather than vice versa.

There is one additional recommendation: be sure to take the form of thiamin called thiamin hydrochloride. Thiamin Hydrochloride was the compound used in the 1951 study and the Center's study with adults. There are a number of other forms of thiamin, but none of the opinions offered in this book can relate to them because they have not been studied.

It has been demonstrated that the maximum amount of thiamin that can be used by the body at any one time is 5mg. Indeed, the total amount of thiamin that typically can be found in the body is 30mg. So the question remains: why 300mg? The answer probably lies somewhere in the extremely complex process of absorbing thiamin and getting it past a structure called the blood-brain barrier.

The blood-brain barrier is a membrane surrounding brain cells designed to protect them from infections caused by bacteria circulating in the blood as well as from certain environmental hazards. The design of the barrier resists these intrusions but lets other substances pass. For example, most of us are aware of the fact that alcohol easily passes the barrier. What we are not aware of is that all the water-soluble vitamins, including thiamin hydrochloride, cannot pass the barrier.

But there are two processes that can be brought to bear to bring thiamin into the brain. One makes use of chemical carriers that transport a small amount of thiamin across the barrier. But that source is saturable (essentially limited in its capacity). There is another process that is much less saturable. It is called diffusion and it occurs when chemicals in high concentration on one side of a membrane are passively driven across that membrane to equalize the concentrations on both sides of the membrane.

It has been shown that if one takes 100mg of thiamin three times a day, its density in the blood will continue to rise over the first 7 – 10 days, finally reaching a steady state after that time. Interestingly, it would appear that total levels of 100 or 200mg of thiamin per day taken orally simply do not produce sufficient blood densities to diffuse enough of the vitamin across the barrier to produce much of an effect upon stuttering, while at 300mg per day, for almost a third of adult males, it does.

Thus choosing 300mg a day as the level for the adults in the Center's study came about as a result of trying different levels of the vitamin clinically and choosing the lowest level that would appear to make a difference in stuttered speech for a significant number of individuals.

Interestingly, if one visits a vitamin shop and looks at the levels of thiamin available, one may be surprised to see a large number of bottles marked 500mg. These are routinely recommended by physicians for alcoholics. Alcohol gravely depletes the body of thiamin by interacting with it in many ways thereby effectively eliminating it.

There are other issues, besides alcohol, that can have negative impacts as well. Here, for example, is an enumeration of just a few of the many conditions that have been shown to interfere with thiamin absorption:

Alzheimer's Disease

Anorexia

Diabetes

Hyperthyroidism

Infection

Malabsorption syndrome

Inadequate thiamine intake can also occur with diets containing a lot of the following:

Blueberries, red beets and red cabbage

Brussel Sprouts

Coffee and tea

Foods high in simple carbohydrates

Sulfites (a food preservative)

White rice, raw freshwater fish and raw shellfish

Certain medications can deplete vitamin B1. These include:

Acid blockers

Antacids

Antibiotics

Anticonvulsants

Antivirals

Aromatase inhibitors

Blood pressure drugs

Bronchodilators

Cardiac glycosides

Diuretics

Hormone replacement therapy

Oral contraceptives

Sulfonamides

Even storing, processing and cooking can have negative consequences on thiamin content. For example:

Thiamin is not stable in storage

Conventional cooking reduces the thiamin content of food by 20-50%

Rice loses about 80% of its thiamin when converted from brown to white

A similar loss occurs going from whole wheat to white flour

The modern food industry has responded to the fragility of thiamin by moving toward thiamin enrichment of grain products. In a sense, this has been effective – for without it, thiamin deficiency would be a much more common phenomenon.

People with heart failure and gastrointestinal disease have an increased risk of thiamin deficiency. In each of these groups, restoring normal thiamin levels may prevent some of the worst complications of the disease.

Even in the absence of any of these diseases, elderly people are at increased risk of thiamin deficiency. This is, at least in part, due to a reduction in the ability to absorb dietary thiamin that occurs as we age.

Popular fad diets that greatly reduce intake of grains and legumes make thiamin deficiency more likely. For instance, after eight weeks on the Atkins diet, people following this popular low-carbohydrate strategy saw their thiamin intake drop by 40%.

As can be seen from the above, even the recommended dietary allowance (RDA) of 1.2mg of thiamin per day, given the minefield of obstacles standing in its way, may be difficult to achieve. Some nutritionists consider thiamin to be the most deficient vitamin in the diet.

Chapter 5

The trigger for stuttering

This chapter describes the trigger for stuttering, how it arises and develops in childhood, the stresses that provoke it and the ways individuals eventually learn to cope with it. This sets the stage for the later explanation of the role of thiamin in producing fluent speech. It also explains why a technique (Intent Therapy), also to be described later, works as well as it does.

Before beginning, one important point must be made:

THIAMIN CAUSES ROUGHLY ONE THIRD OF ADULT MALES WHO STUTTER TO GO INTO REMISSION NOT BECAUSE IT ADDRESSES STUTTERING BUT BECAUSE IT ADDRESSES THE TRIGGER FOR STUTTERING.

We begin with an abstract. Words in parenthesis throughout the text are used to clarify concepts for the non-specialist; numbers refer to a reference list in the appendix. The chapter is segmented into eleven parts. It is recommended that the reader take their time with each part because many of the concepts presented are alternative.

Abstract

Using information obtained from studies of early childhood stuttering, carefully documented clinical observations and

data on the physiology of normal speech, the hypothesis is drawn that the trigger for stuttering is a pattern of sensory impulses that, under conditions of appropriate and sufficient stress, arises from receptors within and around the vocal cords to signal the brain that a specific level and concordance of speech-arresting muscle tensions has been attained at the vocal cords.

The behaviors that characterize stuttering are learned attempts to prevent or release these speech-arresting levels of vocal cord tension. The learned behaviors can be diverse. For example, at the onset of stuttering in children, the most typical immediate response to a sudden vocal cord immobilization is a syllable repetition. Such repetitions can occur with varying degrees of tension and frequency and often become habits.

After a period of time, which can vary greatly, the child may become aware of and react to these repetitions by engaging in one or more of a number of behaviors. These can be avoidance activities such as changing words, forceful confrontational attacks such as violent head shaking, or more subtle pre-speech maneuvers such as swallowing, inhaling deeply or speaking on expiratory reserve volume (the very last bit of air left in the lungs). All of these behaviors can become learned because all have the power to either prevent the unique pattern of sensory impulses (the trigger) from occurring or to extricate from it once it has occurred – and thus are rewarding.

The implication of this is that the treatment of stuttering would best be served by developing appropriate techniques for preventing the trigger from occurring rather than attempting to deal with its learned responses.

Part 1: The introduction

Stuttering is often defined in terms of its overt symptoms. Typically these are described as repetitions, prolongations and blocks⁽¹⁾ - although a great many other behaviors have been described as well. In concert with this definition, most people tend to think of stuttering as a disorder of speech production.⁽²⁾ As a result, cognitive issues, more specifically, the thoughts of people who stutter, tend to receive much less attention.⁽³⁾

One important cognitive issue for people who stutter is their ability to look ahead and 'see' feared sounds, words, and speaking situations approaching.⁽⁴⁾ This anticipatory mental activity, called *scanning*, leads to a variety of behaviors, one of which is the avoidance of feared words and feared speaking situations.⁽⁵⁾ Clinical experience indicates that scanning and avoiding are not present at the onset of stuttering but develop over time to become habits.⁽⁶⁾

Scanning and avoiding are not the only habits. A number of studies have shown that most, if not all, of the overt struggles that characterize stuttering are also learned.⁽⁷⁾ Part of this understanding comes from learning-theory models of

stuttering⁽⁸⁾ and part as a result of the successful use of behavior modification techniques in treatment.⁽⁹⁾ (Behavior modification techniques are used to change learned behaviors.)

If stuttering is composed of learned struggle, scanning and avoidance behaviors, an adequate definition must include all three. For purposes of this explanation, stuttering is defined as:

All the things that people who stutter do when they get 'stuck' in their speech and/or all the things that people who stutter do to avoid getting 'stuck' in their speech.

This definition may appear simplistic, but notice what it accomplishes. First, it takes the point of view of the person who stutters, not the point of view of an observer. Second, it takes an exclusive orientation away from a catalog of overt struggle behaviors and provides for the very real possibility of important cognitive components. Finally, it strongly emphasizes that overt stuttering, scanning and avoidance behaviors are responses to something else - the 'getting stuck' or, what is hypothesized in this chapter to be the event that elicits the trigger for stuttering.

To begin our quest for the trigger, we begin with a *rewording* of a number of questions that have repeatedly been asked about overt stuttering. In these questions, the concept of a 'trigger' for stuttering is substituted for the traditional focus upon behavioral signs. So, for example, why does the *trig-*

ger for stuttering occur more often among males than females, why does it tend to run in families, why is it absent or markedly reduced when a person who stutters talks to themselves out loud alone, or when they sing, or whisper, or speak to the rhythm of a metronome, or speak chorally, or against a masking noise, or when using a foreign accent or talking to pets?⁽¹⁰⁾ And why does the trigger typically not occur when, in the course of speech development, a child is using single words to express himself - only to appear suddenly months or years later when the child begins using sentences?⁽¹¹⁾ And when it does appear, why does it typically manifest itself at the beginnings of sentences rather than being evenly distributed throughout?⁽¹²⁾

And then there are the interesting issues raised by the cognitive component. For example, a child who recently started using full sentences suddenly begins to stutter. In response, he reverts to using single words to convey his ideas. This child is, by the above definition, still stuttering since he is responding to his awareness of his stuttering by electing to avoid it by not using sentences. How are we to react to this child?

