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Strategy training during inpatient rehabilitation may prevent apathy symptoms after acute stroke

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Abstract

Objective—Apathy, or lack of motivation for goal-directed activities, contributes to reduced engagement in and benefit from rehabilitation, impeding recovery from stroke. We examined the effects of strategy training, a behavioral intervention used to augment usual inpatient rehabilitation, on apathy symptoms over the first 6 months after stroke.

Design—Secondary analysis of randomized controlled trial.

Setting—Acute inpatient rehabilitation.

Participants—Participants with acute stroke who exhibited cognitive impairments (Quick Executive Interview Scores ≤ 3) and were admitted for inpatient rehabilitation were randomized to receive strategy training ($n=15$, one session per day, 5 days per week, in addition to usual inpatient rehabilitation) or reflective listening ($n=15$, same dose).

Methods—Strategy training sessions focused on participant-selected goals and participant-derived strategies to address these goals, using a global strategy training method (Goal-Plan-Do-Check). Reflective listening sessions focused on participant reflections on their rehabilitation goals and experiences, facilitated by open-ended questions and active listening skills (attending, following and responding).

Main Outcome Measurements—Trained raters blinded to group assignment administered the Apathy Evaluation Scale at study admission, 3 and 6 months. Data were analyzed with repeated measures fixed-effects models.

Results—Participants in both groups had similar subsyndromal levels of apathy symptoms at study admission (strategy training, $M=25.79$, $SD=7.62$; reflective listening, $M=25.18$, $SD=4.40$). A significant group by time interaction ($F_{2,28}=3.61$, $p=.040$) indicated that changes in apathy

symptom levels differed between groups over time. The magnitude of group differences in change scores was large ($d=-0.99$, $t_{28}=-2.64$, $p=.013$) at month 3, and moderate to large at month 6 ($d=-0.70$, $t_{28}=-1.86$, $p=.073$).

Conclusion—Strategy training shows promise as an adjunct to usual rehabilitation for maintaining low levels of post-stroke apathy.

Introduction

Apathy is defined as a lack of motivation or interest in goal-directed activities.¹⁻² Apathy is characterized by changes in three dimensions of motivation: (1) diminished goal-directed cognition (lack of interest and value attributed to productivity and socialization), (2) diminished goal-directed behavior (lack of effort, productivity, initiative, or persistence), and (3) diminished emotional responsivity to goal-directed activities (flat affect).²⁻³ Apathy has been observed in a variety of neurological and psychiatric disorders, including stroke, traumatic brain injury, and dementia.⁴⁻⁵

With regard to stroke, apathy is associated with significant functional disability and poor rehabilitation outcomes.⁶⁻⁸ Although estimates vary, apathy may occur in 15–71% of individuals in the acute phase of stroke recovery, with an estimated prevalence of 34–36%.⁹⁻¹⁰ In the acute phase, apathy can be dissociated from other common stroke-related related problems such as cognitive impairments or depression.¹¹ Nonetheless, post-stroke apathy is associated with significantly higher incidence of executive cognitive impairments and depression,^{6,9-10,12} with one systematic review reporting Odds Ratios of 2.90 for executive cognitive impairments, and 2.29 for depression.⁹

Evidence addressing efficacious interventions for post-stroke apathy is limited.¹³ Case reports have examined a range of pharmacotherapy agents, including dopamine agonists (amantadine, bromocriptine), monoamine oxidase inhibitors (selegiline), stimulants (methylphenidate), and non-benzodiazepine hypnotics (zolpidem).¹⁴⁻¹⁸ One quasi-experimental study examining acetylcholinesterase inhibitors (galantamine, donepezil) and a secondary analysis of a randomized controlled trial examining a nootropic agent (nefiracetam) both demonstrated moderate effect sizes for reduction in apathy symptom levels relative to baseline (Cohen's $d=0.63$), and relative to placebo (Cohen's $d=0.71$), respectively.¹⁹⁻²⁰

As for non-pharmacological interventions, there is a dearth of studies addressing post-stroke apathy. Problem solving therapy and behavioral activation have shown promise for treating apathy in other neurological and psychiatric populations.²¹⁻²⁵ Although there are some differences, these programs share many key active ingredients: goal setting and planning, self-evaluation and self-monitoring of behavior, and activity-based training in problem solving skills.²¹ Emphasis is placed on initiation and completion of goal-directed activities. Only one study applied these methods to post-stroke apathy, reporting that problem solving therapy demonstrated some efficacy in the prevention of post-stroke apathy.²⁵

