Acute Poisoning by Propylhexedrine

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Nasal decongestants may be bought without prescription. We report a case of extreme illness resulting from ingestion of the contents of one nasal inhaler. The illness was followed by the syndrome of "shock lung."

Case Report

A man aged 22 was depressed but otherwise in good health. He swallowed the contents of a Benzedrex nasal inhaler containing 250 mg of propylhexedrine and several hours later was awakened by violent palpitation, headache, and severe central chest pain. On examination he was found to be breathless, with cold sweaty skin and dilated pupils. Initially the blood pressure was 110/70 mm Hg and the pulse rate 120/min, but he became increasingly shocked and the blood pressure fell to 50/0 mm Hg. The E.C.G. showed multiple ventricular extrasystoles, and chest *x*-ray examination showed bilateral pulmonary oedema (Fig. 1). The arterial blood Po_2 was 18 mm Hg.

He was treated supportively with mechanical ventilation, fluid replacement, hydrocortisone (for 48 hours), digoxin, practolol, and lignocaine. Next day the blood pressure was normal but the E.C.G. showed an anterior myocardial infarct and atrioventricular dissociation with an accelerated junctional pacemaker. Improvement, however, continued and on the third day he was taken off the ventilator.

Next day a pericardial effusion developed without cardiac tamponade and chest *x*-ray examination showed surgical emphysema in the right lung field, mediastinum, and neck (Fig. 2). The following day he passed a melaena stool but blood transfusion was not required. A barium-meal examination showed nothing abnormal but the platelet count was 26,000/mm^3.

He then made progressive improvement. The pericardial effusion and surgical emphysema resolved and the pulmonary oedema disappeared. He remained cyanosed, however, with increasing dyspnoea. No abnormalities in the chest or evidence of cardiac failure were found but the arterial blood Po_2 remained at 66-68 mm Hg on air with a normal Pco_2. After a week the chest *x*-ray film showed reappearance of mottled shadowing in both lung fields (Fig. 3). This suggested the syndrome of shock lung (Hardaway *et al.*, 1967), and prednisone 40 mg daily was started with dramatic improvement. Within 48 hours dyspnoea completely cleared and the arterial blood Po_2 on air rose to 95 mm Hg. Over the next two weeks FEV_1/FVC rose from 1·4/1·45 to 3·0/3·25 litres and the pulmonary shadowing disappeared.

Steroids were continued for three weeks and then tailed off completely. There was no recurrence of symptoms and the arterial blood gases remained normal. The pulmonary shadowing did not recur and the patient was well at the time of writing.

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Comment

Propylhexedrine, a sympathomimetic amine, was introduced to replace amphetamine as a nasal decongestant as it has little stimulant action on the central nervous system. Its main action is as a vasoconstrictor with both α- and β-peripheral adrenergic effects (Goodman and Gilman, 1965).

Myocardial infarction has been reported after the administration of sympathomimetic amines (Smith and Logue, 1960) and the pericardial effusion in this case was presumably secondary to the myocardial infarct. The E.C.G. changes of frequent ventricular extrasystoles followed by atrioventricular dissociation were also similar to abnormalitites reported after the therapeutic administration of noradrenaline (Littler and McKendrick, 1957). Atropine was found by these authors to prevent the emergence of arrhythmias, and it may be that prophylactic use of atropine would have helped the present patient. In addition the use of α- and β-adrenergic blocking agents would seem logical. Practolol was used in this case with beneficial effect.

Surgical emphysema probably occurred after bronchial rupture owing to mechanical ventilation but its advent was delayed, and an alternative explanation is prolonged vasoconstriction in the bronchial mucosa leading to ischaemic damage.

The pathogenesis of the condition has not been fully elucidated. Most of the patients with "shock lung" in the series of Hardaway *et al.* (1967) deteriorated and died. In the present case the patient was started on prednisone after the syndrome had developed and made a dramatic recovery, suggesting that this may be the treatment of choice.

Requests for reprints should be addressed to Dr. P. Marsden.

References

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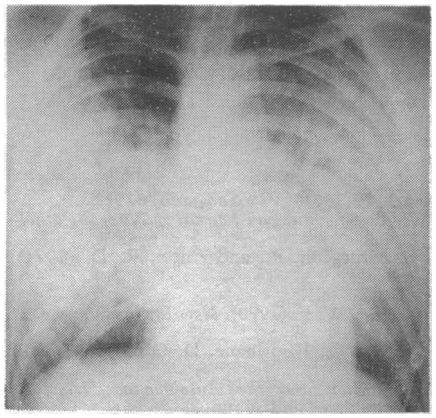

FIG. 1—Chest *x*-ray film on admission (16 January 1971) showing severe pulmonary oedema.

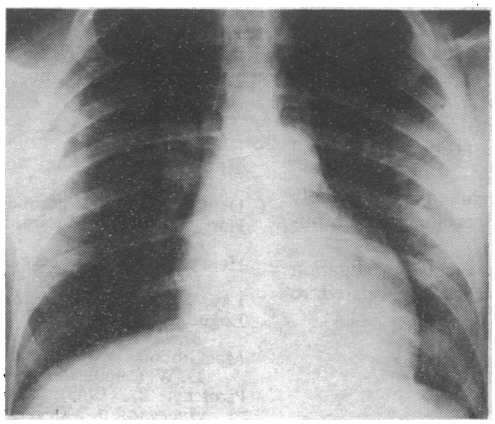

FIG. 2—Chest *x*-ray film on 21 January 1971. Pulmonary oedema has cleared. Note pericardial effusion and mediastinal emphysema.

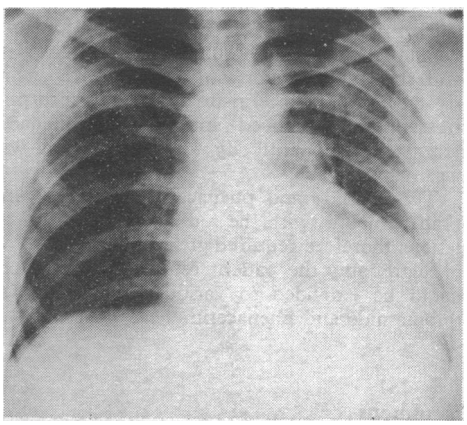

FIG. 3—Chest *x*-ray film on 27 January 1971. Reappearance of lung-field shadowing.