Journal of Chromatography, 449 (1988) 337–340
Elsevier Science Publishers B.V., Amsterdam — Printed in The Netherlands

CHROM. 20 763

Note

Validity of *post mortem* chest cavity blood ethanol determinations

ROBERT D. BUDD
Los Angeles County Medical Examiner-Coroner, 1104 N. Mission Road, Los Angeles, CA 90033 (U.S.A.)
(Received June 28th, 1988)

In many *post mortem* cases it is medically and/or legally important to determine whether a decedent was under the influence of ethanol at the time of his/her death. Blood specimens taken from the heart or a major artery are the specimens of choice for these laboratory ethanol concentration determinations. Occassionally, due to the massiveness and/or the nature of the decedent's injuries, blood for such an alcohol determination is unavailable or available in insufficient quantity from the heart or femoral or corotid arteries. In some of these cases the autopsy surgeon may scoop blood from the chest area for use by the toxicology laboratory to determine the concentration of ethanol present. There is no question that this scooped chest blood is a non-recommended source[1–3]. There is a high potential for direct contamination of this blood with ethanol from the stomach contents if the stomach and diaphragm are perforated and, with the passage of time and even if the stomach and diaphragm are not damaged, contamination may occur via *post mortem* diffusion of ethanol from the stomach contents[2]. Both of these factors, if operating to any extent, will yield falsely elevated ethanol determinations.

In such cases where blood cannot be obtained in sufficient quantity in any other way and, out of necessity then, scooped chest blood is used for the ethanol determination, the question arises both practically and legally as to whether this scooped chest, thorax or pleural blood is a valid specimen. The pathologist can take several steps to enhance the validity of the chest blood specimen. First, the possibility of direct contamination by ethanol from the stomach contents can be noted, *i.e.* were the stomach wall or diaphragm perforated. Second, *post mortem* diffusion can be minimized by a prompt autopsy. When the pathologist has done all that can be to verify that the chest blood has been minimally contaminated and it is deemed by him/her to be a valid specimen, the question arises as to how the ethanol concentration determined correlates with that of heart or arterial blood.

Over the years the ethanol concentrations in many body fluids and tissues have been determined and correlated with blood ethanol concentrations from the same individuals[4–11]. Now, to examine the validity of scooped thorax, chest or pleural blood specimens, all Los Angeles County Coroner's cases over the last eight years where both chest blood and heart or arterial bloods were obtained and submitted to the laboratory for ethanol determinations were studied. The results are correlated, summarized, and discussed.

0021-9673/88/$03.50

EXPERIMENTAL

Samples

The blood specimens analyzed in this study were from persons who died in Los Angeles County between February 1, 1980 and February 9, 1988 and were from cases where a blood specimen was obtained both from the heart as well as from the thorax, chest cavity or pleural cavity.

Reagents

The reagents used were 10% aqueous sodium tungstate solution and an acidic internal standard solution consisting of 0.667 *N* sulfuric acid containing 2 ml/l of *tert.*-butanol.

Blood analysis

To a 12-ml centrifuge tube 1 ml of blood, 1 ml of the 10% sodium tungstate solution, and 1 ml of the internal standard solution were added. The mixture was shaken for 10 s, then the tube was centrifuged at 6000 g for 10 min.

Gas chromatography

A 3-μl volume of the supernatant was injected into the gas chromatograph: a Hewlett-Packard Model 5750 instrument with a 6-ft. $\times$ 0.85 in. I.D. metal column packed with Porapak Q, operated at 191°C. The alcohol concentration was calculated from the peak heights through the use of reference standards extracted similarly to the blood specimens.

RESULTS AND DISCUSSION

Although blood from the heart and from the chest cavity, thorax or pleural cavity are rarely taken simultaneously from a decedent here at the Los Angeles County Medical Examiner-Coroner's Office, about 25 cases were found over the last eight years. Of these cases, fifteen showed at least one blood specimen positive for the presence of ethanol (concentration $>$ 0.019%, w/v).