Strategy training is a behavioral intervention that includes each of these key active ingredients.²⁶⁻²⁷ In a previous trial, we demonstrated that strategy training showed promise

for reducing disability and improving executive cognitive functions (i.e., inhibition, cognitive flexibility) among individuals with cognitive impairments after acute stroke.²⁸ Given the reported associations among apathy, disability, and executive cognitive functions in previous studies, we posited that strategy training may also address apathy symptoms after stroke. Our hypothesis was further supported by the noted similarities between strategy training and interventions that have previously shown promise for addressing apathy in other neurological and psychiatric populations (problem solving therapy and behavioral activation programs). Thus, we conducted a secondary analysis of data from our previous trial comparing strategy training and an attention control intervention and examining post-stroke apathy symptoms over the first 6 months after stroke.

Methods

The parent study was a single-blind Phase II pilot study examining strategy training among adults with cognitive impairments after stroke.²⁸ Participants were 30 individuals with acute stroke admitted to inpatient rehabilitation, and demonstrated cognitive impairments (Quick Executive Interview = 3).^{29–30} Individuals with severe aphasia (Boston Diagnostic Aphasia Examination Severity Rating Scale = 1);³¹ physician diagnosis of dementia prior to stroke onset (as documented in the medical record); major depressive disorder, bipolar, or psychotic disorder (Primary Care Evaluation of Mental Disorders);³² or recent substance abuse (within 3 months, Mini-International Neuropsychiatric Interview);³³ or anticipated length of stay less than 5 days were excluded from the study. Eligible participants were randomized to intervention group (strategy training, reflective listening) using a simple randomization scheme (1:1 ratio, generated through a random numbers table), developed and maintained by the research coordinator. Recruitment staff, therapists, and raters did not have access to the randomization scheme.

Intervention

Both intervention groups received usual inpatient rehabilitation, plus one 45 minute research intervention session (strategy training or reflective listening) per day, 5 days per week, for the duration of inpatient rehabilitation. Research intervention sessions were delivered according to standardized procedures described elsewhere^{26–27} by trained rehabilitation personnel who were masked to the opposing protocol. Delivery of both interventions was monitored using rigorous fidelity procedures described in detail elsewhere.²⁷ Participation in intervention sessions was assessed with the Pittsburgh Rehabilitation Participation Scale,³⁴ and understanding of the intervention was assessed (1=minimal understanding, 2=acceptable understanding, 3=excellent understanding) in both groups.

Briefly, participants in both groups engaged in a structured goal identification activity, using the Canadian Occupational Performance Measure.³⁵ Participants described a typical daily routine, and based on this identified daily activities that they thought were important and difficult to perform after their stroke. From this exercise, participants in both groups identified 4–6 goals to address in rehabilitation. Strategy training sessions focused on coaching participants to address their self-selected activity-based goals through self-evaluation of performance, self-derived strategies to address performance (using a global

strategy training method, Goal-Plan-Do-Check),³⁶ and application of learned principles across self-selected goals. In contrast, reflective listening sessions focused on participants' reflections on their goals and their rehabilitation experiences, facilitated by scripted open-ended questions and active listening skills (attending, following and responding). Reflective listening sessions were dose-matched and controlled for the non-specific effects of attention.

As this is a secondary analysis of data from a completed trial, it is worth noting that the strategy training intervention was designed incorporating principles of meta-cognitive instruction,³⁶ for the purposes of reducing disability and improving executive cognitive functions. Although not the original intent of the initial trial, noted similarities between the principles of meta-cognitive instruction (that have shown promise for reducing disability and improving executive cognitive functions) and the active ingredients of problem solving therapy and behavioral activation programs (that have shown promise for addressing apathy) raised the question as to whether strategy training in its original form had any effect on apathy symptoms over the course of the trial. These noted similarities included a focus on goal-setting and planning (addressing activation or initiation), self-monitoring, and problem solving (addressing goal completion).

