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Evidence-based practice in stuttering: Some questions to consider

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Abstract

A recent forum in *JFD* (28/3, 2003) evaluated the status of evidence-based practice in fluency disorders, and offered recommendations for improvement. This article re-evaluates the level of support available for some popular approaches to stuttering therapy and questions the relative value placed on some types of programs endorsed by the forum. Evidence-based practice is discussed within the context of emerging concerns over its application to non-medical interventions and suggestions are made for grounding fluency interventions by reference to empirically supported principles of change. A popular, evidence-based intervention for stuttering in young children (the Lidcombe program) is evaluated within the suggested parameters.

Educational objectives: After reading this article, the reader will be able to: (1) evaluate the status of evidence-based practice in fluency disorders; (2) list concerns about the impact of narrow interpretation of EBP on research and practice in the field of fluency disorders and other non-medical domains; (3) articulate the role of theory and basic research in selecting among and evaluating therapy approach options.

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In a recent issue of *JFD* (28, 3) (hereafter, 28/3), a number of authors (Bothe, 2003; Finn, 2003; Ingham, 2003; Onslow, 2003) proposed certain standards to which stuttering treatment should be held and further suggested that only a limited number of therapy programs currently meet such evidence-based practice (EBP) standards. In

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29 the same series of articles, some authors appeared to imply that treatment programs
30 not meeting the specified standards, and the clinicians who administer them, may in
31 fact be engaging in less than ethical clinical practice, since they volitionally forgo
32 a small set of “validated” techniques for those seemingly supported by a lesser evi-
33 dence base. In this space, I would like to further discuss such implications as well
34 as related, seemingly provocative issues. I will also address larger issues in evaluat-
35 ing the degree to which EBP is currently fully “ready for prime time” implementa-
36 tion in the field of fluency disorders. In doing so, I will frame my comments in the
37 form of a series of questions that I think we need to ask and consider answering be-
38 fore applying some of the extended principles of EBP to the field of stuttering interven-
39 tion.

40 On the surface, evidence-based practice is a noble concept and goal. Indeed, it would seem
41 nonsensical to argue for therapeutic practice that is not based on some body of evidence.
42 I will not, therefore, position myself as saying that evidence-based practice is wrong. At
43 the same time, certain embodiments and extensions of evidence-based practice seem less
44 obviously of value and may in fact pose difficulties for researchers, clinicians and their
45 patients.

46 In framing this article partially as a response to the authors in 28/3, I prefer to start
47 with areas of agreement. I wholeheartedly concur with the obvious need for practition-
48 ers to document the rationale for their selection of therapy approaches. As such, I also
49 most wholeheartedly agree with the general consensus of the authors in the issue that
50 we need far more research into therapeutic efficacy in stuttering treatment. Thus, I agree
51 with [Ingham \(2003\)](#) that researchers need to develop more interest in therapy trials, and
52 that our funding agencies, particularly the American National Institutes of Health, need
53 to invest in them more aggressively. It is possible to conjecture that the lack of funded
54 research in therapy efficacy is at least in part due to the relative paucity of such applica-
55 tions when weighed against the bulk of submissions that propose basic research ques-
56 tions. Despite these strong areas of agreement, however, my affinity for some, if not
57 many, of the arguments raised in the issue begins to wane considerably, because they
58 raise a number of vexing questions. I address what I view to be the most important of
59 these questions in the remainder of this article. Among the issues that I will consider
60 are:

- 61 1) The nature and scope of “evidence” and its relationship to clinical practice;
- 62 2) The limitations that may be associated with the use of a single framework to imple-
63 ment evidence-based practice across medicine and the many health-related profes-
64 sions;
- 65 3) The role of different types of evidence in determining the value of specific therapy
66 approaches in stuttering;
- 67 4) The role of theory in evaluating treatment approaches;
- 68 5) Potential barriers to the gathering of clinical evidence and its implementation by prac-
69 tioners; and finally,
- 70 6) Some logical “next steps” that will be required if practitioners and researchers
71 are to bridge the perceived gaps between evidence and practice in stuttering treat-
72 ment.

73 1. What is evidence?

74 Is evidence a “fuzzy category”? A “fuzzy” category in psychology or linguistics is one
75 that seems to have an identity that can be agreed upon, but has features that are difficult to
76 specify exactly (Rosch, 1973; Rosch & Mervis, 1975). Fuzziness has also been applied to
77 the evaluation of data, as in fuzzy logic (Zadeh, 1965). In the past 30 years, “fuzziness” has
78 spilled over to accounts of logical decision-making in the physical, biological and social
79 sciences, indicating that one person’s data may or may not be sufficient to be useful to
80 another’s evaluation of a set of facts or features.

81 In much the same way that semantics has argued about the boundaries that separate cups
82 from bowls, it is not clear that any field can arrive at a perfect definition of evidence, or list of
83 its features, although some will find it easier to define operationally, such as pharmaceutical
84 interventions in medicine. Narrow criteria going into an evidence-based test of intervention
85 effectiveness will improve the likelihood that professionals will agree on the value of the
86 evidence that is produced. For example, if the question is whether a drug lowers blood
87 pressure, there are only a few accepted measures of outcome.

88 In medicine, many questions which could appear simple are complicated by the “messi-
89 ness” of the typical patient, who rarely presents with a single problem, or canonical features.
90 Thus, the problem of treating the “whole person” appears to create a sense of fuzziness in
91 dealing with evidence—to what extent will carefully gathered treatment evidence bear a
92 reasonable relationship to the actual case under consideration? How are treatment outcomes
93 in one domain related to the overall functioning of the individual in other domains? This
94 fact has generated a certain degree of tension between certain groups in medicine regarding
95 the importance of personal experience with well-defined individuals in one’s application of
96 evidentiary meta-analyses derived from large groups (Pope, 2003). There is a growing body
97 of reports in which physicians, for example, argue that the complexity of individual profiles
98 supercedes evidence from carefully controlled trials. Thus, there is growing documentation
99 that some medical practitioners “resist” evidence, when, in fact, they appear to allocate
100 value to different types of evidence in making individual clinical decisions. An important
101 issue in such resistance is not so much a devaluation of new evidence, but personal experi-
102 ence with previous existing treatments that “work” for the clients one typically sees. This
103 type of evidence can be called “anecdotal” as various authors in 28/3 note, but is powerful
104 when the clinician is both gatherer and applier of the data, something quite different from
105 anecdotal evidence associated with authority of peers or other respected professionals (so-
106 called “authority-based” evidence; Onslow, 2003). One could say that there is evidence that
107 one reads about and evidence that one encounters in everyday practice.

108 2. Evidence of *what*?

109 This may seem like a silly question, but if one wants to evaluate evidence, one must
110 ask what concept, notion, or idea the evidence has been designed to inform. In fluency
111 treatment, this is not necessarily straightforward, although some would like to limit the
112 nature of the “primary” evidence of interest to measures of stuttering frequency as outcomes
113 of certain interventions. Ingham (2003), for example, is quite open in her assessment that

114 observable behavior “trumps” less observable concomitant or predisposing components of
115 the disorder, and that is a reasonable opinion to adopt. I would like to point out that the
116 notion that one treats a condition only by addressing its observable symptoms is somewhat
117 controversial, however. For example, going beyond the field of stuttering, it is not clear that
118 current psychological theory uniformly endorses such an approach, which might presume,
119 for example, that maladaptive classroom behavior is best treated symptomatically, rather
120 than by searching for its root causes, motivators, or maintainers. Modern criminology, for
121 instance, looks for the roots of delinquent behavior in a plethora of preceding and underlying
122 factors, rather than merely focusing on deterrence (selective consequence of undesirable
123 behaviors). As an example, in their large-scale meta-analysis, [Petrosino, Turpin-Petrosino,](#)
124 [and Buehler \(2002\)](#), noted that cognitive-behavioral interventions are far more effective
125 in preventing recidivism in juvenile delinquency than punitive system responses. In such
126 models of treatment, frequency of incarceration is ultimately compelling evidence, but the
127 treatment targets are not the same as the outcome targets. In such studies, evidence also
128 comes in “stages,” first those that show an immediate, targeted change, and then those that
129 show achievement of the final, different goal. In our field, this would be similar to asking
130 whether or not attitude change eventually induces fluency changes, even if this cannot be
131 measured in a short-term study.

