CLINICAL REPORTS

Since it has been suggested that the negative inotropic and dromotropic effects attributable to the local anesthetic effects of propoxyphene and norpropoxyphene may play a role, in certain cases, in the demise of individuals consuming excessive amounts of propoxyphene, case reports wherein cardiac abnormalities were reported in association with propoxyphene overdose are reviewed below.

In 1964 McCarthy and Kennan (J.A.M.A., *187*:164–165, 1964) published one of the first reports of fatal propoxyphene overdose. A 15-year-old girl took 1280 mg propoxyphene hydrochloride with suicidal intent. When she arrived at the emergency room she was comatose and cyanotic with shallow respiration. Persistent generalized convulsions began almost immediately, and succinylcholine chloride was given intravenously in an effort to relax the respiratory muscles so that artificial ventilation could be established. Cardiac arrest occurred at this point. An endotracheal tube was inserted, and artificial ventilation and external cardiac massage immediately begun. Nalorphine and levarterenol were administered intravenously. Shortly thereafter blood pressure was noted at 90/40 mm Hg, pulse 120 per minute. The patient remained deeply comatose with continued convulsive seizures separated by periods of apnea. Intravenous and intramuscular diphenylhydantoin and intravenous paraldehyde were administered to control convulsions. Peritoneal dialysis was begun, during which a bigeminal cardiac rhythm was noted that "responded well to intravenous procaine amide hydrochloride" (an antiarrhythmic agent with local anesthetic properties). The patient's course was slowly downhill, complicated by electrolyte imbalance and infection, and she expired 5 days later. Autopsy revealed cerebral edema, atelectasis, focal pneumonia, pleural effusion, and necrosis of the brain. The authors ascribed the episode of cardiac arrest to the severe hypoxia. They also observed that the bigeminal rhythm was easily controlled with procaine amide and that this rhythm disturbance had been noted previously and should be considered in the management of such patients.

Comment: The cardiac arrest and bigemial rhythm almost certainly were engendered by the severe anoxia and cyanosis (and the acidosis that undoubtedly developed). It would seem unlikely that the bigeminal rhythm was caused by the local anesthetic effects of propoxyphene or norpropoxyphene inasmuch as the abnormal rhythm was reported to have responded well to intravenous procaine amide, itself a local anesthetic.

Qureshi (J.A.M.A., *188*:470–471, 1964) reported cardiac and other findings in an 18-year-old woman who ingested 832 mg propoxyphene hydrochloride at one time in a suicidal attempt. She promptly became disoriented and had generalized convulsions followed by cyanosis, coma, and circulatory shock. At examination, heart rate was 110, and apical systolic murmur, respirations slow and shallow with cyanosis. More convulsions, coma, and deep cyanosis followed. ECG revealed sinus tachycardia, nonspecific ST–T changes, and QRS 0.2 seconds, suggestive of intraventricular conduction delay. Appropriate measures were taken to counteract CNS depression and shock, and the patient improved and was discharged after an uneventful course, apparently well, three days later. With clinical improvement the ECG returned to normal.

Comment: In his commentary the author notes that "the cardiac findings in this case may have been due to a direct toxic effect of propoxyphene on the heart; however, the possibility that these cardiac manifestations may have been due to myocardial hypoxia associated with the respiratory depression cannot be excluded."

Sigurd and Jensen (Danish Med. Bull. *18*:166–168, 1971) reported a case of propoxyphene poisoning "complicated by circulatory arrest caused by asystole followed by reversible heart pump failure." The patient was a 45-year-old man without known heart disease who was jailed because of public drunkenness only to be discovered 9½ hours later to be comatose. It was determined later that he had taken a barbiturate and propoxyphene, in addition to alcohol, in an attempt to commit suicide. On admission he was cyanotic and deeply comatose. Cyanosis rapidly became severe, and cardiac arrest occurred; an ECG at that time revealed no cardiac electrical activity. Cardiopulmonary resuscitation measures were undertaken, including intravenous bicarbonate solution and adrenalin I.V. and intracardially. The ECG then revealed widened QRS and absent P waves, rate 80 per minute, no perceptible pulse. A solution of isoprenaline in isotonic glucose was infused, and the ECG then showed less-widened