Measures

To characterize the sample, we collected relevant demographic (age, gender, race) and medical information (stroke type, hemisphere, onset) from the medical record. We measured medical burden with the Cumulative Illness Rating Scale³⁷ and baseline level of disability with the Functional Independence Measure.³⁸ We measured stroke severity using the National Institutes of Health Stroke Scale, a standardized neurological examination that characterizes type and severity of neurological impairment after stroke.³⁹ We also measured executive cognitive functions (Delis Kaplan Executive Functioning System Color Word Interference),⁴⁰ and mood symptoms (Hamilton Rating Scale for Depression).⁴¹

We measured apathy symptoms with the Apathy Evaluation Scale⁴² at study admission, 3 months and 6 months later using trained raters who were masked to group assignment. The Apathy Evaluation Scale is a clinician-rated structured interview that measures lack of motivation or interest in goal-directed activities. Eighteen items addressing initiative, effort, productivity, emotional responsivity, novelty seeking or curiosity, perseverance, and social engagement are scored on a 4 point ordinal scale, indicating the degree to which participant responses are characteristic of motivation or interest (1=greatly characteristic, 4=not at all characteristic). Item scores are summed, with a total score of 37 or higher deemed indicative of apathy. The Apathy Evaluation Scale has demonstrated good convergent and divergent validity, and good intra-rater and inter-rater reliability,⁴² and has been used to characterize apathy in participants with stroke and traumatic brain injury.⁴²⁻⁴³

Data Analyses

All statistical analyses were performed using SASR version 9.3 software (SAS Institute, Inc., Cary, North Carolina). We used the intent-to-treat principle including all participants assigned to intervention groups, regardless of intervention received or study completion. We computed descriptive and inferential statistics to examine baseline differences between the

groups, using *t* tests and chi square tests, as appropriate. Prior to model testing, we examined descriptive statistics (measures of central tendency and variability) and distributional plots for the dependent variable (Apathy Evaluation Scale) and potential predictors (National Institutes of Health Stroke Scale, Delis Kaplan Executive Functioning System Color Word Interference Subtest, Hamilton Depression Rating Scale) for each intervention group over the three time points (baseline, 3 months, and 6 months). Missing values of predictors were imputed using multiple imputation with fully conditional specification.⁴⁴ We used repeated measures fixed-effects models to examine group (strategy training, reflective listening) and time (baseline, 3 and 6 months) main effects, as well as the group*time interaction. Model parameters were estimated using the full maximum likelihood technique. Next, predictors (National Institutes of Health Stroke Scale, Delis Kaplan Executive Functioning System Color Word Interference Subtest, Hamilton Depression Rating Scale) were added to the model separately, then in pairs, and finally all together to determine the contribution to the model fit. We used a nested likelihood ratio test with deviance values from the full (model with predictors) and reduced (initial model) models to select the model that best fit the data.⁴⁵ Analyses were first completed using all available data; and then a sensitivity analysis was conducted by reanalyzing models for participants with complete data. Post hoc analyses were conducted using planned comparisons to interpret significant fixed effects and effect sizes were computed using model-derived change score estimates. Interpretation of effect sizes was based on the recommendations of Cohen (0.2=small, 0.5=medium, 0.8=large).⁴⁶

Results

Sixty participants enrolled in the study (Figure 1). Of these, 27 participants were excluded because they did not meet eligibility criteria, and 3 participants withdrew during the screening process prior to randomization. Thus, we randomized 30 participants to the intervention groups. All participants received and actively participated in the allocated intervention (Pittsburgh Rehabilitation Participation Score: 1=no participation, 5=very good participation; strategy training $M=4.69$ $SD=0.80$, attention control $M=4.99$, $SD=0.90$). Participants in both groups demonstrated an acceptable understanding of the intervention they received (1=minimal understanding, 2=acceptable understanding, 3=excellent understanding; strategy training $M=2.28$ $SD=0.35$, attention control $M=2.87$, $SD=0.28$).

There were no significant differences between groups with respect to demographic or stroke-related variables, with the exception of stroke severity (Table 1). Strategy training participants had significantly greater stroke severity compared to reflective listening participants. Strategy training and reflective listening participants exhibited similar levels of apathy symptoms at study admission. These levels were subsyndromal (1 strategy training participant and 1 reflective listening participant met criteria for syndromal apathy).⁴² Yet, there was a range of apathy symptoms in the sample. There were no differences in baseline executive cognitive functions (both groups demonstrated severe impairments) or depressive symptoms (both groups demonstrated minimal symptom levels).