132 Similarly, stuttering frequency is certainly one logical outcome measure for documenting
133 therapeutic effectiveness when treating people who stutter, but not all agree it should be the
134 *only* one or the first one ([St. Louis, in press](#)). In particular, if a program diminishes percent
135 stuttered syllables (%SS) by 50% but maladaptive attitudes are neither assessed at intake or
136 therapy dismissal, or are shown to remain unchanged, how is such evidence to be evaluated
137 or balanced with other observations of the same individual? At the same time, it is not clear
138 that attitude change unaccompanied by any changes in stuttering frequency or quality can
139 be considered evidence of fully successful therapy.

140 3. Can all fields use the same evidence-gathering framework?

141 Another way to phrase this question is, “how is evidence best produced?” One cannot
142 argue the merits of progressively more demanding tests of treatment effectiveness in any
143 discipline. However, requiring all evidence to fit within the medical community’s current
144 hierarchy of evidence types may not be possible for fields such as ours. Fields such as
145 physical therapy, education and social work face difficulty in implementing components
146 of Randomized Clinical Trials (RCTs) such as blinding, placebos, and other common con-
147 structs. They don’t seem to be a natural fit for all therapies within the discipline ([Swinkles,](#)
148 [Albarran, Means, Mitchell, & Stewart, 2002](#)), despite being hailed as a gold standard for
149 determining therapeutic efficacy in medicine, and now, stuttering ([Ingham, 2003](#)). For ex-
150 ample, placebo pills are clearly easier to administer than placebo behavioral treatments.
151 “Washout” phases (to assess the effects of removing a treatment), so appropriate for short-
152 duration medications, are not as easily evaluated when treatments involve client learning
153 of techniques, concepts, reactions, etc. Once some treatments have been applied, they are
154 not readily “un-applied.” Thus, while many fields agree that evidence can be derived from
155 RCTs ([Feinstein & Horwitz, 1997; Sailor & Stowe, 2003](#)) not all fields would agree that it

156 has to be, and in some cases, argue that it should not be. Even in medicine, [Culpepper and](#)
157 [Gilbert \(1999, p. 831\)](#) note that

158 . . . the emphasis on (EBT) . . . has raised the Randomised Clinical Trial (RCT) to
159 a position above other forms of biomedical research. Yet, primary care research,
160 with its emphasis on outcomes, natural history, human behavior, cultural issues, and
161 questions related to care rather than cure, is of vital importance to an understanding
162 of our patients, their problems, and the way we can best serve them. These issues are
163 suited to qualitative research designs, descriptive work, and case-control or cohort
164 designs; RCT designs rarely apply. That we neglect these critical research priorities
165 in favor of (RCTs) . . . is tragic.

166 One apparent concern with EBP, as identified by [Sailor and Stowe \(2003\)](#), is that we
167 cannot allow social policy to view program efficacy as either a race or as a construct only
168 evaluable by application of a tightly specified subset of research methods. I will not bore
169 readers with their detailed and hopelessly familiar analogy of the drunk seeking his keys
170 only under the lamppost because “the light is better there,” but will note their concern that
171 in the messier, real-world contexts of behavioral change in childhood and beyond, we can-
172 not limit ourselves only to those questions which lend themselves to randomized clinical
173 trial designs; nor can we necessarily view an RCT within a discipline as superior to other
174 forms of therapeutic evidence being gathered concurrently. Sailor and Stowe are adamant
175 in their distaste for the RCT as the model for educational research, a close colleague of
176 the work performed by speech language pathologists. [Salkovskis \(2002\)](#) echos their con-
177 cern in the application of RCTs to cognitive-behavioral treatments. As noted earlier, it
178 is not clear that RCTs will apply to situations where conditions cannot be counterbal-
179 anced, blinded or “withdrawn” to verify the impact of the intervention on the behavior
180 of interest. Only operant or similar behavioral approaches to fluency treatment seemingly
181 fit this model. Therefore, blanket acceptance that RCTs represent the gold standard for
182 speech-language intervention may artificially constrain which approaches to intervention
183 are even eligible for equal consideration. It is not clear that a fluency shaping or mod-
184 ification skill, once taught, can be untaught, or the components of cognitive therapy be
185 undone.

186 **4. What are the qualities of good evidence?**

187 First, in choosing from the available data, publication in a peer-reviewed journal is often
188 considered the default, as it should be. Our best evidence is to be found there, rather than
189 in books or workshops, neither of which are held to rigorous peer-review standards Jour-
190 nals tend to be quite concerned by the design of trials that report therapeutically relevant
191 evidence. To raise the bar, we could set minimum standards for the number of subjects
192 required to provide evidence of therapeutic effectiveness, require thorough baselining, de-
193 mand evidence of skill generalization, durability of results, etc., in order to publish any
194 results; however, to date, we have not set such standards. And the lack of such standards
195 is probably fortuitous given the fact that historically, such requirements would be likely
196 to stultify progress. Witness the fact that many major discoveries in medicine, psychology

197 and other fields have begun with publication of individual case reports or case series that
198 spurred further basic and applied research (Vandenbroucke, 2001).

199 Beyond the features of individual research reports, an important component of the value
200 of evidence is its replication by sites not viewed as major “stakeholders,” something recog-
201 nized even in 1984 by Andrews, Craig, Feyer, Hoddinott, Howie, and Neilson in their
202 review of stuttering therapies, and certainly held as important in other fields. Why is this
203 relevant? In stuttering, of the roughly 50 published articles on the effectiveness of the be-
204 haviorally or operant-oriented Lidcombe program of intervention (Onslow, Packman, &
205 Harrison, 2003), virtually all have program authors as article authors. As we improve the
206 level of evidence-based practice in fluency disorders, we will clearly need more evidence
207 from clearly independent sites (Lohr, DeMaio, & McGlynn, 2003) that have neither actual
208 nor perceived personal, professional or financial interests in the outcomes.

209 It is interesting that, outside the field of speech–language pathology, other fields have
210 moved from asking that therapies or interventions be grounded in evidence to asking deeper
211 questions about the nature of the evidence itself, rather than the design of trials meant to
212 produce evidence. For example, an important concept now operative both in medicine
213 and psychology is that therapy programs should be “dismantle-able” into their component
214 efficacious parts (Rosen & Davidson, 2003) to isolate “empirically supported principles
215 of change” or enable understanding of the “causal processes and mechanisms” (Michie &
216 Abraham, 2004). I will return to this issue later.

217 5. Is there evidence in stuttering treatment?

218 Yes, of many differing approaches and components. Ramig and Bennett (1997) and
219 Culatta and Goldberg (1995) are among those who provide annotated summaries of the
220 degree to which extant therapies are evidence-based. Surveys of practicing clinicians suggest
221 that they employ a healthy mix of these evidence-supported approaches, including fluency
222 shaping, attitude modification, and operant techniques. Cooper and Cooper (1996) reported
223 that only 7% of practicing clinicians disagreed with the premise that a “combination of
224 operant and self-evaluative-type therapies (sic) is probably the most efficacious type of
225 therapy for stutterers,” a finding that Cooper and Cooper (1996) believed to be “reassuring”
226 (p. 132).

