A BEHAVIORAL APPROACH TO MANAGEMENT OF NEUROLEPTIC-INDUCED TARDIVE DYSKINESIA: PROGRESSIVE RELAXATION TRAINING

by

Philip R. Johnson

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As members of the Dissertation Committee, we certify that we have read the dissertation 
prepared by Philip R. Johnson 
entitled A Behavioral Approach to Management of Neuroleptic-Induced Tardive 
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and recommend that it be accepted as fulfilling the dissertation requirement for the 
Degree of Doctor of Philosophy 

Charlene Kampfe, Ph.D.  
Date: April 22, 2009 

Chih-Chin Chou, Ph.D.  
Date: April 22, 2009 

William Downey, Ph.D.  
Date: April 22, 2009 

Carl Liaupsin, Ed.D.  
Date: April 22, 2009 

Final approval and acceptance of this dissertation is contingent upon the candidate’s 
submission of the final copies of the dissertation to the Graduate College.

I hereby certify that I have read this dissertation prepared under my direction and 
recommend that it be accepted as fulfilling the dissertation requirement. 

Dissertation Director: Charlene Kampfe, Ph.D.  
Date: April 22, 2009
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DEDICATION

To Barbara, for your understanding, steadfast love, and support, and A. E. Kiser, for planting a seed so many years ago.
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ABSTRACT

The effectiveness of progressive relaxation training in decreasing the severity of neuroleptic-induced tardive dyskinesia (TD) was examined in the current study. Three residents at a county-owned nursing home who had been receiving neuroleptic medications for a number of years to treat severe mental illness participated in this study. A multiple baseline across subjects design was used to evaluate the effect of progressive relaxation training on the participant’s orofacial TD symptomatology. The severity of each participant’s orofacial TD was observed to improve when the intervention was introduced. Treatment integrity and IOA data that were collected indicate that the intervention was implemented at a high level of fidelity and that data were reliable. Thus, a clear functional relationship was established between progressive relaxation training and severity of orofacial TD in this study. Although the present study was preliminary in nature, the results that were obtained provide a basis upon which to develop a behavioral treatment protocol for managing TD.
“Somewhat like happiness—which is readily understood until one is asked to define it—the apparently straightforward, causative relationship between neuroleptic treatment and development of tardive dyskinesia unravels under scrutiny”

Gardos & Cole, 1997, p. 113

Neuroleptic Medications

Since their introduction in the early 1950s, neuroleptic, or antipsychotic, medications have greatly influenced psychiatric care and treatment (Keltner & Folks, 2005; Rivas-Vazquez, Blais, Rey, & Rivas-Vazquez, 2000). Prior to their use, hundreds of thousands of individuals with severe psychiatric problems were hospitalized, at times under poor conditions (Keltner & Folks, 2001). Physical restraint; social isolation; chemically induced sedation, comatose states, and/or seizures; and occasional treatment with aggressive measures (e.g., psychosurgery, electroshock therapy) were commonly used to control patients before the widespread use of neuroleptic agents (Keltner & Folks, 2001; Minchin & Csernansky, 1996). Persons who were the recipients of these treatments were rarely able to resume productive social or occupational functioning (Keltner & Folks, 2001).

Paul Charpontier, a French scientist, synthesized the first neuroleptic agent, chlorpromazine (Thorazine), in 1950 while he was attempting to develop an effective
antihistamine (Minchin & Csernansky, 1996). Although its antihistaminic properties were found to be mild, chlorpromazine was highly sedating. The sedating effects of the drug attracted the attention of Henri Laborit, a French surgeon at the military hospital of Val-de-Grâce. Laborit found chlorpromazine to be effective in alleviating patients’ anxiety during surgery. Chlorpromazine produced what Laborit referred to as an ataractic effect (literally "without emotion"), leading him to hypothesize that it might be useful in psychiatry (Ayd, Jr., 1991; Deniker, 1970; Deniker, 1989; Guidry et al., 1988).

Encouraged by Laborit’s findings, Jean Delay and Pierre Deniker, two French physicians, began treating psychiatric patients with chlorpromazine (Deniker, 1989). In 1952, the two researchers published six reports pertaining to their findings on the use of chlorpromazine as an antipsychotic agent (Ayd, Jr., 1991; Deniker, 1970; Deniker, 1989; Keltner & Folks, 2005). Delay and Deniker found that “agitation, aggressiveness, and delusional conditions of schizophrenia improved” (Deniker, 1970, p. 158) when chlorpromazine was administered to their patients. The drug was relatively ineffective, however, in treating the negative symptoms of schizophrenia (i.e., affective flattening, apathy, anhedonia, alogia, and avolition) (Deniker, 1970). The reports by Delay and Deniker drew the attention of the psychiatric community and generated worldwide clinical trials of chlorpromazine (Ayd, 1963; Ayd 1991). The drug was introduced in public hospitals in about 1954 (Keltner & Folks, 2005). Chlorpromazine, and the many psychotropic drugs that followed, literally transformed treatment for mental illness and played a major role in the restructuring of the state hospital system in the United States (Keltner & Folks, 2005).
Although neuroleptic medications have primarily been prescribed to manage psychoses in persons with serious mental illness (Kane, 2006; Keltner & Folks, 2005) as well as psychiatric disturbances and severe behavioral problems in persons with developmental disabilities (Advokat, Mayville, & Matson, 2000), these agents have also been used for treating a variety of other conditions and disorders. These disorders include refractory major depression, delusional depressive disorder, anorexia, anxiety, hallucinogen-induced psychosis, agitation in dementia and depression, Huntington’s disease, impulsivity, refractory obsessive-compulsive disorder, porphyria, refractory hiccups, itching, antiemetic uses, and personality disorders (Keltner & Folks, 2005).

**Tardive Dyskinesia**

Unfortunately, neuroleptic medications are associated with a number of side effects, many of which can be quite serious. Neuroleptic-induced extrapyramidal symptoms (EPS), which manifest as abnormal involuntary movements, are among the most worrisome of these side effects (Deniker, 1970; Gebhardt et al., 2006; Keltner & Folks, 2005; Rivas-Vazquez et al., 2000). Tardive dyskinesia (TD), a potentially irreversible involuntary movement disorder that is characterized by constant movements of the mouth, lips, tongue, jaw, trunk, and/or extremities has been referred to as "the most dreaded" (Keltner & Folks, 2005, p. 492), “the most serious” (Cortese, Jog, McAuley, Kotteda, & Costa, 2004, p. 32), “one of the most severe” (Kučerová, 2002, p. 421), and “the principle adverse effect” (Margolese, Chouinard, Kolivakis, Beauclair, & Miller, 2005, p. 541) of these neuroleptic-induced EPS.
Neuroleptic refers to the adverse, rather than beneficial, effects associated with antipsychotic medications. Delay and Deniker proposed the use of the term neuroleptic (from the Greek: “which takes the nerve”) in January 1955 to underscore the tendency of these medications to produce significant EPS (Deniker, 1989). The qualifier, tardive, which means "delayed" or "belated onset," was introduced by Faurbye et al. (1964) because TD symptoms typically manifest after long-term exposure to neuroleptic medications (Faurbye, Rasch, Peterson, Brandborg, & Pakkenberg, 1964; Fountoulakis, 2006; Gebhardt, 2006; Keltner & Folks, 2005). It is important to note, however, that TD can manifest after short-term exposure to neuroleptic medications (Margolese & Ferreri, 2007; Skidmore, Weiner, & Burke, 2005). Today, the term tardive dyskinesia is frequently used to indicate a disorder that is induced by neuroleptic or antipsychotic medications. Hence, neuroleptic- or antipsychotic-induced tardive dyskinesia is typically referred to as tardive dyskinesia (DSM-IV-TR, 2000).

The first-generation of neuroleptic medications, usually referred to as conventional or traditional neuroleptics, are particularly prone to inducing EPS, whereas the second-generation, or atypical neuroleptic medications (i.e., those developed after 1990), present less risk of inducing these involuntary movement disorders (Correll, Leucht, & Kane, 2004; Farah, 2005; Gebhardt et al., 2006; Keltner & Folks, 2005; Pierre, 2005; Remington, 2007). Table 1 provides a list of traditional and atypical neuroleptic medications. The terms neuroleptic and antipsychotic are frequently used interchangeably; however, it has been argued that the atypical agents should be referred to exclusively as antipsychotics because they demonstrate a reduced tendency to produce
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Note. Information for this table was obtained from the following source: *Psychotropic Drugs* (4th ed.), by N. L. Keltner and D. G. Folks, 2005, Elsevier Mosby.
EPS (Correll, Leucht, & Kane, 2004; Ross, Thomas, Booth, & Weinborn, 2005). Due to their dopamine-antagonist properties (discussed below), atypical neuroleptic medications can most definitely induce the same movement disorders that are commonly associated with the traditional neuroleptic medications (Keltner & Folks, 2005; Physicians' Desk Reference, 2009; Remington, 2007). For this reason, the terms neuroleptic and antipsychotic will be used interchangeably in this report.

**Description of Symptoms**

Tardive dyskinetic movements are typically choreatic (repetitive, jerky, irregular, usually short amplitude) (Pierre, 2005); however, they may also present as athetoid (slow, sinuous, or writhing), dystonic (slow and sustained muscle contractions), stereotypic (rhythmic and repetitive), or as a combination of any of these movements (Sachdev, 2000). The most common clinical presentation of TD involves the buccolingual masticatory syndrome, also referred to as orofacial dyskinesia (grimacing; tongue protrusion; lip-smacking, puckering, and pursing) (Gebhardt et al., 2006; Kane, 2001, Vaddadi, Hakansson, Clifford, & Waddington, 2006). It is unknown why TD involves the lower facial musculature in most individuals (Lohr, Kuczynski, & Niculescu, 2003). In addition, TD is associated with impaired finger and toe movements that resemble piano or guitar playing movements (Pierre, 2005) and, although it is rare, choreic movements can also manifest in the trunk and extremities (Rodnitsky, 2002). In many cases, TD is initially detected by family members and clinicians because individuals with this condition are often unaware of, or undisturbed by, the movements (Pierre, 2005).
The symptoms of TD can vary from minimal to disabling, although the majority of cases are mild (Fabbrini, Barbanti, & Aurilia, 2001; Oosthuizen, Emsley, Maritz, Turner, & Keyter, 2003). The involuntary movements may be momentarily worsened by emotional arousal, stress, and distraction during voluntary movements in areas of the body that have not been affected (DSM-IV-TR, 2000). Stimulants, withdrawal from neuroleptic agents, and anticholinergic medications also tend to worsen the symptoms (DSM-IV-TR, 2000). Dyskinetic movements are momentarily reduced by relaxation and by voluntary movements in affected areas of the body; they are typically absent during sleep. TD may be temporarily suppressed by increased doses of neuroleptics or sedatives (DSM-IV-TR, 2000). Finally, the severity of TD may vary over time, improve during the course of neuroleptic therapy, and, in rare cases, spontaneously remit (Margolese & Ferreri, 2007).

Etiology

TD results from long-term exposure to dopamine antagonists (i.e., chemical agents that block dopamine receptors) (Soares-Weiser & Fernandez, 2007; Stahl, 2000). Conventional neuroleptics, as a class, are dopamine antagonists (Stahl, 2000). Although the etiology of TD is not clearly understood, it is hypothesized that chronic blockade of the postsynaptic dopamine (D₂) receptors in the nigrostriatal dopamine pathway results in dopamine receptor supersensitivity, which produces TD (Brasic, Bronson, & Chun, 2007; Soares-Weiser & Fernandez, 2007; Stahl, 2000). The dopamine receptor supersensitivity theory has guided TD research since 1970; however, several inconsistencies suggest that
this theory does not fully explain the pathogenesis of TD (Hyde, Apud, Fisher, & Egan, 2005; Sachdev, 2000). These inconsistencies include the following: (a) supersensitivity develops within 2 to 4 weeks of initiating neuroleptic treatment, whereas TD typically manifests after long-term use; (b) most subjects in animal studies develop supersensitivity, although TD develops in only a fraction of patients; and (c) supersensitivity declines within weeks of discontinuing neuroleptic treatment, whereas TD can be permanent (Hyde et al., 2005; Sachdev, 2000).

The shortcomings of the dopamine supersensitivity hypothesis have led to the consideration of additional etiological models. Soares-Weiser and Fernandez (2007) suggest that the effect of neuroleptics may not be restricted to the dopaminergic system. Fibinger and Lloyd (1984), for example, proposed that TD is caused, at least to some degree, by neuroleptic-induced degeneration of the gamma-aminobutyric acid (GABA) neurons in the basal ganglia. These neurons play a central role in the subcortical regions that produce abnormal movements (Hyde et al., 2005). Abnormalities in other neurotransmitter pathways (e.g., acetylcholine, serotonin, norepinephrine, and enkephalin) have also been implicated in the pathophysiology of TD (Wonodi, Hong, & Thaker, 2005). It has been further hypothesized that dopamine receptor blocking agents cause an increased synthesis of hydrogen peroxide which generates free radicals that damage cell components (Lohr, 1991). Free radicals are reactive chemical species that are generated through a number of pathologic and physiologic processes. They have been implicated in a number of neuropsychiatric conditions, many of which manifest as psychopathologic symptoms and movement disorders (Lohr, 1991).
Atypical neuroleptics are serotonin-dopamine antagonists (Stahl, 2000). Like the conventional agents, atypicals block D2 receptors; however they also bind to postsynaptic serotonin 2A (5-HT$_{2A}$) receptors and block them. In each dopamine pathway, serotonin interacts with the 5-HT$_{2A}$ receptors on the dopamine neurons and inhibits the release of dopamine. Blocking the 5-HT$_{2A}$ receptors disinhibits the dopamine neurons, allowing them to release dopamine. When this occurs, dopamine can compete with the atypical antipsychotic for the D$_2$ receptor and reverse the inhibition at that site (Stahl, 2000). Atypical agents have a higher affinity for 5-HT$_{2A}$ receptors over D2 receptors in the nigrostriatal dopamine pathway. It is hypothesized that this high serotonin-to-dopamine receptor blockade ratio generates an increase in dopamine release that balances out the dopamine blockade effect at the D$_2$ receptor sites, resulting in a lowered risk of TD and other EPS (Meltzer, Matsubara, & Lee, 1989; Remington & Kapur, 1999; Stahl, 2000). This effect is finite, however, and can be decreased or eliminated as D2 occupancy increases with the administration of higher doses of atypical agents (Remington & Kapur, 1999).

Rate of Incidence

Estimates on the overall prevalence of TD vary. The disparity in findings can be attributed to the use of different methods of assessment; differences in diagnostic criteria; differences in patient age, gender, and psychiatric diagnoses; duration and type of neuroleptic exposure; and possible comorbid medical and neurological illnesses (Chouinard, 2004 b; Hyde et al., 2005; Wonodi, Hong, & Thaker, 2005). In general,
however, at least 20% of those treated with neuroleptics develop TD (Soares-Weiser & Fernandez, 2007), and approximately 5% are expected to develop the condition with each year of exposure to these agents (Kane, Woerner, & Lieberman, 1988). This rate is cumulative. It has been estimated that approximately 70% (i.e., 68.4%) of individuals who have been exposed to typical neuroleptic agents continuously for 25 years will develop TD (Glazer, Morgenstern, & Doucette, 1993). It is not known why some individuals, but not others, develop TD (Margolese & Ferreri, 2007).

Published reports appear to support the claim that atypical neuroleptic medications present a lower risk for causing TD. Correll, Leucht, and Kane (2004), for example, reviewed 11 long-term neuroleptic trials (i.e., at least 1 year in duration) and found the weighted mean annual incidence risk for TD associated with atypical neuroleptics to be 2.1%. However, Correll et al. also found that the data from these trials “seem to indicate” that the benefits of lower TD risk may be reduced with higher doses of atypical neuroleptic medications. This is an important concern, because the doses of atypical neuroleptic medications used in clinical settings are increasing (Pierre, 2005). In another study, conducted on 162 hospitalized patients at Central State Hospital in Petersburg Virginia (Ross, Thomas, Booth, & Weinborn, 2005), TD was present in 40% of the participants, the majority of whom (i.e., 94%) were taking neuroleptic medications. Of those with TD, 40% were taking only typical neuroleptics, 39% were taking only atypical neuroleptics, and 47% were taking both typical and atypical neuroleptic medications.
Risk Factors

Advanced age has consistently been identified as a risk factor for the development of TD, particularly with the first-generation agents (Caligiuri, Jeste, & Lacro, 2000; Dolder & Jeste, 2003; Fabbrini, et al., 2001; Fountoulakis et al., 2006; Vaddadi et al., 2006). For persons older than 50 years who are beginning treatment with typical antipsychotics, the rate of TD has been found to be three to five times greater than for younger age groups (Woerner, Alvir, Saltz, Lieberman, & Kane, 1998). On the other hand, Correll et al. (2004) found that the incidence of TD in adult and elderly patients receiving second-generation antipsychotics “appears to be” about one-fifth of the risk found with first-generation antipsychotics. Other findings related to TD risk factors have often been inconsistent (Müller, Shinkai, De Luca, & Kennedy, 2004).

Although gender is frequently cited as a factor associated with higher risk for TD, findings from research is conflicting. Initial studies indicated higher rates of TD in females; however, those findings were confounded by variations in age or treatment variables between groups (Egan, Apud, & Wyatt, 1997). Results obtained from subsequent studies that controlled for these factors showed higher rates of TD only in women over the age of 65, with no apparent gender effects in younger cohorts (Egan et al., 1997).

Additional conditions or factors that may increase the risk for developing TD include diabetes (Caligiuri & Jeste, 2004; Elkashef & Wyatt, 1999), the appearance of EPS shortly after antipsychotic treatment has been initiated (Glazer & Saltz, 2006), the presence of affective symptoms (Elkashef & Wyatt, 1999; Fountoulakis et al., 2006),
stereotypic movement disorder (Bodfish, Newell, Harper, & Lewis, 1996), negative symptoms in persons with schizophrenia (Egan et al., 1997), brain damage (Fountoulakis et al., 2006), and smoking (Chong, Tan, Tan, & Mythily, 2003). Treatment variables associated with increased risk for developing TD include medication dosage (i.e., higher doses are associated with increased risk) (Fountoulakis et al., 2006), duration of exposure to antipsychotic agents (i.e., risk increases exponentially with duration of treatment) (Fountoulakis et al., 2006; Miller et al., 2005), and treatment with anticholinergic agents (Miller et al., 2005).

