Effectiveness of Retraining Phoneme to Grapheme Conversion

Elizabeth Hillis Trupe
Medical Rehabilitation Center of Maryland, An Affiliate of HEALTHSOUTH Rehabilitation Corporation, Baltimore, Maryland

J.S., a 36-year-old right-handed female, suffered an embolic stroke during surgery following a motor vehicle accident. A CT scan later revealed a large ischemic infarct in the left middle cerebral artery distribution, involving the posterior frontal, anterior parietal, and superior temporal lobes. At 3 months post onset, J.S. presented with severe aphasia affecting all language modalities and profound oral-verbal apraxia. Six months later, her apraxia persisted, but her auditory and reading comprehension had improved to a functional level for most daily interactions. Boston Diagnostic Aphasia Examination (1972) scores were compatible with a classification of Broca’s aphasia. Her speech was limited to a single perseverative utterance, “God-God.” J.S. communicated by writing, augmented with a variety of gestures and drawing. Her writing consisted exclusively of content words, predominantly nouns. Most responses were correctly spelled real words, with frequent semantic paraphasias. A Words + Portable Voice, a computer system with typed input and synthesized speech output, was purchased to enable J.S. to interact, with her limited content and structure, via telephone and with nonreaders. However, intelligible output by the Portable Voice requires that many words be misspelled to approximate programmed “rules” of phoneme-grapheme correspondence. For example, the word “pizza” should be spelled, “p-e-e-t-s-a” for recognizable production. J.S. was unable to formulate alternate spellings. She was also unable to identify her own semantic paraphasias in writing or to spell unfamiliar words or syllables to dictation, suggesting impaired phoneme-to-grapheme conversion processes.

The subject understood spoken and printed words, as demonstrated by word-to-picture matching tasks (Table 1), but could not match printed to spoken words reliably. Writing to dictation was much less accurate than spontaneous writing. These discrepancies are also consistent with poor phoneme to grapheme conversion.

A model of cognitive processes involved in letter-writing to dictation tasks was considered in planning remediation for writing target phoneme sequences. This model (Figure 1) was described by Mills and Kaufman in 1978, adapted from Weigl and Fradis (1977).

The model is based on postulated transcoding processes, by which units of one code system are transposed into appropriate units of another code system. According to Weigl and Fradis, damage to the cognitive system may disrupt any of the transcoding systems. Since J.S. accurately reproduced letters and wrote serial letters, Type 2 transcoding (from the visual perceptual code to the graphic response) appeared to be intact. However, Type 1 transcoding (from the auditory perceptual code to the visual perceptual code) was considered grossly impaired, indicated by her inability to point to letters corresponding to phoneme stimuli. Since direct training and drills of phoneme-to-grapheme conversion rules failed completely, a treatment plan was designed to access the concept code, or semantic system, to intervene in phoneme-to-grapheme transcoding. The model in Figure 2 was postulated, based on that of Mills and Kaufman. Terms in parentheses are my own interpretation.
Table 1. Pre- and post-test performance.

<table>
<thead>
<tr>
<th>Task</th>
<th>Pre-Treatment (%)</th>
<th>Post-Treatment (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Writing Monosyllabic Words to Dictation:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consonant-vowel sound (e.g., bay)</td>
<td>12</td>
<td>64</td>
</tr>
<tr>
<td>Consonant-vowel-consonant (e.g., lip)</td>
<td>20</td>
<td>84</td>
</tr>
<tr>
<td>Minnesota Test for Differential Diagnosis of Aphasia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visuomotor and Writing Disturbances:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reproducing letters</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Writing letters to dictation</td>
<td>31</td>
<td>88</td>
</tr>
<tr>
<td>Written spelling (words to dictation)</td>
<td>50</td>
<td>90</td>
</tr>
<tr>
<td>Writing sentences to dictation</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>Visual and Reading Disturbances:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Matching letters (1:5)</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Matching words to pictures (2:1)</td>
<td>94</td>
<td>94</td>
</tr>
<tr>
<td>Matching printed to spoken words (2:1)</td>
<td>63</td>
<td>97</td>
</tr>
<tr>
<td>Auditory Disturbances:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recognizing common words</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(pointing to named pictures, 1:6)</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Recognizing letters</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(pointing to named letters, 1:5)</td>
<td>23</td>
<td>88</td>
</tr>
<tr>
<td>Discriminating between word pairs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(pointing to named pictures, 1:2)</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure 1. A model of normal writing to dictation.
Figure 2. A model for training phoneme to grapheme transcoding via key words.

