Cerebrospinal Fluid Amines and Higher-Lethality Suicide Attempts in Depressed Inpatients

J. John Mann and Kevin M. Malone

Previous studies have found that not all suicide attempters with major depression have reduced serotonergic activity based on low cerebrospinal fluid 5-hydroxyindoleacetic acid (CSF 5-HIAA) levels. In this study we hypothesized that serotonergic function is lower in depressed patients who have carried out high-lethality suicide attempts resulting in more medical damage, which might explain differences in serotonergic activity among depressed suicide attempters. We assessed the relationship of CSF 5-HIAA and other amine metabolites to the most lethal lifetime suicide attempt in 22 drug-free inpatients with major depression. CSF 5-HIAA levels were lower in depressed patients with a history of a high-lethality or well-planned suicide attempt compared to depressed patients with a history of only low-lethality suicide attempt(s). Other CSF monoamine metabolites did not correlate with suicidal behavior. Low serotonergic activity may correlate with a predisposition to more lethal suicide attempts in major depression. © 1997 Society of Biological Psychiatry

Key Words: Suicide attempt, cerebrospinal fluid, 5-hydroxyindoleacetic acid, homovanillic acid, 3-methoxy-4-hydroxyphenylglycol, depression

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Introduction

Reduced serotonergic function has been associated with completed suicide and attempted suicide. Brain stem levels of serotonin (5-HT) and 5-hydroxyindoleacetic acid (5-HIAA) are modestly reduced in five of seven studies of suicide victims (Shaw et al 1967; Pare et al 1969; Lloyd et al 1974; Korpi et al 1986; Cochrant et al 1976; Bourne et al 1968; Beskow et al 1976), and this reduction appears to be independent of the associated psychiatric diagnosis. Reductions in serotonin transporter binding and increases in serotonin 5-HT$_{2A}$ and 5-HT$_{1A}$ receptor binding have been reported by most studies in the prefrontal cortex of suicide victims (Mann et al 1996), consistent with reduced presynaptic function and perhaps compensatory postsynaptic receptor up-regulation.

Suicide attempt behavior is more complex than suicide completion, and perhaps that is the reason why the results are not as clear-cut in suicide attempters. Ten of 15 published studies (Nordin 1988; Secunda et al 1986; Roy et al 1986; Roy-Byrne et al 1983; Vestergaard et al 1978; Banki and Arató 1983; Jones et al 1990; Westenberg and Verhoeven 1988; Edman et al 1986; Ågren and Niklasson 1986; Lopez-Ibor et al 1985; van Praag 1982; Träskman et
al 1981; Palaniappan et al 1983; Åsberg et al 1976) found lower cerebrospinal fluid (CSF) levels of 5-HIAA in depressed patients who had made a suicide attempt, compared to depressed patients who were nonattempters. This finding may not extend to all types of depression, because studies of suicide attempters with bipolar disorder have not consistently reported lower CSF 5-HIAA, (see Åsberg 1989 for review). Moreover, even in studies reporting lower CSF 5-HIAA in suicide attempters, not all suicide attempters have low CSF 5-HIAA. For example, one study (Åsberg and Träskman 1981) found that low CSF 5-HIAA characterized 53% of suicide attempters compared to 23% of nonattempters. These results raise the question as to whether there is a subtype of suicide attempt that is correlated with low serotonergic activity.

Stengel and others (Stengel 1973) have described two categories of suicide attempts. The first category has been termed failed suicide and involves a highly lethal attempt with evidence of planning and a low chance of rescue. The second category encompasses an impulsive, unplanned attempt involving little medical damage, and a high chance of rescue. It has been called pseudocide, parasuicide or suicide gesture (Lennard-Jones and Asher 1959; Stengel 1973). Despite this wide range of types of suicidal acts, the relationship of the type of suicidal act to serotonergic function has hardly been studied. Given that low CSF 5-HIAA may predict future suicide (Nordström et al 1994; Åsberg et al 1976; Träskman et al 1981), it may also be related to the potential for highly lethal suicide attempts. The relationship of CSF 5-HIAA to the most lethal lifetime suicide attempt has not been studied. Moreover, extrapolating from the association of low CSF 5-HIAA with impulsive aggression against person or property (Virkkunen et al 1987; Brown et al 1979, 1982; Linnoila et al 1983; Lidberg et al 1985), it has been hypothesized that impulsive suicide attempts, but not nonimpulsive suicide attempts, will correlate with low serotonergic function; however, only one study has quantified planning of suicide attempts and evaluated its relationship to serotonergic function. We previously reported in another set of patients that depressed patients with a recent highly planned suicide attempt had low CSF 5-HIAA relative to nonattempters and patients who had a history of a recent poorly planned suicide attempt (Mann et al 1992).

