

Selective Serotonin Reuptake Inhibitor Discontinuation Syndrome: Understanding, Recognition, and Management for Psychologists

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Psychologists increasingly provide psychotherapeutic services to patients receiving concomitant pharmacotherapy. Ongoing experience with the relatively new selective serotonin reuptake inhibitors and other atypical antidepressants has revealed a discontinuation syndrome that emerges following abrupt or tapered withdrawal from these agents. The syndrome has both somatic and psychological characteristics, which tend to be mild and transient but can also be more distressing and become temporarily debilitating. Psychologists need to be aware of the potential for these events, as they are in an optimal position to collaborate with physicians and other prescribing clinicians to prevent or manage this syndrome.

Discontinuation syndrome, a cluster of symptoms that appears when a patient abruptly or gradually terminates long-term medication, can be seen with various medication classes, such as antipsychotics, antidepressants, and mood stabilizers (Schatzberg et al., 1997b). For antidepressants, the first reports of discontinuation symptoms were published 40 years ago (Mann & MacPherson, 1959) for the tricyclic antidepressant (TCA) imipramine. Discontinuation syndromes were subsequently identified for other

TCA, as well as for monoamine oxidase inhibitors (MAOIs; Dilsaver & Greden, 1984; Dilsaver, Greden, & Snider, 1987; Halle & Dilsaver, 1993). With the increasing use of the selective serotonin reuptake inhibitors (SSRIs) and other atypical agents, discontinuation events have begun to be noted for these antidepressants as well, the first reports appearing in the literature in 1993 (Black, Wesner, & Gabel, 1993; Mallya, White, & Gunderson, 1993). Now recognized is an SSRI discontinuation syndrome that shares features similar to those noted with discontinuation from other classes of antidepressant, as well as presenting certain novel symptoms (Schatzberg et al., 1997b). Given the large number of patients who are receiving concomitant psychotherapy and pharmacotherapy with these newer antidepressants (Hollon et al., 1992), it is incumbent on psychologists to remain up to date as to developments in the field of psychopharmacology (Rivas-Vazquez & Blais, 1997). In addition to having psychiatrists and other prescribing professionals educate patients as to the possibility of discontinuation events, it is important for psychologists to understand and be able to recognize this syndrome to further educate the patient, increase adherence with the medication regimen, and collaborate with the prescribing clinician in managing the syndrome should it emerge (Kaplan, 1997; Rosenbaum & Zajecka, 1997).

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Recognizing SSRI Discontinuation Syndrome

Discontinuation syndromes associated with TCAs and MAOIs range from relatively transient and mildly distressing symptoms to more severe, emergent events. For example, TCA discontinuation can result in flu-like symptoms (consisting of gastrointestinal distress, nausea, and vomiting), sleep disturbance, movement disorders (e.g., akathisia, parkinsonism), behavioral activation (possibly leading to hypomanic or manic states), and cardiac arrhythmias. More severe reactions, such as delirium, catatonia, agitation, thought disorganization, aggression, or severe cognitive impairment, can result from abrupt discontinuation of MAOIs (Dilsaver

et al., 1987; Haddad, 1997; Lejoyeux & Ades, 1997; Schatzberg et al., 1997b). The SSRI discontinuation syndrome, although it shares an overlap with TCA discontinuation phenomena, presents some novel symptoms and is characterized by both somatic and psychological features.

