P&A Newsletter

PAPE & ASSOCIATES, INC.

Specializing in Toxicology

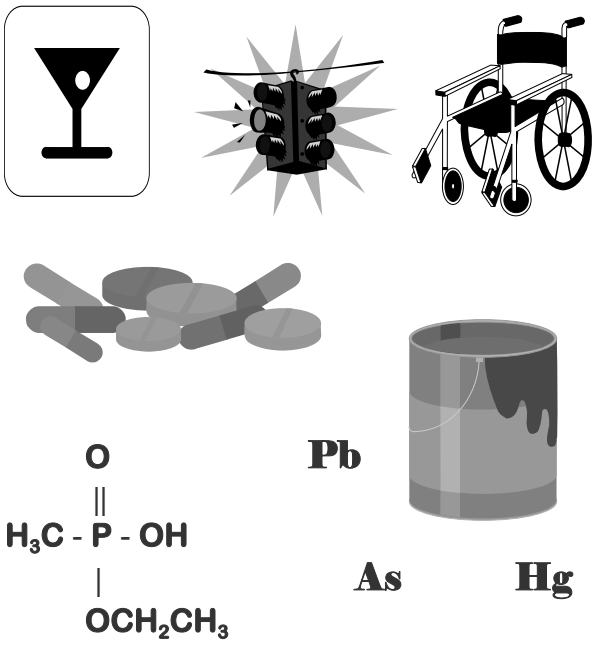
TOXICOLOGY REPORTER

Alcohol-and-Accident

Liquor Liability

Drugs-and-Accident

Toxic Torts



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Specializing in Toxicology

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Dr. Brian Pape specializes in toxicology and related sciences. From 1986 to 1997, he held a faculty appointment as Clinical Associate Professor of Pathology, University of Massachusetts School of Medicine.

From 1982 to 1985, he was Senior Associate Consultant for Mayo Clinic (Rochester, MN), and Director of Toxicology at New England Toxicology Services (Woburn, MA).

From 1973 to 1982, Dr. Pape was Director of Toxicology and Associate Professor in the Department of Pathology at the University of Missouri School of Medicine (Columbia, MO), where he was a member of the Emergency Room Committee, Environmental Trace Substances Research Center Advisory Committee, Medical School Committee on Pathogens-Toxins-and-Carcinogens, and the Technical Advisory Committee of the Missouri Association of Crime Laboratory Directors.

Dr. Pape has published papers, abstracts, and professional articles relating to alcohol and drugs, pesticides and toxic chemicals, analytical chemistry, forensic science, and general toxicology. He currently writes the *Toxicology Reporter.*

He has served as a technical and expert consultant to business, labor, and governmental agencies; and he has been qualified as an expert in toxicology and related sciences in State and Federal Courts.

His expertise has been recognized by American Men and Women of Science, Who's Who in Technology Today, Who's Who in Medicine/Healthcare, and the scientific honorary Sigma Xi.

Board-certifications include the American College of Forensic Examiners (BCFE) and the American Board of Forensic Medicine (BCFM).

Dr. Pape has testified in State and Federal Courts on a wide range of issues relating to clinical, analytical, and forensic toxicology. He has also consulted regarding risk assessment, reliability of laboratory testing, and pre-trial evaluation of expert testimony.

To request additional copies of this or future newsletters or Dr. Pape's participation in a CLE program, fax him at 800-736-9096. Dr. Pape can also be reached at 800-736-0503.

TOXICOLOGY REPORTER

Alcohol-related Accident

Motor vehicle accidents:

There is scientific consensus that alcohol causes deterioration of driving skills beginning at 0.05% BAC or even lower, and progressively serious impairment at higher BACs. *JAMA 255:522-7 (1986).* Research indicates that each 0.02% increase in BAC doubles a driver's risk of being in a fatal crash; the risks increase even more rapidly for drivers under age 21; and, the risks also increase more rapidly for women. Reference to Zador in *Alcohol and Health Res World 17(1):28-34*

Accidental falls:

A large percentage of falls are related to alcohol. Compared to the population, studies of fatal falls among alcoholics report odds ratios of 2.9 to 16. Determination of the odds ratios of significant BACs for fall-related patients compared to disease-related patients yielded odds ratios from 2.5 to 10. 53% of patients injured in accidental falls in the evening in Helsinki Finland and 15% of the time-, site-, and sex-matched control pedestrians were alcohol-involved. Relative risk of injury (if 1.0 at zero BAC), was 3 at BACs of 0.060-0.100%, 10 at 0.101-0.150%, and about 60 at BACs greater than 0.151%. The authors concluded that **(**1) alcohol increases a pedestrian's risk of accidental fall somewhat more than it does a driver's risk of traffic accident; (2) the relative risk of a fall increases with an increase in the pedestrian's BAC; and (3) the risk at BACs greater than 0.100% is so high that practically all cases with such BACs can be considered to have been caused by alcohol. *J Stud Alcohol 44(2):231-245 (1983)*