We now report results of a study of the relationship between serotonergic activity and the most lethal lifetime suicide attempt as determined in inpatients being evaluated for major depression. The goal of this study was to determine how dimensions of severity of suicidal acts such as degree of medical damage, suicidal planning, and subjective lethal intent are associated with serotonergic activity, especially in relation to the most lethal lifetime suicide attempt for each case. We also extended our previously published study by assaying cerebrospinal fluid levels of monoamine metabolites, which were compared between depressed patients with a lifetime high medical damage suicide attempt, and depressed patients with a history of only low medical damage attempts. Thus, both groups had major depression and differed only in terms of the degree of medical damage caused by their most lethal lifetime suicide attempt. Since degree of medical damage ultimately distinguishes completed suicide from attempted suicide, we hypothesized that the group more closely resembling "failed suicides" (a higher level of medical damage) could be distinguished from other suicide attempters by lower levels of CSF 5-HIAA.

Methods

Recruitment

Patients were included in the protocol after admission to a private university psychiatric hospital for evaluation and treatment of depression and having given written informed consent for this study as required by the Institutional Review Board for Biomedical Research. The group included 22 inpatients meeting criteria for a current major depressive episode who had made a lifetime suicide attempt. The duration of the drug-free status of patients was established by a combination of a drug screen, observation in hospital, and a history obtained from the patient's family and the referring physician. Patients had to be free for at least 14 days from drugs known to affect the serotonin system, including all monoamine oxidase inhibitors, tricyclics, selective serotonin reuptake inhibitors, serotonin agonists, and new nonserotonergic antidepressants. The drug-free interval was longer for drugs with a long half-life such as fluoxetine (6 weeks) and oral antipsychotics (4 weeks). When the drug-free period was recorded, it was arbitrarily limited to 365 days. The mean drug-free period was 100 days (SD = 122), largely because many of these patients had been untreated prior to admission. Diagnosis was established according to DSM-III-R criteria at a consensus conference based on results of the Structured Clinical Interview for DSM-III-R (SCID) (Spitzer et al 1989) and independent, supplemental interviews by two research psychiatrists (JJM, KMM). All patients had a current major depressive episode. In addition, 3 patients had psychotic features and 1 had a bipolar disorder. Patients had a physical examination and routine laboratory screening tests to detect neurological disease and any active physical disease that could affect their mental status or CSF 5-HIAA. Exclusion criteria included cardiac failure, renal or liver disease, malignancy, obstruc-
tive airway disease, endocrinopathies, and neurological disorders.

Assessment of Clinical Symptoms

The severity of depression was assessed by the Hamilton Depression Rating Scale (HDRS) (Hamilton 1960). Suicidal behavior was measured by the Suicide Intent Scale (SIS) (Beck et al 1975) and medical damage by the Medical Damage Rating Scale (Beck et al 1975). The data reported on suicide attempts refer to the most medically damaging, lifetime suicide attempt. This suicide attempt was further classified as nonviolent (drug overdose) or violent (gunshot, cutting, stabbing, jumping, or hanging). Suicide methods also refer to the method of the most lethal lifetime attempt and included overdose ($n = 16$), cutting ($n = 2$), hanging ($n = 2$), jumping ($n = 1$), and shooting ($n = 1$). The Medical Damage Rating Scale scores degree of medical damage from 0 to 8 ($0 = $none, $8 = $dead; $0 - 7$ applicable in this study). Anchor points are different and specific for each type of method. All suicide attempt data were gathered on a Suicide History Form (copyright MHCRC, unpublished), which uses clinical probes and anchor points to identify suicide attempts chronologically and requires careful documentation of the method and medical damage of each suicide attempt such that reliable lethality scores are generated for all suicide attempts. Data were analyzed both using the score as a continuous variable and by dividing patients into high and low lethality attempters on the basis of a median split of lethality scores. The Suicide History Form also incorporates the Beck Scale for Suicidal Ideation (Beck et al 1979) and the Beck Suicide Intent Scale (Beck et al 1975). Components of suicide intent were rated for the most lethal attempt based upon the two-factor solution resulting from our group’s factor analysis (Mieczkowski et al 1993) of the Beck Suicide Intent Scale (Beck et al 1975). Factor I, called Lethal Intent, contains items pertaining to subjective intent. Factor II, called Planning, contains items pertaining to the objective degree of planning for the attempt. Two subscale scores were generated by summing the scores of items included within each factor.