Treatment hinged on teaching the subject to associate each phoneme with a familiar "key word" that she could spell.

METHOD

A multiple baseline across behaviors design (McReynolds and Kearns, 1983) was chosen to analyze the effects of treatment. The design consisted of a baseline phase, followed by sequential treatment phases. Thirty English phonemes with common graphemic representations were randomly divided into three sets (A, B, and C) to be trained separately. Baseline data were collected by presenting the instruction, "Point to the letter that makes the sound (phoneme)." No feedback was provided. J.S. was required to identify the letter on a keyboard, rather than write the letter, in order to focus treatment on transcoding Type 1 to the exclusion of transcoding Type 2 (Figure 1). Responses were considered correct if J.S. identified any letter that could be associated with the stimulus phoneme in English (e.g., c, k, or ck were accepted for /k/). Pre- and post-tests are listed in Table 1. Treatment involved presentation of the following hierarchy of stimuli:

1. Point to the letter that makes the sound (phoneme).
2. Think of a word that starts with (phoneme).
3. A word that starts with (phoneme) is (key word). Point to the letter that makes the first sound of (key word).
4. Write (key word). Point to the letter that makes the first sound of (key word).
5. (target grapheme) makes the first sound of (key word). (phoneme) is for (key word) (e.g., "B makes the first sound of baby. /b/ is for baby.")

When the target grapheme was identified in response to any stimulus, subsequent stimuli were not presented. Only the response to the initial
stimulus was scored. Treatment continued with set A stimuli until J.S. achieved 80% accurate phoneme-to-grapheme matching for two successive sessions. When she reached criteria for set A, sets B and C were individually and sequentially trained to criteria. Maintenance of previously treated sets involved treatment every third session.

RESULTS

Figure 3 shows that the intervention (training to associate phonemes with key words) increased phoneme to grapheme matching when introduced in each set of phonemes.

![Figure 3. Percent correct phoneme to grapheme matching.]

Mean baseline performance was below 20% accurate for each set, and performance improved abruptly with intervention, reaching 90-100% accuracy for all sets. Generalization to untrained phonemes was not clearly demonstrated. However, J.S. reached criterion more quickly for each successive set. Generalization to untrained tasks was suggested by increased accuracy of spelling words to dictation, pointing to named letters, and matching printed to spoken words (Table 1). Spelling of monosyllables not only improved in accuracy, but also showed a shift in predominant error type, from semantically related words (e.g., "eye" for see) before treatment to graphemically related words (e.g., "gan" for gas) after treatment. Good maintenance of learned associations was demonstrated in retesting of all phonemes after one and twelve months with no specific treatment.

CONCLUSIONS

Phoneme-to-grapheme matching can be trained by accessing the semantic code through use of trained key words. The resulting system can be used and maintained for writing short words to dictation, for writing unfamiliar words, and for reducing paraphasic errors in writing.
DISCUSSION

Evidence that J.S. actually used the key word to transcode from the phoneme to the grapheme was revealed in her errors. Her cognitive processes became more evident as she began to produce telegraphic speech. When instructed to write the letter for the stimulus, /s/, J.S. wrote m. When asked the key word, she wrote "Mindy" (the name of one daughter) and said, "Sam" (the name of her other daughter and the key word for /s/). Apparently, J.S. followed a learned decoding, transcribing, encoding process appropriately, but showed a disruption between her concept code and her graphic-lexical code, as in the following example.