And, later in life, if this child, as an adult, masters word substitution and situation avoidance to the point where he never stutters overtly but still lives in constant fear of inadvertently stuttering - are we not to call him a person who stutters? These are some of the clinical questions that arise when a traditional overt-symptom-response-only approach is taken

with respect to the understanding and treatment of stuttering. These issues lead to an inevitable conclusion, herein offered as a question:

If overt stuttering, scanning and avoiding are learned responses to the occurrence or anticipation of a trigger, wouldn't it be better to ignore these learned responses and treat the trigger instead?

The following illustrates this alternative way of thinking. Let us equate overt stuttering with the knee-jerk reflex. The striking of the patellar tendon (the place just under the kneecap) causes a unique pattern of sensory impulses to arise from that area, go up the leg and enter the spinal cord. This pattern of impulses is the trigger. The trigger stimulates other nerves which, in turn, send a message back out to the leg to cause a muscle in the thigh to contract which causes the foot to fly up. The foot flying up is the response and, in this example, is analogous to a stutter.

Imagine, however, that the patellar tendon cannot be seen. All that can be seen is the foot, and its flying movements are inappropriate, unwanted and cause considerable stress whenever they occur. Attempts by therapists to hold the foot down or to modify its movements (analogous to traditional speech therapy) often fail. One day someone discovers the patellar tendon and observes the relationship between it being struck and the subsequent foot-flying event. Knowledge of this relationship quickly leads to a solution.

One might argue about the validity of the above example, but it still retains its power to explain what is here contended to be a common mistake in therapy (attempting to treat stuttering instead of its trigger). It also explains a similar and common mistake made by researchers who try to uncover the core brain mechanisms underlying stuttering by exhaustively mapping the brain's equivalents to what are essentially learned responses.

There is another common mistake. People who stutter often believe 'stress' to be the trigger for their stuttering. To them, it seems a simple stimulus-response event. For example, a feared speaking situation, such as talking on the telephone, elicits a high degree of anticipatory stress (a cognitive response conditioned by prior experience) which, in turn, elicits a stutter. The logical assumption, derived from the experience, is that anticipatory stress causes stuttering. A closed loop cycle is proposed, with anticipatory stress triggering stuttering which, in turn, reinforces the anticipatory stress. In other words, a self-fulfilling prophecy.

But both psychotherapy and speech therapy have long histories of not being very successful in the treatment of chronic stuttering.^(13, 14) Both disciplines often rationalize their relative lack of success by saying that since stuttering is a complex, multi-factorial problem, starting early in life and of long-standing duration, it is highly resistant to permanent extinction. Rarely, if ever, is there mention of an intervening step, such as a triggering event, that might be going unaddressed.

It is the contention of this chapter that there is a trigger for stuttering and that it always occurs during the time period between the occurrence of stress and an overt stutter or, in the case of an avoidance response, it is a cognitive event, the perceived threat of a stutter, which occurs in response to stress, that evokes the avoidance response.

Part 2: The origin of the trigger

In our quest for the origin of the trigger, we examine the beginnings of stuttering. We do so for one reason: unencumbered by the overlay of years of conditioning, important clues to the trigger reveal themselves more readily at this early stage.

We begin with general observations about the onset of stuttering. The first is that most stuttering begins between the ages of two and five, with a major peak of onset of occurrence between two and a half and three and a half years of age.⁽¹⁵⁾ The second is that most children who stutter have been speaking fluently for a period of time before their stuttering begins. The average length of time the child has been speaking fluently before the onset of stuttering is seventeen months, but it can vary widely.⁽¹⁶⁾ Third, many children who stutter can have periods when they are totally fluent. These periods, sometimes called remissions, can last for days, weeks, months, years or, in the case of outgrowth, forever.⁽¹⁷⁾ Fourth, at the onset, most stuttering appears at the beginnings of phrases or sentences.⁽¹⁸⁾ And fifth, by far, the most

common initial expressions of stuttering are repetitions.⁽¹⁹⁾

Given these observations, it is obvious that the trigger, whatever it is, is not occurring all or even most of the time. And if the trigger is a response to stress, then the stress apparently comes and goes quickly, and often without apparent rhyme or reason. It is strange to think, for example, that a stress and its associated trigger could be present on the first word of a sentence and not on the rest of the sentence, or that Johnny could be overheard talking fluently with his action figures when playing alone in the family room, only to stutter instantly when someone comes into the room and asks him a question.

If we think for a moment about the concept of a trigger we see that for one to occur, a threshold of some sort must be crossed. Whether it be the response of a thermostat to a certain temperature, triggering the turning on of a furnace or an air conditioner, or an ion channel in a cell's membrane, which is shut when the potential is near its resting potential, only to open suddenly when the potential increases to a precisely defined level, the concept of a trigger always implies the concept of a threshold.

For the purposes of this chapter, the threshold for the trigger is defined as: *A level of physiological activity capable of eliciting an overt stutter or of provoking an avoidance response.* When the magnitude of the physiological activity *crosses the threshold*, an overt stutter response occurs; when it *ap-*

proaches the threshold, its threat potential, as a result of prior conditioning, can initiate an avoidance response - but obviously only in children and adults who scan.

At the onset of stuttering, there is no scanning. Anticipatory stress, as a learned response, takes time to develop. This means that at the onset of stuttering there is no attempt to avoid supra- (above) threshold trigger events. And since, without scanning, the trigger events are essentially all unanticipated, they evoke what are probably the most basic of responses.

It is a contention of this chapter that the most basic of responses are the ones that occur whenever there is a sudden, unanticipated stoppage of a learned and generally smoothly functioning forward progression of preprogrammed neuromuscular events.

So, for example, both speech and walking are forward-movement processes. Both have beginnings, midpoints, and endings. Walking and speaking are both learned activities and both have central nervous system mechanisms that plan, execute and monitor the respective behaviors. Once learned, both behaviors appear to be automatic and the users of such behaviors fully expect the ongoing nature of the behaviors to proceed smoothly and without interruption.

Interesting things happen when an unanticipated interruption occurs to a normally smoothly functioning ongoing neuro-

muscular activity. What follows is a personal example:

A number of years ago I was traveling by train from New York to Washington, DC. I was sitting in a coach next to the dining car. As people passed through the coach on their way to the dining car, they were required to press a bar on the door at the end of the coach that would cause the door to slide open. Sitting near that door, I noticed that something was wrong with the bar's actuating mechanism. When the bar was pressed, the door did not open immediately, as expected, but only after a delay of several seconds. I watched how people reacted to this sudden, unexpected stoppage of a normal-on-going-forward-movement process (the progression of walking to the door, pressing a bar, expecting the door to open immediately and then continuing to walk).

There were three types of responses. First, and by far the most frequent, were those individuals who repeatedly pressed the bar in an effort to get the door to open. Call these actions repetitions. The second group, much less numerous than the first, were those who applied pressure continuously to the bar in the effort to open the door. Call these actions prolongations. And the third group were those who paused, seemingly impatiently waiting for the door to open so they could continue walking. Call these inactions (or hesitations) blocks.

Here, I thought, was an obvious and efficient explanation for the beginning of developmental stuttering. As indicated earli-

er, children had been speaking for a while prior to the onset of stuttering. The learning process had progressed without undue incident. Speech was becoming smoother and more automatic each day. Suddenly, in the midst of this ongoing movement process, the progress was abruptly and unexpectedly halted. (The equivalent to the 'getting stuck' part of the definition). The reaction to this event was as non-thinking, automatic and similar as the behaviors exhibited by my fellow travelers on the train that day: mostly repetitions with occasional prolongations and/or hesitations. Interestingly, research on the earliest stuttering signs show this to be precisely the case, with the onset of signs typically being mostly repetitions, with occasional prolongations and/or hesitations. ⁽²⁰⁾

One final point to the story. One of my fellow travelers that day, a young man in his late teens, dressed in what I would call 'Elvis' attire, had occasion to go to the dining car a second time. He was hard to miss. I noticed that as he approached the bar, almost without breaking stride, he lifted his foot and gave the bar a forceful kick. One may conclude from this that on the basis of apparently just a single prior experience (*one-trial learning*), this young man had devised a strategy for dealing with his anticipation of a trigger. Here, I thought, was the analog for quickly-learned severe, pre-emptive (anticipatory) stuttering.

Part 3: Early responses to repetitions

We shall now examine some of the early behaviors devel-

oped by children to deal with repetitions since they are, by far, the most common initial expressions of stuttering. Note that repetitions can persist for variable periods of time (from a day to years) in unmodified form. This is due to the fact that since they often work to release the 'stuck' condition, they are rewarded and thus learned.

Eventually, however, the child might react to the repetitions, find them unacceptable, and seek to eliminate them. A number of options are available. He can, if he scans, choose to eliminate the repetitions by looking ahead and changing feared words and avoiding feared speaking situations. Or he might confront the 'getting stuck' head-on with forceful struggles - as 'Elvis' did on the train. Or he might replace the repetitions with other, seemingly less obvious struggle behaviors. It is this last group that interests us as we again look to certain behaviors that appear early on in the development of stuttering but do not seem to persist into the adult form of the disorder.

There are three of note. The first is swallowing. Children who stutter will sometimes swallow just before speaking.⁽²¹⁾ It is as if the process of swallowing somehow facilitates the onset of speech production. If one asks a bright 7-year-old who has been exhibiting this behavior for a while: "Does swallowing always work?", he will sometimes smile, revealing that he fully understands the question and usually answers: "Not always." To which, if one asks, "Does it sometimes take two or three swallows to get things going?", he will often say "yes".

So swallowing, when it occurs, is usually a conscious anticipatory behavior designed to either disable or prevent a trigger from occurring.