Lifetime history of aggressive behavior was measured using the Aggression History Questionnaire developed by Brown and Goodwin (Brown et al 1979, 1982). A categorical diagnosis of a borderline personality disorder (BPD) was based on DSM-III-R criteria (Spitzer et al 1989) and established by the International Personality Disorder Examination (IPDE) structured clinical interview (Loranger et al 1987, 1991). Ratings of current psychopathology assessed the patients’ clinical status over the 2 weeks prior to the sampling of CSF. Trait-related measures such as the Aggression History Questionnaire and IPDE were carried out after stabilization of the patients’ acute status. These latter ratings were almost always done after the CSF sampling and close to the timing of discharge.

The Lumbar Puncture

The lumbar puncture was performed at about 08:30 hours after the patient had been kept in bed at rest and fasted from midnight. We did not employ a low-monoamine diet in this study or in our previously reported study (Mann et al 1992). CSF was withdrawn from the L4–L5 interspace with the patient in the left decubitus position. After the removal of 1 mL of CSF into the first sample tube, a further 15 mL of CSF was collected in a second, single tube and immediately transferred on ice water to be centrifuged at 4°C. This 15-mL sample was then divided into 1-mL aliquots for storage at −70°C until assay. CSF monoamine metabolites were assayed in a 1-mL aliquot of the 15-mL sample.

CSF 5-HIAA, homovanillic acid (HVA), and 3-methoxy-4-hydroxyphenylglycol (MHPG) were assayed by high-performance liquid chromatography (HPLC) with electrochemical detection (Scheinin et al 1983). The within-run and between-run coefficients of variation of the assay method were less than 10%. The level of sensitivity of the assay for 5-HIAA was 0.5 pmol/injection. All assays were done by laboratory staff blind to the clinical data.

Cross-classification analyses were performed to test for significant differences in categorical demographic and clinical characteristics. Statistical comparisons between the high- and low-lethality attempter groups were made using Student’s $t$ test and analysis of variance (ANOVA). The Newman–Keuls post hoc multiple comparison procedure was used to identify which groups were significantly different when a significant group effect was found by ANOVA. Stepwise logistic regression analysis was used to examine the relationship of lethality and planning with CSF 5-HIAA. Results are reported as mean ± standard deviation, and all probabilities are two tailed. To distinguish high-lethality and low-lethality suicide attempters a priori, we did further comparisons dividing suicide attempters into those characterized by high (score ≥ 3) and low (score < 3) medical damage based on a median split of the Medical Damage Rating Scale scores. Subsequent examination of the results of this median split revealed a clinically meaningful cutoff score, because a score of ≥ 3 on the Medical Damage Rating Scale corresponds to a degree of medical damage requiring medical hospitaliza-
Table 1. Demographic and Clinical Features of High versus Low Lethality Suicide Attempters

<table>
<thead>
<tr>
<th>Variable</th>
<th>Low lethality (n = 8)</th>
<th>High lethality (n = 14)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>28.9 ± 6.9</td>
<td>34.9 ± 9.9</td>
<td>-1.49</td>
<td>0.15</td>
</tr>
<tr>
<td>Hamilton depression scale (17 item)</td>
<td>19.7 ± 5.1</td>
<td>20.0 ± 6.9</td>
<td>-0.10</td>
<td>0.92</td>
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<td>Beck hopelessness score</td>
<td>13.7 ± 6.1</td>
<td>11.7 ± 6.1</td>
<td>0.68</td>
<td>0.51</td>
</tr>
<tr>
<td>Barratt impulsivity score</td>
<td>61.7 ± 8.6</td>
<td>54.7 ± 9.1</td>
<td>1.29</td>
<td>0.22</td>
</tr>
<tr>
<td>Brown aggression history</td>
<td>23.4 ± 5.4</td>
<td>21.8 ± 8.3</td>
<td>0.45</td>
<td>0.66</td>
</tr>
<tr>
<td>Buss-Durkee hostility score</td>
<td>37.6 ± 9.9</td>
<td>41.4 ± 10.4</td>
<td>-0.73</td>
<td>0.48</td>
</tr>
<tr>
<td>No. of suicide attempts</td>
<td>4.0 ± 3.8</td>
<td>3.6 ± 2.6</td>
<td>0.26</td>
<td>0.79</td>
</tr>
</tbody>
</table>

