Symptomatic Overlap of Cocaine Intoxication and Acute Schizophrenia at Emergency Presentation

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Abstract

Cocaine intoxication and acute abstinence alter brain dopaminergic functioning, resulting in behavioral changes closely mimicking the positive and negative symptoms of schizophrenia. In emergency room settings, recent cocaine abuse can be mistaken for schizophrenia and may cause inappropriate diagnosis and in some instances medical mismanagement. Schizophrenia patients presenting with recent cocaine abuse may also present with significant diagnostic and treatment dilemmas. This study attempts to distinguish between cocaine and schizophrenic psychosis by examining patients who present with both recent cocaine abuse and acute schizophrenia (CA+SZ), cocaine intoxication without schizophrenic illness (CA), and acute schizophrenia with no comorbid substance abuse (SZ) within the first 24 hours after arrival at the Bellevue psychiatric emergency service. Clinical assessment included the Brief Psychiatric Rating Scale, the Schedule for the Assessment of Positive Symptoms, and the Schedule for the Assessment of Negative Symptoms. Both cocaine abusing groups were required to have positive urine toxicology screens for inclusion in the study. Multivariate analysis of variance showed the CA+SZ patients present with a clinical profile that overlaps with CA patients on mood and negative symptom dimensions and overlaps with SZ patients on most positive symptoms. CA+SZ patients differed from both groups, however, by presenting with significantly more hallucinatory experiences than cocaine abusing or schizophrenia patient counterparts. Despite considerable overlap, each group of patients presented with a discernible cross-sectional symptom pattern.

Key words: Cocaine intoxication, psychosis, emergency assessment.

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Comorbid substance abuse has been found to be present in almost half of all schizophrenia patients presenting for emergency service treatment (Atkinson 1973; Barbee et al. 1989). Recent cocaine abuse has also been found to be a major factor in precipitating the use of emergency psychiatric services by schizophrenia patients (Atkinson 1973; Barbee et al. 1989; Dixon et al. 1990). Cocaine abuse, moreover, can significantly alter schizophrenia patients' clinical presentation in acute treatment settings (Atkinson 1973; Brady et al. 1990; Sevy et al. 1990; Seibyl et al. 1993; Serper et al. 1995, 1996).

Psychostimulant administration in both controlled (e.g., Angrist et al. 1982; van Kammen and Boronow 1988; Goldberg et al. 1991) and naturalistic (Serper et al. 1995) settings has been associated with a reduction in schizophrenia patients' negative symptom presentation. In our recent study, for example, we found cocaine abusing schizophrenia patients who presented to the psychiatric emergency service had significantly fewer negative symptoms than nonabusing schizophrenia emergency service patients (Serper et al. 1995). At 4-week retest on the inpatient service this pattern reversed itself. Seeing a similarity to past studies using controlled administration of psychostimulants, we suggested that the temporary reduction in negative symptoms resulted from the pharmacodynamic actions of cocaine. Similarly, Goldberg et al. (1991), in a controlled laboratory investigation, found improvements in negative symptoms in schizophrenia patients after administration of moderate amounts of dextroamphetamine (0.25 mg/kg per day). Goldberg suggested that amphetamine may work to decrease negative symptoms by stimulating cortical dopamine D1 receptors.

In addition to affecting negative symptoms, acute and chronic cocaine abuse in both schizophrenia and non-

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schizophrenia patients, as well as in laboratory animals, has been associated with increased anxiety and depression (Gawin and Ellinwood 1988; Brady et al. 1990; Sevy et al. 1990; Yang et al. 1992; Serper et al. 1995). Sevy et al. (1990), for example, found cocaine abusing schizophrenia patients showed significant elevations in depression compared with nonabusing schizophrenia inpatients. Similarly, cocaine abuse in patients without psychiatric disorders has been associated with panic attacks, chronic depression, and increased suicidal ideation (Gawin and Ellinwood 1988; Gold 1993).