Aquatic accidents:

Alcohol is associated with an increased risk of neck fracture and spinal cord injury. One study found that 44 percent of the 220 hospital admissions for neck fracture from diving accident showed evidence of alcohol use; and more than 22 percent had BACs greater than 0.10%. Another study reported that subjects who sustained spinal cord injury from diving were four times more likely than controls to have consumed alcohol. Perrine et al. reported that diving performance is impaired at a BAC of 0.04%. *J Stud Alcohol 55:517-524 (1994)*

Snowmobile accidents:

Studies have found strong association between the consumption of alcohol and snowmobile accidents. 64% of the drivers were OUI and fatalities had a ratio of 4.3 for the use of alcohol; 86% of the fatally injured drivers were OUI and had a mean BAC of 0.17%; alcohol was implicated in 69% of the fatally injured and 59% had a BAC over the legal limit; and 80% (24 of 30) of the fatally injured drivers were found to have been DWI. A review of these studies leads to a general overall impression that fatal snowmobile accidents are most frequently associated with the use of alcohol and sub optimal lighting conditions and young male drivers. *Ann Emer Med 24(5):842-8 (1994); Artic Med Res 51(Suppl 7):56-8 (1992); J Can Med Assoc 146(2):147-52 (1992); J Trauma 22(12):977-82 (1982)*

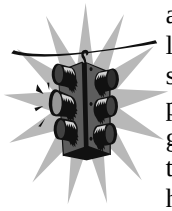
Electrical deaths:

References:

RK Wright and JH Davis, The Investigation of Electrical Deaths: A Report of 200 Fatalities, *J For Sci, 25 (3), 514-21 (1980).* PF Mellen et al., Electrocution: A Review of 155 Cases with Emphasis on Human Factors, *J For Sci, 37 (4), 1016-22 (1992)*

Pedestrian-MVAs:

Of the 10,000+ pedestrians killed and 100,000+ pedestrians injured each year as a result of pedestrian-motor vehicle accident (Ped-MVA), the consumption of alcohol plays a contributing factor in a large percentage of these accidents. Case studies have reported that at least 50 percent of the pedestrians having a BAC greater than 0.10% were responsible for the accident. Studies of risk analysis have reported that as the pedestrian's BAC increased, the apparent risk of accident increased more rapidly than the increase in the pedestrian's BAC. This finding regarding risk assessment is similar to that reported for a driver's risk of MVA. *Reference not cited.*

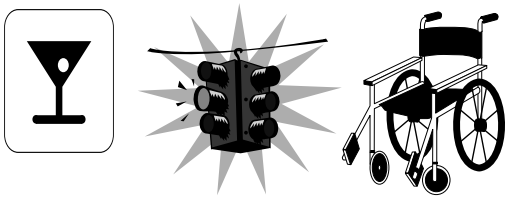


Other types of accident:

Other types of alcohol-related accident include bicycle, boating, hunting, and fire-related accident or death.

Some of the most important aspects of case analysis in an alcohol-related matter are discussed below in "Liquor Liability".

Liquor Liability



Trial testimony by an alcohol expert usually focuses on five case-specific issues:

Alcohol Concentration Test Results

Total Alcohol Consumption

BAC at the Time of Last Service

Reported Appearance-Behavior-Demeanor

Expected Effects Based on BAC

Alcohol test results

In most liquor liability cases, the relevant post-accident alcohol concentration tests are done in hospitals using blood serum. The reliability of a blood serum alcohol concentration test result (BSAC) should be reviewed; and the BSAC should be expressed as an equivalent whole blood alcohol concentration (BAC).

As previously discussed, BSACs are about 15 percent higher than the equivalent BAC. To convert a BSAC test result to an equivalent BAC, divide the BSAC by 1.15. An example conversion of BSAC to BAC follows:

115 mg/dl BSAC = 0.115% BSAC
= 0.100% BAC

Alcohol consumption

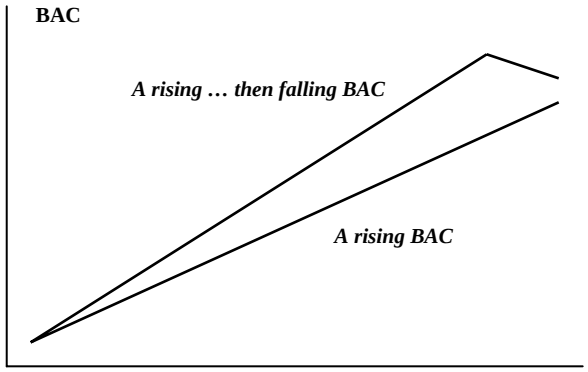
The estimation of a person's total alcohol consumption (TAC) should account for the total amount of alcohol eliminated (TAE) between the time alcohol was first consumed and the time the alcohol test specimen was drawn or the time of death plus the total body alcohol (TBA) in all tissues and fluids necessary to account for the BAC test result.

