Accidental Intra-arterial Injection of Promethazine HCl during General Anesthesia: Report of a Case

H. Mostafavi, M.D., F.A.C.A.,* and M. Samimi, M.D.*

To our knowledge, there has been no report of accidental intra-arterial injection of promethazine hydrochloride (Phenergan) during general anesthesia.

Report of a Case

A 64-year-old man was admitted to Pahlavi Medical Center complaining of chronic pain and tenderness of the right upper quadrant of approximately a year's duration. Except for opium abuse for more than 20 years, the history disclosed no abnormalities. Physical examination and laboratory studies disclosed no abnormalities except a mass and tenderness in the right upper quadrant. Elective exploratory laparotomy was scheduled.

The patient was premedicated with atropine, 0.5 mg, and meperidine, 50 mg, im, 45 minutes before induction of anesthesia. The back of the left hand was chosen for intravenous infusion of 5 per cent dextrose in water, and a 20-gauge needle was inserted into the vein.

Anesthesia was induced with 2.5 per cent thiopental sodium, 400 mg, followed by succinylcholine, 80 mg, iv, and the trachea was intubated. Anesthesia was maintained with halothane–oxygen–nitrous oxide.

At the time of abdominal incision, the patient developed a generalized skin reaction (erythema type), which was thought to be an allergic response, perhaps due to succinylcholine. He was given an antihistaminic. Since the initial intravenous infusion was infiltrated, promethazine HCl, 25 mg, was injected through a 19-gauge regular needle which we thought was in the right antecubital vein.

When the needle was connected to the iv tube, it was noticed that a pulsating column of blood was entering the tubing. At this point, the diagnosis of accidental intra-arterial injection was made. The needle was kept in place, and approximately three minutes later, the patient developed cyanosis of the forearm and fingers. In spite of the presence of a radial pulse, the hand was cold and cyanotic. Ten milliliters of 1 per cent xylocaine were injected immediately through the same needle, with alleviation of the cyanosis, and the fingers became warm. Twenty minutes later, however, the hand again showed cyanosis, coldness, and edema. The ulnar pulse was absent, but the brachial and radial pulses were present. Twenty milligrams of papaverine were given intra-arterially, without any visible effect. This was followed by another 10 ml of 1 per cent xylocaine solution, which improved the patient's condition. Since the surgeon had done a liver biopsy and blood was oozing from the site of the biopsy, the patient could not be heparinized.

An hour post-promethazine injection, stellate ganglion block was performed; this alleviated the cyanosis and mottling of the skin. Two more stellate ganglion blocks were done during the first 12 hours, with excellent results. The patient's condition was evaluated repeatedly during the next 36 hours and found to be completely satisfactory. He did not complain of pain at any time.

Approximately 48 hours post-promethazine injection, the patient developed erythema which was accompanied by mild edema at the site of injection. At this time, we noticed a large vesicle on the back of his hand, with scattered patches of pallor and erythema of the hand and forearm.

On the fourth day necrotic and gangrenous changes developed on the back of the hand and tips of the fingers. In the next ten days the necrotic changes were reduced in size by simple dry dressings, but finally necessitated skin graft. However, the gangrenous changes of the tips of third, fourth, and fifth fingers progressed, eventually necessitating amputation of these fingers at the distal interphalangeal joints.

Comment

Several theories have been proposed to explain the pathologic responses to intra-arterial injection of various drugs. It was initially believed that tissue necrosis secondary to intra-arterial injection was the result of severe spasm, perhaps because of high alkalinity of the drug, and arterial blockage by formation of acid crystals. Evans and Gray[1] believed that norepinephrine release is the cause of the vessel spasm with intimal damage and thrombosis. Waters[2] suggested that the combination of intravascular erythrocyte hemolysis, crystal formation, and platelet aggregation caused the tissue damage. Cohen[3,4] demon-

* Assistant Professor.
Received from the Department of Anesthesiology, Tehran University Medical School, Tehran, Iran.

strated the importance of arterial damage and thrombosis in initiating the tissue damage. Kinmonth and Shepherd [5] supported this conclusion with animal studies, and also showed that surgical sympathectomy and heparinization reduced the area of gangrene of the ear in rabbits. Biopsy evidence indicates that regardless of the various causative factors, thrombosis eventually develops in all cases in which tissue damage occurs. Although no reported study has dealt with intra-arterial injection of promethazine, the development of necrosis and gangrene in our patient probably involved thrombosis of the arterioles. Phenergan is an aqueous solution of 10 per cent promethazine HCl, *p*H 5.3, recommended for use im or iv.[6]

At present, there is no documented explanation for the development of gangrene after intra-arterial injection of promethazine HCl. The effect of any treatment is difficult to evaluate, since the condition is not clearly understood. However, in treating our patient we could not use heparin. Stellate ganglion block improved the condition temporarily, but could not prevent the development of gangrene.

References

1. Evans FT, Gray TC: General Anesthesia. First edition, volume 1. London, Butterworths, 1959, p 365
2. Waters DJ: Intra-arterial thiopentone: A physiochemical phenomenon. Anesthesia 21:346, 1966
3. Cohen SM: Accidental intra-arterial injection of drugs. Lancet 2:361–371, 1948
4. Cohen SM: Accidental intra-arterial injection of drugs. Lancet 2:409–416, 1948
5. Kinmonth JB, Shepherd RC: Accidental injection of thiopentone into arteries. Brit Med J 2:914–918, 1959
6. Wood-Smith FG, Stewart HC: Drugs in Anaesthetic Practice. Second edition. Washington, Butterworths, 1964, pp 230–232

Pulmonary Embolism Following Spinal Anesthesia: Report of a Case

Adrian F. Newens, LCDR, MC, USN, M.D.

Spinal anesthesia is frequently accompanied by decreases in blood pressure of various degrees. Decreased peripheral resistance, pooling of blood, and diminished cardiac output have been shown to be responsible for the hypotension.[1] The following case history documents sudden hypotension accompanying spinal anesthesia which was relatively resistant to the usual modes of therapy and was associated with cardiac arrhythmia and pulmonary embolism.

Report of a Case

A 42-year-old Negro man was transferred from another hospital with an intracapsular fracture of the left femoral neck, suffered in a fall five days earlier. Treatment had consisted of bed rest, Buck's traction, and analgesics. The patient had been medically retired from military service because of a seizure disorder, related to chronic alcoholism. Maintenance doses of diphenylhydantoin and phenobarbital had kept him seizure-free for two years. He denied cardiorespiratory disease. No other significant facts were documented by medical history.

Physical examination was within normal limits except for the hip fracture. Chest x-ray disclosed no abnormalities. An electrocardiogram showed nonspecific ST–T wave changes and sinus rhythm. Open reduction of the fracture was scheduled for the following day.

After premedication with morphine sulfate, 10 mg, hyperbaric spinal anesthesia was administered using 12 mg of tetracaine, while the patient remained awake and cooperative. The sensory skin anesthetic level was determined to be T-8, and the left leg was elevated for surgical preparation. Blood pressure was 140/90 mm Hg, and the pulse was regular at 80 beats/min. A total of 4 ml of Innovar was administered in increments for further sedation. Within five minutes the blood pressure was inaudible; then it was determined to be 60 torr by cuff occlusion and palpation of the

Received from the Anesthesiology Service, Naval Hospital, St. Albans, New York 11425.

Reprint requests should be addressed to: Chief, Research Unit, Naval Hospital, St. Albans, New York 11425.

The opinions contained herein are the private ones of the writer and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.