Self-Inhibition and the Disruptive Effects of Internal and External Feedback in Skilled Behavior*

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This paper examines the hypothesis that following activation, the components for producing a skilled behavior undergo a brief period of self-inhibition during which they are difficult to reactivate. Evidence for this self-inhibition hypothesis is reviewed from a wide range of areas: electrophysiology, electromyography, misspellings by dysgraphics, the perception and recall of briefly viewed misspellings by normal subjects, the nature of omission errors in speech production and transcription typing, and between-language regularities in the pattern of phoneme repetition within words.

The paper begins with a theoretical rationale for the concept of self-inhibition, based on a recently proposed theory of the relationship between the perception and production of speech (MacKay 1982, in press). I next outline the evidence for self-inhibition and finally examine some disruptive effects of feedback that can be explained as a side-effect of self-inhibition. I focus in particular on the role of self-inhibition in the disruption of ongoing speech production that results when auditory feedback is amplified and delayed about 200 ms.

SELF-INHIBITION AND THE NODE STRUCTURE THEORY

Nodes are the basic units for perception and production in the node structure theory of MacKay (1982, in press) and some nodes take part in both perception and production, whereas others do not. The unshared components include a system of sensory analysis nodes for representing patterns of sensory experience in perception and a system of muscle movement nodes for representing patterns of muscle movement in production. The shared components are mental nodes which represent phonological units such as segments and syllables, and sentential units such as words and phrases (see MacKay 1982).

Nodes have two states of activity: priming and activation. Activation is required for perception and action and is always sequential in nature. Moreover, activation always requires a special triggering mechanism to determine when and in what order the relevant nodes become activated. The numbers

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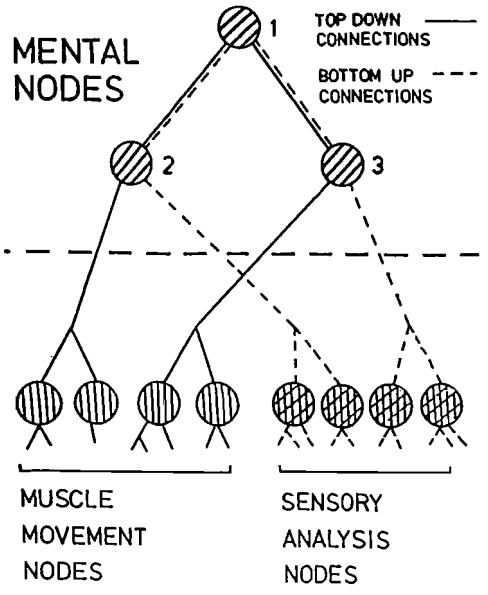

Fig. 1. The relation between mental nodes, sensory analysis, and muscle movement nodes

adjacent to the nodes in Fig. 1 represent this order of activation, but the special activating mechanisms required to achieve this order have been left out. (However, see MacKay 1982.) During its period of self-sustained activation, a node automatically primes all nodes connected to it. Priming refers to a transmission across the connection between nodes. This transmission produces an increased subthreshold activity which readies the connected node for potential activation. Priming summates over time, but cannot by itself cause activation. Activation instead requires application of an activating mechanism which causes the most primed node in its domain to become activated at the time when it is applied. All nodes become activated under this "most-primed-wins principle."

Domains consist of natural categories of nodes (e.g., noun nodes, verb nodes), all of which share the same activation mechanisms. Finally, errors in action and perception occur when an "extraneous" node (i.e., a node from outside a hierarchy such as the one in Fig. 1) receives greatest priming at the time when the activating mechanism is applied. Consider, for example, Freudian slips of the ear such as the misperception of *carcinoma* for *Barcelona* in the case of an individual listening to a sentence about *Barcelona* while simultaneously being concerned or preoccupied with the disease known as *carcinoma*. The misperception occurs because priming for the node representing *carcinoma*, arising from the preoccupation (top-down) and from aspects of the acoustic stimulus (bottom-up) exceeds the priming for the node representing *Barcelona*, arising from the input itself. As a consequence, the extraneous node representing *carcinoma* becomes activated under the most-primed-wins principle rather than the one representing *Barcelona* when the activating mechanism for nouns is applied. The theory predicts a general tendency for errors to involve substitutions from within the same domain. For example, nouns will substitute with nouns and verbs with verbs (see MacKay 1982).