Cocaine abuse in schizophrenia and nonschizophrenia patients has, in some studies, been associated with causing or worsening positive symptoms (Siegel 1984; Brady et al. 1990, 1991; Cleghorn et al. 1991; Satel and Edell 1991). Brady et al. (1990), for example, found that cocaine abusing schizophrenia patients present with significantly more paranoia than nonabusing patients. Similarly, cocaine abuse in nonschizophrenia patients has also been associated with potentiating psychotic symptoms including paranoia and hallucinations (McLellan et al. 1979; Siegel 1984; Gold 1993) and a high rate of psychiatric emergency service visitation (Breslow et al. 1996). Specifically, cocaine-intoxicated patients without a history of psychiatric disorders have been found to present for psychiatric emergency treatment with significant paranoia and hallucinations that can be mistaken for schizophrenia (Siegel 1984; Brady et al. 1991; Breslow et al. 1996). Psychosis usually lasts from several hours to days, but longer periods of psychosis and disorganization have been reported (Siegel 1984). McLellan et al. (1979) found that prolonged stimulant abuse (6 or more years) was associated with emergence of persistent schizophrenia-like symptoms.

In acute treatment settings, consequently, the behavioral concomitants of cocaine intoxication and withdrawal in nonschizophrenia patients can be mistaken for schizophrenia or other psychiatric conditions, and acute cocaine intoxication can be overlooked or undetected in psychotic schizophrenia patients. In either case, inappropriate diagnosis and, in rare instances, medical mismanagement may result (Barbee et al. 1989). Recent binge cocaine use, for example, may result in significant diagnostic and emergency treatment complications such as seizures, myocardial infarction, aggressive outbursts, respiratory failure, and sudden death (Jonsson et al. 1983; Schachne et al. 1984; Barbee et al. 1989; Nadamanee et al. 1989). It is therefore important for clinicians to characterize and recognize clinical manifestations of acute cocaine presentations in schizophrenia and nonschizophrenia patients.

No study to date has directly compared the acute symptom profile of nonpsychiatric cocaine-abusing patients with schizophrenia patients who abuse cocaine and with nonabusing schizophrenia patients presenting for emergency psychiatric treatment. As a followup to our previous report (Serper et al. 1995), we were interested in determining the symptomatic overlap and divergence between cocaine-abusing and non-cocaine-abusing schizophrenia patients on several key dimensions of psychopathology, including positive symptoms, negative symptoms, and anxiety/depression. Consistent with our earlier findings, we hypothesized that cocaine-abusing patient groups would present with more anxiety/depression and fewer negative symptoms than the nonabusing schizophrenia patients. We did expect, however, that cocaine-abusing schizophrenia patients would present with significantly more negative signs than nonpsychiatric cocaine-abusing patients. Considering the combined effects of recent psychostimulant use and acute psychosis, we hypothesized that cocaine-abusing schizophrenia patients would manifest more psychotic symptoms than schizophrenia patients and cocaine-abusing nonschizophrenia patients. It should be noted, however, that our patients self-selected for cocaine abuse and, consequently, our conclusions must be limited because of the naturalistic design.

Methods

Subjects. Three groups of subjects were studied: 54 schizophrenia patients with no current substance abuse diagnoses (SZ), 32 cocaine-abusing schizophrenia patients with or without other substance use (CA+SZ), and 30 cocaine-abusing patients (CA) without current or past history of schizophrenic disorder. Because we were interested in examining acute effects of cocaine abuse, we initially selected all subjects from patients successively entering the psychiatric emergency service (PES) at Bellevue Hospital who had an initial chart diagnosis of schizophrenia or cocaine abuse/dependence and gave voluntary written informed consent to participate. Table 1 presents the demographic characteristics of the three groups.

