2) perphenazine, 8 mg b.i.d., for 6 months; 3) two courses of bilateral ECT (five treatments initially, followed by six treatments 7 months later); 4) trazodone, 50 mg t.i.d., for 6 weeks; 5) desipramine, 25 mg t.i.d., for 8 weeks; 6) clomipramine, 25 mg t.i.d., for 6 weeks (both the desipramine and the clomipramine were at therapeutic serum levels despite the low doses); and 7) combined carbacholazepine, 200 mg t.i.d., and lithium carbonate, 450 mg b.i.d., for 4 weeks (both with therapeutic serum levels). These treatments had little or no effect.

Finally, Mr. A received lorazepam, 2 mg i.m. Initially there was no response, but 4 hours after the injection he suddenly started talking. He was alert and oriented and exhibited normal psychomotor activity. He denied symptoms of depression and smiled freely during the interview. He had somatic complaints but denied abnormalities of thought content or perception. He could not explain his former mutism and psychomotor retardation. He exhibited thought blocking and loosening of associations and had no insight regarding his illness.

This effect lasted approximately 12 hours. His medications were changed to oral doses of pimozide, 12 mg h.s.; benztropine, 2 mg b.i.d.; and diazepam, 10 mg h.s. (Diazepam was chosen for its longer half-life.) Mr. A's psychomotor activity increased but never became completely normal. He no longer exhibited waxy flexibility. He remained mute with the staff but started to speak to his mother and his wife. Soon after, he was able to be discharged to the care of his family.

The mechanism of action of parenteral lorazepam in relieving catatonia is unclear (2, 3). However, this case shows that it may have an important role in relieving longstanding schizophrenic catatonia that has proven resistant to other forms of therapy. It also demonstrates the previously reported benefit of adding diazepam to the medication regimen (4).

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Ms. A, a 62-year-old Caucasian housewife, was first seen at home at the request of her family doctor. Over the preceding few weeks she had complained of having AIDS and was feeling depressed and guilty about this. She boiled her eating utensils, washed her clothes separately, and had taken to her bed to avoid contact with other members of her family. She had lost an indeterminate amount of body weight as a result of her reduced appetite.

There was no antecedent history of psychiatric or major physical illnesses. Ms. A's sister and two sons suffered from schizophrenic illness, and her daughter had recently been admitted to a hospital because of a brief psychotic illness. There was no evidence of personality problems or drug or alcohol abuse.

On admission to the hospital, Ms. A was found to be depressed, with psychomotor retardation and delusions of having AIDS and being infectious. She had no other delusions and was cognitively unimpaired.

Soon after admission, she made a low-visibility suicide attempt by cutting both her wrists and needed surgical intervention. She later stated that she did this to prevent the spread of the disease. She refused food and medication but was treated with eight ECTs after the appropriate procedures under the 1983 Mental Health Act had been followed. She made a remarkable recovery and remained well at 6-month follow-ups while on a regimen of antidepressant medication.

It is well recognized that the content of delusions is subject to pathoplastic sociocultural influences. This case is an example of this. Since AIDS has been widely discussed in the media and is said to have high fatality, it is understandable that collective anxiety in the general population is high. However, it is important, especially for mental health professionals, to be aware that the presenting complaint of "fear of AIDS" may be just one aspect of much more serious underlying psychopathology. In this patient's case, it was not very difficult to suspect and treat the psychotic depression, but it might be more easily overlooked in younger, high-risk patients.

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An Attempt to Commit Suicide by Contracting AIDS

SIR: I read with great interest the letter by Richard J. Frances, M.D., and associates (1) on contracting acquired immune deficiency syndrome (AIDS) as a means of committing suicide and the article by Daniel K. Flavin, M.D., and associates (2) in which they reported on four alcoholic homosexual men who deliberately sought to be infected by AIDS with the purpose of committing suicide. I would like to report briefly on a similar suicide attempt by a woman.

Depressive Illness With Delusions of AIDS

SIR: With the increase in the incidence of acquired immune deficiency syndrome (AIDS) in recent years, there has been a spate of articles in medical journals about the physical and psychiatric aspects of AIDS. Miller et al. (1) discussed a case of depression and a case of anxiety in homosexual men. Many other articles (e.g., 2, 3) have discussed psychiatric morbidity in high-risk groups of patients. I would like to report the following unusual case.
Sex Differences in Panic Disorder

Sir: The preponderance of women among persons with panic disorder is a consistent epidemiological finding (1). The explanations for this are mostly speculative and range from socioeconomic to psychological. The significance of this difference, however, could be etiologically relevant to the disorder.

Having conducted hundreds of lactate infusions in patients with panic disorder, we have reported changes in a great number of parameters before, during, and after the infusions (2) but have found few significant sex differences. Recently identified ventilatory and acid-base changes during lactate infusions (3), however, have brought into focus a relatively neglected area of research: the respiratory physiology of panic. In order to evaluate the exact acid-base status of panic patients, we started collecting arterial blood samples during lactate infusions. The procedure, described in detail elsewhere (2), consists of a 30-minute saline infusion (baseline) followed by a 20-minute infusion of 0.5 M sodium lactate.

Analysis of the baseline arterial blood gas samples revealed that male panic disorder patients who subsequently panicked when given lactate (N=5) were in a state of mild, chronic, compensated, respiratory alkalosis (normal pH, low arterial PCO2, and low HCO3 level) and developed superimposed, acute respiratory alkalosis (significant drop in arterial PCO2). In fact, an arterial PCO2 of 40 torr or higher at the time of the switch to lactate virtually precluded lactate panic in male patients. Male panic patients who did not panic when given lactate (N=8) maintained normal pH, arterial PCO2, and HCO3 values and did not hyperventilate during the baseline period.

By contrast, there were no significant acid-base changes for female patients during the baseline period and no significant baseline acid-base differences between female panic patients who panicked when they received lactate (N=10) and those who did not (N=11). Female patients as a group maintained blood gas values consistent with mild, chronic, compensated respiratory alkalosis. At baseline, female patients who panicked after lactate were indistinguishable from female patients who did not panic. Acute hyperventilation thus seems necessary for lactate panic to occur in men but not in women. A tendency to hyperventilate at baseline may be a characteristic feature of lactate-sensitive male, but not female, panic patients.

We do not have data about the time of the procedure in the female patients' menstrual cycles. This factor could significantly alter these findings. A larger sample size is clearly needed to substantiate these preliminary results. However, if they are replicated, differences in respiratory physiology could indicate a distinct characteristic of lactate-sensitive male panic patients that differentiates them from female patients with panic disorder.

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