resenting supraventricular tachycardia with aberrant ventricular conduction. Rapid administration of digitalis was started, and one hour later the rate dropped abruptly to 50 beats per minute. Intravenous therapy with isoproterenol, dopamine, and sodium bicarbonate was administered, and the rate varied between 70 and 160 beats per minute.

Eight hours after admission, a consultant in cardiology reviewed the patient's electrocardiograms and course and believed that the findings were best explained by adrenal insufficiency with hyperkalemia, despite normal levels of sodium and potassium at admission, tachyarrhythmia rather than bradyarrhythmia, and clear absence of significant dehydration. Determinations of serum electrolyte levels were reordered and revealed a sodium level of 123 mEq/L and a potassium level of 9.8 mEq/L. Corticosteroids were administered intravenously. A variety of disturbances in rhythm occurred, including atrial flutter with rapid and slow ventricular responses.

A transvenous atrial pacemaker was inserted, which produced ventricular capture at a rate of 160 beats per minute. An hour later, asystole that was unresponsive to the pacemaker occurred, and the infant was pronounced dead. On the following morning the sample of serum obtained at admission, from which the normal values for sodium and potassium levels had previously been obtained, was located, and the determinations were repeated. On this occasion the serum sodium level was found to be 122 mEq/L, and the serum potassium level was 10.1 mEq/L. At necropsy the adrenal glands of this patient were found to be approximately the same size as the kidneys (a combined adrenal weight of 18.6 gm).

**DISCUSSION**

In Figure 1 are selected leads from the first complete ECG (obtained seven hours before the patient's death), showing a rapid rate of 160 beats per minute, which is at the upper limits of normal for a ten-day-old infant. In leads 1 and 2, the broad QRS complexes and tall T waves, if seen in the context of a bradycardia, would have to be considered diagnostic of hyperkalemia. Looking at leads 3 and aVR, the wave forms are so bizarre as to make it difficult for the nonelectrocardiographer to decide which is a QRS complex and which is a T wave, although such bizarre wave forms should make one entertain hyperkalemia in the differential diagnosis.

We have previously observed that diagnostic problems arise in male infants who have bizarre ECGs. These patients are frequently referred for cardiologic consultation before the total clinical picture of adrenal insufficiency becomes apparent. This case further broadens the spectrum of clinical findings when a patient having the adrenogenital syndrome may exhibit and indicates the need for continuing vigilance because of the propensity of this endocrine and electrolytic disturbance to mimic primary cardiovascular disease.

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**Acute Ventilatory Failure from Sniffing Paint**

**To the Editor:**

No anatomic cause has been apparent in sudden deaths due to sniffing volatile hydrocarbons. Recently, I had an opportunity to observe the progression of clinical events in a patient who had been sniffing volatile hydrocarbons. A medical intensive care unit in a modern hospital provided the unique setting for these observations.

**CASE REPORT**

A 24-year-old man was admitted to the Phoenix (Ariz) Veterans Administration Hospital with a three-day history of muscular weakness which progressed from the lower extremities to the upper extremities. He was having considerable respiratory distress, and studies of arterial blood gas levels revealed acute respiratory failure combined with metabolic acidosis (arterial oxygen pressure [PaO₂], 31 mm Hg; arterial carbon dioxide tension [PaCO₂], 67 mm Hg; and arterial pH 7.11). After suctioning of secretions from the upper airway and administration of oxygen therapy, there was a transient improvement of ventilatory failure (PaO₂, 74 mm Hg; PaCO₂, 41 mm Hg; and pH 7.18). Profound muscular weakness was present in all extremities. The patient, although fully alert, was unable to talk and unable even to hold a pen in either hand in order to sign a consent form for nasotracheal intubation in the event this became necessary. The cough reflex and the gag reflex were absent. The serum level of
potassium at this time was 2.5 mEq/L. The velocity of conduction in the ulnar nerve was within normal limits. The chest x-ray film was normal. The spinal fluid was normal with respect to the presence of cells and the protein content.

Three hours after his admission, while being monitored in a medical intensive care unit, the patient suffered respiratory arrest after a period of labored breathing and hyperventilation. At this time a nasotracheal tube was inserted, and ventilation was maintained with a volume ventilator. The patient became comfortable again, but for several hours, he had complete paralysis of respiratory muscles, with no respiratory excursion at all without the ventilator. The patient required support with the ventilator for 72 hours, at the end of which his vital capacity rose to 1,050 ml. Subsequent spirometric studies showed a complete return to normal values. Hypokalemia and muscular weakness also resolved completely after 72 hours. The patient had received intravenous solutions of potassium chloride and sodium bicarbonate. Additional findings included a urinary pH of 5.5, moderate proteinuria, and numerous red blood cells in the urine. Continuous electrocardiographic monitoring, including continuous computerized analysis for ventricular arrhythmias, failed to reveal any cardiac arrhythmias.

Only after the resuscitation of the patient was his history of sniffing spray paint over a period of four years revealed by the patient's wife. The ingredients of the spray paint included toluene and distillates of petroleum. There had been a recent increase of up to 25 times a day that the patient sprayed this paint into a wash cloth and inhaled it. Only once before did he notice some weakness in the legs and inability to cough effectively.

DISCUSSION

Thousands of children and young people in this country and abroad have been deliberately exposing themselves to repeated inhalation of volatile agents, such as paint, paint thinners, and glue, in order to experience inebriation. Sudden death, toxic polyneuropathy, renal tubular acidosis, and hypokalemia are some of the consequences. Speculation regarding the cause of death includes asphyxiation by the plastic bag, the anesthetic effect of the hydrocarbon, severe cardiac arrhythmia, and respiratory arrest. The case presented here demonstrates, in a single patient, several serious consequences described in different reports and, at the same time, unfolds the chain of events which could have led to death unless appropriate action had been taken.

The progression of generalized muscular weakness was observed in this patient for several hours after the cessation of sniffing the paint. Absent cough and gag reflexes and hypoventilation progressed to respiratory muscular paralysis with retention of alertness until the onset of respiratory arrest. Onset of respiratory arrest was anticipated, and very little time was lost in the establishment of an airway and in management via a ventilator. Muscular weakness was probably associated with hypokalemia, but motor neuropathy cannot be ruled out as a cause. Biopsies of nerve or muscle were not performed. Progression of marked neuropathy and weakness for three months after the cessation of exposure has been described. Anesthesia and severe cardiac arrhythmia can be discounted in this patient.

The speculation regarding asphyxiation becomes interesting when one considers that if the patient described had continued to inhale the paint with a plastic bag over his head, he would eventually have become unable to remove the bag from sheer muscular weakness. His death would have appeared to be from asphyxiation by the bag in an unconscious patient. Yet, the patient was fully conscious, and death would really have occurred from ventilatory failure. Respiratory muscular paralysis or asphyxiation from the bag with consciousness maintained until before death (or both) could account for this. Profound weakness of the muscles of respiration is being suggested as a possible cause of death in sniffers of solvents.

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Pulmonary Varix

To the Editor:

I read with interest the report on pulmonary varices by Romanoff et al in Chest and would like to call attention to our recent report on the same topic. The vascular nature of our patient's lesion was not suspected before mediastinoscopy. We did not believe that resection was indicated, and our patient is still being followed; however, I agree that the lesion may not always be without serious complication and suggest that immediate resection be considered in any patient in whom bleeding or systemic embolism is suspected.

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