The somatic symptoms of SSRI discontinuation consist of disequilibrium (e.g., dizziness, lightheadedness, or vertigo), gastrointestinal symptoms (e.g., nausea or vomiting), flu-like symptoms (e.g., fatigue, lethargy, chills, muscular aches), sensory disturbances (e.g., tingling, paresthesias, burning or electric shock sensations), sleep disturbances (e.g., insomnia, experiencing vivid dreams), headaches, and movement-related symptoms (e.g., tremor, akathisia, parkinsonism; Coupland, Bell, & Potokar, 1996; Haddad, 1997; Lejoyeux & Ades, 1997; Schatzberg et al., 1997b). Coupland et al. (1996) reported that the sensation of dizziness has typically been described by patients as a "swimming," "spaced out," "drunken," or "buzzing" quality, which is exacerbated by movement. Psychological symptoms associated with SSRI discontinuation include lowered mood, anxiety, agitation, irritability, tearfulness or crying spells, and aggressive or impulsive behavior, with less frequent reports of confusion, decreased cognitive performance (e.g., slowed thinking, inattention, or poor memory), and depersonalization (Haddad, 1997; Schatzberg et al., 1997b). Table 1 summarizes the most common symptoms of discontinuation events associated with SSRI treatment interruption; these symptoms were obtained from a retrospective chart review study of 352 patients (Coupland et al., 1996), a compilation of 25 case reports appearing in the literature (Zajacka, Tracy, & Mitchell, 1997), and an open-label study of 222 patients comparing three different SSRI agents (Rosenbaum, Fava, Hoog, Ascroft, & Krebs, 1998).

It is critical to distinguish discontinuation phenomena from the emergence of adverse reactions due to other causes such as side

effects, rebound symptoms (the reemergence of the original symptoms, possibly in more intense form, following discontinuation of an agent), relapse or recurrence of a depressive episode, or even psychological reactions to discontinuing pharmacotherapy. It is possible for the clinician to mistake the symptoms for the flu and consequently disregard signs of the syndrome as events unrelated to treatment. Conversely, patients may erroneously overinterpret the symptoms as an indication that they have become "addicted" to the antidepressant, thereby compromising motivation for any necessary treatment during future episodes of depression (Rosenbaum & Zajacka, 1997). Antidepressant discontinuation syndrome needs to be conceptualized as being quite distinct from the withdrawal states associated with discontinuation of addicting agents, such as benzodiazepines, sedative hypnotics, alcohol, or barbiturates. Withdrawal events from these agents can range from diaphoresis, tachycardia, and jitteriness to more emergent conditions such as seizures, coma, cardiovascular failure, and possibly even death. It is also important to note that long-term exposure to these substances can lead to increased physiological tolerance, as well as to drug-seeking behavior and an increase in the individual's craving for the substance (Schatzberg et al., 1997b). Finally, since discontinuation symptoms can include alterations in mood, affect, appetite, and sleep, it is important not to misinterpret these symptoms as a relapse into depression, thereby inappropriately continuing antidepressant treatment (Lejoyeux & Ades, 1997). Discontinuation symptoms will emerge within 24 to 72 hr, whereas signs of a depressive relapse are not likely to manifest themselves for 2 to 3 weeks (Rosenbaum & Zajacka, 1997).

In an effort to better delineate this syndrome, a consensus panel of experts recently proposed the following model for SSRI discontinuation syndrome (Schatzberg et al., 1997b):

1. The symptoms are not attributable to other causes.
 2. The symptoms emerge after medication is abruptly discontinued, following periods of intermittent noncompliance (missed or forgotten doses, drug holidays), or, less frequently, following prescribed dose reduction.
 3. The symptoms are generally mild and transient but can be more distressing and troublesome and can lead to brief impairments in functioning or productivity (e.g., missed work).
 4. Symptoms are self-limiting, typically resolving within 2 to 3 weeks.
 5. The syndrome is rapidly reversed by reintroducing the original medication or by substituting another agent that is pharmacologically similar.
 6. Occurrence or severity of the syndrome is minimized either by a slow tapering of the medication or by utilization of a similar agent that has an extended half-life, such as fluoxetine (Prozac).
- Although no standardized assessment instruments exist to date, investigators in a recent study devised a checklist based on the signs and symptoms of discontinuation syndrome that have been reported in the literature to date (Rosenbaum et al., 1998). The Discontinuation-Emergent Signs and Symptoms (DESS) Checklist is a 43-item instrument that is clinician administered and rated and that queries the patient as to the presence and/or status of the spectrum of somatic and psychological symptoms typically associated with the syndrome. Although it requires validation, the DESS Checklist offers clinicians a systematic strategy for appropriate recognition of discontinuation syndrome.

