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Ronnie G. Stout,[1] *Ph.D. and Rokeya S. Farooque,*[1] *M.D.*

Claims of Amnesia for Criminal Offenses: Psychopathology, Substance Abuse, and Malingering

ABSTRACT: The forensic psychiatric examiner often encounters defendants who deny memory for their offense. Past research proposes a variety of factors to account for offense amnesia. To date there have been few systematic studies of offense amnesia in relation to psychiatric diagnosis, either alone or in combination with other known factors such as substance use and malingering. We studied 53 pretrial felony defendants who had been referred for psychiatric examination; 40% claimed amnesia for their offense. Examinees with psychotic disorders in general, and schizophrenia in particular, were relatively less likely to claim amnesia than were examinees with other diagnoses. Substance use at the time of the offense and associated substance use disorder diagnoses were positively associated with offense amnesia. Malingering diagnosed by general clinical criteria was a poor predictor of amnesia claims. These data suggests that two prominent reasons for referral for forensic psychiatric evaluation include the presence of psychotic symptoms and claims of amnesia for the offense.

KEYWORDS: forensic science, forensic psychiatry, amnesia, crime, mental disorder, substance abuse

The forensic psychiatric examination is often complicated by the subject's claim of inability to remember the alleged offense. Various studies indicate that between one-fourth and two-thirds of homicide offenders claim amnesia for their offense, as do between 10% and about a third of offenders generally (1–5). It is thought that offense amnesia is associated with more violent offenses, especially violent physical attacks upon others (3,6). For example, 19 of the 203 offenders studied by Taylor and Kopelman (5) claimed amnesia for the offense, and in each instance, the subject had been charged with a violent offense, half of these offenses being murder or manslaughter. At the same time, research suggests that defendants who claim amnesia for the offense have an increased likelihood of referral for pretrial psychiatric assessment (2,7).

A number of factors have been proposed to account for, or to be associated with, offense amnesia. Several writers have suggested that offense amnesia may result from extreme emotional arousal, possibly coupled with repressive or dissociative mechanisms, although there is controversy around this issue (1,3,6,8,9). Personality traits such as introversion or "hysterical" traits may be correlated with offense amnesia (10–12). At least one study found lower IQs in examinees who claimed amnesia, but the authors suspected that the amnesia was often feigned (13).

The intersection of memory, substance misuse, violence, and mental disorder is of unique importance to the forensic examiner. There are clear associations between substance use and crime, particularly violent crime (14), and it is well established that offense amnesia is often associated with intoxication (1). A wide variety of drugs impair memory, including alcohol, benzodiazepines, barbiturates, and cannabis derivatives, among others. The typical effect of such substances is impairment of long-term memory while short-term memory is preserved. The consequence for memory is impaired encoding and/or consolidation of new information which may, if sufficiently severe, constitute anterograde amnesia (15–17). In the Taylor and Kopelman (5) sample of offenders, alcoholism was prevalent among subjects claiming offense amnesia. Moreover, more than half of the amnesic subjects had been under the influence of alcohol in the hours before their offense, and amnesia was overrepresented among those who had consumed the most alcohol. In other studies, over 85% of offenders who claimed amnesia reported being under the influence of alcohol and/or drugs at the time of the crime (18,19). A link between offense amnesia and alcohol blackouts has been proposed (3,4,20). Pyszora et al. (2) found that, besides being associated with crimes of passion and psychiatric disorder, amnesia was associated with a history of alcohol abuse and blackouts. Importantly, blackouts are more common when alcohol is combined with other intoxicating substances (21,22).

Furthermore, there is a high prevalence of alcohol and drug use among people with serious mental disorders, which may worsen symptoms as well as heighten risk for violence (23–28). No clear relationship between offense amnesia and psychiatric illness has been identified, but there have been few systematic efforts (1). For example, Cima et al. (13) studied a sample of psychiatric inmates, but aside from psychopathy and IQ, they did not analyze the relationship between diagnosis and amnesia. Depression, dissociation, and schizophrenia have all been mentioned in connection with offense amnesia (11,12).

