Schizophrenia Patients With a History of Severe Violence Differ From Nonviolent Schizophrenia Patients in Perception of Emotions but Not Cognitive Function

Henry Silver, M.B.B.S., B.Med.Sci., D.P.M., F.R.A.N.Z.C.P.; Craig Goodman, Ph.D.; Gabriella Knoll, M.D.; Victoria Isakov, M.D.; and Ilan Modai, M.D., M.H.A.

Background: Impaired processing of emotions may relate to violent behavior in schizophrenia patients. We compared emotional function in schizophrenia patients with and without a history of severe violent behavior.

Method: Tests of identification and differentiation of facial emotions were performed to compare 35 patients with chronic schizophrenia or schizoaffective disorder (DSM-IV criteria) and a history of severe violent behavior with 35 nonviolent schizophrenia patients and 46 healthy controls. Tests of executive function, attention, visual orientation, working memory, memory for faces and objects, and motor function were also administered.

Results: Violent and nonviolent schizophrenia patients showed impaired emotional and cognitive function compared with controls. Violent patients showed a significantly better ability to identify facial emotional expressions but a poorer ability to discriminate between intensity of emotions than nonviolent schizophrenia patients. There was no difference in cognitive performance between the 2 patient groups.

Conclusion: Violent schizophrenia patients may have a better ability to identify facial emotional cues than nonviolent schizophrenia patients but may be less able to assess the intensity of these cues. This trait may contribute to conflict generation and failure to recognize resolution signals, leading to conflict escalation and violence in violence-prone schizophrenia patients.

(J Clin Psychiatry 2005;66:300–308)

Received May 2, 2004; accepted Sept. 2, 2004. From the Brain Behavior Laboratory (Drs. Silver, Goodman, Knoll, and Isakov), Sha'ar Menashe Mental Health Center (all authors), Hadera; and the Bruce Rappaport Faculty of Medicine, Technion Institute of Technology, Haifa (Drs. Silver and Modai), Israel.

Supported by a grant from the Ministry of Immigrant Absorption, Jerusalem, Israel (Drs. Silver and Goodman).

The authors acknowledge the assistance of Pazit Ben Bassat Harar in data management and of Rena Kurs in preparation of the manuscript.

Corresponding author and reprints: Henry Silver, M.B.B.S., Sha'ar Menashe Mental Health Center, Mobile Post Hefer 38814, Israel (e-mail: mdsilver@tx.technion.ac.il).

Most schizophrenia patients are not violent, but studies confirm a small but significant association between schizophrenia and violence.[1] Although the causes are unknown, severity of clinical symptoms, substance abuse, neurologic dysfunction, electroencephalogram abnormalities, and head injury have been linked with violent behavior in schizophrenia.[2,3] The relationship to neuropsychological function is unclear, with reports of worse,[4] similar, or even better test performance in violent compared with nonviolent patients.[5,6]

Processing of facial emotional stimuli can be distinctly affected in schizophrenia,[7–11] although it is not clear whether this is part of the general cognitive impairment associated with the illness or an independent deficit.[12–14] Impaired emotional processing may affect functional outcome independently of symptoms[15] and has been associated with abnormal activation of limbic areas.[16]

Schizophrenia patients benefit less than healthy individuals from greater stimulus intensity when recognizing facial emotions[17] and are poor at using contextual clues in social judgments.[18] Furthermore, there may be differential impairments in recognition of different emotions and of emotional versus neutral facial expressions.[17] Interpretation of emotional signals is important in conflict generation and resolution,[19] so it is possible that impairment in emotion processing may be related to violent behavior independently of cognitive or clinical aspects of the illness.

Most violent incidents occur between familiar individuals involved in ongoing relationships. The ability to correctly interpret the intentions of the perceived opponent is a key aspect of conflict resolution.[19] Schizophrenia patients are particularly vulnerable to the emotional intensity of environments such as occur in families with high levels of expressed emotion.[20] In emotionally intense settings, rapid and accurate assessment of emotional valence and its intensity may be critically important in determining the nature of the interaction. Emotional intensity is associated with greater schizophrenia relapse rates and is often accompanied by aggressive behavior.[21] Specific interpersonal and social contexts may increase the risk of violence in schizophrenia, and patients who perceive hostility from others are more likely to engage in hostile threats and acts.[22] Failure to recognize the intensity of

critical or hostile attitudes of others may lead violence-prone patients to remain in emotionally charged high-risk situations rather than withdraw.[23]

Deficits in processing of affective information have been described in criminal offenders with psychopathic personality,[24] but we found no studies of relationships between emotion processing and violence in schizophrenia. To provide such data, we examined the relationship between emotional function and violence in schizophrenia patients and tested the hypothesis that impaired emotion processing is related to violent behavior in schizophrenia.

Methods of assessing emotion processing vary, and this may influence findings.[25] We chose to study recognition of facial emotional expressions. This function has been consistently reported to be impaired in schizophrenia patients[7–13,17] and can be assessed with tests that have been proven reliable and sensitive to change.[11,13,26,27]

We asked the following questions: (1) Do violent schizophrenia patients differ from nonviolent schizophrenia patients in the ability to identify facial emotional expressions? (2) Do violent schizophrenia patients differ from nonviolent schizophrenia patients in the ability to discriminate between the intensity of facial expressions of the same valence? (3) If both patient groups have similar levels of cognitive function, do they differ in emotion recognition?

