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MEDICAL TOXICOLOGY CONSULTATION:

Male, deceased

File No: 10159

Date: 7/6/04

Medical toxicology consultation on the above-captioned matter is requested by (Attorney) by way of letter dated June 29, 2004.

For purposes of evaluation, I am provided the deposition of Trooper taken May 12, 2004; Accident Report; and report of the post mortem examination of Male; and analyses performed for cocaine metabolites on 18-wheeler driver.

The records reveal an accident having occurred at 12:05 p.m. on April 29, 2002, involving a collision between an 18-wheeler in the process of making a U-turn and a vehicle driven by Male that rear-ended the 18-wheeler. Male died at the scene of the accident. Post mortem examination of Male concluded that death occurred as a result of blunt force injury. Among other post mortem findings were presence of severe coronary artery atherosclerosis, remote posterior myocardial infarct, but status post coronary artery bypass grafts times four; and left ventricular hypertrophy and dilatation.

Approximately four hours after the accident the 18-wheeler driver provided a urine specimen for drug screening. The specimen tested positive for the cocaine metabolite, benzoylecgonine. The positive screening test was referred for definitive analysis using gas chromatography/mass spectroscopy, which revealed a concentration of benzoylecgonine of 471 nanograms per milliliter of urine (ng/ml). At the time of the accident, the 18-wheeler driver was cited for making an illegal turn, and just over a year later, was cited for criminal intoxication manslaughter based upon the discovery that the urine drug test was positive for "cocaine."

In his deposition, Trooper, who attended the accident, did not cite the 18-wheeler driver for driving under the influence of drugs because he found no reason to suspect that drugs were involved in the accident, describing 18-wheeler driver as appearing totally normal with no stigmata that he would identify as drug-related impairment.

I am requested to provide an opinion concerning the significance of the laboratory finding of cocaine metabolites as it may relate to the question of impairment as a result of cocaine use having been present at the time of the accident.

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The specimen was collected approximately four hours after the occurrence of the accident. In his deposition, Trooper was asked to opine whether cocaine use would result in impaired behavior that could have contributed to the accident. There was tacit assumption that the urinalysis demonstrated the presence of cocaine, which it did not. The test identified the presence of benzoylecgonine which has no pharmacologic activity. The presence of benzoylecgonine has never been demonstrated by any researcher or any human experience to cause impairment. In this case, the presumption that benzoylecgonine in the urine proved the presence of cocaine was erroneous.

When cocaine enters the body by any route, there is a period of time during which active cocaine is present in the body and physiological responses related to cocaine do occur. The duration of these effects depends on the amount and route of cocaine entering the body. The period of "cocaine-related impairment" varies from a few minutes to as long as two hours depending on the route by which it is absorbed. While cocaine is circulating in the body for a period varying from 30 minutes to several hours, depending on the sensitivity of the test used to detect cocaine, small amounts of cocaine appear in the urine. Under almost all circumstances, the urine becomes negative for cocaine within six to 12 hours. No cocaine was demonstrated to be present in the urine of the 18-wheeler driver at the time the urine specimen was taken. Consequently, cocaine-related impairment at the time of the accident could not have been a causative factor for the accident.

There is widespread misinterpretation of the significance of benzoylecgonine in the urine. There is no relationship whatsoever between benzoylecgonine present at any concentration and cocaine-related impairment. There is only a gross relationship between concentrations of benzoylecgonine in the urine and the duration of time between the most recent use of cocaine and the collection of the urine specimen. This, of course, is a function of the amount of cocaine that was used and the frequency of use of cocaine.

The concentration of benzoylecgonine of 471 ng/ml identified in the 18-wheeler driver is very low, compared to the concentrations measured when cocaine impairment is present.

Based upon the evidence manifest in this case, it may be assumed in reasonable medical probability that use of cocaine was remote by an undetermined period of time, probably exceeding 12 to 24 hours. Consequently, the positive urine test has no relevance except to establish that at some time during several days prior to the collection of the urine specimen, cocaine had been used.

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The widespread use of urine as a screening procedure in the workplace and in safety-sensitive employment has as its purpose the identification of persons who have used cocaine, but has no relevance whatsoever for determination of the presence of impairment. There is no reliable association between the presence of benzoylecgonine and impairment.