TAC = TAE + TBA

For example: If a 180 pound male had a BAC of 0.15% after drinking for five hours, the TAE would be equivalent to about three drinks and the TBA would be equivalent to about six drinks. Therefore, the TAC would be equivalent to about nine drinks or the amount of alcohol in about 13 ounces of 80-proof liquor.

Estimation of BAC at a specific time

The estimation of a drinker's BAC at the time of last service is usually based on a process of extrapolation that is applied to case-specific information.



A Case-Example Time-Line

[_____ … __]

1 2 3 4

When (1) is the time alcohol was first consumed, (2) is the time alcohol was last served, (3) is the time of last consumption, (4) is the time the alcohol test specimen was collected or the time of death, and (1-4) is the time period during which alcohol was eliminated.

There are a variety of ways to estimate BAC at a specific time and, indirectly, the alcohol-related effects:

- Retrograde extrapolation from a BAC test
- Forward extrapolation based on consumption
- Range extrapolation based on all variables
- Evaluation of witness statements and testimony

In most cases, the drinker's BAC is rising: The drinker's BAC at the time of last service is usually lower than the BAC at the time of a post-service accident; and the post-accident BAC is sometimes the peak or highest BAC experienced by the drinker.

… at the time of last service, there is usually alcohol in the drinker's stomach and, by definition, there is alcohol in the last alcoholic beverage served to the drinker, … and the drinker usually experiences a rising BAC that does not reach a peak level until sometime after the last swallow of alcohol.

Retrograde extrapolation is the calculation of BAC at a specific time based on one or more alcohol concentration test result(s) at some later time(s) and what are sometimes unspecified or implicit assumptions. These assumptions usually include the following:

Assumptions that (1) the subject was fully post-absorptive and experiencing a falling BAC between the time of interest and the time of the alcohol concentration test, (2) that the rate of elimination used to calculate the

fall in the subject's BAC is reliable, (3) that there were no special factors affecting the subject's post-accident BAC, and (4) that the alcohol test result was reliable.

Example: Assuming that the subject was post-absorptive at the time of the MVA and his BAC was 0.09% 90 minutes after the MVA, what was the BAC at the time of the accident? **Solution:** During the 90 minute post-accident period, the subject's BAC fell about 0.023% (1-1/2 hours x an elimination rate of 0.015% per hour). Therefore, his BAC at the time of the accident was about 0.11%. *Note: The most frequently reported average rate of elimination for adult males is 0.015% BAC per hour.*

Example: Assuming the same facts and a second BAC test result of 0.04% four hours post-MVA, what is the highest reasonable estimate of the subject's BAC at the time of the accident? **Solution:** Assuming that the subject's apparent decline in BAC between the two post-MVA tests reflected the subject's true rate of elimination (0.05% over 2-1/2 hours) at prior times, the apparent rate of elimination would be 0.02% per hour. Therefore, the subject's true BAC at the time of the accident (90 minutes before the first BAC test) would be 0.12%.

Forward extrapolation: Forward extrapolation is based on the analysis of factors that include the time(s) of alcohol service and consumption as well as the time-course and extent of the absorption, distribution, and elimination of alcohol. An alcohol test result is not required; but, when available, the extrapolated BAC and any available BAC test result(s) should be compared.

Range extrapolation: A process that explicitly considers all of the reasonable variables affecting the reliability of the factors involved in the extrapolation of the person's BAC.

Clinical indicia: Depending on the case-specific circumstances, clinical indicia (i.e. visible and/or obvious signs) of intoxication might include witness testimony regarding the subject's appearance-behavior-demeanor. Indicia of intoxication can sometimes be related to an estimate of the subject's BAC.

Effects of alcohol

In almost every alcohol related case, the physical-and-behavioral effects of alcohol and the risk of alcohol-related accident are important issues.

In a liquor liability case, expert case-analysis regarding whether or not a person would be expected to exhibit visible or obvious indicia of intoxication should include the consideration of all reasonable-and-relevant points of comparisons. Examples follow.