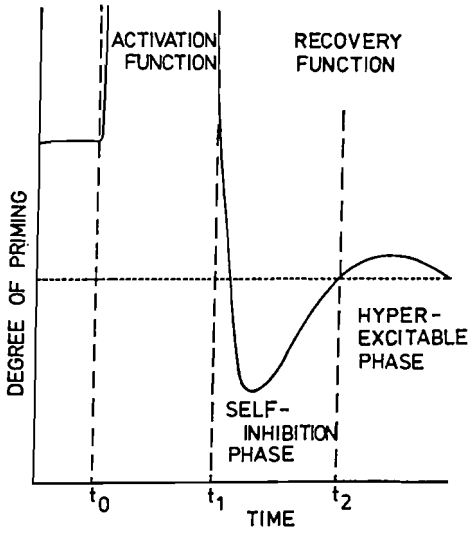

Fig. 2. The recovery cycle for a single node: self−inhibition begins at t_1 following activation at t_0. The period between t_2 and t_3 corresponds to the hyperexcitable phase

A RATIONALE FOR SELF−INHIBITION AND THE RECOVERY CYCLE

Self−inhibition becomes necessary whenever a node receives both top−down and bottom−up connections in an output hierarchy. Because only mental nodes are used both for producing and for perceiving cognitive units such as phrases, words, syllables, and segments, the self−inhibition postulate applies only to mental nodes and not to sensory analysis or muscle movement nodes. The reason can be seen in Fig. 1, which illustrates the detailed relations between sensory analysis nodes, mental nodes, and muscle movement nodes. Bottom−up connections are represented by the broken lines and top−down connections by the solid lines, and, as can be seen, mental nodes receive both top−down and bottom−up connections. These two−way connections lead to the possibility of reverberatory effects at every level in a system. The reasoning can be illustrated for two hierachically connected, but otherwise arbitrary nodes: A (superordinate) and B (subordinate). During production, A becomes activated and primes B via the top−down connection. However, when B subsequently becomes activated, this could lead to reactivation of A because of the bottom−up connection required for perception involving both of these same nodes. The self−inhibition that follows activation therefore ensures that higher level nodes do not become reactivated due to bottom−up priming arising from the activation of subordinate nodes.

I come now to the question of how nodes recover following self−inhibition. My basic assumption is that self−inhibited nodes undergo a normal recovery cycle, including a period of hyperexcitability or postinhibitory rebound during which self−priming first rises above and then returns slowly to resting level. Figure 2 illustrates the recovery cycle for a single node.

Activation begins at time t_0, when the activating mechanism is applied and ends at t_1 with the onset of self–inhibition. During the recovery phase in Fig. 2 priming first falls below resting level and then rebounds (the hyperexcitability phase) before returning to resting level again. The duration of the entire recovery cycle (from self–inhibition until the return to resting level) is assumed to vary with the level of the node in the system. For the lowest level nodes, the entire recovery cycle lasts only a few milliseconds. However, for higher level nodes, the cycle can last up 300 ms with a refractory phase of about 50 ms and a peak in self–priming at about 200 ms.

EVIDENCE FOR SELF–INHIBITION AND THE RECOVERY CYCLE

In this section I discuss various sources of evidence for self–inhibition and recovery with the general characteristics discussed above.

Electrophysiological Evidence

Although the figure of 200 ms for the peak period of hyperexcitability for mental nodes is only approximate, it receives support from the electrophysiological literature. For example, Tunturi (1958) found a postinhibitory rebound that follows activation by as much as 200 ms in central neuronal aggregates.

Electromyographic Evidence

Electromyographic activity without the occurrence of full–fledged muscle movement indicates that muscle movement nodes are being primed in the theory (MacKay 1981). Since spontaneous priming is assumed to peak at about 200 ms following self–inhibition, the theory therefore predicts a peak in electromyographic activity in the appropriate muscles 200 ms after production of a segment. Evidence for such a peak is found in a study by Ohala and Hirano (1967). They had subjects produce the syllable /pa/ while recording electromyographic activity in the obicularis oris musle of the lips and observed two peaks of subthreshold electromyographic activity. The first occurred just prior to the contraction of the lip muscles, and the other, smaller peak occurred about 200 ms later, as if reflecting rebound hyperexcitability.