Studies of the combined memory effects of substance abuse and major mental disorder in nonforensic settings have yielded mixed results. Given that alcohol is the most frequently abused substance among people with schizophrenia, there is noteworthy evidence of greater memory impairment among outpatients with both schizophrenia and alcohol abuse or dependence than among comparison outpatients with schizophrenia alone (29). Cocaine use may confer a similar liability among people with schizophrenia (30). However, Pencer and Addington (31) found no relationship between substance abuse and cognitive function among patients with first-

[1]Forensic Services Program, Middle Tennessee Mental Health Institute, 221 Stewarts Ferry Pike, Nashville, Tennessee 37214.

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episode schizophrenia-spectrum disorders. But it is also true that studies of memory in mental disorder, with or without accompanying substance abuse, have seldom looked at autobiographical memory. Thus, the relevance of such research for offense amnesia is uncertain.

This study was an effort to characterize an available sample of pretrial detainees who had been referred for psychiatric examination and who claimed amnesia for their offenses, in comparison with examinees who did not claim amnesia, in relation to *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV) diagnosis (32), substance use, and malingering. Of particular interest was evidence of any link between psychiatric diagnosis—especially psychotic disorder—and claims of offense amnesia, and the concurrent influence of substance use status.

Method

Subject Selection

We reviewed records of defendants in criminal proceedings who had been referred to a specialty inpatient forensic psychiatric center for a 1-month examination to furnish opinion concerning competence to stand trial and insanity defense. Our institutional review board approved the study.

While remaining blind to all clinical data other than diagnosis, we first screened the available sample of dischargees for substance use disorder diagnoses, to capture a sizeable number of defendants who had likely used drugs or alcohol around the time of the offense. In this manner, we selected 30 subjects with discharge substance use disorder diagnoses between March 1998 and August 2001. Employing a similar blind, we then selected a comparison group of 34 subjects without discharge substance use disorder diagnoses from the same timeframe. Of these, two were omitted because information about their substance use habits could not be corroborated, as described below. One additional patient was omitted because he claimed only partial amnesia. We then reviewed the remaining 61 records and omitted eight who denied committing the alleged offense. Thus, the final sample was composed of 53 examinees.

Data Coding

Diagnoses were arrived at by consensus between one forensic psychiatrist and one of two psychologists. Each diagnostician had at least 20 years of clinical experience. Consultation services such as laboratory and radiology services were available as needed to aid in patient assessment. For purposes of data analysis, multiple Axis I diagnoses were allowed, but mood disorder and schizophrenia were considered non-overlapping diagnoses. Patients diagnosed with schizoaffective disorder were coded as having schizophrenia. An organic brain syndrome category included examinees with a seizure disorder or history of encephalitis. Malingering was a codable diagnosis, but the presence of malingering did not exclude other (genuine) psychopathology. A DSM-IV diagnosis of malingering was made clinically (33), as when there were reports from collateral informants of functioning discrepant from that observed in the hospital, or when two or more of the following features were present: (1) functioning on the residential unit discrepant in characteristic ways from behavior presented in examinations; (2) flagrantly unlikely reports of psychotic symptoms; (3) psychological test findings of feigning or gross exaggeration of symptoms, or of cognitive performance below expectancy given history and presentation. For example, a defendant who presented himself as too distractible to participate in interviews, yet was observed at other times reading a newspaper with evidently good concentration, would be suspected of malingering. Likewise, performance on cognitive tests suggestive of severe cognitive impairment would point toward malingering in an individual who has functioned adequately and independently in the community. The malingering diagnosis did not specifically target offense memory.

The presence of substance use proximal to the offense was determined on the basis of examinee self-report buttressed by reports by collateral informants of a corresponding pattern of substance use in the examinee. Typical informants were family members or close friends. Defendants whose self-reports could not be corroborated by a collateral report were omitted. We aimed to establish substance abuse at least within a period of hours before the offense. For example, ingestion the day before, but not the day of, the offense was not counted as proximal substance use.