1. Decoding of auditory stimulus [s]
2. Transcoding [s] to auditory perceptual code /s/
3. Accessing auditory lexical code /Sam/
4. Transcoding /Sam/ to concept code, "oldest daughter"
5. Transcoding concept code ("oldest daughter") to lexical graphic code, Sam (correct) or Mindy (semantic confusion)
6. Grapho-motor encoding of lexical graphic code
7. Graphic response g (or m if semantic confusion occurred)

This study supports models of cognitive processes used for spelling described by Goodman and Caramazza (1985). These authors have proposed a dual system by which normal writers spell novel or unfamiliar words via phoneme-to-grapheme conversion rules ("assembled orthography") and spell familiar words via a lexical-semantic system ("addressed orthography"), as shown in Figure 4. Analogous constructs referred to earlier in the model by Mills and Kaufman are inserted in the figure.

Figure 4. Lexical, non-lexical, and post-graphemic processes used for spelling.

J.S. needed to access both systems of orthography to spell novel words because her phoneme-to-grapheme conversion rules were disrupted by her stroke. Her resulting system, modeled in Figure 5, is slow and occasionally in error secondary to breakdown in her lexical-semantic system. Perhaps continued reinforcement will eventually result in condensation of compensatory cognitive processes, allowing her to more efficiently transcode auditory stimuli into graphic output. However, 12 months after treatment ended, J.S. persisted in using key words to transpose phonemes into graphemes and to spell monosyllabic words. In writing longer words or sentences to dictation, she continued to rely on impaired addressed orthography, resulting in frequent semantic confusions and inconsistent error awareness.

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Figure 5. A model of J.S.'s writing to dictation.

J.S. started reading aloud and repeating monosyllables at 11 months post onset, during the maintenance phase for all phoneme sets. Independent production of words emerged later. It is tantalizing to hypothesize that treatment of phoneme-to-grapheme transcribing resulted in de-blocking of grapheme-to-phoneme transcribing, since improved writing of monosyllabic words to dictation (16% to 74% correct) was concurrent with improved oral reading of the same words (0% to 80% correct). Weigl (1974) discussed the possible role of intact phonemic-graphemic correspondence rules and repetition in de-blocking word comprehension. He studied aphasics who initially had intact auditory and reading comprehension of words, but could not match printed to spoken words (Table 2). Both Weigl's subjects and J.S. confirm the existence of phoneme-to-grapheme transcribing rules separate from the semantic system. This study demonstrates that treatment can result in utilization of the semantic system to transcode phonemes to graphemes when these rules are disrupted by brain damage.

REFERENCES


DISCUSSION

Q: In the early days of the treatment of epilepsy, it was assumed, first of all, that the epileptic had seizures because he or she masturbated. There was evidence that bromides reduced sex drive. Somebody took those two observations and put them together, and decided that the best way to treat epilepsy was to give the epileptic bromides. Sure enough, seizures were reduced, although it probably didn't have anything to do with reducing the sex drive; it probably had to do with something about the influence of bromides on neurofunction. Lest the implications of that not be clear, let me just ask you if you have other studies in mind that will help to confirm your feeling that response of the kind that patient gave you to the kind of therapy that you did does indeed suggest the reality of the model that you approached the treatment with. What do you have in mind as a way of further exploring the power of that particular model to guide our treatment? And as a companion question, are there other explanations for the finding that are not as exotic as transcoding?

A: Maybe, but I don't find the concept of transcoding exotic to explain the responses that she was giving when I asked her to point to a letter associated with a given sound. That's a fairly simple task that she could not initially accomplish. I think what allowed her to accomplish it after treatment was that when she heard a sound, she thought of a particular word. She could spell that word, and she could visualize that word. And she could think of what letter it started with, and then point to that letter. The concept of transcoding is simply moving from the representation of the sound, which she was given, to the representation of the letter, which she then responded with. That seems to me a very simple construct to describe the process, so I didn't search for other ones. At this moment, I can't think of other ones. Can you?

