(anion gap >12–16 mmol/L), plasma lactate levels (>1 mmol/L), decreased arterial pH (often severe), relatively normal PaO_2 and SaO_2 saturation with elevated peripheral venous pO_2 (>40 mmHg) or SaO_2 saturation (>70%).

Arsenic ingestion produces a "garlic-like" breath odor that is easy to recognize. Cyanide produces a "musty" or "bitter almonds" breath odor that many persons cannot recognize. The cause of death in acute arsenic poisoning most often is hypovolemia from "third-spacing" of fluids and gastro-intestinal bleeding with hypotension and cardiovascular collapse. Administration of potent vasodilating amyl and sodium nitrite cyanide antidotes may be dangerous especially in this setting. Chelators are used to treat acute arsenic poisoning, but survival is determined mainly by supportive measures (volume repletion, transfusion). Late sequellae of arsenic poisoning include peripheral polyneuropathy and bone marrow depression with anemia, leukopenia, and pancytopenia, while a Parkinsonian-like condition is the major sequella of severe acute cyanide poisoning. Pancytopenia in the survivors from the Wakayama event led to the diagnosis of arsenic poisoning.

References:

1. Hall AH: Arsenic and arsine. In: *Clinical Management of Poisoning and Drug Overdose*, Haddad LM, Shannon MW, Winchester JF (Eds), 3rd ed. WB Saunders Company, Philadelphia, PA, 1998, pp 784–789.
2. Hall AH, Remake BH: Cyanide and related compounds, In: *Clinical Management of Poisoning and Drug Overdose*, Hatted LM, Shannon MW, Winchester JF (Eds), 3rd ed., WB Saunders Company, Philadelphia, PA, 1998, pp 899–905.
3. Hall AH: Systemic asphyxiants, In: *Irwin & Rippe's Intensive Care Medicine*, Irwin RS *et al* (Eds), 4th ed. Lippincott-Raven Publishers, Philadelphia, PA, 1999, pp 1797–1806.

Keywords: arsenic; cyanide; laboratory studies; poisoning; signs, clinical

PN6-2
Detection and Identification of Unknown Poisonous Substances: A Poisons Centre Perspective

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In accidents or terrorist actions involving possible poisonous substances, the toxic agent might be difficult to identify. When facing such a situation of poisoning, it is important to define the most appropriate strategy to undertake. Apart from technical examinations performed by the police and other agencies, the medical professionals must take action to guarantee treatment of victims in the best manner.

In unclear cases of poisoning, careful observation and documentation of the clinical signs and symptoms should gear further activities. Could these signs and symptoms be connected to a specific toxic exposure?

For most poisonings, symptomatic treatment alone is sufficient to manage the poisoned patient to full recovery. However, in a certain of poisonings, specific treatment with antidotes might be of crucial importance. In order not to miss important treatment possibilities, it is mandatory to identify or exclude those exposures and poisonings where specific treatment is possible.

Substances that should be included among chemical exposures for which antidote treatment may be important are listed below. In this context, poisonings by pharmaceuticals are not included, because they are less likely to be involved in these situations.

aniline	copper	lead	nitriles
arsenic	crimidine	lewisite	nitites
barium	cyanides	mercury	nitrobensen
bromate	ethylene glycol	methanol	organophosphates
carbamate	fluorides	mustard gas	phenol
chlorate	hydrofluoric acid	nerve gases	phosphorus, white

Keywords: antidotes; chemicals; detection; diagnosis; identification; pharmaceuticals; poisoning; substances; toxicity; treatment

PN6-3
Intoxication with Arsenic Mixed in Curry in Wakayama

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Sixty-seven patients were poisoned with arsenic and four of them died after ingestion of curry and rice in which someone mixed arsenic at a self governing summer festival last year (1998) in Wakayama, Japan. However, it required one week to detect the precise substance that caused poisoning. The purpose of this presentation is to describe the Japanese system for detection of and information about poisonous substances obtained from patient material that resulted from this incident of arsenic intoxication.

The 67 patients ingested the curry and rice at about at 18:00 hours on 25 July 1998. They were taken to 13 hospitals; six were taken to the Wakayama Medical College Hospital at 20:30 hours. Their main symptoms were nausea, vomiting, abdominal pain, and headache. After emergency treatment, serum ChE and urine paraquat levels were measured because there remained doubt about the nature of the poison responsible for the intoxication. Serum ChE was normal and qualitative tests of the urine for paraquat also was negative. The doctor notified the police that he was suspicious that these cases were related to poisoning, and asked for help to detect the presence of a toxic substance in vomit. The police reported the following day at six o'clock that cyanide was detected in the vomit. About half of the victims then were treated with sodium thiosulfate. After one week, the police published that arsenic was detected from patient material. Since one week already had passed since the poisoning, the patients were not treated using BAL that is an antidote for arsenic. Urine arsenic concentration reached normal levels in almost every patient within two months of the incident.

The symptoms and the laboratory data during acute phase of poisoning were not specific for arsenic intoxication, except for the changes in the electrocardiogram (ECG): long Q-T interval and negative T-wave. Thus,