**Treatment**

Treatment strategies for managing the symptoms of TD can be generally classified into three categories: (a) pharmacological, (b) neurosurgical, and (c) behavioral (Johnson, 2002). No treatment for TD has been found to be completely satisfactory or effective (Correll et al., 2004; Fabbrini, et al., 2001; Paulson, 2005; Soares-Weiser & Fernandez, 2007); therefore, preventative strategies are important (Brasic, Bronson, & Chun, 2007; Fabbrini et al., 2001; Margolese & Ferreri, 2007; Oosthuizen et al., 2003; Sachdev, 2000). Limiting exposure to conventional neuroleptic agents whenever possible has been the primary means of preventing TD (Hyde, Apud, Fisher, & Egan, 2005). Margolese and Ferreri recommend treatment with atypical antipsychotics first, introducing conventional antipsychotics only when (a) atypical agents fail to reduce psychosis, (b) patients prefer conventional agents, or (c) patients cannot tolerate atypical agents. Additional preventative strategies include (a) using the lowest neuroleptic dose
needed to achieve the desired effect, (b) periodically assessing the need for continued neuroleptic therapy whether or not TD is present, and (c) withdrawing neuroleptic therapy at the soonest possible opportunity (Brasic et al., 2007; Hyde et al., 2005; Soares-Weiser & Fernandez, 2007).

Although TD can be irreversible, it is possible to reverse or decrease the severity of symptoms if the condition is treated during the initial stages (Keltner & Folks, 2005). Discontinuing the offending drug, reducing the dose, or switching to an antipsychotic with potentially lower risk of causing TD (e.g., switching from a typical to an atypical antipsychotic) should be considered when involuntary movements first begin to manifest (Kane, 2006; Sachdev, 2000). Withdrawal of antipsychotic therapy, however, should only be considered in persons for whom antipsychotic treatment is not regarded as necessary (Damier et al., 2007). Some individuals have shown noticeable improvement in symptoms following discontinuation of neuroleptic treatment, although complete resolution of this condition is rare (Damier et al.) and withdrawing treatment with antipsychotics increases the risk of psychotic relapse (Gilbert et al., 1995). In severe cases, when TD poses a risk to health, impairs an individual’s ability to engage in everyday activities, or is otherwise significantly troublesome (e.g., causes problems with breathing, eating, walking, or sleeping), increasing the dose of the offending neuroleptic or switching to a high-potency typical neuroleptic may suppress the symptoms (Hyde et al., 2005). Suppression with typical neuroleptics is considered risky due to the following reasons: (a) suppression of TD is not always achieved when a neuroleptic dose is increased, (b) a breakthrough dyskinesia that is more difficult to treat may manifest, (c)
the likelihood of eventually achieving long-term remission of symptoms may be decreased, and (d) long-term worsening of the condition is a possibility (Egan et al., 1997; Hyde et al., 2005; Miyasaki & Lang, 1995; Sachdev, 2000).

A number of adjunctive medication strategies for the treatment of TD have been studied, although definitive findings in this area are lacking (Pierre, 2005). Soares-Weiser and Fernandez (2007) recently conducted an extensive literature search and found no evidence-based pharmacological interventions (i.e., those examined in randomized clinical trials) to be definitive for treating TD. In addition, many medications used for treating TD are associated with adverse effects. For example, common side effects of tetrabenazine, a catecholamine depleter used for treating TD, are drowsiness, Parkinsonism, depression, insomnia, nervousness, and akathisia (Sachdev, 2000). Tetrabenazine has also been associated with “florid psychiatric symptoms” (e.g., panic attacks, depressive and guilty thoughts, obsessional ruminations) (Bruneau et al., 2002). Atypical antipsychotics are associated with weight gain, metabolic disturbances, hyperprolactinemia, cardiac events, and sexual dysfunction (Wirshing et al., 2003). Wirshing et al. contend that these side effects “are emerging as the tardive dyskinesia equivalent of the conventional antipsychotic medications” (p. 165). It should be noted that the FDA has yet to approve any agents for treating TD (Glazer & Saltz, 2006).

Surgical intervention should only be considered for those patients with persistent (> 1 year) severe and disabling TD that is resistant to medical treatment (Damier et al., 2007). Neurosurgical treatments for TD include deep brain stimulation of the internal globus pallidus (Damier et al.; Schrader, Peschel, Petermeyer, Dengler, & Hellwig,
2003), pallidotomy (Lenders et al., 2005; Wang, Turnbull, Caine, Stoessl & Caine, 1997; Weetman, Anderson, & Gregory, 1997), thalamotomy (Druckman, Seelinger, & Thulin, 1962; Hillier, Wiles, & Simpson, 1999), and lesioning in the red nuclei (Nashold, 1969). With one exception (Damier et al.), the reports cited here have included only single cases. Although reports of successful neurosurgical treatment of antipsychotic-induced movement disorders are rare (Schrader et al., 2003), “substantial improvement” (Wang et al.) and “nearly complete abolition of TD” (Lenders et al.) have been achieved with pallidotomy. In addition, deep brain stimulation of the globus pallidus “immediately suppressed” oro-facial-lingual TD in one patient (Schrader et al.) and led to “a clear improvement” (i.e., a decrease of more than 40% in the main outcome measure at 6 months) in 10 patients who were experiencing severe TD (Damier et al.). Neurosurgical interventions for treating TD are costly and associated with serious potential side effects, including infection, damage to the optic tract and other vital structures, intracerebral hemorrhage, depression, and behavioral disorders (e.g., suicidality) (Damier et al., 2007; Hyde et al., 2005). 

Behavioral interventions for managing TD and similar involuntary movement disorders (e.g., dystonia) include biofeedback (Abrams, 1986; Albanese & Gaarder, 1977; Cotton, 1986; Fudge & Sison, 1997; Fudge, Thailer, Alpert, Intrator, & Sison, 1991; Sherman, 1979); overcorrection and positive and negative feedback (Taylor, Zlutnick, & Hoehle, 1979); binary feedback, self-monitoring, and videotape feedback (Frederiksen & Rosenbaum, 1979), discreet-discrete prompting and video feedback (Jackson, Schonfeld, & Griffith, 1983); and progressive relaxation training and covert
reinforcer sampling (Wisocki, 1993). Each of these interventions was found to decrease the dyskinetic movements in study participants.

Adverse effects from behavioral treatment approaches for TD were discussed in two reports (Abrams, 1986; Wisocki, 1993). The participants in Abrams’s study “appeared confused and were unable to recognize the relationship between their abnormal movements and the increasing tone [of the biofeedback apparatus]” (p. 61). As a result, Abrams modified the biofeedback procedure by providing “verbal information to the subject about the adequacy of his or her course of relaxation” (p.62). It should be noted that the participants in this study ($n = 10$) were individuals with chronic schizophrenia, institutionalized at a state hospital, who were difficult to manage due to behavioral problems. As a general rule, however, adverse effects associated with biofeedback are minimal (Grazzi 2007). Behavioral interventions may require consistent and intense work over an extended period of time to achieve a desired outcome. The participant in Wisoki’s study, for example, became tired of maintaining the behavioral treatment regimen and requested medication as an “instant cure” for her TD.

Problems that may arise during progressive relaxation training include muscle cramping, intrusive thoughts (e.g., anxiety-producing thoughts, sexual arousal), strange and unfamiliar feelings (e.g., disorientation; sensation of floating; feelings of warmth, tingling, or coolness), and a sense of losing control (Bernstein, Borkovec, & Hazlett-Stevens, 2000). Bernstein et al. recommend strategies for addressing each of these variables that may impede progressive relaxation training (see Appendix A for a list of these strategies).
Statement of the Problem

Tardive dyskinesia is a difficult to treat, potentially irreversible movement disorder that is caused by exposure to neuroleptic medications. Tardive dyskinetic movements are uncomfortable and disfiguring (Guthrie, 2002; Tandon & Jibson, 2002). Consequently, TD is frequently associated with shame, guilt, anger, depression (Margolese et al., 2005), social isolation, stigma (Correll, Leucht, & Kane, 2004; Kane, 2001; Oosthuizen et al., 2003; Tandon & Jibson, 2002), and poor employment prospects (Kane, 2001; Tandon et al., 2000). It is also associated with increased mortality (Tandon & Jibson, 2002) and suicidality in schizophrenia (Margolese, Chouinard, Walters-Larach, & Beauclair, 2001, as cited in Chouinard, 2004 a). TD may render a person’s speech unintelligible; cause respiratory distress, falls (Margolese et al., 2005), and musculoskeletal pain (Schoonderwoerd, 2005); make it difficult to retain dentures and eat (Fabbrini, Barbanti, & Aurilia, 2001); and impede rehabilitation (Kane, 2001; Tandon, Kasper, Kane, & Juncos, 2000). This disorder has doubled in prevalence over the past 20 years despite the introduction and widespread use of atypical agents (Soares-Weiser & Fernandez, 2007). TD most definitely continues to present a significant problem to those who are being treated with neuroleptic medications.

Research Question

The purpose of this study was to determine whether a specific behavioral intervention, progressive relaxation training, would result in a reduction in the severity of dyskinetic movements in persons diagnosed with neuroleptic-induced TD. The study
addressed the following research question: Does progressive relaxation training have any effect on TD that is manifested in the orofacial region? It was hypothesized that progressive relaxation training would indeed result in a decrease in severity of TD symptomatology as evidenced by a reduction in the rate of occurrence of orofacial movements in the study participants.

Significance of the Study

Pharmacological, neurosurgical, and behavioral strategies for treating TD were briefly discussed in this chapter. Many of the drugs used for treating TD are associated with significant side effects. Moreover, evidence to support the efficacy of pharmacological interventions is both lacking and of poor quality (Soares-Weiser & Fernandez, 2007). Reports of successful neurosurgical treatment of TD are rare. Neurosurgical procedures are both costly and associated with the risk of very serious side effects; therefore, this option should only be considered for severe and disabling TD that is resistant to treatment. Behavioral interventions, in contrast to pharmacological and neurosurgical treatments, have been found to be effective in decreasing the severity of TD without the risk of significant side effects.

Numerous studies have been conducted on pharmacological treatments for TD (see, for example, the review by Soares-Weiser & Fernandez, 2007), while only a minimal amount of research has been conducted on behavioral interventions for treating this disorder. An extensive search of the literature that was conducted for this dissertation resulted in only nine studies pertaining to behavioral treatment for TD and three studies
pertaining to behavioral treatment for movement disorders that are similar to TD. These studies, the most recent of which was published in 1997, are reviewed in detail in the following chapter. Procedural and methodological shortcomings that posed threats to both internal and external validity were found in most of the studies that were reviewed. In addition, many of the researchers utilized subjective rather than objective methods for measuring the dependent variable (i.e., severity of TD) and did not assess the reliability of the data.

When designing the current study, an attempt was made to address the shortcomings of earlier research pertaining to behavioral management of TD. The objectives of this study were to (a) augment the existing body of research pertaining to behavioral treatment for TD, (b) utilize an experimental design that will provide for a higher degree of control against threats to internal validity than much of the previous research conducted in this area, and (c) lay the groundwork for the development of a behavioral treatment protocol for managing TD.

Definition of Terms

Affective Flattening: Diminished, or absence, of emotional response to a situation or condition.

Akathisia: A condition associated with exposure to antipsychotic medications that is characterized by subjective complaints of restlessness and an irresistible need to move. Akathisia typically manifests as pacing, rocking from foot to foot, fidgety movements of the legs, and an inability to sit or stand still. Irritability, fear and anger, impatience,
unbearable inner torment, and an inability to concentrate may sometimes accompany these symptoms.

**Alogia:** A general lack of spontaneous content in conversation.

**Anhedonia:** An inability to experience pleasure from activities that are normally pleasurable.

**Anticholinergic Medications:** Agents that block parasympathetic nerve impulses. These medications are often used to treat extrapyramidal side effects caused by exposure to antipsychotic medications.

**Antipsychotic Medications:** Medications used primarily in the treatment of psychoses.

**Apathy:** A state of indifference or lack of emotion.

**Athetoid:** Slow, irregular, twisting, snakelike involuntary movements.

**Atypical Antipsychotic Medications:** Antipsychotic agents developed and released since 1990. Atypical antipsychotic drugs are associated with a lower risk for causing EPS and TD than conventional agents.

**Avolition:** A general lack of initiative or motivation.

**Biofeedback:** An operant conditioning technique that uses instrumentation to record, amplify, and immediately feed back information in the form of a signal (typically a tone or a light) to a trainee regarding the strength of a subtle physiological response over which he or she seeks to gain control (e.g., blood pressure, muscle contractions, heart rate).

**Buccal Cavity:** The cavity between the jaws and the cheeks.
Chorea: Repetitive, jerky, irregular, relatively rapid involuntary movements involving the limbs or facial muscles.

Conventional, or Traditional, Antipsychotic Medications: Antipsychotic agents developed and released prior to 1990. Conventional antipsychotic drugs are associated with a higher risk for causing EPS and TD than atypical agents.

Dementia Agitans: A degenerative disorder of the central nervous system characterized by tremor and impaired muscular coordination.

Dementia with Lewy Bodies: A type of dementia characterized by progressive cognitive decline, in combination with the following three features: (1) marked variability in alertness and attention (e.g., frequent drowsiness, lethargy, lengthy periods of time spent staring into space, or disorganized speech), (2) recurring visual hallucinations, and (3) parkinsonian motor symptoms (e.g., rigidity and loss of spontaneous movement).

Dopamine: A neurotransmitter involved in arousal levels and motor activity that is thought to play a major role in schizophrenia (i.e., drugs that inhibit dopamine reduce the symptoms of schizophrenia, while drugs that enhance the action of dopamine can induce symptoms similar to those of schizophrenia).

Dopamine Antagonists: Chemical agents that inhibit the action of dopamine by blocking dopamine receptors.

Dyskinesia: Abnormal involuntary movements characterized by spasmodic or repetitive motions or lack of coordination that typically manifest in the extremities, trunk, or jaw.

Dystonia: Prolonged involuntary positioning or spastic contraction of the muscles that may cause twisting of body parts, repetitive movements, and increased muscular tone.
**Electromyographic (EMG) Biofeedback Training:** Biofeedback training that uses an electromyogram, i.e., an instrument that measures the electrical activity of muscles. EMG biofeedback uses electrodes placed on the skin to generate a feedback signal (e.g., a tone or a light) in response to muscle activation so that trainees can learn to gain control over muscular tension.

**Extrapyramidal Symptoms (EPS):** A set of side effects associated with the use of antipsychotic medications and other dopamine antagonists. EPS can manifest as unpleasant sensations of restlessness, involuntary movements, uncontrollable muscular contractions, muscular rigidity, slowed movement, or tremor. EPS include Parkinsonism, akathisia, dystonia, and tardive dyskinesia.

**Neuroleptic Medications:** Antipsychotic medications.

**Momentary Time Sampling Data Recording:** A method for measuring behavior in which a specified behavior is scored as either occurring (“+”) or not occurring (“—”) depending on whether it is occurring at the end of brief observation intervals.

**Neurotransmitter:** Chemicals released from nerve cells that enable them to transmit impulses to one another.

**Orofacial Dyskinesia:** Abnormal involuntary movements involving the mouth and face (e.g., licking, chewing, lip smacking, tongue protrusion, grimacing).

**Progressive Relaxation Training:** Progressive relaxation training is a process for teaching a person to relax each of the major muscle groups in his or her body in a systematic fashion (i.e., one muscle group at a time).
**Psychosis:** A disorder characterized by severe loss of contact with reality that manifests as delusions, hallucinations, disorganized speech, and bizarre or catatonic behavior.

**Psychotropic Drugs:** Drugs used for treating psychosis, depression, mania, anxiety, and behavioral problems (e.g., aggression, self-injurious behavior, destructive behavior).

**Receptors:** Sites on cells that receive neurotransmitters.

**Serotonin:** A neurotransmitter that plays an important role in sleep-wake cycles, obsessive-compulsive behaviors, depression, and eating.

**Serotonin Dopamine Antagonist:** Chemical agents that inhibit the action of serotonin by blocking serotonin receptors.

**Stereotypic movements:** Non-goal-directed rhythmic, repetitive, coordinated, and patterned movements, postures, or vocalizations that are executed almost identically during each repetition (e.g., head banging, grimacing, finger waving, body rocking, snorting, and groaning).

**Tardive Dyskinesia:** A disorder characterized by repetitive, involuntary, purposeless movements that manifest as a side effect of exposure to dopamine antagonists (e.g., neuroleptic medications).

**Torticollis:** A painful condition in which a person experiences recurrent but transient contractions of the muscles in the neck, which cause the head to twist or turn to one side and the chin to be elevated and turned toward the opposite side.
CHAPTER TWO
LITERATURE REVIEW

“Tardive dyskinesia remains an enigmatic phenomenon…It will constitute a challenge for basic and clinical researchers for generations to come.”

Wolf, Yassa, & Llorca, 1997, p. 7

In the previous chapter, TD was identified as a significant and serious side effect associated with exposure to neuroleptic medications for which no treatment has been found to be completely satisfactory or effective. This chapter provides a critical review of the literature pertaining to behavioral interventions for managing TD. Several methods of assessing the severity of TD were used in these studies; concerns regarding diagnosis of the disorder were also raised. For this reason, this chapter begins with brief reviews on diagnosis and assessment of TD.

The literature cited in this review was obtained from extensive searches conducted on the Google Scholar electronic database and on the Alt HealthWatch, Academic Search Alumni Edition, MEDLINE, PsychINFO, and PsycARTICLES electronic databases accessed through the University of Arizona Library EBSCOhost Web Research Databases. Literature reviewed included the following: articles in peer-reviewed scholarly/professional journals, government reports, dissertations, books, and chapters in edited books. Articles accessed through on-line sources were also reviewed. Additional articles and books were obtained from searches conducted on the University
Diagnosis of Tardive Dyskinesia

TD is generally defined as a disorder of involuntary abnormal movements that manifests following exposure to neuroleptic medications over a period of at least 3 months (or 1 month if age 60 years or older), that cannot be attributed to other conditions (e.g., Huntington’s disease, Sydenham’s chorea, hyperthyroidism) (DSM-IV-TR, 2000; Llorca, Chereau, Bayle, & Lancon, 2002). Although these criteria may appear straightforward, accurate diagnosis of the disorder can be problematic due to the following factors, which should be considered in a differential diagnosis:

1. A number of medical, neurological, and physiological conditions, such as Huntington's disease (an inherited disease of the central nervous system that causes chronic progressive chorea and mental deterioration), Wilson's disease (a hereditary syndrome characterized by degenerative changes in the brain, cirrhosis of the liver, enlargement of the spleen, tremor, muscular rigidity, involuntary movements, spastic contractures, psychic disturbances, dysphagia, progressive weakness, and emaciation), hyperthyroidism, and poorly fitting dentures may produce involuntary movements that are indistinguishable from those caused by neuroleptics (Brasic & Bronson, 2007; DSM-IV-TR, 2000).
2. Dyskinetic movements that are topographically similar to TD may appear spontaneously, with no apparent cause, in persons who are elderly (Kane, 2000). The prevalence of spontaneous dyskinesias in this population may be as high as 5% (Kane & Smith, 1982).