Comment: No. I'm not carrying the details of either the model or your method in my head as I stand here, so you'll excuse me if I stumble or even fall down. I'm just wondering if we don't all have the obligation to somehow test these models on a variety of patients, and, in fact, test them in ways that would produce data contrary to the predictions of the model if, for example, the real influence on treatment is merely the systematic presentation of stimuli of a number of sorts and the systematic reward of a variety of responses. I just think so much of what we do in therapy is much like using steroids—that the effects are so general, at least potentially, that we really have the obligation to test a great many patients before we use data from treatment for confirmation of a particular model.

A: I do think that we need to test other patients and that one patient does not confirm a model. But I would like to comment that I used the transcoding model to develop treatment rather than using treatment to specifically confirm the model. I think the treatment was more directly related to what I perceived to be the problem because I had the model in mind before I did the treatment.

Q: You noticed that the patient's semantic paragraphs decreased with your training procedure. Why do you think that happened?

A: I think that she was doing some ongoing self-monitoring. Perhaps when she was dictated the word "see," instead of writing "eye" as she might have originally thought of, she might have thought, "That can't be right,
because "see" starts with /s/." Or she might have started with the /g/ because she made that connection, and she couldn't finish it by writing "eye." So I think she was just doing more ongoing self-monitoring when she was writing.

Q: I guess I have a question about whether your evidence really does support the model, though. At least if I understand your paradigm correctly, what you did is you gave the individual a letter; the individual then had to visually scan the environment to find that letter; and then based on finding that letter, she then thought of words?

A: No, look at the treatment program, the stimuli hierarchy, if you will. First, she was to think of a word, then identify the letter. Initially, if she was given the sound [m] she couldn't identify the corresponding letter. If I said, "Think of a word that starts with the sound [m]," she couldn't do that either. The next stimulus was, "Well, a word that starts with [m] is Mom. Point to the letter that makes the first sound of 'Mom'." Then she pointed to /g/ because she knew how to spell 'Mom' already. She was not speaking when we started treatment, but she was writing content words, which included more than 30 key words that we used to associate with the phoneme stimuli.

Q: The purpose for your going into this was that she was using an automatic voice speaker. After all this training, was she able to use it any better?

A: She was; but she didn't use it, in fact, because by that time, she was starting to produce telegraphic speech. We did not expect that she would ever say anything much but "God-god." As soon as you become at all a functional speaker, you don't want to carry around a little computer to communicate with. So what she in fact does now is speak; or she can more frequently write a word, and then read it after she writes it.

C: I'm not too concerned about the model. I can probably say that the importance of your study to me is that you demonstrated a functional relationship between your treatment and your patient's improvement. In a sense, the model is extra baggage, for me. And I think that rather than reinforcing some of the suggestions for more patients to support the model, I would even go a little further and say that perhaps the methodology isn't the best to look at this type of model. Not that you can't and make those inferences, but I'm less comfortable with it. For example, we might be better off looking at response times for P-codes vs. C-codes as they do with the Sternberg model and things like that. Looking at latencies and then inferring conceptual processes or different stages in processing is more consistent with looking at that type of model. And that's fine, too - but that to me it is still very inferential. But I think that we may get in trouble if we cross methodologies to answer questions that are perhaps not consistent with those different methodologies. So what I'm saying is that your study is important, and it's important to me because it shows a functional relationship between treatment and improvement. But rather than having replications that would perhaps support or not support the model, I would suggest that there are different ways to look at conceptual processes and to make those inferences, and to me that may not be the best way. The importance is that the patient improved, and it's a very direct relationship.
Q: What intrigues me with a treatment that seems to work with one patient is, will it work with others? Then one is led to develop selection criteria so you don’t waste treatment, but you find out in advance whether it’s going to work. Would you think that something as silly as testing a patient up front on a group of irregularly spelled words and phonetically pronounceable non-words might be a selection criterion for this treatment?

A: Absolutely. I tried that with J.S., and she refused. She wouldn’t write non-words because she knew they weren’t words; it was silly, and she shouldn’t have to try to write them.

Q: And she did well with irregularly spelled words?

A: Initially, she had semantic paraphasias, but she did write some. She showed no difference between irregularly and regularly spelled words.