TABLE 1. Age and Serum Cholesterol Levels of 192 Depressed and Normal Subjects

Group	N	Age (years) Mean	Age (years) SE	Cholesterol (mg/100 ml) Mean	Cholesterol (mg/100 ml) SE
Aged 60–85 years					
Depressed men	24	67.0	1.2	255.9	10.4
Depressed women	24	67.8	.8	275.1	11.1
Normal men	24	69.4	.9	260.0	11.4
Normal women	24	67.2	.8	287.1	15.1
Aged 18–59 years					
Depressed men	24	39.9	2.4	201.2	6.3
Depressed women	24	39.8	2.6	218.8	9.0
Normal men	24	42.3	3.6	215.2	10.7
Normal women	24	40.2	3.8	222.1	12.3

DISCUSSION

The data from this study confirm the report of Adlersberg and associates (5) that age and sex account for a significant proportion of the variance in serum cholesterol level. Our data do not support the findings of Lang and Haits (1) that serum cholesterol level is higher among either young or old patients with major depression than among age- and sex-matched normal controls. Thus, an abnormally high serum cholesterol level is not selective for major depression and therefore is not a useful biological marker for major depression.

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Agranulocytosis Associated With Amoxapine

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Agranulocytosis developed in a woman who had taken amoxapine for 10 weeks. The author is unaware of any previous reports of this complication of amoxapine therapy.

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Amoxapine is a relatively new tricyclic antidepressant with a chemical structure which is chemically distinct from that of other tricyclic antidepressants (1). Agranulocytosis is a relatively rare, but potentially lethal, side effect of tricyclic antidepressants (2). In this report I present a case of agranulocytosis that developed in a woman who had been treated with amoxapine for 10 weeks. I am unaware of any previous reports of agranulocytosis associated with amoxapine.

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CASE REPORT

Ms. A, a 42-year-old woman, was treated with amoxapine, 50 mg/day, for severe panic disorder complicated by depressive symptoms. On day 32 of amoxapine therapy, she was hospitalized on the psychiatric unit of a community hospital because of intensification of her symptoms of panic. She remained there for 2 weeks, and her dose of amoxapine was increased to 150 mg/day. She was taking no other medications. The results of her physical examination were normal. Her WBC count was 4,200/mm^3 with 57% neutrophils, 27% lymphocytes, 6% monocytes, 5% eosinophils, and 5% band cells. Her psychiatric condition stabilized and she was discharged from the hospital. On day 65 of amoxapine therapy, Ms. A contacted her physician because of malaise, weakness, fever, and chills that had persisted for 3 days. Her WBC count was 200/mm^3 with 0% neutrophils, 92% lymphocytes, and 8% monocytes. Amoxapine therapy was discontinued. A stormy 47-day hospitalization ensued, with a lengthy stay in the intensive care unit. Her agranulocytosis was complicated by staphylococcal septicemia, possible endocarditis, pleural effusion, empyema, arterial embolism, exfoliative dermatitis, and acute respiratory distress syndrome. Bone marrow aspiration showed hypocellular

bone marrow with marked myeloid suppression. Treatment included leukocyte transfusions, ventilatory support, parenteral antibiotics, and anticoagulation therapy. On day 82, her WBC count was 11,300/mm^3 with 15% bands and 71% neutrophils. Ms. A was discharged on day 114. Her condition has been stable since then, with normal complete blood counts.

DISCUSSION

This case demonstrates an association between amoxapine and agranulocytosis. The risk of potentially fatal toxic bone marrow suppression should be considered in the prescription of most psychotropic medications. The period of maximum risk of drug-induced agranulocytosis is reported to be at about weeks 3–4, but the risk continues to at least weeks 8–12 (3). In this case, agranulocytosis occurred after 10 weeks of amoxapine therapy. Our enthusiasm for using the newer antidepressants that are being marketed in this country as safe products (4) should be tempered by an awareness of the risks associated with them.

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ECT-Induced Delirium and Further ECT: A Case Report

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*The authors report the occurrence of a severe confusional state (*DSM-III *delirium) in a patient who received bilateral sinusoidal ECT. Unilateral brief-pulse ECT was then used to successfully treat the patient's depression without the redevelopment of delirium.*

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ECT, when administered 2–3 times a week, seldom produces a severe organic brain syndrome (1, 2). It has been suggested (2) that the incidence of this complication can be reduced by the use of unilateral electrode placements, threshold rather than supra-threshold levels of electrical energy, and oxygenation during ECT. The present study reports the occurrence of delirium in a patient who received only four bilateral sinusoidal treatments. Delirium did not redevelop after subsequent administration of unilateral brief-pulse ECT.