3. Abnormal movements similar to TD were reported in individuals with psychosis long before neuroleptic medications were introduced (Kraepelin, 1919; Richard, O’Brien, & Kurlan, 2005; Turner, 1989). Terms such as grimace, fidget, jerky, and twitching were frequently used descriptors in the casebooks of Ticehurst House Asylum in Sussex England between 1850 and 1889; the majority of the movements were manifested in the arms and face (Turner, 1989).

4. Stereotypic behaviors—rhythmic, repetitive, coordinated, and patterned movements, postures, or vocalizations that are executed almost identically during each repetition—are common among persons with mental retardation and autism (Richard et al., 2005; Shulman, Sanchez-Ramos, & Weiner, 1996). Stereotypic movements may be difficult to distinguish from TD. In one study (Meiselas et al., 1989), for example, experienced raters were unable to reliably differentiate stereotypic behaviors from neuroleptic-induced TD when viewing videotapes of children with autism. The raters were blind to the children’s exposure, if any, to neuroleptic medications.

As noted in the previous chapter, the onset of TD may occur following short-term exposure to neuroleptic agents; there is no fundamental distinction between early and late onset cases (Skidmore et al., 2005). In addition, TD symptoms can appear for the first time or increase in severity when neuroleptic treatment is discontinued or when there is a
reduction in neuroleptic dose (Dixon et al., 1993; Keltner & Folks, 2005). This phenomenon is referred to as withdrawal dyskinesia (Keltner & Folks, 2005). Because withdrawal dyskinesia only appears when neuroleptic agents are withdrawn or reduced in dosage, it is apparent that in some cases neuroleptics can effectively mask the symptoms of TD (Kane, Woerner, & Lieberman, 1988).

Tardive Dyskinesia Assessment

*Observational Rating Scales*

Direct observational rating scales are the standard means of assessing TD in clinical research (Kane, 2000). The Abnormal Involuntary Movement Scale (AIMS) (Brasic et al., 2007; Glazer & Saltz, 2006; Wonodi, Hong, Avila, & Thaker, 2005), developed by the Psychopharmacology Research Branch of the National Institute of Mental Health (Guy, 1976) is the most frequently used observational rating scale. The AIMS is a multi-item rating scale designed to record the presence and assess the severity of abnormal movements in seven areas of the body: muscles of facial expression; lips and perioral area; jaw; tongue; upper extremities; lower extremities; and neck, shoulders, and hips. Severity is rated according to a five-point scale: 0 = none, normal; 1 = minimal (may be extreme normal); 2 = mild; 3 = moderate; and 4 = severe (Guy). The AIMS is popular largely because it is straightforward and easy to use (Cassaday, Thaker, Summerfelt, & Tamminga, 1997).

A variety of observational rating scales have been developed in addition to the AIMS. Among these are the Rockland Simpson Tardive Dyskinesia Rating Scale
(Simpson & Angus, 1970), the Barnes Scale (Barnes & Trauer, 1982), the Maryland Psychiatric Research Center Involuntary Movement Scale (MPRC) (Cassaday et al., 1997), the Dyskinesia Identification System Condensed User Scale (DISCUS) (Sprague, Kalachnik, & Shaw, 1989), and the Extrapyramidal Symptom Rating Scale (ESRS) (Chouinard, Ross-Chouinard, Annable, & Jones, 1980). Although multi-item observational rating scales continue to be widely used, they do have shortcomings.

According to Kane (2000), the following problems are associated with using observational rating scales for assessing TD: (a) clear distinctions often are not made regarding the determination of abnormal versus normal movements (e.g., infrequent tongue protrusion or lip movements may not represent TD); (b) the observed severity of movements could be based on frequency, duration, quality, or intensity and there is no clear means of integrating these aspects; and (c) there are no established criteria for TD that could be used to demonstrate the concurrent validity of these scales. As noted earlier in this chapter, special populations such as persons with serious mental illness, older people, persons with mental retardation, and persons with autism may present particular challenges in assessing involuntary movements (e.g., spontaneous dyskinesia), underscoring the need for comprehensive training of raters. Further, according to Lohr and Caligiuri (1992), it is often difficult to use subjective procedures such as rating scales to assess TD because people tend to suppress involuntary movements in clinical settings.
Other Methods for Assessing TD

Frequency counts and instrumentation are the two most frequently used alternatives to multi-item rating scales for assessing TD (Kane, 2000). Frequency counts provide the number of abnormal movements that occur within a given time frame. The procedure is straightforward, can be repeated numerous times, does not require rater training, and needs no equipment other than an instrument for measuring the passage of time (e.g., a stopwatch) (Gardos, Cole, & La Brie, 1977). In addition, data obtained from frequency counts are easy to handle statistically (Gardos, Cole, & La Brie). This method of assessment, however, does have some limitations. A frequency count can be difficult to perform when multiple movements are present (Kane, 2000) or if movements shift from one area of the body to another (Gardos, Cole, & La Brie). The overall severity of the disorder with regard to the magnitude of the movements and the body areas involved may not be accurately reflected by a frequency count (Kane, 2000). Finally, it is difficult to compare individuals who have different types of movements when using this method of assessment (Kane, 2000).

Instrumentation (e.g., electromyography, ultrasound, accelerometers, force gauges, position transducers, digital image processing, and polygraph) has been utilized in an attempt to provide a more objective and precise means of assessing dyskinetic movements (Caligiuri, 1997; Caligiuri et al., 1997; Gardos, Cole, & La Brie, 1977; Gattaz & Büchel, 1993; Jus, Jus, & Villeneuve, 1973; Kane, 2000; Lohr & Caligiuri, 1992). A designation of “mild” or “severe” on an observational rating scale, for example, is subjective and based on the experience of the rater; whereas, when instrumentation is
used, the criteria for severity is explicitly defined and movement abnormalities are measured as a continuous variable on an interval scale (Caligiuri, 1997; Caligiuri et al., 1997). Often, it is difficult to distinguish the presence of coexisting motor disorders in the same area of the body (Lohr & Caligiuri); however, some instrumental techniques are able to differentiate TD from other simultaneously occurring movement disorders, such as neuroleptic-induced parkinsonism (Caligiuri, Lohr, Bracha, & Jeste, 1991; Wirshing, Freidenberg, Cummings, & Bartzokis, 1989). Instrumentation may also facilitate identification of early or subclinical cases of TD (Kane, 2000; Lohr & Caligiuri).

Instrumental procedures for assessing TD are associated with a number of limitations. These include the following: (a) the necessary data processing equipment and technical support may not be readily available; (b) some instrumental techniques, such as ultrasound, may be quite expensive; (c) procedures such as electromyography may be invasive, difficult to administer, and produce variable findings with repeated measures; (d) some individuals, such as persons with dementia or persons with paranoia, may be unable to participate in studies that involve instrumentation; and (e) instrumental assessments provide no information about the functional significance of an involuntary movement, a potentially important outcome variable in treatment studies (Caligiuri, 1997; Caligiuri et al., 1997).
A Critical Review of Studies Pertaining to Behavioral Approaches for Treating Tardive Dyskinesia

A review of the literature pertaining to behavioral treatment of neuroleptic-induced TD revealed nine reports in which behavioral interventions were used to manage involuntary movements associated with TD. Three additional reports were found in which dyskinetic movements not associated with exposure to neuroleptic medications were successfully managed through the use of behavioral techniques. These additional studies are included in this review because the involuntary movements targeted for intervention were topographically similar to TD (i.e., spontaneous orofacial dyskinesia, L-dopa-induced dyskinesia, spasmodic torticollis, and dystonia). The most recent of the 12 reports reviewed in this section was published in 1997, 11 years prior to the present study.

It is customary in the rehabilitation literature to refer to individuals who participate in research as “consumers” or “participants.” In the majority of the reports reviewed below, however, research participants are referred to as “patients” because they were receiving treatment in in-patient settings and/or from medical personnel. For the purpose of this study, participants will be referred to as patients in the reviews of the reports wherein they were identified in this manner.

Biofeedback

The majority of the published reports on behavioral treatment of TD pertain to research on electromyographic (EMG) biofeedback training (Abrams, 1986; Albanese &
Biofeedback training is an operant conditioning technique that uses instrumentation to record, amplify, and immediately feed back information in the form of a signal (typically a tone or a light) to a trainee regarding the strength of a subtle physiological response over which he or she seeks to gain control (e.g., blood pressure, muscle contractions, heart rate). When treating dyskinetic movements, a trainee learns to control the intensity of the biofeedback signal in order to develop the ability to control the amount of tension in the affected muscles necessary to manage the involuntary movements.

Farrar (1976), a dentist, published the first report in which a behavioral intervention (i.e., EMG biofeedback training) was used to treat dyskinetic movements. The patient, a 51-year-old tugboat captain, developed an orofacial dyskinesia following the extraction of his teeth and subsequent treatment with complete dentures. Farrar described the movements as “severe dystonic muscular contractions of the face, lips, neck, tongue, and jaws” (p. 385). The patient’s speech was described as “stammering” and it was further noted that he “was apprehensive and obviously emotionally disturbed” (p. 385). No history of antipsychotic drug use was noted. The patient had been unable to perform his occupational duties for 2 months as a result of the dyskinetic movements.

During the initial phase of treatment, the patient’s dentures were modified and an isometric exercise regimen was initiated. Within 3 weeks, the patient’s jaws and tongue were restored to more normal function and he was able to return to full-time employment. Tremors persisted, however, in the muscles of the tongue, lip, jaws, and neck. Additional
methods of treatment including drugs, speech therapy, and other exercises were attempted without success.

After a period of approximately 2 years, during which there was no further improvement in the patient’s condition, the decision was made to treat the dyskinetic movements with EMG biofeedback. Testing was initially performed with advanced clinical-type biofeedback equipment and a small portable feedback unit. The portable unit was found to be satisfactory to monitor the hyperkinetic muscles. The portable device, which was equipped with a small earplug and electrodes, was small enough to be carried in the patient’s shirt pocket. The electrode elements were situated so as to monitor the electrical activity of the chin and mouth muscles; a tone was emitted when the involved muscles contracted. The patient received training regarding use of the device and was instructed to use it for 2 hours each day.

A significant reduction in orofacial movements was noted when the patient returned following 3 weeks of treatment with the portable biofeedback unit. Dystonic movements in the muscles of the face and lips were nearly absent, the patient’s speech was improved, and there was “a marked change” in his personality. The patient was reportedly “elated about his progress” and “no longer apprehensive about his condition.” While these results are encouraging, the causal nature of the relationship between the dependent and independent variables was not firmly established due to the pre-experimental characteristics of the research design that was used. The absence of baseline data, which would have provided a standard for evaluating the effect of the intervention on the dependent variable; subjective, rather than objective, measurement of the
dependent variable; and the lack of control over extraneous variables (inclusion of baseline and withdrawal conditions would have provided sufficient control), weakened the level of both the reliability of the measurements and the validity of the results that were obtained.

Korein et al. (1976) reported on the use of sensory feedback therapy (i.e., biofeedback) to treat 48 patients with spasmodic torticollis and 7 patients with dystonia. Spasmodic torticollis, also referred to as cervical dystonia, is a painful condition in which one experiences recurrent but transient contractions of the muscles in the neck, causing the head to twist or turn to one side. Dystonias are slow and sustained involuntary muscle contractions that cause repetitive movements and often twisting of body parts.

Potential contributing factors to the dystonias were obtained from less than half of the patients. These included a family history of torticollis, or “head tremor;” perinatal factors (e.g., difficulties in maternal pregnancy or birth); infection; viral illness; and trauma to the head or neck region. Almost all of the patients had received various forms of physical and/or drug therapy. These therapies were found to have been either ineffective or moderately effective.

Prior to treatment, baseline neurological and physiatric examinations were conducted to determine the characteristics of the movement abnormality and the state of the muscles involved. EMG recordings were obtained by means of surface electrodes placed on the involved muscle groups. Recordings were made while the patient performed various head movements. Videotape recordings and/or still photographs were taken with the patient performing the same movements. This documentation was
established on all patients so that the clinical examinations and physiological and video recordings could be repeated following 8 to 12 weeks of treatment to permit before and after comparison of the patient’s condition.

Biofeedback sessions were approximately 45 minutes in length with intermittent rest periods and were scheduled from three to five times per week. Therapeutic goals included relaxation of the involved spasmodic muscles followed by strengthening of the atrophied muscles. Patients were given a course of home exercises as they progressed with therapy. The goals of the home exercise regimen were relaxation of the spasmodic muscles and isometric contraction of the atrophied muscles.

Significant improvement was noted in 32 (58%) of the patients after receiving 8 to 12 weeks of treatment. This included return to work, improvement of social or marital life, and return of the ability to drive a car. Eighteen patients were able to control the position of their head to varying degrees and duration during feedback sessions; however, there was little to no carryover and no significant change in activities of daily living. Five patients experienced “virtually no change whatsoever.” In four of these patients, failure to improve was attributed to the following conditions: (a) leukemia and organic mental syndrome, (b) cervical root and bilateral accessory nerve section, (c) long-standing torticollis for 35 years with severe fixed arthritic changes, and (d) severe long-standing torticollis.

Thirty of the 32 patients that experienced significant improvement were followed from 3 months to up to 3 years. Regression to baseline conditions occurred in 8 of these
patients after discontinuation of feedback therapy (27%). This represents an overall improvement in 40% of the entire group (n = 55).

This study is noteworthy because it is one of the few pertaining to behavioral treatment of dyskinetic movements that used a large number of participants. Further, EMG recordings provided an objective measurement of the dyskinetic movements. The internal validity of the study was compromised, however, because the researchers chose to use a quasi-experimental design (i.e., one-group pretest posttest design). Without a control group, it is not possible to rule out that differences observed between pre- and post-intervention clinical examinations and physiological and video recordings were solely a function of the biofeedback training and not a function of some other variable(s).

Szekeley, Turner, and Jacob (1982) reported on the use of meditation to treat dyskinesia in a 44-year-old male graduate student with idiopathic Parkinson’s disease. The dyskinetic movements, which appeared following an increase in dosage of Sinemet (i.e., L-dopa), manifested as 180° lateral head turning with simultaneous lateral jerking of the left arm and left leg. Each of these movements occurred from 20 to 30 times per minute. Episodes of dyskinetic movements lasted from 1 to 6 hours, with periods of cessation ranging from a few minutes up to 2 hours. The jerking interfered with eating, dressing, and shaving, made reading and writing “virtually impossible,” and “grossly impaired” his gait. The patient had to suspend his graduate studies due to his inability to read. In addition, the patient had episodes of “severe” contraction headaches which were followed by stiffness and fatigue.
During the initial phase (i.e., baseline) of this study, the patient self-monitored the severity (scale of 0 – 10) and duration of episodes of left extremity jerking. Exploratory monitoring of EMG levels in various muscle groups was initiated during the second week in preparation for biofeedback therapy. At that time, a decrease in the dyskinetic movements was noted. When questioned, the patient reported that he had been meditating which he described as “concentration, focusing on breathing, and thinking of nothingness” (p. 445). Consequently, the decision was made to focus on developing the patient’s meditation strategy rather than switching to biofeedback.

The patient practiced meditation twice a day for 30-minute periods throughout the second phase of the study. He was seen four times by the researchers for exploratory EMG monitoring, observation of his dyskinesia, and discussion of progress during this 18-day period. The patient decreased the dosage of his medication without consulting with the researchers during this phase. The lower dose failed to adequately control his Parkinson symptoms.

During the third phase of the study, the patient continued meditation twice daily and gradually increased the Sinemet dose back to what it had been during Phase 1. He was seen twice during this 30-day period and then discharged. The researchers report that the level of dyskinesia was “remarkably lower” in Phase 3 than it had been during Phase 1. In addition, the average severity of the movements decreased to a rating of approximately 1 and the average duration of the episodes decreased to under one-half hour. The patient’s headaches and muscle soreness subsided and he was able to read without difficulty and resume his graduate studies.
The patient was seen again 4 months later because the dyskinetic movements returned after he stopped meditating regularly. During the fourth phase, meditation was reintroduced and treatment with Sinemet was maintained. The movements gradually decreased over a 3 week period to levels similar to those observed during the third phase.

This study is particularly interesting because, by ceasing meditation following the third phase, the patient in effect initiated a withdrawal condition. Replication of results obtained during the third phase, following the reintroduction of meditation during the fourth phase, suggests that the intervention (i.e., meditation) was responsible for the decrease in dyskinetic movements. A stronger argument regarding this effect would have been possible if the authors had used an objective and reliable method of measuring the dyskinetic movements rather than patient self-monitoring. In closing, Szekely et al. point out that the meditation technique practiced by this patient is in actuality a form of relaxation training and maintain that it is safe to assume that other types of relaxation therapy would be “equally effective.”

Albanese and Gaarder (1977) were the first to report on the effects of EMG biofeedback training specifically for treating neuroleptic-induced TD. Their paper provided brief case reports on 2 “well-motivated” male patients: a 39-year-old stockroom manager who experienced an episode of acute paranoid psychosis shortly after his wife divorced him and a 51-year-old farmer who had experienced several episodes of psychotic depressive illness as an adult. Both patients manifested severe orofacial dyskinesias following treatment with traditional neuroleptic medications (the stockroom
manager had been treated with trifluoperazine and fluphenazine decanoate; the farmer had been treated with fluphenazine decanoate).