Tests were chosen to assess a comprehensive range of neuropsychological functions. Based on evidence from previous studies that identification and discrimination of facial emotional expressions may be different psychological processes,[13] we assessed emotion recognition by testing the ability to identify facial emotional expressions and the ability to discriminate between the intensity of 2 facial expressions of the same valence, placed side by side.

Cognitive testing was conducted with the aim of assessing neuropsychological functions served by different brain regions and systems. Multiple cognitive deficits are well recognized in schizophrenia patients and have been proposed to be linked to violence,[2–4] but specific associations have not been demonstrated.[28] We included tests of "frontal" functions such as executive processing and working memory, tests of general cognitive processes such as attention and motor processing, and domain-specific tests reflecting the function of localized cortical and subcortical neural substrates.

Choices of domain-specific tests were guided by evidence from lesion[29–33] and functional magnetic resonance imaging[34,35] studies that perception of faces and of objects is organized by a well-circumscribed neural system that includes the fusiform gyrus and ventral occipitofrontal cortex. Transient memory for faces is associated with activity within the inferotemporal cortex, a brain region concerned with recognizing faces.[35–37] The systems may be lateralized to the right hemisphere.[38]

Studies of frontal processes are complicated by the inconsistent application of terms such as *executive control, abstract thinking, planning, inhibition,* and *decision-making*.[39–41] Furthermore, even when agreement exists on their definitions, these terms are descriptive. They may help systematize behavioral data but do not indicate the neural mechanisms involved or the functional relations between them.[42] Furthermore, traditional "frontal" tests such as the Wisconsin Card Sorting Test measure several cognitive components including information maintenance, abstraction, set shifting, and inhibition of previously rewarded responses.[43] To overcome some of these difficulties and provide simpler measures of frontal function, we chose an executive test designed to segregate simple abstraction from storage and maintenance requirements[44] and included tests of working memory.

To reduce stimulus variability and intersubject variability and enhance the cross-cultural generalizability of the results, we used only visually presented, nonverbal stimuli and limited participation to clinically stable male patients. We included a healthy control group for comparison.

METHOD

Study Population

The schizophrenia patient group with a history of violence (VH) included 35 men hospitalized in a maximum-security unit at Sha'ar Menashe Mental Health Center (Hadera, Israel), a national tertiary referral facility for treatment of patients who have committed serious violent crimes and whose subsequent violent behavior in closed wards of standard psychiatric hospitals required transfer to a maximum security environment. Crimes committed by patients in the sample included murder, rape, and recurrent violent acts against persons. All patients were hospitalized in the facility for at least 6 months prior to recruitment to the study.

The patient control group consisted of 35 male chronic schizophrenia patients with no history of severe violence (NVH) who were hospitalized for at least 6 months in the standard wards of the same hospital.

Inclusion criteria for recruitment to the study included fulfillment of DSM-IV diagnostic criteria for schizophrenia or schizoaffective disorder and a minimum of 2 years' duration of illness. Patients with clinically significant depression (DSM-IV diagnosis), brain damage, or abuse of alcohol or illicit drugs while in the hospital, while on leave from the hospital, or in the 6 months prior to admission were excluded. Diagnosis was by consensus among treating and research clinicians (all senior psychiatrists), based on clinical interview and observational, case note, and collateral information.

Diagnoses in the VH group included the following schizophrenia subtypes: paranoid (N = 31), disorganized

(N = 1), residual (N = 2), and schizoaffective (N = 1). Three patients had an additional lifetime diagnosis of substance abuse. The diagnoses in the NVH group included the following schizophrenia subtypes: paranoid (N = 24), residual (N = 6), undifferentiated (N = 1), and schizoaffective (N = 4). One patient had an additional lifetime diagnosis of substance abuse.

The proportion of patients with the paranoid subtype of schizophrenia did not differ significantly between the 2 groups (Fisher exact test, p = .08).

Participants were treated with the same medication continuously for at least 3 months with a constant dose for at least 4 weeks prior to the study. All received antipsychotic drugs: 9 patients in the VH group (risperidone or clozapine) and 33 in the NVH group (risperidone, olanzapine, or clozapine) received atypical antipsychotics, and the rest received various typical antipsychotics. Fourteen patients in the VH group (biperiden N = 10, trihexyphenidyl N = 4) and 11 patients in the NVH group (biperiden N = 10, amantadine N = 1) received antiparkinsonian drugs.

A control group of 46 healthy male volunteers drawn from hospital staff and the community was also tested. Control participants reported no history of violence, psychiatric illness, brain injury, or drug or alcohol abuse on questioning. They were matched for age (mean = 35.47 years, SD = 10.54) and education levels (mean = 11.36 years, SD = 1.73) with the patient group.

All participants provided written informed consent for participation in the study after receiving a full explanation of the test procedures. The study was approved by the institutional ethics committee.

Clinical Assessments

Clinical symptoms were assessed using the Scale for the Assessment of Positive Symptoms (SAPS)[45] and Scale for the Assessment of Negative Symptoms (SANS),[46] and extrapyramidal side effects were assessed with the Simpson-Angus Scale for Extrapyramidal Side Effects (SAS).[47]

Critical incidents. Hospital policy requires reporting of all incidents involving aggression, and these reports were gleaned from ward records. In the VH group, the mean number of reported critical incidents in the 6 months preceding the study was 2.91 (SD = 4.62; range, 0–15). No critical incidents were recorded in the same time period for patients in the NVH group.