CASE REPORT

Mr. A was a 57-year-old man with a bright-normal to superior Shipley IQ. He had an 18-year history of intermittent depression, which had begun soon after the accidental drowning of a son. Four months before Mr. A's admission to our hospital, his father had died, after which he became psychotically depressed, with vegetative signs and symptoms; mood-congruent delusions; suicidal, homicidal, and persecutory ideation; and auditory hallucinations. Before transfer to our hospital, Mr. A was hospitalized and treated with maprotiline (125 mg/day), desipramine (75 mg/day), and trifluoperazine (5 mg/day) without therapeutic response.

A physical examination of Mr. A after transfer was normal except for left-sided ptosis, increased muscle tone with mild cogwheeling, mild gait disturbance, and a positive snout reflex. An EEG was slightly abnormal, with some intermittent irregular slowing in the left midtemporal and anterior temporal regions. A heat stroke 9 months before the current

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TABLE 1. Neuropsychological and Behavioral Test Results of a Patient Given Bilateral Sinusoidal and Unilateral Brief-Pulse ECT

Test	Bilateral Sinusoidal ECT (treatments 1–4)		Unilateral Brief-Pulse ECT (treatments 6–10)	
	2 Days Post-ECT	6 Days Post-ECT	2 Days Post-ECT	6 Days Post-ECT
Orientation (<10=impaired)	3	7	10	10
Right-left orientation (<4=impaired)	0	2	4	4
Visuographic dyspraxia (<3=impaired)	0	3	4	5
Perseveratory behavior (<45=impaired)[a]	7	40	45	45
Dysnomia (<4=impaired)	0	4	4	4
Verbal fluency (<23=impaired)	7	14	18	26
Attention/concentration (<30=impaired)[a,b]	23	29	42	52
Visuographic double tracking (<24=impaired)[a,c]	2	3	10	15
Immediate memory (6–7=unimpaired)[a,d]	6	7	6	6
Short-term memory (<15=impaired)	2	8	22	23
Digits backward (<4=impaired)[a]	0	3	3	4
Zung depression scale (<50=normal)[e]	—	56	—	51
Hamilton depression scale (<8=normal)[f]	—	17	—	6

[a]Alternate form used.
[b]1-minute trial.
[c]Trails B test; choices given in 124 seconds.
[d]Digits forward test.
[e]SDS index.
[f]17 items rated by one examiner.

hospitalization was thought to be secondary to neuroleptic treatment. Two months later, Mr. A developed a persistent hypesthesia of the left hand. Neurologic evaluation for possible cerebrovascular disease (including a CAT scan) was negative. Pre-ECT spine X-rays revealed a widespread variety of degenerative changes in the spinal column, some severe. He also had had spinal decompression surgery.

After Mr. A gave informed consent for ECT, he was randomly assigned to receive bilateral sinusoidal ECT as part of an ongoing research investigation. Medication for agitation (oral thioridazine, 50 mg as needed) was discontinued 3 days before ECT. ECT was administered on Mondays, Wednesdays, and Fridays and was modified by atropine, .6–1.2 mg subcutaneously 30 minutes before treatment; methohexital, 60 mg i.v.; succinylcholine, 60–90 mg i.v.; curare, 3–4.5 mg i.v.; and 100% oxygen. The small dose of curare was needed to aid in the achievement of total relaxation because of the patient's vertebral disease. Electrical stimulation consisted of a bidirectional sinusoidal stimulus (140 V, 60 Hz, .5–.6-second duration, mean stimulus energy of 56 J, and mean seizure duration of 73 seconds). Seizure duration was measured electroencephalographically.

By the fourth treatment, Mr. A had become profoundly confused and dysnomic. Neuropsychological testing 2 days after the fourth ECT (see table 1) resulted in a low level of performance on a variety of tests (3). Neurological examination at this time revealed no other change from the baseline evaluation. CAT scan with enhancement revealed no abnormalities, but an EEG performed 8 days after the fourth ECT showed a mild to moderate diffuse slowing of background rhythms, with an accentuation in the left temporal region of greater degree than that seen before ECT. We concluded that ECT had induced an unusually severe and prolonged acute confusional state (*DSM-III* delirium). Repeat neuropsychological testing 6 days after the fourth ECT corroborated this conclusion, in that most cognitive functions were improving (4).