Each patient was evaluated for biofeedback training one month after discontinuation of neuroleptic treatment. Although he was not manifesting any psychotic symptoms, the first patient (i.e., the stockroom manager) continued to engage in “almost constant contorting and chewing movements of the mouth, some foot tapping, and slight choreiform movements of the fingers” (p. 1149). The second patient engaged in “constant severe chewing movements and rhythmic protrusion of the tongue” (p. 1150); no movements were manifested in his extremities.

Albanese and Gaarder worked directly with both patients on masseter movement using EEG feedback (the masseters are the muscles that close the mouth). Input from the muscle was fed to an electronic instrument that provided the patients with three types of feedback: (a) a sound that increased in pitch with increasing muscle tension, (b) a threshold-controlled beep-like sound that signaled the same input, and (c) visual feedback provided by a meter that was in front of the patients. The patients were instructed to eliminate the beep-like sound, reduce the sound pitch level, and lower the meter reading. They were also encouraged to take note of the sensation in the masseter muscle when they successfully achieved the same feeling (i.e., a reduction in tension) while at home.

Treatment sessions, provided once a week, were 45 minutes in duration. Significant improvement was observed in both patients within 3 weeks. The first patient received 10 training sessions and was “totally without dyskinetic symptoms” during a 3-month follow-up session. No follow-up data are available for the second patient because
his wife became ill and the family moved out of town following his ninth session. Albanese and Gaarder report, however, that during the second patient’s final session, the only abnormal movement manifested was of the tongue within the closed mouth. When interpreting these results, it should be noted that the functional relationship between the dependent and independent variables was not adequately demonstrated and that the reliability of the data was not established. Baseline data were not reported for either patient, there was no indication regarding how Albanese and Gaarder measured the dependent variable, and there was inadequate control for threats to internal validity.

Sherman (1979) reported on the use of EMG biofeedback to control TD in a 44-year-old white male with a history of prolonged chlorpromazine use and over 20 years of chronic alcohol and multiple drug abuse. The patient had a 5-year history of TD that did not abate during a 6-month pre-hospitalization period; he stopped using chlorpromazine during this time. A precise description of the dyskinetic movements was not provided; however, Sherman did report that the patient had orofacial dyskinesia that manifested as random movements of the tongue, mouth, and jaw. He also had frequent tension headaches and muscle tremors in his hands that were only apparent when he was upset.

The patient received treatment while he was on a Veterans Hospital’s Alcohol Rehabilitation Unit; treatment consisted of two independent phases. He was initially given EMG feedback from the frontalis (muscles situated in the forehead) two times a week in the laboratory. EMG feedback was visually displayed on a meter. The patient was also instructed to use progressive muscle relaxation exercises, similar to Jacobson’s (i.e., a procedure during which the trainee is instructed to tense and then relax specific
muscle groups throughout the body and to observe the difference in the sensations of both conditions) when he was on the ward and at home. These procedures were implemented to reduce the patient’s anxiety and to assure him that he could control his physiological responses to anxiety (e.g., muscle tension and tension headaches). The frontalis EMG decreased from 23 microvolts at the end of the first session to seven by the end of the fourth session (average plus or minus 2 microvolts); the patient was able to sustain this low level for 2 months. By the end of the first phase the following subjective indications of improvement were noted: (a) improved interaction with other patients and non-treatment staff, (b) reduction of hand tremors when tense, and (c) elimination of the tension headaches. There was no evidence of improvement in the patient’s TD, however.

The second treatment phase was initiated after the patient had left the program. During this phase, the patient received five 20-minute sessions of auditory feedback training of masseter activity; no absolute measures of masseter tension were made. The patient was also instructed to use his newly acquired skill of muscle tension awareness outside of the laboratory so as to familiarize himself with the movements of his mouth. The jaw movements, which gradually decreased following the second session of feedback from the masseter, were completely absent by the fourth session. Two months after the second phase was initiated, the patient reported that the movements were “entirely absent” outside of the laboratory setting. The dyskinetic symptoms were not apparent at a 15-month follow-up session. The patient reported that feedback from the masseter had increased his awareness of his jaw movements, which enabled him to control the movements as soon as they started. These results are promising; however, as in the
studies reported by Farrar (1976) and Albanese and Gaarder (1977), Sherman did not use an objective and reliable method for measuring masseter movements (i.e., severity of the dyskinetic movements was based on the patient’s self-report). In addition, no baseline data were collected on the dependent variable and Sherman did not provide sufficient control for threats to internal validity.

Abrams (1986) studied the effect of treatment with EMG biofeedback of the masseter muscles on the severity of orofacial TD in 8 psychiatric inpatients. Abrams noted that all of the participants were maintained on psychotropic medication throughout the study. She acknowledged that the severity of the TD ratings may have been reduced due to medication-induced masking of TD symptomatology. Participants were determined to have TD if they received an overall rating of one or greater on the Simpson-Angus Scale (an observational rating scale) where symptoms included orofacial movements. Abrams reported that an “attempt” was made to match participants according to age, diagnosis, severity of TD, and length of medication use. She did not clearly report on how she did this nor on whether her attempt was successful.

Three pre-treatment sessions were conducted for each participant during the first 3 weeks of the study. The Simpson-Angus Scale was administered during the first week and ratings were recorded for a single pre-treatment measure. Dyskinetic movements were monitored by an EMG recording of the masseter for a period of 10 minutes during each pre-treatment session to obtain a total of three measures. Post-treatment measurements were collected in the same manner during the 3 weeks following the final treatment session. It should be noted that Abrams used both an objective and a subjective
method for measuring the dependent variable in order to enhance the validity and reliability of the study. She argued that objective methods for measuring TD (e.g., EMG recordings) have “better reliability but less certain validity,” while subjective methods for measuring TD (e.g., observational rating scales) “have the highest validity,” but “require the demonstration of reliability.” Abram’s hypothesized that (a) Simpson-Angus scores and EMG recording of masseter muscle measures would be significantly positively correlated, (b) there would be a significant decrease in participants’ orofacial dyskinetic movements after receiving biofeedback training as measured by EMG recording of masseter muscle activity, and (c) there would be a significant decrease in participants’ orofacial dyskinetic movements after receiving biofeedback training as measured by the Simpson-Angus Scale.

Treatment with biofeedback was initiated during the fourth week. Each participant received two 30-minute training sessions per week over an 8 week period. There were three separate treatment components: (a) relaxation training, using a protocol developed by Lazarus (a procedure involving tensing and relaxing of various muscle groups, similar to Jacobson’s method described above), (b) training to recognize sensations associated with the onset of muscular contraction, and (c) training in the use of deep muscle relaxation strategies to inhibit involuntary movements. According to Abrams, participants in this study “presented varying degrees of psychotic pathology—delusions and hallucinations” that interfered with their ability to utilize the biofeedback equipment. Consequently, the training procedure was modified to alleviate confusion
caused by the auditory feedback signal: participants were provided with verbal feedback from the researcher, instead of receiving direct feedback from the biofeedback apparatus.

An analysis of the data revealed that the correlation between Simpson-Angus scores and EMG measures was not statistically significant and that neither the Simpson-Angus scores nor the EMG measures revealed significant decreases in participants’ orofacial dyskinetic movements following the biofeedback training. Significant reductions in EMG measures were observed, however, during the training sessions; these effects did not carry over into subsequent trials. Abrams hypothesized that the participants were unable to maintain learning from one trial to the next because they were “highly dependent” on the researcher’s cues and the structured biofeedback training procedure. She maintained that the dependence on structure and cuing demonstrated an attribute of psychotic behavior: “schizophrenics have difficulty in focusing on relevant stimuli and in sustaining efficient performance” (p. 67).

Abrams used a one-group pretest-posttest design was used for this study. This type of pre-experimental design is noted for having poor internal validity because changes in the dependent variable may be attributable to history, maturation, instrumentation, or testing (Patten, 2000). The external validity of this study is also weak, due to the small sample size. In addition, Abrams did not provide interobserver agreement (i.e., interrater reliability) data on the Simpson-Angus Scale measures. This is surprising, given she correctly noted that subjective measures “require the demonstration of reliability.” A good argument could be made that the EMG recordings by themselves were sufficient for measuring the dependent variable (i.e., objective, precise, high level of
reliability and validity) and that it was not necessary to use the Simpson-Angus Scale at all (see *Assessment of Tardive Dyskinesia* earlier in this chapter). It is indeed fortunate that EEG recordings were collected throughout the course of the study. Without these recordings, Abrams would not have been able to determine that there were reductions in EMG measures within the biofeedback training sessions.

Cotton (1986) reported on the use of progressive relaxation training and EMG biofeedback to control neuroleptic-induced TD in a 26-year-old male with schizophrenia. Onset of the dyskinetic movements had occurred 5 ½ years prior to the study, after 2 years of neuroleptic treatment. Cotton described the participant’s involuntary movements as “obvious and severe choreiform movements in his fingers, wrists, and arms” (p. 4) that were also apparent to a lesser degree in his lower extremities. Orofacial movements (i.e., chewing and coordinated tongue and mouth movements), although not pronounced, were also present.

Upper extremity movements interfered with the participant’s ability to resume employment as an automobile body worker. For this reason, EMG biofeedback training on the forearm extensor muscle was initiated. An A – B – BC – B – BC design was used, in which A was the baseline phase, B the progressive relaxation training phase, and BC the combined progressive relaxation training – biofeedback training phase. A series of six sessions were provided in each phase. Cotton did not use a withdrawal design due to the potential for the effects from the relaxation training to persist during the withdrawal phase(s). The participant maintained the following medication regimen throughout the
course of the study: perphenazine (8 mg., b.i.d.), cogentin (2 mg., b.i.d.), and halcion (0.25 mg q.h.s.).

During the baseline phase, the participant was seated for approximately 15 minutes with the biofeedback electrodes attached to the forearm extensor muscle; feedback was not provided. EMG measurements were then recorded for a period of 5 minutes. After the EMG measurements were obtained, the participant rated the severity of the movements using a 10-point scale (1 = total absence of movements and 10 = most severe the movements had ever been). During the progressive relaxation training phases, the participant received a 15-minute progressive relaxation training session following Bernstein and Borkovec’s protocol (1973), which is similar to Jacobson’s technique described above. As in the baseline phase, the electrodes were attached but the participant received no feedback. The progressive relaxation training session was followed by 5 minutes of EMG recording and another participant rating. During the progressive relaxation training – biofeedback phases, the participant was again seated with the electrodes attached. Progressive relaxation training with feedback was implemented for approximately 10 minutes, after which the participant received 5 minutes of auditory feedback by means of variable rate clicking sounds. The progressive relaxation training – biofeedback session was followed by 5 minutes of EMG recording and a participant rating.

An analysis of the data revealed an immediate decrease in EMG level and self-rating of the dyskinetic movements upon implementation of progressive relaxation training. This change was maintained across all phases, leading Abrams to conclude “Use
of progressive relaxation training procedures, with or without the addition of EMG biofeedback appears to result in a decrease in choreiform movements” (p. 7). No difference was reflected in the participant’s self-ratings of the two conditions, although he reportedly tended to prefer the progressive relaxation training with biofeedback condition over progressive relaxation training alone. The participant received six “refresher” EMG sessions, at his request, during the 7 months following training. While the hand movements were never completely absent, the participant was able to find employment doing automobile body work after 5 months. He reported that he occasionally had to stop and make a concerted effort to relax in order to control the movements, but that doing so did not significantly interfere with his performance at work.

EMG level recordings provided an objective means for measuring the participant’s dyskinetic movements. The inclusion of baseline measures and continuous assessment as well as the relatively marked decrease in EMG levels upon initiating progressive relaxation training suggest that progressive relaxation training may have been responsible for the changes that were observed. It is quite possible, however, that some effects of progressive relaxation training carried over into the progressive relaxation training-biofeedback condition (i.e., multiple treatment interference). The effect of the latter condition on the dependent variable, therefore, cannot be adequately evaluated. It should also be noted that a functional relationship between the dependent variable and the independent variables cannot be firmly established because Cotton was not able to include a withdrawal phase (Richards et al., 1999).
Fudge et al. (1991) reported on the efficacy of EMG feedback training in reducing the frequency and intensity of oral-lingual movements in 20 adult male inpatients who were diagnosed as having neuroleptic-induced TD. Inclusion criteria for the study were: (a) a 9-month history of TD, (b) a minimum of 2 years of neuroleptic treatment, and (c) AIMS scores indicating at least mild TD in two of four AIMS areas involving facial movements or a single AIMS score indicating moderate TD in one of those areas. The AIMS was administered prior to training to verify inclusion criteria and then weekly during the course of the study to monitor TD severity. The AIMS raters were calibrated with one another during the baseline rating for each participant and then at weekly intervals. Patients were excluded from participating in the study if they had a concurrent neurological disorder, severe dental abnormalities that might mask or contribute to dyskinetic symptoms, or unable to cooperate due to severe dementia. The ages of the participants ranged from 45 to 72 years.

Prior to initiating the biofeedback phase of the study, neuroleptic and anticholinergic (i.e., agents used to treat EPS) medications were systematically reduced in order to establish the lowest therapeutic dose for each participant. Anticholinergic medications were eliminated for all but one participant; all participants were receiving one neuroleptic medication for the duration of the study. Participants were stabilized for a minimum of one month on their lowest doses of medications before feedback training was initiated. They were then randomly assigned to one of two groups—“true feedback” or “false feedback”—with ten participants per group.
Training sessions, given on 10 consecutive days, were 14 minutes in duration. EMG activity in the genioglossus (a muscle involved in tongue movements), masseter, and frontalis muscles was recorded during the sessions. Feedback for the “true feedback” group (i.e., experimental group) was presented contingent on an increase in genioglossus activity, whereas feedback for the “false feedback” group (i.e., control group) was computer generated at random intervals (i.e., noncontingent feedback). Participants in the “false feedback” group received identical instructions as those given to the “true feedback” group. This study was conducted under double-blind conditions—feedback contingencies were unknown to the research technician and the participants.

An analysis of the EMG recordings indicated that a “modest” reduction in oral dyskinetic movements was produced within sessions with contingent feedback training. This effect was specific to the genioglossus; a similar suppression of EMG activity was not shown in masseter or frontalis activity. These results were further supported by a statistical analysis in which significant differences were found between the true and false feedback groups. The authors attributed these results to specific training effects because (a) the study was conducted under tightly controlled conditions (i.e., double-blind with placebo), (b) participants were randomly assigned to the treatment groups, and (c) there were a sufficient number of participants to support a statistical analysis. The weekly AIMS scores did not differ significantly between the true and false feedback groups.

In a subsequent review of this study, Fudge and Sison (1997) reported that both groups’ AIMS scores were identical with pre-training scores at 1 week and 2 weeks after the training. They noted that biofeedback training effects often fail to persist beyond the
clinical setting and suggested that an individualized training program might be more
effective for TD suppression than the “quite rigorous approach” taken in their study. Had
Fudge and his colleagues instructed participants to practice the skills they acquired during
biofeedback training, as Albanese and Gaarder (1977) and Sherman (1979) did, treatment
effects may have persisted.

This study is noteworthy for the following reasons: (a) EMG recordings provided
an objective means for measuring the dependent variable; (b) the AIMS raters were
calibrated with one another, thereby increasing the reliability of the AIMS scores and; (c)
although the researchers used a convenience sample, the study was conducted under tight
double-blind conditions and the sample was large enough to support a statistical analysis.
It should be noted that Fudge et al. (1991) acknowledged the following weaknesses they
encountered with the AIMS: (a) differences were observed between the severity of
movements during examination and those seen while participants were engaged in
everyday activities (i.e., questionable concurrent validity), and (b) the instrument’s
sensitivity to extremely low-amplitude movements or those that were long in duration
was poor. For these reasons, the authors questioned the reliability of the AIMS as a
measure of TD severity for behavioral approaches to treatment.

Additional Behavioral Approaches

Published reports on the use of biofeedback training outnumber those pertaining
to other behavioral approaches for treating TD and related movement disorders.
Additional behavioral methods, however, have been shown to be effective in reducing
dyskinetic movements. Following is a review of several reports on management of TD using behavioral strategies that have not been discussed thus far in this report.

*Overcorrection and positive/negative feedback.* Taylor, Zlutnick, and Hoehle (1979) used a multiple treatments design to study the effects of several behavioral procedures on orofacial dyskinesia in a 66-year-old male and on head-bobbing movements in a 66-year-old female. Both participants had a prolonged history of neuroleptic use. The male participant received three separate sessions over 3 consecutive weeks. All three sessions included an initial 10-minute baseline phase, during which the participant was seated alone in the observation room and instructed that the therapist would be in to see him in a few minutes. Instructions, positive and negative feedback, overcorrection strategies, and a final withdrawal condition were implemented during the first session (A – B – C – D – A). During the instructions condition, the participant was reminded intermittently that the therapist wanted to see how well he could hold his jaw still. Under the positive and negative feedback condition, the participant received praise from the therapist provided he did not move his jaw for a period of 10 seconds (i.e., “You haven’t moved your jaw again, very good.”). When he did move his jaw, the participant was immediately told “You’re moving your jaw again.” During implementation of the overcorrection condition, the participant was instructed to exaggerate specific dyskinetic movements (i.e., overcorrection) for a period of 10-seconds following their involuntary occurrence. The session concluded with a withdrawal condition.
The instructions condition “had little apparent effect” on the dependent variable. Consequently, the protocol was modified and implemented as follows during the remaining two sessions: (a) the instructions condition was omitted, (b) overcorrection and positive and negative feedback were combined, (c) a self-control condition was added, and (d) a withdrawal condition was implemented following the self-control condition (A – B – C – A). During the overcorrection/positive and negative feedback phase, the participant was informed that the therapist would ask him to engage in overcorrection when the jaw movements occurred. If the jaw movements were absent for a period of 10 minutes, the participant received verbal reinforcement (i.e., “Very good.”) from the therapist. Following the overcorrection/positive and negative feedback condition, the participant was instructed to try to control the movements on his own (i.e., self-control condition) while the therapist left the room and observed him for 10 minutes through an observation mirror. The self-control condition was followed by a final baseline phase.

The female participant received two sessions, during which positive and negative feedback, self-control, and generalization strategies were implemented; overcorrection was not implemented. The positive and negative feedback and self-control methods were implemented as they were for the first participant, although head bobbing movements, rather than jaw movements, were targeted for intervention. During the generalization phase of each session, the therapist told the participant that the experiment was finished and instructed her to sit in the waiting room until her taxi arrived. The occurrence of head bobbing was then recorded by an observer who was located at a secretary’s desk in the waiting room. It should be noted that the generalization condition was quite similar to the
baseline condition in that the participant was left alone and then surreptitiously observed. The participant’s dyskinetic movements returned to baseline levels upon implementation of this phase during both sessions.