Neuropsychological Assessments

Emotions

Identification of facial emotions (Penn Emotion Acuity Test[48]). The Penn Emotion Acuity Test (PEAT) contains 40 black-and-white pictures depicting happy (N = 10), sad (N = 10), and neutral (N = 20) facial emotional expressions. The pictures are presented one at a time, and the participant is asked to rate the emotional valence of each face on a 7-point scale: very happy, moderately happy, somewhat happy, neutral, somewhat sad, moderately sad, and very sad. The percentages of total correct responses and of correct responses for happy, sad, and neutral emotional expressions were the outcome measures.

Differentiation of facial emotions.[14] For the differentiation of facial emotions (EmDiff scale), the participant is asked to indicate whether the emotional expressions on a pair of faces placed side by side differ in intensity. The pairs express the same valence (happy or sad) and use the same set of pictures as the PEAT[48] with an equal number of pictures for each valence (20 happy and 20 sad pairs). There are 3 possible responses (A = B, A > B, A < B). The outcome measure used was the mean of the percentage of correct responses for happy and for sad expressions.

Cognition

Abstraction, Inhibition, and Working Memory task.[44] This test is designed as a measure of abstraction and concept formation with and without additional working memory loads. It presents subjects with 5 shapes: 2 in the upper right and 2 in the upper left corner of a computer screen, with a fifth target object appearing in the center of the screen, below the other stimuli. The participant's task is to pair the target object with the objects on either the left or right. On half of the trials, an additional working memory maintenance requirement is superimposed on this basic module by adding a delay between the presentation of the target and other objects. The total number correct for trials without working memory requirements was selected as the performance measure.

Penn Face Memory Test.[49] This test consists of 20 target faces and 40 foils (20 for each test trial). Stimuli are black-and-white photographs of faces balanced for gender and age. All faces are of neutral emotional expression, as determined by 12 raters. The total number of true positive responses for short delay periods was the performance measure.

Visual Object Learning Test.[50] This test was designed as a spatial analog of the California Verbal Learning Test.[51] It uses 20 Euclidean shapes as learning stimuli that are presented over 4 learning trials, followed by short and long delay test recall. New distracter shapes are used in every test trial. The total number of true positive responses for short delay was selected as the performance measure.

Computerized Judgment of Line Orientation.[52] This is a computerized adaptation of the original paper-and-pencil task. Participants are shown 2 lines at an angle and are asked to indicate the corresponding lines on a simultaneously presented array. The number of correct responses was chosen as the performance measure.

Table 1. Sociodemographic and Illness Variables in Schizophrenia Patients With and Without a History of Severe Violence

	Violent Patients (N = 35)		Nonviolent Patients (N = 35)		Statistics	
Variable	Mean	SD	Mean	SD	F	p
Age at first hospitalization, y	26.15	9.30	26.88	9.38	0.09	.77
Length of illness, y	11.54	9.79	13.10	10.65	0.30	.58
No. of hospitalizations	4.60	6.95	3.00	4.23	1.41	.24
Length of current hospitalization, y	1.87	0.31	5.20	0.85	0.69	.41
SANS score	56.19	21.72	42.22	20.77	6.22	.02
SAPS score	24.84	23.37	27.14	17.75	0.18	.67
Simpson-Angus Scale score	6.10	5.53	1.96	2.58	11.06	.001

Abbreviations: SANS = Scale for the Assessment of Negative Symptoms, SAPS = Scale for the Assessment of Positive Symptoms.

Penn Continuous Performance Test.[53] This test is a measure of sustained attention developed for use in functional neuroimaging studies. During this task, the participant is asked to respond to a set of vertical and horizontal lines (a 7-segment display) whenever they form a digit. The total number of true positive responses was the performance measure.

Digit span (verbal working memory). The backward digit span test from the Wechsler Adult Intelligence Scale Version 1[54] was used to assess verbal working memory.

Dot Test Modified (visual working memory).[55] The subject is presented with a card on which a mark is present. After a 10-second interval, the paper is removed, and the subjects are immediately asked to reproduce the mark on a blank card. To facilitate measurement, a cross was used instead of a dot and 10 cards were presented. The sum (in millimeters) of the distance between the target mark and that recalled by the subject was the outcome measure.

Mini-Mental State Examination.[56] General cognitive function was tested with the Mini-Mental State Examination.

Finger Tapping Test.[57,58] The Finger Tapping Test examines the ability to make rapid repetitive movements. The test was modified, and patients were asked to tap with the index finger on 2 points set 30 cm apart as rapidly as possible. Each hand was tested separately. The outcome measure was the number of taps per minute with the dominant hand.

The clinical rater was independent of the neuropsychological tester and unaware of test results.

Data Analysis

Test scores in the 2 groups were compared using t tests. Analyses of variance (ANOVAs) with clinical symptoms as covariates were used to control for these as potential confounds. Not all participants completed all tests, and therefore the sample sizes differ slightly. Spearman r, which is less affected by outlying values than Pearson r, was used for correlations.

Two-tailed significance tests with the significance level set to 5% were used.

RESULTS

The VH and NVH patient groups did not differ in age, length of illness, number of admissions, duration of current hospitalization, age at first hospitalization, or SAPS scores, but VH patients had significantly higher SANS and SAS scores (Table 1). To test whether the differences in negative symptoms between the patient groups may be explained by differences in extrapyramidal symptoms (SAS score), we performed an ANOVA with SANS scores as dependent variables, group (VH, NVH) as independent variable, and SAS scores as covariate. This analysis showed no significant difference in SANS scores between the 2 groups ($F = 0.25$, $df = 1,53$; $p = .6$).