227 Thus, in some respect, much of what seems to be at stake in positioning “camps” in
228 stuttering treatment is the relative focus on, contribution(s) and merits of potential therapy
229 components involving stuttering modification, cognitive therapies or counseling in addition
230 to negative contingencies, and/or fluency shaping components and/or GILCU (to para-
231 phrase Ingham, 2003, pp. 203–204). Despite some expressed pessimism about the level
232 of empirical support for many popular approaches to stuttering therapy (Ingham, 2003),
233 one can find ready evidence for the value of many therapy components in the treatment
234 of stuttering. This body of evidence includes documented support for the so-called “tradi-
235 tional” approaches (fluency shaping with or without components of stuttering modification;
236 Baumeister, Caspar, & Herziger, 2003; Conture, 2001; Conture & Melnick, 1999; Gottwald
237 & Starkweather, 1999; Runyan & Runyan, 1999). Within therapy programs that address
238 the cognitive and affective concomitants of stuttering, there is evidence of the effective-

ness of interventions to improve self-efficacy and locus of control (I note that some of these are from outside the field of speech–language pathology, a concept I will return to later; Bandura & Locke, 2003; Graves, 2003; Multon, Brown, & Lent, 1991). Again, from outside the field of speech–language pathology, there is excellent evidence of the value of cognitive–behavioral therapy (CBT), an empirically supported treatment approach (Salkovskis, 2002), shown to be effective in treatment for Generalized Anxiety Disorder (Lohr et al., 2003). CBT has also been effective in reducing speech anxiety that not only impacts speakers' comfort in communication but can logically hinder everyday generalized application of other therapeutic strategies for more fluent speech (Cho, Smits, & Telch, 2004; Heimberg, 2002). Desensitization in stuttering treatment is also validated by analogy to other disorders (e.g., Fava et al., 2001). Studies of relapse, for example, demonstrate the effects of anxiety on skilled motor practice (Craig, 1998; Woodman & Hardy, 2003). Self-reports of successfully treated and self-treated adults (Finn, 1996; Yaruss et al., 2002), which tend to emphasize some degree of cognitive–behavioral readjustment, should not be dismissed as forms of evidence when they are congruent with other types of data, as they appear to be. There is a substantial difference between such survey results and testimonials that cannot be reconciled with the larger body of basic and applied science in a domain (e.g., the use of copper bracelets in healing cancer). Vos, Willems, and Houtepen (2004) note the value of patient testimonial in evaluating appropriate treatment options for conditions which at this point in time are “medically inexplicable,” as stuttering would seem to be. Finally, there is a very large literature on the effectiveness of operant/behavioral treatments (some of which is summarized in the 28/3 series), which I am neither explicitly nor implicitly trivializing. I simply believe that data derived from such treatment approaches do not represent the only “decent” evidence out there. Furthermore, I believe that some existing treatment effectiveness data could benefit from better integration with evolving data and theories regarding the nature and etiology of stuttering, as I will discuss further below.

6. What will affect the “shape” of future evidence?

It seems reasonable to suggest that at least some of the information being gathered in “basic research” on stuttering will have to be integrated into the design and interpretation of efficacy research in fluency disorders. For example, most researchers now agree that approximately 80% of children who begin to stutter will spontaneously remit from symptoms, primarily within a window that is between one and two years post-onset (Curlee & Yairi, 1997). This relatively recent set of facts “ups the ante” for demonstrations of clinical effectiveness in preschool stuttering therapy because it leaves open the possibility that even very efficacious therapies look to be so because of sampling error. It is probable that we are looking for a small effect at play among the 20% of children who are not expected to improve simply through the “tincture of time.” As such, failure to balance clinical populations varying in inherent risk for chronic stuttering may produce outcomes that unduly reflect either spontaneous recovery or speeding of the natural course of spontaneous recovery. Fixing a problem earlier than it might have resolved on its own, even if it is quite dramatically linked to treatment timing, is not trivial but is not the same thing as efficacy

281 in treatment of chronic stuttering. Specifically to the point, in preschoolers, even so-called
282 treatment control groups, such as those reported by Harris, Onslow, Packman, Harrison, and
283 Menzies (2002) show a tendency for both untreated and treated children to show similar
284 improvement curves, although the treated children showed more improvement during the
285 treatment period. Thus, a primary challenge for all interventions, particularly those involv-
286 ing preschool children who stutter, is to show that they are demonstrably better than delayed
287 therapy or no therapy at all, something that would be particularly difficult for small scale
288 studies to prove or disprove. To date, to this writer's knowledge, no large scale published
289 studies have contrasted multiple types of intervention for early childhood stuttering, a crit-
290 ical step if we are to rule out what appears to be the case in contrasting legitimate forms
291 of psychotherapy, that is, that most such psychotherapies are quite effective (Wampold et
292 al., 1997). As we now gather information regarding predictors of childhood persistence and
293 recovery (see Yairi & Ambrose, 2004), all treatment studies in children will need to address
294 and report these patient variables. Likewise, it is rare to see information about adult clients
295 that includes potential outcome-influencing data, such as concomitant problems (Watson et
296 al., 1994), and the degree to which the perceived handicap of stuttering (Yaruss, 1998) may
297 impact broad and lasting generalization of skills. This is, of course, not simply a problem
298 in stuttering or speech–language pathology. There has been a recent cross-disciplinary sea
299 change (in medicine, allied health, etc.) to include more patient-centered outcome mea-
300 sures and a broader model of impairment (Swinkles et al., 2002) in evaluating therapy
301 effectiveness.

302 7. Do the therapies themselves matter?

303 For some readers, this will seem like a foolish question, but it shouldn't be if we examine
304 the process of evaluating therapy effectiveness in other fields. To date, we simply lack
305 sufficient numbers of comparison studies to perform the required meta-analyses in fluency
306 therapy, but in psychotherapy, specific techniques have been found to account for no more
307 than 15% of variance in therapy outcomes (Chwalisz, 2001; Lambert & Barley, 2001). This
308 makes behavior change therapy, like that used in psychology or stuttering, very different
309 from the types of interventions typically studied in evidence-based medicine. It is not
310 clear that the beneficial effects of drugs require any interpersonal skills on the part of
311 those who prescribe or dispense them. A surgeon's technical prowess would seem to be
312 much more important than other aspects of his or her involvement with the patient. In
313 such fields, we might say that interpersonal skills don't matter much in one's treatment
314 outcome—but what about the interpersonal talents of one's speech–language pathologist,
315 teacher, or psychologist? Given a high degree of evidence that specific therapies cannot in
316 fact be favored over one another in the treatment of certain disorders, it has already been
317 recommended that clients should seek out specific therapists, rather than specific therapies
318 (Ahn & Wampold, 2001). The notion that all therapies appear to produce winners doesn't
319 negate the importance of asking and trying to answer this question (Wampold et al., 1997).
320 Taken to extremes, there seem to be two diametrically opposed views of the value of the
321 therapy approach itself in achieving outcomes. In the first, exemplified by the Ahn and
322 Wampold (2001) recommendation, the most active component of some treatments is the

therapist. This notion may remind some of detailed observations made by Van Riper (1973) regarding the potent roles of clinical suggestion and persuasion in promoting historically popular approaches to stuttering therapy, and is certainly one basis for the placebo effect across health-related disciplines (e.g., Benson & Epstein, 1975).