For purposes of discussion, I am attaching a section from Baselt's "Disposition of Toxic Substances and Chemicals in Man," discussing cocaine and which provides further details on the rational application of analyses for cocaine and cocaine metabolites in forensic toxicology. The general consensus among the appropriate professional groups that benzoylecgonine has no psychoactivity is reiterated in this discussion, and the persistence of benzoylecgonine in both blood and urine over hours to days beyond the use of cocaine is widely accepted. It is noteworthy that, as reported in the medical literature as well as in my personal professional experience, where specimens have been collected within an hour or less of the time of cocaine use during which time the person may be expected to manifest cocaine adverse effects, these demonstrate quantities into the tens of thousands, if not hundreds of thousands of nanograms of benzoylecgonine per milliliter of biological fluids. Clearly, this did not apply in the instance of 18-wheeler driver.

Also attached are the position statement of the American College of Medical Toxicology concerning cocaine metabolites in urine, and the discussion of the American Medical Association Council on Scientific Affairs concerning scientific issues of urine drug testing.

Should any additional information become available that is relevant to a decision on causation, I reserve the right to review this material.

Eric G. Comstock, M.D.

ECG/cw

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cc:

Attachment:

Baselt RC. Cocaine. In: **Disposition of Toxic Drugs and Chemicals in Man, 2000; pp. 205-210.**

Interpretation of Urine Analysis for Cocaine Metabolites. **American College of Medical Toxicology Position Statement, 2001.**

Council on Scientific Affairs. Scientific Issues In Drug Testing. **JAMA 257(22):3110-3114, June 12, 1987.**

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ADDENDUM REPORT:

Male, deceased

File No: 10159

Date: 7/14/04

Subsequent to my report of July 6, 2004, concerning the above-captioned case, I have received additional material consisting of a report generated by, Ph.D., dated June 8, 2004.

Ph.D. briefly summarizes the only facts available on this case that are relevant to issues of possible causation for alleged impairment of 18-wheeler driver at the time of the accident as a result of the use of cocaine. Ph.D. correctly notes that a urine specimen was collected approximately four hours after the accident and tested positive for "metabolites of cocaine" in a screening test. This was followed by confirmation by gas chromatography/mass spectroscopy of the presence of benzoylecgonine at a concentration of 428 ng/ml. However, in reviewing my report of July 6, 2004, I note that the results were reported as 471 ng/ml. Further it is noted by Ph.D. that an analysis of the specimen further confirmed the presence of benzoylecgonine in the urine specimen.

Ph.D. continues to review in great detail the patient's drug use history, stating that it is important since at issue is the state of the 18-wheeler driver at the time of the accident with regard to alleged cocaine-related impairment. Past history of drug use is irrelevant and prejudicial, failing to address in any way whether the 18-wheeler driver was impaired by virtue of use of cocaine at the time of the accident.

Ph.D. then cites a publication, "Ambre (J. Anal. Toxicol. v.9, p.263, 1995)." Referring to the paper cited by Dr. Wimbish, Figure 2 (page 245) plots plasma levels against time, with injection of 100 milligrams of cocaine intravenously, demonstrating that cocaine itself disappears even at a 100 milligram dose within 10 hours of injection.

The same Figure 2 shows the presence of benzoylecgonine in urine persists for up to 60 hours. Since no cocaine was detected in the urine specimen collected from the 18-wheeler driver, this would place the use of cocaine at some time prior to 10 hours before the urine collection.

It is universally accepted that the metabolite, benzoylecgonine, which persists much longer, is inactive. Consequently, the publication cited by Ph.D. supports that no active cocaine was present in the blood of the 18-wheeler driver near term to the occurrence of the accident.

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The use of the nomogram prepared by Dr. Ambre in his publication has been the subject of considerable discussion and criticism concerning the many variables in the concentration of benzoylecgonine at various times in various individuals and by various modes of administration, causing great uncertainty as to any effort to predict blood levels from a urine specimen, detecting a substance that has no psychological activity. This particular publication has been discussed at length by the Forensic Toxicology Interest Group of the American Academy of Clinical Toxicology during past years, a group of which I am Co-Chairman. The consensus is that prediction of cocaine-related impairment at any particular time as a result of any single urine analysis for benzoylecgonine is unreliable. In the Ambre publication, it is indicated that this is a proposed method to arrive at blood levels based on the more universally available data that concerns benzoylecgonine in urine rather than cocaine in blood. While it is an interesting attempt, it is a technique that has not been subject to correlation with actual impairment measured in individuals with predicted concentrations of the inactive derivative in the urine.