A second behavior is the use of inspiratory gestures:⁽²²⁾ brief, deep, rapid inhalations just before the onset of speech. Sometimes these behaviors occur as a series of sniffs, sometimes as deep oral inhalations. Again, they appear just before the beginnings of sentences and appear to facilitate speech onsets. They are less prominent than swallowing and are often seemingly employed without conscious awareness.

The third behavior is speaking on expiratory reserve volume or what is commonly called: supplemental air.⁽²³⁾ The child will appear to exhale almost completely and then, on the minimum amount of air left in the lungs, produce as many words as he can. Again, this is apparently used to initiate speech. Owing to the forceful nature of this expiratory maneuver, the child is often aware of using it.

Notice that since swallowing, rapid, deep inspirations, and speaking on supplemental air are techniques employed by these children in the silence *before* speech begins, it would appear they are being used to prevent or disable a trigger. This interpretation gains credence by the fact that when these behaviors are employed to deal with repetitions, they usually cease and the speech that emerges usually sounds normal.

Part 4: The silence before speech

Since the trigger occurs in response to stress and both apparently occur in the silence before speech, it would seem reasonable to consider what events might be taking place during this time period that could be interpreted as stressful. There are several obvious candidates. To begin, if the child is speaking in sentences, as they typically are when stuttering begins, there are a number of choices that have to be made before speech can begin. The vocabulary, grammar, syntax, intonation and linguistic stress of the entire sentence have to be selected, integrated and sequenced.⁽²⁴⁾ And these activities, and the neurological processes that sub-serve them, have to take place in fractions of a second and in a brain that, on average, is two and a half to three and a half years old.

It is a contention of this chapter that these complex and diverse activities, occurring quickly and simultaneously in a relatively immature central nervous system, constitute multiple stresses that express themselves as heightened levels of muscle tension in the vocal tract during the silence before speech begins.

In predisposed individuals, these heightened levels of muscle tension can cross a threshold yielding a potentially speech-arresting condition. The repetitions that then typically ensue are not 'problems' saying the first word or syllable; they are, instead, the normal responses to a sudden, unanticipated stoppage of a learned and relatively smoothly functioning

forward progression of preprogrammed neuromuscular activities. *They are the repeated pressing of the bar required to open the 'stuck door' at the end of the railway car.*

Part 5: Other sources of 'stress' in the silence before speech

There are three other, less obvious sources of 'stress' that occur during the silence before speech. The first is called *coarticulation*.⁽²⁵⁾ Co-articulation is the influence one sound has upon another during ongoing speech. For example, in the word 'queen' which is written in phonetics as [kwɪn], notice that before the [k] is made, the lips are already rounded for the [w]. This occurs because the [k] requires no lip involvement and so the lip rounding for the [w] is free to move forward in time to facilitate the smooth transition from the [k] to the [w]. This happens continuously and is a normal feature of speech production.

But co-articulation can be more extensive. It has been shown, for example, that given the correct phonetic environment, the effect of the articulatory (speech) requirements for the last word of a sentence can sometimes be detected on the first word of that sentence.⁽²⁶⁾ This means that continuous speech is not merely a concatenation of one word independently following another in time, but instead, prior to the onset of the sentence, that is, during the silence that precedes it, a large part of the articulatory organization for the entire sentence can take place.

The second not-so-obvious event occurring in the silence before speech is the 'stress' associated with speaking too rapidly. More specifically, it is the speed of the first word. It is called *speed stress* and it manifests itself in the following manner: As the length of a sentence is increased, the duration of each of the syllables in the sentence decreases.⁽²⁷⁾ This means, that as the sentence gets longer, the speaker talks more rapidly. And although this is true throughout the sentence, nowhere is it apparently more prominent than on the first word of the sentence.⁽²⁸⁾ So the speed of the first word is particularly sensitive to the number of words in the sentence. This is a normal feature of speech production.

Let us consider what happens as the sentence is lengthened and the first word is spoken more rapidly. To understand this, imagine we are in a physiology lab and are going to study the behavior of the triceps muscle as it functions in an arm-extension maneuver. Surface electrodes have been attached to the muscle to record its electrical activity. The three conditions to be studied are a slow-speed extension of the arm, a moderate-speed extension, and a rapid one. The activity to be examined in detail is the behavior of the triceps muscle a fifth of a second *before* the movement begins.

The test is completed and the data collected and analyzed. Not surprisingly, one finds the greatest degree of electrical activity, that is, the most tension on the triceps, occurs before the rapid extension, a lesser degree of tension before the moderate-speed extension, and the least before the slowest-

speed extension.

The basic principle of physiology demonstrated is that in order to have progressively greater initial velocities, one must recruit (involve) progressively more muscle fibers *before* the movement begins and that this recruitment is reflected in the greater average tensions recorded *prior* to the onset of the faster extensions.

The same is true during the silence before a sentence versus the silence before a word spoken in isolation. The tension is always greater before the sentence and, within limits, the longer the sentence, the greater the pre-speech tension. Research shows that the longer the sentence, the greater the amount of stuttering⁽²⁹⁾ and that most early onset stuttering occurs on the first word of phrases or sentences.⁽³⁰⁾

Stocker developed a treatment for young children based on this understanding.⁽³¹⁾ Her approach was to have children return to using single- word utterances to express what formerly had been expressed as stuttered sentences. This elemental form of speech, relieved of a great deal of its pre-speech linguistic programming and most of its speed stress, was fluent. Then, gradually, as fluency became more firmly established, she proceeded to two-word utterances, three-word utterances and so on - guiding the child through to progressively greater levels of linguistic complexity and speed stress.

The third event of importance occurring in the silence before

speech is called the *pre-phonatory tuning of the larynx*.⁽³²⁾ There are two types. In one type, if speech is to start with the correct pitch and loudness, the vocal cords must assume specific postures and tensions before speech. Were pre-phonatory tuning of the larynx not present, speech onsets would be marked by rapid shifts of vocal cord activity, as cortical (brain) control of the larynx, making use of auditory feedback (what was heard), *hunted* to find the right initial pitch and loudness. A moment's reflection indicates this is not the case and a simple example convincingly makes the point: If a person is asked to speak in falsetto, they know, *a priori* (before the sound is even made), using various proprioceptors (non-audible sensations from the body), they can do it. There is no need for the person to hunt for falsetto voice after phonation has begun.

The second type is the position required of the vocal cords for the first sound in an utterance. As indicated earlier, it is not always the case that the vocal cords adduct (come together and touch one another) just before speech. Quite often the opposite is true. A little less than half of the consonants in English are voiceless, that is, they require the vocal cords to be apart. So, for example, the voiceless 'p', 't', and 'k' sounds are produced with the vocal cords apart, while their voiced articulatory equivalents, 'b', 'd' and 'g', are produced with the vocal cords together.

Part 6: The base level tension

All of the above tensions are superimposed upon a fluctuating 'base level tension'. *The 'base level tension' is defined as the tension on the vocal cords when a person is not speaking and not intending to speak.*

There are many sources for this tension. Some can be said to be systemic. These might be: fatigue, nutritional deficiencies, allergies, illnesses, and hormone fluctuations. Others might be reactions to environmental conditions such as extremes of temperature, humidity - or positive ions, drugs, alcohol, pollutants, etc.

Still others might be considered 'psychological'. These might be the loss of a loved one, a disagreeable boss, a mean teacher, an unhappy experience, etc. Collectively and typically, these can be stressful situations and/or memories.

The combined strength of these various sources at each moment in time, constitutes the 'base level tension'. These sources can be individually high or low and in phase or out of phase with each other - yielding 'good', 'average' or 'bad' speaking days, weeks, months, etc.

Thus the often-experienced and reported variability in the severity of the disorder, the variability that speaks to the need for repeated measures in both research and clinical settings has to do with shifting base level tensions.

Part 7: Marshaling the evidence in support of the site of the trigger

There are thus at least ten separate events taking place simultaneously in the silence before phrases or sentences in normal speech production. They are: the selection of vocabulary, grammar, syntax, intonation and linguistic stress, the effects of co-articulation, speed of the first word, and the two forms of pre-phonatory tuning of the larynx: correct pitch and loudness at onset and correct voiced/voiceless distinction at onset - and the base level tension. *Add to this an eleventh event, the role of anticipatory stress, scanning - in the case of the individual who has learned to look ahead for feared sounds, words and speaking situations - and the relatively complete picture of stress is painted.*

Managing these events correctly represents a substantial undertaking when viewed within the constraints of the time available and the immaturity of the nervous system at the typical age of the onset of stuttering. Clearly this creates a multiple-stress situation and, as indicated earlier, a common expression of stress is an increase in muscle tension.

The question remains, where is the most likely site of the tension? *The purpose of this chapter is to suggest that the answer lies at the narrowest aperture along the vocal tract from lungs to lips: the space between the two vocal cords.* A small increase in muscle tension at this location can, given its crucial role in so many of these activities, create more difficulty in

speech than an increase in muscle tension at any other location. Furthermore, if the critical source of tension were elsewhere, let us say, the lips, the tongue, or the soft palate, the primary initial signs of stuttering, repetitions, would show articulation and/or resonance defects indicative of struggle in these areas, but they do not. On the contrary, most clinicians often describe the very early onset repetitions as seemingly 'effortless'.

It is thus the contention of this chapter that the pre-speech focus of stress-induced muscle tension must be at the larynx (the vocal cords). When the tension level at the larynx reaches a threshold, the vocal cords become essentially immobilized in either an adducted (closed) or an abducted (open) position, depending upon whether the intended first sound is voiced or voiceless. This state of vocal-cord immobilization, commonly called a block and sometimes described as a 'hesitation' is felt to be the core of stuttering.

