



NEWS AND INFORMATION  
**POISON  
NETWORK**

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*From the Washington Poison Center Medical Director*

## Seattle hosts North American Congress of Clinical Toxicology

Last September more than 700 people from around the world attended the North American Congress of Clinical Toxicology, held in Seattle for the first time in 30 years. Three decades ago, the meeting drew barely 70 delegates.

### Pre-Congress Symposia

In two days of outstanding pre-Congress Symposia, the American College of Medical Toxicology focused on controversial questions that have plagued the discipline for at least 30 years.

- Steve Borron of Washington, DC set the stage by examining the relationships between **toxicokinetics and toxicodynamics**. He explained how an understanding of the metabolic pathway provides great insight into how dose-response curves can better be interpreted on the clinical scene. As an example, fomepizole is effective when ethylene glycol levels are high and glycolate levels are low—it blocks the metabolism of the conversion. In contrast, when glycolate levels are high after the ethylene glycol levels have fallen, fomepizole is of no benefit.
- South Carolina's Eric Lavonas wrestled with just who needs **dialysis for lithium overdose**—or for salicylate poisoning—and what the goal of such treatment really is. (As one who tried to cope with lots of aspirin poisoning before dialysis became available, his presentation

really hit the mark.)

- Boston's Michele Burns zeroed in on the **serum digoxin level** as it pertained to the possible use of fragments, and Alabama's Erica Liebelt highlighted the overall picture of lead as a poison—providing different inferences for different attendees depending on their home territories.
- Seattle's Pete Rainey cleared the air about day-to-day quandaries of testing for **drug abuse** and approaches to levels of action.
- Jeff Nemhauser of the National Institutes of Health followed with the latest on occupational organic **mercury exposure**—and the persistent data deficit when it comes to plausible treatment.
- Connecticut's Chuck McKay posed the question whether **drug levels actually mean anything**. He concluded that they mean dollars for those promoting the use of levels. And they mean a lot of confusion—often leading to unproven treatments. Let us agree that we need far more and better formal evaluations of blood levels.
- New York's Bob Hoffman stretched our imaginations by examining “sur-

rogate markers” for TCAs, dig, APAP, CO, etc., suggesting caution at the very least.

- At the American Academy of Clinical Toxicology session, speakers zeroed in on the mechanisms and treatments for **toxin-induced liver disease**. Texas's Kathleen Delaney really went “Around the Liver in 45 Minutes” and no one will forget the trip. Other speakers updated us on the metabolic mechanisms thought to be involved, how to recognize the resultant problems and the general principles of hepatic carcinogenesis. Seattle's Bob Carithers reviewed how to recognize and manage fulminant hepatic failure. He followed that with an analysis of idiopathic hepatotoxicity, emerging concepts of how APAP damages the liver and what alternative and complementary medications can do to damage the liver.

The FDA's John Senior concluded with current thoughts about how best to determine the “true incidence” of liver damage from drugs. While we know far more today than half a century ago, there is lots of room for improvement.

### Congress platform and poster presentations

The formal Congress then convened with a mixture of 251 platform and poster presentations. Particularly notable were the following:

**Valdecoxib (Bextra) Overdose: A Case Series.** Before all the bad publicity about COX-2 inhibitors, Ne-

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## North American Congress

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braska, Hennepin and Rocky Mt. Poison Centers had combined their cases and learned that overdoses were very uncommon and that single-agent exposure of less than 5 mg/kg did little and could be managed at home. Recall that unlike the conduct of randomized clinical trials where statistical comparisons are crucial, case series are likely to be far less reliable in coming up with precise probabilities. They do give us ballpark projections to be prudent about following the patient's clinical course.

**Reasons for Outpatient Pediatric Medication Error.** Stremski from Milwaukee's Poison Center examined its experience with the topic. I include his contribution because in prior Congresses there has been a dearth of presentations on drug-related errors. It's almost as if they didn't exist, even after the Institute of Medicine dropped its bomb five years ago. Stremski was confident that dose errors were more common than timing errors and both far exceeded giving the wrong drug. Fortunately, in his series, there were few serious consequences, unlike the report released in January 2005 from the combined experiences of Minnesota's hospitals. There, deaths from medication errors were all too common, but that series tended to use deaths as the sentinel event instead of medication errors.

**Take Too Many ASAs and Call Me From the Morgue.** Susan Smolinski and colleagues from Detroit's Poison Center provided a great reminder that one must be very careful with interpreting salicylate levels when there are significant co-ingestants, particularly among patients who are >50 years of age. In truth, this message came out of Canada twenty years ago, and it seems to become truer every year. Yes, the Done nomogram can provide some

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## CME questions

**Retain your answers and claim 0.5 hours of Category 1 CME credit.**

**1.** Sales of illegal "moonshine"—ethyl alcohol—continue to escalate. Holstege and colleagues completed the first study since 1960 of contaminated moonshine confiscated by law enforcement agents. Of their 48 samples, what percentage was contaminated with lead?

- A. 10%
- B. 30%
- C. 50%
- D. 70%
- E. 90%

Forty-three of the 48 samples (90%) had easily detected lead levels, from a low of 5 to a high of 599 ppb—with more than 60% exceeding the EPA's worry level for drinking water. The alcohol content ranged from 10% through 66% with a mean of 41.2%.