In the opposing view, assuming that the therapist understands the precepts of the treatment, all treatments are the treatment: when the treatment isn't working well, it has to be the therapist's fidelity in applying the approach; he or she may need more guidance or experience. The approach itself cannot be at fault if it has been experimentally validated somehow. In fact, this is an observation made in the recent issue of *JFD* (Onslow, 2003), when variations in the rate of clinical progress are to be considered. Between such extremes, there is of course the thorny problem that not all treatments work for all individuals, something medicine knows well (e.g., paradoxical (opposite) response to an intervention or lack of response to medication). There is also, in any literature, a published tendency to "blame" patient or family compliance for lack of success in therapy, a problem sometimes disguised in the completion or drop-out rate.

8. Can treatments arise in the absence of theory?

Theory is basic to any discipline, even when we do not directly appeal to it in framing interventions or evaluating them. Sailor and Stowe (2003) note that the age-old opposing philosophies of positivism (empiricism) and constructivism (subjectivism) have not made "good bedfellows" in the behavioral sciences, and thus, sometimes underlying theories are not overtly addressed in framing social policy.

Can effective therapy arise in the absence of well-articulated theory that addresses the nature and etiology of stuttering? Asking this question is akin to asking "should and why does this therapy work?" in addition to "does this therapy work? In fact, specification of causal mechanism or mechanisms of action is a component of Phase I studies aimed at approving new pharmaceuticals (www.fda.gov). While there are those who would argue that some approaches to treatment render theory unnecessary (after Skinner, 1950), there would appear to be tangible consequences if a treatment does not appear consistent with practitioners' views on how behavior change is achieved. First, there is likely to be lack of "buy in." Clinicians seem to have more of an affinity for programs whose mechanisms they understand. In speech pathology, this may be a factor in the declining popularity and/or sole use of operant-based approaches in treating stuttering, which have been less than vocal in linking themselves to a theory of stuttering, especially recent biological models. The current tempering of enthusiasm for using or only using operant approaches in treating stuttering has been seen earlier, in the decline of popularity of similar programs in treating language problems in children (Kamhi, 1999).

Lipsey and Cordray (2000) summarize recent concerns from a number of fields that are currently evaluating EBP by referring to "grounded" or "program" theory. Many interventions may share characteristics that lead to successful outcomes (both active or non-specific factors; Lohr et al., 2003) and some components may mediate outcomes in ways not anticipated by program framers. Thus, it is critical to explain what program outcomes are or are not actually produced by unique program actions—in other words, determining the

causal mechanisms through which the program produces the outcomes of interest. Lohr et al. (2003) express concern that there is a tendency to replace analytical efficacy studies that test and examine putative causative agents of change with studies of comparative efficacy. Treatment outcome studies also face difficulty in equating any number of non-specific but potentially active outcome factors such as degree of attention, the personality and expertise of the clinician as well as his or her allegiance to the intervention being employed.

Thus, using the experimental method to validate interventions is of course extremely valuable, but has its limitations. As Lipsey and Cordray (2000) note, speaking about educational interventions, using the experimental method to ask whether an intervention is effective only provides a “yes” or “no” answer. In today’s social climate, because we wish to optimize correct answers and improve on less successful attempts, pragmatic assessment of efficacy “puts a premium on *why* (italics added) an intervention is successful or ineffective . . . in short, the explanation of program effects, or lack thereof, requires some THEORY that can serve as a framework for organizing and interpreting information from both descriptive and experimental components of an outcome evaluation” (p. 358). The inverse, therapy with no apparent theoretical grounding, that attempts to specify precise content or form of intervention, represents a technical approach to behavior change that reduces the role of clinicians (and researchers) as decision-makers and innovators (Evans, 1996).

Thus, model building is important in the applied social sciences (Dishion & Patterson, 1999). Models provide a way of conceptualizing the processes that lead to the outcomes of interest in the intervention sciences. Good models are testable, falsifiable and revisable. They can be improved by adding or changing theoretical constructs or reconceptualizing relationships among model components. Eifert (1996) contends that, “a theory-driven hypothesis-testing approach to conducting therapy that is both flexible and patient-focused, is critical for the long-term survival and prosperity of behavior therapy in an era of managed health care.” Lohr et al. (2003) put it more concisely: we must “do the right clinical things for the right theoretical reasons.”

Lack of an underlying theory for a therapy approach also leads to an inability to refine initial proposals to be more efficacious or can lead to “reinventing the wheel rather than re-applying it” (Michie & Abraham, 2004). In other words, if we understand what parts of a therapy are achieving its desired goals, we can “fine-tune” the therapy to maximize “facilitating” components while reducing apparently non-effectual components that do not seem to exert much effect on the outcome. Unfortunately, in some cases, the cost of a disconnect between a therapy and its theory is a spate of initial successes that don’t hold up under scrutiny because post hoc explanation of a potential relationship between a therapy and its mechanism of action turned out to be inaccurate (e.g., the embarrassing case of eye movement desensitization therapy (EMDR) in psychology; Follette & Beitz, 2003).

One way to choose among therapy approaches is to relate them theoretically and empirically to beliefs about what we think stuttering is, etiologically. Increasing amounts of basic research suggest that stuttering reflects underlying neurological disfunction, and interacts with speech and language planning demand (see Anderson & Conture, 2004; Buchel & Sommer, 2004; Conture, Zackheim, Anderson, & Pellowski, 2004; Cuadrado & Weber-Fox, 2003; Forster & Webster, 1991, 2001; Kleinow & Smith, 2000; Smith & Kelly, 1997 for representative but not exhaustive examples). As stuttering becomes chronic, there is also evidence that it is shaped by experience. But little empirical data exist to suggest that

410 stuttering is “learned” per se, and thus, it is not clear why operant approaches (the primary
411 ones favored by authors in *JFD* 28/3 with the exception of Langevin & Kully, 2003) should
412 work, for instance. Moreover, most of the therapy presentations and efficacy data reports for
413 operant procedures do not attempt to ground the therapy in theoretical or evidence-based
414 models of the underlying disorder. Years ago, Andrews et al. (1983) noted that:

415 The purely operant model of stuttering . . . by implication involves some qualitative
416 differences in the child-rearing practices of stutterers’ parents . . . behavior that has
417 been looked for and not found. The model . . . (also) predict(s) that stuttering behaves
418 as a lawful operant. Even the evidence for this last proposition is somewhat equivocal
419 . . . hedonically ambiguous stimuli such as verbal stimuli have been employed (in
420 studies, and) . . . Clearly the data on the effect of punishment are unsatisfactory . . .
421 (p. 237)

422 Attanasio (1999) suggests that operant programs are atheoretical by nature and do not
423 require grounding in models or theories, but others would vociferously disagree (Eifert,
424 1996; Lohr et al., 2003; Rosen & Davison, 2003; Salkovskis, 2002). In fact, there is real
425 risk that treatments not based on any articulated theory may well “paint themselves into
426 corners” when challenged by the need for refinement.

427 While questions in evidence-based practice can be phrased as *Yes/No* or *More/Less* (Does
428 this treatment work? If so, which treatment is better than another?), there is an important
429 role for questions that ask, “How” and “Why.” We need to know *why* therapies succeed, so
430 “lessons can be learned and improvements can be made . . . program stakeholders . . . want
431 an explanation for the results . . .” (Lipsey & Cordray, 2000), not just the results themselves.
432 Across other fields, there is growing concern that evidence-based practice should be super-
433 imposed on a coherent body of knowledge of pathophysiology and clinical observation,
434 not the other way around; as van Weel and Knottnerus (1999) note, we need to test “key
435 elements of the intervention as identified from a conceptual basis” (p. 918).

