Only eight cases diagnosed during life as a cyst of the thoracic duct have been reported. Although Steinberg and Watson reported some cases with a jugular lymph sac at the site of entrance of lymphatic channels into the vein, these cases were thought to be different from the other reported cases. Steinberg and Watson concluded that a persistent embryonic jugular lymph sac may become evident in the neck because of incompetence or absence of valves. The case reported by Barlow and Gracey may have the same etiology as a jugular lymph sac. Histologically, our case had inflammatory changes which indicated an acquired origin. In addition, several reports mentioned atherosclerotic or inflammatory changes in the wall of the cyst. Considering some congenital factors, the origin of cysts of the thoracic duct remains the subject of debate.

Although some cases have been reported as symptomless pain in the chest, dyspnea, and dysphagia apparently due to the mediastinal compression by the cyst were significant in our case. An increase in symptoms after meals, as mentioned by Cervantes-Perez and Fuentes-Maldonado, is important and a clue to this diagnosis, but there was no close relationship to the ingestion of a meal in our case. Lymphangiographic studies should be strongly recommended for posterior mediastinal masses when other diagnostic methods fail to make a diagnosis. Surgery may be unnecessary for a symptomless case. A cyst of the thoracic duct should be included in the list of differential possibilities for posterior mediastinal masses.

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Toxic Synergism of Disopyramide and Hyperkalemia*

Bill D. Maddux, M.D.,† and Richard B. Whiting, M.D.‡

A patient is presented whose electrocardiogram became remarkably abnormal and who developed hypotension while receiving disopyramide and potassium. This report documents a potassium-disopyramide synergism leading to life-threatening toxicity. These abnormalities were corrected by glucose, insulin and bicarbonate therapy, suggesting that serious disopyramide toxicity may be enhanced by hyperkalemia and reversed by lowering the serum potassium.

Disopyramide is an effective antiarrhythmic agent which has many of the properties of quinidine. Like quinidine, this drug has variable effects depending on the serum potassium level. This report documents an instance of profound electrocardiographic abnormality and hypotension due to combined disopyramide and potassium intoxication. There was prompt reversal of both the electrocardiographic abnormality and the low blood pressure by correction of the serum potassium.

CASE REPORT

A 65-year-old black man entered the hospital with complaints of epigastric fullness and early satiety. He had ischemic heart disease with a known left ventricular aneurysm, and both mitral and tricuspid insufficiency. At the time of admission, the blood pressure was 110/80 mm Hg, pulse 80 and regular. The jugular veins were distended with prominent V waves evident. The chest was clear to examination. Auscultation of the heart revealed murmurs consistent with mitral and tricuspid insufficiency, and there was a prominent apical S4. His liver measured 16 cm in the midclavicular line and was pulsatile. Medications included disopyramide 150 mg qid, digoxin 0.125 mg daily, furosemide 40 mg daily, and potassium chloride 20 mEq bid. Pertinent initial laboratory data included: Na 141, K 4.9, Cl 108, CO2 22, BUN 30, and creatinine 1.6. The ECG on admission is depicted in Figure 1A.

Because the patient also had known peptic ulcer disease, the diagnosis of gastric outlet obstruction was entertained at the time of admission. For this reason, a nasogastric tube was inserted for continuous suctioning. He was maintained on intravenous fluids, including potassium chloride at the rate of 80 mEq per day. His digoxin, disopyramide, and furosemide were continued. Five days later, the nasogastric tube was removed and oral potassium re-started at 20 mEq bid. The serum potassium ranged between 5.0 and 6.1 during the period on IV fluids. The day after removal of the nasogastric tube, the patient complained of dizziness and weakness. He was noted to have a blood pressure of 60 mm Hg systolic, and

*From the Division of Cardiology, Department of Medicine, University of Missouri Medical Center, Columbia.†Fellow in Cardiology.‡Associate Professor of Medicine and Director, Coronary Care Unit. Reprint requests: Dr. Whiting, Cardiology C-7, University of Missouri Medical Center, Columbia 65212
treated with glucose, insulin, and bicarbonate before the serum electrolyte values were known.