Two weeks after the initial PES assessment, all schizophrenia subjects received a Structured Clinical Interview for *DSM–III–R* (SCID; Spitzer et al. 1990) and a formal *DSM–IV* (American Psychiatric Association 1994) diagnosis was made. All SCID interviews of SZ and CA+SZ patients were conducted 2 weeks after admission to the psychiatry inpatient service in order to limit the effects of transient acute drug-induced psychosis on the CA+SZ interview results. Of 30 CA patients entering the emergency service, 28 were released within the first 24 hours of arrival, precluding our ability to conduct the SCID interview. Consequently we set the following criteria for

Table 1. Demographic Information

	Group					
	SZ		CA+SZ		CA	
Variable	M	SD	M	SD	M	SD
Age, yrs	39.4	10.1	34.20	7.40	35.0	8.70
% male	93.0	—	91.00	—	87.0	—
Chlorpromazine equivalent dose	462.3	299.6	552.30	282.10	268.3	8.90
Education, yrs	11.5	2.3	10.50	2.00	10.60	2.80
Number of previous hospitalizations	11.7	8.3	12.80	7.50	6.00	7.80
Amount spent on cocaine in 24-hr. period before PES arrival	—	—	$108.58	$153.16	$107.46	$113.89

Note.—SZ = schizophrenia patients; CA+SZ = cocaine-abusing schizophrenia patients; CA = cocaine-abusing patients; M = mean; SD = standard deviation; PES = psychiatric emergency service.

study inclusion of CA patients: they received a current medical chart *DSM–IV* diagnosis for cocaine abuse or dependence; they currently had a positive urine toxicology screen for cocaine; they reported using cocaine within the last 24 hours; they received no current diagnosis or rule-out diagnosis of schizophrenia, schizoaffective disorder, any *DSM–IV* Axis I disorder with a psychosis component, or any *DSM–IV* Axis II odd cluster diagnosis; and a review of available past medical chart information revealed they had no past discharge diagnoses of schizophrenia or schizoaffective disorders. Diagnostic subgroupings for cocaine abuse were also supplemented by the Bellevue Substance Abuse Questionnaire (BSAQ; Munsey et al. 1992). Positive urine toxicology for cocaine was required in order to be entered into either the CA or CA+SZ patient group. Two patients who reported a history of cocaine abuse but who had clean urine were not included in the protocol. In addition, no CA+SZ or CA subject tested urine-positive for opiates, methadone, barbiturates, or benzodiazepines. Diagnoses for psychoactive substance abuse and schizophrenia were generated independently by a trained SCID rater and then subject to independent diagnoses generated by a senior attending psychiatrist during diagnostic consensus meetings. Diagnostic reliability between the raters and the psychiatrist was high: 0.87 (kappa) for schizophrenia; 0.82 (kappa) for cocaine abuse/dependence for all cases. Diagnostic disagreements were settled by discussion. No subject with a history of definite brain illness or injury or any disorder of the central nervous system was entered into the protocol. No subjects with onset of SZ symptoms before the age of 16 or after the age of 65 were included.

Although an effort was made to assess patients in a medication-free state, 17 SZ, 10 CA+SZ, and 3 CA subjects received neuroleptic medication prior to testing. The three CA subjects receiving neuroleptic received one dose as needed in the emergency service. Chlorpromazine medication dose equivalents (Davis 1974) for each of the patient groups are presented in table 1. There were no significant group differences in medication at time of testing. Cocaine abuse profiles obtained from the BSAQ are presented in table 1. Both abusing groups reported similar cocaine use profiles according to the amount of money they reported spending on cocaine within the 24 hours before arrival at the PES. Twenty-six subjects withdrew from the study and are not included in the analysis. There were no significant group differences on any demographic or clinical variable between patients who completed the protocol and those who did not ($F < 1$).