Offense amnesia was coded in those defendants who claimed to have no memory of the act(s) constituting the charged offense, even though there might have been memory for events before or after the offense.

Results

Table 1 gives the characteristics of the final sample. Of the total sample, 21 examinees (40%) claimed offense amnesia. In three of these, there was evidence of partial or full recovery of memory during the 1-month evaluation period.

As predicted, there was a significant relationship between substance use at the time of the crime and claims of offense amnesia. In 14 of the 21 examinees who claimed amnesia (67%) there was

TABLE 1—*Sample characteristics.*

Age		
Median	30	
Range	19–51	
	n	%
Sex		
Male	47	89
Female	6	11
Ethnicity		
Caucasian	37	70
African American	16	30
Offense		
Property Offense	10	19
Sex Offense	6	11
Assault	17	32
Murder/Attempted Murder	20	38
Diagnosis*		
Schizophrenia	15	28
Any Psychotic Disorder	23	43
Organic Brain Syndrome	8	15
Mood Disorder	20	38
Substance Use Disorder	26	49
Borderline/Mental Retardation	22	42
Malingering	8	15
Substances Used at Time of Offense†		
Alcohol	17	32
Benzodiazepines	6	11
Cannabis	9	17
Cocaine	5	9
Opiates	1	2
Methamphetamine	2	4
Hallucinogens	1	9
Any	25	47
None	28	53

*Due to multiple diagnoses, percentages sum to greater than 100. †Some defendants used more than one substance.

evidence of use of some substance at the time of the offense, whereas proximal substance use was established in only 11 of the 32 who did not claim amnesia ($\chi^2 = 5.31$, df = 1, $p = 0.021$). Of the remaining seven of 21 amnesia cases who did not have proximal substance use, three were diagnosed with malingering. Thus, 81% of those claiming amnesia either had proximal substance use or were deemed to be malingering. Consistent with these findings, a discharge diagnosis of substance use disorder was significantly associated with claims of amnesia ($\chi^2 = 4.32$, df = 1, $p = 0.038$). Of the 26 defendants with a substance use disorder, 14 (54%) claimed amnesia for the offense.

Proximal alcohol use bore the strongest relationship to amnesia. There was evidence of alcohol use in 48% of those who claimed amnesia, but in only 22% of those who did not ($\chi^2 = 3.86$, df = 1, $p = 0.049$). Of the 17 offenders who had consumed alcohol at the time of the offense, 10 (59%) claimed inability to remember their offense. In addition, the presence of alcohol use overlapped considerably with use of cannabis, cocaine, and benzodiazepines in offenders who claimed amnesia. Proximal cannabis use was present in nine offenders, and six of these claimed amnesia. However, five of these six were also using alcohol at the time of the offense. Four out of five cocaine users claimed amnesia, but two of these four were also using alcohol. Of the six benzodiazepine users, four claimed amnesia, and three of these four had consumed alcohol.

Examinees diagnosed with schizophrenia were less likely to claim amnesia than those without this diagnosis ($\chi^2 = 6.04$, df = 1, $p = 0.014$). Of 15 defendants so diagnosed, only two claimed offense amnesia. Moreover, the correlation between schizophrenia and proximal substance use was near zero. Thus, the inverse relationship between schizophrenia and amnesia was not mediated by an absence of substance use in this diagnostic group; in fact, proximal substance use was present in 40% of the examinees with schizophrenia. About a third of them were drinking at the time of the offense, and only one of these claimed amnesia. There was a similar trend toward inverse association between offense amnesia and psychotic diagnoses in general. However, this trend did not achieve statistical significance and was likely due to the high correlation between psychotic diagnosis and schizophrenia. Of 23 defendants with psychotic diagnoses, 15 (65%) were diagnosed with schizophrenia.