Swallowing, rapid, deep inspirations, and speaking on supplemental air - all prevent repetitions because they interfere with the buildup of tension leading to the threshold of laryngeal blocks, but they do so in different ways. For example, in the case of an abrupt, deep, inspiratory gesture, a rapid and large laryngeal patency (opening) is produced by a reflexive contraction of two muscles, the posterior cricoarytenoid and cricothyroid⁽³³⁾ to accommodate the greater inflow of air. This neural command to the laryngeal abductors (muscles that open the vocal cords) supersedes the intent to speak, with

the result that most of the sources of pre-speech tension are temporarily suspended.

Speaking on supplemental air (just the opposite of the previous approach) also accomplishes the same thing, but in a more involved way. On the one hand, pleural (lung), diaphragmatic (the abdominal diaphragm), intercostal (the muscles between the ribs) and abdominal wall (muscles on the front and sides of the abdomen) receptors (sensory nerve endings), detecting lung volumes dropping dramatically below tidal volumes (the amount of air moving in and out of the lungs during normal quiet breathing), prepare the organism for imminent inspiration by again initiating abduction (opening) of the vocal cords,⁽³⁴⁾ while, at the same time, laryngeal adductors (muscles that close the vocal cords) are at work trying to create sufficient trans-glottal resistance (enough closure at the vocal cords) to enable the build-up of the requisite subglottic air pressure (air pressure under the vocal cords) to initiate phonation.⁽³⁴⁾ This activity also interferes with the intent to speak by temporarily suspending most of the normally attendant pre-speech laryngeal tensions.

Finally, at the end of the swallow reflex, after transport of the bolus (mouthful of food) to the esophagus has taken place, with its concomitant transient deglutition apnea (complete, protective blocking of the airway during swallowing to prevent food from going down the wrong pipe), the larynx drops abruptly and the vocal cords open widely to enable resumption of respiration.⁽³⁵⁾ This behavior, as part of the swallow re-

flex, like the others, interferes with the preparations that must be made to get ready for speech.

Swallowing, rapid inspirations, and speaking on supplemental air all engender alterations of the tensions within and around the larynx just prior to the onset of speech that are both independent of and, to a certain extent, incompatible with the speech that follows. The three behaviors are not struggles in speech; they are, instead, activities before speech that temporarily substitute basic, reflexive mechanisms⁽³⁶⁾ for neurologically higher-order speech commands to the larynx. Said somewhat differently, *they all temporarily suspend the intent to speak and, in so doing, subtract most of the pre-speech tensions on the vocal cords.*

Part 8: More evidence in support of the larynx as the site of the trigger

There are a whole host of other observations that further implicate the larynx as the site of the trigger. Here are just a few: Occasionally, in the effort to speak without stuttering, a child will resort to whispering. The altered physiology of the larynx during whispering to a more abducted state of the vocal folds would support the role of the larynx as the site of the trigger.⁽³⁷⁾ The use of the electro larynx - a device used by laryngectomees (individuals who have had their voice boxes removed - usually because of cancer) to produce voice has elicited fluent speech when used by non-laryngectomized individuals who stutter.⁽³⁸⁾ Changing the pitch of the voice, and

thus the internal and external laryngeal tensions and postures, has produced similar effects.⁽³⁹⁾ Mouthing words fluently with no attempt at phonation further supports the contention.⁽⁴⁰⁾ Fluent esophageal speech (speech made by swallowing air into the esophagus and releasing it in a sort of controlled burp) following total laryngectomy with individuals who had stuttered preoperatively has been reported.⁽⁴¹⁾ The therapeutic use of gentle onsets of phonation to ease into vocal fold vibration also is corroborative.⁽⁴²⁾ And the research that shows, in a similar vein, that individuals who stutter have longer Voice Onset Times (take longer to get their vocal cords vibrating) than people who don't suggests an inherent state of neural-based inertia at the vocal cords.⁽⁴²⁾ All of this admittedly circumstantial, but all, nevertheless, suggestive of a laryngeal involvement.

There is yet a further intriguing piece of evidence that points to the larynx as the site for the trigger. One of the most common activities for lessening or interfering with the prephonatory tensions in and around the larynx is the use of verbal starters. A starter may be a sound, a syllable, a word or words (real or nonsense) or a phrase *that has no contextual linguistic function* and is used before the onset of the feared utterance to get the vocal cords vibrating in the expectation that they will continue to vibrate directly into and through the feared utterance. Verbal starters have a reputation for working initially and then failing. The reason this happens is that after a while, the starter becomes so automatic it becomes incorporated into the overall plan for the sentence. Said

somewhat differently, *verbal starters stop working when they begin to assume the role of the first word and start to co-articulate with the rest of the sentence. It is at this point that the starter becomes part of the intent to speak and thus acquires many of the sources of pre-speech tension that lead to a block.*

Part 9: The trigger for stuttering

It is the contention of this chapter that the trigger for stuttering is a unique afferent neural array (pattern of sensory impulses) that arises from sensory receptors within and around the larynx that signal the central nervous system that a threshold level for a specific concordance of speech-arresting laryngeal tensions has been reached or soon will be reached. It is this unique afferent neural array, or its threatened occurrence, that constitutes the trigger for the learned behaviors called stuttering.

Change the afferent array and the conditioned stutter responses cease. The most efficient way for making changes to the array is to subtract laryngeal tensions or to create new ones, ones that have no cue value. So while Pavlov's dogs were conditioned to respond to the acoustic spectral array of a bell by salivating, no such response occurred to the acoustic spectral array of hand clapping since only the bell's spectra had been the recipient of conditioning.

Similarly, singing, whispering, speaking to the rhythm of a

metronome, shouting, swallowing, rapidly inspiring, speaking on residual air, head jerks, coughing (throat clearing), stress-reduction techniques, starters, changing words, etc. all have the capability of changing the afferent array, as do all activities that distract attention away from speech, have no conditioned anticipatory stresses associated with them (like speaking alone or with a foreign accent), and any or all of the activities that reduce linguistic and/or speed demands.

Part 10: The basal ganglia

Recent research suggests that children who do not stutter are subject to the same linguistic and speech stresses as children who do, but that what separates the two groups may be a genetically-based predisposition for excessive tension to be developed at the larynx in stuttering children.⁽⁴³⁾ Current findings implicate the basal ganglia (a part of the brain) and its tendency to respond inappropriately when under conditions of stress - as the source of this predisposition.⁽⁴⁴⁾ The reasons for this are not entirely known, and it may be nothing more than a maturational delay that eventually catches up and is not found in adults (this may account for why a substantial majority of children who stutter spontaneously outgrow it), but attempts to understand the mechanism are currently underway.⁽⁴⁵⁾

We do know, however, that since the basal ganglia are critically involved in memory, cognition, learning, sequencing of complex motor commands and have direct connections with

the limbic system⁽⁴⁶⁾ (the emotional parts of the brain) and projections to and from a laryngeal area on the motor cortex, ⁽⁴⁷⁾ the basal ganglia, more than any other central nervous system structure, has the potential of being the source of the trigger for stuttering.

In the next chapter we shall provide more detailed information about the basal ganglia and a theoretical discussion regarding precisely how its behavior, in predisposed individuals, elicits the trigger for stuttering. In association with that discussion, we will theorize how thiamin alters the behavior of the basal ganglia and perhaps certain other central nervous system areas to eliminate the trigger.

Part 11: A final overview

We posit a model for stuttering based on a series of events that lead to a trigger for the disorder. The events are as follows: In a neurologically predisposed child, diverse stresses occur simultaneously to result in a buildup of tension at the larynx which reaches speech-arresting levels in the silence before speech. A state of vocal-cord immobilization, *commonly called a block, then exists. This block constitutes the core of stuttering.*

The most typical immediate response to a block is a syllable repetition. Repetitions can occur with varying degrees of tension and frequency and often become habits. After a period of time, which can vary greatly, the child becomes aware of

and reacts to the repetitions by engaging in one or more of a number of activities. These can be avoidance behaviors, direct, forceful confrontational attacks, or more subtle maneuvers such as swallowing, inhaling rapidly and deeply, or speaking on expiratory reserve volume.

All of these behaviors, with the exception of the core behavior, the laryngeal block, are learned. The core behavior is represented by a unique laryngeal afferent array that, through conditioning, becomes the trigger for all of the subsequent learned behaviors associated with it. Any activity that alters this unique array can prevent the conditioned stutter responses from occurring.

Chapter 6

Laryngeal reciprocal inhibition and GABA

We now understand that to prevent the trigger for stuttering from occurring one must somehow prevent the buildup of tension that occurs at the vocal cords under conditions of stress. In the previous chapter we specified eleven stresses that have the potential of adding to the buildup of that tension. In this chapter, we start by taking a closer look at the behavior of the vocal cords during normal speech as well as during moments of stuttering.

In part 5 of the previous chapter we pointed out that some sounds are made without voicing (that is, the vocal cords are apart and not making any sound) and other sounds are made with voicing (that is, the vocal cords are together and vibrating to making sound). In most languages of the world, this voiceless/voiced distinction is often the only characteristic separating two sounds that are, in all other respects, identical.

For example, in English, the [s] sound is voiceless, the sound heard as an [s] is generated in the mouth as air passes through a narrow channel between the tongue and anterior hard palate and becomes turbulent. No sound is made at the vocal cords. Its voiced equivalent [z] is the same as the [s] in terms of how it's made but, in addition, the vocal cords are vibrating, thus adding a second source of sound.

