DATA EVALUATION RECORD

(1) **CHEMICAL**: Trichlorfon

(2) **TYPE OF FORMULATION**: Unspecified


(4) **REVIEWED BY**:

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(5) **APPROVED BY**:

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Date: ____________________________

(6) **TOPIC**: Case History
(7) MATERIALS AND METHODS:

(a) Poisoning Victim: The patient was a 30-year-old man.
(b) Poisoning incident: He consumed 250 g of vodka at mealtime and then, while drunk, (accidentally?) drank a container of concentrated trichlorfon solution.
(c) Amount of exposure: Unknown
(d) Route of exposure: Oral
(e) Symptoms: 15-20 minutes later, he experienced nausea, vomiting, and frequently loose stools. At admission, he was in serious condition. He was unconscious and had alcohol on his breath. His skin was pale and diaphoretic. His reflexes were sharply diminished. His pupils were constricted to the size of pin points and reaction to light was sluggish. On percussion, lung sounds were heard with weak vesicular respiration.

Heart tones were muffled and rhythmic. The pulse was 72 and slightly full. Blood pressure was 115/75. The abdomen was soft, rumbling and painful in the left and right iliac regions. Gastric lavage was not successful.

Five hours later, the patient was conscious and abdominal pain had diminished. He developed a cough with a heavy mucous discharge. This cough and mucous discharge with dyspnea persisted for "a long time." The pupils remained constricted for a total of 8 days. Two weeks after the poisoning, the patient still felt weakness in his legs but had begun to walk, using a cane.
Neuropathologic evaluation revealed slight horizontal nystagmus when looking to the right. Accomodation and convergence were maintained. Photoreaction of the pupils was satisfactory. Tendon reflexes in the arms were "brisk," but sluggish in the legs. The Achilles reflex was depressed, and the plantar reflex was not elicited. There was mild hypesthesia in the lower extremities of the distal type. The Barre syndrome (lower) was positive. The abdominal reflex was elicited. The gait was paretic. Lasègue's sign was positive on both sides. The diagnosis was polyneuritis superimposed on trichlorophon intoxication.

After treatment for the polyneuritis, the pains in the lower extremities and the paresis were somewhat relieved.

(f) **Emergency treatment**: A dose of 40 ml of 40% glucose was given intravenously with 1.0 ml each of vitamins B₁ and B₆ (concentration unspecified), and 1.0 ml of 0.05% strophanthinh, 5 units of insulin, 2.0 units of 20% camphorated oil, 2.0 units of sordiamine, and 2.0 units of 0.1% atropine were given by injection.

(g) **Continued treatment**: At a "later date" (unspecified), 1.0 unit of 0.1% atropine was given intravenously together with 5.0 units of 0.5% novocaaine three times a day. (The patient received 0.05 g of pure atropine sulfate in total over the course of treatment.) Prednisolone, cocarboxyl, vitamins B₁ and B₆, penicillin, streptomycin, and chromosmon
were also administered and the patient received carbolin "internally."

Treatment for the toxic polyneuritis was not reported.

(g) **Clinical Laboratory findings:** Blood analyses revealed: erythrocytes 4,520,000, hemoglobin 84 units, color index 0.93, leukocytes 8,100, eosinophils 0%, p. (band neutrophils?) 3%, segmented neutrophils 75%, lymphocytes 17%, monocytes 5%, and erythrocyte sedimentation rate 27 mm/hour.

Urine analyses revealed no albumin. Bilirubin by Van den Berg was 0.64 mg%, and residual nitrogen results were 56 and 40 mg%, at 1 and 8 days, respectively, after ingestion of trichlorfon.

Radiographs of the chest organs revealed no changes in the heart and lungs.

**CORE CLASSIFICATION:** Not applicable

(8) **REPORTED RESULTS:** Acute trichlorfon poisoning was complicated by polyneuritis. The combined action of trichlorfon and alcohol may have increased the severity of the intoxication and affected the onset of polyneuritis.

(9) **TECHNICAL REVIEW TIME:** 1.5 hours