With respect to age, the group of examinees who claimed amnesia was evenly distributed across a range of 19–44 years. In terms of discharge diagnosis, this group of defendants was dominated by mood disorder, subnormal intellectual functioning, and substance use disorders: 53% (11/21) of examinees claiming amnesia had a mood disorder diagnosis, 67% (14/21) had a substance use disorder diagnosis, and 53% (11/21) had a diagnosis of borderline intellectual functioning or mental retardation. Similarly, substance use at the time of the crime was prominent in conjunction with these clinical diagnoses; 86% (18 of 21) of amnesic defendants had a mood disorder diagnosis or proximal substance use, or both (seven of 21 had both), whereas only three out of 21 (14%) of the amnesic patients had neither problem; 76% of amnesic patients had subnormal intellectual functioning or proximal substance abuse, or both (nine of them had both), whereas only five of 21 (24%) had neither problem. There were 12 cases that lacked all three of these features, and only one of these cases claimed amnesia. By comparison, there were six examinees in whom all three of these features were present, and five of the six claimed amnesia.

The relationship between malingering and amnesia was nonsignificant—$\chi^2 = 2.06$, df = 1, $p = 0.15$. To further examine the possible effect of malingering on other significant relationships, we removed the eight subjects with a malingering diagnosis (leaving $n = 45$) and re-analyzed the data. The relationship between amnesia and proximal substance use remained evident ($\chi^2 = 8.01$, df = 1, $p = 0.005$), as did the negative relationship between amnesia and schizophrenia ($\chi^2 = 4.01$, df = 1, $p = 0.045$).

Discussion

Reconciling this study with past research focuses the problem of defining "amnesia" for empirical study. We chose to operationalize amnesia as a claim by the offender of no memory at all for the criminal act itself, regardless of memory for events immediately before or after the crime. However, other investigators have recognized memory impairment in different degrees, differentiable by the presence of memory for events before and after the criminal act (considered by some a partial, but not full, amnesia) and whether memory for the act itself is described as completely absent versus "partial" or "hazy" (1,2,34). Thus, it is possible that we omitted cases of incomplete amnesia that other researchers might have included as positive cases, while including cases that others would regard as "partial" amnesia cases.

The prevalence of the malingering diagnosis in our sample was in line with suspected base rates in populations of criminal examinees, that is, about 10–20% (7). However, malingering was not reliably associated with offense amnesia. Hence, malingering as diagnosed by general clinical criteria may not be particularly useful in identifying specific claims of offense amnesia. Formal tests of malingering specific to the memory sphere may improve identification of malingering with respect to offense recall (9,35,36).

Like other researchers, we found that ingestion of alcohol and other drugs was prevalent among examinees who claimed offense amnesia. Out of all the variables we examined, the association between substance use and amnesia was the strongest, and alcohol was the most commonly used substance. Succinctly stated, about two out of three of our subjects who claimed amnesia reported substance use proximal to the offense, and about two out of three subjects who did not claim amnesia did not report proximal substance use.

The validity of claims of alcohol-induced amnesia—i.e., alcohol blackout—or of offense amnesia due to other substances besides alcohol, has been a point of controversy in the literature on offense amnesia. Moskowitz and others (8,37) adopt the view that alcohol blackout does not account for offense amnesia because a person in a blackout state would be too intoxicated to engage in complex behavior; however, Goodwin (16) and White et al. (16,21,38) argue otherwise. Examples of amnesia for complex behavior from studies of drugs besides alcohol include the Daderman et al. (39,40) documented cases of complex violent acts under the influence of Rohypnol, with no evident later memory for the behavior.

Consideration of factors contributing to offense amnesia is further complicated by suggestions that dissociative processes come into play. Some authorities have assigned a role to dissociative mechanisms in impairment of offense memory and have suggested that such mechanisms may result from high emotional arousal or from trauma experienced by the offender in the context of the offense (1,6,8). With relevance to this study, it has been suggested that substance use may foster a state of dissociation that leads to amnesia (8,18). Cooper et al. (19) presented data showing higher reports of state dissociation at the time of the offense among offenders who claimed amnesia. There was, however, a high frequency of intoxication among amnesic subjects, and higher reports of state dissociation among those who had been intoxicated. Our own position is that memory blackouts due to ingestion of alcohol and certain drugs are a real phenomenon with a reasonably well

understood physiological substrate (15,21), and possibly a genetic susceptibility (41). Complex behavior does occur during blackouts. Amnesia due to blackouts should be considered a form of organic amnesia (9) and to adduce the concept of dissociation to explain memory effects associated with alcohol or drug use is unparsimonious and may lead to needless confusion.