Assessment Instruments. Assessment instruments included the Brief Psychiatric Rating Scale (BPRS; Overall and Gorham 1962), a scale that assesses a wide variety of psychiatric signs and symptoms, including anxiety/depression, thought disturbance, anergia, hostility, and activation subscales; the Schedule for the Assessment of Positive Symptoms (SAPS; Andreasen 1984*a*), divided into four subscales: hallucinations, delusions, bizarre behavior, and positive thought disorder; and the Schedule for the Assessment of Negative Symptoms (SANS; Andreasen 1984*b*), including items for affective flattening, alogia, avolition-apathy, anhedonia-asociality, and attention subscales. SAPS and SANS global rating items for each subscale were omitted from subscale total scores and summed to produce a global SAPS and global SANS rating for each patient group. Trained raters conducted the BPRS, SAPS, and SANS ratings on all subjects. The inter-rater agreement for the scales, based on 20 percent of the total sample, using intraclass correlation coefficients (Shrout and Fleiss 1979), was high: 0.91 for BPRS, 0.89 for SAPS, and 0.87 for SANS.

Data Analyses. Demographic data were examined using one-way analyses of variance (ANOVA). Data analyses for each clinical rating scale measure (i.e., BPRS, SAPS,

and SANS) were conducted using multivariate analyses of variance (MANOVA) on each subscale score. For each subscale on the SAPS and SANS, global rating items were not used to compute the subscale total score. Instead, global ratings for each subscale were summed to form SAPS global and SANS global subscale measures. When multivariate diagnostic group differences were found to be significant, ANOVA with Tukey-Cramer (i.e., Tukey HSD) pairwise comparisons were employed to examine univariate effects.

Results

The first analyses examined group demographic characteristics of the sample. The data serving as the basis for these analyses are presented in table 1. ANOVA found significant differences in the subjects' ages ($F = 4.24$, $df = 2,113$, $p < 0.05$). Tukey-Cramer tests revealed the SZ patients were significantly older than CA+SZ, but CA did not significantly differ from either group. In addition, CA+SZ and SZ patients reported significantly more previous hospitalizations than CA patients ($F = 6.08$, $df = 2,99$, $p < 0.003$). (Because they could/would not report how many previous hospitalizations they had had, 6 SZ, 5 CA+SZ, and 2 CA patients were excluded from this analysis. There was a nonsignificant trend toward more years of education for SZ than the other groups ($F = 2.48$, $df = 2,113$, $p < 0.08$).

The next set of analyses examined clinical scale characteristics of the sample. Patients' subscale scores on the SAPS, SANS, and BPRS are presented in table 2. SAPS and SANS global scores for each group are presented in figure 1.

On the SAPS, the overall multivariate effect was significant (Pillais approximate $F = 3.72$, $df = 10,220$, $p < 0.001$). When univariate ANOVAs were performed, significant differences were detected on the SAPS global ($F = 3.45$, $df = 2,113$, $p < 0.05$), thought disorder ($F = 7.51$, $df = 113$, $p < 0.001$), hallucinations[1] ($F = 7.38$, $df =$

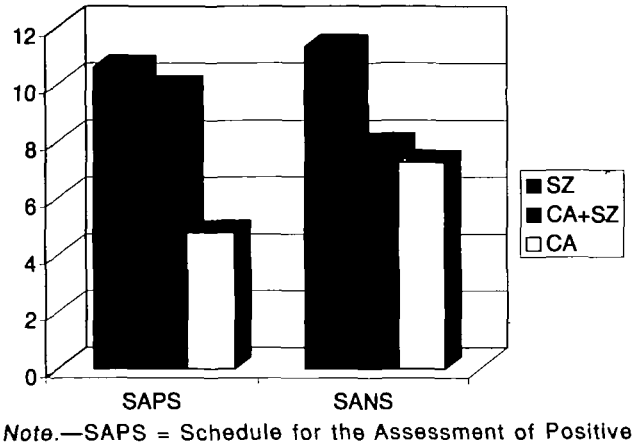
Table 2. Clinical scale scores for patient groups