Table 1
*Symptoms of Selective Serotonin Reuptake Inhibitor
Discontinuation Syndrome Events
(in Descending Order of Occurrence)*

Coupland et al. (1996) ^a (N = 158)	Zajacka et al. (1997) ^b (N = 25)	Rosenbaum et al. (1998) ^c (N = 185)
Dizziness	Dizziness	Worsened mood
Movement-related	Fatigue/malaise	Irritability
Lethargy	Nausea	Agitation
Paresthesias	Headaches	Dizziness
Nausea	GI symptoms	Confusion
Vivid dreams	Abnormal dreams	Headache
Lowered mood	Insomnia	Nervousness
Headaches	Myalgias	Crying
Insomnia	Incoordination	Fatigue
Anxiety	Tremulousness	Emotional lability
Irritability	Electric sensations	Insomnia
	Vomiting	Dreaming
	Agitation	Anger
	Chills	Nausea

GI = gastrointestinal.

^a Medications included paroxetine, fluvoxamine, sertraline, and fluoxetine. ^b Medications included paroxetine, fluvoxamine, sertraline, fluoxetine, and venlafaxine. ^c Medications included paroxetine, sertraline, and fluoxetine.

Pharmacologic Factors Determining SSRI Discontinuation Syndrome

Following its release into the synaptic cleft by the presynaptic neuron, a neurotransmitter—in this case, serotonin—either binds to receptors on the postsynaptic neuron, is reabsorbed by the presynaptic neuron by means of a reuptake pump, or is broken down and degraded into its main metabolite(s) by the enzyme monoamine oxidase. By binding to the reuptake pump, the SSRIs predominantly inhibit the presynaptic neuron's reuptake of serotonin, thereby increasing the pharmacological effects of serotonin by allowing it to act in the synapse for an extended period. As increased levels of available serotonin are sustained following chronic administration of SSRIs, a negative feedback mechanism causes the serotonin receptors on the postsynaptic neuron to functionally decrease in density—a process referred to as *down-regulation* (a more detailed account of the mechanism of action of the SSRIs and newer antidepressants is summarized concisely by Stahl, 1998). When SSRI treatment is discontinued, there is a resultant decrease in levels of synaptic serotonin, as the presynaptic reuptake pump is no longer blocked. Although the precise mechanism underlying the discontinuation syndrome is unknown, it is speculated that the symptoms that are observed following discontinuation or interruption of treatment are caused by the temporary deficiency of synaptic serotonin in the face of down-regulated (or reduced) serotonin receptors. Furthermore, because other neurotransmitter systems are affected, albeit to a lesser degree than serotonin, it is felt that acute alterations in dopamine, norepinephrine, or gamma-aminobutyric acid levels may also play a role in the emergence of the syndrome (Schatzberg et al., 1997a).

Antidepressants demonstrate structural and functional differences that dictate their pharmacological activity, typically referred to as *pharmacodynamics* (how and where the drug affects the body) and *pharmacokinetics* (how the body processes the drug, in terms of absorption, distribution, metabolism, and elimination). Ultimately, both pharmacodynamic and pharmacokinetic properties of a drug will determine therapeutic efficacy, amount of medication required for clinical response, required frequency of administration, side effect profile, drug-drug interactions, and toxicity. Pharmacodynamic elements that are most relevant to the emergence of discontinuation syndrome consist of the agent's selectivity or affinity for a particular type of receptor, as well as the agent's strength or potency of action at that binding site. For example, although the SSRIs demonstrate a higher degree of selectivity for serotonin receptors relative to other neurotransmitter receptors, there is a variability across agents as to their affinity for other receptor sites, such as acetylcholine, dopamine, and norepinephrine. Similarly, antidepressants demonstrate differences in terms of their potency for blocking the reuptake pump, which consequently affects synaptic serotonin levels.

