

Postmortem Hemorrhage

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A recent case I was asked to review involved a victim who had been shot multiple times. In the first two trials the jury was unable to reach a verdict whether the shooting of the victim was self defense or murder. The prosecution decided to try the shooter a third time. I was subsequently asked to review some of the trial testimony, police reports, autopsy report and the scene and autopsy photographs.

In the autopsy the Forensic Pathologist states there was 750 cc of blood in the right pleural cavity; 450 cc in the left pleural cavity; 30 cc in the pericardial sac; and 100 cc in the abdominal cavity. At the time of the trials, the Prosecutor inquired of the Forensic Pathologist, the significance of the blood in the respective cavities and pericardial sac. To that inquiry the Forensic Pathologist testified as follows: **“For there to be bleeding the heart must still be pumping.”** The question that needs to be asked is this a true statement. The answer to that question is ‘No’, it is not a true statement, for it discounts the phenomenon of **postmortem hemorrhage (postmortem bleeding)**.

The victim had been shot multiple times with some of the gunshot wounds involving the right and left lung, heart, ascending aorta, arch of the aorta, descending aorta, pulmonary artery, liver, pancreas, spleen, left kidney, and intestines. Thus, there was a clear source for the accumulation of blood in the right and left pleural cavities, pericardial sac and abdominal cavity. However, he had also been shot in the head with involvement of his brainstem, the point of entrance being the oral cavity. There was no blood identified in his stomach, larynx or bronchial tree. There was also minimal evidence of intracranial hemorrhage and no evidence of significant brain swelling, which suggested the the victim died very quickly. That being said, then how do you account for the blood in the pleural cavities, pericardial sac and abdominal cavity. The blood can be accounted for by **postmortem bleeding or hemorrhage**.

In evaluating intrathoracic hemorrhage any injury to the chest wall, lungs, heart, ascending aorta, arch of the aorta, and the thoracic aorta (descending aorta) is referred to as **antimortem bleeding** if the bleeding occurs before death. Other potential causes of significant intrathoracic hemorrhage are tearing or perforation of the intercostal and, less often, the mammary arteries, as well as the branches of the arch of the aorta, such as the brachiocephalic, left common carotid and left subclavian arteries. Several thousand ml of blood and or clot may accumulate in the left and or right pleural cavities. What must be remembered, whatever the source of the antimortem bleeding,

postmortem bleeding can add substantially to the total volume found in the chest at the time of autopsy. **Due to the great variability of postmortem coagulation, and subsequent lysis, much of the blood found at autopsy may not have been there at the moment of death. Thus, it is considered unwise to dogmatically state the amount of blood lost before death or to make a statement, “For there to be bleeding the heart must still be pumping.”**

Postmortem bleeding from any antemortem (before) death injury may continue for a considerable time. Even though the heart ceases to function and thus, is no longer pumping blood throughout the vascular system, blood may continue to flow from injured vessels and traumatized tissues.

There are a number of fundamental points that need to be considered. When large blood vessels have been injured and hemorrhage is absent, that does not necessarily mean that the wound was postmortem in origin. It could mean that shock or a brainstem injury was a major factor in causing death. On the other hand, a great amount of blood in either the pleural or abdominal cavities does not always indicate that all blood was antemortem in origin: some wounds could continue to bleed postmortem, especially if they were situated in a dependent part of the body, under the influence of gravity or the body had been moved. In such cases, the quantity of the blood lost could be considerable. Also, the amount of postmortem bleeding depends on the fact that in most cases, the blood is liquid in the postmortem period in many parts of the body. Thus, the greater the quantity of blood that is liquid, will in turn lead to a greater quantity of postmortem accumulation. Additionally, the quantity of blood lost in the postmortem period is very much dependent on the size of the injured vessels. For example, there is the potential for a greater quantity of postmortem bleeding from an injured thoracic aorta versus a small peripheral lung vessel. In an article published in the *Am J Forensic Med.* 2004 Mar; 25(1):20-2, written by Nikolic S *et al.*, they showed the amount of postmortem bleeding following postmortem cutting of the thoracic aorta ranged from 100 to 1300 cc, with an average of 620 cc.