**2.** Chicago's Jerry Leiken and associates reviewed their collective experiences with some 20 patients who had presented to a toxicologic clinic with subjective symptoms compatible with a single "toxic trigger." None described any "chemical sensitivities," but 18 of the 20 could clearly describe a single, one-time exposure 1 month to 6 years previously to which they ascribed their ongoing symptoms. Even after repeated normal toxicologic findings and normal medical work-ups, how many of the 20 patients were able to accept the negative findings?

- A. 8/20
- B. 6/20
- C. 4/20
- D. 2/20
- E. 0/20

Despite all of the negative findings, this group of patients—as well as a number of others cited by the authors—went on searching for confirmation of their belief system. Unfortunately, the authors fail to say how many paid their bills; I suspect the large majority did so.

**3.** Hughes and Dart from Denver's "RADARS" program decided to explore the possibility of seasonal variations in the abuse of prescription opioids. They gathered data from seven other poison centers and accumulated a sample of 4,394 intentional exposures. Which of the following seven opioids proved to be the most frequent culprit?

- A. Hydrocodone
- B. Hydromorphone
- C. Oxycodone
- D. Fentanyl
- E. Oxycontin

The authors found a clear—and surprising—winner. They also found that there are definite seasonal variations in the popularity profile, with oxycodone jumping up in the summer. RADARS continues to monitor this issue, giving a tad of balance to the reports from the Drug Enforcement Administration.

**4.** Smollin and Nelson decided to track just how many of the 237 abstracts presented at the 2001 annual Toxicology Congress ever saw the light of day of publication. They searched PubMed from a year before the abstracts appeared—they were suspicious!—until three years after the meeting. Which of the following best represents the actual percent published in "indexed medical journals"?

- A. 5%
- B. 15%
- C. 25%
- D. 35%
- E. 45%

The time delay until publication averaged only 16 months, but both the content and the number of authors changed significantly. The 57 published articles appeared in 21 different journals. The others are still floating around in cyberspace.

*Answers: (1.) E; (2.) E; (3.) A; (4.) C.*

## North American Congress

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guidance, but it was developed exclusively on kids. Our group confirmed 40 years ago that it had some relevance to adults, but the time-since-ingestion is far less accurate among adults. “Acute-on-chronic” overdoses make the problem even worse. If you have any doubts—and the patient seems to be getting worse—call on a consultant to help decide what is the best course to follow.

### Serotonin Syndrome (SS) From Acute Olanzapine Overdose.

Overall, serotonin syndrome has been only rarely associated with atypical antipsychotics—and a number of toxicologists have urged that antipsychotics be used to treat SS. Suchard and Erickson describe a 36-year-old man who presented with an intentional drug overdose. When he arrived at the ED, he already evidenced many of the symptoms of SS, and he went on to develop more. The staff reviewed his list of medications and suspected that the problem stemmed from sertraline and not the olanzapine. Responding well to management, he regained consciousness. He claimed that he had only consumed a one-time dose of olanzapine—and the laboratory confirmed sub-therapeutic levels in the blood and no sertraline whatsoever. The authors postulate olanzapine displaces serotonin from its 5-HT<sub>2</sub> and 5-HT<sub>3</sub> receptors, increasing activity at the unopposed 5-HT<sub>1A</sub>

receptor. Obviously, much remains to clarify the relative risks of such an unusual event.

**Status Epilepticus after Massive overdose of NAC.** Bailey and colleagues from Montreal reported the case of a 30-month-old girl who got into a lot of APAP. She was started on the British IV NAC routine, only to have the first dose of NAC be a four-times overdose and the second one a forty-times overdose—2500 mg/kg instead of 200. Within five hours she began to develop myoclonus that progressed to irregular breathing and unresponsiveness to pain, going on to acute cerebral edema, intracranial hypertension and death. What can one say, except that at least the treatment was warranted even if it was erroneous dosing.

### Stability of 2-PAM after Discharge from a Mark-1 Autoinjector.

Wolowich and associates sought to determine if 2-PAM degraded after it left the autoinjector (perhaps spurred on by the observation that atropine did not degrade after 50 years in the unventilated basement of New York’s Bellevue Hospital). They emptied the autoinjector into a sterile plastic vial and found that the 2-PAM remained chemically intact for at least 48 hours—letting them conclude that they could transfer the 2-PAM from the autoinjector designed for treating adults into another vial aimed at letting them administer the correct dose

for a child. (But 48 hours does really differ from 50 years!)

**Lead Poisoning Mistaken for Acute Porphyria.** Kansas’s Sawyer and colleagues describe a 47-year-old woman with central nervous system changes, cerebral edema and seizures that had been ascribed to porphyria until someone learned that she had been shot 20 years before and had retained bullet fragments near her C-1 vertebra. The blood lead level was 111 micrograms/dL so she was chelated with difficulty, but eventually her lead level dropped—and the fragments were removed from her spine. Her signs and symptoms gradually improved as the levels of lead fell, but the authors refrain from saying just how “well” she eventually got. In my experience, mistaking porphyria for lead poisoning is a far more common scenario.

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