LAST WORD SOCIETY

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Tragedy at Gunpowder River: Amtrak-Conrail Collision, Chase, Maryland

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ABSTRACT: The National Transportation Safety Board (NTSB) reported that the collision between an Amtrak train and three Conrail locomotives on 4 Jan. 1987 was caused by impairment of the engineer of the Conrail locomotives from marijuana and by the failure of and lack of safety devices. Evaluation of NTSB's report does not support marijuana impairment as a cause of the accident, but it does reveal many safety problems.

KEYWORDS: Last Word Society, accidents, train accidents

On Sunday, 4 Jan. 1987, at the end of New Year's weekend, an Amtrak train carrying 660 passengers and 5 crewmen rear-ended a stopped train of three Conrail locomotives with a crew of two, an engineer and a brakeman. The Amtrak engineer and 15 passengers were killed and 174 passengers were injured. The damage to the railroad was estimated at $16.5 million [*1*].

The Amtrak train had two locomotives, three food service cars, and nine coaches. At the area where the crash occurred, the Conrail locomotives had switched to the north track. Five trains were scheduled to use the two tracks between the time of the 1:30 p.m. crash and about 1:40 p.m. (see Fig. 1). None of the trains was running on schedule.

The Conrail train left the yard on a side track about 14 miles (22½ km) south of the accident site at 1:16 p.m. The Amtrak train left Baltimore 17 miles (27 km) south of the site at 1:16 p.m., about 5 minutes late. The Amtrak train, which was restricted to 105 mph (169 km/h), left 4 min ahead of a Metroliner train that was running 47 min late and was to travel at 125 mph (201 km/h) on the same track as the Amtrak and Conrail trains. At about the same time, two trains were coming south. One was 10 min late and had a speed limit of 90 mph (145 km/h), the other, on a separate track and 2 min behind the first, was running 10 min late and had a speed limit of 105 mph (169 km/h). The plan was to have the northbound trains clear the northbound track so that the two southbound trains could use both of the tracks into Baltimore. The Conrail train was running at its

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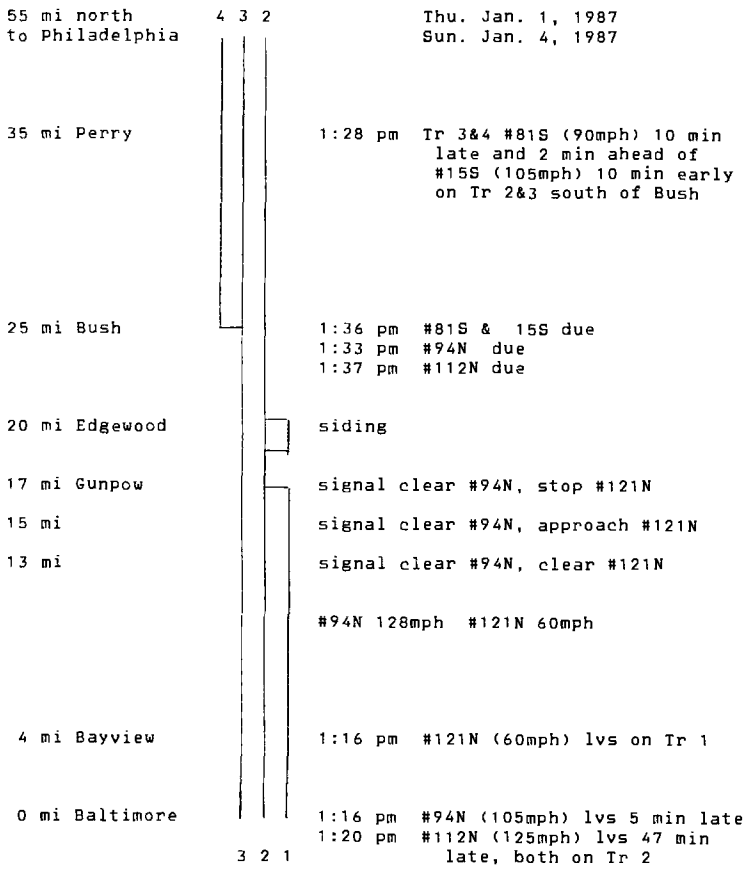

FIG. 1—*Details of the Amtrak-Conrail Crash.*

proper speed of about 60 mph (97 km/h) on the side track when it was alleged that the two crewmen missed a signal warning the crew to restrict the train's speed and to be prepared to stop. On seeing a red signal, the Conrail engineer immediately applied the train's brakes. This train had stopped about 350 ft (169 m) beyond the red signal, with the rear of the last unit standing on the switch, when it was struck by the Amtrak locomotive, whose brakes had dropped its speed of 128 mph (206 km/h), which was 23 mph (37 km/h) over its restricted speed of 105 mph (169 km/h), to about 105 mph (169 km/h) before it crashed into the rear of the Conrail train.