Evans (1) has criticized a reliance on self-report as a "measure" of intoxication at the time of the offense. We attempted to improve on self-report by means of corroborating informants. Beyond this, it is unclear whether indices like blood alcohol content (BAC) would be of much additional benefit. BAC is imperfectly related to blackouts (20,22). Moreover, there is evidence that blackouts are more likely with concurrent use of alcohol and other substances. Benzodiazepines in particular are suspect, but other substances, e.g., marijuana, may have combinatory or synergistic effects with alcohol as well (21). Furthermore, observer reports would mean little, because in many respects, a person in a blackout can appear normal. Because a blackout state may not be evident to the external observer (17,42,43), first-response criminal investigators may collect few observations about a defendant's state of intoxication even when the offender is arrested soon after the offense. At the same time, the detainee may lack insight regarding degree of cognitive impairment when in a state of intoxication, although he or she may later recognize a memory gap (17,44).

Controversies about the validity of blackout claims intersect with forensic questions. Although claims of inability to remember the offense are rarely beneficial, offenders may nevertheless issue such claims for strategic purposes, perhaps because they wrongly believe that doing so will help them in court (4,5,7,45). Merckelbach and Christianson (7) describe several self-serving motives for claiming amnesia aside from circumscribed issues of insanity or diminished capacity. Additionally, at least one authority has observed that it is difficult to assess intent relative to acts that are submerged in amnesia and has argued that a blackout is a state of "absence of mind" that the law should recognize as exculpatory (much like an "automatism") in instances when the blackout was the unanticipated result of social drinking (46). To gain perspective on amnesia claims, it is important to note that past research has not focused exclusively on pretrial detainees as we did. For example, Pyszora et al. (2) studied postconviction inmates who were serving life sentences. Nearly a third of their sample initially reported they were amnesic, and over 40% of those continued to claim complete lack of offense memory 3 years beyond conviction.

This study suffers from the same weaknesses as any retrospective characterization of a clinical sample; in particular, any evident association in the data is subject to cross-validation in other samples. Nevertheless, we were struck by the finding that amnesia claims were so uncommon among examinees with schizophrenia, or with psychotic disorders in general. We suspect that these data reflect two prominent reasons for referral for forensic psychiatric evaluation, whereby defendants are referred for evaluation either because they appear to have psychotic features or because they claim amnesia for the offense (perhaps in the presence of other identifiable, but nonpsychotic, mental health problems). It has been shown that defendants with a psychotic disorder are much more likely to be referred for pretrial evaluation of competence (47). At the same time, there is parallel evidence that a claim of offense amnesia is also likely to trigger referrals for mental examination (2,7).

This study found no evidence that psychotic disorder in general, or schizophrenia in particular, confers a higher likelihood of offense amnesia. It appears that among pretrial examinees there may be only limited overlap between those patients with psychotic features and those who deny memory for their offense. We maintain that substance use around the time of an offense, and in some cases clumsy attempts at malingering, probably account for the great majority of amnesia claims (though not all), and that both these features should be considered in the course of the examination before alternative explanations are sought.