Because Mr. A had shown a partial therapeutic response to ECT, we decided to continue with a less "toxic" form of ECT, namely, unilateral nondominant brief-pulse ECT. However, he was left-handed, showed other signs of mixed cerebral dominance according to d'Elia's criteria (5), and had a family history of sinistrality. Consequently, cerebral dominance was determined by a sequence of brief-pulse bilateral, right, and then left unilateral treatments (one of each) as described by Clyma (6). This trial was initiated 12 days after the last sine wave bilateral treatment. Mr. A's verbal responses were delayed longer after left than after right unilateral ECT. It was therefore concluded that his left cerebral hemisphere subserved language functions. He was then given three more right unilateral brief-pulse treatments. All brief-pulse treatments used a bidirectional electrical stimulus (800-mA peak amplitude, 60 pulse-pairs/second, .75-msec pulse width, 1.00–1.25-second duration, mean stimulus energy of 16 J, and mean seizure duration of 79 seconds). A good therapeutic response was attained.

Mr. A was retested 2 and 6 days after his 10th (last) treatment (see table 1). The test results indicate that cognition was still improving despite the interpolated unilateral pulse treatments. This improvement is significant because there usually is some cumulative buildup of confusion during a treatment course, even with unilateral nondominant ECT (7). A repeat EEG performed 8 days after the last ECT revealed very mild generalized slowing, at times with left temporal predominance, but both findings were present to a lesser degree than after the smaller number of bilateral sine wave treatments.

DISCUSSION

The etiology of this ECT-induced delirium is unclear. The presence of focal neurologic signs and preexisting focal EEG abnormalities suggests the possibility of a focal CNS lesion that was not evident radiographically, although these findings were inconsistent with each other on the basis of laterality.

The present case indicates that delirium after standard bilateral sinusoidal ECT does not necessitate the discontinuation of ECT (2), since delirium did not redevelop after subsequent administration of unilateral nondominant brief-pulse ECT. This result is consistent

with the lesser degree of memory and EEG abnormalities known (8) to occur with the use of unilateral nondominant brief-pulse ECT in patients without CNS abnormalities.

Less neuropsychological impairment in Mr. A after unilateral brief-pulse ECT than after bilateral sinusoidal ECT may have been related to any of the following factors: 1) less electrical stimulation of the dominant cerebral hemisphere (and the possible site of a preexisting CNS lesion) with unilateral ECT, 2) less electrical energy with the use of brief-pulse ECT, 3) less seizure generalization with unilateral brief-pulse ECT, or 4) natural resolution of the delirium unaffected by subsequent ECT administration (2, 4).

In addition to improved neuropsychological functioning, Mr. A's affect continued to improve. Some investigators (9) report that unilateral brief-pulse ECT is not always as effective as bilateral sinusoidal ECT in treating depression, whereas other investigators (10) report similar antidepressive efficacy. In any event, the decrease in neuropsychological and depressive pathology in the present case argues for using unilateral nondominant brief-pulse ECT in situations where a severe organic brain syndrome has occurred with standard ECT techniques or when the patient is felt to be at increased risk for this to occur.

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Adult Onset of Tourette's Syndrome: A Case Report

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A 45-year-old man fulfilled all DSM-III *criteria for the diagnosis of Gilles de la Tourette's syndrome except for early onset of the disease. He first developed tics at age 35 and coprolalia at age 40.*
(Am J Psychiatry 140:924–925, 1983)

Gilles de la Tourette's syndrome is an illness that usually develops between age 2 and age 15 (1 and *DSM-III*). Isolated reports of later onset have involved incorrect diagnoses or have not been regarded as typical cases of the syndrome (2). A male patient is described here who met all the diagnostic criteria of *DSM-III* and Shapiro and associates (1) for the syndrome except that he developed symptoms after the age of 35. The other symptoms for Tourette's syndrome are the following: presence of recurrent, involuntary repetitive, rapid, purposeless motor movements affecting multiple muscle groups; multiple vocal tics; ability to suppress movements voluntarily for minutes to hours; variations in the intensity of the symptoms over weeks or months; and duration of symptoms for more than 1 year.

CASE REPORT

Mr. A, age 45 years, has been treated at our institution for 3 years. He first complained of "strange sensations" in his stomach muscles and jaw at age 35. One year later he observed a "restlessness" in his stomach muscles, arms, legs, and jaw and commented, "It was as if my muscles developed a life of their own." At age 40 he felt the urge to utter the word "shit" and subsequently uttered the word louder and

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