**Degree of Medical Damage Resulting from a Suicide Attempt and CSF 5-HIAA**

Suicide attempters who had made a high medical damage lifetime suicide attempt (≥ 3 on Medical Damage Rating Scale) had significantly lower mean CSF 5-HIAA compared to low medical damage attempters (83.7 ± 24.2 vs. 109.7 ± 28.2 pmol/mL; t = 2.29, df = 20, p = .03) (see Figure 1). There was also a trend for CSF 5-HIAA to correlate negatively with the maximum degree of medical damage inflicted by a lifetime suicide attempt (r = −.38, n = 22, p = .07). The mean interval between the most lethal suicide attempt and sampling of CSF was 785 ± 1627 days (range 4–7128). A two-way analysis of variance, including recency of suicide attempt, confirmed that high medical damage suicidal behavior remained significantly associated with lower mean CSF 5-HIAA (F = 4.3, df = 1, p = .05), whereas the effect of recency of suicide attempt was not significant (F = 1.15, df = 1, p = .29). Age was not significantly different between high and low lethality suicide attempters (34.9 ± 10.9 vs. 28.9 ± 7, t = -1.49, p = .15).

**Suicidal Planning and CSF 5-HIAA**

High suicidal planning score, as defined by a median split (≥ 9) of the score on Factor II of the Suicide Intent Scale, was also associated with lower CSF 5-HIAA. Sixty-six percent of high-planning attempters had low (< 81 pmol/mL) CSF 5-HIAA, whereas only 20% of low-planning attempters had low CSF 5-HIAA (χ² = 4.23, p < .04). Moreover, there was a trend for suicidal planning to correlate negatively with CSF 5-HIAA (r = −.41, n = 19; p = .08). Subjective suicidal intent (Factor I) did not correlate with CSF 5-HIAA (or HVA or MHPG).

**Clinical Characteristics and CSF 5-HIAA**

No differences in CSF 5-HIAA were found between violent and nonviolent suicide attempters (94.5 ± 25.3 vs. 111.4 ± 27.9 pmol/mL, t = -1.17, df = 17, p = .26) or between psychotic and nonpsychotic attempters (91.3 ± 31.9 vs. 90.4 ± 19.1 pmol/mL, t = 0.07, df = 20, p = .94). No correlations were found between CSF 5-HIAA and severity of depression (Hamilton Depression Rating Scale; r = -0.22, n = 22, p = .19).

**Other CSF Amine Metabolites**

In contrast to the findings in the serotonergic system, no associations were found between CSF HVA or MHPG levels and high (≥ 3) versus low (< 3) medical damage suicide attempts (CSF HVA: 170 ± 63.8 vs. 198.8 ± 56.3 pmol/mL).
Figure 1. Distribution of CSF 5-HIAA in high versus low lifetime lethality suicide attempters ($t = 2.29$, $df = 20$, $p = .03$). Median values for the two groups are numerated.

$$\text{pmol/mL, } t = 0.99, \ df = 18, \ p = .34; \ 
\text{CSF MHPG: } 46.4 \pm 8.1 \ vs. \ 43.3 \pm 10.7 \ \text{pmol/mL, } t = -0.79, \ df = 20, \ p = .44).$$

Similarly, high versus low suicidal planning was unrelated to CSF HVA or MHPG (CSF HVA: $176.2 \pm 66.6 \ vs. \ 178.6 \pm 56.9 \ \text{pmol/mL, } df = 18, \ p = .94; \ 
\text{CSF MHPG: } 46.8 \pm 9.8 \ vs. \ 46.0 \pm 8.4 \ \text{pmol/mL, } df = 18, \ p = .84).$$

No differences in CSF HVA or MHPG levels were found in psychotic versus nonpsychotic depressed patients (HVA: $223.8 \pm 86.4 \ vs. \ 180.83 \pm 59.2 \ \text{pmol/mL, } df = 20, \ p = .18; \ 
\text{MHPG: } 46.4 \pm 6.6 \ vs. \ 43.9 \pm 10.5 \ \text{pmol/mL, } df = 20, \ p = .56).$$

Moreover, no differences were found in CSF HVA or MHPG between violent and nonviolent attempters. No linear correlations were statistically significant between HVA and MHPG with degree of medical damage or planning.

