are well documented after sodium nitroprusside (SNP) infusions but toxicity has not correlated well with CN^- levels. Appropriate management remains unclear.

In this case a 1 year old 9.8 kg male with sepsis and multisystem failure was treated with a SNP infusion to augment cardiac output. On day 2 of SNP (then at 6 mcg/kg/min) rising whole blood CN^- levels were noted. 10 mg of IV hydroxycobalamin (OHCo) failed to lower the CN^- level (2.3 mcg/ml before and after OHCo). CN^- levels fell rapidly after administration of 3.15 gm of IV sodium thiosulfate (4.0 mcg/ml before, 0.45 mcg/ml 1/2 hour after). Thiocyanate (SCN^-) levels were not significantly elevated (1.3 mg/dl, 1.6 mg/dl before and after OHCo; 1.4 mg/dl, 2.1 mg/dl before and after sodium thiosulfate). Monitoring of clinical course, arterial blood gas values, venous hemoglobin oxygen saturation, and arterial-venous oxygen saturation difference failed to demonstrate significant evidence of CN^- toxicity despite elevated CN^- levels.

This case demonstrated markedly elevated CN^- levels without evidence of toxicity. Venous oxygen saturation data were not clinically useful in assessing CN^- toxicity in this patient. Thiosulfate alone was rapidly effective in lowering CN^- levels.

90. **MAO INHIBITOR/MDMA INTERACTION: AGONY AFTER ECSTASY.** Smilkstein MJ, Smolinske SC, Kulig KW, Rumack BH. Rocky Mountain Poison & Drug Center, 645 Bannock Street, Denver, Colorado 80204-4507.

The use and abuse of MDMA (3,4-methylenedioxy-methamphetamine, "ecstasy") has generated tremendous controversy. Despite apparent popularity, there have been no well documented reports of serious toxicity due to MDMA.

In this case a 50 year old male on chronic monoamine oxidase (MAO) inhibitor therapy ingested a single "natural tranquilizer" (later identified as MDMA) followed one hour later by his usual dose of phenelzine (15 mg). One-half hour after phenelzine he developed hypertension (210/100), diaphoresis, altered mental status, and marked hypertonicity. He was treated with supportive care and recovered fully after a 5-6 hour symptomatic period. The clinical course was typical of interaction between MAO inhibitors and some sympathomimetics including amphetamines. Sympathomimetic-MAO inhibitor interactions can cause excessive release of endogenous bioactive amines. Hypertensive crisis, intracranial hemorrhage, hypertonicity, and hyperthermia have occurred from sympathomimetic-MAO inhibitor interactions.

MDMA shares structural and pharmacologic features with other agents capable of causing these interactions. This case suggests that MDMA can cause significant toxicity in patients taking MAO inhibitors.

91. SEVERE ADVERSE REACTION TO 3,4-METHYLENEDIOXYMETHAMPHETAMINE (MDMA). Brown CR, McKinney H, Osterloh JD, Shulgin A, Peyton J, Olson KR. San Francisco Regional Poison Center, San Francisco General Hospital, University of California, San Francisco.

MDMA (Ecstasy), a substituted amphetamine, had been used in psychotherapy and recreationally with increasing frequency until July, 1985 when it became a Schedule I drug. Although few adverse reactions have been seen, we report a 32 year old woman who ingested a "standard" dose (approximately 200 mg) and rapidly developed visual hallucinations, confusion, and agitation. Pulse was 150, blood pressure 90/50, respiration 36, and temperature 41.6. She rapidly became comatose and hypotensive followed by the development of pulmonary edema and hypoxia. The ensuing 5 days were complicated by persistent hyperthermia, tachycardia, emotional lability, dizziness, tremulousness, insomnia, anorexia, rhabdomyolysis, leukocytosis, coagulopathy, toxic hepatitis, and a herpetic-like rash all of which eventually resolved. MDMA was measured in the drug sample, blood, and urine. Serum MDMA levels were 7 ug/ml. No other amphetamines or stimulant drugs were found. Contrary to popular believe, MDMA is a potentially dangerous drug.

92. PROLONGED ABSORPTION AND TOXICITY FOLLOWING CUTANEOUS EXPOSURE TO NICOTINE. Benowitz NL, Lake T, Keller KH, Lee BL. San Francisco Bay Area Regional Poison Center, San Francisco General Hospital, University of California, San Francisco.

Poisoning with nicotine can occur following ingestion of tobacco or pesticides containing nicotine, use of tobacco-based enemas or after cutaneous exposure to nicotine. We report a case of a patient who had nicotine intoxication, after soaking her skin with a diluted solution of Black Leaf 40 (40% nicotine sulfate.) The patient presented with lethargy, weakness, nausea, vomiting and abdominal cramps. The plasma concentrations of nicotine were 200-300 ng/ml, ten times those found in most smokers. The nicotine level remained above 200 ng/ml over 8 hours while cotinine, the major metabolite of nicotine, increased indicating continued absorption. The patient had clinical improvement by 13 hours, despite constant elevation of blood concentrations of nicotine, indicating the development of tolerance. Smokers are known to develop tolerance to the noxious effects of nicotine, but this is the first documented case of tolerance developing to toxic levels.
The therapeutic implication of this case is that cutaneous nicotine poisoning may be prolonged due to continued absorption despite skin decontamination. Clinicians should anticipate this possibility and be prepared to give intensive care for at least 12 to 24 hours.

93. ACUTE INGESTION OF LEAD NAPHTHENATE/1,1,1-TRICHLOROETHANE. Aleguas A, Johnson PN, Lewander WJ. Rhode Island Poison Center, 593 Eddy St., Providence, RI 02902

A 32 year old depressed male attempted suicide by drinking 8 ounces of an octane-booster containing 55% lead naphthenate (20% elemental lead), 30% 1,1,1-trichloroethane, and 15% mineral oil. He spontaneously vomited shortly thereafter. After initial decontamination (syrup of Ipecac) at an outlying hospital, he was transferred to a tertiary-care center and admitted to the ICU. On admission he was lethargic and ataxic with a whole blood lead level of 130 mcg% and a FEP of 20 mcg%. He was treated with IM BAL and IV EDTA. Subsequent serum levels dropped by day four to 20 mcg% and FEP levels peaked at 35 mcg%; 24 hour urine lead determination reached 1989 mcg during chelation therapy. The patient initially had periods of apparent confusion and intermittent complaints of sensory hallucinations. An EMG performed on hospital day 3 was assessed as normal. The patient was discharged on hospital day 8 to a regional facility for psychiatric and continued chelation therapy.