Impairment Estimation Procedure (IEP)

While a recent study suggests that a behavioral-based Impairment Estimation Procedure (IEP) can be used to estimate BAC as well as alcohol impairment, the results are not conclusive.

IEP cues for severe impairment seem extreme. Examples include (1) social interaction that is uncontrolled (e.g. urinating), hostile (e.g. cursing), withdrawn (e.g. reclusive), or confused (e.g. loss of memory); (2) physical appearance that is sloppy (e.g. slovenly); and (3) motor coordination that is stumbling (e.g. weaves or falls) or fumbling (e.g. shaky).

IEP cues seem to improve the likelihood that the moderately impaired person will be identified. However, the use of IEP cues (and, presumably, responsible beverage service practices) does not ensure the identification of the moderately impaired patron. Some patrons who are chemically impaired (based on BAC) may be difficult to detect ... presumably due to an acquired tolerance to alcohol or learned behavior intended to avoid detection as visibly or obviously intoxicated.

One study reports 32 cues that were observed while assessing the reliability of IEPs including speaking very loudly, unusual or expanded gestures, and flushed or red-faced. It may be helpful to compare a list of cues with case-specific facts or testimony.

Tolerance to alcohol

One of the few clinical studies regarding tolerance at high BACs was reported in the Journal of Forensic Science. A summary follows.

110 consecutive alcoholics who voluntarily entered a detoxification center were studied to determine their ability to perform certain designated functions (a) while under the influence of alcohol at admission and (b) four days later, after they had undergone detoxification. The findings indicate that alcoholics develop an increased tolerance to alcohol at BACs that are extremely high including levels generally considered potentially fatal.

Witness testimony

The deposition testimony of witnesses often seems to provide information favorable to the defense; and, at trial, deposition testimony is often the defendant's best "*home base*". For both plaintiff and defendant, the approach to taking the deposition of witnesses is very important!

Examination of an alcohol expert

Case-decisions regarding deposition, voir dire, and cross-examination at trial

Case-evaluation and case-strategy are two important considerations when an attorney is deciding if-when-how to examine an adversarial expert. When considering these and other case-specific options, counsel will usually benefit from a discussion with an experienced liquor liability expert.

Expert deposition
There are at least three good reasons to consider deposing an expert:

- You know little or nothing about the expert's approach to case-analysis and his/her ability to defend the approach taken, case-assumptions, case-calculations including BAC and TAC, and relevant scientific studies.

- You want to establish the nature, scope, and limits of the expert's case-analysis as presented in a written report and/or you want to "marry" the expert to a flaw in the case-analysis or written report.

- You want to settle the case and hope to indirectly affect the negotiations by diminishing the perceived impact of the expert's testimony.

When should the expert be deposed? As a general rule, as late as possible ... after you have obtained a detailed report or exhausted all related attempts to define the expert's opinions and/or anticipate the expert's testimony as well as the expert's reaction to cross-examination.

Voir dire
A voir dire is an under-utilized technique. While you might be hesitant to disclose your approach to cross-examination at a pre-trial deposition, you should be much less concerned when conducting a voir dire.

What's in his file?	*What's not there?*
What has he done?	*What has he charged?*
What does he know?	*How does he react?*

Compared to a discovery deposition, a well-devised voir dire can have a much greater impact. The expert is usually not able to effectively rehabilitate his/her lack of case-specific knowledge or approach to case-analysis: *"Isn't it true that when I questioned you about 20 minutes ago, you were not able to …?*

Cross-examination
The effectiveness of your examination is based in large part on your preparation, your anticipation of the content of expert's testimony, the expert's usual behavior, your confidence, the use of control techniques, and a goal of providing the members of the jury with both information and explanation.

Does your cross-examination of the expert reflect a consistent case-strategy that includes ways to effectively present information about the witness's … ?

Qualifications
Knowledge of case-specific facts
Focus (i.e. what he did and did not do)
Implicit and explicit assumptions
Disregard for case-relevant factors
Gaps in testimony re relevant issues
Accuracy when describing the case analysis

Do you visualize and then construct a cross-examination that is organized, understandable, easy to follow, relevant, to the point, interesting-informative-and-illustrative, and persuasive?

Are you able to control the expert?
Are you familiar with the scientific literature, the expert's implicit or unspoken assumptions, and the expert's usual appearance-behavior-demeanor … such that you can confidently and effectively use techniques to control the expert? Are you able to effectively use different types of questions to control both the flow of the examination and the expert's response to the particular question?

Isn't it true that …
Are you able to …
Are you familiar with …
Why didn't you tell the members of the jury …
Have you ever published anything in …
Did you …

Are you able to follow-up?