Here are some other examples of English voiceless/voiced cognates: [p]/[b], [t]/[d], [k]/[g], [f]/[v]. So the vocal cords are constantly being actively opened and actively closed in ongoing speech depending upon whether

the consonant is voiced or voiceless (the vowels are all voiced). Also note that this is happening with great speed (in thousandths of a second) and with exquisite precision. This important requirement for speech has been almost entirely neglected by researchers and clinicians working with people who stutter. However, as we will discover, it is of utmost importance in understanding the mechanism underlying the trigger for stuttering.

The muscles that oppose each other in opening and closing the cords are said to be 'antagonistic' to one another. Most muscles or muscle groups have their antagonist muscles or muscle groups.

So, for example, I bend my arm at the elbow by contracting the biceps muscle and extend it by contracting the triceps muscle. The biceps and triceps are said to be antagonists. But also, and very important, while one muscle is actively contracting to produce the desired movement the other, its antagonist, is *actively relaxing*. This means that if one looks closely at my triceps when my biceps is actively contracting, not only is my triceps not opposing the contraction, but the normal subtle background contracting present to keep my triceps 'warmed up', so to speak, so that it is ready to work – is suddenly absent.

To alternately contract and extend my arm rapidly, my biceps and triceps must thus alternately actively contract and, just as importantly, alternately *actively relax*. This alternating relaxing component of the movement process between antagonistic muscles is called *reciprocal inhibition*. It is sometimes called Sherrington's law. Sir Charles Scott Sherrington was a neurophysiologist who received the Nobel Prize in 1932 for discovering it.

As indicated earlier, much new research in stuttering seems to implicate a part of the brain called the basal ganglia. Recent advances in knowledge of the basal ganglia suggest that this part of the brain does not initiate movement. Instead, when voluntary movement is initiated by other brain structures (most likely, for speech, the cerebral cortex and the cerebellum) the basal ganglia act to inhibit competing motor mechanisms that might otherwise interfere with the desired movements. In other words, one of the things the basal ganglia does is to support the reciprocal inhibition that must be seen at the vocal cords during ongoing speech.

Furthermore, while the basal ganglia receives information from most of the cerebrum, it sends almost all of its information back only to the frontal cortex. As such, the frontal cortex and basal ganglia can be said to constitute an integrated neural loop. The frontal cortex and basal ganglia appear to specialize in different, but related aspects of learning. The frontal cortex acts when new behaviors have to be learned while the basal ganglia take over when the behaviors have become learned. It is quite clear that the obvious struggle behaviors which characterize the disorder are learned, but it is also quite likely that most of the tensions developed at the vocal cords which trigger the disorder are not.

GABA (gamma-amino butyric acid), is the most extensive inhibitory neurotransmitter in the brain - and it is mostly concentrated in the basal ganglia.

Thiamin is required for the proper functioning of GABA.

GABA is also found in the frontal cortex. As indicated earlier, the neurons in the frontal cortex receive inhibitory commands from the basal ganglia. These commands, under normal conditions, are then communicated to the

muscles of the vocal cords via a set of pathways between the frontal cortex and the vocal cords. The fact that thiamin produces a remission of symptoms for almost of third of adult males who stutter would suggest that these individuals are GABA-function deficient. If this is true and if stress causes a further decrease in GABA function and a commensurate increase in excitatory neurotransmitter function, especially neurotransmitters called dopamine and glutamate, then the stage is set, not only for a loss of the normal laryngeal reciprocal inhibitions required for speech, but beyond that, to a spasm of the vocal cords.

Direct evidence of a vocal cord spasm as the likely core of stuttering has been provided by a widely known and respected study published in 1978 in the Journal of Speech and Hearing Research. Investigators, using electrodes implanted in the vocal cords of individuals who stutter, found that just before and during moments of stuttering the muscles that both open and close the vocal cords during speech were both being contracted forcefully and simultaneously. In other words, there was clearly a loss of the reciprocal inhibition at the vocal cords that one would expect during speech.

Although a spasm of the vocal cords was hypothesized to be the core of stuttering as long ago as 1849, it wasn't until Freeman and Ushijima, authors of the aforementioned 1978 study, published their findings that we had the first direct empirical evidence to support this theory. Bear in mind that the constricting movements of the vocal cords I had seen earlier through the use of ultrasound did not permit direct verification - so while the assumption was that these constricting movements were the result of excessive tension it remained just that, an assumption, until the 1978 study was published.

Here is the abstract from that study:

“Laryngeal muscle activity during fluent and stuttered utterances was investigated via electromyography. Analysis revealed that stuttering was accompanied by high levels of laryngeal muscle activity and disruption of normal reciprocity between abductor and adductor muscle groups. Results are interpreted as demonstrating the existence of a laryngeal component in stuttering and showing a strong correlation between abnormal laryngeal muscle activity and moments of stuttering.”

Notice that the authors had not quite gotten their interpretation of their findings correct. They viewed the aberrant laryngeal behavior as “a laryngeal component in stuttering”, not as the core behavior of the disorder. Others have made this mistake, even to this day, failing to fully grasp the overwhelming evidence that demonstrates that the obvious supra (above) and infra (below) - laryngeal struggle behaviors that so often characterize the disorder are learned.

Investigators have tried giving GABA to people who stutter. But GABA, in its pure form, apparently does not cross the blood-brain barrier and thus has little effect. So various pharmaceutical derivatives of it have been developed that cross the barrier and have been tried with people who stutter. In the main, the results of these studies have been positive, with statistically significant enhancements of fluency demonstrated. But the problem with most of these pharmaceutical agents is that they often come with unacceptable side effects and none has ever been able to produce the kind

of total remission of symptoms produced by thiamin.

If one carefully examines these studies, it becomes obvious that the experimenters, while purportedly looking at a speech problem were, in fact, unaware that the overt struggle behaviors they were studying were learned. Instead, had they been aware of the core of the disorder (the laryngeal spasm) and had they known about the behavior of the larynx during speech, they would have fully understood that what they should have been examining is the laryngeal reciprocal inhibition required for the rapid alternation between voiced and voiceless consonants in ongoing speech.

There is, however, one study where the investigator was aware of this. Martin Adams, in an article published in the *Journal of Speech and Hearing Research* in 1971, made an important contribution to our understanding of stuttering.

In his study, Adams created two distinctly different reading passages. The first was composed entirely of voiced consonants. The second contained a combination of both voiced and voiceless consonants. So the first passage did not require any laryngeal reciprocal inhibition because the vibration was continuous throughout. The second, on the other hand, required the usual reciprocal inhibition that occurs in normal, ongoing speech. Said somewhat differently, in the first passage, once the vocal cords started to vibrate, they continued to do so in uninterrupted fashion until the end of the passage; while in the reading of the second passage, there was the typical closed/open alternation between voiced and voiceless consonants - and its associated requirement for reciprocal inhibition.

Adams noted that apart from this difference, the two passages were care-

fully matched along several other linguistic and phonetic parameters. Fourteen people who stuttered read each passage out loud five times in different random orders. The moments of stuttering were tabulated and a statistical analysis revealed significantly less stuttering with the all-voiced passage. This finding confirmed the importance of fully understanding the elements of speech and the importance of laryngeal reciprocal inhibition in ongoing speech.

Please note, however, that had his subjects been instructed, prior to reading the passages for recording, to rehearse reading them out loud alone in an empty room, with no one listening, many of them would have been fluent. For these individuals, without an audience, presumed or real, with its attendant conditioned stresses, there would be no stuttering.

So it is clear that the neurotransmitter issue becomes a non-issue if the stresses present (remember, there usually are eleven of them) are not enough to reach the trigger threshold for stuttering. When the overall tension level is below the threshold, the mechanisms involved in speech production apparently work without difficulty. When the level is above the trigger threshold, the system breaks down and the conditioned responses are elicited.

There is another interpretation that can be made with respect to GABA function and the vocal cords. It is well known that higher centers of the brain (mediated by inputs from the basal ganglia) often serve to inhibit lower ones and that stress can create a condition of disinhibition which permits lower, more primitive reflex responses to emerge. The basic, reflexive functions of the vocal cords are to close tightly during swallowing to prevent food or liquid from entering the lungs, and to open widely, after

swallowing, to enable resumption of breathing. With insufficient GABA function the amount of stress required to elicit these primitive reflex responses would be much less than ordinarily required.

There are direct connections from the basal ganglia to and from a laryngeal area on the motor cortex. There are connections from this laryngeal area on the motor cortex (via a pathway called the corticobulbar tract) to the nucleus ambiguus in the medulla. A majority of laryngeal motoneurons in the nucleus ambiguus are GABA sensitive. The nucleus ambiguus contains the cell bodies of nerves that innervate not only the muscles of the larynx but also muscles that are activated during the pharyngeal phase of swallowing.

Since GABA is present in the basal ganglia, on the motor cortex, and there are GABA-sensitive motor neurons in the nucleus ambiguus, it is tempting to speculate that this linkage may represent the neural pathway responsible for the laryngeal spasm that generates the trigger for stuttering - especially when there is the presumption of a deficiency of GABA function.

However, it is again important to remember that with enough thiamin it has been shown that, regardless of the stress present, for almost a third of adult males, there can be a *complete* remission of stuttering. So even if the individual's base level tension is high, even if they are speaking very rapidly, even if they are ill, even if they 'see' all the difficult words and speaking situations approaching – in spite of all of this, for slightly less than one in three, if they take 300mg of thiamin per day, they simply do not stutter.

This, I submit, is the remarkable thing about Thiamin.

Chapter 7

The magnesium connection

In this chapter we discuss an enhancement to Thiamin. It came about as a result of a series of events that occurred while I was director of The National Center For Stuttering. In that capacity I was made aware of many so-called 'remedies' for stuttering - from having clients sleep with a horse hair under their pillow to having them read from the Bible each morning in a loud voice, to suggesting they have their lingual frenulum cut (thinking that stuttering was the result of tongue-tie), to the often suggested "just relax and slow down".