Pharmacokinetic factors that determine the emergence of discontinuation events consist of the agent's half-life, the activity of the agent's metabolite(s), and whether the agent exhibits linear versus nonlinear kinetics. A drug's half-life, which is usually expressed in hours, refers to the time required to eliminate half (50%) of the drug from blood plasma. An agent's half-life determines the time it takes to reach the steady-state plasma concentration that is needed for optimal clinical effect (which usually occurs in approximately 4 to 5 half-lives), as well as the frequency

of dosing (the shorter the half-life, the more frequently the agent has to be administered to achieve constant serum levels). A drug's metabolites are the breakdown products of that drug and can be either active or inactive (referring to whether or not the metabolite exhibits pharmacodynamic activity similar to that of the parent compound). Finally, linearity refers to the relationship between the blood levels of an agent and the amount ingested. For example, doubling the dose of an agent with linear kinetics should result in a twofold increase in blood levels, whereas doubling an agent with nonlinear kinetics leads to a greater-than-proportional increase in blood levels (i.e., a fourfold increase). More pertinent to discontinuation syndrome, an agent with nonlinear kinetics evidences a disproportionate decrease in blood levels relative to the dosage reduction (Pies, 1998). Table 2 summarizes pharmacokinetic properties of several agents that have been associated with discontinuation events.

Variations in the reported frequency of discontinuation events for the SSRIs and the newer atypical agents are based on the different pharmacologic properties demonstrated by each agent (Preskorn, 1993). It is readily apparent that the factors noted above determine the residual availability of synaptic serotonin following discontinuation or interruption of treatment. Consequently, agents demonstrating a pharmacologic profile that results in a precipitous decline in serotonin are more likely to produce discontinuation events. For example, paroxetine (Paxil) has a short half-life, no active metabolites, and nonlinear kinetics, and is a highly potent inhibitor of serotonin reuptake. It is not surprising, then, that paroxetine has been associated with the highest incidence of discontinuation symptoms in two separate reviews of the literature (Lejoyeux & Ades, 1997; Zajecka et al., 1997). Additionally, paroxetine is distinct from the other SSRIs in that it has some affinity for cholinergic receptors, which may contribute to the symptoms that emerge when this agent is abruptly discontinued (Pies, 1998).

Conversely, discontinuation events have been reported much less frequently for fluoxetine, which has the longest half-life and an active metabolite and is not as potent in inhibiting serotonin reuptake as the other SSRIs (Dominguez & Goodnick, 1995). Even though fluoxetine exhibits nonlinear kinetics, its long elimination rate and persistent pharmacologic activity due to its active metabolite appear to serve as a buffer to minimize the emergence of discontinuation events (Lazowick, 1995; Schatzberg, 1998; Thompson, 1998).

In a prospective study examining discontinuation phenomena in 222 patients, Rosenbaum et al. (1998) used the DESS Checklist

Table 2
Pharmacokinetics of Selective Serotonin Reuptake Inhibitors and Atypical Antidepressants

Drug	Trade name	Daily dosage range (mg/day)	Half-life (hr)	Active metabolite	Linear kinetics
Paroxetine	Paxil	10–60	21	No	No
Fluvoxamine	Luvox	50–300	15	Unclear	No
Sertraline	Zoloft	50–200	26	Yes	Yes
Fluoxetine	Prozac	20–80	48–72	Yes	No
Venlafaxine	Effexor	75–375	3–7	Yes	Yes
Nefazodone	Serzone	200–600	2–4	Yes	No

Note. Data are from *Physicians' Desk Reference* (1999).

and other instruments to observe and compare the frequency of discontinuation events associated with paroxetine, sertraline (Zoloft), and fluoxetine treatment interruption. Results indicated that discontinuation events were statistically less for fluoxetine-treated patients versus those treated with paroxetine and sertraline, with the greatest number of events observed for the paroxetine group. These findings are consistent with the anecdotal cases that have been reported in the literature.

