QRS, absent P waves, rate 150, and a "well filled pulse." The patient continued to improve slowly (BP 130/80 mm Hg on second day), but he remained unconscious during the first two days, without convulsions. The ECG subsequently revealed "tall double-peaker P waves" which remained unchanged during the nine days he was on the ward. At no time were signs of myocardial ischemia seen in the ECG and no rise occurred in serum lactate dehydrogenase.

His hospital course was complicated by a psychosis and transient renal functional impairment in association with a mild diabetes insipidus-like syndrome, possibly as a result of the anti-ADH effect of propoxyphene. (Bower et al., Proc. Soc. Exp. Biol. and Med., *120*:155–157, 1965; McCarthy and Keenan, *vide supra*).

Propoxyphene was detected in the urine by thin layer chromatography, and serum barbiturate was 0.3 mg% (as aprobarbitate, W.H.O.). No plasma electrolyte data are included in the report.

Comment: The patient ingested three CNS-depressant agents in attempting suicide and was not seen medically for 10 hours or more. He was comatose and cyanotic, and cardiac arrest occurred within moments following admission. Undoubtedly the myocardium had sustained injury during the long interval of coma and anoxia. Acidosis was treated promptly, and cardiopulmonary resuscitation ultimately saved his life. There were many factors, obviously, contributing to myocardial ischemia and injury (cardiac arrest persisted 8 minutes). The role, if any, of the local anesthetic effects of propoxyphene and norpropoxyphene in the disturbance of cardiac function in this patient can only be surmised.

The serial electrocardiograms presented in this report suggest the presence of hyperkalemia. The infusion of glucose and the intravenous bicarbonate that the patient received would have tended to improve the ECG. Any hyponatremia accompanying the mild diabetes insipidus syndrome that developed would enhance any electrocardiographic manifestation of hyperkalemia.

Gustafson and Gustafson (Acta Med. Scand., *200*:241–248, 1976) reviewed pertinent laboratory and clinical findings in eleven cases (10 patients, one of whom was admitted twice) of propoxyphene overdose observed at University hospital in Lund, Sweden. None had ingested propoxyphene alone (5 had ingested one or more additional CNS-depressant drugs). In addition, alcohol intake was reported in six.

The principal clinical findings consisted of (1) coma (six patients were in deep coma on admission, four of whom had taken tablets containing barbiturate and one a phenothiazine preparation), (2) depressed respiration, 15/minute or less, in 7 cases (two patients were apneic and severely acidotic and required a mechanical respirator), (3) circulatory abnormalities, (4) metabolic acidosis, (5) convulsions. (The absence of any mention of cyanosis is curious in view of the presence of respiratory depression and periods of apnea.)

With respect to cardiovascular function: One patient on first admission had a systolic blood pressure of 80 mm Hg. On second admission he manifested circulatory arrest (ventricular fibrillation) associated with severe acidosis. Sinus tachycardia was present on admission in five patients, and in the remaining cases the heart rate was normal. The ECG revealed QRS widening in four patients (two of whom had severe acidosis), and in one patient a bundle branch block was noted on admission that was present at discharge 33 hours later, suggesting the prior existence of this conduction defect.

Acidosis was noted in four patients (one of whom was admitted with acidosis on two occasions). Convulsions were noted in only one patient, and severe acidosis was present in this patient.

The two patients with severe acidosis merit additional discussion. One was a 21-year-old man who was first admitted (case 1) comatose after excessive ingestion of alcohol and a propoxyphene-barbiturate-aspirin preparation. Systolic blood presure was 80 mm Hg, but there were no signs of respiratory depression. He recovered over a 20-hour period, uneventfully. However, he was admitted again (case 5) 8 months later, again having imbided heavily and having taken 650 mg propoxyphene, 3.5 gm aspirin, and 500 mg vinbarbital. On admission he was pulseless and apneic. Defibrillation was successful, and intracardiac adrenalin and intravenous isoprenaline were administered. At this time the ECG revealed widened QRS (0.14 seconds), but an hour later the ECG showed sinus rhythm and normal QRS. Mechanical respiration was continued, and a metaraminol drip established to maintain blood pressure. However, his cardiac function ceased 15 hours later. Plasma propoxyphene concentration on admission was 0.74 micrograms/ml, norpropoxyphene 0.39 micrograms/ml.