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References

1. Evans C. What violent offenders remember of their crime: empirical explorations. Aust NZ J Psychiatry 2006;40:508–18.
2. Pyszora NM, Barker AF, Kopelman MD. Amnesia for criminal offences: a study of life sentence prisoners. J Forensic Psychiatry Psychol 2003;14:475–90.
3. Schacter DL. Amnesia and crime: how much do we really know? Am Psychol 1986;41:286–95.
4. van Oorsouw K, Merckelbach H, Ravelli D, Nijman H, Mekking-Pompen I. Alcoholic blackout for criminally relevant behavior. J Am Acad Psychiatry Law 2004;32:364–70.
5. Taylor PJ, Kopelman MD. Amnesia for criminal offenses. Psychol Med 1984;14:581–8.
6. Swihart G, Yuille J, Porter S. The role of state-dependent memory in "red-outs." Int J Law Psychiatry 1999;22:199–212.
7. Merckelbach H, Christianson SA. Amnesia for homicide as a form of malingering. In: Christianson SA, editor. Offenders' memories of violent crimes. Chichester, West Sussex, UK: John Wiley and Sons, 2007;165–90.
8. Moskowitz A. Dissociation and violence: a review of the literature. Trauma Violence Abuse 2004;5:21–46.
9. Cima M, Merckelbach H, Nijman H, Knauer E, Hollnack S. I can't remember your honor: offenders who claim amnesia. German J Psychiatry 2002;5:24–34.
10. Gudjonsson GH, Hannesdottir K, Petursson H. The relationship between amnesia and crime: the role of personality. Pers Individ Dif 1999;26:505–10.
11. Vivian SE, Gudjonsson GH. Denial and memory performance in two cases of homicide. Med Sci Law 1986;26:72–6.
12. Porter S, Birt A, Yuille J, Herve H. Memory for murder: a psychological perspective on dissociative amnesia in legal contexts. Int J Law Psychiatry 2001;24:23–42.
13. Cima M, Merckelbach H, Hollnack S, Knauer E. Characteristics of psychiatric prison inmates who claim amnesia. Pers Individ Dif 2003;35:373–80.
14. Boles SM, Miotto K. Substance abuse and violence: a review of the literature. Agg Violent Behav 2003;8:155–74.
15. Ghoneim MM. Drugs and human memory (part 2): clinical, theoretical, and methodological issues. Anesthesiology 2004;100:1277–97.
16. Goodwin DW. Alcohol amnesia. Addiction 1995;90:315–7.
17. Dowd SM, Strong MJ, Janicak PG, Negrusz A. The behavioral and cognitive effects of two benzodiazepines associated with drug-facilitated sexual assault. J Forensic Sci 2002;47:1–7.
18. Parwatikar SD, Holcomb WR, Menninger KA. The detection of malingered amnesia in accused murderers. Bull Am Acad Psychiatry Law 1985;13:97–103.
19. Cooper BS, Cuttler C, Dell P, Yuille JC. Dissociation and amnesia: a study with male offenders. Int J Forensic Psychol 2006;13:69–83. Available from: http://ijfp.psyc.uow.edu.au/IJFPArticlesIssue3/Cooper.pdf. Accessed August 13, 2007.
20. Granacher RP. Commentary: alcoholic blackout and allegation of amnesia during criminal acts. J Am Acad Psychiatry Law 2004;32:371–4.
21. White AM. What happened? Alcohol, memory blackouts, and the brain. Alcohol Res Health 2003;27:186–96.
22. Hartzler B, Fromme K. Fragmentary and en bloc blackouts: similarity and distinction among episodes of alcohol-induced memory loss. J Stud Alcohol 2003;64:547–50.