The report from the National Transportation Safety Board (NTSB) on the accident [*1*] stated

> The National Transportation Board (NTSB) determined that the probable cause of this accident was the failure, as a result of impairment from marijuana of the engineer of the Conrail train to stop his train in compliance with home signal 1N before it fouled track 2 at Gunpow, and the failure of the Federal Railroad Administration (FRA) and Amtrak to require Conrail to use automatic safety backup devices on all trains on the Northeast Corridor.

Other quotations which follow were also taken from the NTSB report [*1*].

> Contributing to the accident were (1) the failure of the brakeman of the Conrail train to observe signal aspects and to alert the engineer when they became restrictive; (2) the failure of the crew members of the Conrail train to make the automatic cab signals (ACS) test; (3) the muting of the ACS alerter whistle on the lead unit of the Conrail train; and (4) the inadequacies of the FRA oversight of Amtrak's and Conrail's supervision of corridor trains.
>
> Operation of the Amtrak train at 125 mph, rather than its restricted speed of 105 mph, contributed to the severity of the accident.

It also put the Amtrak train near enough to the Conrail train for it to be unable to stop in time.

What evidence did the NTSB have to conclude that the engineer may or may not have been impaired from the use of marijuana (and alcohol)?

> The time lapse between the accident and the collection of blood and urine samples from the Conrail engineer and brakeman precluded precise interpretation of the test results. . . . Furthermore, since CAMI (Civil Aeromedical Institute) had exhausted most of the Conrail engineer's blood specimen in the test procedure, it was not possible for CHT (Center for Human Toxicology, University of Utah) to accurately determine the level of psychoactive cannabinoids (delta-9-tetrahydrocannabinol) or (THC) that may have been present in his blood. Nonetheless, the Safety Board strongly believes that the test results provided by CHT were sufficient, along with known research findings, to permit analysis of the toxicological factors in the accident.

The first set of serum and urine specimens from the Conrail engineer and brakeman were submitted to CAMI by air express the night of the crash. Unused portions of these specimens were submitted to CHT on 16 Jan. 1987. On 3 April 1987 a "second set" of specimens was submitted to CHT. The reports of the results from the examination of the second set of specimens were dated 18 Aug. 1987.

The evidence of impairment was based on the findings of no delta-9-tetrahydrocannabinol (THC) but 52 ng/mL of THC carboxy acid (THC-A) in the second set of the engineer's blood specimens, which had been collected 5 h after the crash. The concentration of 52 ng/mL of THC-A led to the speculation that the THC concentration was in the range of 1 to 10 ng/mL, that the laboratory's lower detection limit was 3 ng/mL, and that the THC concentration would be less than 3 ng/mL but would have been considerably greater at the time of the accident. The lower limit of detection of the method used has been claimed to be less than 1 ng/mL of THC. The 52 ng/mL of THC-A could characterize the engineer as a "heavy" or "frequent" user. If the blood concentration of THC-A was 52 ng/mL, the serum or plasma concentration should have been almost double, or about 100 ng/mL. Only one of five subjects was reported in a major study [*2*] to have a concentration exceeding 100 ng/mL, and this subject's concentration dropped below 101 ng/mL 3 h after smoking marijuana, at which time the plasma THC was 3.7 ng/mL (about 1.8 ng/mL in the blood). The same subject had a plasma THC concentration of 0.44 ng/mL four days later.

The speculation continued, referring to the results of two uncontrolled studies of cannabinoids which provided no drug concentrations but extended the reported effects of marijuana from 3 to 4 h [*3*] up to 7 [*4*] and 24 h [*5*]. It has been alleged that the 24-h "preliminary report" [*5*] of pilot impairment is being repeated with controls. The impairment was measured by the amount of the pilot's deviations and corrections from the center of the runway and glide path on landings using a flight simulator. The other study, which was not without problems, alleged that a critical tracking breakpoint decrement persisted up to 7 h after smoking marijuana. Would the above decrements have any bearing on the proper operation of a train?

The lack of THC in the engineer's blood did not prevent speculation of undefined

marijuana impairment, but in addition, the lack of alcohol in his blood was back-extrapolated 5 h to a possible blood alcohol concentration of 0.06% at the time of the accident. Apparently this convoluted thinking was the basis for speculation that the engineer "was impaired from the effects of marijuana, possibly combined with the effects of the use of alcohol the night before the accident."

Many factors were responsible for this tragedy. The close scheduling of five trains which were not on schedule and were to use two tracks at the same time. The speed of the Amtrak train of 128 mph (206 km/h), rather than its restricted speed of 105 mph (169 km/h), would require about 9000 ft (2740 m) to stop it. The Amtrak engineer, who was alone in the cab, had been cited for motor vehicle speeding 11 times between 1969 to 1984. "A tissue sample from the Amtrak engineer was sent to CAMI for testing; the test was negative for alcohol." The toxicological report also stated that the specimen was unsuitable for further analysis. It must be assumed that it is not known whether the Amtrak engineer was impaired by other drugs or whether he was impaired by some prior alcohol consumption.

