emergency response. This hospital DISASTER paradigm has the following elements:

1. detect the event if occult and determine the event characteristics that will modify in-hospital emergency response;
2. inform persons who immediately need to know of the event, activate the hospital emergency incident command system (HEICS) according to pre-determined criteria or judgment, and initiate key critical emergency response elements (e.g., deploying a portable decontamination facility, postponing elective surgical cases, and deploying chemical antidotes to the emergency department;
3. implement safety and security measures (including an assessment of hazards);
4. accommodate arriving patients through surge capacity, assign personnel to various HEICS units, and assess ongoing patient needs and capacity requirements;
5. sort arriving patients (triage) with the first triage question being whether they are contaminated or infectious;
6. treat arriving patients;
7. empty the emergency department (and other hospital areas as needed) through admission, discharge, or secondary distribution to other facilities; and
8. record patient and event data and implement recovery efforts (including mental health services).

This paradigm was used to summarize the complex elements in previously written emergency department emergency operations plans for: (1) trauma and burn emergencies; (2) chemical emergencies; (3) radiation emergencies; and (4) emergencies with pediatric patients (with biological emergencies and emergencies with mental health needs in progress).

Conclusion: The ADLS DISASTER paradigm may be modified for hospital emergency preparedness and may serve as a convenient tool for logically organizing the complex elements of in-hospital emergency response.

Keywords: disaster; education; emergency department; hospital emergency incident command system (HEICS); preparedness

Prehosp Disast Med 2005;20(5):s156–s157.

What is the Threshold Value for Mixed Venous Oxygen Saturation (SvO_2) in Patients with Acute Cyanide Poisoning?

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Introduction: Although cyanide increases mixed venous oxygen saturation (SvO_2) through its blockade of mitochondrial respiration, the diagnostic value of SvO_2 is unclear in patients with cyanide poisoning.

Objective: This study sought to determine SvO_2 in patients with suspected cyanide poisoning before antidotal therapy.

Methods: This was a prospective study of SvO_2 in six patients who were admitted to Lariboisière Hospital with suspected cyanide poisoning. SvO_2 was determined in blood specimens that were collected from Swan Ganz catheters in patients 1, 2, 4, and 5, and femoral veins in patients 3 and 6 before antidotal therapy was administered.

Results: The mean age of the six patients was 39 years of age (SD = 15 years of age). Four patients were comatose, one had altered mental status, and one had normal neurological status. Relevant data are shown in Table 1. The lowest SvO_2 measured was 83.7%. Only three patients had SvO_2 >90%, a threshold suggesting inhibition of oxygen utilization, including two patients with femoral vein samples.

Conclusion: Inhibition of oxygen utilization may be only transient and area-dependent in humans who are severely poisoned with cyanide.

Keywords: cyanide; cyanide poisoning; mixed venous oxygen saturation; oxygen utilization

Prehosp Disast Med 2005;20(5):s157.

Case Report: Cyanide as an Unrecognized Cause of Neurological Sequelae in a Fire Victim

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Introduction: Early and late neurological manifestations of smoke-induced cyanide poisoning are controversial.

Objective: This presentation describes a fire victim with severe cyanide poisoning, but without carbon monoxide poisoning, which resulted in early and late neurological sequelae.

Methods: This is a case report using emergency medical services and hospital records.

Results: A 60-year-old woman was found apneic, pulseless, and comatose at the scene of a fire. Endotracheal intubation and mechanical ventilation were performed and hydroxycobalamin (Cyanokit®) 2.5 g IV was given at the scene. Before hydroxycobalamin administration, carboxyhemoglobin and cyanide levels were 11% and 69 μmol/L respectively. After hydroxycobalamin administration, her blood pressure was 120/80. Further antidotal therapy was not given. The patient gradually awoke over the next several days. On Day 8, she spontaneously opened her eyes and reacted to pain appropriately. On Day 30, choreoathetotic movements and dysarthria appeared, resolving over the course of one month. An extrapyramidal hypertonia, predominantly involving the left upper arm and face, persisted for six months. On Day 7, a magnetic resonance imaging (MRI) of her brain revealed hypointensity on T1-W and hyperintensity on T2-W1 in the putamini, globus pallidi, and caudate nuclei. On Day 17, computerized tomography of her