The extremely moderate polymorphonuclear infiltration

...projection and then to massive mucosal herniation,

ward the mucous membrane, giving rise first to folds and

nary disease. The ... failure to clarify their mechanism of formation.

... duplicating the enigmatic structure of the nasal polyps in their variable fibrous, myxomatous and vascular components. The microscopic structure of the polyp in our case well fits in the latter category.

... has considered the nasal polyp a disease of the vascular bed, in the sense that increased capillary permeability and immigration of inflammatory cells can be produced by a variety of injuries, including sensitization to bacteria or to other allergens. One is tempted to apply the same concept to the interpretation of our bronchial polyp. Vascular congestion and tissue edema were prominent histologic features. It may well be that the pressure resulting from the accumulation of intracellular and extracellular-bound edema fluid pushed forward the mucous membrane, giving rise first to folds and projections and then to massive mucosal herniation, which ultimately was responsible for the occlusion of the bronchial lumen and the ensuing pulmonary atelectasis. The extremely moderate polymorphonuclear infiltration can find a likely explanation in the increased capillary permeability, from trauma or secondary infection, or a combination of both.

REFERENCES


Treatment of Propoxyphene Overdose with Naloxone*

Edward S. Kersh, M.D.

Reported are two patients, one child and one adult, with propoxyphene-induced coma successfully reversed with naloxone. We believe that this is the first report on the use of naloxone for propoxyphene overdose.

The clinical manifestations of overdose with propoxyphene and of overdose with opiates have been noted to be similar and include apnea, pinpoint pupils, pulmonary edema, CNS depression, and convulsions. There have also been reports of true dependence to propoxyphene and manifestations of withdrawal syndrome, including perspiration, tremulousness, nausea and delirium.

Although regarded as a non-narcotic, the chemical formula of propoxyphene is quite similar to methadone. Because of these similarities, trials of nalorphine and levallophan have been suggested in propoxyphene overdose. Their efficacy has been difficult to assess. We report here the first cases of propoxyphene overdose treated with naloxone hydrochloride, a new narcotic antagonist.

CASE REPORTS

CASE 1

The patient, a 22-month-old black girl, was well until the day of admission when her mother noted her to be drowsy and of unsteady gait. The child had been seen playing with her grandmother's handbag, whereupon four propoxyphene 65 mg capsules were noted to be missing. On arrival at the emergency room, the child was lethargic, pulse rate 110/min, respiratory rate 10/min, and weight 11.4 kg. Syrup of ipecac 15 ml failed to produce emesis. Fifteen minutes later, she became comatose and responded to painful stimuli only. Her pupils were constricted, equal, but unreactive to light. Deep tendon reflexes were hypoactive and symmetrical. Emergency laboratory studies included serum electrolytes: sodium 134 mEq/L, chlorides 102 mEq/L, potassium 4.5 mEq/L and CO2 19 mEq/L. Blood glucose and urea nitrogen were normal. Within one minute following the administration of 0.08 mg of naloxone hydrochloride intravenously, she became alert, active and her pupils were dilated. One half hour later, she was somnolent with constricted pupils. Repeat admin-
TREATMENT OF PROPOXYPHENE OVERDOSAGE

The patient, a 24-year-old black woman, was brought to the emergency room in a comatose state. She had a history of tubal pregnancy, pelvic inflammatory disease, and several urinary tract infections. She had been scheduled for cystoscopy on the day of admission. Two months prior to admission, she had undergone a myomectomy of leiomyomata with a cystoscopy. Thereafter, she had been placed on propoxyphene 65 mg, and diazepam 5 mg. She had refilled her prescriptions on the day prior to admission. One hour prior to admission, she was found comatose in her bed by her husband who poured a glass of milk down her throat in an attempt to revive the patient.

An endotracheal tube was inserted and ventilation was assisted with a Bird respirator using 100 percent O₂. One half hour later, repeat blood gas studies revealed pH 7.27, PCO₂ 50, PO₂ 80 mmHg, and O₂ saturation 93 percent. Three hours later, the patient became alert and extubated herself. Chest x-ray film the following morning showed no infiltrates. She was discharged again to taking a bottle of propoxyphene and diazepam, but could not give an accurate statement as to the number of capsules and tablets taken. The patient was discharged the third day to return for cystoscopy in one month. Three weeks later, the patient was again brought to the emergency room in a comatose state with apnea, cyanosis and pinpoint pupils. Vital signs included blood pressure 100/85, pulse rate 110/min, respirations zero. Physical examination was unchanged from her previous admission. She was given 0.4 mg naloxone hydrochloride intravenously in three doses one minute apart whereupon she became alert with dilated pupils immediately after the third dose. She admitted again to taking an entire bottle of propoxyphene and diazepam, but could not give an accurate statement as to the number of capsules and tablets taken. The patient was discharged the third day to return for cystoscopy in one month. Three weeks later, the patient was again brought to the emergency room in a comatose state with apnea, cyanosis and pinpoint pupils. Vital signs included blood pressure 100/85, pulse rate 110/min, respirations zero. Physical examination was unchanged from her previous admission. She was given 0.4 mg naloxone hydrochloride intravenously in three doses one minute apart whereupon she became alert with dilated pupils immediately after the third dose. She was discharged again to taking a bottle of propoxyphene, this time without diazepam. Chest x-ray film and blood sugar were normal and the patient was discharged after 24 hours of uncomplicated hospitalization.