Another suggestion that occurred several times, and in slightly varying forms, was to take the juice of half a lemon, add a tablespoon of blackstrap molasses to it, combine the two thoroughly, then add a glass of warm water, stir and take just before every meal. This drink was described by one source as 'the elixir for stammering'.

I decided to look at this 'elixir' in more detail because since it was taken internally it might have systemic implications. I found that drinking the juice of half a lemon in a glass of water upon arising was a frequently recommended suggestion for 'good health'. I further discovered that lemon juice, although obviously containing citric acid, actually produces an alkaline condition in the body. Some health professionals suggest that an alkaline condition of the body (tested from saliva or urine using commercially available test strips) is more resistant to infection and other diseases than an acidic condition. There have also been anecdotal reports about

individuals whose stuttering has been helped by eating an alkaline-producing diet (fruits, vegetables, nuts) as opposed to an acid-producing one (meat, processed foods, refined sugar).

The second component, molasses, appears to have also been recommended more than once. I thought originally it was just to counter the tartness of the lemon juice. But sometimes it was recommended by itself.

Unfortunately, when I first encountered this suggestion, I simply dismissed it as having no basis in fact and it was only when I was well into the consideration of the importance of thiamin to stuttering that I began to look back at this nutritional recommendation and start to appreciate its significance. Later, a blog devoted to a consideration of thiamin and stuttering mentioned two anecdotal reports further confirming the use of molasses as an 'antidote' to stuttering in children.

I discovered that molasses, a product of sugar refining, is essentially a distillate of much of the nutrition in the plant. The roots of sugar cane grow deep, some 10 - 15 feet down, well below the depleted surface soils and gather much of the vitamins and minerals found at these subsurface levels. Most of these are in small amounts - with one exception: magnesium. The amount of magnesium in one tablespoon of unsulphured blackstrap molasses turns out to be 200 percent greater than the RDA for magnesium for children ages six to eight.

My research further revealed that vitamin C aids the absorption of magnesium and that magnesium, in turn, is required to convert thiamin into several forms, one of which is required for absorption. So the vitamin C in the lemon juice was facilitating the absorption of the magnesium and the

magnesium, in turn, was facilitating the absorption of thiamin which, of course, was required for the production of GABA. Finally, to come full circle recent research has shown that magnesium directly binds to and activates GABA receptors

The significance of all of this became clear in 1991 with a study reported in the Polish Journal of Otolaryngology. Fifty-three children who stuttered and twenty-two who did not were examined in terms of levels of magnesium in their bloodstream. In 47% of the stuttering children, there was a deficiency of magnesium; virtually none was found in the non-stuttering children.

There are a number of reasons why a magnesium deficiency might exist. It is known that magnesium is depleted by stress. Other causes of magnesium deficiency can be diarrhea, diabetes, Crohn's disease, certain drugs and the relative absence of other minerals in the diet, minerals such as calcium and zinc, which are required to prevent magnesium loss.

But there is more. The dietary intake of magnesium, in general, is usually deficient. The process of refining foods strips nutrients, including magnesium. Here are some examples:

1. Dry-roasting nuts removes the oils which contain magnesium.
2. Milling flour from grains strips magnesium from the grain.
3. Sugar in anything consumes magnesium.
4. Fluoridated, softened, and distilled water depletes magnesium.
5. Some processed foods, like lunch meat, contain phosphates that bind to magnesium and flush it out of the body.
6. Alcohol blocks magnesium.

7. Coffee works like a diuretic to flush magnesium out of the body.
8. Some foods - like raw nuts and seeds, soybeans, spinach, and chard - contain compounds called phytic acid and oxalic acid which cause magnesium to be eliminated from the body.

In view of the above, and the often-quoted statistic that eighty percent of Americans have diets that are magnesium deficient, we began our investigation into magnesium by recommending its RDA (recommended dietary allowance). The RDA represents the average daily dietary intake level sufficient to meet the nutrient requirement of nearly all - 97 to 98% of - healthy individuals in a group.

But magnesium, a mineral, must be in compound form to be taken. The problem is that these compounds vary considerably in their ability to be absorbed. The most common form, magnesium oxide contains the highest percentage of magnesium but only 4% of it is absorbed. After some research, it was decided to use magnesium glycinate, a highly absorbable form of magnesium with few, if any, side effects when taken at RDA levels.

There is another reason for using magnesium glycinate which makes it superior to other magnesium compounds. Glycine, an amino acid to which the Magnesium is bound, is yet another inhibitory neurotransmitter. It is often released in conjunction with GABA and seems to serve similar functions.

For the typical adult male we decided to use, as a standard recommendation, 400mg of magnesium glycinate per day. 130mg tablets are available and our recommendation is that one 130mg tablet of magnesium glycinate accompany one 100mg tablet of thiamin hydrochloride at breakfast, lunch

and dinner. It is appropriate to take them at the same time since thiamin and magnesium are both absorbed largely in the small intestine.

We have been evaluating magnesium clinically for some time now. Our goal is to ferret out features that might compromise a randomized, double-blind study. Gaining clinical experience as a precursor to doing formal research has proven to be a productive way of gaining new knowledge.

Our preliminary impression is that magnesium at RDA levels seems to help largely those who are at least partially helped by thiamin. We are also trying magnesium at more than RDA levels to see if higher levels might perhaps cross a threshold of clinical significance - as thiamin did when we began to use 300mg per day. It is also quite clear though that magnesium, alone, has no impact on adult stuttering.

One note of caution before taking magnesium. Running throughout this book is the clear recommendation that before trying any supplement, one must consult with one's physician. For example, persons with kidney disease or a severe cardiac condition should only take magnesium under a doctor's close supervision. But, in addition, there are so many medications that have the potential of interacting negatively with a supplement, that can lose their effectiveness when taking a supplement or that can interfere with supplement absorption, that to proceed without medical clearance would be most unwise.

Chapter 8

The Thiamin Protocol

In this chapter, we outline a three-week test that one can take to determine if thiamin can benefit their stuttering. We call the combination of 300mg of thiamin hydrochloride with 400mg of magnesium glycinate taken daily in a specific way: THE THIAMIN PROTOCOL. The Protocol is for adult males. Adult females and children require lesser amounts. Also, before taking, remember again to obtain the OK from your health care provider.

Taking these two supplements also requires, as a precursor, and as part of The Protocol Test, the elimination of anything that might possibly interfere with their absorption and/or function. As indicated earlier, thiamin and magnesium have dietary requirements for maximum absorption and function. If this aspect of the test is neglected, one runs the risk of having a false negative outcome, that is, incorrectly concluding that the Thiamin Protocol is ineffective.

So what follows must be done to create the supportive environment for the three-week test.

For one week prior to and two weeks during the test:

Eliminate coffee and tea or any sugar-laden beverages.

Refrain from all sources of simple carbohydrates.

Do not eat sushi.

Try to avoid antacids, barbiturates and/or diuretics.

Try to avoid taking antibiotics.

Do not eat 'junk' foods.

Refrain from smoking.

In addition, try to be on an alkaline-promoting diet rather than an acid-promoting one.

Here is a sampling of alkaline-promoting foods:

Almonds

Apples

Apricots

Asparagus

Bananas

Broccoli

Carrots

Cauliflower

Cherries

Green beans

Hazelnuts

Honey

Mineral water

Potatoes

Radishes

Spinach

Tomatoes

Watermelon

Zucchini

And here is a sampling of acid-promoting foods:

American/cheddar cheese

Brown rice

Butter

Chicken

Cooked salmon

Canned sardines

Cooked turkey bacon

Dry roasted peanuts

Hard boiled egg

Hot dogs

Lean beef

Lentils

Low-fat yogurt

Mozzarella

Oats

Parmesan cheese

Pepperoni

Plain bagel

Walnuts

White bread

White canned tuna fish

Whole Milk

Whole-wheat spaghetti

Wild cooked trout

For a complete listing of both alkaline and acid-forming foods, the reader is advised to go to the internet and search under the term: alkaline/acid-

forming foods. Basically, if one can elect an alkaline-forming diet for the three-week period, one would have a much better idea of the true impact The Thiamin Protocol will have on one's stuttering. As an aside, the taking of a magnesium supplement has been shown to have an alkalizing effect on the body, something that is desirable for general health reasons. Also note that the taking of a magnesium supplement is often recommended to reduce or eliminate muscle spasms.

The goal is not to radically change your diet. The goal is to create the optimum conditions, just prior to and during the test, for The Thiamin Protocol to reduce or eliminate your stuttering. If you experience a positive outcome, you may then slowly add back acid-forming foods to determine the optimum acid/alkaline balance for you.

Since our main focus is upon thiamin, let us now move on to a bit more detail about it. There are many types of thiamin; some cross the blood-brain barrier, others do not. Although thiamin hydrochloride does not cross the barrier it has a long history of safety and it has been scientifically tested for efficacy for people who stutter. It may very well be that some of the other forms of thiamin may turn out to be more effective (and in some clinical reports this has been suggested) but, in the absence of scientific data, they cannot be supported. Also, some of these newer forms of thiamin may have the potential of engendering side effects that are less than desirable.

So it is best to take thiamin hydrochloride. But not all brands of thiamin hydrochloride are the same. While the process for creating the vitamin may be essentially the same, issues of purity, accuracy of the label as to contents, the particular excipients used (excipients are additional chemi-

cals used to add bulk to a pill, enhance its bioavailability, permit it to retain its strength over extended storage periods, etc.) and consistency from batch to batch are to be considered. One brand of thiamin may therefore have a different combination of excipients than another and their effects upon the efficacy of thiamin have yet to be studied.