Fixed Effects Models - All Data

Raw means for apathy symptoms by group over time are presented in Figure 2. Analyses of the initial model (group, time and group*time interaction) revealed a significant group*time interaction ($F_{2,28}=3.61, p=.040$), indicating that changes in levels of apathy symptoms differed between strategy training and reflective listening participants over the three time points. Tests of main effects showed that levels of apathy symptoms were significantly different over time ($F_{2,28}=3.56, p=.041$), and just missed significance between treatment groups ($F_{1,26}=4.05, p=.054$). Examination of each predictor (National Institutes of Health Stroke Scale, Delis Kaplan Executive Functioning System Color Word Interference Subtest, Hamilton Depression Rating Scale) separately, then in pairs, and all together showed that there was no improvement in model fit between the full and reduced models. The group*time interaction and main effect of time remained significant in all models; however, the predictor effects (including executive cognitive functions and depressive symptoms) were non-significant. Because the predictors did not improve model fit, our final model included group, time, and group*time with no additional covariates.

Fixed-Effects Models - Complete Data Only

Analyses of models using only participants with complete data at each time point ($n=12$) produced a significant group*time interaction ($F_{2,20}=4.45, p=.025$) and a significant main effect of time ($F_{2,20}=3.94, p=.036$), and a non-significant main effect for treatment group ($F_{1,10}=2.11, p=.176$). Predictors were non-significant and did not improve model fit. The final model included group, time, and the group*time interaction. Thus, the results of these analyses were very similar to the analyses using all participants.

Post Hoc Contrasts and Effect Sizes

Post hoc contrasts and effect sizes for group differences from baseline to month 3 and from baseline to month 6 are reported in Table 2. The magnitude of between group differences in month 3 apathy change scores was large and statistically significant ($d=-0.99, t_{28}=-2.64, p=.013$), while the magnitude of the between group differences in month 6 apathy change scores was moderate to large, and not statistically significant ($d=-0.70, t_{28}=-1.86, p=.073$).

Given the small sample, we did not conduct additional statistical models examining differences between groups within each of the dimensions of the Apathy Evaluation Scale (i.e., cognitive, behavioral and emotional) as originally defined by Marin.⁴² However, descriptive plots of mean summary scores within each dimension suggest that differences between groups in overall apathy scores may have been driven by differences in cognitive and behavioral dimensions (Figure 3). The descriptive plots do not indicate any differences between groups in the emotional dimension.

Discussion

Strategy training was associated with significantly lower levels of post-stroke apathy than reflective listening (an attention control condition) over the first 6 months after stroke. In contrast to the pharmacological agents discussed earlier in this paper, the effect size of differences between groups was large at 3 months and moderate to large at 6 months

(although not statistically significant at 6 months). Of note, reflective listening participants demonstrated an increase in apathy symptoms at 3 and 6 months, relative to baseline. These findings are consistent with longitudinal studies that report increases in apathy symptoms in the first 3 to 6 months after stroke and leveling off after that point.⁷ Strategy training participants demonstrated very little change in apathy symptoms at 3 months, and a small reduction in apathy symptoms at 6 months. These data suggest that benefits of strategy training administered in the acute phase may not be in the reduction of apathy symptoms per se, but in the prevention of increased levels of apathy symptoms that may occur absent intervention.

There may be several reasons why strategy training may influence apathy symptoms in acute phase after stroke. It may be that strategy training – through its focus on goal-setting and planning, self-monitoring and problem solving – addresses the core deficits of apathy (i.e., activation and perseverance). Alternatively, it may be that strategy training addresses a broader constellation of sequelae that influence apathy symptom levels. We previously reported that strategy training showed promise for reducing disability and improving executive cognitive functions in the first 6 months after stroke.²⁸ It could be that the reductions in disability sustain motivation for goal-directed behavior, and thus have indirect benefits for apathy symptoms. Similarly, improvements in executive cognitive functions may prevent loss of interest and initiation of goal-directed behavior that typically emerges after stroke. Strategy training may also address mood. However, the data from the current trial do not appear to support these explanations. Neither executive cognitive functions nor depressive symptoms altered the effect of intervention group on apathy symptoms over time. These findings suggest that while apathy symptoms may be related to executive cognitive functions and depressive symptoms, apathy symptoms may be driven by factors other than those detected through our standardized neuropsychological tests and psychiatric interviews. We pose this premise cautiously, given that participants in our sample had subsyndromal and not syndromal levels of apathy (and subsyndromal depressive symptom levels) at baseline, and that an association between apathy and traditional cognition and mood assessments may be more apparent with higher levels of apathy symptoms.⁴⁷