The Production of Misspellings by Dysgraphics

A dysgraphic is someone who chronically misspells common words with very high probability, due perhaps to cerebral injury, but not to a lack of schooling or a general inability to learn. Lecours (1966) examined the misspellings of one dysgraphic (Lee Harvey Oswald) and discovered a repeated letter effect, which was analyzed further in a study by MacKay (1969a). Oswald often dropped a repeated letter in a word, e.g., misspelling *Elderly* as *Eldery*, but he sometimes added a repeated letter of his own in *Habitituated* and *Decemember*. MacKay (1969a) showed that deletions of repeated letters were significantly more common than additions and that deletions of the second of two repeated letters, e.g., *Eldery* were significantly more common than deletions of the first,

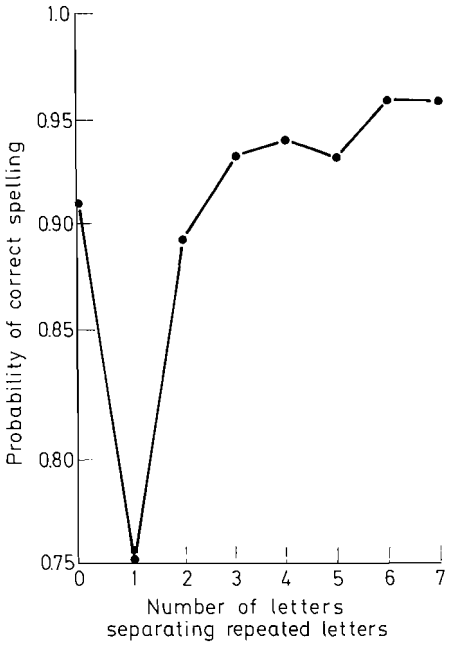

Fig. 3. The probability of correct spelling (vs letter deletion) as a function of the number of letters separating a repeated letter (from MacKay 1969a)

e.g., *Ederly*, as would be expected if repeated letter deletions reflected self−inhibition of nodes which are to be activated in sequence.

However, MacKay's most interesting data bearing on the self−inhibition hypothesis concerned the degree of separation of a repeated letter which became deleted. Oswald frequently misspelled repeated letters which were close together as in *Anlyze*, but he rarely dropped a widely separated repeated letter as in *Misspellng*. The probability of *correctly* spelling a word containing repeated letters in the sample is shown in Fig. 3 as a function of degree of separation of the repeated letters. For example, the repeated letter error in *Coperation* would be assigned Separation 0 since no letters separate the repeated letters in the error, that in *Anlyze* is Separation 1 since one letter separates the repeated letter in the error, and so on up to Separation 7.

As can be seen in Fig. 3, the probability of correct spelling was moderately high for immediately repeated letters (Separation 0), dropped dramatically for repeated letters with Separation 1 and increased to its highest level at Separation 6. Except for the moderately high probability of correctly spelling immediately repeated letters, the function in Fig. 3 can be seen to resemble and be explained by the recovery cycle function in Fig. 2. Moreover, the special status of immediately repeated letters is to be expected: unlike other repeated letters, immediately repeated letters are generally not pronounced in English, do not represent separate phonemes, and are coded via special orthographic rules which do not apply to repetitions with greater separation. All three factors suggest that zero separation letter repetition data should be disregarded or treated separately in this and subsequent figures. This granted, the data suggest that dysgraphic deletions may reflect an

abnormally strong and extended recovery cycle following self–inhibition of nodes for writing out the letters in words.

The Perception and Recall of Misspellings

Another source of evidence for the recovery cycle hypothesis comes from a study of the detection of experimentally constructed misspellings. MacKay (1969a) examined how easily normal subjects could perceive and recall misspellings which resembled the ones produced by the dysgraphic discussed above. The results showed that normal individuals experienced greatest difficulty in perceiving and recalling those experimentally constructed misspellings which were similar to the ones that the dysgraphic produced more frequently. The misspellings were planted in sentences which the subjects read at about 77 ms per letter. They then attempted to recall the sentence, writing it out exactly at it was spelled, guessing at misspellings if necessary. Following recall, the sentences were presented again and subjects were shown each spelling error in turn and asked whether they had noticed it when reading the sentence.