Analysis of the data revealed that implementation of overcorrection and positive and negative feedback strategies resulted in a reduction of dyskinetic movements. The inclusion of baseline measures and withdrawal conditions, continuous measurement of the dependent variable across all phases, and marked decreases in the frequency of dyskinetic movements suggest that the intervention (i.e., overcorrection and positive and negative feedback) was indeed responsible for the observed treatment effects. A significant shortcoming of alternating treatments designs, however, is the possibility that effects of one or more of the treatments may carry over into subsequent phases (i.e., multiple treatment interference), thereby weakening both the internal and external validity of the study (Barlow and Hersen, 1984; Morgan & Morgan, 2009; Richards et al., 1999). Finally, frequency counts, which were performed by under-graduate psychology students trained in behavioral observation, provided an objective and reliable means for measuring the dependent variable. Reliability ranged from 91.0% to 100% for all observations (M = 96.4%).

**Binary feedback, self-monitoring, and videotape feedback.** Frederiksen and Rosenbaum (1979) evaluated the effectiveness of binary feedback, self-monitoring, and videotape feedback in suppressing neuroleptic-induced orofacial dyskinesia in 2 psychiatric patients at a VA hospital. One of the participants, a 63-year-old male,
“exhibited sucking and licking movements of his mouth and tongue” (p. 300). The other participant, a 56-year-old male, “displayed marked tongue protrusions and lip smacking” (p. 300).

The researchers chose to use withdrawal designs for each participant (A – B – A – C – A – D – A and A – B – C – D – A, respectively). In addition to the baseline and withdrawal phases, the following fixed length experimental phases were implemented: self-monitoring, videotape feedback, and binary feedback. These phases were implemented in sequential order differing across participants. During baseline and withdrawal phases, the participants were instructed to make themselves comfortable. The target responses were then videotaped. No instruction was provided regarding inhibiting or controlling the dyskinetic movements during the baseline phases. During the self-monitoring phase, participants received and were shown how to use a reciprocating hand-held counter at the beginning of the session. They were given definitions of the target responses (i.e., tongue protrusion and lip sucking) and then instructed to record each occurrence of either target response or the combined behavioral chain (i.e., tongue protrusions and lip sucking). Participants were also instructed to use self-monitoring to aid them with inhibiting the occurrence of the movements. During the videotape feedback phase, participants were told that they could simultaneously view themselves on a television monitor as they were being videotaped. They were then instructed to watch the monitor and use the videotape feedback to aid them in suppressing dyskinetic movements. Finally, during the binary feedback phase, participants attempted to suppress buccolingual movements through the use of a digital timing device that stopped at the
onset of dyskinetic movements and resumed only when the movements were terminated. Participants were instructed to keep the timer moving as long as possible.

Binary feedback and self-monitoring resulted in “marked decreases” in dyskinetic movements for both participants, while videotape feedback resulted in a reduction in movements for only one of the participants. Frequency counts provided a means for objectively measuring the dyskinetic movements. Each occurrence of tongue protrusion, lip sucking, or the combined behavioral chain was regarded as one response. Reliability ranged from 69% to 100% (M = 89.7%) over 18 checks obtained for the first participant, while reliability ranged from 81% to 100% (M = 91.6%) over 16 checks obtained for the second participant. Frederiksen and Rosenbaum likely included withdrawal phases in order to control for multiple treatment interference. Inclusion of withdrawal phases in single subject designs with multiple treatments, however, will not eliminate the possibility of multiple treatment interference (Kazdin, 1982).

Visual feedback/TV monitor and discreet-discrete prompting. Jackson, Schonfeld, and Griffith (1983) evaluated the effectiveness of two behavioral treatments on decreasing orofacial TD in a 62-year-old female client at a community aging program. The dyskinetic movements “consisted of frequent lateral movements to either the left or right in addition to a forward puckering of the lips” (p. 548). An alternating treatments design was used for this study. The researchers recorded the frequency of mouth movements per 10-second interval. Interobserver reliability was checked at least once during each phase of the study. Reliability ranged from 90% to 100% over all checks.
The following conditions were implemented during the course of this study: group activity setting baseline, TV monitor baseline, visual feedback/TV monitor, self-control, discreet-discrete prompting (DD prompting), and generalization and maintenance. The participant was engaged in a group activity with 8 to 10 other community aging program clients during the group activity setting baseline condition. During the TV monitor baseline condition, she watched a daily scheduled program on the TV monitor. The participant did not receive any feedback or instructions to inhibit mouth movements during the TV monitor baseline condition. During the visual feedback/TV monitor condition, the participant received verbal instructions at 5-minute intervals to observe the area around her mouth on a television monitor and to hold it “as still as possible.” The participant was instructed to keep her mouth still while viewing a daily television program on the monitor during the self-control condition. During the DD prompting condition, a portable electronic prompting device delivered an "unobtrusive" .5 second duration tone at 10 second intervals while the participant viewed a daily television program. The tone was presented to remind the participant to hold her mouth still. Finally, during the generalization and maintenance procedure condition, the participant was given a prompting card that provided instructions for her to (a) practice holding her mouth as still as possible whenever she looked in the mirror and (b) practice holding her mouth as still as possible for approximately 1 minute whenever a person spoke to her. The participant was told to keep the card with her at all times and to practice the instructions that were printed on it.
Analysis of the data indicates that visual feedback and DD prompting were both effective in decreasing the frequency of the participant’s orofacial movements and that the generalization and maintenance condition resulted in generalization of the treatment effects outside of the treatment environment. While frequency counts provided an objective means for measuring the dependent variable, the possibility of multiple treatment interference cannot be ruled out because an alternating treatments design was utilized for this study. In addition, Jackson, Schonfeld, and Griffith (1983) correctly point out that further research with additional participants is needed to determine the extent to which the results of this study may be generalized.

*Progressive relaxation training and covert reinforcer sampling.* Wisoki (1993) reported on the successful use of progressive relaxation training and covert reinforcer sampling to decrease neuroleptic-induced orofacial dyskinetic movements that manifested as “excessive tongue-thrusting movements, occurring, on average, 26 times per minute.” The participant, Mary, a 77-year-old female resident of a nursing home, “complained bitterly” about the movements. She avoided contact with her family and avoided participating in activities in the community because she was embarrassed by the movements. Mary had been institutionalized from time-to-time for “manic depressive psychosis” during the 40 years prior to her admittance to the nursing home. She had been given phenothiazines (i.e., traditional neuroleptic medications), as needed, to manage anxiety since arriving at the nursing home; however, she had not taken any neuroleptic agents for at least 6 months prior to beginning the study. The following medications were
given to Mary throughout the course of this study: lithium carbonate for depression, Sinequan for sleep at night, and placebos three times per day for “nerves.”

A withdrawal design with changing conditions was used for this study (A – B – C – D – A – B – D). During the 2-week baseline phase, a total of nine frequency counts of tongue-thrusting movements were taken each day during three 5-minute intervals in the morning, the afternoon, and the evening. Mary was unaware of the therapist’s presence during two of the three frequency counts in each interval; however, she joined the therapist in recording the dyskinetic movements during the third count. The therapist was a 29-year-old female registered nurse who had been working at the nursing home for one and a half years prior to the study. Frequency counts were taken throughout the course of the study, which occurred over an 11-week period.

The following treatment conditions were implemented during this study: self-control, relaxation training, and covert reinforcer sampling. The 1-week self-control instructions phase was implemented in the presence of the therapist during the second week of the study. Mary was instructed to “try her best to relax her tongue” during each of the nine 5-minute intervals throughout the day during this phase. The purpose of the self-control instructions condition was to ascertain the extent to which Mary could exercise voluntary control over the dyskinetic movements and the degree to which she would respond to “motivating instructions.” The therapist taught Mary a modified Jacobsonian relaxation procedure and practiced it with her for 30 minutes twice each day during the third and fourth weeks. The relaxation exercises involved the muscles of the
entire body with particular attention focused on relaxation of the facial and tongue muscles.

A covert reinforcer sampling phase was implemented during the 6th and 7th weeks of the study. After practicing the relaxation exercises, Mary was asked to imagine a pleasurable scene chosen from a list that she had prepared previously (e.g., a view of the mountains in the fall, sitting at the beach, a trip to her son’s home). The therapist provided a continuous narrative of the scene, integrating sensory material (i.e., taste, smell, touch, and sound imagery) as she did so, with particular attention directed on relaxing the tongue. Mary was instructed to practice the covert reinforcer sampling scenes at least twice a day. At the conclusion of each covert reinforcer sampling session, Mary was instructed to keep her tongue still for as long as possible for a 5-minute period of time. This condition was included to demonstrate to Mary that she had some degree of voluntary control over the dyskinetic movements. It also provided the researchers with a comparative measure of the movements obtained when Mary was not distracted.

A withdrawal phase was implemented during weeks 8 and 9. Mary was instructed to practice the relaxation and covert reinforcer sampling exercises by herself at least twice a day throughout this phase. During the 10th week, a self-control practice phase was implemented. Mary was asked to keep her tongue still for as long as she possibly could during this condition which was implemented twice each day. Finally, covert reinforcer sampling was re-implemented without the relaxation practice during the last (i.e., 11th) week of the study.
An analysis of the data obtained during this study revealed that progressive muscle relaxation training and covert reinforcer sampling both resulted in substantial decreases in tongue-thrusting movements. During the initial baseline phase of this study, Mary typically experienced 130 tongue-thrusting movements per 5-minute interval. She averaged less than 40 tongue-thrusting movements per 5-minute interval at the conclusion of the study. Mary reported that the techniques were very effective and that she felt substantially less agitated. She became more actively involved in her personal development, requested physical therapy for an old hip injury, started to talk about playing the piano again, and arranged to leave the nursing home to meet her daughter-in-law for lunch. Unfortunately, 2 months after the study concluded, Mary reported that she was growing weary of having to work to keep her tongue still and asked for medication as an “instant cure.” In spite of having been informed of the results of the study, the attending physician granted Mary’s request and increased the dosage of phenothiazines. Although the medication was initially effective, within several months Mary was again experiencing the effects of TD.

In her discussion regarding the results of this study, Wisoki acknowledged that medications often produce rapid effects, whereas behavioral interventions require consistent and often intense work to produce gradual change over time. For this reason, clients are “often unwilling” to practice behavioral techniques regardless of the degree to which a behavior may be causing them problems. According to Wisoki, if the treatment program had been designed so as to address this problem, maintenance may have been achieved. Finally, while frequency counts of tongue-thrusting movements provided an
objective means for measuring the dependent variable, the possibility of multiple treatment interference cannot be discounted because several interventions were implemented during this study.

Summary

TD is a condition that is caused by long-term exposure to antipsychotic medications. Diagnosis of TD, however, is not straightforward and requires the elimination of other conditions that may also produce dyskinetic movements. Dyskinesias may appear spontaneously in persons who are elderly or persons with psychiatric conditions, such as schizophrenia, whether or not they have a history of taking antipsychotic medications. Many persons with developmental disabilities and autism engage in stereotypic behaviors, such as grimacing, that may be difficult to distinguish from TD. In addition, a number of physiological, neurological, and medical conditions may produce dyskinetic movements that are indistinguishable from TD (e.g., Huntington’s disease, Sydenham’s chorea, and hyperthyroidism).

Assessment of Severity

Assessing the severity of TD presents challenges as well. Observational rating scales are the most widely-used instruments for assessing TD. These scales are straightforward and easy to use; however, the ratings are subjective in nature and based upon the experience of the rater. Observational rating scales do not provide a means for distinguishing between abnormal versus normal movements and stereotypic versus
dyskinetic movements. They also do not indicate whether the observed severity of movements is based on frequency, duration, quality, or intensity or on a combination of any two or more of these measures.

Frequency counts and instrumentation provide an objective alternative means of assessing TD. Frequency counts are clear-cut in procedure and the data can be easily analyzed statistically. They can be difficult to conduct, however, when multiple movements are present or when movements shift from one area of the body to another. In addition, frequency counts may not accurately reflect the overall magnitude of the movements or the areas of the body that are involved. It is also difficult to compare individuals who manifest different types of dyskinetic movements with one another when frequency counts are used.

Instrumentation provides perhaps the most accurate and objective means for measuring TD, although it does have its limitations. Equipment and technical support, both of which can be very expensive, need to be readily available. Some procedures may be difficult to administer and invasive and may produce variable findings with repeated measures. Some individuals may not be able to participate in studies involving instrumentation due to confusion caused by some aspect of the procedure (e.g., a feedback tone) or by the invasive nature of the equipment (e.g., electrodes attached to the skin over the affected muscles). Finally, instrumental procedures do not provide information about the functional significance of a movement disorder.
Treatment

No treatment for TD has been found to be completely satisfactory or effective. Thus, prevention is critical to effective management of this condition. Limiting exposure to conventional antipsychotic medications through the use of atypical agents is the primary means of preventing TD. The use of conventional agents is recommended only when atypical agents fail to produce intended results, when patients cannot tolerate atypical agents, or when patients express a preference for conventional agents. Withdrawing the offending agent should be considered when dyskinetic movements first begin to manifest, provided antipsychotic treatment is not regarded as necessary.

Approaches to managing TD symptoms can be generally classified into the following categories: (a) pharmacological, (b) neurosurgical, and (c) behavioral. Of these three approaches, pharmacological interventions remain the preferred treatment for managing TD, although there is insufficient evidence to recommend this approach, and many of the pharmacological agents used for treating TD are themselves associated with serious side effects. Neurosurgical interventions for treating TD should only be considered as a last resort for those patients with severe disabling TD that is resistant to treatment. Neurosurgical treatment is costly and is associated with the potential for very serious side effects. Behavioral interventions, on the other hand, have been shown to be effective in managing TD symptomatology without the risk of causing the serious side effects associated with pharmacological and surgical interventions.
Summary of the Research Pertaining to Behavioral Interventions for Treating Tardive Dyskinesia

Nine reports of studies pertaining to behavioral interventions for treating TD were found and reviewed. Three additional reports pertaining to behavioral treatment of dyskinetic movements that were not neuroleptic-induced were found and reviewed because the movements were topographically similar to TD. In a number of these studies, methodological limitations make it difficult to adequately evaluate the effect of the interventions. These include (a) lack of baseline measures of the dependent variable (Albanese & Gaarder, 1977; Farrar, 1976; Sherman, 1979), (b) use of subjective methods for measuring the dependent variable (Albanese & Gaarder, 1977; Farrar, 1976; Sherman, 1979; Szekeley et al., 1982), (c) inadequate or weak control for threats to internal and external validity (Abrams, 1986; Albanese & Gaarder, 1977; Cotton, 1986; Farrar, 1976; Frederiksen & Rosenbaum, 1979; Jackson et al., 1983; Korein et al., 1976; Sherman, 1979; Szekeley et al., 1982; Wisoki, 1993), and (d) failure to assess the reliability of the data (Abrams, 1986; Albanese & Gaarder, 1977; Farrar, 1976; Sherman, 1979; Szekeley et al., 1982; Wisoki, 1993).

Most of the studies on behavioral treatment of TD were conducted using one or two participants. The internal validity of several of these studies (Albanese & Gaarder, 1977; Farrar, 1976; Sherman, 1979) would have been stronger if (a) objective and reliable methods of measurement had been used, (b) baseline measures had been collected and reported, and (c) a withdrawal condition had been incorporated into the study design. The treatment integrity of the study conducted by Szekeley et al. (1982)
was compromised by the participant (i.e., initiating meditation during the baseline phase, discontinuing medications, and discontinuing meditation). The possibility of multiple treatment interference presented a significant threat to the internal validity of several studies that evaluated the effectiveness of more than one intervention (Cotton, 1986; Frederiksen & Rosenbaum, 1979; Jackson, et al., 1983; Taylor et al, 1979; Wisoki, 1993).

According to Kazden (1982), multiple treatment interference is a “major issue” of multiple treatment designs. Although there are advantages to using multiple treatment designs (e.g., a withdrawal condition is not necessary, baseline data may not be required, and alternative treatments can be compared within a relatively short period of time), the possibility that the effects of a particular condition may be altered in some manner by a condition(s) that preceded it cannot be discounted (Kazden). It is interesting to note that Frederiksen and Rosenbaum included withdrawal phases prior to implementing each new intervention. This practice, however, does not eliminate the possibility of multiple treatment interference (Kazden). It should be noted that multiple treatment interference also poses a major threat to the external validity of a study because the results obtained may only apply to individuals who receive the intervention(s) either in the same way or in the same sequence (Kazdin, 1982; Patten, 2000; Salkind, 2006).

Only three of the studies that were reviewed in this chapter used more than two participants. Korein et al. (1976) used the largest sample of participants (n = 48); however, the absence of a control group makes it impossible to rule out that changes observed in the dependent variable may have been a function of some factor(s) other than the intervention. Eight psychiatric in-patients participated in the study conducted by
Abrams. The external validity of this study was weak due to the small sample size. The internal validity was also weak because Abrams used a pre-experimental design (i.e., one-group pretest-posttest design). Furthermore, participants received verbal feedback from Abrams on the dependent variable instead of receiving it from the biofeedback equipment. This modification of the typical biofeedback training procedure may have produced different results than those that would have been obtained if the participants had received direct feedback from the biofeedback apparatus. Finally, Fudge et al. (1991) conducted the most tightly controlled study among those that were reviewed. Participants were randomly assigned to experimental and control groups, there was a significant number of participants to support a statistical analysis \((n = 20)\), objective and reliable methods were used to measure the dependent variable (i.e., EMG recordings and AIMS raters that were calibrated with one another), and the study was conducted under double-blind with placebo conditions.

The studies reviewed above have their limitations, some of which are significant. The results obtained from them, however, suggest that behavioral interventions have consistently produced a positive effect on TD symptomatology. Progressive relaxation training was a component of Abrams’ (1986) and Sherman’s (1979) treatment protocols, although the effect of the training on TD was not specifically examined. Cotton (1986) and Wisoki (1993) did, however, examine the effects of relaxation training and found that the training resulted in a reduction in the severity of dyskinetic movements. Szekely et al. (1982) examined the effect of meditation, a form of relaxation training, on L-dopa-
induced dyskinesia and reported on improvements that were observed upon its implementation.