Outside of the SSRI class, discontinuation phenomena similar to those observed with the SSRIs have also been reported for the atypical antidepressant venlafaxine (Effexor), which is a serotonin-norepinephrine reuptake inhibitor (Agelink, Zitzelsberger, & Lieser, 1997; Farah & Laurer, 1996). Given its short half-life, venlafaxine may be as likely as paroxetine to produce discontinuation events. Parker and Blennerhassett (1998) reported that venlafaxine discontinuation produced symptoms that could be quite distressing and that could occur after missing even a single dose. A tapering schedule has been recommended for any patient who has been treated with venlafaxine for 1 week or longer (Rosenbaum & Zajecka, 1997). Recently, the extended-release formulation of venlafaxine was studied in order to determine whether this preparation also produced symptoms upon abrupt discontinuation (Fava, Mulroy, Alpert, Nierenberg, & Rosenbaum, 1997). Results indicated that extended-release venlafaxine produced a statistically greater frequency of discontinuation symptoms when compared with a placebo, prompting investigators to recommend gradual tapering when terminating or changing treatment from this formulation of venlafaxine.

The first case report of possible discontinuation phenomena associated with the atypical antidepressant nefazodone (Serzone) recently appeared in the literature (Benazzi, 1998). Although it has some atypical features, nefazodone shares a similar pharmacology with the SSRIs (Stahl, 1998), and the patient in this report developed dizziness, nausea, vomiting, sweating, insomnia, and restlessness following 1 day of abrupt discontinuation. The symptoms persisted for 3 days and spontaneously remitted. To date, there have been no reports in the literature of discontinuation events for the atypical antidepressants bupropion (Wellbutrin) or mirtazapine (Remeron). Studies conducted with two new antidepressants, citalopram (Celexa), which is a highly specific SSRI, and reboxetine, which is a selective norepinephrine reuptake inhibitor, reported that no discontinuation events upon abrupt discontinuation had been observed with either of these two agents (Mucci, 1997; Muldoon, 1996).

Emergence of SSRI Discontinuation Syndrome

Aside from pharmacologic properties that affect the occurrence of this syndrome, the rate of discontinuation represents a critical determinant in the emergence of events. Acute discontinuation of an agent, due to either intentional or accidental noncompliance, places patients at increased risk for experiencing symptoms, as compared with the incidence of discontinuation events that emerge when medication is slowly and gradually tapered following completion of pharmacotherapy (Lejoyeux & Ades, 1997). Length of treatment also appears to be a factor determining the emergence of discontinuation syndrome, with patients being at increased risk following several months of SSRI treatment; it is unusual for patients to exhibit discontinuation phenomena when they have

been treated with an SSRI for less than 7 weeks (Thompson, 1998). As noted above, venlafaxine may be unique in this regard, as a tapering schedule has been recommended for patients treated with this agent for periods as brief as 1 week. Treatment duration of 6 months or longer is not felt to increase risk for the syndrome (Coupland et al., 1996; Rosenbaum & Zajecka, 1997; Schatzberg et al., 1997b).

Approximately one third of patients who terminate SSRI treatment exhibit discontinuation syndrome (Rosenbaum & Zajecka, 1997). Symptoms begin to emerge within 1 to 3 days following the last dose or commencement of tapering (particularly for paroxetine), although onset may be delayed in the case of an agent with an extended half-life, such as fluoxetine, in which case symptoms may emerge a week or longer following the final dose. Discontinuation from venlafaxine may tend to precipitate rapid emergence of symptoms, possibly within 24 hr (Lejoyeux & Ades, 1997). Duration of symptoms ranges from several days to several weeks (Coupland et al., 1996), with most symptoms remitting within 2 weeks (Rosenbaum & Zajecka, 1997). Symptoms are usually mild and transient but can be more severely distressing and debilitating, leading to disruption of psychosocial activities (e.g., missed days at work) and requiring some type of intervention (Pacheco, Malo, Aragues, & Etxebeste, 1996).