Isn't that because …
Would you agree with a statement that …
Let's review …

Do you practice and test your trial skills?
Think through example outlines of case-specific questions-and-answers, your reaction to potential adverse answers, techniques you can use to maintain or regain control of the witness and/or focus on your strategic "home-base", follow-up questions and/or illustrations, checklists used to ensure that you have provided the jury with necessary information, and a strong closing.

Have you ever asked an expert to assume the role of an adversarial expert witness … agreeing to a summary of the expert's anticipated testimony and then conducting a telephonic cross-examination?

Elements Key to an Effective Cross-examination

Preparation - Anticipation - Knowledge - Control

Case exhibits for mediation or trial

Well prepared exhibits can focus attention on allegations, relevant case law, case-specific questions, the state of the evidence, implicit or explicit assumptions relied on by an expert witness, reasonable alternative explanations, and case analyses!

It has been said that well prepared case exhibits resemble very effective story-boards.

Drug-related Accident or Incident

Motor Vehicle Accidents:

The use of antidepressants and opiod analgesics by older drivers was associated with increased risk of injurious motor vehicle collisions. *Epidemiology 5(6):591-98 (1994)*

Persons who use minor tranquilizers were 4.9 times as likely to be involved in serious road accidents as those who did not use tranquilizers. Ref. to Skegg et al. in *Am J Psych 142(5):543*

Marijuana-and-MVA:

A case vignette: At 12:15 a.m., an 18 y.o. driver was involved in a two-car MVA. MedFlight took him to University Hospital. Over the next four hours, he received approximately 4.5 liters of i.v. fluids and the equivalent of 0.75 liters of packed red blood cells and 0.5 liters of whole blood. He died at 5:15 a.m. Drug test results follow:

Blood THC	**9 ng/ml**
Blood THC-COOH	**9 ng/ml ***
Urine drug screen	**THC +**

**THC-COOH is a major metabolite of marijuana (THC). THC-COOH has no effect on a person (i.e. it is not biologically active).*

Case review included the following:

The passage of time

The deceased's clinical course did not suggest that the rate of elimination of THC was substantially reduced between the time of the accident and the time of death. The estimation of THC concentration at the time of the MVA would have to take into account the passage of time and the expected rate of elimination.

Even though marijuana was smoked near to the time of the MVA, the deceased's blood THC level was likely falling over the 4-plus hours between the MVA and the time of death. During this period of time, the level of marijuana metabolite (THC-COOH) was likely increasing due to the metabolism of THC → THC-COOH and THC-COOH's slower rate of elimination. *Studies have reported that the ratio of THC to THC-COOH can be used to estimate when marijuana was last smoked.*

The infusion of fluids and blood products

While the dilution-effects on levels of THC and THC-COOH were not estimated, these effects probably caused a reduction of less than 10 percent.

The postmortem blood specimen

Because most studies relating to THC levels and the effects of THC are based on plasma specimens and the reported blood:plasma ratio for THC is about 0.5, the reported postmortem blood THC level was doubled to get the expected equivalent plasma THC level of 18 ng/ml.

Concentration-related effects

Compared to alcohol, the effects of THC are less predictable; but some studies have reported concentration-related effects at THC levels as low as 2.5 ng/ml/. However, a set of effects … even a set of adverse effects … does not always establish a "substantially increased risk of MVA".

Behavioral Effects: Drugs-Alcohol-Assault

Cocaine-ethanol-cocaethylene-and-assault:

As previously described, the consumption of cocaine and alcohol results in the formation of cocaethylene (CE or BEEE).

Cocaine + Ethanol → Cocaethylene

The individual and/or additive effects of cocaine, CE, and ethanol have been associated with adverse behavioral effects including deviant or violent behavior.

Other drugs that have been associated with aggressive behavior include benzodiazepines such as diazepam or Valium.

Drug Use and Aggressive Behavior:

A Common Theory: Alcohol and Drugs

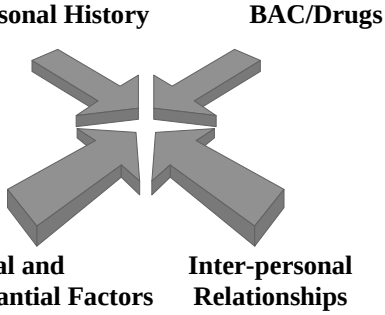
Alcohol-related theories of aggressive behavior are probably applicable to a consideration of the relationship between the use of some drugs and aggressive behavior.