The results showed that repeated–letter misspellings such as *Eldery* were harder to perceive than nonrepeated–letter misspellings such as *Eldely*. Similarly, given that the subject claimed to perceive an error when reading the sentence, misspellings that involved repeated letters were more difficult to recall than those that did not. Moreover, the functions relating degree of separation of the repeated letters in both perception and recall resembled both the dysgraphic function (Fig. 3) and the recovery cycle function (Fig. 2). The perception function appears in Fig. 4 (left ordinate), which plots the probability of correctly detecting a repeated–letter misspelling as a function of the degree of separation of the repeated letters. As can be seen there, repeated–letter misspellings (the broken line in Fig. 4) were only harder to detect than nonrepeated misspellings (the solid horizontal line in Fig. 4) when 0 to 3 letters separated the repeated letters. However, when more than three letters separated the repeated letters, detecting a repeated–letter error was as easy or easier than detecting a nonrepeated–letter error.

The recall function also appears in Fig. 4 (right ordinate), which plots the probability of correctly recalling repeated–letter misspellings that have been detected. As can be seen there, repeated–letter misspellings (the broken line in Fig. 4) were only harder to recall than nonrepeated–letter misspellings (the horizontal solid line) when 0 to 2 letters separated the repeated letters. With more than three intervening letters, recalling a repeated–letter error was as easy or easier than recalling a nonrepeated–letter error. This degree of separation effect is readily explained under the recovery cycle hypothesis.

Limitations of MacKay (1969a)

Although the results of MacKay (1969a) support the recovery cycle hypothesis, there are a number of ways that the procedures could be extended and refined using computer–controlled stimulus presentation. Consider, for example, a task where the subject must detect a set of possible target letters in a rapidly presented sequence of displays, as in Shiffrin and Schneider (1977) except for the minor modification that a target letter can be repeated in the sequence. The subject has three tasks. The top priority task is to respond as quickly as possible to indicate detection of the first

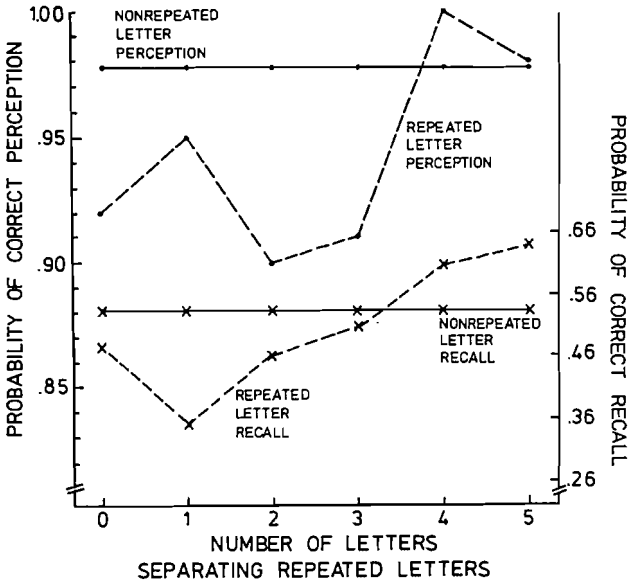

Fig. 4. The probability of detection (left ordinate dots) and recall (right ordinate crosses) for repeated letter mispellings (broken lines) and nonrepeated letter misspellings (solid lines)

target; the other, subordinate tasks are to indicate whether one or two targets were presented and what the target(s) were. In the special case where the same target letter is repeated (in either identical of different location in the display), the node structure theory predicts that occurrence of the first target will reduce detection of the second target if the intervening interval is short (up to 50 ms, say), but will facilitate detection of the second target when the intervening interval approximates some critical value corresponding to the hyperexcitable phase (say 200 ms).

Repeated Phonemes in Fast Speech

As expected under the recovery cycle hypothesis, speakers tend to drop immediately repeated phonemes (Separation 0) when speaking rapidly. However, determining exactly which nodes are responsible for this phenomenon is difficult. For example, when speaking rapidly, speakers of English often drop one of the /k/'s in *take care*, but it is often difficult to tell which /k/ was droped or whether the omission was intentional (Heffner 1964). Moreover, a single segment node cannot be responsible for this effect since these segments are produced by two different segment nodes, /k/ (final consonant group) and /k/ (initial consonant group). The omission can only reflect self–inhibition of nodes below the segment level.