> On May 26, 1987, the CAMI biochemist pleaded guilty to Federal felony charges of providing false information to the FRA. According to the FRA, the CAMI laboratory had provided falsified serum test results in some previous train accidents that occurred after the FRA test regulations were implemented early in 1986.

This revelation should invalidate the report "Summary of Post-Accident Testing Events, Feb. 10, 1986–Jan. 15, 1987" [*6*].

The Conrail train lacked an automatic train control, which would have automatically stopped the train far short of the switch. The cab signal whistle was muted with duct tape. The "deadman" foot pedal had been made inoperative. One of the four bulbs of the cab signal was missing. The console radio, which was defective, produced a transmission that was weak and broken by static when received by a Conrail unit only 50 yd (46 m) away. The portable radio transmitted properly but only over a short distance.

The inspection of the safety equipment was the responsibility of the Conrail engineer, as well as observing and responding to the external signals. The railroads are responsible for scheduling and for allowing Conrail to operate without a most vital safety system, the automatic train control. The NTSB interpretation of impairment from marijuana based on the alleged finding of 52 ng/mL of THC-A in the engineer's blood specimen is scientifically indefensible.

Subsequent to this report, a statement was given at a criminal hearing that the brakeman shared a marijuana cigarette with the engineer in the cab a few minutes before the crash.

> According to the trainmaster, he was familiar with the crew members, and they appeared to him to be normal and unimpaired. . . . He [had] received two days training in recognizing individuals under the influence of alcohol and (other) drugs.

Could it be established with any degree of scientific certainty that the Conrail engineer was impaired by marijuana (and alcohol)? What would a qualified forensic toxicological expert's opinions be concerning the following hypothetical questions? Assuming that the engineer was operating a train that was involved in a crash and his blood obtained 5 h after the crash contained 52 ng/mL THC-COOH and no alcohol—specifically, how would his performance be altered? How would the operation of the train be affected? What expert opinion would be offered if it was also assumed that the engineer and brakeman had each smoked part of a marijuana cigarette?

Would an expert give an opinion that the engineer was most probably impaired by marijuana (and alcohol)? What scientific foundation is there for such an opinion? Would there be a degree of impairment that would cause the crewman to cut off the deadman control, not notice that the ACS alerter whistle was muted, not observe that a safety

bulb was missing, operate with a defective radio, not observe external warning signals, and not react promptly to them? Any impairment would have nothing to do with scheduling or with operating a train without a most important safety device, an automatic train control system. This device, which acts completely independent of the crew, would have prevented this and many other tragedies.

Would any urine testing program have prevented this crash? The men appeared normal and unimpaired to a "trained observer," the trainmaster; therefore, there would have been no cause for testing. The results would not have been available for several days. It is probably more cost-effective and more likely to increase safety if those responsible are required to check motor vehicle driving records to discover risk takers and to install and maintain automatic and other safety equipment on trains.

References

[1] "Railroad Accident Report—Rear-End Collision of Passenger Train 94 and the Colonial Rail Corporation Freight Train ENS-121, on Northeast Corridor, Chase, MD. Jan. 4, 1987," NTSB/RAR-88-01, National Transportation Safety Board. Washington, DC, 25 Jan. 1988.

[2] Peat, M. A., McGinnis, K. M., Johnson, J. R., Galinsky, R. E., Foltz, R. L., and Jones, R. T., "The Disposition of Delta-9-Tetrahydrocannabinol, 11-Hydroxy-Delta-9-Tetrahydrocannabinol, and 11-Nor-9-Carboxylic Acid-Delta-9-Tetrahydrocannabinol in Frequent and Infrequent Marijuana Users," Insurance Institute for Highway Safety, Washington, DC, 1989.

[3] McBay, A. J., "Interpretation of Blood and Urine Cannabinoid Concentrations," Letter, *Journal of Forensic Sciences*, Vol. 33, No. 4, July 1988, pp. 875–883.

[4] Barnett, G., Licko, V., and Thompson, T., "Behavioral Pharmacokinetics of Marijuana," *Psychopharmacology*, Vol. 85, 1985, pp. 51–56.

[5] Yesavage, J. A., Leirer, V. O., Demari, M., and Hollister, L., "Carry-Over Effects of Marijuana Intoxication of Aircraft Pilot Performance: A Preliminary Report." *American Journal of Psychiatry*, Vol. 142, No. 11, Nov. 1985, pp. 1325–1329; see also Letters to the Editor, Vol. 144, No. 2, Feb. 1987, pp. 259–260.

[6] "Summary of Post-Accident Testing Events, Feb. 10, 1986–Jan. 15, 1987," USDOT FRA, U.S. Department of Transportation, Washington, DC.

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