Among the various behavioral techniques for treating TD that have been studied, biofeedback training has received the most attention. It should be noted, as Cotton (1986) correctly pointed out, that EMG biofeedback training is frequently used as a relaxation technique. Indeed, most of the studies that examined the effect of biofeedback training on TD focused the training on reducing muscle tension and relaxing specific muscles that were involved in producing the dyskinetic movements. As reported above, results obtained from Cotton’s study indicate that EMG biofeedback was no more effective than progressive relaxation training alone. Moreover, during the second week of implementing the progressive muscle relaxation phase in Wisoki’s study, the rate of the participant’s tongue-thrusting movements was essentially equal to that observed during the final week of the study. The progressive muscle relaxation phase was implemented prior to implementing the covert reinforcer sampling condition in Wisoki’s study.

Progressive relaxation training was not examined by itself in any of the studies that were reviewed in this chapter. It was, however, studied either as a component of specialized intervention protocols or in comparison to one or more other interventions in single-subject studies. It is interesting that results obtained from these studies suggest that the severity of TD symptomatology was reduced in those participants who received progressive relaxation training. Advantages of progressive relaxation training over biofeedback training include the following: (a) special equipment (e.g., a biofeedback unit) is not required; (b) it is not necessary to attach anything, such as electrodes, to the
participants; (c) feedback signals, which can be confusing to some individuals, are not used (see for example the review of Abram’s study earlier in this chapter); and (d) participants can practice the techniques between sessions without assistance. Given these considerations, the effect of progressive relaxation training on TD symptomatology merits further scrutiny than it has received thus far. When conducting this research, every attempt was made to avoid the methodological limitations that have compromised the reliability and validity of the results obtained from previous studies pertaining to behavioral management of TD.
CHAPTER THREE
METHODOLOGY

This chapter provides a description of the procedures that were used in the collection and analysis of the data for this study. Included in this chapter are a description of the participants and setting in which the study was conducted; an operational definition of the dependent variable; and descriptions of the independent variable, the method used for measuring the dependent variable, the means of collecting data on the dependent variable, the environment in which the intervention was implemented, the study design and procedure, and the method used for data analysis. Also included is a discussion regarding the methods used for collecting interobserver and treatment integrity data.

Setting and Participants

This study was conducted in a county-owned nursing home and outpatient facility in Southern Arizona. Specialty care (e.g., behavioral health care, traumatic brain injury care, neurological care, ventilator and respiratory care, wound care, and total parenteral nutrition care) is provided at the facility, primarily to persons who are enrolled in the Arizona Long-Term Care System (ALTCS), Arizona's capitated Medicaid program for long-term care beneficiaries. This particular site was chosen because a number of older people reside there who have been receiving antipsychotic medications for many years to treat serious mental illness. As noted earlier, advanced age, exposure to conventional antipsychotics, and extended duration of antipsychotic treatment have been identified as
increasing the risk for developing TD. A number of the residents at this facility meet all of these criteria.

The study participants were selected from the residents at the nursing home. Each participant had been diagnosed with having neuroleptic-induced TD prior to the study. The facility’s Medical Director of Behavioral Health Services assisted with the recruitment process by identifying potential participants and arranging for them to meet with the principal investigator (PI) of the study. No information pertaining to potential participants was disclosed to the PI during the recruitment process. Once they had been identified, the PI met individually with each of the potential participants to obtain their informed consent (See Appendix B for the Informed Consent document) and their permission to review their nursing home medical records (see Appendix C for the Participant Authorization for Use and Disclosure of Protected Health Information for Research document). The PI reviewed both consenting documents with the potential participants and answered questions pertaining to the study prior to obtaining their signatures.

The participants ranged in age from 51 to 72 years; all had been receiving antipsychotic medications, including traditional agents, for a number of years. Complete medical records were not available, so it was not possible to determine the participants’ length of exposure to neuroleptics. Information regarding the participants’ ages, psychiatric diagnoses, and medication regimen at the time the study was being conducted was obtained by a review of records provided by the nursing home.
Participant 1

Participant 1 was a 72-year-old male who had received the following diagnoses: dementia with Lewy bodies, dementia with behavioral disturbance, paralysis agitans (i.e., Parkinson’s disease), bipolar disorder unspecified, and depressive disorder. At the time of this study, Participant 1 was receiving the following psychotropic medications: lorazepam (Ativan, an anxiolytic medication), carbidopa-levodopa (Sinemet, a medication used to treat parkinsonism), trazodone (an antidepressant medication), and quetiapine (Seroquel, an atypical antipsychotic medication). Participant 1’s orofacial dyskinesia manifested as jaw movements (i.e., slow chewing movements), thrusting of the lower lip, and tremor in the cheek and mouth muscles.

Participant 2

Participant 2 was a 51-year-old male who had received the following diagnoses: anoxic brain damage, mood disorder, intracranial injury without skull fracture, chronic meningitis, and anxiety state. At the time of this study, Participant 2 was receiving the following psychotropic medications: valproic acid (Depakote, an antiseizure medication), lithium (to treat depression), clonazepam (Klonopin, to treat anxiety), and trazodone (to treat insomnia). Participant 2’s orofacial dyskinesia manifested as movements of the jaw (i.e., chewing), tongue movements (i.e., licking the lips), eye-rolling, forehead and eyebrow movements, biting the lower lip, and lip pursing and smacking.
Participant 3

Participant 3 was a 60-year-old male who had received the following diagnoses: mental retardation, seizure disorder, schizoaffective disorder, adjustment reaction disorder, and depression. He had also been experiencing anxiety, depression, paranoia, insomnia, visual and auditory hallucinations, and suicidal ideations. At the time of this study, Participant 3 was receiving the following psychotropic medications: phenytoin (Dilantin, an antiseizure medication) and mirtazapine (Remeron, an antidepressant medication). Participant 3’s orofacial dyskinesia manifested as jaw movements (i.e., chewing and mouth opening and closing), tongue movements (primarily within the buccal cavity, particularly behind the lower lip), blinking, eye-rolling, forehead and eyebrow movements, grimacing, and lip smacking.

Dependent Variable

The dependent variable, orofacial dyskinesia, is defined as any movement involving the muscles of the face, mouth, jaw, or tongue. These movements include protrusion of the tongue; pushing of the tongue into the cheek or any other area within the oral cavity; tongue movements within the buccal cavity; licking of the lips; chewing, sucking, puckering, pursing, and smacking movements of the lips and mouth; opening and closing of the jaw; biting; forehead furrowing; eyebrow movements; grimacing; blinking; eye rolling movements; and tremors in any area of the face, mouth, jaw, or tongue.
Design and Procedure

A multiple baseline across subjects design was used to evaluate whether progressive relaxation training is effective in reducing the severity of orofacial movements in the study participants. Multiple baseline designs involve implementation of an intervention across two or more participants at different points in time. Staggering the implementation of the intervention serves to minimize alternative explanations for changes in behavior; the number of baselines contributes to the strength of the demonstration (Morgan & Morgan, 2009). Most researchers consider two or three participants to be the minimum necessary to provide an adequately convincing conclusion in a multiple-baseline design (Kazdin, 2001; Morgan & Morgan, 2009).

This single subject design was chosen over a withdrawal design for the following reasons: (a) the effects from relaxation training cannot be withdrawn or reversed (Cotton, 1986), (b) participants may have found it undesirable to withdraw the intervention due to the severity of their dyskinetic movements, and (c) the additional baselines would serve as controls against which the effects of the treatment could be assessed. In addition, an attempt was made to meet the following conditions, which are based on criteria identified by Richards et al. (1999) as being essential to the successful implementation of multiple baseline across subjects designs: (a) the participants displayed the same target behavior (i.e., orofacial dyskinesia) in the same setting (i.e., the relaxation training room), (b) the participants were similar enough to one another to expect that each would respond to the relaxation training procedure and yet not likely to respond until such time as the training was specifically implemented to treat their orofacial dyskinetic movements, (c) there was
a reasonable expectation that the same variables (i.e., independent and extraneous) would exert the same influence on each of the participants, (d) the relaxation training procedure was likely to have a similar effect on each participant, (e) a consistent recording procedure was selected for measuring orofacial movements across participants and a criterion level for initiating the intervention for each participant was identified, and (f) there was reasonable expectation that the resources would be available to maintain data collection and interventions for the duration of the study.

The participants in this study agreed to receive two sessions per day, on consecutive days, for a total of 17 sessions. Participants did, however, miss some sessions. Reasons for missed sessions are detailed in the following chapter.

Method of Measuring the Dependent Variable

The PI videotaped the participant’s faces for the duration of each session. A digital video camcorder was mounted on a tripod that was situated at a distance of approximately 4 feet and directly in front of the reclining chair that the participants were seated in during the sessions. The PI began recording as a participant was entering the room. After the participant was seated and the chair had been reclined to its lowest position, the PI adjusted the video camcorder so that the participant’s face and head was within the video frame. The PI stopped the video camcorder as the participant was leaving the room.

Following the sessions, the PI transferred the videotape data to a compact disc. The PI then reviewed the videotape data and recorded momentary time sampling data on
the dependent variable, using 10-second intervals, for a period of 5 minutes. Momentary time sampling is a time-based method of recording behavior during which an observation period is divided into discrete units of time (i.e., intervals) and the behavior is scored either as occurring (“+”) or not occurring (“—“) at the conclusion of each interval (Umbreit, Ferro, Liaupsin, & Lane, 2007). This method of measuring the dependent variable was chosen for the following reasons: (a) momentary time sampling is practical when measuring nonuniform behaviors (i.e., behaviors, such as tardive dyskinesia, that tend to vary in length) that are likely to persist for extended periods of time (Richards et al., 1999; Sulzer-Azaroff & Mayer, 1991; Umbreit et al., 2007) and (b) momentary time samples using short intervals tend to more accurately depict the rate of occurrence of frequently occurring behaviors (Sulzer-Azaroff & Mayer, 1991).

**Relaxation Training Room**

The relaxation training sessions were conducted in a 12 foot by 17 foot room located within the nursing home’s Activity Room. This room is used by staff and by residents for group therapy sessions and meetings. The room is also used for storing a few folding chairs and office chairs as well as three televisions and videocassette recorders that are situated on stands with wheels. A reclining chair was centrally located in the room for the participants to sit in during the sessions. The PI sat in a straight chair that was located to the right of the recliner. A compact disc player was placed on a folding chair to the immediate left of the PI. The compact disc player was used for playing a recorded surf sound during the progressive relaxation training sessions (see
Relaxation Training Protocol, Appendix D). A single floor lamp, positioned against a wall directly behind the PI, provided lighting during the sessions. Finally, a long rectangular table was located along the wall of the room to the left of the recliner and a hutch was located against the wall behind the recliner.

**Baseline Phase**

During the baseline condition, the participants were asked by the PI to enter the training room and to sit on the reclining chair. Once the participant was seated, the PI slowly reclined the chair and then instructed the participant to make himself comfortable while the video camera was being adjusted. Once the camera was adjusted, the PI sat down in the chair to the right of the recliner and then asked the participant if he was comfortable. The session began when the participant indicated that he was comfortable. During the session, the PI conversed with the participant for an average of 15 minutes and then asked the participant to sit quietly for 5 minutes so that baseline data could be collected on the dependent variable. After the 5 minute baseline data collection period, the PI instructed the participant that the session was over, returned the reclining chair to an upright position, thanked the participant, and then escorted him to the door of the training room.

**Intervention Phase**

Progressive relaxation training was initiated for the first participant after a stable rate of occurrence of orofacial movements had been obtained across three baseline
sessions. During the intervention condition, the PI asked the participant to enter the training room and sit on the reclining chair. The PI slowly reclined the chair and then instructed the participant to make himself comfortable while the video camera was being adjusted. Once the camera was adjusted, the PI sat down in the chair to the right of the recliner and then asked the participant if he was comfortable. The progressive relaxation training procedure was initiated after the participant indicated that he was comfortable. The protocol used for this study was adapted from a procedure developed by Ferreira (1994) (see Appendix D for the Progressive Relaxation Training Protocol). Specific emphasis was placed on relaxing facial and tongue muscles during the relaxation training session. Sessions were approximately 35 minutes in duration.

Participants were observed throughout each training session for the purpose of monitoring their overall relaxation responses. The PI recorded these observational data during the sessions on a Relaxation Response Monitoring Form (see Appendix E). Problems related to implementing the progressive relaxation training procedure that arose during the course of the study (e.g., laughter, talking, anxiety-producing thoughts, and external noise) were addressed according to strategies recommended by Bernstein, Borkovec, and Hazlett-Stevens (2000) (see Appendix A).

The intervention was initiated for the second participant after the first participant had achieved a downward trend in the rate of orofacial movements across four consecutive sessions. Finally, the intervention was initiated for the third participant following a downward trend in the rate of the second participant’s orofacial movements across five consecutive sessions.
Each participant was offered a compact disc recording of the progressive relaxation training procedure that was used in the study during their final session. The progressive relaxation training recording will enable participants to continue practicing the relaxation techniques if they choose to do so.

Data Collection

Baseline phase. Collection of momentary time sampling data from videotaped baseline sessions was initiated after the participants had responded to the PI’s request to sit quietly for 5 minutes so that their facial movements could be videotaped (see Appendix F for the form that was used for collecting momentary time sampling data). A response to this request was determined to have occurred after participants had sat in the recliner without talking or moving their arms, legs, or torso for a period of 30 seconds (i.e., three consecutive time sampling intervals). Momentary time sampling data on the dependent variable were recorded for a period of 5 minutes during the baseline sessions, using 10-second intervals. A MotivAider® (MotivAider, 2000) was utilized for measuring the intervals during both the baseline and the intervention phases.

Intervention phase. Collection of momentary time sampling data from videotaped intervention sessions was initiated after (a) the Relaxation Training Protocol had been implemented through step 8 (see Appendix D) and (b) following step 8, 30 seconds had elapsed during which no orofacial dyskinetic movements were observed to have occurred (i.e., three consecutive time sampling intervals). It should be noted that, when collecting
data during the intervention phases, the PI (a) started each videotape at a random point following step 8 (i.e., observation periods were not predetermined), (b) observed the videotape, and (c) initiated time sampling data collection after 30 seconds had elapsed without the appearance of dyskinetic movements. When the above criteria were not met (e.g., a participant was unable to relax his orofacial muscles for 30 seconds following step 8), data collection was initiated following implementation of step 9 of the Relaxation Training Protocol (see Appendix D). Momentary time sampling data on the dependent variable was recorded for a period of 5 minutes during the intervention sessions, using 10-second intervals.

The following procedure was employed to respect and protect the participant’s privacy during the data collection process: (a) participants were randomly assigned a numerical code (i.e., 001, 002, and 003), (b) all data were designated by the number “8,” (c) medical records data were designated by the letter “R,” (d) observational data were designated by the letter “O,” (e) videotape data were designated by the letter “V,” and (f) momentary time-sampling data were designated by the letter “T.” “003-8V,” for example, designated videotape data for Participant 3.

Data Analysis

Visual inspection of graphically displayed data is the typical means of evaluating whether the experimental criterion (i.e., determinations regarding whether the intervention is responsible for changes in the dependent variable) have been met in a single subject experiment (Kazdin, 1982). Graphically displayed momentary time
sampling data were used to determine whether the independent variable was responsible for changes observed in the rate of orofacial dyskinetic movements following the initiation of the intervention phases for each of the participants.

*Interobserver Agreement (IOA)*

A research collaborator was trained in the response definition (i.e., orofacial dyskinetic movements) by the PI. This was accomplished by reviewing the operational definition for the dependent variable, as described above, and then providing visual examples of specific dyskinetic movements from videotape data collected during the course of the study. After receiving the training, the research collaborator independently recorded momentary time sampling data on the dependent variable from the videotape data that were collected during the sessions. Videotape data for these reliability checks were selected by the collaborator after the PI had shuffled the compact discs containing the data and spread them out on a table. The collaborator was blind to specific participants and sessions during this selection process. Momentary time sampling data were collected by the collaborator on the dependent variable for one baseline session and two intervention sessions for each participant. The timing of the IOA observation periods coincided with the timing of the 5-minute observation periods during which the PI had previously collected momentary time sampling data (the PI had written the timings of the observation periods on the compact discs so that they could be located for IOA data collection). The momentary time sampling data collected by the collaborator were then compared with the PI’s momentary time sampling data from the same session. Reliability
estimates were computed by dividing the number of agreements (i.e., number of identical intervals that received the same score by the PI and the collaborator) by the total number of intervals and multiplying the result by 100% (see Appendix G for an example of IOA data collection and computation).

_Treatment Integrity_

Treatment integrity of the relaxation training procedure (i.e., the degree to which the intervention was implemented as designed) was assessed by the research collaborator for six sessions during the intervention phases. This was done to verify the functional relationship between the independent and dependent variables. Treatment integrity data were collected by having the research collaborator rate either the presence or the absence of each component of the relaxation training protocol on a treatment integrity form (see Appendix H for the form that was used to assess treatment integrity). Session integrity was computed by dividing the total number of components that were actually completed during each session by the number of components that should have been completed (i.e., 15) and multiplying the result by 100%. Component integrity was computed by dividing the total number of times each component was completed by the total number of times treatment integrity was assessed and multiplying the result by 100%.
CHAPTER FOUR

RESULTS

As mentioned in the previous chapter, visual inspection of graphically displayed data is typically employed when analyzing results obtained from single-subject research. Figure 1 graphically depicts the percentage of momentary time sampling intervals in which orofacial movements occurred during the 5-minute observation periods that were conducted within each session (Figure 1).

Rate of Orofacial Dyskinetic Movements

As shown in Figure 1, Participant 1’s orofacial dyskinetic movements occurred during 100% of the observed intervals during the baseline phase and decreased markedly during the intervention phase ($M = 25\%$; range $= 0\% - 100\%$). It should be noted that muscle tremors observed in the orofacial area were scored as dyskinetic movements because it was not possible to differentiate between parkinsonian symptomatology and TD symptomatology. Further, there was a considerable amount of noise outside of the relaxation training room throughout sessions 11 and 14 (i.e., several residents were playing cards in the Activities Room and speaking loudly during session 11; workers were testing the fire alarm system in the building during session 14). This noise could be heard clearly in the relaxation training room and may account for the higher scores obtained during those sessions. Session 17 (i.e., the final intervention session) was terminated at the request of Participant 1 because the level of sound filtering in from the
Figure 1. Percentage of Dyskinetic Movements
environment outside of the relaxation training room was quite high (a church group was performing Easter music for the residents). No data were collected for this session.