Omission Errors in Speech and Typing

MacKay (1969c) identified a class of speech errors labeled motor masking (Type I) that unlike the rapid speech omissions, are undeniably inadvertent: the speaker unintentionally drops the second of two segments that must be repeated in close succession. An example of a German error from Meringer and Meyer (1895) is *der iese* instead of *der Riese*, where the immediately repeated /r/ has been dropped. Although such errors support the self–inhibition hypothesis (see MacKay 1969c), the use of experimental induction procedures seems advisable for studying these errors, which are relatively rare and require that the observers make subtle perceptual discriminations under less than optimal observational conditions (see Cutler 1982).

However, omission errors in skilled transcription typing exhibit a similar phenomenon which cannot be attributed to observer error. High–speed videotapes indicate that typestrokes proceed in three stages (Grudin 1983): a movement of the finger toward the key, a rapid downstroke to strike the key, and an equally rapid rebound or lift–off from the key. During an omission error the finger either fails to move toward the key or fails to execute the downstroke. Like Type I masking errors, typestroke omissions are usually preceded by an identical letter or finger movement, either in the preceding word or within the word itself. For example, Grudin had skilled typists transcribe a text as rapidly as possible and found that half of the typists had omitted the third /i/ in the word *artificial*. Moreover, in Grudin's overall sample of omission errors, the left–to–right masking (omission) effect was strong enough to override a general tendency for word–initial letters to be correctly typed. Example errors are, *the nitre* for *the entire* and *keep utting* for *keep putting*. Such errors suggest that following activation, the nodes for typing a letter and the movement components for striking the key undergo self–inhibition, which interferes with the subsequent execution of an identical movement due to follow closely in time.

The Structure of Phoneme Repetition

Somewhat indirect, but nevertheless interesting, support for the recovery cycle hypothesis comes from a study of phoneme repetition in the structure of words (MacKay 1970a). The rationale of the study was as follows: If a recovery cycle determines how easy it is to repeat an element in a word, then the structure of repeated elements in the word should reflect that recovery cycle. Immediate repetition of an element should be rare, reflecting the self–inhibitory phase of the recovery cycle. But at some point following self–inhibition, repetition should become more likely than would be expected by chance, reflecting the hyperexcitable phase of the recovery cycle. This of course assumes that speakers normally produce the repeated segments in a word at relatively fixed times on the average and do not alter their rate of speech in the middle of a word, depending say on the nature of its components, but such assumptions seem reasonable in view of phenomena such as constant relative timing (see Shapiro 1977). To summarize, the prediction is that immediate repetition of phonemes in the structure of words should be rare due to the recovery cycle constraint. However, repetition of a phoneme in a word should be highly likely after a degree of separation corresponding to the hyperexcitable phase.

To test this prediction, MacKay (1970a) examined the patterns of phoneme repetition in two very different languages, Serbo–Croatian and Hawaiian. The pattern of phoneme repetition in words of various lengths in the two languages appears in Fig. 5 and is remarkably similar for both

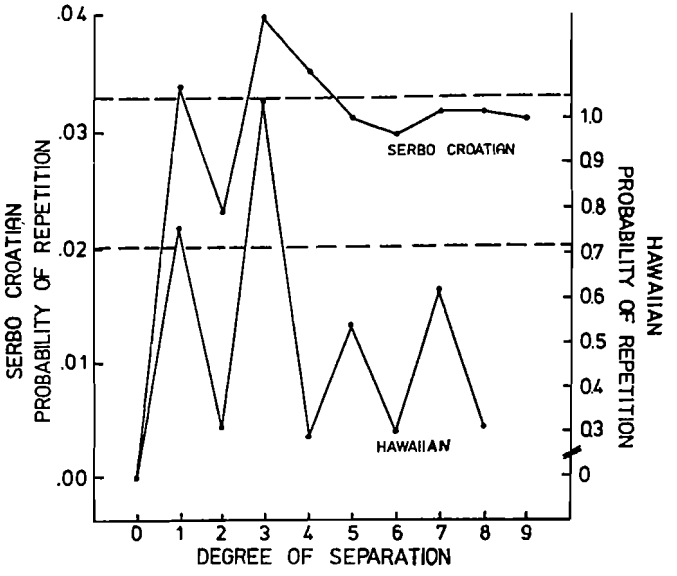

Fig. 5. The probalitity of phoneme repetition in Croatian (left ordinate dots) and Hawaiian (right ordinate crosses) as a function of degree of separation of the repeated phonemes. The horizontal lines represent probabilities under the hypothesis that phoneme repetition is a random event

languages. A very large and random sample of words was examined and the probability of segment repetition at the various separations was corrected on the basis of word length (since long separations for short words are impossible). As can be seen in Fig. 5, immediate repetition of phonemes was highly unlikely and significantly less likely than would be expected if phoneme repetition in a word were a random event (the dashed line in Fig. 5). However, the probability of repetition differed for consonants vs vowels, peaking with one intervening element of vowels in both Serbo–Croatian and Hawaiian, and peaking with about three intervening segments for consonants in both languages.