The findings indicate that early aggressive behavior leads to an increase in alcohol use and alcohol-related aggression, but that levels of alcohol use are not significantly related to later aggressive behavior. Thus, the study data suggest that alcohol-related aggression is engaged in by aggressive people who drink. These data

lend support to other research indicating that early aggressive and antisocial behavior is predictive of later alcohol-related problems. Other studies suggest that environmental and situational variables are important.

Alcohol and Aggression: What do most people believe? From Paglia, A and Room, R*; J Subst Abuse 10(2): 199-216 (1998)*: **Over 75 percent of the respondents obtained in a survey of Canadian adults believed that alcohol is associated with aggression** … 92% believed that an intoxicated person is responsible for any behavior and that alcohol is not an acceptable excuse.

Alcohol/drugs and Behavior: Case Factors



Behavioral theories include the following:

Physiological disinhibition theory: Alcohol/drugs increase aggression directly by depressing the brain center that normally inhibits aggressive behavior.

Expectancy theory: Alcohol/drugs increase aggressive behavior because people expect it to increase aggressive behavior.

Indirect cause theory: Alcohol/drugs increase aggression by causing changes within the person that increase the probability of aggression (e.g. by reducing intellectual functioning).

Based on the uncertainties regarding the application of only one of these theories to a case-specific situation, it is prudent to consider all reasonable theories and relevant factors as part of a case analysis.

Worker's Compensation: Death Case

Information and analyses related to the investigation of a suspect drug-related death

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Case Vignette: As the result of a work-related accident, the deceased was permanently disabled and prescribed medication for chronic pain (narcotics), muscle spasm (cyclobenzaprine), and depression (amitriptyline). He was last seen by his wife about 12 hours prior to discovering his body on the family room couch. He was pronounced dead at the scene and his body was transported to the Office of the Medical Examiner. An autopsy was done 24 hours later.

Prior medical history

Prior medical history included concerns expressed by treating physicians regarding the deceased's dependence on narcotics and poor-compliance with the prescribed use of antidepressants. Three physicians were identified; and review of the patient's medical records disclosed some duplication of prescriptions for narcotics.

Prior pharmacy records

24-month records from three local pharmacies were reviewed and compared with the available medical records and prescriptions. The pharmacy records indicated that three physicians prescribed narcotics (oxycontin or hydrocodone) in the 12-months prior to death. The records of one of these physicians were not included with the deceased's prior medical history. Discovery was expanded to include this physician.

Prior medical-pharmacy claims

This review disclosed claims related to one out-of-state pharmacy that provided oxycontin 11 months prior to death. A related medical record was never identified and further discovery was not pursued.

Police report of death

The police report of the death investigation referred to a suicide note, concern regarding depression, and reference to a recent suicidal ideation. One empty prescription container for the most recent month's prescription of cyclobenzaprine was found in a waste basket located in the kitchen. Assuming that month's medication was used as directed beginning with the day it was refilled, 22 pills were missing.

Physical evidence

Physical evidence recovered by the police included six containers from pharmacies-of-record. The evidence was consistent with an impression that the deceased had accumulated narcotics by *doctor shopping*.

Statements relative to the 24 hours prior to death

The deceased was last seen alive in the late evening hours … about 8 hours prior to the time he was discovered. He was described as being quiet but in generally good spirits.

Statements of close friends or others

No other statements were obtained.

Ambulance report

The EMS record indicated initial rigor consistent with death at least six hours prior to arrival.

Death-related medical records

There was no emergency medical treatment.

Autopsy report and related notes

The autopsy report included findings of pulmonary edema and pre-existing heart disease. Stomach contents, heart blood, and urine were submitted for toxicology.

Toxicology test results

Stomach contents were positive for amitriptyline. Urine was not tested. Blood drug test results follow:

Amitriptyline	3450 ng/ml
Nortriptyline	1005 ng/ml
Oxycodone	120 ng/ml
Cyclobenzaprine	22 ng/ml

Results interpretation

While the test results for the antidepressant (amitriptyline) and its biologically active metabolite (nortriptyline) were well in excess of the therapeutic range and potentially lethal, these postmortem drug levels could have been increased as a result of a perimortem disequilibrium effect or postmortem diffusion and/or redistribution effects. Nevertheless, assuming a doubling of the actual premortem drug levels, the adjusted tricyclic antidepressant concentrations would still be considered potentially lethal. *Both amitriptyline and nortriptyline are potentially cardiotoxic and the subject of case reports of fatal cardiac arrhythmia.*

Cyclobenzaprine is also potentially subject to a postmortem redistribution process, and the results should be interpreted with caution. The result did not explain the 22 missing pills.

Oxycodone is not thought to be subject to a postmortem redistribution process. The level was in the toxic range.