In a similar manner, the mean level of Participant 2’s orofacial dyskinetic movements decreased markedly from 97% (range = 83% - 100%) during the baseline phase to a mean level of 31% during the intervention phase (range = 0% - 90%, see Figure 1). The environment outside of the relaxation training room was quite noisy while implementing the intervention with Participant 2 during sessions 11 and 14 as well. This may have affected his response to the intervention, as evidenced by the higher scores obtained during those sessions. Participant 2 elected to withdraw from the study following session 14. He did not indicate his reason for withdrawing.

Participant 3’s orofacial dyskinetic movements occurred during 100% of the observed intervals during the baseline phase and decreased slightly to a mean level of 87% during the intervention phase (range = 43% - 100%, see Figure 1). Several factors should be considered when analyzing the data for Participant 3. These include the following: (a) Participant 3’s dyskinetic movements were more pronounced than those of the other participants; (b) Participant 3 missed a total of five baseline condition sessions after he was admitted to a psychiatric hospital for reporting that he was experiencing suicidal thoughts and auditory and visual hallucinations; and (c) when he resumed the sessions, Participant 3 continued to report he was experiencing suicidal thoughts, auditory and visual hallucinations, and that “the medications still aren’t working.” Participant 3 did insist, however, that the sessions were “helping” and indicated his desire to continue to participate in the study. It should further be noted that the magnitude of
Participant 3’s orofacial dyskinetic movements appeared to improve upon implementing the intervention. This would likely have been detected if instrumentation (e.g., EMG recordings) or an observational rating scale (e.g., the AIMS or the DISCUS) had been used for measuring the dependent variable. Momentary time sampling is not designed to measure the magnitude of a behavior.

Finally, it should be noted that none of the participants was able to sustain relaxation of the orofacial muscles (i.e., a complete absence of orofacial dyskinetic movements) for the remainder of a session once the criteria had been met for initiating data collection. Participants were often able, however, to sustain relaxation of orofacial muscles for several periods of time with a session. It was beyond the scope of this study to determine why the participants were unable to sustain orofacial muscular relaxation.

Interobserver Agreement Data

The IOA scores obtained for the Phase 1 baseline condition were: Participant 1, 100%; Participant 2, 100%, and Participant 3, 100%. The IOA scores obtained for the Phase 2 intervention condition were: Participant 1, 100% and 97% (M = 99%); Participant 2, 100% and 97% (M = 99%); and Participant 3, 100% and 80% (M = 90%).

Treatment Integrity Data

Session integrity and component integrity were maintained 100% of the time during the sessions in which treatment integrity data were obtained. The treatment integrity data are presented in Table 2.
Table 2

Treatment Integrity Data

<table>
<thead>
<tr>
<th>Observation Sessions</th>
<th>Component</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>Component Integrity</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Greeted the participant</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>2. Instructed the participant to sit in the reclining chair</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>3. Reclined the reclining chair to its lowest position</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>4. Asked the participant to make himself comfortable</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>5. Asked the participant if he was comfortable</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>6. Instructed the participant to take three deep breaths</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>7. Turned on the ocean surf recording</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>8. Instructed the participant to relax each muscle group</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>9. Repeated the procedure for relaxing each muscle group</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>10. Allowed the participant several minutes to enjoy the feeling of being relaxed</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>11. Repeated the procedure for relaxing each muscle group</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>12. Turned off surf sound and instructed the participant to take three deep breaths</td>
<td></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
</tbody>
</table>
### Table 2 (continued)

<table>
<thead>
<tr>
<th>Component</th>
<th>Observation Sessions</th>
<th>Component Integrity</th>
</tr>
</thead>
<tbody>
<tr>
<td>13. Raised the reclining chair to its upright position</td>
<td>1 1 1 1 1 1 1 100%</td>
<td></td>
</tr>
<tr>
<td>14. Asked the participant how he was feeling</td>
<td>1 1 1 1 1 1 1 100%</td>
<td></td>
</tr>
<tr>
<td>15. Accompanied or assisted the participant to the door of the training room</td>
<td>1 1 1 1 1 1 1 100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 1 1 1 1 1 1 100%</td>
<td>Overall Mean = 100%</td>
</tr>
</tbody>
</table>

**Session Integrity**

| 100% 100% 100% 100% 100% 100% | Overall Mean = 100% |

**Directions:** Write a “1” if the component was completed and a “0” if the component was not completed. Compute session and component integrity at the conclusion of the study.
CHAPTER FIVE

DISCUSSION

This chapter provides an analysis of the results presented in Chapter Four. Also included are a discussion regarding the relationship of this study to similar research, a discussion regarding the limitations of this study, recommendations based on the findings, and a discussion regarding implications for future research.

Analysis of the Results

The purpose of the present study was to determine whether progressive relaxation training would reduce the severity of dyskinetic movements in persons diagnosed with neuroleptic-induced TD. The study was specifically designed to address the following research question: Does progressive relaxation training have any effect on TD that is manifested in the orofacial region? Further, it was hypothesized that progressive relaxation training would result in a decrease in the severity of TD symptomatology as evidenced by a reduction in the rate of occurrence of orofacial movements in the study participants.

Visual inspection of Figure 2 in Chapter Four reveals that the mean rates of occurrence of orofacial dyskinetic movements decreased markedly in Participants 1 and 2 upon implementation of the intervention. The mean rate of occurrence of orofacial dyskinetic movements also decreased in Participant 3 after the intervention was introduced; however, the initial effect was not as dramatic as that observed in the other
two participants (i.e., a decrease was not observed until the fourth intervention session). Possible explanations for this variation in the response rate include the higher level of magnitude of Participant 3’s dyskinetic movements, the presence of potentially interfering cognitive stimuli (as evidenced by self-reports of suicidal ideation and visual and auditory hallucinations), and missed sessions during the baseline phase. It should be pointed out again that the magnitude of Participant 3’s orofacial dyskinetic movements appeared to improve upon implementing the intervention; it was not possible, however, to detect this change with momentary time sampling. Finally, environmental conditions, such as noise level, appeared to affect each participant's response to the intervention.

The severity of each participant’s orofacial TD improved (i.e., the rate of occurrence decreased) when the intervention was introduced. Treatment integrity data indicate that the intervention was implemented at a high level of fidelity. Likewise, IOA data reveal a high level of reliability in the data that were collected on the dependent variable during the course of the study. Therefore, a clear functional relationship was established between the independent variable (i.e., progressive relaxation training) and the dependent variable (i.e., severity of orofacial TD). These data indicate that progressive relaxation training can provide an effective means for decreasing the severity of orofacial TD.

Relationship to Similar Research

A number of the studies pertaining to behavioral management of TD that were reviewed in Chapter Two contain methodological limitations that make it difficult to
firmly establish the functional relationship between the independent and dependent variables. As noted previously, these limitations include lack of baseline measures of dependent variables, use of subjective methods for measuring dependent variables, inadequate or weak control for threats to both internal and external validity, and small sample size (i.e., most of the studies were conducted using one or two participants). Reliability of data collected on the dependent variable was only assessed in a few of the reviewed studies (i.e., Frederiksen & Rosenbaum, 1979; Jackson, Schonfeld, & Griffith, 1983; Taylor, Zlutnick, & Hoehle, 1979); treatment integrity data was not reported in any of them. In spite of these shortcomings, however, the results obtained from these studies suggest that behavioral interventions can be effective in reducing the severity of TD symptomatology.

Several types of behavioral interventions were examined in the reviewed studies (i.e., biofeedback training, meditation, progressive relaxation training, overcorrection, positive/negative feedback, binary feedback, self-monitoring, videotape feedback, visual feedback/TV monitor, discreet-discrete prompting, and covert reinforcer sampling). Among these interventions, biofeedback training, with a focus on relaxing muscles specifically involved in producing dyskinetic movements, received the most scrutiny. Progressive relaxation training, on the other hand, received far less attention and was not examined in the absence of other interventions within any given study. Rather, progressive relaxation training was studied either as a component of specialized intervention protocols or in comparison to one or more other interventions in single-subject studies (e.g., EMG biofeedback training and covert reinforcer sampling); it is not
possible to rule out the threat of multiple treatment interference on both the internal and external validity of these studies.

In the current study, an attempt was made to address the methodological limitations of previous research pertaining to behavioral treatment for TD. Use of a multiple baseline design was critical to accomplishing this task. A small sample of participants was included in this study; however, replication of the effect of the independent variable on the dependent variable during the intervention phases served to minimize alternative explanations for the observed changes. To further strengthen the internal validity of this study, baseline levels of the dependent variable were obtained for each participant, the dependent variable was monitored for each participant throughout the study, an objective method for measuring the dependent variable was utilized, criteria were established for determining the data collection periods, and reliability data were collected and assessed. Finally, the manner in which the intervention was operationalized was described in detail in Chapter Three, each component of the independent variable was specified in the Relaxation Training Protocol (see Appendix D), and treatment integrity data were collected and assessed so as to insure that the intervention was implemented as specified. Although overlooked in the studies that were reviewed for this dissertation, these latter factors are essential to establishing the scientific and applied validity of any study (Morgan & Morgan, 2009).
Limitations

The present study has several limitations. First, only three participants were included in the study. A large pool from which to recruit participants was not available due to the low incidence of TD in the general population. Replication of this study is necessary in order to determine whether the results can be extended to other persons with TD. Second, although every attempt was made to address each of the conditions that were identified in Chapter Three as being essential to implementing a successful multiple baseline across subjects design (Richards et al., 1999), it was not possible to ascertain whether they were, in fact, all met (e.g., it was not possible to determine whether participants were going to experience psychotic symptomatology during the course of the study). Third, as noted above, it was not possible to detect variations in the magnitude of the dependent variable that may have occurred during the course of the study with momentary time sampling. Further, momentary time sampling can either overestimate or underestimate the occurrence of the behavior that is being assessed (Sulzer-Azaroff & Mayer, 1991). Short intervals, however, have been found to minimize this distortion (Sulzer-Azaroff & Mayer). In the present study, 10-second intervals were used during the momentary time sampling periods in an attempt to minimize distortion. Finally, the fact that it was not possible to control extraneous variables in the environment, such as noise level, appears to have affected participant’s responses to the intervention during at least two sessions.

Although an attempt was made to standardize the data collection procedure (i.e., data collection was initiated during the baseline and intervention conditions according to
criteria specified in Chapter Three), internal validity would have been stronger if an instrumental approach had been utilized for assessing the data. Doing so would have (a) allowed for objective and continuous assessment of the dependent variable for the duration of each session, (b) either minimized or eliminated any distortion that may have occurred due to momentary time sampling, and (c) made it possible to record and assess both the rate of occurrence as well as the magnitude of the dyskinetic movements. A computer program designed to analyze dyskinetic movements from video signals would likely be the best method for instrumentally assessing orofacial TD. The means for collecting the data would be no more intrusive than the method that was utilized for collecting videotape data in the current study. Alternative methods for collecting data instrumentally (e.g., EMG recordings) typically involve attaching electrodes to the skin over affected muscles. The presence of electrodes would probably make it difficult for people to relax orofacial muscles.

A means of digital image processing of a video signal, digital movement analysis (DMA), has been examined for assessing TD (Nilsson, Hansen, Büchel, Gattaz, & Gerlach, 1996) and found to be “a useful supplement to classical TD rating [scales]” (Nilsson et al., 1996). Nisson et al. acknowledged the need for further validity evaluation of this instrument; however, no additional reports on DMA were found during a search of the electronic databases identified in Chapter Two. Additional examination of this method for instrumentally assessing TD (i.e., computer analysis of videotape data) is most definitely warranted.
Implications and Recommendations for Future Research and Practice

The results obtained during the present study appear to be similar to those obtained from previous research pertaining to behavioral management of TD in that the severity of the participant’s orofacial dyskinetic movements decreased when the independent variable was introduced. This study is significant in that progressive relaxation training was examined by itself; it was not a component of a treatment protocol, nor was it compared with other interventions in an alternating treatments design. As noted in Chapter Two, there are considerable advantages to progressive relaxation training over other behavioral interventions that have been examined for TD management (i.e., the procedure is relatively straightforward; special equipment is not needed; no sensors are attached to the trainees; feedback signals, which can be confusing to some trainees, are not used; and the techniques can be practiced by trainees between sessions without assistance). The decrease in severity of the participants’ orofacial dyskinetic movements suggests that progressive relaxation training may provide a viable option to more invasive pharmacological and surgical approaches for managing this disorder.

Although attempts were made to control the environment in which the training was implemented, noise from outside of the training room was quite audible during several sessions. Extraneous noise did appear to have a negative effect on the participants’ responses to the intervention (i.e., the severity of the dyskinetic movements increased dramatically when the level of ambient noise increased). This underscores the
importance of insuring that extraneous noise is minimized when conducting progressive relaxation training sessions.

It was beyond the scope of this study to address participant’s generalization of their responses to the intervention to situations and settings outside of the training environment. Future research should address this critical issue. A possible way of doing so would be through implementation of cue-controlled relaxation training, a procedure during which a word such as “calm” or “relax” is presented contingent upon the trainee’s relaxed response. Once established, cue words have been found to produce a relaxation response without the need to engage in the full progressive relaxation procedure (Lindsey, Fee, Michie, & Heap, 1994). This variation on the basic procedure of progressive relaxation training should be explored for TD management because it may enable individuals to self-manage dyskinetic movements outside of the clinical environment.

Finally, in addition to orofacial dyskinetic symptomatology, Participant 1 also experienced a persistent tremor in the orofacial area due to Parkinson’s disease. This tremor was scored as a dyskinetic movement when it occurred during the observation sessions. It is noteworthy that the tremor was observed to subside during implementation of the intervention. This finding suggests that progressive relaxation training should be examined with regard to management of parkinsonian symptomatology.
Conclusion

Neuroleptic medications have greatly influenced psychiatric care and treatment since their introduction in the 1950s. The fact that neuroleptics may, and often do, cause a number of significant side effects is the downside to their use as therapeutic agents. Tardive dyskinesia, a potentially irreversible abnormal involuntary movement disorder, is regarded as being among the most serious side effects associated with these medications.

While no approach to treatment has been found to be completely effective in all cases for managing the disorder, behavioral interventions have been shown to decrease the severity of TD with far fewer, less severe adverse side effects than the pharmacological and neurosurgical alternatives that are currently available. The present study was designed to examine the effects of a specific behavioral intervention, progressive relaxation training, on the severity of orofacial tardive dyskinesia. A clear functional relationship was established between the dependent and independent variables in this study, indicating that progressive relaxation training may be of benefit either as an alternative or as an adjunct to pharmacological and surgical methods for managing TD. In conclusion, although the current study was preliminary in nature, the results that were obtained provide a foundation upon which to develop a behavioral treatment protocol for managing TD.
### APPENDIX A

**STRATEGIES FOR ADDRESSING POTENTIAL RELAXATION TRAINING PROBLEMS**

<table>
<thead>
<tr>
<th>Problem</th>
<th>Strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle cramps</td>
<td>The therapist should encourage the trainee to move the cramped muscles while keeping the muscles in other areas of the body relaxed. For more severe cramps, the trainee may need to massage the cramped muscles.</td>
</tr>
<tr>
<td>Movement</td>
<td>The therapist should remind the trainee that he or she is not to move.</td>
</tr>
<tr>
<td>Laughter or talking</td>
<td>Ignore laughter. Ignore talking unless the trainee is reporting a serious problem (e.g., extreme discomfort). If the trainee continues to talk or laugh, the therapist should reiterate that he or she should remain quiet during the training session.</td>
</tr>
<tr>
<td>External noise</td>
<td>The therapist should proceed as though the noise does not exist.</td>
</tr>
<tr>
<td>Spasms and tics</td>
<td>If the spasms or tics do not appear to be disrupting the relaxation process, the therapist should state to the trainee that the movements are not a cause for concern. After the session, the therapist should explain that spasms and tics are associated with muscle relaxation and are commonly experienced prior to sleep.</td>
</tr>
</tbody>
</table>
APPENDIX A (continued)

<table>
<thead>
<tr>
<th>Problem</th>
<th>Strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety-producing thoughts</td>
<td>The therapist should talk more so as to maintain the trainee’s attention on the instructions. The therapist may also, with the trainee’s assistance, identify a set of neutral or pleasant images for the trainee to focus on during sessions. The therapist should then incorporate some feature of the image into the relaxation dialog.</td>
</tr>
<tr>
<td>Sexual arousal</td>
<td>The therapist should reassure the trainee that sexual arousal during a relaxation session is not unusual, that it is not a “major difficulty,” and that there is no need to be alarmed.</td>
</tr>
<tr>
<td>Sleep</td>
<td>If the trainee appears to be asleep, the therapist should continue to make requests or give suggestions in an increasingly louder voice until he or she responds. To avoid sleeping, the therapist should (a) instruct the trainee to sleep for at least 8 hours on the nights before training sessions, (b) avoid scheduling early morning training sessions or scheduling sessions immediately after a trainee has eaten, (c) try speaking louder and less monotonously, and (d) provide direct instructions to the trainee to focus on the sound of his or her voice (i.e., the therapist’s voice) while keeping muscles deeply relaxed.</td>
</tr>
<tr>
<td>Problem</td>
<td>Strategy</td>
</tr>
<tr>
<td>-------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Coughing and sneezing</td>
<td>The therapist can usually ignore coughing and sneezing; however, the session should be postponed if disruptive coughing and sneezing is the result of an illness.</td>
</tr>
<tr>
<td>Inability to relax specific muscle groups</td>
<td>This may stem from the presence of intrusive thoughts (e.g., anxiety-producing thoughts or sexual arousal) and may be addressed by the therapist as indicated above.</td>
</tr>
<tr>
<td>Strange or unfamiliar feelings during relaxation</td>
<td>The therapist should inform the trainee that these feelings are often experienced when learning relaxation techniques and encourage the trainee to enjoy, rather than fear, them. The therapist can also ask the trainee to (a) open his or her eyes, (b) look around the room, (c) look at his or her own body, and then (d) close his or her eyes and continue relaxing.</td>
</tr>
<tr>
<td>“Losing control” during relaxation</td>
<td>The therapist should identify and discuss the subjective experiences the trainee will likely perceive during the training sessions. It is important to emphasize that the trainee, and not the therapist, is producing the sensations and that they will be very pleasant. After each session, the trainee should be encouraged to report any novel sensations. The therapist should continue to assure the trainee that the sensations are frequently reported consequences of deep muscular relaxation.</td>
</tr>
</tbody>
</table>
APPENDIX A (*continued*)

<table>
<thead>
<tr>
<th>Problem</th>
<th>Strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td>“Internal” arousal</td>
<td>At times, trainees will report feeling “tight” or tense “inside” at the conclusion of a relaxation training session, even though there is no tension in any muscle groups. The therapist should explain that the relaxation training procedure directly affects those muscles that are under voluntary control, whereas internal tension involves muscles that are not under voluntary control. Further, the therapist should explain that the voluntary and involuntary systems are interrelated and that, with practice, relaxation of those muscles under voluntary control will eventually produce relaxation internally.</td>
</tr>
</tbody>
</table>

*Note. Information for this table was obtained from the following source: New Directions in Progressive Relaxation Training: A Guidebook for Helping Professionals, by D. A. Bernstein, T. D. Borkovec, and H. Hazlett-Stevens, 2000, Praeger Publishers.*
APPENDIX B

INFORMED CONSENT

Title of Project: A Behavioral Approach to Management of Neuroleptic-Induced Tardive Dyskinesia: Progressive Relaxation Training

Introduction

You are being invited to participate in a research study. The information in this form is provided to help you decide whether or not to take part. Study personnel will be available to answer your questions and provide additional information. If you decide to participate in this study, you will be asked to sign this consent form. A copy of this form will be given to you.