	Group					
	SZ		CA+SZ		CA	
Scale	M	SD	M	SD	M	SD
SAPS						
Hallucinations[1]	7.75	7.58	12.68	7.74	6.20	5.68
Delusions[2]	15.63	9.68	17.87	10.49	8.06	9.49
Thought disorder[2]	7.90	7.10	6.00	8.53	1.83	3.83
Bizarre behavior	3.38	3.37	3.09	3.45	2.33	1.98
Global[2]	10.59	13.94	9.87	4.46	4.80	3.48
SANS						
Alogia[3]	4.48	3.54	3.65	3.00	2.29	2.59
Flat affect	11.34	8.37	7.70	6.19	8.29	5.88
Anhedonia[4]	9.46	5.37	6.25	6.25	7.00	5.07
Avolition/Apathy	3.92	2.80	2.84	2.04	2.74	2.09
Inattention[3]	2.64	2.49	2.34	1.94	1.22	1.58
Global[5]	11.31	5.17	7.87	4.29	7.26	3.10
BPRS						
Thought disturbance[2]	16.83	6.03	16.83	5.42	11.24	5.15
Activation	5.29	2.09	5.26	1.89	4.16	2.24
Anergia[4]	7.50	4.26	5.43	2.83	6.68	3.50
Hostility	6.16	2.69	5.93	2.62	6.24	1.81
Anxiety/Depression	10.72	4.18	12.84	3.85	12.70	4.23

Note.—SZ = schizophrenia patients; CA+SZ = cocaine-abusing schizophrenia patients; CA = cocaine-abusing patients; M = mean; SD = standard deviation; SAPS = Schedule for the Assessment of Positive Symptoms; SANS = Schedule for the Assessment of Negative Symptoms; BPRS = Brief Psychiatric Rating Scale.

[1]CA +SZ > SZ, CA
[2]SZ = CA + SZ > CA
[3]SZ > CA
[4]SZ > CA + SZ
[5]SZ > CA + SZ, CA

Figure 1. Global SAPS and SANS scores at ER presentation



Note.—SAPS = Schedule for the Assessment of Positive Symptoms; SANS = Schedule for the Assessment of Negative Symptoms; ER = emergency room; SZ = schizophrenia patients; CA+SZ = cocaine-abusing schizophrenia patients; CA = cocaine-abusing patients.

2,113, $p = 0.001$), and delusions ($F = 8.57$, $df = 2,113$, $p < 0.001$) subscales. Tukey-Cramer post-hoc tests found the CA+SZ group to have more hallucinations than either the CA or the SZ patients, who did not differ, and the SZ and CA+SZ groups to have significantly more global positive symptoms than CA patients, with CA+SZ falling in the middle, not differing significantly from the other two groups.

On the SANS, the overall multivariate effect was significant (Pillais approximate $F = 2.34$, $df = 10,216$, $p < 0.02$). When univariate ANOVAs were performed, significant group differences were found on all the subscales: affective flattening (F=3.54, $df = 2,112$, $p < 0.03$), alogia ($F = 5.54$, $df = 2,112$, $p < 0.01$), anhedonia-asociality ($F = 4.77$, $df = 2,112$, $p < 0.01$), avolition-apathy ($F = 3.15$, $df = 2,112$, $p < 0.05$), inattention ($F = 4.03$, $df = 2,112$, $p < 0.02$), and global ($F = 10.11$, $df = 2,112$, $p < 0.001$). Tukey-Cramer post-hoc followup tests revealed that SZ patients had significantly more global negative symptoms than CA+SZ or CA, who did not differ, and significantly more anhedonia-asociality than CA+SZ, with CA patients not significantly differing from either group. CA patients had significantly less inattention than either CA+SZ or SZ patients, who did not differ, and CA patients had significantly less alogia than SZ, with CA+SZ not significantly differing from either group. For the affective flattening and avolition-apathy subscales, no Tukey-Cramer test differences were detected.