**Modeling Serotonin Biology and Suicidal Behavior**

Using a stepwise logistic regression analysis with CSF 5-HIAA as the independent variable, and lethality as the dependent variable, the overall sensitivity of the model was $82\%$ ($B = -0.04, \ SE = 0.02, \ Wald = 3.83, \ p = .05$) (see Figure 2). There were insufficient data on planning (available for 19/22 cases) to also include this variable in the model.

**Discussion**

We found that reduced levels of CSF 5-HIAA were associated with two measures of severity of suicidal behavior: primarily, a greater degree of medical damage resulting from the most lethal lifetime suicide attempt and secondarily, a higher degree of planning associated with the same suicide attempt.

Despite the apparently small sample size in this study, it is the fifth largest population of suicide attempters with major depression out of 16 studies with CSF amine levels assayed (see Introduction for references), and the first to use DSM-III-R criteria to diagnose major depression utilizing the SCID (Spitzer et al 1992). Several interesting findings emerge from our study, despite these size limitations.
**Biological vs. Clinical Measures**

There is a significant difference in CSF 5-HIAA between high- and low-lethality suicide attempters in the absence of measurable clinical differences between the groups, suggesting that CSF 5-HIAA was a superior discriminator of the depressed patient group with a history of serious suicidal behavior compared with clinical measures. Åberg and colleagues made a similar observation in their comparison of suicide attempters and nonattempters with major depression. They found CSF 5-HIAA was lower in suicide attempters, but severity of depression did not differ from nonattempters (Åberg and Träskman 1981). We found evidence for a relationship between degree of medical damage in the most lethal lifetime suicide attempt and CSF 5-HIAA. A logistic regression model demonstrated that lower CSF 5-HIAA levels are predictive of a history of more medically damaging suicide attempts with an overall sensitivity of 82%. Almost all previous studies have not quantified suicide lethality and therefore there are few studies available for comparison.

In an overlapping group of patients, we have reported elsewhere (Malone et al in press) that the prolactin response to fenfluramine, another index of serotonergic function, is blunted in the group with a high-lethality lifetime suicide attempt. Thus, results using two different probes of the serotonergic system, CSF 5-HIAA in this study, and the prolactin response to fenfluramine, are concordant. In an earlier study, involving another patient group (Mann et al 1992), we found that low CSF 5-HIAA was associated with a greater degree of planning of the most recent suicide attempt, but degree of medical damage of the most recent lifetime attempt did not appear related in that study. A subsequent analysis, when that original study group was doubled in size, did demonstrate a relationship between low CSF 5-HIAA and greater medical damage (Mann et al in press). That study also confirmed the relationship of planning to low CSF 5-HIAA. The lack of statistical significance of suicidal planning in the current study may be due to the fact that we had planning data on only 19/22 subjects, too few to incorporate the planning variable in the logistic regression model. The contribution of suicidal planning to this model needs to be more fully assessed in a larger patient population. No other published studies have quantified
planning so as to evaluate its relationship to biological indices.

Based on these results, at least one reason why only a proportion of suicide attempters appear to have low CSF 5-HIAA, is because of the seriousness of the suicide attempt. Individuals with low CSF 5-HIAA appear more likely to make serious suicide attempts; however, as our data (Mann et al 1992) and Asberg and Träskman (1981) have shown, the distribution of CSF 5-HIAA levels overlaps in attempters and nonattempters, and therefore CSF 5-HIAA is a relative risk factor among several.

Specificity to Serotonin System

The absence of correlations of the other amine metabolites (HVA and MHPG) to dimensions of suicide attempt behavior suggests that these findings are relatively specific to the serotonin system. This observation strengthens the probability that the results are correct, and are reinforced by results of postmortem studies of the brain of suicide victims.