Tardive dyskinesia (TD) is a serious side effect that develops from taking certain medications. People with TD experience involuntary movements that can affect their face, tongue, jaw, trunk, and/or arms and legs. The movements can be uncomfortable and painful and, for some people, embarrassing. Unfortunately, no treatment for TD has been found to be completely effective. Often, TD is treated with medication. These medications sometimes cause additional side effects. Another type of treatment, behavioral treatment, teaches people how to control the movements without using medication. Behavioral treatments for TD have been found to be effective without causing serious side effects.

Purpose of the Study

The purpose of this study is to see whether a specific type of behavioral treatment, progressive relaxation training, will help people with TD to control the movements in their face and mouth. This study is being conducted because I want to find a way to help people control their TD in a way that will not cause more side effects.

Why you were asked to Participate

You are being asked to participate in this study because you are a resident at Posada del Sol Healthcare Center who has been identified as having TD.

Number of People Participating in Study

A total of four people are being asked to participate in this study.
APPENDIX B (continued)

What will happen during this Study

If you agree to participate in this study, you and I will begin by measuring the level of the movements in your face and mouth. During this part of the study, you will be asked to come into the relaxation training room and sit on a reclining chair. I will recline the chair and ask you to remain seated for approximately 30 minutes. I will be videotaping your face throughout each of these sessions. We will continue to do this for a few sessions before we start the relaxation training. I cannot tell you exactly how many sessions we will have before starting the relaxation training because I need to start each person in the study at a different time. We could meet as few as three times or as many as fifteen to twenty times before we begin the relaxation training. Once we do begin the relaxation training, however, we will continue to do it until the study is finished. You should receive between five and twenty-five relaxation training sessions before the study is over.

During the relaxation training sessions, you will still come into the training room and sit on the reclining chair. After the chair is reclined, I will begin to instruct you on how to relax the muscles in your body. I will not touch you during the training unless, with your permission, I assist you with positioning yourself in the reclining chair. We will start by relaxing the muscles in your hands and then gradually move from one muscle group to another until you have relaxed the rest of the muscles in your body. We will spend some extra time on relaxing the muscles in your face and around your mouth. The relaxation training sessions will last about 30 minutes. You may end a relaxation session at any time if you wish to do so.

Length of Study

About 25 to 30 sessions will be needed to complete the study. If possible, I would like to see you twice a day until the sessions are completed. Each session will take about 30 minutes to complete.

Possible Risks

The things that you will be doing have very little to no risk. If you become uncomfortable during the relaxation training in any way or for any reason, you can stop participating immediately and, if you wish for me to do so, I will ask a nurse or a staff person to help you.

Benefits

You will not receive any benefit from participating in this study; however, most people enjoy relaxation training and say that they feel good at the end of each session. Most
people also say that relaxation training helps them to feel less anxious and less stressed out.

Costs

Aside from your time, there are no costs for taking part in this study.

Payment for Participation

You will not be paid for your participation; however, I will give you a relaxation training CD so that you can continue to practice the relaxation techniques after the study is over.

Videotaping

I would like to make a video recording during each of your sessions so that I can accurately record the movements in your face and around your mouth. I will make the video recordings only if you check the box below:

- I give my permission for video recordings to be made of me during my participation in this research study

- I do not give my permission for video recordings to be made of me during my participation in this research study

Confidentiality

The only persons who will know that you participated in this study will be the research team members, Philip Johnson, M.S. (the principal investigator for this study) and Teresa Kolodny (a Ph.D. candidate in the Rehabilitation Program at the University of Arizona). Ernestina Pelayo, M.D. will also know that you participated because she assisted in recruiting you for the study. Your records will be confidential. You will not be identified in any reports or publications resulting from the study. Representatives of regulatory agencies (including the University of Arizona Human Subjects Protection Program) may access your records.

Participation in this Study

Your participation in this study is voluntary. You may decline to participate in this study; this will not affect your present or future care at Posada del Sol Healthcare Center and will not cause any loss of benefits to which you are otherwise entitled. You may also choose to stop the study at any time with no affect on your present or future care at
APPENDIX B (continued)

Posada del Sol Healthcare Center and no loss of any benefits to which you are otherwise entitled. Any new information discovered about the research will be provided to you. This information could affect your willingness to continue your participation.

Additional Information

You can obtain further information about the research or voice concerns or complaints about the research by calling the Principal Investigator, Philip Johnson, Ph.D. Candidate at (520) 626-8126. If you have questions concerning your rights as a research participant; have general questions, concerns, or complaints; or would like to give input about the research and can’t reach the research team, you may call the University of Arizona Human Subjects Protection Program office at (520) 626-6721 (out of state use the toll-free number: 1-866-278-1455). If you would like to contact the Human Subjects Protection Program via the web, please visit the following website: http://www.irb.arizona.edu/contact/

Signature

By signing this form, I affirm that I have read the information contained in the form, that the study has been explained to me, that my questions have been answered, and that I agree to take part in this study. I do not give up any of my legal rights by signing this form.

____________________________________________________  ________________
Printed Name of Participant          Date Signed

____________________________________________________
Participant’s Signature

____________________________________________________
Participant’s Signature          Date Signed

Printed Name of Participant’s Legal Representative (if necessary)

____________________________________________________
Signature of Participant’s Legal Representative       Date Signed

____________________________________________________
Relationship to the Participant
APPENDIX B (continued)

Printed Name of Witness to Legal Representative Consent (if necessary)

____________________________________________________
Signature of Witness            Date Signed

Statement by Person Obtaining Consent

I certify that I have explained the research study to the person who has agreed to participate and that he or she has been informed of the purpose, the procedures, the possible risks, and the potential benefits associated with participation in this study. Any questions raised have been answered to the participant’s satisfaction.

____________________________________________________
Printed Name of Study Principal Investigator

____________________________________________________
Study Principal Investigator Signature            Date Signed
Title of Project: A Behavioral Approach to Management of Neuroleptic-Induced Tardive Dyskinesia: Progressive Relaxation Training

The United States government has issued a new privacy rule to protect the privacy rights of individuals enrolled in research. The Privacy Rule is designed to protect the confidentiality of an individual’s health information. This document hereafter known as an “Authorization for Use and Disclosure of Protected Health Information for Research” describes your rights and explains how your health information will be used and disclosed for this study.

Purpose

You are being invited to participate voluntarily in the above-titled research project. The purpose of this project is to see whether progressive relaxation training will help people with tardive dyskinesia (TD) to control the movements in their face and mouth.

Use and Disclosure of Protected Health Information

The principal investigator, Philip Johnson, Doctoral Candidate, will review your medical records at Posada del Sol Healthcare Center in order to obtain the following information about you: (a) age, (b) history of neuroleptic medication use, (c) reason(s) for prescribing neuroleptic medications, (d) list of current medications, (e) date of tardive dyskinesia diagnosis, (f) tardive dyskinesia symptoms, and (g) history of treatment for tardive dyskinesia. This information will be useful when Mr. Johnson is studying how you respond to the relaxation training.

The information will be coded in order to protect your identity. During the study, the information will be kept in a locked file cabinet in Philip Johnson’s office in the Department of Special Education, Rehabilitation, and School Psychology at the University of Arizona. When the study is finished, the information will be kept in a locked file cabinet in Dr. Charlene Kampfe’s office in the Department of Special Education, Rehabilitation, and School Psychology at the University of Arizona. The information will be destroyed 2 years from the date the study is completed.

This information will be used in the study report to provide some basic background on you and the other participants in the study. You will not be personally identified in this report. The information will also be used as it relates to the way you respond to the relaxation training.
APPENDIX C (continued)

Ernestina Pelayo, M.D. will be providing your medical records to Philip Johnson. Mr. Johnson will only review those records that provide the information listed above. You have the right to access your protected health information that may be created during this study as it relates to your treatment.

Contacts

You can obtain further information from the principle investigator, Philip Johnson, Ph.D. Candidate at (520) 626-8126. If you have questions concerning your rights as a research subject, you may call the Human Subjects Protection Program office at (520) 626-6721.

Authorization

I hereby authorize the release of my medical records by Posada del Sol Healthcare Center. I may withdraw this authorization at any time by notifying the principle investigator in writing. The address for the principle investigator is

Philip Johnson  
University of Arizona  
College of Education  
Department of Special Education, Rehabilitation, and School Psychology  
P.O. Box 210069  
Tucson, AZ 85721-0069

If I do withdraw my authorization, my information previously disclosed cannot be withdrawn and may continue to be used. Once information about me is disclosed in accordance with this authorization, the individual or organization that receives this may redisclose it and my information may no longer be protected by Federal Privacy Regulations. I may refuse to sign this authorization form. If I choose not to sign this form, I cannot participate in the research study. Refusing to sign will not affect my present or future care at Posada del Sol Healthcare Center and will not cause any loss of benefits to which I am otherwise entitled. This authorization will expire on the date the research study ends. I will be given a copy of this signed authorization form.

__________________________________________________________________________  ________________
Participant’s Signature            Date Signed

__________________________________________________________________________
Printed Name of Participant
APPENDIX C (continued)

_______________________________________________      ________________
Signature of Participant’s Legal Representative (if necessary)       Date Signed

_______________________________________________
Printed Name of Participant’s Legal Representative

________________________________________________________
Relationship to the Participant

_______________________________________________             ________________
Printed Name of Witness to Legal Representative Consent (if necessary)              Date Signed

_______________________________________________
Signature of Witness       Date Signed
APPENDIX D

RELAXATION TRAINING PROTOCOL

The following progressive relaxation training procedure was implemented by the PI with each study participant:

1. Greet the participant as he enters the training room.

2. Instruct the participant to sit in the reclining chair.

3. Recline the reclining chair to its lowest position.

4. Asks the participant to make himself comfortable.

5. After a few moments, ask the participant if he is comfortable. When the participant indicates that he is comfortable, instruct him to take three diaphragmatic breaths in the following manner: “We always begin by taking three very slow and gentle deep breaths; breathing in through your nose, holding your breath for a few moments, and then breathing out through your mouth. Now let’s take your first breath. Breathe in through your nose [researcher models inhaling], and hold it…hold it…hold it…, and now breathe out through your mouth [researcher models exhaling]. That’s it, thinking about how good it feels to relax. Now let’s take your second breath [researcher models inhaling], and hold it…hold it…hold it…, and breathe out through your mouth [researcher models exhaling]. Very good. And now let’s take your last breath [researcher models inhaling]… hold it…hold it…hold it…, and breathe out through your mouth [researcher models exhaling].”
6. Turn on a compact disc recording of an ocean surf sound while telling the participant, “As I turn on the surf sound, I’d like you to continue to think about how good it feels to relax.” Six to seven surfs are generated per minute on the ocean surf compact disc recording.

7. Begin to instruct the participant to relax by suggesting “think about letting your hands relax.” The researcher times the word “relax” so that it stated simultaneously with the rhythm of the surf sound. The final consonant of the word “relax” is extended and gradually faded to mimic the fading of each surf sound. The participant is instructed to relax his hands at least three more times, using statements such as “letting your hands relax,” “easy and relax,” and “more and more relax.”

   a. Additional instructions may be used, including “Feeling comfortable and relax;” Breathing easy and relax;” “It feels so good to relax;” “Very, very good, just relax;” “Thinking only of relax;” Deeper and deeper relax;” “Feeling rested and relax;” “Remembering that the more relaxed you are, the better you feel;” and “Feeling peaceful and relaxed.”

8. Continue the relaxation training process until each of the following areas have been addressed: hands, arms, shoulders, forehead, eyes, face, mouth, jaw, neck, upper back, lower back, chest, stomach, legs, and feet.

9. Repeat the procedure for relaxing all of the areas of the body, giving additional attention to relaxing the muscles of the face, mouth, and jaw.
APPENDIX D (continued)

The word “keeping” may be substituted for the word “letting” during this step (e.g., “Think about ‘letting’ your face relax. ‘Keeping’ your face relaxed.”).

10. Allow the participant several minutes to enjoy the feeling of being relaxed: “I’m going to stop talking for a few minutes so that you can enjoy the feeling of being relaxed.”

11. Repeat the procedure of relaxing all of the areas of the body identified above. Two to three areas may be addressed simultaneously during this step (e.g., “Think about keeping your hands, arms, and shoulders relaxed. Keeping your hands, arms, and shoulders relaxed.”).

12. The relaxation session is terminated as follows: Turn off the ocean surf compact disc, then state “We always end each session by taking three slow and gentle deep breaths. Breathe in through your nose [researcher models inhaling], and hold it…hold it…hold it…, now breathe out through your mouth [researcher models exhaling]. That’s it, very, very good. Let’s take the second breath [researcher models inhaling], and hold it…hold it…hold it…, and breathe out through your mouth [researcher models exhaling]. Good. Feeling rested and relaxed. And now, taking the last breath [researcher models inhaling]… hold it…hold it…hold it…, and breathe out through your mouth [researcher models exhaling]. Remembering to stay relaxed for the rest of the day.”
APPENDIX D (continued)

13. Wait for several seconds, then raise the reclining chair to the upright position,
    shake the participant’s hand, and ask how he is feeling. Respond appropriately to
    the participant’s subjective report.

14. Thank the participant and then accompany him to the door of the training room.
## RELAXATION RESPONSE MONITORING FORM

Name: _____________________ Date: ______________ Time Start: ________  
Rater: _____________________ Pre-score: __________ Time Stop: ________  
Session: __________ Post-score: __________

<table>
<thead>
<tr>
<th>Muscle Group</th>
<th>Tense</th>
<th>Relaxed</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Right Hand &amp; Forearm</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>2. Right Bicep &amp; Shoulder</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>3. Left Hand &amp; Forearm</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>4. Left Bicep &amp; Shoulder</td>
<td>1</td>
<td>2</td>
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<tr>
<td>5. Neck</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>6. Forehead</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>7. Eyes</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>8. Mouth</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>9. Abdomen</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>10. Right Leg</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>11. Left Leg</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>12. Entire Body</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

**Note:**
- Relax = loose, limp muscle tone, supported by a subjective verbal report
- Tense = rigid, tight muscle tone, supported by a subjective verbal report
- Relaxation Score = \( \frac{\text{Score Achieved}}{60} \times 100 = \_\_\% \)

**Observations/Notes:**

---

*Note. Adapted from Relaxation Training for People with Severe Mental Retardation (p. 8), by Ferreira, 1994, presented at the American Association on Mental Retardation National Convention, Boston, MA.*
APPENDIX F

MOMENTARY TIME SAMPLING DATA COLLECTION FORM

Name of Participant: ____________________

Name of Data Collector: __________________

Date: _____________

Starting Location on Videotape (in minutes and seconds): __________

Interval length: 10 seconds

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<td>13</td>
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<td>25</td>
<td>26</td>
<td>27</td>
<td>28</td>
<td>29</td>
</tr>
</tbody>
</table>

Key:

+ = occurrence
— = nonoccurrence
APPENDIX G

INTEROBSERVER DATA COLLECTION EXAMPLE

Name of Participant: 001

Date: 3-31-09

Starting Location on Videotape (in minutes and seconds): 21:35

Interval length: 10 seconds

Name of Data Collector: PJ

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<th>4</th>
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<th>6</th>
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</table>

Name of Data Collector: TK

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</table>

Agreement

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<th>16</th>
<th>17</th>
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<td>y</td>
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</tbody>
</table>

Agreement = 27/30 = .90 x 100% = 90%
## APPENDIX H

### TREATMENT INTEGRITY FORM

<table>
<thead>
<tr>
<th>Component</th>
<th>Observation Sessions</th>
<th>Component Integrity</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Greeted the participant</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Instructed the participant to sit in the reclining chair</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Reclined the reclining chair to its lowest position</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Asked the participant to make himself comfortable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Asked the participant if he was comfortable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Instructed the participant to take three deep breaths</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Turned on the ocean surf recording</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Instructed the participant to relax each muscle group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Repeated the procedure for relaxing each muscle group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Allowed the participant several minutes to enjoy the feeling of being relaxed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Repeated the procedure for relaxing each muscle group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Turned off surf sound and instructed the participant to take three deep breaths</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### APPENDIX H (continued)

<table>
<thead>
<tr>
<th>Component</th>
<th>Observation Sessions</th>
<th>Component Integrity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1  2  3  4  5  6</td>
<td></td>
</tr>
<tr>
<td>13. Raised the reclining chair to its upright position</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14. Asked the participant how he was feeling</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15. Accompanied or assisted the participant to the door of the training room</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Session Integrity</td>
</tr>
</tbody>
</table>

Directions: Write a “1” if the component was completed and a “0” if the component was not completed. Compute session and component integrity at the conclusion of the study.

*Note.* Adapted from *School-Based Interventions: The Tools You Need to Succeed* (p. 135), by K. L. Lane and M. E. Beebe-Frankenberger, 2004, Boston: Allyn & Bacon
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