treatments very well; she had no further significant cardiac problems. It has been suggested that genetic factors may be associated with the determination of these patterns and their clinical correlates.

Ms. A was a 68-year-old woman who was suffering from her sixth episode of severe major depressive disorder. Her antipsychotic drug treatment and psychotherapy but partially remitted with ECT. Each episode lasted approximately 2 years and was followed by complete remission and return to normal functioning. Because the depressive episode she was in at the time of this report had not responded to an outpatient trial of pharmacotherapy, Ms. A was hospitalized to receive more intensive treatment. Her medical history included a myocardial infarction with a brief hospitalization. She was 63 years old and multinodal diffuse coronary artery disease was discovered at that time and had been treated since then with low doses of insulin. The results of admission laboratory tests were normal except for a fasting blood sugar level of 317 mg/dl and an ECG that showed the signs of the anteroseptal myocardial infarction and nonspecific ST-T wave abnormalities. The results of a physical examination were unremarkable except for a cataract in one eye. A previous history included diabetes mellitus, which had been diagnosed 15 years before the onset of the cardiac arrest. Her blood pressure was 180/120 mm Hg (diastolic = 100-110/180). After unsuccessful attempts to control her anxiety with reassurance and support, we decided to proceed with the treatment with Ms. A's consent. Before ECT, she was given 1 mg i.v. of propranolol, administrated and the standard administration of atropine was omitted. Intravenous pentothal, 100 mg (1.8 mg/kg), and succinylcholine, 30 mg (0.5 mg/kg), were used to induce general anesthesia and muscle relaxation, respectively. From a mask with positive pressure. The treatment was continuously monitored with a bipolar electroencephalogram (EEG) and an ECG. A bilateral electrical stimulation was achieved by a shock-induced activation of the autonomic nervous system and the use of propranolol may have contributed to the cardiac arrest by exacerbating the parasympathetic response associated with the concomitant stimulation.

Cardiac arrest is a well-documented, although rare, complication of ECT and is not necessarily related to B blockers. Indeed, it may be argued in this case that the likelihood of a cardiac arrest was heightened by the lack of preventive use of atropine, the subcutaneous administration of propranolol may have contributed to the cardiac arrest by exacerbating the parasympathetic response associated with the concomitant stimulation. In particular, Fitts and associates (8) demonstrated anecdotal evidence supporting the efficacy of propranolol in the treatment of ECT-related tachyarrhythmia and exaggerated sympathetic outflow suggesting caution when combining B blockers and ECT.

A hypertensive patient with a history of diabetes and ischemic heart disease was given propranolol (8.4 mg) before an experimental cardiac arrest after an uncontrolled episode of tachycardia and hypertension. The incidence and severity of these complications appear to increase in the presence of certain variables relating to the patient's condition such as preexisting cardiovascular illness and old age, and certain variables relating to treatment conditions, such as oxygenation and premedication (4).

Anecdotal evidence supports the efficacy of propranolol and other B blockers in the treatment of ECT-related tachycardia and exaggerated sympathetic outflow (1-3) and, at least in theory, in the prevention of myocardial ischemia that may occur in patients with ischemic heart disease (3). To our knowledge, the oral or intravenous administration of B blockers as a pre-ECT medication has not been reported to be associated with adverse side effects, but in the case reported here the use of intravenous propranolol immediately before ECT in a patient with preexisting cardiac pain and a history of ischemic heart disease was associated with a reversible cardiac arrest.

**Cardiac Arrest During ECT Modified by B-Adrenergic Blockade**

Paolo Decina, M.D., Sidney Maliz, M.D., Harold A. Sackei, Ph.D., John Helzer, M.D., and Stuart Yudofsky, M.D.

A hypertensive patient with a history of diabetes and ischemic heart disease was given propranolol (8.4 mg) before an experimental cardiac arrest after an uncontrolled episode of tachycardia and hypertension. The incidence and severity of these complications appear to increase in the presence of certain variables relating to the patient's condition such as preexisting cardiovascular illness and old age, and certain variables relating to treatment conditions, such as oxygenation and premedication (4).