Cognitive Functions

As expected, patients performed worse than healthy participants on all cognitive tests (Table 2). There was no significant difference in cognitive test performance between the VH and NVH patient groups.

Identification of Emotions

Patients performed worse than healthy controls in the identification of facial emotional expressions (total PEAT score) (Figure 1). Among patients, the VH group performed significantly better than the NVH group. ANOVA with total PEAT score as the dependent measure and group (NVH, VH, control) as the between-subject factor showed a significant group effect ($F = 18.83$, $df = 2,111$; $p = .0001$). Post hoc analysis of contrasts (Bonferroni) showed significant differences between controls and the NVH (CI = 0.1445 to 0.2824, $p = .0001$) and VH (CI = 0.0173 to 0.1552, $p = .015$) groups and between the NVH and VH (CI = –0.2011 to – 0.0533, $p = .001$) patient groups.

Comparison of identification of emotional and neutral stimuli. To compare identification of facial expressions conveying emotion (sad or happy) with that of neutral facial expressions, we calculated an identification of emotions measure by computing the mean of scores for identification of sad and happy facial expressions and comparing it with identification of neutral facial expressions. We performed an ANOVA with

Table 2. Cognitive Function in Violent and Nonviolent Patients and Healthy Controls[a]

	All Patients (N = 70)		Nonviolent Patients (N = 35)		Violent Patients (N = 35)		Healthy Controls (N = 46)	
Measure	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Visual orientation	19.99	6.02	20.35	7.45	19.63	4.28	23.80	4.01
Attention	30.99	7.41	29.71	9.56	32.26	4.11	35.22	1.81
Verbal working memory	4.51	1.93	4.65	1.99	4.37	1.88	8.47	2.43
Spatial working memory[b]	140.20	68.14	148.07	84.60	131.89	44.48	111.38	29.16
Executive	20.99	3.88	20.50	3.98	21.46	3.78	24.35	1.75
Memory for faces	27.48	3.42	27.50	2.83	27.46	3.96	30.26	2.50
Memory for objects	13.12	2.61	13.29	2.60	12.94	2.64	15.98	2.29
Motor	99.17	27.05	95.03	25.48	103.54	28.33	129.70	33.15
Mini-Mental State Examination	25.76	3.24	26.05	3.28	25.44	3.22	29.23	1.32

[a]Patients versus controls: significant at $p < .001$ on all tests. Nonviolent versus violent patients: nonsignificant on all tests.
[b]Lower score indicates better performance.

Figure 1. Identification of Happy, Sad, and Neutral Facial Expressions in Patients and Controls

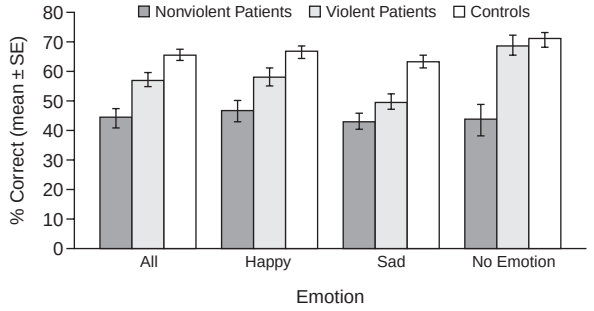
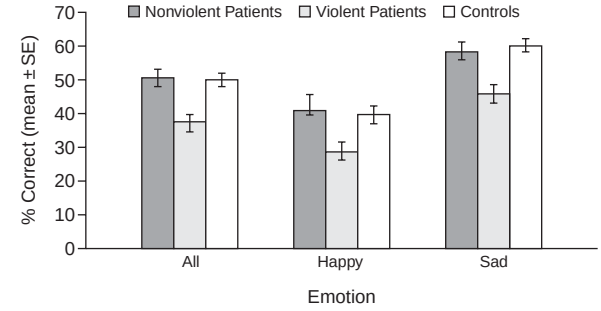


Figure 2. Differentiation of Facial Emotional Expressions in Patients and Controls



emotion (emotion measure vs. neutral) as within-subject variable and group (NVH, VH, control) as between-subject factor. This analysis showed significant effects of emotion ($F = 22.19$, $df = 1,111$; $p = .0001$) and of group ($F = 17.81$, $df = 2,111$; $p = .0001$) and significant emotion-by-group interaction ($F = 11.006$, $df = 2,111$; $p = .0001$).

ANOVAs conducted within each group showed significantly better identification of neutral expressions than of emotional expressions within the VH ($F = 46.91$, $df = 1,33$; $p = .0001$) and control ($F = 8.95$, $df = 1,45$; $p = .004$) groups but no difference within the NVH group ($F = 0.241$, $df = 1,33$; $p = .62$).

Differentiation of Facial Emotions

VH patients performed worse than NVH patients and healthy controls on the test of differentiation of emotions (EmDiff) (Figure 2). We performed an ANOVA with total EmDiff score as the dependent variable and group as the between-subject factor. This analysis showed a significant effect of group ($F = 11.085$, $df = 2,112$; $p = .0001$). Post hoc analysis of contrasts (Bonferroni) showed significant difference between the control and VH (CI = 0.0680 to 0.1907, $p = .0001$) and NVH and VH groups (CI = 0.0687 to 0.2004, $p = .0001$) but not between the control and NVH groups (CI = –0.0670 to 0.0566, $p = .868$).

