ceived ECT, similar comparisons were not made for the

young adult group.

Fifty-one geriatric patients (10 ECT patients and 41 medication patients) were reliably followed for 24 months. Of the four patients who received ECT and antidepressant medication, three relapsed within 24 months. Of the six patients who received ECT alone, two relapsed. Of the 41 patients who received medication alone, 15 (36.6%) relapsed within 24 months. Similar comparisons for the young adult patients were not made because of their chaotic follow-up patterns and scant numbers.

Results from this small retrospective study suggest the following conclusions. 1) Giving antidepressant medication to elderly patients receiving ECT does not increase the risk of delirium or prolong hospitalization. 2) Hospital stays were longer for geriatric and young adult patients who received ECT than for patients receiving medication alone. We believe this represents a severity of illness factor (ECT patients were sicker) and does not suggest that ECT caused prolongation of hospitalization. 3) Readmission rates (our measure of relapse) for all geriatric patients was high, regardless of treatment, and approximated the 53.8% 1-year relapse rate found by Georgotas et al. for geriatric patients receiving nortriptyline as maintenance medication (4). Future investigations should examine more refined physiologic measures of ECT, such as total seizure time, and correlate physiologic measures with outcomes such as length of hospital stay, relapse rate, and risk of delirium.

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Use of Maintenance ECT for Elderly Depressed Patients

SIR: Commenting on the review by Robert F. Prien, Ph.D., and Alan J. Gelenberg, M.D. (1) of preventive treatment of bipolar disorder, Charles Kellner, M.D., and associates (2) raised the issue of maintenance ECT. In our department seven patients have now been treated with maintenance ECT for nearly 3 years (mean±SD=34.0±1.9 months, range=28-44 months).

Among these elderly patients (mean±SD age=75.0±3.5 years, range=56-86 years) responding poorly to mood stabilizers—in particular, lithium and carbamazepine—one suffers from bipolar disorder according to the DSM-III-R criteria, five from recurrent major depression, and one from schizoaffective disorder; their first episodes occurred be-

tween 7 and 52 years ago (mean±SD=31.2±5.9). Before inclusion in the maintenance program, a positive response had been obtained with a curative series of six to 10 ECTs (mean±SD=8.0±0.4).

The mean±SD total number of maintenance ECTs in the group is presently 25.8±2.4 (range=19-36), and four patients are continuing in the program. Maintenance treatment was delivered every 3-5 weeks for the first 4 months and every 4-8 weeks from the 12th month onward. The mean±SD scores on the Hamilton Rating Scale for Depression (31.4±1.7 [absence of symptoms=1]) and the Brief Psychiatric Rating Scale (29.5±2.2) remained low and stable during the maintenance period. Whereas patients had spent a mean ±SD of 58.2 ± 11.0 weeks in the hospital before maintenance ECT, the average time in the hospital, apart from the periodic 36-hour stay to receive the maintenance ECT, dropped to 3.7 ± 1.1 weeks during the maintenance period. This last figure should be compared to the 27.4±43.0 weeks spent in the hospital during the previous 3 years (Wilcoxon test adjusted to a 36-month period after ECT, p<0.01). Similarly, 4.7±1.6 recurrences were observed in the 3 years preceding maintenance ECT and only 1.4±0.6 under maintenance treatment (Wilcoxon test, p<0.03), with two (28.6%) of these patients, who often do not respond to treatment, showing no evidence of further mood disorders. Maintenance ECT appears to be a valuable alternative in the treatment of recurrent mood disorders.

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The Clonidine Test in Posttraumatic Stress Disorder

SIR: Several findings in animals and human beings support the idea of noradrenergic abnormalities in posttraumatic stress disorder (PTSD) (1). The catecholamine-mediated behavioral sequelae of inescapable shock in animals closely parallel the negative symptoms of PTSD in human beings. Fewer platelet α_2 binding sites and sustained elevation of urinary norepinephrine levels have been found in PTSD patients. Moreover, antidepressants acting on the noradrenergic system improve PTSD symptoms.

The growth hormone (GH) response to clonidine has already been used as an indirect index of noradrenergic function in various pathological conditions, particularly depressive disorders, with very promising results (2). To our knowledge, this method, however, had never been used in PTSD, and we would like to describe here the results of this neuroendocrine assessment in a PTSD patient before and

after successful treatment.

Mr. A, a 20-year-old patient, was hospitalized with a typical case of PTSD, according to the DSM-III-R criteria, following a car accident 2 months earlier. He had not received any psychotropic drug for his current symptoms. The traumatic event was reexperienced in recurrent dreams, and the patient persistently avoided stimuli associated with the trauma. Furthermore, persistent symptoms of increased arousal—irritability, difficulty concentrating, and hypervigilance—were present.