436 Thus, reasonable questions in selecting a preferred therapy approach are: How do we
437 think therapy works, when it does seem to, and why wouldn’t it be expected to work when
438 it doesn’t seem to? As Kamhi noted, discussing approaches to language remediation in
439 children: “One reason not to use a . . . treatment approach is that we do not understand what
440 it is about the treatment approach that makes it work” (Kamhi, 1999, p. 96). Onslow (2003)
441 chastises clinicians for employing “assertion-based practice.” However, it is possible that an
442 educational emphasis on the growing body of basic research on the nature of stuttering has
443 led clinicians to apply an educationally enculturated form of clinical bias in dealing with
444 accumulated evidence of neurophysiological, physiological, neurolinguistic and cognitive
445 factors in the disorder. Because so much research into developmental disorders now empha-
446 sizes “constitutional” factors in their etiology, I do not find it surprising that surveys show
447 a recent slight downturn in interest in the use of purely operant programs (Crichton-Smith,
448 Wright, & Stackhouse, 2003; Kuhr, 1994) by clinicians. The “traditional” approaches tend
449 to emphasize components that include fluency shaping, stuttering modification, desensiti-
450 zation and establishment of self-efficacy and locus of control (LOC), each of which has
451 therapy goals grounded in part in neurophysiological research. Thus, it is relatively easier
452 to understand WHY these approaches should work, and it is merely a next step to further
453 document their application to this population. There are also, contrary to the assertion of

454 some, already data to show that they work, as noted earlier. The fact that such components
455 appear to have such wide-spread application (Cooper & Cooper, 1996) and that adults report
456 relatively good success with them (Yaruss et al., 2002) is distinctly at odds with claims that
457 they lack efficacy. The data are consistent to some degree with a claim that their current
458 profile of efficacy data may not be as compelling when contrasted with a small but intense
459 body of data on a small number of fairly singular approaches.

460 However, supportive data are there if one chooses to look for them, both in stuttering and
461 related fields. For example, attitude change does in fact bear on the risk of relapse (Andrews
462 & Craig, 1988; Craig, 1998). In social phobia, a meta-analysis of exposure therapy has
463 confirmed its efficacy in desensitizing patients (Fava et al., 2001). This is not “assertion-
464 based practice.” To believe that clinicians prefer to utilize non-efficacious techniques even
465 when they do not work for their clients requires a more misanthropic view of our professional
466 peers than seems warranted, and threatens the type of collegial dialogue that best fosters
467 cooperative design, collection and analysis of therapy outcome studies.

468 **9. Will we need more than one theory? Will we need more than one therapy** 469 **approach?**

470 There is already some consensus that the theory of what causes stuttering is not sufficient
471 to explain what stuttering becomes. In health professions that emphasize behavioral change,
472 “no single theory dominates . . . the most useful approach is to combine concepts from
473 more than one theory to address a problem, and to bring these together in a comprehensive
474 plan. . . . no model or theory will get it right all the time, and in practice, often a single
475 theory (approach) explains only a small amount of the variance in targeted behaviors”
476 (Cockburn, 2004). As noted earlier, there is evidence that speech–language pathologists
477 already believe this, and use a healthy mix of therapy approaches, including fluency shaping,
478 attitude modification and operant. The concept that more than one fluency therapy may work
479 equally well already appears documented for older children who stutter (Hancock et al.,
480 1998), and future research may wish not only to enlarge the approaches that are compared,
481 but seek predictors of which clients profit best from which therapies.

482 **10. Applying evidence to theory: a case in point**

483 A number of the contributors to the JFD symposium cite the Lidcombe program (Onslow
484 et al., 2003) as one that has better efficacy data than popular alternatives for the treatment of
485 stuttering in very young children. I would like to illustrate some of my concerns about the
486 application of EBP by applying some of the concepts in this article to this value judgment.

487 First, as noted, Lidcombe is grounded as an operant program. As mentioned earlier,
488 operant procedures have fallen out of favor in stuttering, as well as across many fields of
489 human behavioral intervention. Indeed, such procedures appear to be primarily used with
490 profoundly impaired children in the field of communication disorders and related educa-
491 tional and behavioral domains. In language, there is consensus that operant programs did
492 not meet the clinical challenge, even in Pervasive Developmental Disorder, where general-

493 ization of results has not met expectations, even when trial by trial documentation appears
494 promising (Delprato, 2001).

495 Moreover, in other fields, there is a trend to reconceptualize some older operant terminol-
496 ogy in order to better explain effects of certain interventions. In particular, I find more recent,
497 non-operant interpretations of concepts such as “reward” and “reinforcement,” which I will
498 enlarge on below. However, I find few parallel conceptualizations of extinction, a concept
499 clearly at play in Lidcombe, where “correction” (earlier termed “punishment”) is the pre-
500 sumed agent of stuttering reduction. Negative consequence of behavior in most current
501 behavioral therapies implies some level of relative patient choice of action and definition of
502 reward that can impact action, such as a broad group of maladaptive and habitual behaviors.
503 Bandura and Locke (2003) reflect that, “If knowledge and skills could be acquired only by
504 negative response feedback, human development would be greatly retarded . . .” Finally, as
505 I note in more detail below, punishment of the stuttered moment (or time-out from positive
506 reinforcement) does not easily map onto our understanding of the mechanisms that produce
507 or prevent children’s stuttered events.

508 Typically, a therapeutic program is most attractive to discerning, experienced clinicians if
509 they extract principles that permit them to understand, adapt and trouble-shoot the program.
510 In other words, if I or any other clinician knows why a component is integral to the program,
511 if the child does not respond as predicted, we can fine-tune using similar principles. Right
512 now, in operant programs such as Lidcombe, the contingency schedule appears to be the only
513 manipulable component, which limits the degree to which one can fine-tune or “dismantle”
514 (Rosen & Davidson, 2003) the program to investigate its mechanisms of action or refine
515 its level of effectiveness for a particular client. Simply put, when we teach a skill, we can
516 think of other ways to convey the information or skills we seek to instill. When we change
517 behavior through contingencies, there is no teaching, only learning, of a sort. When learning
518 does not occur, therapeutic course adjustments or options appear relatively restricted.

519 Still another way to endorse the outcomes of a program is to be able to interpret the
520 program from other theoretical perspectives, and there is actually a wealth of ways to view
521 a purportedly operant program such as Lidcombe. I will take this opportunity to speculate
522 on only a few. For example, it is relatively easy to hypothesize that a potential mechanism of
523 action in Lidcombe is manipulation of linguistic demand. Parents must design activities that
524 progressively create a proportion of fluent and stuttered utterances and it is possible that they
525 help children shape less ambitious language attempts as they gain fluency. Intake/outcome
526 language measures for Lidcombe children (Bonelli, Dixon, Bernstein Ratner, & Onslow,
527 2000) suggest that spontaneous language tendencies of children who have completed the
528 program have been modified to be more age-appropriate than they were when the child was
529 first referred for intervention. This may have occurred if children associated correction of
530 dysfluent utterances with parental disapproval of relatively long or complicated utterances.
531 This does not mean that children ‘lost language’ skills as a result of the reinforcement
532 schedule, but rather that they may have been encouraged to keep their language production
533 attempts more comfortably within their fluency abilities (Bonelli et al., 2000). Such data are
534 consistent with recent data reported by Watkins and Johnson (2004) that show spontaneous
535 recovery to be linked to slowed pace of expressive language growth post-onset of stuttering
536 symptoms, while persistence was linked to more developmentally advanced spontaneous
537 language profiles.