Deposition of a *knowledgeable person*

The deceased's wife testified that her husband had not consumed oxycodone for at least two months prior to his death. She also testified regarding her discussions with her husband and his treating physicians regarding his apparent reliance on "pain medication".

A written report addressed all relevant topics:

Doctor Shopping and/or drug accumulation

Expected drug dose:concentration relationships

Amounts of drugs consumed

Self over-medication or overdose

Interpretation of drug test results

Cause of death

Inconsistency of other case testimony

Two of the most common toxic torts are discussed in the next section.

Toxic Torts

Carbon Monoxide (CO):

Absorption-Distribution-Elimination of CO

CO is readily absorbed through the lungs; it binds with hemoglobin in red blood cells; it is distributed throughout the body in proportion to the blood volume and heme-containing proteins; and CO is largely eliminated post-exposure as a result of a relatively slow process involving exchange with oxygen. Blood carboxy-hemoglobin (CO-Hgb) concentration is usually expressed as a percent saturation (i.e. the % of hemoglobin combined with CO).

Mechanism of CO Toxicity

Toxicity associated with a decrease in the availability of oxygen at the cellular level is due to a reduction in the oxygen-carrying capacity of red blood cells as a result of the formation of carboxy-hemoglobin (CO-Hgb), a decrease in the dissociation of the available oxygen carried by RBCs (due to a shift in the oxygen dissociation curve), and the binding of CO to other heme-containing proteins including the cytochrome system responsible for cellular respiration.

CO + Hgb → CO-Hgb → Effects

Signs and Symptoms of CO Intoxication

Organs with the highest metabolic rates (oxygen requirements) are the most sensitive. For example, heart and brain tissue. Other factors that relate to target-organ toxicity include rate of respiration, metabolic requirements (exercise), and anemia. As a general rule, CO-Hgb level and effects are related to the level of inspired CO, the degree of physical activity, the duration of exposure, and pre-existing cardiovascular or cerebrovascular disease.

Following CO poisoning, potential neurological effects include change in mental status, coma, decerebrate rigidity, decreased comprehension-coordination-spatial reasoning-visual acuity, and short-term memory loss. Later complications include ARDS, myocardial damage, renal insufficiency, and neurological abnormalities. Longer-term neuropsychological sequelae of CO intoxication include deafness, blindness, impairment of memory, mental retardation, Parkinson-type syndrome, and/or personality change.

Diagnosis of CO Poisoning

The diagnosis of CO poisoning includes history, physical examination, clinical laboratory testing, response to treatment, and psychometric testing. *Ref.: Comp. Rev. in Toxicology, PD Bryson (1996)*

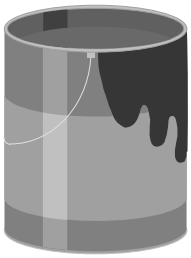
The most controversial CO-related complaints are attributed to multiple chemical sensitivities.

Lead Paint in a Home:

Expert review-consultation-report-and-testimony in a lead paint case usually includes the following:

Environmental surveys for lead

Evaluation of the methods used to test for lead or biochemical markers of lead exposure



Computerized biokinetic modeling of factors relating to the exposure-absorption-distribution-and-elimination of lead and comparison with measured blood lead levels

Construction and evaluation of time-lines and test results relating to locations (e.g. residences), sources of lead (i.e. potential exposures), blood lead (BPb) and erythrocyte protoporphyrin (EP) levels and BPb:EP ratios as indicators of the onset-duration-extent of exposure, medical treatments-effects, re-exposure to lead, the effects of lead, and confounding factors

Computer-based searches of the most current scientific literature, a *paper-chase* or search for expert publications or prior inconsistent statements, qualification of experts, trial exhibits, examination of experts, written report, and expert testimony

A written report usually includes the following:

What were the sources, duration, and extent of the person's exposure(s) to lead?

Factors include residential time-lines, residential and environmental sources of lead and the associated lead levels, as well as time-specific events such as lead abatement(s).

What were the increases in the person's body burden of lead associated with these exposures?

Factors include the absorption-distribution-elimination of lead … allowing for computer-based biokinetic modeling of blood lead (BPb) levels.

What were the physiological, biochemical, and behavioral indicators that are consistent with lead? How sensitive and selective are these indicators for lead-related effects compared to other conditions?

In same cases, a toxicologist is also asked to report and/or testify regarding the generally accepted adverse effects of lead including neuropsychological effects; however, this would usually not eliminate the need for an experienced medical expert and a neuropsychologist.