The results were the same for happy as for sad expressions. We performed an ANOVA with valence (happy, sad) as within-subject variable and group (NVH, VH, control) as the between-subject factor. This analysis showed a significant effect of valence ($F = 145.56$, $df = 1,112$; $p = .001$) and of group ($F = 11.085$, $df = 1,112$; $p = .0001$) but no significant valence-by-group interaction ($F = 1.032$, $df = 2,112$; $p = .36$).

Effect of Clinical Symptoms on Identification and Differentiation of Emotions

To examine the effect of negative symptoms, we repeated the ANOVA analyses with the PEAT or EmDiff test scores as dependent variables, group (VH/NVH) as the independent variable, and SANS score as covariate. The results remained highly significant. The results of the analyses were also unchanged when extrapyramidal side effect severity (SAS score) was added to the SANS score as a second covariate. The effect of positive symptoms was tested in a similar way by using SAPS score as covariate, and no effect of positive symptoms was found.

The analyses were repeated in the subgroup diagnosed with paranoid schizophrenia and showed similar results.

Relation Between Perception of Emotions and Recent Aggressive Behavior

There was no correlation between the number of recent critical incidents (a measure of violent behavior in the period preceding the study) and performance on cognitive or emotional tests in the VH group.

To examine further whether differences in perception of emotions between VH and NVH patients were related to presence of recent violent behavior, we repeated the analyses in a subgroup of patients with no reported critical incidents in the 6 months preceding the study (no violent "state" in relation to testing). The results were similar to those of the entire patient group.

Performance on tests of identification of emotions (PEAT) showed no significant correlation ($r = -.05$, $p = .7$) with discrimination of emotions (EmDiff) in the study population or within the patient or healthy groups.

DISCUSSION

The impaired neuropsychological performance found in our patients compared with healthy individuals is consistent with extensive evidence of cognitive impairments in schizophrenia.[11,13,59–61]

Violence and Emotional Recognition

Novel results of this study include the findings that VH patients performed differently on tests of recognition of emotions than NVH patients while showing similar cognitive functions.

VH patients identified facial emotional expressions more accurately than NVH patients, although, consistent with previous studies,[7–14] both patient groups were significantly impaired compared with healthy individuals. We interpret this finding as indicating that violence-prone schizophrenia patients may be more aware than nonviolent schizophrenia patients of emotional content of facial expressions and hence more readily stimulated by emotional clues.

We found no previous reports of a relationship between violence and emotional processing in schizophrenia patients, but studies reporting that paranoid schizophrenia patients show better facial affect perception than nonparanoid patients have been published.[62,63] Herpertz et al.[24] reported impaired processing of affective information in criminal offenders with psychopathic personality disorder. It has also been reported that schizophrenia patients may have greater impairment in emotional processing than affectively ill persons.[64,65]

Identification of Emotional Versus Neutral Expressions

The ability to identify emotional relative to neutral facial expressions differed between VH and NVH patients. NVH patients were equally impaired in identifying emotional (happy or sad) and neutral facial expressions. This finding is consistent with Kohler et al.,[17] who, using color pictures of faces representing basic emotions, found that schizophrenia patients were more impaired in recognition of neutral expressions than happy or sad expressions compared with healthy controls. In contrast, VH patients, like healthy participants, were better at identifying neutral than emotional expressions. The greater ability of VH patients to identify the absence of emotional cues in facial expressions may paradoxically have some undesirable consequences. Schizophrenia patients demonstrate a bias when identifying neutral emotional expressions,[10,66] tending to misinterpret neutral expressions as happy or sad.[17] Therefore, in potentially confrontational situations, nonviolent patients may (mistakenly) interpret neutral facial expression as conveying appeasing (sad or happy) signals favoring conflict resolution. In contrast, violence-prone patients lacking this bias will recognize the expression as neutral and not be appeased.

Identification Versus Differentiation of Emotions

The second novel finding was that VH patients were more impaired than NVH patients in discriminating between facial emotional expressions placed side by side. The dissociation between identification of facial emotions and their discrimination is consistent with previous findings that these are separate skills.[13] The findings are consistent with evidence that schizophrenia patients benefit less than healthy individuals from greater stimulus intensity when recognizing facial emotions[17] and are poor at using contextual clues in social judgments[20] and indicate that violence-prone patients may have selectively greater impairment compared with nonviolent patients in using intensity clues.

Based on our findings, we propose that violent schizophrenia patients, although impaired in emotional processing compared with normal controls, may be better able than nonviolent schizophrenia patients to use stimulus and contextual clues to identify the presence (or absence) of emotional signals in facial expressions but less able to use these clues to accurately assess the intensity of the emotional expressions. This greater attentiveness to emotional aspects of facial expression may lead to more intense emotional engagement with others in violence-prone patients than in less emotionally aware schizophrenia patients who are not violence-prone. Once violence-prone patients are emotionally engaged, their poorer ability to judge the intensity of emotional expressions can lead to responses that are not appropriate to the emotional message sent.

Since our patient groups were categorized by a history of violence and not current violent behavior (many in the VH group had no aggressive behaviors recorded in the 6 months preceding the study), we interpret these differences in perception of emotions as reflecting trait- rather

than state-like characteristics of schizophrenia patients at risk for violent behavior.