Comment

Naloxone hydrochloride has recently been introduced as a narcotic antagonist. Like other narcotic antagonists, it is the N-ally1 derivative of a potent narcotic analgesic, but in contrast to nalorphine and levallorphan, naloxone does not possess agonist actions (respiratory depression, psychotomimetic effects, analgesia, pupillary constriction, etc) and it is effective in reversing the agonist properties of other narcotic antagonists such as pentazocine, cyclazocine and levallorphan. Naloxone hydrochloride has been recommended for the treatment of respiratory and cerebral depression induced by natural and synthetic narcotics.

Propoxyphene is an analog of the narcotic methadone and, as such, its pharmacologic properties are very similar to those of other opiates. In rodents, naloxone hydrochloride increases the convulsive and mortality threshold of propoxyphene. Pupillography studies in man have shown that naloxone hydrochloride will reverse propoxyphene-induced miosis. Therefore, it seemed reasonable to evaluate the effectiveness of naloxone hydrochloride in the treatment of propoxyphene overdose.

Results in the present report demonstrate that nal-
oxone hydrochloride will reverse coma, respiratory depression and miosis due to propoxyphene overdosage. The effectiveness of naloxone hydrochloride in reversing propoxyphene overdosage provides a significant addition to the emergency room physician's armamentarium. It is important to note that the duration of action of propoxyphene may exceed that of naloxone hydrochloride. Continued patient observation is necessary since repeated doses of naloxone hydrochloride may be required.

As demonstrated in this report, when the etiology of the depression is unknown or due to more than one type of drug, naloxone hydrochloride may serve as a safe and useful diagnostic as well as therapeutic agent in the management of known or suspected cases of propoxyphene overdosage.

ACKNOWLEDGMENT: The naloxone hydrochloride (Narcan) used in this investigation was supplied by Endo Laboratories, Inc, subsidiary of E. I. du Pont de Nemours & Company (Inc), Garden City, New York.

REFERENCES
6 Fraser HF: Propoxyphene antidotes. JAMA 204:299, 1968
8 Kallos T, Smith TC: Naloxone reversal of pentazocine-induced respiratory depression. JAMA 204:932, 1968

Adverse Effect of Surgical Repair of an Atrial Septal Defect in a Patient with Associated Left Ventricular Disease*

Kenneth L. Wanderman, M.D.;** Ruth Jortner, M.D.; Maurice M. Augen, M.D.; and Morris Levy, M.D., F.C.C.P.

The case of a seven-year-old child with atrial septal defect and associated left ventricular disease is presented. Following surgical repair of the defect, her condition deteriorated and she died of progressive heart failure. It is postulated that prior to operation the atrial septal defect provided a mechanism for decompression of the left atrium. After removal of this protective mechanism, the left ventricular disease was manifested by increased left atrial and pulmonary capillary pressure leading to her death. Such a course of events in a child is unusual and illustrates the difficulty in assessing left ventricular function in patients with atrial septal defect.

The development of progressive left ventricular failure following the surgical closure of an isolated atrial septal defect is an unusual course of events. In the case herein reported, such a course took place in a child over a period of three and a half years following operation, and terminated fatally. The case is reported in order to emphasize the difficulty in evaluation of left ventricular function in patients with atrial septal defect, and to demonstrate the hemodynamic deterioration which may occur as a result of repair of the defect, when there is associated left ventricular disease.

CASE REPORT

A seven-year-old girl, born in Morocco, was first admitted to Bellinson Hospital in October, 1964, for diagnostic studies. Her birth weight was 2 kg and she was kept for four weeks in an incubator. At the age of five years she was admitted to another hospital because of congestive heart failure and a cardiac arrhythmia, and was digitalized. Following that, the child had several admissions because of heart failure and was maintained on digitalis and diuretics. Family history revealed that two siblings died at a young age of unknown cause.

Physical examination revealed a poorly developed child, without cyanosis or edema. The neck veins showed prominent "a" and "v" waves. Her lungs were clear. A hyperkinetic systolic sternal lift and apical retraction were palpated. The pulse was regular, 90 per minute. First heart sound was normal and was followed by a grade 3/6 ejection type pulmonic systolic murmur. The second sound was closely split.

*From the Cardiopulmonary Institute and Thoracic Cardiovascular Surgery Department, Bellinson Hospital, Petach Tikvah, Israel.
**Department of Internal Medicine, Shaare Zedek Hospital, Jerusalem. Work partly supported by the Herschus Fund of Shaare Zedek Hospital.
Reprint requests: Dr. Augen, Bellinson Hospital, Petach Tiqva, Israel

CHEST, VOL. 63, NO. 1, JANUARY, 1973