Strategy training appeared to address the cognitive (interest in goal-directed activities) and behavioral (initiation and completion of goal-directed activities) dimensions of apathy symptoms, but not the emotional dimension of apathy symptoms. Examining the active ingredients of strategy training – self-directed goal setting and planning, self-monitoring, and problem solving – these findings make sense. The incorporation of self-selected goals may enhance interest and motivation for goal-directed activities, and the development of goal planning, self-monitoring, and problem solving skills may promote activation and perseverance. However, there are no components that explicitly address limited emotional responsivity – or enthusiasm for goal-directed activities. This may be better served through pharmacological augmentation, an approach that has demonstrated efficacy in the management of depression.⁴⁸ Co-activation through pharmacotherapy (addressing biological substrates) and interventions like strategy training (addressing behavioral substrates) may provide the necessary biological and behavioral augmentation to maximize benefit from rehabilitation in the early phase of recovery, and promote the best outcomes.⁴⁹

Recent evidence suggests that the neurobiological bases for activation or energization of effortful goal-directed behavior (appetitive or preparatory behavior) can be distinguished from the completion or achievement of goal-directed behavior (consummatory behavior).⁵⁰ Among adults with post-stroke apathy, deficits in appetitive or preparatory behavior have been consistently noted, whereas consummatory behavior remains relatively intact.⁵¹ Thus, post-stroke apathy appears to be more strongly associated with deficits in activation compared to the maintenance or completion of goal-directed behavior. By incorporating active ingredients of “behavioral activation” – particularly goal setting and planning – strategy training may be specifically addressing this deficit in post-stroke apathy. Certainly interventions incorporating similar active ingredients have demonstrated promise in adults with apathy attributed to stroke and traumatic brain injury (see Lane-Brown and Tate, Mikamiet al., for selected examples).^{23–25} Future investigations seeking to clarify the behavioral phenotypes and biological substrates of apathy across disorders may consider isolating active ingredients in these interventions, and exploring the impact of these components on appetitive and consummatory behaviors. This could not only advance our understanding and characterization of apathy, but also may point to more focused and efficient intervention approaches.

Due to exclusion of individuals with severe aphasia, our sample contained twice as many participants with right hemisphere stroke as left hemisphere stroke. This may have implications for the interpretation of study findings. Although there have been some reports of laterality of post-stroke apathy (i.e., right hemisphere stroke is more frequently associated with post-stroke apathy than left hemisphere stroke),¹² evidence suggests that lesions impeding transmission to and from the medial frontal lobes and basal forebrain will culminate in apathy regardless of hemisphere affected by stroke.⁸ Post-stroke apathy is frequently associated with lesions impairing the medial orbitofrontal regions (particularly the anterior cingulum), the ventral striatum (particularly the nucleus accumbens), and the medial dorsal thalamic nuclei.¹³ These regions are particularly vulnerable to stroke, given the vascular supply to many of these regions from branches of the middle cerebral artery, the most common site for stroke in the United States.⁵²

Although promising, we must acknowledge limitations in the analyses that prohibit wide scale generalization of these findings. Chief among these is our focus on apathy symptom levels, rather than syndromal apathy. This was a secondary analysis and recruitment for the parent study was not focused on admitting participants with syndromal apathy. Thus, our findings do not generalize to individuals with syndromal apathy in the acute phase of stroke. In addition, due to our small sample, we were unable to detect predictor*time effects, influencing our choice of the final model. Furthermore, complete data on the Apathy Evaluation Scale at each time point was only present in a portion of the sample. The primary reason for missing data was acuity of medical illness prohibiting accurate assessment either at baseline or at follow-up. We evaluated the impact of missing data through sensitivity analyses, and concluded that the final model was stable. Nonetheless, we urge caution in generalizing these findings. Finally, there may be a number of additional unmeasured factors that could have influenced outcomes, including premorbid function, post-acute rehabilitation course, and social support.