Mr. A's clinical condition was assessed with the Impact of Event Scale (3) during his first day of hospitalization, and he received a total score of 45 (intrusive subscale, 20; avoidance subscale, 25). No significant associated depressive features were noted. The clonidine challenge, performed 3 days after admission according to a previously described method (2), showed a blunted GH response (baseline GH level=0.1 ng/ml; peak GH level=4.7 ng/ml).

Implosive therapy was initiated after Mr. A had been taught and had practiced progressive muscle relaxation and guided imagery. Then, the original traumatic scene was presented until satisfactory control of anxiety was achieved. The treatment yielded good improvement of symptoms, with a decrease in the total Impact of Event Scale score to 17 (intrusive subscale, 10; avoidance subscale, 7) after 2 weeks and to 4 (intrusive subscale, 2; avoidance subscale, 2) after 3 weeks. A second clonidine challenge test performed the following week elicited a normal GH response (baseline GH level=0.1 ng/ml; peak GH level=11.1 ng/ml).

Since GH response to clonidine seems to reflect the sensitivity of postsynaptic α_2 -adrenergic receptors, the initially blunted response noted in our patient supports the idea of decreased noradrenergic sensitivity, which could reflect the down-regulation of noradrenergic receptors. This result is consistent with the decrease in platelet α_2 binding sites in PTSD patients described by Perry et al. (4). In addition, the normalization of the clonidine test results after successful therapy suggests that noradrenergic disturbances in PTSD are limited to the pathological period and therefore might represent a state marker rather than a trait marker of PTSD, in contrast to what they indicate in endogenous depression (5). This hypothesis should obviously be tested in a larger group of PTSD patients.

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Delusions of Pregnancy in a Man

SIR: There have been reports of delusions of pregnancy in men in association with organic cerebral pathology (1) or schizoaffective disorder (2, 3). We should like to report a case of delusions of pregnancy in a male patient without any evidence of such pathology.

Mr. A, a 51-year-old man with a 20-year history of chronic delusional disorder, persecutory type, was admitted to our hospital after having inserted a knife in his anus in order to facilitate delivery of a baby. This patient had been treated for years with depot neuroleptic drugs, but he had interrupted the treatment 3 months before he was admitted. Nine years earlier, he had left the country of his birth, where he was working as a reporter, for political reasons. When he arrived in France, he was married and had a son. His wife did not want to have a second child. A week before his hospitalization, he went to a gynecology clinic; he was excited and expressed the firm belief that he was pregnant. Confronted by physicians' denial of his pregnancy, he exhibited autoaggressive tendencies.

On admission Mr. A complained of abnormal sensations that he misinterpreted as pregnancy: his abdomen was swollen and he felt some abdominal spasms. He thought a baby was moving in his abdomen. He had gained 10 lb, which to him constituted proof of his assumptions. When we pointed out to him that men could not become pregnant, he denied it, explaining that he was "intersexual" (a man and woman at the same time), making a pregnancy possible.

His delusive belief had started after surgical treatment of an anal fistula 4 months previously. He was convinced that a uterus had been grafted into his abdomen during the operation, which took 4 hours. To make certain that he was recovering from the fistula, he had been given a rectoscopic examination. He explained to us that during this investigation, the surgeon had done an anal artificial insemination. Furthermore, he claimed that the depot neuroleptic drugs he took had been used to prepare his body for this pregnancy; he thought they were estrogens. The clue was that during depot neuroleptic treatment, his chest had increased in size and galactorrhea had appeared. In the past he had experienced some homosexual relationships and suffered from difficulties with women, which he related to the abnormal shape of his penis (his genitals were completely normal).

After 4 months of treatment with chlorpromazine, 400 mg/day, the delusion of pregnancy completely resolved. Mr. A continued to receive psychiatric care, and some persecutory delusions still remained.

The number of recorded instances of delusions of pregnancy in men is very small. Organic brain damage plays a strong part in the development of delusions of pregnancy in these patients. However, our patient was free of organic cerebral pathology. He was of normal intelligence, and results of physical and neurological examinations were normal, as were his blood biochemistry and an EEG. There was no history of drug or alcohol abuse and no family history of psychiatric illness or perinatal complications. The delusion of pregnancy constituted a short event in his chronic persecutory type of delusional disorder. This delusion appeared as a secondary phenomenon to misinterpretation of primary somatic sensations.