We examined only happy and sad emotions in this study. The generalizability of our findings to other emotions is as yet unknown and is under investigation. The impaired ability of violence-prone patients to correctly assess the intensity of happy or sad emotions may impede conflict resolution, as appeasement signals may not be identified. There is evidence that schizophrenia patients and healthy individuals may be more accurate in identifying happy or sad than angry facial expressions[17] (H.S., manuscript in preparation), but the relative ability of violent and nonviolent patients to discriminate between degrees of angry stimuli has not been tested. If the discrimination of anger intensity is similar to that of happy and sad expressions, it can be postulated that in emotionally charged settings violence-prone patients would be more likely than nonviolent patients to misinterpret mild annoyance as rage, resulting in an aggressive response to a perceived threat.

Relation Between Violence and Cognitive Function

The finding that VH and NVH patients showed similar cognitive performance, including on tests of prefrontal lobe functions such as working memory and executive tests, is consistent with that of Lapierre et al.,[5] who found no relation between neuropsychological dysfunction and aggressive behavior. In contrast, Adams et al.[67] found a strong relationship between neurologic impairment and lifetime history of antisocial behavior in violent incarcerated schizophrenia patients.

Evidence regarding the relationship between cognitive function and violent behavior is unclear. In a meta-analysis of reported studies, Morgan and Lilienfeld[28] found evidence for poorer executive function in antisocial than comparison groups but noted that evidence for specificity of executive deficits relative to other neuropsychological tasks was inconsistent. Lapierre et al.[68] compared psychopathic and non-psychopathic criminals and found a relation between psychopathy and ventral frontal deficits.

Our findings indicate that cognitive performance, including performance on some tests of executive functions, does not differentiate between violent and nonviolent schizophrenia patients. However, we studied a chronically ill and cognitively impaired population, and the possibility that poor cognition may be a risk factor in less impaired patients cannot be excluded, nor can the possibility that cognitive impairment may be a general risk factor for violent behavior across different diagnostic and nonpatient groups.

Differential Impairment Between Cognition and Emotional Recognition

The presence of emotion recognition differences between patient subgroups who did not differ in cognitive performance is consistent with evidence that processing of facial emotional stimuli can be affected independently of cognitive functions in schizophrenia.[7–11] However, our study was not specifically designed to test for a differential deficit, and it is possible that factors such as task difficulty or psychometric properties of the tests influenced results. Hence, the possibility that emotional impairment is part of a more general cognitive impairment[12–14] cannot be excluded. Since both patient groups were cognitively impaired compared with controls, cognitive functions that can explain the differences in emotional processing between violent and nonviolent patients may be difficult to identify. The dissociation between the ability to identify facial emotions and to differentiate between degrees of intensity suggests that emotional processing may be heterogeneous and involve several neural substrates.

Study Limitations

All of our patients were treated, and the effect of medication on the results is unknown. More VH than NVH patients received typical antipsychotics, reflected in more severe extrapyramidal symptoms in that group, but this is unlikely to explain the findings. The NVH group received more atypical antipsychotics, which do not impair perception of emotion and may improve cognitive function,[69] yet the group showed poorer performance on PEAT. Likewise, medication effects are unlikely to explain selective differences in neuropsychological performance.

Our findings were not explained by differences in negative symptoms between the groups, consistent with other reports showing a lack of a relationship between negative symptoms and perception of emotions.[13,70,71] Nor were the findings explained by differences in paranoid symptoms, which have been associated with better facial affect perception compared with nonparanoid schizophrenia,[62,63,72] as similar results were found in the subgroup of paranoid schizophrenia patients as in the other patients.

We studied severely impaired, chronically ill patients, and generalization to less impaired patients must be done with caution. In particular, the possibility that cognitive factors may influence violent behavior in less impaired patients cannot be excluded.

We did not study emotions such as anger and fear, which may show different patterns of impairment in schizophrenia than happy or sad emotions,[17] so generalization to other emotions, as mentioned above, must be cautious.

Our findings support the need for further investigations of emotional processing in relation to violent behavior in schizophrenia in a broader range of patients and emotions.

Drug names: amantadine (Symmetrel and others), biperiden (Akineton), clozapine (Clozaril, Fazaclo, and others), olanzapine (Zyprexa), risperidone (Risperdal).