23. Grant BF, Stinson FS, Dawson DA, Chou P, Dufour MC, Compton W, et al. Prevalence and co-occurrence of substance use disorders and independent mood and anxiety disorders. Arch Gen Psychiatry 2004;61:807–16.
24. RachBeisel J, Scott J, Dixon L. Co-occurring severe mental illness and substance use disorders: a review of recent research. Psychiatr Serv 1999;50:1427–34.
25. Negrete JC. Clinical aspects of substance abuse in persons with schizophrenia. Can J Psychiatry 2003;48:14–21.
26. Hall W, Degenhardt L, Teesson M. Cannabis use and psychotic disorders: an update. Drug Alcohol Rev 2004;23:433–43.
27. Baethge C, Baldessarini RJ, Khalsa HMK, Hennen J, Salvatore P, Tohen M. Substance abuse in first-episode bipolar I disorder: indications for early intervention. Am J Psychiatry 2005;162:1008–10.
28. Crawford V, Crome IB, Clancy C. Co-existing problems of mental health and substance misuse (dual diagnosis): a literature review. Drugs Educ Prev Policy 2003;10(Suppl.):S1–74.
29. Bowie CR, Serper MR, Riggio S, Harvey PD. Neurocognition, symptomatology, and functional skills in older alcohol-abusing schizophrenia patients. Schizophr Bull 2005;31:175–82.
30. Serper MR, Bergman A, Copersino ML, Chou JCY, Richarme D, Cancro R. Learning and memory impairment in cocaine-dependent and comorbid schizophrenic patients. Psychiatry Res 2000;93:21–32.
31. Pencer A, Addington J. Substance use and cognition in early psychosis. J Psychiatry Neurosci 2003;28:48–54.
32. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th ed. Washington, DC: American Psychiatric Association, 1994.
33. Resnick PJ, Knoll J. Faking it: how to detect malingered psychosis. Curr Psychiatry 2005;4(11):13–25.
34. Stanton J, Simpson AIF. The aftermath: aspects of recovery described by perpetrators of maternal filicide committed in the context of severe mental illness. Behav Sci Law 2006;24:103–12.
35. Delain SL, Stafford KP, Ben-Porath YS. Use of the TOMM in a criminal court forensic assessment setting. Assessment 2003;10:370–81.
36. Jelicic M, Merckelbach H, van Bergen S. Symptom validity testing of feigned amnesia for a mock crime. Arch Clin Neuropsychol 2004;19:525–31.
37. Marshall WL, Serran G, Marshall LE, Fernandez YM. Recovering memories of the offense in ''amnesic'' sexual offenders. Sex Abuse 2005;171:31–8.
38. White AM, Jamieson-Drake DW, Swartzwelder HS. Prevalence and correlates of alcohol-induced blackouts among college students: results of an e-mail survey. J Am Coll Health 2002;513:117–31.
39. Daderman AM, Lidberg L. Flunitrazepam (Rohypnol) abuse in combination with alcohol causes premeditated, grievous violence in male juvenile offenders. J Am Acad Psychiatry Law 1999;27:83–99.
40. Daderman AM, Fredriksson B, Kristiansson M, Nilsson LH, Lidberg L. Violent behavior, impulsive decision-making, and anterograde amnesia while intoxicated with flunitrazepam and alcohol or other drugs: a case study in forensic psychiatric patients. J Am Acad Psychiatry Law 2002;30:238–51.
41. Nelson EC, Heath AC, Bucholz KK, Madden PAF, Fu Q, Knopik V, et al. Genetic epidemiology of alcohol-induced blackouts. Arch Gen Psychiatry 2004;61:257–63.
42. Verwey B, Eling P, Wientjes H, Zitman FG. Memory impairment in those who attempted suicide by benzodiazepine overdose. J Clin Psychiatry 2000;61:456–9.
43. Verwey B, Muntendam A, Ensing K, Essink G, Pasker-de Jong PCM, Willekens FLA, et al. Clinically relevant anterograde amnesia and its relationship with blood levels of benzodiazepines in suicide attempters who took an overdose. Prog Neuropsychopharmacol Biol Psychiatry 2005;29:47–53.
44. Merritt P, Hirshman E, Hsu J, Berrigan M. Metamemory without the memory: are people aware of midazolam-induced amnesia? Psychopharmacology 2005;177:336–43.
45. Brandt J. Malingered amnesia. In: Rogers R, editor. Clinical assessment of malingering and deception. New York: Guilford Press, 1988;65–83.
46. Merikangas J. Commentary: alcoholic blackout—does it remove *mens rea*? J Am Acad Psychiatry Law 2004;32:375–7.
47. Viljoen JL, Zapf PA. Fitness to stand trial evaluations: a comparison of referred and non-referred defendants. Int J Forensic Ment Health 2002;12:127–38.

Additional information-Reprints not available from author:
Ronnie G. Stout, Ph.D.
Middle Tennessee Mental Health Institute
221 Stewarts Ferry Pike
Nashville, TN 37214
E-mail: Ron.Stout@state.tn.us