While there are a number of good brands in the marketplace, the brand used in our studies was Solgar. The reasons had to do with the reputation of the company and its products and the product's ready availability in many health food stores, not only across the United States, but throughout the world.

The Thiamin Protocol requires that an individual take 300mg of thiamin hydrochloride per day as three 100mg pills dispersed throughout the day. So the best approach would be to take one pill with each meal together with one 130mg magnesium glycinate pill. There are many good brands of magnesium glycinate available. The one often used is by Pure Encapsulations (available through distributors online), largely because of its rigorous quality testing. As an aid to enhancing bioavailability, one might recommend taking the pills with a glass of water into which the juice of half a lemon has been squeezed and to take just prior to each meal.

The reason one is not advised to take all 300mg of thiamin at once is that, as indicated earlier, too much at any one time will be quickly excreted through the urine. Furthermore, it has been shown that continuous ingestion of thiamin in a dispersed manner, as indicated earlier, enables blood levels to rise continuously for between seven and ten days before leveling off, thereby enabling progressively greater diffusion of the vitamin across the blood-brain barrier. Interestingly, this agrees with the frequent clinical

observation that when thiamin does produce a remission of symptoms, it does so usually between the first and second week.

If, however, after two weeks there has been no noticeable improvement taking the supplements, the odds are they will not work for you, even at higher levels. However, this does not necessarily mean that there is no hope. Instead, one can initiate a new and different approach, an approach that will be described in the next chapter.

Finally, as usual, do not start the Thiamin Protocol without the express consent of your physician.

Chapter 9

Intent Therapy

In this chapter we propose a solution for those for whom the supplement approach is ineffective, as well as for those for whom it is effective but want eventually to be weaned off it. The solution combines the use of a very simple, but unique, way to get set to speak together with a contemporary version of an electronic device that enhances the speed of acquisition of new behaviors. We begin with the new way to get set to speak.

Let us start by reiterating some of the stresses that are associated with the production of a sentence. These stresses occur during the time one gets set to speak. They are stressful not because they are inherently so, but because there are so many of them occurring simultaneously and with such great rapidity.

In the production of a sentence we noted that there are a number of choices that have to be made before speech can begin. The vocabulary, grammar, syntax, intonation and linguistic stress of the entire sentence have to be selected, integrated and sequenced (in fractions of a second).

We further indicated three additional sources of stress. Co-articulation, where the effect of the speech requirements for a subsequent word in a sentence can sometimes be detected on the first word of that sentence - indicating that some of the organization for all or part of the entire sentence can take place even before the start of the sentence. The speed of the first word (*speed stress*) - as the sentence is lengthened the first word

is spoken more rapidly suggesting that the longer the sentence, the greater the pre-speech tension (because faster movements require more tension before the movement begins). And the third event of importance, the *pre-phonatory tuning of the larynx* - which we noted was comprised of two types: In one, if speech is to start with the correct pitch and loudness, the vocal cords must assume specific postures and tensions before speech. And two, the open or closed position required of the vocal cords for the first sound in an utterance depending upon whether the first sound is voiceless or voiced.

These pre-speech sources of tension are present when an individual *intends* to say a sentence. *Many of them are not present when there is no intent to say a sentence.* This is an extremely important point. It bears repeating: *without the intent to say a sentence many of the pre-speech sources of tension cannot be there.*

The goal is to *reduce the pre-speech tensions to a level below the trigger threshold for stuttering by altering how much a person intends to say before they speak.* The technique that accomplishes this is called: Intent Therapy.

But first, some additional observations. If a person says just one word, it is because they have intended to say just one word. In that circumstance, many of the stresses associated with saying sentences are not present. So while vocabulary stress remains, grammar, syntax, intonation and linguistic stress do not. And while pre-phonatory tuning of the larynx remains, co-articulation between words and much of speed stress do not.

So of the potential sources of stress, only intra-word co-articulation,

vocabulary, and pre-phonatory tuning of the larynx remain. If we could create that low level of pre-speech tension before sentences, we would really have a powerful tool.

What follows is a set of instructions given to clients designed to achieve just that goal:

“What you are going to learn is a sport that has four rules. You are going to take pride in how well, how smoothly, how slickly, how undetectably you follow these rules as you play your sport. And, as a byproduct of playing your sport correctly, you will not stutter.

We start with sentences that begin with one-syllable words. We are going to slow the first word of each sentence. But this will not be ordinary slowing. *It is going to be very different.* Here are the four rules:

1. The first rule is that when you say a sentence, you are to put a ‘mental comma’, a pause, between that first word and the rest of the sentence. You don't have to slow the first word; all you have to do is put the comma in and your brain will do it for you automatically.
2. Here's the second rule. You are to say the first word as if it were the only word you were going to say, with the part that comes after the comma, the rest of the sentence, spoken as an *afterthought*.
3. Here's the third rule. There shall be no influence of the second word on the ending of the first word. The comma is a real wall separating the first word from the rest of the sentence.

4. Rule number four. Never inhale during the comma because if you do, your brain will think you have begun a new sentence without technique and then you may stutter.

If you closely examine these four rules, you will notice that the first three say the same thing - but in different ways. What has to be learned is that you must intend to say just one word. Once you have successfully accomplished this, then and only then can you shift your intent and say the rest of the sentence. Do this correctly and you have stripped most of the pre-speech sources of tension from the vocal cords that normally occur before sentences - while still retaining the sentence form.

One way to practice is to create a long list of one-syllable words. Say each one as if it were the only word you were going to say and then, and only then, make up and say the rest of a sentence. The important thing is not to have the rest of the sentence in your mind before you say the first word. If you do, the other forms of pre-speech tension will automatically creep back in.

If you have trouble doing this, take 50 file cards (3"x5") and write a single one-syllable word on each. Shuffle them, turn them face down, and read them one at a time, adding a different made up rest of the sentence to each - *as an afterthought*. With no foreknowledge of the word to come, the temptation to create the sentence in advance is reduced.

Practice this several times a day, for weeks at a time, until it becomes second nature to you. What you are practicing is not reading single words and then adding made-up sentences. What you are practicing is shifting from saying a word with the intent to only say that word to saying the rest

of the sentence as if it were an afterthought. You are practicing Intent Therapy. This is the 'sport' you must master.

When a sentence begins with a multi-syllable word, an entirely different issue is encountered. This issue is very important. An illustration makes the point of exactly how important it is:

If you ask 100 people who stutter to think of a difficult word, a word they have trouble saying, with the exception of their name, more often than not the word will be multi-syllabic. As a matter of fact, at least nine times out of ten it will be. The question is: why are multi-syllable words reported as difficult nine times more often than mono-syllable words? What is so special about them? Here is the answer:

It would appear that the basic unit of speech, in so far as the brain is concerned, is the one-syllable word - that we were originally meant to speak only in monosyllables. But when one does it sounds unnatural. For example, read the following four sentences out loud and see for yourself: "I would like to tell you how to buy a car. If you go to a street near my house you will find a man there who sells used cars. He has been there for a long time. You can trust him." Most unnatural, wouldn't you say? It has almost a metronomic quality to it.

Interestingly, when you ask people who stutter to speak in synchrony with a metronome they rarely stutter.

Now consider this sentence: "I would like to go to Mississippi this fall." Every word in this sentence is one syllable except Mississippi, which is four. Read it out loud. Notice how quickly the syllables in Mississippi are

spoken. You may have missed it. Read it out loud again. The speeding of the syllables in Mississippi occurs completely automatically so there is no reason to pay any attention to it.

It is as if the brain is saying: “There is a certain amount of time slotted for the saying of each word in the sentence. That amount of time is equivalent to the time it takes to say a one-syllable word. If you want to say a four-syllable word in that sentence you are obliged to say it in the same time slot it would take to say a one-syllable word. You don’t get any more time.” So you automatically rush the syllables of the multi-syllable word to fit them into the one-syllable-long time slot.

As indicated, most people are totally unaware of doing this. It is completely automatic. But acoustic phoneticians, who have measured speech durations, have long known that the speed of the syllables in a multi-syllable word in a sentence is always faster than the speed of a one-syllable word in a sentence. As a matter of fact, even when a person is asked to say a multi-syllable word by itself, a similar, but lesser degree, of speeding occurs.

Remember the arm extension example in Chapter 5? Faster arm extensions require more tension prior to the movement than slower arm extensions. Equally, faster speech requires more tension before speaking than slower speech. So it is fair to say that both in sentences and out of sentences, syllables in multi-syllable words, since they are almost always spoken more rapidly than monosyllable words are almost always spoken with more tension. And since it is tension that triggers the threshold for stuttering, it is far more likely one will hit one’s threshold on a long word

than a short one. And that's why long words are so much more often reported as the 'difficult' ones.

Recognizing the difference in speed between multi- and mono-syllable words has led to an addition to rules 1 - 3. The addition states that when a sentence begins with a multi-syllable word it is to be spoken at a rate of 108 beats or 108 syllables per minute. Speaking at this rate counters the tendency to rush multi-syllable words. To facilitate practicing this, one can purchase an inexpensive metronome or gain free access to an one online at www.metronomeonline.com.

To practice, prepare a list of two-syllable words. Set the metronome to a rate of 108 beats per minute and begin by reading from the list, with each syllable spoken in synchrony with each beat of the metronome. When reading, make sure the first syllable melts directly into the second one. There are to be no pauses between the syllables in multi-syllable words. Do a lot of two-syllable word reading each day for weeks. Become comfortable with it. Think of it as metronomic speech at 108 syllables per minute applied to two-syllable words - but don't use an unnatural or sing-song voice. Strive for total naturalness when reading them. They're just slowed a bit, that's all.

