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Alcohol Levels in Intracranial Blood Clots

In 1967 a routine alcohol determination was performed on the brain and blood of a 43-year-old male (HVB) who had been found unconscious, lying at the foot of a stairway in his home. He had sustained a fracture of the skull and survived nine hours in the hospital.

Autopsy revealed fractures of the skull involving the right temporal bone with extension into the right middle fossa and across to the sella turcica. Another fracture line extended anteriorly to the right optic foramen. There were extensive subdural and subarachnoid hemorrhages with clots in the subdural space and the brain was lacerated.

The entire brain and accompanying hemorrhage and clots were placed in a container for toxicological analysis. A specimen of heart blood, liver, kidneys, stomach, and contents were also submitted. As was the custom in our laboratory, 500 g of brain were used for the initial steam distillation. No attempt was made to separate brain tissue from the accompanying blood and clots. Ethanol was found in the screening tests and a 100-g portion analyzed quantitatively resulted in the presence of 0.15% weight/weight (W/W) ethanol, while tests on the specimen of heart blood revealed only a faint trace. This apparent discrepancy made us repeat the analysis, with the same results.

On reviewing the entire case, it occurred to one of us (AWF) that the fact that the deceased had lived nine hours in the hospital after sustaining the fractured skull could be significant. If hemorrhage into the subarachnoid and subdural spaces occurred at the time of the fall, such blood would be separated from the circulation and not subject to elimination of the alcohol through oxidation and excretion. In essence, the subject had provided us with a specimen of blood and clots containing the amount of alcohol at the time of the injury. The alcohol in the heart blood continued to undergo the gradual processes of elimination during the nine hours of survival. To put this theory to a test, we returned to the bucket containing what was left of the brain and separated (as best we could) brain tissue from blood clot. This resulted in the presence of 0.13% (W/W) alcohol in the brain, while the combined brain tissue and blood clot had contained 0.15% (W/W) alcohol.

Although not entirely conclusive, this finding seemed to explain the wide difference in the amounts recovered from the heart blood and the brain. We alerted our pathologists that in the event another case occurred in which a head injury caused death and the victim survived a number of hours, all intracranial clots should be separated from brain tissue and we would test them individually.

This particular set of circumstances does not occur frequently. Most severe head injury victims rarely survive more than one hour. In our office, where 1200 autopsies are

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performed annually, the next opportunity to put the above theory to a test did not come until 17 months later.

On 1 December 1968, an autopsy was performed on an adult male (GH—exact age unknown) who had died at the county hospital at 4:20 a.m. that day. He had been found in coma in the basement of a “senior citizens’ home” and taken to a local hospital at 5:25 p.m. on 30 November 1968. He was subsequently transferred to the county hospital, where more adequate neurosurgical facilities were available. He did not respond to medical emergency measures and died at least 11 hours after the injury had occurred.

Analysis of the heart blood showed 0.02% weight/volume (W/V) alcohol, the clot separated from brain showed 0.09% (W/W) alcohol, and the brain tissue showed 0.13% (W/W) alcohol. The brain, at autopsy, showed multiple focal areas of hemorrhage in the gray and white matters of the temporoparietal lobes, bilaterally, and some softening of the brain tissue.

The relatively small amount of alcohol in the heart blood, compared to that in the intracranial clot, appeared to corroborate our theory. However, the larger quantity found in the brain led us into further study of the metabolism of alcohol in brain tissue after severe head injury.

The third case occurred on 29 May 1971. A 43-year-old male (RJW), with a history of drinking heavily for the past 10 years, had been on a “spree” for three weeks. He was found “intoxicated” on the sidewalk. His sister-in-law was called, he was put in her car, and she drove him home. On arrival, he was snoring in the back seat so she left him there. Some time later the sister-in-law checked and found him apparently dead. He was taken to a local hospital where he was pronounced dead on arrival.

Autopsy revealed extensive fractures of the skull. An epidural hematoma 8.0 cm in diameter and 4.5 cm thick was found in the left temporoparietal area.

Toxicological analysis showed alcohol 0.05% (W/W) in the brain and 0.18% (W/W) in the epidural clot. There was no alcohol in the heart blood and none in the stomach washings. We were unable to determine the interval between the occurrence of the injury and the time of death.

The fourth case (AC) was autopsied on 22 November 1971. He was a 46-year-old male who had attended a reunion the day before. He allegedly became drunk and nasty and the manager was asked to have him removed. It was later learned that he had fallen outside and had been brought to a local hospital in coma. He was admitted at 5:30 a.m. on 21 November 1971 and died at 6:37 p.m. the same day, having lived slightly more than 13 hours.

Autopsy revealed extensive fractures of the skull and a subdural hematoma measuring 8.0 cm in length, 5.0 cm in width, and 1.5 cm in thickness.

Toxicological analysis revealed 0.09% (W/W) alcohol in the brain, 0.08% (W/W) alcohol in the clot separated from the brain, and no alcohol in the peripheral blood or in the stomach contents.

Discussion

The first case (16 July 1967) was our first indication that intracranial blood clots in victims who survive a number of hours after injury could be used to ascertain the state of intoxication at the time of accident. Subsequently, three additional cases occurred in our laboratory to corroborate this finding. In a recent publication [1] the authors state, “We do not claim priority for the concept of using sequestered blood specimens for chemical studies. It is one of the ‘tricks of the trade’ in forensic pathology which is not widely

utilized or appreciated by hospital pathologists." We could find no other reference in the literature on this subject.

During the performance of our analyses we were impressed with the apparent retention of alcohol in brain tissue for hours after severe head injury. This will be the subject of a subsequent report.

Summary and Conclusions

1. Four cases (Table 1) are presented in which severe head injury occurred and the subjects survived a number of hours prior to death.

2. The alcohol content of the intracranial clot, when present, may be an indication of the state of intoxication at the time of the accident.

3. In such cases, blood alcohol in itself will contribute little to an understanding of the facts surrounding the injury.

TABLE 1—*Summary of alcohol content in heart blood, intracranial clot, and brain with duration of survival between injury and death.*

| Case | Date | Survival Time, h | Alcohol Found, % | | | |
|----------------|----------|------------------|------------------------|--------------------------|-------------------|------------------|
| | | | Peripheral Blood (W/V) | Intra-cranial Clot (W/W) | Brain (W/W) | Stomach Contents |
| HVB 67-1374 | 7-16-67 | 9 | faint trace | 0.15 ^a | 0.13 ^a | ... |
| GH 68-2215 | 12-1-68 | at least 11 | 0.02 | 0.09 | 0.13 | ... |
| RJW 71-1089 | 5-29-71 | unknown | none | 0.18 | 0.05 | ... |
| AC 71-2355 | 11-20-71 | 13 | none | 0.08 | 0.09 | none |

^aClot plus brain tissue showed 0.15% alcohol and brain tissue alone showed 0.13%.

Reference

[1] Hirsch, C. S. and Adelson, L., *American Journal of Clinical Pathology*, Vol. 59, 1973, pp. 429-433.

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