On page 3 of his report, Ph.D. assumes that a 10 milligram dose of cocaine by intravenous administration is "an effective dose for the desired euphoria." This assumes that the cocaine was administered intravenously and fails to define "the desired euphoria" or the time frame over which the desired euphoria may ensue. With the use of cocaine that would closely approximate peak flow levels at the same time as an intravenous dose, the "desired euphoria" occurs in a few minutes and persists for only 15 or 20 minutes. This is based upon my personal professional experience of 25 to 30 years of involvement with the patients who are commonly involved in cocaine use.

Ph.D. goes on to express the opinion that if it is not a "high" that is impairing an individual, it must be withdrawal that is impairing an individual. One cannot have both at the same time. The assumption that after a single injection of cocaine there is symptomatically manifest impairment of function as a result of withdrawal is blatantly absurd. Based upon my long term professional experience with the intermittent use of crack cocaine, normal functioning resumes after the transient "high." The concept of cocaine withdrawal (crash) is a concept derived from repeated use of cocaine over frequent intervals in a short period of time resulting in a continuous state of hyperstimulation from which there ensues a "crash" upon cessation of cocaine use. There is considerable question as to whether the cocaine "crash" is the result of sleep deprivation or frank fatigue as a result of overstimulation for a protracted period of time. Neither of these events have any relevance to the issue of whether the 18-wheeler driver was impaired at the time of the accident by virtue of cocaine use.

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Absent impairment as the result of acute use of cocaine alleged to have occurred in this patient, the backup assumption of Ph.D. is that there is evidence that long term chronic use of cocaine results in impairment that is demonstrated by common neuro-psychological testing. There are a number of publications where highly refined neuro-psychological testing on chronic cocaine users has demonstrated varying degrees of neuropsychological impairment. However, nowhere is there any correlation established and generally accepted that changes in neuropsychological testing parameters are related directly to impairment of functional proficiency under usual workplace environments, so that there is no indication and no way of measuring (even if neuropsychological functions are impaired) the relevance of this to the presence of impairment in the 18-wheeler driver at the time of his accident.

Ph.D. notes the occurrence of cocaine psychosis with long term use of cocaine. Cocaine psychosis occurs with repeated heavy use of cocaine over short intervals and more commonly is manifest at the peak levels of cocaine in the blood stream. Certainly, if the 18-wheeler driver had been suffering from cocaine psychosis at the time of his interaction with the police officer following the accident, such an event or condition could not have avoided perception by the police officer, however gross his perceptive abilities might be.

The facts of the circumstances surrounding this accident consists solely of the presence of an inactive derivative of cocaine in the urine of the 18-wheeler driver in a sample collected four hours after the accident. The substance that was identified has no psychoactivity. Consequently, there are no facts that support the occurrence of impairment related in any way to any amount of cocaine. Further, there are no facts that establish the presence of cocaine in the body of the 18-wheeler driver at the time the accident occurred. Any statement that the 18-wheeler driver was impaired by virtue of cocaine is purely speculation, and history of psychological problems and drug use in the past has no relevance whatsoever to events that prevailed at the time of the accident.

It is therefore my opinion after careful consideration of Ph.D. report that, in reasonable medical probability, impairment related to cocaine use cannot be established as a contributing factor to the accident.

Attached please find the full text of the Ambre publication. Your attention is called to the attachments in my report of July 6, 2004, where I opined there is nothing to support the presence of cocaine-related impairment of the 18-wheeler driver at the time of the accident.

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Introduction of the patient's psychiatric history and introduction of the purely speculative opinion of cocaine involvement in the accident are blatant attempts to introduce prejudice unsupported by the facts.

My opinion is based upon training in toxicology, experience in psychiatric issues related to drug abuse and dependence, a composite of approximately 25,000 patient years in the management of drug abuse and dependence, and delivery of primary care to patients involved in drug dependence and abuse. In addition to the publications cited in my initial report, I am attaching at this time an editorial from Journal of Analytical Toxicology, 2003, concerning review of blood and urine data collected in postmortem cases by the Office of the Chief Examiner of the State of Maryland and the Armed Forces Institute of Pathology. The authors conclude that "urine BE concentrations should be considered cautiously as they do not generally correlate with blood cocaine concentrations; thus, the quantitation of urine concentrations is not necessary when direct prediction of impairment is desired." It is noteworthy that for more than 50 years, the Armed Forces Institute of Pathology has been considered the ultimate authority in disputes in pathology and toxicology.

READ AND SIGNED ELECTRONICALLY

Eric G. Comstock, M.D.

EGC/cw

cc:

Attachment:

Ambre J. The Urinary Excretion of Cocaine and Metabolites in Humans: A Kinetic Analysis of Published Data. **J. Anal. Toxicol.** 9:241-245, Nov/Dec 1985.