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**Case Report**

Ms. A was a 68-year-old woman who was suffering from her sixth episode of severe major depressive disorder. Her antipsychotic drug treatment and psychotherapy but partially remitted with ECT. Each episode lasted approximately 2 years and was followed by complete remission and return to normal functioning. Because the depressive episode she was in at the time of this report had not responded to an outpatient trial of pharmacotherapy, Ms. A was hospitalized to receive more intensive treatment. Her medical history included a myocardial infarction with a brief hospitalization. She was 63 years old and multinodal diffuse coronary artery disease was discovered at that time and had been treated since then with low doses of insulin. The results of admission laboratory tests were normal except for a fasting blood sugar level of 317 mg/dl and an ECG that showed the signs of the anteroseptal myocardial infarction and nonspecific ST-T wave abnormalities. The results of a physical examination were unremarkable except for a cataract in one eye. A previous history included diabetes mellitus, which had been diagnosed 15 years before the onset of the cardiac arrest. Her blood pressure was 180/120 mm Hg (diastolic = 100-110/180). After unsuccessful attempts to control her anxiety with reassurance and support, we decided to proceed with the treatment with Ms. A's consent. Before ECT, she was given 1 mg i.v. of propranolol, administrated and the standard administration of atropine was omitted. Intravenous pentothal, 100 mg (1.8 mg/kg), and succinylcholine, 30 mg (0.5 mg/kg), were used to induce general anesthesia and muscle relaxation, respectively. From a mask with positive pressure. The treatment was continuously monitored with a bipolar electroencephalogram (EEG) and an ECG. A bilateral electrical stimulation was achieved by a shock-induced activation of the autonomic nervous system and the use of propranolol may have contributed to the cardiac arrest by exacerbating the parasympathetic response associated with the concomitant stimulation. In particular, Fitts and associates (8) demonstrated anecdotal evidence supporting the efficacy of propranolol in the treatment of ECT-related tachyarrhythmia and exaggerated sympathetic outflow suggesting caution when combining B blockers and ECT.

**Clinical and Research Reports**

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**Case Report**

Ms. A was a 68-year-old woman who was suffering from her sixth episode of severe major depressive disorder. Her antipsychotic drug treatment and psychotherapy but partially remitted with ECT. Each episode lasted approximately 2 years and was followed by complete remission and return to normal functioning. Because the depressive episode she was in at the time of this report had not responded to an outpatient trial of pharmacotherapy, Ms. A was referred for ECT. The results of ECG were normal, but her blood sugar had been elevated for 1 month and had been treated with insulin. Her systolic blood pressure was 100 mm Hg (100 mm Hg) and she was maintained on oral propranolol and other medications. There were no significant side effects, but she had been treated with propranolol for a total of 2 months and was hospitalized for a cardiac arrest. Her medical history included a myocardial infarction with a brief hospitalization. She was 63 years old and multinodal diffuse coronary artery disease was discovered at that time and had been treated since then with low doses of insulin. The results of admission laboratory tests were normal except for a fasting blood sugar level of 317 mg/dl and an ECG that showed the signs of the anteroseptal myocardial infarction and nonspecific ST-T wave abnormalities. The results of a physical examination were unremarkable except for a cataract in one eye. A previous history included diabetes mellitus, which had been diagnosed 15 years before the onset of the cardiac arrest. Her blood pressure was 180/120 mm Hg (diastolic = 100-110/180). After unsuccessful attempts to control her anxiety with reassurance and support, we decided to proceed with the treatment with Ms. A's consent. Before ECT, she was given 1 mg i.v. of propranolol, administrated and the standard administration of atropine was omitted. Intravenous pentothal, 100 mg (1.8 mg/kg), and succinylcholine, 30 mg (0.5 mg/kg), were used to induce general anesthesia and muscle relaxation, respectively. From a mask with positive pressure. The treatment was continuously monitored with a bipolar electroencephalogram (EEG) and an ECG. A bilateral electrical stimulation was achieved by a shock-induced activation of the autonomic nervous system and the use of propranolol may have contributed to the cardiac arrest by exacerbating the parasympathetic response associated with the concomitant stimulation.
A Case of Mania Secondary to Vitamin B₁₂ Deficiency

Frederick C. Goggans, M.D.

