CASE REPORT

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Suicide by Ingestion of Propranolol

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ABSTRACT: A 60-year-old man with a one-year history of angina committed suicide by ingesting a three-month supply of propranolol. The postmortem anatomical and toxicologic findings are discussed, as is the mechanism of propranolol toxicity.

KEYWORDS: toxicology, suicide, propranolol, beta-adrenergic drugs

Suicide is usually accomplished or attempted with objects or drugs familiar and readily available to the victim. In chronically ill patients whose mobility is restricted by their disease, suicide may be accomplished by the ingestion of drugs not commonly considered suicidal agents.

Case Report

A 60-year-old man first complained of palpitations and dyspnea on exertion in 1976. He was treated with propranolol, 20 mg four times a day, and did well until 1979, when angina developed. Nitroglycerin was added to the therapeutic regimen, but the patient attempted suicide by unspecified means in late 1979 "because of the pain." In May of 1980, he filled a prescription for a three-month supply of propranolol (360 twenty-milligram tablets). The next day, he ingested his entire supply (9600 mg) and informed his family that this was a sincere suicide attempt that he hoped would be successful. He was rushed to the emergency room where he suffered a cardiac arrest 45 min after the ingestion. Following resuscitation he developed profound hypotension refractory to dopamine, isoproterenol, and epinephrine and died 8 h after ingesting the drug.

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Postmortem Findings

The heart weighed 425 g and demonstrated biventricular dilatation. Atherosclerotic narrowing of the coronary arteries varied from 90 to 95% and there was acute thrombosis of the right coronary artery. The 2500-g liver displayed massive fatty metamorphosis, and bilateral aspiration pneumonitis was evident.

Toxicologic Studies

An emergency blood ethanol determination performed on admission was negative. Specimens of blood, gastric contents, liver, lung, kidney, and brain obtained at autopsy were examined for acidic, basic, and neutral drugs. Except for drugs documented as being administered during resuscitative efforts, propranolol was the only drug detected. Propranolol was identified by gas-liquid chromatography (GLC) and quantitated by ultraviolet spectrophotometry as described by Sunshine [*1*] (Fig. 1). The amount of propranolol concentrated in various tissues (Table 1) was determined by GLC (Fig. 2a) in a Perkin-Elmer Model Sigma 1B gas chromatograph equipped with flame ionization detectors and a 1.8-m by 4.0-mm inner diameter glass column containing 2.5% SE30 on 80–100 mesh Chromosorb G. Analyses by GLC were performed with helium carrier gas at 30 mL/min and injector and detector temperatures of 280°C and a column temperature of 180°C. The identity of propranolol was

TABLE 1—*Propranolol concentrations.*

Specimen	Present Case	Previous Reports [*2,3*]
Blood	20 mg/L	9, 4, 167 mg/L
Gastric contents	190 mg/kg	130, 390 mg (totals)
Lung	69 mg/kg	...
Kidney	26 mg/kg	...
Liver	10 mg/kg	140, 170, 958 mg/kg
Brain	6 mg/kg	...

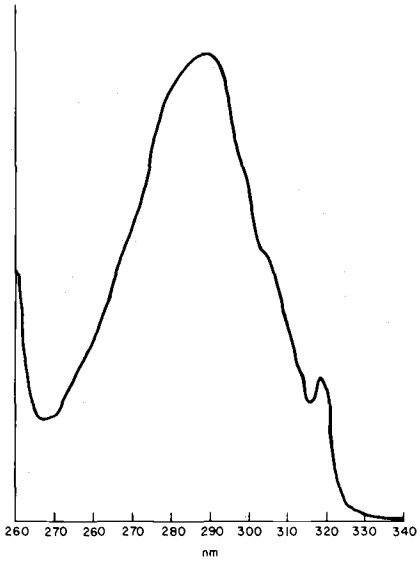

FIG. 1—*Ultraviolet absorption spectrum of propranolol (AFIP Negative 81-12300).*

further confirmed in the Division of Toxicology at the Armed Forces Institute of Pathology with a Hewlett-Packard 5985 mass spectrometer (Fig. 2b).

Discussion

Suicides involving propranolol are rare, and in one of the few reported cases [*2,3*] a combination of propranolol and quinidine was involved [*4*]. In three previously reported cases, the survival time following ingestion is not known [*2,3*]. The discrepancy between the low liver level (10 mg/kg) in the present case and the higher levels in these instances may be a function of metabolism during our patient's 8-h post-ingestion survival. It can be postulated that the victims in the previously reported cases died soon after ingestion since our patient suffered a cardiac arrest 45 min after ingesting the drug.

Propranolol is a potent beta-adrenergic blocking agent with a therapeutic range of 0.03 to 0.2 mg/L of blood [*2*]. Propranolol is used to treat cardiac arrhythmias and in therapeutic doses it causes a decrease in heart rate and blood pressure. A direct myocardial depressant effect has also been postulated [*5*]. The refractory period of the atrioventricular (AV) node is increased and the normal response of the AV node to catecholamines is blocked.

Early signs of toxicity are manifested through a beta-adrenergic blockade, which may cause either slow or rapid development of congestive heart failure. High doses of propranolol

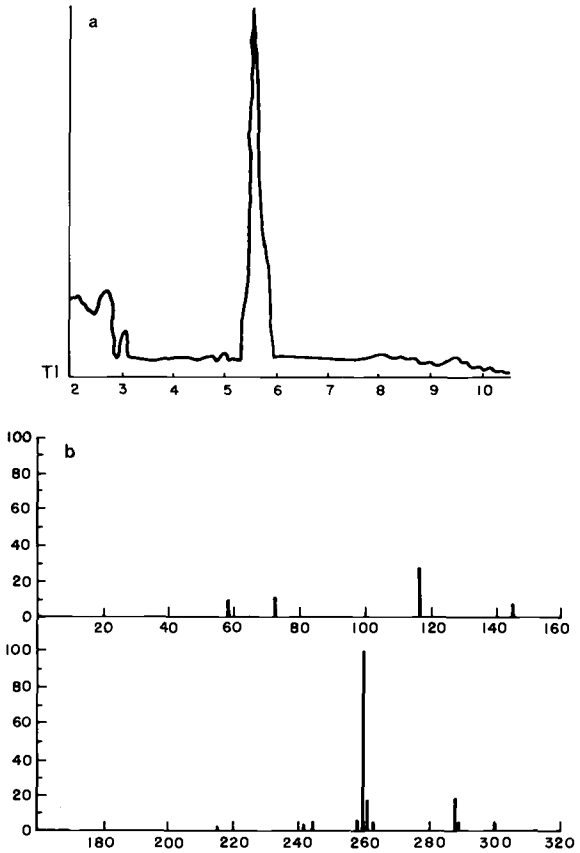

FIG. 2—*Propranolol as determined by* (a) *gas-liquid chromatography (AFIP Negative 81-12302) and* (b) *mass spectrometry (AFIP Negative 81-12301).*

are associated with the rapid appearance of asystole. In the present case, the victim was treated with three powerful beta-adrenergic agonists—dopamine, epinephrine, and isoproterenol—but he failed to respond with either an elevation of blood pressure or the resumption of a normal cardiac rhythm. This lack of response probably was the result of the high concentration of the propranolol saturating all available beta receptors and the inability of beta-stimulating agents to overcome the blockade.

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