538 It is *not* just a matter of semantics to ask if one can describe an operant program
539 such as Lidcombe in alternative terms. Casting therapy components in a new light may
540 allow us to test mechanisms of action. One can ask, for example, if Lidcombe “binds
541 parental anxiety” (Bernstein Ratner & Guitar, *in press*) by making parents active partic-
542 ipants in their children’s therapy. Reductions in parental anxiety levels have been shown
543 to improve some child therapy outcomes (Cobham, Dadds, & Spence, 1998). Similarly,
544 because it acknowledges stuttering openly, does Lidcombe reduce *child* anxiety? This
545 might predict that any structured therapy involving a high degree of parental involve-
546 ment would yield positive outcomes, despite varying specific goals, a notion recently
547 supported by data from Cook, Millard, and Nicholas (2004). Does Lidcombe wind up
548 reinforcing other behaviors that influence the frequency of stuttering? (This is likely to be
549 true of many if not most stuttering therapies.) One can even venture into less comfortable
550 questions, such as, “does it simply drive some behaviors “underground”? (create covert
551 stuttering)?

552 I ask such questions not to diminish the available data, which are truly impressive, but
553 because to a certain degree, the data don’t make a lot of sense when presented in a theoretical
554 and scientific vacuum. For example, one of the most troubling “disconnects” in understand-
555 ing the success of operant techniques with preschool stutterers is the finding that in adults,
556 such therapies appear to work by calling upon extant fluency skills obtained through prior
557 types of therapy (James, Ricciardelli, Rogers, & Hunter, 1989). Since Lidcombe toddlers
558 have not ever been instructed in fluency-enhancing techniques, it is not clear what extant
559 resources the negative feedback spurs into action.

560 Further, because the program is intrinsically linked to reinforcement of fluent speech, it is
561 not trivial to note that the mechanisms by which verbal praise maximizes desired outcomes
562 in children is still poorly understood (Henderlong & Lepper, 2002), but currently, praise
563 is rarely viewed in simple operant terms. Research suggests that the effects of praise vary
564 considerably depending upon the content of the message, the context in which it is delivered,
565 and the way in which it is interpreted by the child. Praise tends to “bond” children to an
566 activity insofar as it increases a child’s motivation to continue participation in an activity. It
567 also is clearly associated with increases in self-efficacy, which maximizes locus of control
568 and coping behavior. All have been linked to fluency improvement in other models of
569 therapeutic intervention.

570 Again, rephrasing the mechanism for change is more than mere semantics. Crucial to
571 questions about the relatively higher success rate of Lidcombe with younger children, it is
572 notable that preschool children do not clearly distinguish between implications of praise
573 and corrections that target effort versus ability, while older children may make more com-
574 plicated assumptions about what is being reinforced and penalized in feedback from adults
575 (Henderlong & Lepper, 2002). Because Henderlong and Lepper review a large literature
576 linking the presumed sincerity and affective value of praise to its effects, it is also inter-
577 esting to speculate that Lidcombe might actually be much less effective if administered
578 by clinicians, rather than parents. Such a notion places a new spin on the therapy design,
579 which seems to have originally involved parents as a way to achieve efficiency in session
580 programming (using parents as therapist surrogates in an environment where therapists
581 are of limited availability). We may be able to predict more and less successful mixes
582 of target populations and therapy providers, or adjust program components by age if we

583 evaluate these potential agents of behavioral change in designing and monitoring program
584 outcomes.

585 If a program such as Lidcombe doesn't recruit some of the mechanisms I have discussed,
586 its efficacious basis is unclear (although I am truly impressed by the efficacy data) since
587 critically, as previously noted, operant programs in adults seem to work best when clients
588 already have techniques to apply to managing fluency (James et al., 1989). I am certainly
589 amenable to trying it with particular children, and using it if it yields results within the
590 promised time frame (a limited number of sessions). But I am less than sanguine, at this
591 state in our knowledge, about setting up this or any other intervention as the paragon of
592 therapeutic virtue to which all other programs must be compared unfavorably.

593 As with many other fluency therapy programs in print, there are some other questions
594 about Lidcombe that we need to answer: The majority of data points represent unknowns,
595 other than overt fluency behaviors going in and coming out; thus, we don't know risk factors
596 such as family history, concurrent language abilities, the child's emotional and cognitive
597 responses to the stuttering problem, the responses of the parents, etc. The recent report
598 from Harrison, Onslow, and Menzies (2004) is a promising beginning but still quite under-
599 specified. Such concerns reflect earlier discussion about what forms of data are considered
600 relevant in the evidence-gathering process. Additionally, with all that we now know about
601 prognostic indicators (Yairi, Ambrose, Paden, & Throneburg, 1996), we have a paucity of
602 information on the developmental profiles of Lidcombe-treated children (and most other
603 young children enrolled in therapy protocols).

604 In sum, I wish to be very clear in my appreciation of the Lidcombe program and its
605 efficacy data. The program's authors are to be respected and commended for providing
606 us with an evidence-based treatment option for young children, as well as for heightening
607 our level of appreciation for rigorous treatment outcomes research. I do believe that the
608 program works for the majority of children who have been enrolled thus far. But I do not
609 understand why it works, or why it should work, and would appreciate answers to these
610 questions before reading more reports telling me that it does.

611 11. What is the alternative to EBP?

612 If there is an alternative to EBP, it is certainly not the endorsement of non-efficacious
613 treatments. However, this is often how the debate is framed, particularly in the many disci-
614 plines now entering the EBP fray (Levant, 2004). In psychology, there is already concern
615 that, "controversy seems to stem from the attempts of some clinical scientists to dominate
616 the discourse on acceptable practice and impose very narrow views of both science and
617 practice" on the field. To some, this might not seem like such a bad thing, but one should be
618 cautious. In psychology, outcomes of Task Forces assigned to evaluate EBPs tended to over
619 represent those approaches that involved highly specified, manual-guided treatments that
620 permit few clinician-selected adjustments. Conflicts among practitioners regarding "best
621 practices" also provided grist for managed care and insurance companies to restrict cover-
622 age for those seeking services, on the grounds that particular therapies had less supportive
623 data or conflicting data (Levant, 2004). This was not the intent of the movement, but it was
624 its outcome.

12. How do we proceed best in this climate?

A first step might be by cooperating to gather more data. And diversity of approaches is probably a good thing, until proven otherwise. Everyone benefits if there is more than one way to “skin a cat”. A dynamic, ever-developing discipline should not be reduced to a single manual of practice, because clients inherently differ in their symptoms and needs. Thus, we need flexibility in planning as well as conducting therapy, rather than strict adherence to a “one-size-fits” all model.

Second, it would appear to be prudent not to start turf wars or philosophical disputes. While interesting (or frustrating) to academics and practitioners alike, a field that seems to be populated by combatants rather than colleagues is likely to suffer casualties in the bid for research funding, since reviewers are more likely to provide support for treatments that appear to be supported by consensus.

It would appear to be imperative to obtain more empirical data regarding the effectiveness of the practices actually employed in the field. The components of stuttering modification therapy are particularly under-researched, which is somewhat surprising given the personal therapy histories of some of the major researcher/clinicians in our field. In stuttering, contrary to some views of the situation, we don’t just have “authority-based practice” (Onslow, 2003). Many of our most visible clinician-researchers who are identified with certain therapy approaches are themselves “walking graduates” of the program orientations that they endorse—that makes us somewhat unique, and I tend as a fellow professional to give credence to therapies that by report helped to give my colleagues fluency. However, more systematic data are clearly needed.

As we do all this, we should derive comfort from the fact that some of our efficacy data come to us by close analogy from psychology and other fields (e.g., CBT). The success of CBT in treating a wide variety of behavioral disorders, including communicative anxiety, should be viewed as endorsement of the wisdom that they can be used to combat speaking fears and anxieties in stuttering. For example, we don’t need to test aspirin in all conditions that produce fever, if we know it reduces the primary symptom.