There were also differences between the two languages. As can be seen in Fig. 5, the repetition pattern was more erratic for Hawaiian than for Serbo–Croatian. The reason is that Hawaiian syllables generally have CV structure, whereas Serbo–Croatian syllables are much more varied and complex in structure. Thus, since vowels and consonants tend to alternate in Hawaiian, segment repetition at even numbered degrees of separation was of necessity infrequent. If syllable structure had been factored out in computing the probabilities of repetition, the Hawaiian and Serbo–Croatian functions would have resembled each other more closely.

As MacKay (1970a) points out, the peaks in Fig. 5 suggest a "law of latent alliteration" and the effect of Hawaiian syllable structure serves to illustrate the statistical nature of that law. Many other factors play a role in determining the phonological structure of words and can thereby override the law of latent alliteration in any one instance. Another important factor, especially for complex or derived words is the nature of the affixes and stems that combine to make up the word. Adding a different prefix could change the structure of repeated phonemes in the word. It is therefore

remarkable that a consistent repetition pattern should appear at all and that it should be so similar for Hawaiian and Croatian, languages that differ in the nature of their segments, number of segments (13 vs 35), and average number of segments per syllable and per word. The data therefore suggest, but do not prove, that the pattern of segment repetition in Fig. 5 is representative of all languages. Of course, if general properties of nervous action are responsible for this pattern, then a similar pattern should appear with only minor variations in all languages.

A great deal of further research is required to fully specify how a word acquires its phonological structure and the pattern of segment repetition within it. We know that phoneme repetition is a factor in the phonological changes that words undergo over time, since repeated segments often become dropped as a language evolves, e.g., the Latin word *stipipendium* changed to *stipendium*, dropping the repeated /p/ (see Meringer and Meyer 1895). These observations fit the prediction that phoneme repetition will represent a factor in the evolutionary changes that words undergo in the history of languages, but the extent to which the hypothesized recovery cycle plays a role in the initial invention of words or in the evolutionary changes that occur over time is currently unknown.

Consider now the limits of the revovery cycle hypothesis for theories of speech and other behaviors. One limit is that neither the recovery cycle nor constraints on repetition apply to all units for producing speech. Consider distinctive features, for example. Immediate repetition of distinctive features in adjacent segments is characteristic of many languages, which suggests that distinctive feature nodes either do not undergo self-inhibition, achieve extremely rapid recovery, or undergo activation for longer periods than segment nodes. This difference is consistent with the fact that distinctive feature nodes lack the two-way subordinate connections of other mental nodes: Because distinctive feature nodes receive bottom-up connections from sensory analysis nodes, but send top-down connections not to these same (sensory analysis) nodes but to different (muscle movement) nodes, the main raison d'être of self-inhibition (reverberatory reactivation) does not apply to distinctive features nodes.

Another limit concerns the potential or maximal rate of segment repetition. At best, data on the structure of segment repetition only suggest a repetition pattern that may be *easily* produced at average rates for everyday speech. The data say nothing about the potential or *maximal* rate for moving the speech muscles or for producing syllables. By exerting greater effort, speakers can undoubtedly exceed the natural repetition rates suggested in Fig. 5. A final limit to the recovery cycle hypothesis concerns the nature of the skill being investigated. Similar constraints on element repetition can only be expected for skills that employ shared input-output nodes. Skills that do not engage mental nodes cannot be expected to display the same recovery pattern.

SELF-INHIBITION AND THE DISRUPTIVE EFFECTS OF FEEDBACK

Self-inhibition is also necessary to prevent disruptive effects of external feedback. By way of illustration, consider the node labeled 3 in Fig. 1, a low-level mental node which becomes activated and gives rise to auditory feedback that I will label A. The sensory analysis nodes automatically process A and eventually prime 3, the node responsible for generating the output in the first place. This feedback-induced priming could cause multiple reactivation of 3, unless it

arrives during 3's period of self−inhibition. Self−inhibition is therefore needed to prevent another potentially catastrophic effect due to *external* feedback.