REFERENCES

1. Walsh E, Buchanan A, Fahy T. Violence and schizophrenia: examining the evidence. Br J Psychiatry 2000;180:490–495
2. Swanson JW, Holzer CE III, Ganju VK, et al. Violence and psychiatric disorder in the community: evidence from the Epidemiologic Catchment Area surveys. Hosp Community Psychiatry 1990;41:761–770
3. Heinrichs RW. Frontal cerebral lesions and violent incidents in chronic neuropsychiatric patients. Biol Psychiatry 1989;25:174–178
4. Krakowski MI, Convit A, Jaeger J, et al. Neurological impairment in violent schizophrenic inpatients. Am J Psychiatry 1989;146:849–853
5. Lapierre D, Braun CM, Hodgins S, et al. Neuropsychological correlates of violence in schizophrenia. Schizophr Bull 1995;21:253–262
6. Planansky K, Johnston R. Homicidal aggression in schizophrenic men. Acta Psychiatr Scand 1977;55:65–73
7. Hooker C, Park S. Emotion processing and its relationship to social functioning in schizophrenia patients. Psychiatry Res 2002;112:41–50
8. Gessler S, Cutting J, Frith CD, et al. Schizophrenic inability to judge facial emotion: a controlled study. Br J Clin Psychol 1989;28:19–29
9. Kring AM, Barrett LF, Gard DE. On the broad applicability of the affective circumplex: representations of affective knowledge among schizophrenia patients. Psychol Sci 2003;14:207–214
10. Heimberg C, Gur RE, Erwin RJ, et al. Facial emotion discrimination, 3: behavioral findings in schizophrenia. Psychiatry Res 1992;42:253–265
11. Silver H, Shlomo N, Turner T, et al. Perception of happy and sad facial expressions in chronic schizophrenia: evidence for 2 evaluative systems. Schizophr Res 2002;55:171–177
12. Kerr SL, Neale JM. Emotion perception in schizophrenia: specific deficit or further evidence of generalized poor performance? J Abnorm Psychol 1993;102:312–318
13. Silver H, Shlomo N. Perception of facial emotions in chronic schizophrenia does not correlate with negative symptoms but correlates with cognitive and motor dysfunction. Schizophr Res 2001;52:265–273
14. Kohler CG, Bilker W, Hagendoorn M, et al. Emotion recognition deficit in schizophrenia: association with symptomatology and cognition. Biol Psychiatry 2000;48:127–136
15. Kee KS, Green MF, Mintz J, et al. Is emotion processing a predictor of functional outcome in schizophrenia? Schizophr Bull 2003;29:487–497
16. Gur RC, Schroeder L, Turner T. Brain activation during facial emotion processing. Neuroimage 2002;16(3 pt 1):651–662
17. Kohler CG, Turner TH, Bilker WB, et al. Facial emotion recognition in schizophrenia: intensity effects and error pattern. Am J Psychiatry 2003; 160:1768–1774
18. Penn DL, Ritchie M, Francis J, et al. Social perception in schizophrenia: the role of context. Psychiatry Res 2002;109:149–159
19. de Waal FB. Primates: a natural heritage of conflict resolution. Science 2000;289:586–590
20. Leff J, Vaughn C. Expressed Emotion in Families. New York, NY: Guilford Press; 1985
21. Butzlaff RL, Hooley JM. Expressed emotion and psychiatric relapse: a meta-analysis. Arch Gen Psychiatry 1998;55:547–552
22. Estroff SE, Zimmer C, Lachicotte WS, et al. The influence of social networks and social support on violence by persons with serious mental illness. Hosp Community Psychiatry 1994;45:669–679
23. Joseph PL, Sturgeon DA, Leff J. The perception of emotion by schizophrenic patients. Br J Psychiatry 1992;161:603–609
24. Herpertz SC, Werth U, Lukas G, et al. Emotion in criminal offenders with psychopathy and borderline personality disorder. Arch Gen Psychiatry 2001;58:737–745
25. Edwards J, Jackson HJ, Pattison PE. Emotion recognition via facial expression and affective prosody in schizophrenia: a methodological review. Clin Psychol Rev 2002;22:789–832. Correction 2002;22: 1267–1285
26. Silver H, Feldman P, Bilker W, et al. Working memory deficit as a core neuropsychological dysfunction in schizophrenia. Am J Psychiatry 2003; 160:1809–1816
27. Silver H, Goodman C, Knoll G, et al. Brief emotion training improves recognition of facial emotions in chronic schizophrenia: a pilot study. Psychiatry Res 2004;128:147–154
28. Morgan AB, Lilienfeld SO. A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. Clin Psychol Rev 2000;20:113–136
29. Benton AL, Van Allen MW. Impairment in facial recognition in patients with cerebral disease. Trans Am Neurol Assoc 1968;93:38–42
30. Haxby JV, Horwitz B, Ungerleider LG, et al. The functional organization of human extrastriate cortex: a PET-rCBF study of selective attention to faces and locations. J Neurosci 1994;14(11 pt 1):6336–6353
31. Sergent J, Ohta S, MacDonald B. Functional neuroanatomy of face and object processing: a positron emission tomography study. Brain 1992; 11(5 pt 1):15–36
32. Kanwisher N, McDermott J, Chun MM. The fusiform face area: a module in human extrastriate cortex specialized for face perception. J Neurosci 1997;17:4302–4311
33. Puce A, Allison T, Gore JC, et al. Face-sensitive regions in human extrastriate cortex studied by functional MRI. J Neurophysiol 1995; 74:1192–1199
34. Gauthier I, Nelson CA. The development of face expertise. Curr Opin Neurobiol 2001;11:219–224
35. Li L, Miller EK, Desimone R. The representation of stimulus familiarity in anterior inferior temporal cortex. J Neurophysiol 1993;69:1918–1929
36. Golby AJ, Gabrieli JD, Chiao JY, et al. Differential responses in the fusiform region to same-race and other-race faces. Nat Neurosci 2001; 4:845–850
37. Farah MJ. Is visual imagery really visual? overlooked evidence from neuropsychology. Psychol Rev 1988;95:307–317
38. Wilson FA, Scalaidhe SP, Goldman-Rakic PS. Dissociation of object and spatial processing domains in primate prefrontal cortex. Science 1993;260:1955–1958
39. Baddeley A, Hitch G. Developments in the concept of working memory. Special section: Working memory. Neuropsychology 1994;8:485–493
40. Perry W, Heaton RK, Potterat E, et al. Working memory in schizophrenia: transient "online" storage versus executive functioning. Schizophr Bull 2001;27:157–176
41. Keefe RSE. Working memory dysfunction and its relevance to schizophrenia. In: Sharma T, Harvey P, eds. Cognition in Schizophrenia: Impairments, Importance, and Treatment Strategies. Oxford, England: Oxford University Press; 2000:16–50
42. O'Reilly RC, Noelle DC, Braver TS, et al. Prefrontal cortex and dynamic categorization tasks: representational organization and neuromodulatory control. Cereb Cortex 2002;12:246–257
43. Koren D, Seidman LJ, Harrison RH, et al. Factor structure of the Wisconsin Card Sorting Test: dimensions of deficit in schizophrenia. Neuropsychology 1998;12:289–302
44. Glahn DC, Cannon TD, Gur RE, et al. Working memory constrains abstraction in schizophrenia. Biol Psychiatry 2000;47:34–42
45. Andreasen NC. Scale for the Assessment of Positive Symptoms (SAPS). Iowa City, Iowa: University of Iowa; 1984
46. Andreasen NC. Scale for the Assessment of Negative Symptoms (SANS). Iowa City, Iowa: University of Iowa; 1983
47. Simpson GM, Angus JWS. A rating scale for extrapyramidal side effects. Acta Psychiatr Scand Suppl 1970;212:11–19
48. Erwin RJ, Gur RC, Gur RE, et al. Facial emotion discrimination, 1: task construction and behavioral findings in normal subjects. Psychiatry Res 1992;42:231–240
49. Gur RC, Jaggi JL, Ragland JD, et al. Effects of memory processing on regional brain activation: cerebral blood flow in normal subjects. Int J Neurosci 1993;72:31–44
50. Glahn DC, Gur RC, Ragland JD, et al. Reliability, performance characteristics, construct validity, and an initial clinical application of a visual object learning test (VOLT). Neuropsychology 1997;11:602–612
51. Delis DC, Kramer JH, Kaplan E, et al. California Verbal Learning Test: Adult Version Manual. San Antonio, Tex: The Psychological Corporation; 1987
52. Benton AL, Hannay HJ, Varney NR. Visual perception of line direction in patients with unilateral brain disease. Neurology 1975;25:907–910
53. Kurtz MM, Ragland JD, Bilker W, et al. Comparison of the continuous performance test with and without working memory demands in healthy controls and patients with schizophrenia. Schizophr Res 2001;48: 307–316
54. Wechsler D. Wechsler Adult Intelligence Manual. New York, NY: New York Psychological Corporation; 1955
55. Keefe RSE, Lees-Roitman SE, Dupre RL. Performance of patients with schizophrenia on a pen and paper visuospatial working memory task with short delay. Schizophr Res 1997;26:9–14
56. Folstein MF, Folstein SE, McHugh PR. Mini-Mental State: a practical method for grading the cognitive state of patients for the clinician.