[1]We also recomputed the ANOVA for the hallucinations subscale, selecting only patients who had hallucinations rated as mild or greater on the SAPS and found the identical pattern of results: CA+SZ had significantly more hallucinations than CA or SZ patients ($F = 3.24$, $df = 2,87$, $p = 0.04$).

On the BPRS, the overall multivariate effect was also significant (Pillais approximate $F = 3.50$, $df = 10,208$, $p < 0.01$). (Two CA+SZ subjects were unwilling to complete the BPRS and were excluded from these analyses.) Univariate ANOVAs revealed a significant difference on the anxiety/depression (F =3.02, $df = 2,111$, $p < 0.05$), anergia ($F = 2.95$, $df = 2,111$, $p < 0.05$), and thought disorder ($F = 8.07$, $df = 2,111$, $p < 0.01$) subscales. Tukey-Cramer followup tests revealed that SZ and CA+SZ groups had significantly more positive symptoms than CA patients as measured by the thought disorder subscale, and CA+SZ had fewer negative symptoms than SZ patients as measured by the anergia subscale; CA patients did not differ significantly from either group. No Tukey-Cramer test differences were detected for the anxiety/depression subscale.

Discussion

CA+SZ patients manifested a mood and negative symptom profile that paralleled CA patients and a global positive symptom profile that closely resembled SZ patients. CA+SZ patients, however, differed from both the SZ and CA groups by presenting with significantly more hallucinatory behavior than either their schizophrenic-diagnosis-only or cocaine-abuse-only counterparts.

Both CA+SZ and CA groups presented with a trend toward more depression and anxiety-related complaints compared with SZ patients. Although nonsignificant, greater anxiety and greater depression in both cocaine-abusing groups suggest that these patients may be beginning to present cocaine withdrawal symptoms (i.e., "cocaine crash"; Gawin and Kleber 1986). Cocaine abuse has been associated with increased depression and suicidal ideation in nonschizophrenia and schizophrenia patients (Sevy et al. 1990; Gold 1993; Serper et al. 1995). In the present report, cocaine-abusing patients were approached within hours after last cocaine use and consequently they may not have reached peak anxiety/depression levels at the time of assessment. Perhaps anxiety and depression symptoms in both cocaine groups would increase during the days following cocaine abstinence (Gawin and Kleber 1986).

For positive symptoms, both schizophrenic groups presented with more psychosis than the nonschizophrenia cocaine-abusing patients. CA+SZ patients, despite their recent stimulant use and increased hallucinatory behavior, did not present as *globally* more psychotic than nonabusing schizophrenia patients. This is consistent with past investigations that found no increase in schizophrenia patients' psychosis after amphetamine or cocaine use

(e.g., Kornetsky 1976; Brady et al. 1990; Serper et al. 1995, 1996). Kornetsky (1976) found, for example, that schizophrenia patients were *hypo*sensitive to the effects of amphetamine, even when they were neuroleptic free. This finding suggests that psychostimulants produce transient or mild elevations of psychosis in schizophrenia patients compared with other groups. Similarly, Brady et al. (1991) found that an increase in psychosis was seldom reported by their cocaine-abusing schizophrenia patients.

Alternatively, it may be the case that cocaine intoxication, unlike recent amphetamine use, infrequently results in psychosis. Angrist (1987), in a review of the history of cocaine use, argued convincingly that cocaine psychosis in nonschizophrenia individuals is an uncommon phenomenon, in part because cocaine has a short half-life requiring continuous and larger doses to produce and maintain a psychosis. This conclusion is consistent with our findings regarding the positive symptom profile of the nonschizophrenia cocaine-abusing patients in the present study. Despite their heavy cocaine use within 24 hours before emergency presentation, CA patients manifested few positive symptoms relative to schizophrenia patients; instead, they sought treatment much more frequently for depression and anxiety symptoms. This behavior is, however, inconsistent with other investigations that report significant psychosis in recently admitted nonschizophrenia cocaine abusers (e.g., Sherer 1988; Brady et al. 1991).