Compatibility with Postmortem Suicide Studies

Several studies (see Arango and Mann 1992 for a review) have reported lower levels of serotonin and/or 5-HIAA in the brain stem of suicide victims compared to controls (Shaw et al 1967; Pare et al 1969; Moses and Robins 1975; Lloyd et al 1974; Korpi et al 1986; Cochrane et al 1976; Bourne et al 1968; Beskow et al 1976). Levels of 5-HIAA in frontal cortex correlate with CSF levels at postmortem (Stanley et al 1985), indicating that CSF 5-HIAA levels may reflect neuronal activity in relevant brain areas. Changes involving multiple serotonin receptors (transporter, 5-HT$_2$ and 5-HT$_{1A}$) have also been found in the brain of suicide victims (Arango et al 1990; Stanley and Mann 1983; Arango and Mann 1992; Mann et al 1989; Paul et al 1984; Stanley et al 1982) (see Arango and Mann 1992 for a review).

Thus, we propose that a relationship exists between suicide completers and at least a subgroup of suicide attempters based on both the behavioral dimensions of degree of medical damage and suicide planning, as well as the biological dimension of altered serotonergic activity. A category of failed suicide was previously described (Beck et al 1975; Stengel 1973) on the basis of clinical similarities in the pattern of the suicidal behavior, namely use of a highly lethal method, minimization of the chances of rescue, and in general, planning the suicide attempt. We now propose that this category of suicide attempt may be further described in biological terms, namely reduced serotonin function.

Correlation between Serotonergic Activity and Past Suicidal Behavior

There are at least two observations that may explain how severity of suicidal acts can correlate with CSF 5-HIAA sampled an average of about 3 years after the most lethal suicide attempt. The first is that CSF 5-HIAA levels are relatively stable over time. This has been demonstrated on test–retest in human (Hildebrand et al 1990) and nonhuman primate studies (Higley et al 1993), and may be explained by the significant degree of genetic control over CSF 5-HIAA (Higley et al 1993). The second observation is that degrees of suicide intent (Beck and Steer 1989) and medical damage (Malone and Mann unpublished data) predict intent and medical damage in subsequent attempts, and are therefore correlated from one suicide attempt to the next. If CSF 5-HIAA can be an indicator of the degree of medical damage incurred from past suicide attempts, it may predict the degree of damage from future suicide attempts.

We previously studied the relationship between CSF 5-HIAA levels and suicide lethality in another group of depressed suicide attempters (Mann et al 1992), where we gathered data on the lethality of most recent suicide attempts only, and found a weak association with CSF 5-HIAA and a significant association with platelet serotonin measures. In a subsequent study on an expanded sample we also found that high medical lethality of recent attempt was related to lower CSF 5-HIAA (Mann et al in press). The present study goes one step further in demonstrating that the most lethal lifetime suicide attempt may correlate with low serotonin function.

Clinical Implications of Serotonergic Activity and Suicidal Behavior

From the opposite perspective, we found that higher levels of CSF 5-HIAA are associated with a history of suicide attempts resulting in less medical damage. This observation is consistent with a potential protective effect of higher levels of CSF 5-HIAA. If a higher level of serotonergic activity is associated with less medically damaging suicide attempts, then it may be hypothesized that treatments that increase serotonergic activity may not only reduce the probability of a future suicide or suicide attempt (Mann and Arango 1992; Träskman et al 1981), but may also reduce the degree of medical damage in the event of an attempt. This suggestion is further supported by the observation that patients with lower levels of CSF 5-HIAA have an increased probability of a future suicide (Nordström et al 1994; Åsberg and Träskman 1981; Träskman et al 1981; Åsberg et al 1976) or suicide attempt (Cooper et al 1992; Roy et al 1989).

We have also found that the degree of medical damage
increases in subsequent suicide attempts (Malone and Mann unpublished data). Prospective studies demonstrating a relationship between lower CSF 5-HIAA levels and the degree of medical damage resulting from future suicide attempts would support the hypothesis that selective enhancement of serotonergic function by pharmacotherapy would reduce the probability of future suicide attempts, or at least the degree of consequent medical injury. Controlled studies evaluating specific serotonin reuptake inhibitors in high-risk suicidal patients are lacking. Published studies have not demonstrated any specific effect on suicidality, but have largely looked at suicidal ideation or low-lethality suicidal behavior (Mann et al 1993; Mann and Kapur 1991). There is a need for controlled pharmacotherapy confirmation and maintenance studies in high-risk populations to adequately test this hypothesis.

Conclusion

Our results indicate that a history of high medical damage suicide attempts in patients with major depression may be associated with low CSF 5-HIAA and therefore reduced serotonergic function.

References