Management of SSRI Discontinuation Syndrome

Upon the emergence of symptoms, the clinician needs to determine their severity and the appropriate management strategy. If the patient has been noncompliant in the context of a successful therapeutic regimen, directives to continue treatment and increase compliance should be made (Kaplan, 1997). When the symptoms emerge during a tapering of the medication at the completion of therapy, supportive interventions, education as to the transient nature of these events, and reassurance that the symptoms are not signs of a depressive relapse or of antidepressant addiction oftentimes suffice.

When pharmacologic intervention is deemed necessary, it is generally felt that reinstating the original antidepressant and recommencing with a slower tapering schedule minimize the occurrence of discontinuation events. Some patients may require a switch to the extended half-life agent fluoxetine prior to proceeding with the tapering schedule. Patients who either are being treated with paroxetine or venlafaxine, have exhibited medication-related anxiety during treatment, or have experienced discontinuation symptoms in the past should be managed with particular caution and undergo a slow and gradual tapering schedule. Rosenbaum and Zajecka (1997) provided recommended rates of tapering for specific medications.

There is a clear role for psychologists in the prevention and management of SSRI discontinuation syndrome. Undoubtedly, discontinuation symptoms require monitoring by a physician or other medical professional, implying that when these events emerge during the course of psychotherapeutic treatment, interventions made by psychologists need to be in collaboration with the prescribing clinician. Interestingly, a recent survey of psychiatrists and physicians in England revealed that "a sizable minority of psychiatrists and a majority of general practitioners said that they were not confidently aware of adverse events associated with antidepressant discontinuation" (Young & Currie, 1997, p. 29).

This issue, coupled with the high number of psychotherapy patients who are receiving concomitant pharmacotherapy, suggests that psychologists need to be fully aware of this phenomenon to participate in its prevention, recognition, and management. As noted elsewhere (Rivas-Vazquez & Blais, 1997), it remains the responsibility and ethical duty of the psychologist to ensure that he or she possesses and can demonstrate the appropriate level of training and command of information prior to engaging in this sphere of clinical work. This having been stated, it appears that there are two primary functions regarding SSRI discontinuation syndrome that psychologists can perform during the course of their psychotherapeutic work: First, they can be instrumental in reducing the risk of discontinuation syndrome by facilitating compliance or adherence with medication treatment. Second, they can assist in the management of the syndrome, should it occur, by providing ongoing education and support.

Noncompliance with pharmacotherapy is a significant factor in the development of discontinuation syndrome. Education about medications and the biological aspects of depression can greatly increase compliance (Katon et al., 1995), and psychologists can be effective in providing this information (Kaplan, 1997; Rosenbaum & Zajecka, 1997). Even though physicians and prescribing clinicians are likely to provide initial education, these messages should be reinforced on multiple occasions, particularly for patients with memory and concentration deficits consequent to depression (Kaplan, 1997). Given the frequency of their contact with patients, psychologists are in an optimal position to review and reinforce this information.