J Psychiatr Res 1975;12:189–198
57. Reitan RM, Davison LA. Clinical Neuropsychology: Current Status and Applications. New York, NY: Hemisphere; 1974
58. Russell EW, Neuringer C, Goldstein G. Assessment of Brain Damage: A Neuropsychological Key Approach. New York, NY: Wiley-Interscience; 1970
59. Saykin AJ, Gur RC, Gur RE, et al. Neuropsychological function in schizophrenia: selective impairment in memory and learning. Arch Gen Psychiatry 1991;48:141–162
60. Morrison RL, Bellack AS, Mueser KT. Deficits in facial-affect recognition and schizophrenia. Schizophr Bull 1988;14:67–83
61. Mandal MK, Pandey R, Prasad AB. Facial expressions of emotions and schizophrenia: a review. Schizophr Bull 1998;24:399–412
62. Davis PJ, Gibson MG. Recognition of posed and genuine facial expressions of emotion in paranoid and nonparanoid schizophrenia. J Abnorm Psychol 2000;109:445–450
63. Lewis SF, Garver DL. Treatment and diagnostic subtype in facial affect recognition in schizophrenia. J Psychiatr Res 1995;29:5–11
64. Walker E, McGuire M, Bettes B. Recognition and identification of facial stimuli by schizophrenics and patients with affective disorders. Br J Clin Psychol 1984;23(pt 1):37–44
65. Gur RC, Erwin RJ, Gur RE, et al. Facial emotion discrimination, 2: behavioral findings in depression. Psychiatry Res 1992;42:241–251
66. Schneider F, Gur RC, Gur RE, et al. Emotional processing in schizophrenia: neurobehavioral probes in relation to psychopathology. Schizophr Res 1995;17:67–75
67. Adams JJ, Meloy JR, Moritz MS. Neuropsychological deficits and violent behavior in incarcerated schizophrenics. J Nerv Ment Dis 1990;178:253–256
68. Lapierre D, Braun CM, Hodgins S. Ventral frontal deficits in psychopathy: neuropsychological test findings. Neuropsychologia 1995;33:139–151. Correction 1995;33:1059
69. Keefe RS, Silva SG, Perkins DO, et al. The effects of atypical antipsychotic drugs on neurocognitive impairment in schizophrenia: a review and meta-analysis. Schizophr Bull 1999;25:210–221
70. Sweet LH, Primeau M, Fichter CG, et al. Dissociation of affect recognition and mood state from blunting in patients with schizophrenia. Psychiatry Res 1998;81:301–308
71. Salem JE, Kring AM, Kerr SL. More evidence for generalized poor performance in facial emotion perception in schizophrenia. J Abnorm Psychol 1996;105:480–483
72. Kline JS, Smith JE, Ellis HC. Paranoid and nonparanoid schizophrenic processing of facially displayed affect. J Psychiatr Res 1992;26: 169–182