Figure 1C shows the electrocardiogram within minutes after the treatment for hyperkalemia. In addition, the blood pressure promptly returned to 110/80 mm Hg and the dopamine was discontinued. Laboratory values then became available and included: Na 130, K 6.9, calcium 9.5 mg/L, arterial pH 7.39, and Po2 240 mm Hg, using an FiO2 of 40 percent. The serum digoxin was within the therapeutic range at 1.9 nanograms/ml. The serum concentration of disopyramide was 10.8 µg/ml—twice the upper limit of the therapeutic range. A repeat determination of the serum potassium following the use of glucose, insulin, and bicarbonate revealed a value of 4.6 mEq/L. A repeat disopyramide serum level nine hours after discontinuing that medication was 6.6 µg/ml.

DISCUSSION

Superficially, the sequence of electrocardiographic changes and clinical events in this patient could be attributed to hyperkalemia alone. However, a serum potassium level of 6.9 mEq/L is unlikely to cause this degree of aberration, since hyperkalemia should not cause widening of the QRS at levels less than 7.5 mEq/L. In fact, the marked QRS widening seen in this patient would not be expected until a level of 9 mEq/L is achieved. Disopyramide toxicity has previously been reported to cause marked widening of the QRS. Published electrocardiograms in these reports are similar to that of our patient, showing very wide QRS complexes, a right bundle branch block configuration, and also a pattern resembling that of hyperkalemia. Previous reports do not clearly show atrial activity during the recorded disopyramide toxic rhythms. The patient presented here exhibited conduction disturbance at the atrial, AV nodal, and intraventricular levels, all of which quickly corrected as the potassium was lowered.

Disopyramide is similar to quinidine in many respects and similar toxic manifestations seem likely. The electrophysiologic effects of quinidine are enhanced at high concentrations of potassium and attenuated at low potassium levels. The electrophysiologic effects of disopyramide are also attenuated by hypokalemia. The toxic effects of disopyramide are enhanced by hyperkalemia in isolated cell perfusion, but this is the first report of a toxic synergism in man.

In conclusion, a patient is described whose electrocardiogram suggested severe hyperkalemia, but whose serum potassium was only modestly increased. The blood level of disopyramide was clearly in the toxic range. This
agent is known to be dependent upon potassium concentration for its effect. Since both the electrocardiographic and hemodynamic alterations promptly responded to corrections of the hyperkalemia, we suggest that disopyramide toxicity may be seriously enhanced by high serum potassium. In such circumstances, disopyramide toxicity may be reversed by lowering the serum potassium. We believe this patient is the first documented example of the toxic synergistic action between potassium and disopyramide in man.

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Pneumocephalus* 
A Complication of Continuous Positive Airway Pressure after Trauma

Claude E. Klopfenstein, M.D.; Alain Forster, M.D.; and Peter M. Suter, M.D.

We report an uncommon and potentially dangerous complication of continuous positive airway pressure (CPAP) applied during spontaneous respiration. A patient with multiple fractures and recurrent atelectasis developed pneumocephalus on the seventh day of respiratory therapy with CPAP via a face mask. A fracture of the base of the skull, not recognized despite neurologic and radiologic evaluation at admission, was at the origin of this complication.

Respiratory therapy with overall intermittent positive-pressure breathing and continuous positive airway pressure (CPAP) during spontaneous breathing is used frequently for the treatment of postoperative and post-traumatic pulmonary complications, especially atelectasis.1-8 These techniques can provide efficient therapy without requiring endotracheal intubation.8 In this re-

*From the Surgical Intensive Care Unit, Institute of Anesthesiology, Hôpital Cantonal, Geneva, Switzerland.
Reprint requests: Dr. Suter, Hospital Cantonal Universitaire, Geneva, Switzerland

Figure 1. Chest x-ray film obtained at admission to intensive care unit, showing atelectasis of right upper lobe.

CASE REPORT

A 30-year-old man was admitted to the emergency room after an automobile accident. At admission, he had an areactive coma, general areflexia, bilateral miosis, and isocoria. The blood pressure was 70/40 mm Hg, and the heart rate was 130 beats per minute. A right clavicular fracture, a large wound of the right elbow, and an otorrhagia on the right side were diagnosed. Roentgenograms of the skull did not reveal any fracture. Decreased breath sounds were noted over the

Figure 2. Roentgenogram obtained on sixth day of hospitalization. Right upper lobe is normally ventilated, but small area of atelectasis is visible in right lower pulmonary field. Right clavicular fracture is evident.