Although a global increase in positive symptoms for CA+SZ patients was not found, CA+SZ patients did present with significantly more hallucinations than either CA or SZ patients. Cocaine has been reported to produce hallucinations in nonschizophrenia individuals (e.g., Brady et al. 1991; Gold 1993). It is possible, in the present study, that additive or synergistic effects of cocaine and acute schizophrenic illness combine to produce more hallucinations than either acute schizophrenia or cocaine use can produce alone. This possibility is less likely because past investigators have failed to find a psychostimulant-induced exacerbation of hallucinations even after direct administration of amphetamines to schizophrenia patients (Janowsky et al. 1973; Kornetsky 1976). It is also unclear why cocaine use by acutely ill schizophrenia patients would specifically produce more hallucinatory behavior and not result in a worsening of other positive symptoms, like paranoia, which also has been associated with cocaine use in schizophrenia and nonschizophrenia subjects (Sherer 1988; Brady et al. 1990, 1991; Satel and Edell 1991).

One possible alternate explanation is that CA+SZ patients' increase in hallucinatory behavior was secondarily caused by sleep deprivation. Prolonged cocaine use has been reported to result in significant sleep disturbance (Gold 1993; Lacayo 1995), and profound sleep disturbances have been related to the development of psychotic symptoms (e.g., Keshavan et al. 1995). It may be that in the present study CA+SZ subjects were severely sleep deprived and thus more hallucinatory. We did not, however, directly assess sleep disturbance symptoms in our subjects, so this hypothesis is highly speculative. Furthermore, this theory does not explain why CA subjects, like their cocaine-abusing SZ counterparts, were also not severely hallucinatory at the time of assessment. Overall, it is unclear why the combined effects of recent cocaine abuse and acute schizophrenic illness on positive symptoms are associated solely with increased hallucinatory behavior.

Consistent with our hypotheses, we also found CA+SZ subjects presented with significantly fewer negative symptoms than non-substance-abusing schizophrenia patients. Past investigations (Angrist et al. 1982; van Kammen and Boronow 1988; Goldberg et al. 1991; Serper et al. 1995, 1996) have found psychostimulants to be associated with fewer negative symptoms in schizophrenia patients. In the present study, we found that the acute CA+SZ negative syndrome not only differed from that of their nonabusing SZ counterparts, but also is reduced to levels that closely resembled the negative symptom profile of nonschizophrenia CA patients.

Three important limitations to the above discussion should be noted. First, the CA group was not given a SCID interview to confirm a substance abuse diagnosis and definitively rule out a schizophrenia diagnosis. Another important limitation is that our patients were not randomly assigned to groups; instead, they self-selected for cocaine abuse. It may be, therefore, that the differences in emergency room symptom presentation between SZ and CA+SZ groups reflect baseline or intrinsic characteristics that are independent of cocaine abuse status. Tsuang et al. (1982), for example, hypothesized that substance abusing schizophrenia patients represent a higher-functioning subgroup and consequently present with fewer negative signs. This possibility cannot be ruled out from the present study's naturalistic design. Finally, the lack of differences in SZ and CA+SZ patients' overall level of psychosis may have been caused by a "psychosis ceiling effect." That is, the SAPS scale may not have been a sensitive enough measure for detecting true differences in the severity of CA+SZ level of psychosis. This possibility is unlikely, however, given each group's mean SAPS score. Longitudinal studies examining well-characterized samples of acute schizophrenia patients presenting consecutively in intoxicated and in drug-free states would help to clarify the effects of cocaine on these patients' emergency presentation.

If confirmed by future studies, the present findings indicate that cocaine and comorbid patient groups present

with overlapping but distinct clinical profiles at emergency presentation. Detection of differential symptom patterns in cocaine-intoxicated and comorbid schizophrenic presentations early in the intake process may help obtain more accurate diagnoses and more appropriate treatment interventions.

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