These fifteen positive cases can be divided into two categories. Eleven cases appeared to be uncompromised (Table I). There was no evidence of stomach or diaphragm perforation; *post mortem* diffusion was minimized by performing the autopsy expediently (within three days) and by keeping the body refrigerated prior to the autopsy; and neither blood specimen was deemed suspect in any way by the pathologist or analyst. These eleven pairs of results agree quite well, have an average ratio of 1.02 (very near unity), have a correlation coefficient of 0.96, and confirm the results found by Jones and Pounder[12] that blood ethanol concentrations are generally independent of the part of the body from which they are taken. The standard deviation of the eleven ratios was 0.20 indicating that when the chest, thorax or pleural blood is not compromised, the ethanol concentration determined can be used to give a good estimate of the actual heart blood ethanol concentration.

Four cases, however appeared to be significantly compromised (Table II). In the first case, the stomach and diaphragm were both perforated, leading to apparent contamination of the chest blood with ethanol from the stomach contents and an

TABLE I

HEART–CHEST ETHANOL CONCENTRATIONS (UNCOMPROMISED)

Case	*Heart blood (9%) (H)*	*Chest/thorax/pleural blood (9%) (CTP)*	*Ratio (CTP/H)*
1	0.22	0.19	0.86
2	0.04	0.03	0.75
3	0.13	0.14	1.08
4	0.04	0.05	1.25
5	0.12	0.13	1.08
6	0.20	0.15	0.75
7	0.05*	0.07	1.40
8	0.11	0.13	1.12
9	0.26	0.27	1.04
10	0.08	0.08	1.00
11	0.10**	0.09	0.90
Average			1.02

* Spleen blood result.
** Calculated from bile result (divided by 1.4, ref. 4).

elevated ethanol concentration. In the second case, the pathologist deemed the heart blood specimen to be suspect and, therefore, to have a falsely lowered ethanol concentration. In the third case, abdominal cavity blood, which is very readily subjected to *post mortem* ethanol diffusion by its proximity to the stomach, had a higher ethanol level despite a prompt autopsy and efforts to keep the body cold prior to the autopsy. In a fourth case, decomposition affected the chest cavity blood, causing the creation of a low concentration of ethanol. In short, none of these four compromised cases yielded accurate or consistent results.

The results of this study indicate that chest cavity blood can be a valid specimen for the determination of the decedent's blood ethanol concentration, quite representative of heart blood (Table I), when precautions are taken to minimize *post mortem* diffusion and when there was no perforation of the stomach walls or the diaphragm. The results do, however, show (Table II) that problems may occur leading to

TABLE II

HEART–CHEST BLOOD ETHANOL CONCENTRATIONS (COMPROMISED)

Case	*Heart blood (9%)*	*Chest/thorax/pleural blood (9%)*
1	0.09	0.21*
2	0.24**	0.32
3	0.13	0.18***
4	0.00	0.03§

* Stomach wall and diaphragm perforated.
** Heart blood declared suspect and unsuitable for testing by pathologist.
*** Abdominal blood used.
§ Decomposed.

inaccurate results. In ethanol determination, if there is any question about compromise of a chest cavity, thorax or pleural cavity blood specimen, this blood should not be used or at least should be corroborated by the determination of ethanol concentrations in other body fluids and tissues.

REFERENCES

1 S. Kaye, *Am. Soc. Clin. Path.*, 74 (1980) 743–746.
2 V. D. Plueckhahn, *Med. J. Aust.*, 2 (1967) 118–124.
3 *Ethanol Determination Procedure Manual*, Los Angeles County Medical Examiner-Coroner, Los Angeles, CA, April 1988.
4 R. D. Budd, *J. Chromatogr.*, 252 (1982) 315–318.
5 P. Neil, A. J. Mills and V. M. Probhakaran, *Can. Soc. Forensic Sci. J.*, 18 (1985) 97–104.
6 A. Kraut and C. A. Purchase, *Can. Soc. Forensic Sci. J.*, 17 (1984) 91–97.
7 B. E. Stone and P. A. Rooney, *J. Anal. Toxicol.*, 8 (1984) 95–96.
8 R. C. Backer, R. V. Pisano and I. M. Sopher, *J. Forensic Sci.*, 25 (1980) 327–331.
9 R. D. Budd, *J. Chromatogr.*, 259 (1983) 353–355.
10 G. Christopoulos, E. R. Kirch and J. E. Gearien, *J. Chromatogr.*, 87 (1973) 455–472.
11 R. W. Prouty and W. H. Anderson, *J. Anal. Toxicol.*, 11 (1987) 191–197.
12 G. R. Jones and D. J. Pounder, *J. Anal. Toxicol.*, 11 (1987) 186–190.