13. How do we assure clinicians “get” the evidence?

Just as our discipline is experiencing the EBP movement later than some other fields, there are some questions we have not yet begun to ask ourselves that will be very important. I do not mean to scare anyone, but the gap between the evidence and actual practice is a big problem for the medical community these days—just because you produce evidence doesn’t mean that it gets used (Davis, Ciurea, Flanagan, & Perrier, 2004). Although no formal studies have been made of the degree to which practicing SLPs keep up with, as well as employ, newer or better supported treatment strategies, there is some awareness practitioners in many fields are unlikely to read their professional journals, and are more likely to “Google” the problem (Nail-Chiwetalu & Bernstein Ratner, 2003, submitted) than consult the appropriate primary literature.

It seems safe to suggest that we need a sea-change in SLPs’ valuation of the need to keep up with the published literature. The new continuing education requirement for the

666 CCC's alone won't do it; journal self-study is one of the least pursued options, and peer
667 review of workshop contents is more or less a formality. In medicine, there is depressing
668 evidence that continuing medical education has not changed physicians' behaviors (Davis
669 et al., 2004). So, a reasonable question to ask is: How do we convince practitioners to gather
670 and use evidence in support of their practice?

671 One way to increase use of "newly evidenced" therapies is clearly philosophical "buy
672 in." In addition to what we see in our own literature and that in psychology, medicine faces
673 similar hurdles. For example, as an extreme example, many physicians are not persuaded
674 by studies of "distant healing," regardless of findings and quality of research design (Astin,
675 Harkness, & Ernst, 2000), because they cannot reconcile such findings with their existing
676 belief systems. We do have some philosophical differences among each other in terms of
677 what we believe stuttering is, as well as how theories and the evidence that supports them
678 should integrate with therapeutic approaches.

679 In such cases, simply "dumping the data" is not going to do the job, as Ingham (2003) and
680 Bothe (2003) observe in *JFD* 28/3. This may be especially problematic in the USA, where
681 training in fluency treatment is much more heterogeneous than in Australia, for example
682 (Attanasio, Onslow, & Menzies, 1996), and requires buy-in of diversely educated professors
683 and clinical supervisors.

684 **14. Caution: there is no finish line ahead!**

685 EBP is not a race, where the earliest finisher ends the discussion, particularly when
686 the evidence base is very immature. Levels of data throughout the profession (e.g., the
687 recent Cochrane report for speech/language therapy in children, Law, Garrett, & Nye,
688 2003) are currently low and need to be thoughtfully built, not just on data, but on sci-
689 entifically and theoretically sound principles. Psychology had an interesting recent "run
690 in" with this problem. As noted earlier, recent analyses of eye movement desensitiza-
691 tion and reprogramming (EMDR) therapy have detected a troubling flaw: recent research
692 clearly indicates that in this hugely "successful" treatment, the putative underlying con-
693 struct upon which the therapy was built is completely dissociated with the outcome
694 (Follette & Beitz, 2003; Wampold, 2001), and that outcomes may reflect a complicated
695 mix of non-specific factors. Thus, Chwalisz (2001) warns that an over-interpretation of
696 EBT might result in the endorsement of dubious treatments that produce superficially
697 compelling results, but do not utilize conceptually and empirically sound principles of
698 change.

699 Because the level of evidence in our field is relatively low when compared to medicine
700 or even psychology, it seems less than appropriate to chastise those who use alternative
701 approaches "early" in the game. It is my personal impression that it has convention-
702 ally been easier for "behavioral" interventions to amass efficacy data in many fields.
703 This concern is not unique to stuttering: in medicine, some approaches that take too
704 narrow a view of the problem space result in "overcontrolled" studies. These in turn
705 can lead to "hyperclaiming" (Rosenthal, 1994), or claims of a research team that a pro-
706 posed intervention is likely to achieve goals that it will not when applied more broadly
707 (or that it is the single acceptable approach to a problem). Faster accumulation of out-

708 come data can obviously lead some programs to document given levels of efficacy be-
709 fore others. This is particularly true when the current climate in EBP is partnered with
710 a proportion of current stuttering research that emerges from national health service-
711 sponsored research programs, or a professional environment in which most practitioners
712 are trained in fairly uniform methods, such as Australia (Attanasio et al., 1996). Above
713 all, at this stage in our development we must not confuse the concept “currently with-
714 out substantial evidence” with “without substantial value” (Kerridge, Lowe, & Henry,
715 1998).

716 American researchers and clinicians, the apparent object of concern by authors of 28/3,
717 face a number of interesting obstacles to rapid treatment efficacy research production, in-
718 cluding more diverse training backgrounds (contrary to the opinions of Attanasio et al.
719 (1996), not all represent the Iowa school via tangible training pedigrees), greater geograph-
720 ical distance among clinical programs, more diversely trained local practitioners to work
721 with and internal competition for public health service grant support. Inequalities in the
722 ease and pace with which therapy outcomes research can reach publication can be particu-
723 larly problematic, because as Wampold et al. (1997) note, in outcomes research, “to receive
724 a prize that is meaningful, the competitors must have a level playing field.” This is also
725 troublesome if one conceptualizes (wrongly) EBT to be some type of race, rather than an
726 ongoing dialogue among researchers and clinicians.

727 It is important to realize that documenting that a program doesn’t work (doesn’t pro-
728 duce results) is different from having no evidence that it does work. “In EBP, there will
729 always be interventions for which no evidence is (yet) available, but that is no reason to
730 withhold the intervention. Where research provides unequivocal evidence of the absence
731 of effects, the situation is much clearer” (van Weel & Knottnerus, 1999). Therefore, it is
732 not yet appropriate to ask why clinicians use therapies having “less evidence” of effec-
733 tiveness than proposed alternatives, as the authors of contributions to the *JFD* symposium
734 suggest.

735 It will also be important to evaluate (or debunk) each opposing approach to practice on
736 its own terms, rather than only as a synchronic comparison of recently compiled efficacy
737 data, since more than one approach is likely to eventually be validated, as noted earlier.
738 Few competing approaches to a problem emerge and achieve equivalent levels of empirical
739 support simultaneously, especially in a sea change of practice guidelines such as the new
740 orientation toward EBP. Immediately below, I will argue that one inevitable outcome to the
741 immediate and wholesale adoption of an EBP approach to treatment selection will be the
742 uneven pace of comparably gathered efficacy data collection across treatment approaches,
743 and across outcome criteria selected for analysis.

744 15. “Branding” of therapy approaches

745 Lidcombe joins a few other recently developed approaches for treatment of commu-
746 nication disorder that are either already proprietary or “branded” through allegiance to a
747 therapist training regimen—FastForWord is a possible parallel for the treatment of SLI,
748 as is Lee Silverman Voice Therapy. In evaluating comparable therapies that are “mar-
749 keted” through training or franchising in other fields, Beutler (2000a, 2000b) and Rosen and

750 Davison (2003) express concern that we should not confuse empirically supported treat-
751 ments with *empirically-supported principles of change*. An important result of a shift in
752 focus to principles or mechanisms of change would be seen in training of future clinicians:
753 since ‘cookbooks’ for all potential treatment regimens cannot be provided in advance, clin-
754 icians in training would need to “learn principles of change and the range of their applica-
755 tions” (Rosen & Davidson, 2003). As noted earlier, among the skills that Rosen & Davidson
756 would teach is how to “dismantle” therapies to identify their process mechanisms. Only
757 by teaching students such concepts, rather than therapies per se do we allow the field to
758 advance in a scientific manner, because “principles of behavior change, after all, cannot be
759 trademarked, for they belong to science.”