In addition to providing basic information on antidepressant medications, coverage of several specific topics may facilitate adherence to pharmacotherapy. Many patients erroneously expect an immediate response to medication, and the failure to obtain this may increase the risk of noncompliance. Discouragement and premature discontinuation of treatment can be reduced by providing information about the actual time course of antidepressant response latency. Psychologists can also educate patients about side effects, another important determinant of noncompliance, and dispense strategies for minimizing the discomfort or consequences of side effects (such as standing slowly if experiencing dizziness or using sugar-free candies to relieve dry mouth). Therapeutic interventions can be used to challenge cognitions that interfere with adherence, such as beliefs that the patient does not deserve treatment or that side effects are a sign of toxicity or even guilt-ridden attitudes about spending money on medication (Fawcett, 1995). Given that most individuals believe that antidepressant medications are addictive (Priest, Vize, Roberts, Roberts, & Tylee, 1996), this misconception should be attended to routinely, as well as other beliefs that may erroneously blur distinctions between antidepressant medication and drugs of abuse or street drugs. Psychologists can also assess whether family members or significant others are reinforcing negative cognitions about pharmacotherapy, thus indicating the need for psychoeducational family interventions (Fawcett, 1995). Finally, as depressive symptoms begin to remit, the reasons for noncompliance may shift: Patients need to understand the importance of continuing to comply with their prescribed medication beyond symptom remission to prevent relapse or recurrence.

Although relatively few studies have examined interventions to improve medication adherence in unipolar depression, available

evidence suggests that the above strategies are effective. In general, psychotherapy offered as an adjunct to pharmacotherapy promotes adherence with pharmacotherapy (Paykel, 1995). More specifically, in the 3-year maintenance therapy study at the University of Pittsburgh, use of the above strategies helped 85% of patients maintain compliance, with less than 10% leaving the trial. Interestingly, patients in the maintenance phase of the trial were seen only once every 6 months by a physician, with other routine visits being conducted by nonphysician primary clinicians, suggesting that much of the work of facilitating and maintaining medication adherence can be conducted effectively by nonphysician health providers (Frank, Kupfer, & Siegel, 1995). In short, preliminary evidence suggests that psychotherapeutic interventions promote compliance.

Beyond providing information to both enhance adherence and prevent the occurrence of discontinuation phenomena, psychologists are also well positioned to intervene when patients in psychotherapy begin to demonstrate symptoms of discontinuation, given that many patients report these events to their therapists before their physicians (Rosenbaum & Zajecka, 1997). If the emergence of symptoms is due to noncompliance, reiteration as to the importance of adhering to the dosing schedule as prescribed by the physician and reestablishment of medication compliance should be a sufficient intervention. When discontinuation events occur during planned cessation of a trial of pharmacotherapy, a collaborative effort by the physician and psychologist can result in the development and implementation of an appropriate management strategy. If the symptoms are being tolerated relatively well, a decision can be made to provide support, education, and reassurance to the patient, particularly as to the transient nature of the syndrome. As noted above, some patients may misinterpret these symptoms as a sign of physiological addiction or of reemerging depressive symptoms, which may heighten their subjective distress and consequently make the discontinuation phenomena less tolerable. Furthermore, these erroneous beliefs may promote a negative view of antidepressant therapy and may prevent a patient from seeking appropriate treatment in the case of future depressive episodes. Although no research has examined psychotherapy during antidepressant tapering, the addition of cognitive-behavioral treatment during benzodiazepine tapering has been shown to facilitate good outcome, as measured by decreased rates of medication use during the follow-up period, for patients with panic disorder (Otto et al., 1993). Extrapolating from this study, it is possible that psychological interventions during antidepressant tapering can facilitate smooth discontinuation, even if discontinuation symptoms should emerge, and can minimize the development of any negative associations with pharmacological treatment.

Summary

SSRI discontinuation syndrome has been observed with patients who either abruptly stop their medication due to noncompliance or are being tapered down during completion or modification of antidepressant treatment. The syndrome has both somatic and psychological characteristics, which can range from mild and transient to more distressing events. Treatment with certain agents may place patients at increased risk for the emergence of this syndrome, and the shorter half-life agents paroxetine, venlafaxine, and sertraline have been associated with greater frequency of

discontinuation events. Psychologists, working in conjunction with the prescribing clinician, can play a significant role in the prevention of discontinuation symptoms by increasing medication compliance, as well as by assisting in the management of the syndrome by providing support and reassurance for patients who are experiencing discontinuation phenomena.

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