Lead Paint Case Review: Special Topic

Corrected BPb and BPb:EP Ratio

BPb:EP ratio as a biological index to the duration of exposure to lead and/or re-exposure

Because blood lead (BPb) and erythrocyte protoporphyrin (EP) levels are affected by a variety of metabolic and physiological factors, it may be useful to evaluate these data in non-traditional ways:

Plot a graph of the specimen collection date versus BPb levels that have been corrected for hemoglobin (Hgb) concentration; then,

Divide the BPb concentration by the Hgb concentration. For example:

BPb/Hgb = 45 mg/dl / 15 gm/dl
= 3 mg Pb per gm of Hgb

Plot a graph of the specimen collection dates versus the EP or FEP levels that have been corrected for the specimen's Hgb level;

Plot graphs of the specimen collection dates versus BPb or corrected BPb and EP or FEP levels; and,

Overlay these graphs with information including dates of residence, lead abatement, chelation treatments, and Pb-related graphs for siblings.

If you have general or case-specific questions, feel free to call Dr. Brian Pape.

Dr. Pape is a board-certified toxicologist with experience in a wide variety of toxic torts including lead paint, pesticides, carbon monoxide, chemical asthma, chemical burns, cosmetics, and solvent vapors. His direct toll-free phone number is (800) 736-0503.

YOUR NOTES:

Brian Pape, Ph.D., BCFE, BCFM

Dr. Brian Pape specializes in toxicology and related sciences. From 1986 to 1997, he held a faculty appointment as Clinical Associate Professor of Pathology, University of Massachusetts School of Medicine.

From 1982 to 1985, he was Senior Associate Consultant for Mayo Clinic (Rochester, MN), and Director of Toxicology at New England Toxicology Services (Woburn, MA).

From 1973 to 1982, Dr. Pape was Director of Toxicology and Associate Professor in the Department of Pathology at the University of Missouri School of Medicine (Columbia, MO), where he was a member of the Emergency Room Committee, Environmental Trace Substances Research Center Advisory Committee, Medical School Committee on Pathogens-Toxins-and-Carcinogens, and the Technical Advisory Committee of the Missouri Association of Crime Laboratory Directors.

Dr. Pape has published papers, abstracts, and professional articles relating to alcohol and drugs, pesticides and toxic chemicals, analytical chemistry, forensic science, and general toxicology. He currently writes the *Toxicology Reporter.*

He has served as a technical and expert consultant to business, labor, and governmental agencies; and he has been qualified as an expert in toxicology and related sciences in State and Federal Courts.

His expertise has been recognized by American Men and Women of Science, Who's Who in Technology Today, Who's Who in Medicine/Healthcare, and the scientific honorary Sigma Xi.

Board-certifications include the American College of Forensic Examiners (BCFE) and the American Board of Forensic Medicine (BCFM).

Dr. Pape has testified in State and Federal Courts on a wide range of issues relating to clinical, analytical, and forensic toxicology. He has also consulted regarding risk assessment, reliability of laboratory testing, and pre-trial evaluation of expert testimony.

To request additional copies of this or future newsletters or Dr. Pape's participation in a CLE program, fax him at 800-736-9096. Dr. Pape can also be reached at 800-736-0503.

TOXICOLOGY REPORTERs

ALCOHOL-RELATED TOPICS

The Fate of Alcohol

Absorption-Distribution-Elimination

Alcohol Testing

Case Circumstances
Specimens and Issues
Test Methods
Results Review and Discovery

Effects of Alcohol

Alcohol and Accident
Behavioral Effects

Liquor Liability

Five Alcohol-related Issues
Analysis of Points and Counter-points
Case Consultation
Examination of Witnesses
Written Report
Expert Testimony

Alcohol-related Terminology

DRUG-RELATED TOPICS

Fate of Drugs

Consumption-Absorption-Distribution-Elimination and Case-illustrations

Therapeutic Drug Monitoring

Premortem Drug Testing

Hospital Drug Testing
Probationary Drug Testing
GC and GC-MS Drug Testing

Postmortem Drug Testing

Discovery – Review – Retesting

Effects of Drugs

Drugs and Accident
Drugs and Behavior

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Drug-related Terminology

TOXIC TORT: TOPICS

Risk Assessment

Carbon Monoxide

Lead Paint

VX Nerve Gas

Organophosphate Pesticides

Cosmetics - Adverse Reactions

Chemical Burns - Acids and Bases

TOXIC TORT: TOPICS

Acetaminophen (*Tylenol*)

Methyl Salicylate (*Oil of Wintergreen*)

Residential Pesticides (*Dursban*)

Adulterated Foods and Beverages

Food Additives – Allergic Reactions

Estimating Airborne Chemical Exposure

Using the Internet

Toxicology Reporters are written by Dr. Pape.

Dr. Pape can be reached at (800) 736-0503.