Stapedial attenuation plays a similar role in the node structure theory. About 100−165 ms before we begin to speak, the stapedius muscle in our middle ear contracts, thereby attenuating amplitude of eardrum vibration when hearing one's own voice. This attenuation continues throughout the period of overt speech and is conventionally considered necessary to prevent damage to the eardrum that might arise from prolonged screaming. However, if this were its sole purpose, one wonders why attenuation also occurs during everyday speech and whispering at levels which cannot possibly damage the eardrum. Under the node structure theory, the purpose of stapedial attenuation is not just to prevent peripheral damage, but to act as another defence against the disruptive effects of external feedback.

Both of these defences can be overcome by means of a special set of experimental circumstances which are readily explained within the self−inhibition hypothesis. For example, when expert generators of Morse code hear the click of their key amplified and delayed by about 0.2 s, they often make repetition errors, producing (n+1) instead (n) *dits* or *dahs*. Repetition errors also become prominent when normal speakers hear the sound of their own voice amplified over earphones and delayed by 0.2 s. As many investigators have noted, the effects of amplified and delayed auditory feedback are similar in many ways to the errors that occur during stuttering. However, other errors accompany delayed auditory feedback repetitions as well: Speakers involuntarily speak with greater amplitude, prolong and substitute speech sounds, and sometimes produce phonemes that are not part of any language familiar to them (Lee 1950). For adults, the disruptive effects of delayed auditory feedback increase as a function of delay up to about 0.2 s and then decrease with longer delays, but never disappear completely even with delays of 0.8 s (MacKay 1968). The delay producing maximal disruption of speech varies with age: 0.2 s for adults, 0.375 s for children aged 7−9, and approximately 0.85 s for children aged 4−6 (MacKay 1968). However, the peak delay does not vary as a function of language familiarity: When bilinguals produce either their more or their less familiar language under delayed auditory feedback, their peak delay remains the same (MacKay 1970b).

Practice or familiarity influences the *degree* of disruption at any given delay. Bilinguals stutter more when producing their less familiar language under delayed auditory feedback (MacKay 1970b) and practice in producing a sentence reduces the probability of stuttering when subjects subsequently produce the practiced sentence under delayed auditory feedback (MacKay 1970b). Probability of disruption also diminishes with age, since practice, familiarity, or experience in producing speech increases as children grow older (MacKay 1968). Mechanical distortion of the returning auditory feedback likewise reduces the disruptions without influencing the delay producing peak interference (Hull 1952; see also MacKay 1969b). In short, neither practice nor rate of speech nor distortion of the returning feedback influences the delay producing maximal disruption of speech, although all three influence the overall amount of interference. This outcome is exactly what one could expect if an individual−specific physiological factor such as recovery from self−inhibition were determining the delay for maximal interference. That is, under the node structure theory, mental nodes responsible for speech become self−inhibited and then undergo a period of hyperexcitability during which they acquire greater than normal sensitivity. The duration of this recovery cycle differs depending upon the level of the node: a few ms for the lowest level muscle movement and sensory analysis nodes, but about 300 ms for phonological segment nodes. That is, I assume that hyper-

excitability peaks at about 200 ms following activation of a phonological segment node and returns to normal spontaneous level about 300 ms following activation. This 200 ms peak would also fit the functions in Fig. 5 for the pattern of phoneme repetition in Hawaiian and Serbo–Croatian.

The maximal influence of delayed auditory feedback with a delay of about 0.2 s therefore seems to reflect an effect of feedback–induced priming arriving with sufficient strength at a critical time in the recovery cycle of just–activated nodes. Amplification of the returning feedback adds further to the priming of the just–activated nodes, and when these combined sources of priming exceed the "top–down" priming for the appropriate nodes in the same domain, these just–activated nodes become reactivated under the most–primed–wins principle, so that the output resembles stuttering (see MacKay and MacDonald 1984).

In summary, the self–inhibition hypothesis not only explains a wide range of relatively directly related phenomena, but also integrates phenomena such as stapedial attenuation and delayed auditory feedback that have hitherto seemed disparate and unrelated.

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