The current investigation raises many important questions about apathy, as well as the associations among apathy, executive cognitive functions, mood, and disability. Recent reports have focused on overlaps between apathy in acquired brain disorders and disruptions of executive cognitive functions and mood. Certainly these sequelae share some common neuroanatomical substrates (i.e., anterior frontal lobes). However, reports vary as to the degree of overlap among these sequelae.^{5,53–54} These varying reports may be due to variations in definitions and manifestations of apathy across neurological and psychiatric disorders. For these analyses we used the neuropsychiatric definition of apathy, originally conceived by Marin and reflected in the recently proposed criteria for apathy in neuropsychiatric disorders.^{1–3} While this remains an accepted definition for most clinical and scientific venues, recent efforts have focused on isolating and studying the dimensions of apathy. This remains an important area of study, and comparisons among multiple fields of study (neuroscience, psychology, psychiatry, rehabilitation), and multiple clinical populations (stroke, traumatic brain injury, dementia, psychiatric disorders) may be informative. In particular, exploration of the behavioral phenotypes (i.e., dimensions) and biological substrates underlying apathy in each of these disorders may add clarity in the characterization, assessment and management of apathy symptoms.

Conclusion

Secondary analyses suggest that strategy training is promising as an adjunctive therapy to rehabilitation for maintaining low levels of apathy symptoms among individuals with subsyndromal apathy symptoms after acute stroke. Prospective examination of strategy training, and strategy training augmented with pharmacotherapy, in individuals with syndromal apathy may illuminate a better understanding of mechanisms and may lead to efficacious and efficient interventions for post-stroke apathy, as well as apathy in other related neurological and psychiatric disorders.

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Figure 1.
CONSORT Flow Diagram

Figure 2.
Apathy Evaluation Scale Scores, Unadjusted Means, By Time Point

Figure 3. Apathy Evaluation Scale Dimension Scores, Unadjusted Means, By Time Point

Note. Dimensions scores derived from sums of items as recommended by Marin.⁴²

Cognitive=Apathy Evaluation Scale items 1,3–5,8, 11, 13, 16; Behavioral=Apathy Evaluation Scale items 2, 6, 9, 10, 12; Emotional=Apathy Evaluation Scale items=7,14.

Table 1

Baseline Characteristics of Participants

	Strategy Training (n=15)	Reflective Listening (n=15)	
Sex, Male, n(%)	9 (60)	11 (73)	$\chi^2_I=0.60$
Age, Years, <i>M(SD)</i>	64.87 (16.59)	71.80 (13.19)	$t_{28}=1.26$
Race, White, n(%)	12 (80)	14 (93)	$\chi^2_I=1.15$
Stroke onset, Days, <i>M(SD)</i>	16.80 (15.58)	18.47 (21.29)	$t_{28}=0.25$
Stroke type, Ischemic, n(%)	10 (67)	11 (73)	$\chi^2_I=0.16$
Hemisphere, Right, n(%)	10 (67)	10 (67)	$\chi^2_I=0.00$
Medical burden, CIRS Total, <i>M(SD)</i> [*]	2.37 (0.47)	2.36 (0.57)	$t_{28}=0.04$
Disability, FIM, <i>M(SD)</i>	65.27 (10.85)	62.60 (16.56)	$t_{28}=0.52$
Apathy, AES, <i>M(SD)</i> [*]	25.79 (7.62)	25.18 (4.40)	$t_{24}=0.23$
Stroke severity, NIHSS, <i>M(SD)</i> [*]	8.87 (2.77)	5.87 (2.72)	$t_{28}=2.99^\dagger$
Executive functions, CWI, <i>M(SD)</i>	3.69 (2.36)	2.73 (2.34)	$t_{26}=1.08$
Depressive symptoms, HamD, <i>M(SD)</i> [*]	6.71 (4.36)	6.75 (4.53)	$t_{24}=0.02$
Research intervention sessions, <i>M(SD)</i>	9.40 (5.31)	9.00 (4.89)	$t_{28}=0.21$

CIRS=Cumulative Illness Rating Scale. FIM=Functional Independence Measure. AES=Apathy Evaluation Scale. NIHSS= National Institutes of Health Stroke Scale. CWI=Color Word Interference (Switching Subscale). HamD=Hamilton Rating Scale for Depression.

^{*} Lower scores=better health.

[†] $p<.05$

Table 2

Post Hoc Contrasts Using Model-Derived Change Score Estimates

	Strategy Training (n=15)	Reflective Listening (n=15)		Coehn's <i>d</i> Effect Size Estimate
Apathy				
Baseline to Month 3, <i>M(SE)</i>	25.17 (2.33)	34.28 (2.67)	$t_{28}=-2.64, p=.013$	-0.99
Baseline to Month 6, <i>M(SE)</i>	21.81 (2.71)	29.19 (2.74)	$t_{28}=-1.86, p=.073$	-0.70

Note: Final Model included group, time, and group*time interaction.