After a couple of weeks, begin practicing three-syllable words, again in isolation, that is, independent of sentences. Again, strive for natural-sounding speech. That is extremely important. No one wants to sound strange. Make doubly certain that your rate remains at 108 beats (syllables) per minute. The greater the number of syllables in a word, the greater the tendency to speed.

After several weeks doing this, making certain your rate does not increase beyond 108 syllables per minute and you do not sound like a robot, go back to two-syllable words, each on a separate 3" x 5" file card, shuffle the cards, and start reading them spontaneously adding words to each two-syllable word to create a new sentence for each. Remember, this is Intent Therapy, and the first word, regardless of its length, is to be spoken as if it were the only word you were going to say, with the *rest of the sentence spoken at normal rates* but as if it were an afterthought. *Thinking of the sentence as a whole before you say it adds back all of the pre-speech tensions and this, more than anything else, leads to stuttering.*

Rule number four is quite important. In so far as the brain is concerned, the basic unit of speech is the breath group - which is defined as all the words spoken on a single breath. So if you inhale again during the comma, or between the syllables of multi-syllable words, even the tiniest of inhalations, you have begun a new breath group or, in other words, a new sentence. So, inhaling during the comma or within multi-syllable words is not allowed.

It must be noted, however, that speakers often take several inhalations when saying long sentences. This is quite normal. But each time they inhale, they effectively signal the brain that a new sentence has begun. So the first word following each inhalation must become the recipient of Intent Therapy. Mid-sentence breaths occur automatically and are often quite brief - so you must be on guard lest one slip by unnoticed.

The best way to practice mid-sentence reapplications of technique is to read from a book or newspaper, noting where a mid-sentence breath might occur and making a deliberate effort to reapply technique at that lo-

cation. You might mark such locations on the page to prompt reapplications. Some of these mid-sentence reapplications should be before monosyllable words; others should be before multi-syllable words.

There are three reasons a person will stutter in the middle of a sentence. Either they have taken a breath in mid sentence, thus effectively making two sentences and are not aware of it; or the stuttered word is multi-syllabic producing a sudden increase in muscle tension; or it is a feared word producing its own increased muscle tensions. In the latter instance, the feared word is also likely to be multi-syllabic - at least 90% of the time it is likely to be. One would be well advised to practice saying feared words at a rate of 108 beats (syllables) per minute. Make a list of them and practice Intent Therapy for each. Practice hundreds of repetitions at a rate of 108 syllables per minute for each word. Do this for weeks. The response has to be so automatic it can withstand the distracting effect of stress.

When you are certain you can consistently produce perfect examples of intent therapy when alone, begin trying to use it in low-stress situations. If you succeed in low-stress situations, move slowly to situations of progressively higher stress. This is a process called systematic desensitization. As you progress, if you move slowly and experience success you will build your confidence.

We fully recognize, however, that life and its stresses are not nicely arranged in a neat hierarchy and you will often be in high-stress situations sooner than you would like. In those circumstances do not be disappointed if you do not succeed. Just continue with your daily practice and your persistence will eventually pay off. Remember, Intent Therapy, like all sports, needs a lot of practice.

In my experience, if you separate your practice into the steps and sequences just outlined, you should, within a few months, be able to handle stressful situations with perfect examples of Intent Therapy.

To facilitate automatic use of technique in conversation, a private reminding device called the MotivAider (www.motiv-aider.com) is used. The device is worn on a belt or in a pocket. Periodically, at pre-set intervals, it emits silent, pulsing signals (inaudible vibrations) that have been programmed in advance.

A Focusing Protocol™ is used to program the MotivAider. As part of the programming protocol, a word is chosen and paired with the desired change. For example, the chosen word might be INTENT and the desired change, to make the next sentence start with perfect technique.

After going through the Protocol, whenever the pulsing signal is felt, the word INTENT is automatically remembered, followed by the desire to use Intent Therapy on the next sentence. The MotivAider is generally set to go off once every fifteen minutes. So four times an hour, throughout the day the wearer is privately reminded to use technique.

After a few weeks of MotivAider prompting, old habits tend to be replaced by new ones. Please bear in mind that the MotivAider is only to be employed after you are certain, when prompted by it, that you can generate a perfect example of Intent Therapy. If you start using the MotivAider before you have attained this capability, you will find yourself being prompted to practice a great deal of imperfect technique - and this would completely defeat the purpose of using the MotivAider.

One final word about Intent Therapy. A slowed first word is a byproduct of good technique. The rest of the sentence, the afterthought, is always to be spoken at a normal rate. But what is a normal rate? Or, said somewhat differently, what is the average duration of a syllable in conversation?

It turns out that 'average' depends on a number of factors, the most important of which is whether the syllable is in a stressed or unstressed word. The average duration of a syllable in an unstressed word is about 125 milliseconds - in a stressed word, about 300 milliseconds. This means that the average rate of an unstressed syllable is about 8 syllables per second, and the average rate of a stressed syllable is about 3.3 syllables per second.

Recall that in Intent Therapy, multi-syllable word starts are to be spoken at a rate of 108 syllables per minute. That is equivalent to an average rate of 1.8 syllables per second - approximately half the rate of an average stressed syllable.

Apparently interesting things happen in the brain when we speak at different rates. In a study published in 2001, researchers, using functional magnetic resonance imaging to study the extent and magnitude of blood flows in the brain, had speakers repeat a syllable at three different rates: 2.5 syllables per second, 4 syllables per second and 5.5 syllables per second.

They found that different rates of speech activated different parts of the brain. In the cerebellum, there was a direct correlation between the syllable rates of 4.0 and 5.5 per second and the spatial extent and magnitude of the blood flows. The cerebellum seemed unresponsive to the slowest

rate. But in the basal ganglia, the area of particular interest in stuttering, there were proportional spatial extents and blood flows for the 2.5 and 4.0 rates, but not for the 5.5 rate. In other words, *the basal ganglia were not responding to faster rates, rates even remotely approaching the rates for unstressed syllables in ongoing speech. And remember, first words of sentences are typically spoken at unstressed rates (a lot of stuttering occurs at the beginning of sentences) as are the syllables of multi-syllable words spoken anywhere in a sentence (most feared words are multisyllables).*

It is tempting to hypothesize that high rates of speech, that is, speech composed of short-duration syllables, may exceed the basal ganglia's reciprocal inhibition capabilities, particularly in the presence of the joint effect of stress-induced reduced levels of GABA and stress-induced enhanced levels of the excitatory neurotransmitters dopamine and glutamate. Also note that recent attempts to train stuttering speakers to reduce the frequency of relatively their short phonation intervals during connected speech have shown enhanced fluency results.

Chapter 10

The sequence

We have several tools that can be used to deal with stuttering and there is a preferred sequence to follow when using them. But first, before beginning, remember to consult with your physician to get clearance. Also, should any undesirable side effects occur, cease taking all supplements immediately and contact your physician.

With that in mind, begin by taking The Thiamin Protocol Test. Remember, the test includes a week of dietary preparation followed by two weeks of supplement ingestion while continuing to maintain the dietary restrictions. Be sure to take the specific compounds and amounts of thiamin and magnesium recommended.

If you are among the 38% or so whose stuttering goes into remission it will be clear and unmistakable.

If you do not observe clear-cut evidence of enhanced fluency during the three-week test period, stop taking the supplements. Also, terminate the diet restrictions and consider moving instead to Intent Therapy.

Intent Therapy is a skill requiring mastery. Think of it as a sport that also addresses the trigger for stuttering. It is very powerful. The MotivAider (www.motiv-aider.com) is a tool to help you focus on this sport. It is a worthwhile investment and has a track record of several decades helping people break old habits and make new ones.

Afterword

The Thiamin Protocol does not work for everyone. The brain is an exquisitely complex structure requiring a number of approaches to the solution of issues. In this report we have presented what we consider to be a simple and effective solution for slightly more than a third of the world's young adult males who stutter.

But much more needs to be done. The goal must be to achieve higher remission rates. The original remission rates using thiamin alone were on the order of 30 percent, with magnesium added it appears to be on the order of about 38 percent. But more research is required to further clarify the optimum thiamin/magnesium ratios for fluency.

In addition, there are complex interactions between the derivatives of thiamin and magnesium, derivatives that cross the blood-brain barrier, that need to be explored. There are other minerals, such as calcium, other vitamins, such as B6 and herbs, such as valerian, that require examination. Even an optimal diet for fluency is probably an area worthy of investigation.

In addition, apart from The Thiamin Protocol, we have also used this report as the vehicle for introducing Intent Therapy. We view Intent Therapy as a viable alternative for those for whom The Thiamin Protocol is ineffective or for those who wish, eventually, to wean themselves off The Protocol. Intent Therapy is a powerful tool for subtracting pre-speech laryngeal tensions, an area that has heretofore not been fully explored.

Some may say that the conclusions drawn in this report regarding the efficacy of The Thiamin Protocol have not been adequately supported by scientific research. I would tend to agree. Obviously much more research needs to be done. But the results of these few studies when combined with the extensive clinical experience gained from a variety of sources tends to lend credence to the conclusions offered.

In addition, when the Thiamin Protocol works, it is as if a switch has been thrown. The results are dramatic and pervasive, producing a remission of symptoms in all speaking situations, a remission that persists as long as the supplements are taken. Nothing in the area of stuttering therapy has ever produced quite so startling a result.

The Thiamin Protocol is, hopefully, the first of a long line of productive outcomes to emanate from an ongoing series of observations that represent a new genre of inquiry. We hope eventually to be able to expand upon the findings described in this report, looking continuously to improve upon them as we learn more about the core of stuttering, its nervous-system underpinnings and how best to study and treat them.

Appendix

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