760 A final concern that is likely to be mitigated by the passage of time is that Lidcombe
761 in particular can show published data virtually exclusively from one research team (even
762 “replications” in Great Britain involved a high degree of participation and authorship by
763 the Sydney team). As Lohr et al. (2003) note, a treatment seeking to be considered “well-
764 established” must additionally supported by research conducted by two independent sets
765 of researchers. While the numbers of published articles on Lidcombe are currently large
766 and impressive, they are still representative of a class of findings that Andrews et al. (1983)
767 termed Class C (findings replicated by a single research center with no conflicting results).
768 Without wide-spread open consideration of the possible effective approaches to stutter-
769 ing treatment, legitimate treatment comparison studies will be impossible because of the
770 well-known allegiance effect—clinicians with truly vested interests in the outcome of par-
771 ticular approaches have remarkable effects on outcomes. Some of the opinions expressed
772 about alternative therapy approaches in 28/3 would seem to be potential markers of exper-
773 imenter bias in testing the very question under consideration (Wampold, Ahn, & Coleman,
774 2001).

775 16. Some concluding thoughts

776 Bertrand Russell once observed that “The most savage controversies are those about
777 matters as to which there is no good evidence either way.” This might be an apt way of
778 describing what we know about stuttering therapy at this time when all is said, done and
779 weighed. Debate is fun for some of us, but:

780 To practitioners, the continued jousting among researchers may seem tedious. When
781 scientists debate clinical practice issues, there is often no middle ground. Scientists
782 need to convince clinicians that their view is the only one that has both theoretical
783 coherence and empirical evidence to support it. This often leads to the use of emo-
784 tionally charged rhetoric and exaggerated claims of the dangers of using the wrong
785 treatment approach and the benefits of using the right one. (Kamhi, 1999; p. 95)

786 We will probably not be able to change some of the inevitable differences in how scientists
787 and clinicians view the major issues in evidence-based practice. What we need to do is find
788 some basic concepts to agree on. Among them might first be an agreement that we need to
789 keep working on the problem. It might be good to work together, since sample sizes required
790 to show program component functionality may be enormous (Harrison et al., 2004). Second,

791 it seems clear that we—scientific clinicians and clinical scientists alike—should keep better
792 data. Further, we need to increase the rate at which successful therapists publish the data.
793 Throughout all this, we must remember to report individual differences even as we evaluate
794 the group data. Ellis Weismer (1991) long ago advised advocates of opposing theoretical
795 models of child language remediation to turn their attention to the variables that might predict
796 which programs are relatively more efficient in producing specific desired outcomes, which
797 may differ from child to child, rather than defend the premise that one type of approach
798 was globally superior to any others. This is sound advice for the field of stuttering as
799 well.

800 We need to join together to increase interest in therapy trials, both from researchers' and
801 funders' perspectives. We need to investigate how we can increase partnering between the
802 actual therapy providers and the publishing "researchers," who are not typically working
803 together in the USA, unlike the case in Australia, for example. I believe that we need to show
804 respect for the values of current providers (e.g., minimizing remarks suggesting that some
805 seem to be endorsing 'an aluminum standard'; Ingham, 2003). These service providers, I
806 believe, want the best for their clients and employ therapy approaches guided by this set of
807 values. And I think we should let the data *and theory* guide further research and therapy but
808 not dictate it, *yet*.

809 A real concern raised by Sailor and Stowe (2003) is that of viewing the recent popularity
810 of EBP, especially as defined by the RCT, as an endgame for current policy. They evaluate
811 the current status of educational research in ways that some of the authors of JFD 28/3 might
812 appreciate: too often "exploratory or descriptive, because the literature is too thin or even
813 nonexistent with regard to many of the issues being investigated . . . still in its adolescence
814 compared with the fully matured science of medicine." They conclude, "It needs time and
815 the opportunity to grow." The same is true in our field.

816 Suffice it to say, now is the time for conciliation and progress, not platforming. We
817 need to stay, as much as possible, collegial. In a small field trying to enter the fray of
818 EBP, some of the arguments raised in JFD 28/3 threaten to weaken rather than strengthen
819 the field. In psychology, where the EBP debate is rampant, advocates of various positions
820 are throwing large bodies of evidence at one another to argue margins of relative efficacy.
821 I personally fear a disciplinary debate where one side claims the other brings virtually
822 nothing to the table (e.g., one forum contributor advised readers to "quit reading" after
823 consulting the preface to ASHA's *Guidelines for Stuttering Treatment*). I also fear that our
824 field may well inherit a wasteland if it polarizes attempts to improve the evidence basis of
825 practice for the field rather than seek to define a set of recommended practices at this point in
826 time.

827 In this writer's opinion, the tone of some past debates is hardly likely to win over
828 the non-believers (cf., Ingham & Cordes, 1999). Rather than castigate researchers and
829 clinicians who do not universally and exclusively endorse some of the approaches rec-
830 ommended by authors in 28/3, we should be proud of those who do not leap to ad-
831 minister treatments inconsistent with the emerging scientific understanding of the ba-
832 sis of stuttering. To me, it implies that such individuals are engaged in reflective
833 thinking and would, therefore, be receptive to strengthened accounts of the causative
834 mechanisms by which supposedly effective and efficacious programs achieve their
ends.

CONTINUING EDUCATION

835

836

QUESTIONS

- 837 1. What does the author conclude about the evidence-base for “traditional” approaches to
838 stuttering therapy?
- 839 a. They are poorly supported by the available data
840 b. They are more strongly supported than operant options
841 c. They are supported by data from fluency intervention and other allied disciplines,
842 such as clinical psychology
843 d. The evidence base is strong, but the basic research is not consistent with it
- 844 2. The author suggests that future therapy outcomes research needs to address which of the
845 following factors that may influence treatment response?
- 846 a. Spontaneous recovery
847 b. Family history of chronicity
848 c. Associated/comorbid conditions
849 d. Perceived emotional and/or cognitive impact of the stuttering on everyday functioning
850 e. All of the above
- 851 3. The author provides a number of reasons why theory and program dismantling are
852 important in developing and evaluating treatment approaches. Which is NOT one of
853 these reasons?
- 854 a. Therapies that cannot be reconciled with theory have no value and should not be used
855 b. Theory assists us in “fine-tuning” therapy approaches to maximize active components
856 and minimize non-active components
857 c. Theory may assist in encouraging practitioner “buy-in” in adopting new treatments
858 d. Theory may help us distinguish between outcomes that are program-specific as op-
859 posed to clinician-specific
860 e. Theory may help us to adapt programs to specific client needs
- 861 4. Which of the following is NOT one of the author’s concerns in evaluating the evidence-
862 base for a treatment approach?
- 863 a. The degree to which it can be reconciled with existing data regarding the underlying
864 nature of stuttering and characteristics of its development
865 b. Whether or not a treatment approach has been evaluated by multiple, unaffiliated
866 researchers/clinicians
867 c. Whether or not a therapy approach can be dismantled into its active components
868 d. The relative number of studies that have appeared to date documenting the program’s
869 effectiveness when compared to other programs
870 e. All of the above
- 871 5. Which of the following statements best summarizes the author’s reaction to the forum
872 in 28/3?
- 873 a. She fully agrees with it, and offers additional support for the viewpoints expressed
874 in the issue
875 b. She believes that there is an evidence-base for many approaches to stuttering inter-
876 vention beyond those highlighted in the forum

- 877 c. She believes that most current disagreements about appropriate treatment for stuttering
878 will be answered by larger and additional RCTs
- 879 d. She believes that no current therapies can be linked to theories about the nature of
880 stuttering
- 881 e. She believes that no adequate treatment approaches for stuttering will be developed
882 until we have a better understanding of the etiology of the disorder than we do know

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