A case of mania apparently secondary to vitamin B₁₂ deficiency appeared without other overt clinical features of pernicious anemia and resolved with B₁₂ replacement. Six months later, the patient was receiving monthly B₁₂ injections and his mental status remained normal.

(Am J Psychiatry 141:300-301, 1984)

Secondary mania is defined as a manic syndrome of organic etiology with clinical features indistinguishable from those of primary mania (1). Numerous factors—including various drugs and toxins, metabolic disturbances, and infections and neoplastic disorders—have been associated with secondary mania (2, 3). Although many psychiatric symptoms have been associated with B₁₂ deficiency, I present here the first report, to my knowledge, of the full manic syndrome secondary to B₁₂ deficiency.

CASE REPORT

Mr. A, an 81-year-old man, was admitted to the hospital with a 1-week history of irritability mixed with hyperactivity, depression, catatonic speech, hallucinations, sexual indiscretion, and reckless and agitated behavior. He felt that

his home town was glooming a day of celebration in his honor to which several Hollywood personalities were invited. Also, he began to physically recognize that six young men were responsible for his admission. As the time of admission to the hospital.

Mr. A had no personal or family history of psychiatric illness, nor had he any history of severe illness, medical disorders, and he had not been taking any medical treatments before admission.

On admission: Mr. A was oriented in all spheres but was easily distractible. He was agitated, loud, and protested and denied any history of physical or psychological disorder. He had no delusions or hallucinations other than those already mentioned.

Administration of 3 mg of intramuscular haloperidol resulted in marked clinical improvement. Oral haloperidol, 5 mg b.i.d., was continued during the evaluation phase of his hospitalization. The results of general physical and neurological examinations were entirely within normal limits. His hemoglobin was 42.4%, his hemoglobin level was 13.8 g/dl, and his white blood count was 5,000 cells/ml. His red cell morphology was normal. His mean corpuscular volume was receiving 1 μm. No hyperpigmentation or hemeuropathy was noted on the peripheral blood smear. The results of general blood chemistry, a thyroid function test, and a comprehensive drug screen were within normal limits. His CAT scan was normal. The initial serum vitamin B₁₂ level was 116 pg/ml (normal 200-600). A repeat level was 56 pg/ml. Folate levels were within normal limits. The EEG showed slow wave slowing with intermittent 4-6 cycle/sec theta activity. A Schilling test was positive; antibodies to both parietal cells and intrinsic factor were present.

Mr. A was treated with daily vitamin B₁₂ replacement, 1,000 μg iv, for 1 week, then with weekly injections at the same dose. Haloperidol was tapered and discontinued during the first week of hospitalization. Mr. A was treated by a completely normal, normal status was well maintained, and he was receiving monthly B₁₂ injections from his general physician. He had returned to full work activities.

REFERENCES


Although involvement of the CNS in vitamin B₁₂ deficiency, this case emphasizes that psychiatric manifestations can occur in the presence of low serum B₁₂ levels but in the absence of the other well-recognized abnormalities of neurological and psychiatric disturbances. It also suggests that vitamin B₁₂ deficiency can occur in the presence of low serum B₁₂ levels, and it also supports other recent reports on the occurrence of secondary mania in patients with vitamin B₁₂ deficiency. This case presents a new aspect of the mania syndrome in patients with vitamin B₁₂ deficiency.