As pointed out above, a major factor determining the quantity of liquid blood in the postmortem period is the state of postmortem coagulation. This in turn is determined by two factors, whether the person's death was rapid or slow and the rate of decline of the pH of the person's blood following death.

In those individuals whose death is rapid their fibrinolytic activity level typically is high, consequently, the blood is more liquid. However, in cases in which the person dies slowly, the fibrinolytic activity is usually low, and thus, the blood will show more clots. The fibrinolytic activity level is in a large part determined by the rate of fall of the pH of the blood following death.

In the antemortem period blood pH is regulated to stay within the narrow range of 7.35 to 7.45. If the blood pH is alkaline (greater than 7.45) or acidic (less than 7) this can lead to death so blood is one of the most regulated systems in the body. Blood pH is regulated by acid-base buffers such as carbonic acid and bicarbonate ion, which exert

their influence principally through the respiratory system and the kidneys to control the acid-base balance. In death, the body buffering system is not maintained and thus, blood pH changes can occur.

In one study involving the death of 11 individuals, the pH change in the first 20 hours following death was 7.0 to 5.5.

The lower pH is a reflection of the accumulation of acidic metabolites with the major ones being lactic acid and formate (methanoic acid), as well as the accumulation of NADH. Lactic acid is produced by lactate dehydrogenase from pyruvate by way of anaerobic glycolysis in skeletal muscle, liver and red blood cells when insufficient oxygen is available for pyruvate to enter the citric acid cycle. This process occurs naturally in muscle tissue during exercise and in normal metabolism inside red blood cells. The normal serum lactate concentration is 0.5-2.2 mmol/L. Circulating lactate is normally oxidized to pyruvate through the actions of lactate dehydrogenase after being taken up by monocarboxylate transport proteins that are differentially expressed in actively respiring cells and tissues. In a recent study of postmortem levels of lactate, lactate in human heart blood increased by 20-fold by one hour after death and 50-70 fold by 24 hours.

Formate (methanoic acid) is the simplest carboxylic acid. Formate is the toxic metabolite of both methanol and formaldehyde and is normally present in mammals in low concentrations (0.12-0.28 mmol/L) following the catabolism of several amino acids including serine, glycine, histidine, and tryptophan, as well as catabolism of methanol from external sources, such as would occur in the alcoholic who has no access to ethanol. In humans, formate is rapidly oxidized to carbon dioxide by the liver to prevent toxic effects in the brain and eyes due to an inhibition of the cytochrome oxidase, a protein complex in the respiratory chain in mitochondria. In one study, the concentrations of formate were elevated in the blood of putrefied (5-44 days) bodies to as much as 5 mmol/L compared to non-putrefied (1-14 days) bodies in which its average level was 0.86 mmol/L. This increase in formate is believed to be due to bacterial action on of lipids and proteins in decomposing bodies.

Nicotinamide adenine dinucleotide (NAD⁺) and its reduced form NADH are substrates that have central roles in cellular metabolism and energy production as hydride-accepting and hydride-donating coenzymes in the citric acid cycle. During hypoxic conditions NAD⁺ cannot be regenerated from NADH, which accumulates due to anaerobic glycolysis and peroxisomal catabolism of fatty acids.

Summary

The most immediate biochemical change, which occurs after death, is a fall in the concentration of oxygen due to the absence of circulation. This in turn results in a switch from the citric acid cycle to anaerobic metabolism. Anaerobic glycolysis in turn results in the accumulation of lactic acid and an increase in the concentration of NADH. Formic acid also increases but requires decomposition to set in. This is because

normally the production of NADH requires enzymes that function at physiological pH as well as NAD⁺ as a co-factor, both of which are not in evidence in the postmortem period. Thus, the increase in formic acid is most probably due to bacterial action as part of the decomposition process in putrefaction. Thus, how much formic acid is formed is determined by the rate of decomposition.

The accumulation of lactic acid and formic acid initiates the fall in pH in the postmortem period. Additionally, due to circulatory stasis, which occurs immediately after death, the blood buffer systems fail. This in turn causes a rapid pH decline as more acidic metabolites are produced. The speed of the plasma pH decrease is quite rapid, which in one study showed a decline in pH from 7.45 to 6.0 within the first 24 hours after death. As discussed above, another study involving the death of 11 individuals, the pH change in the first 20 hours following death was 7.0 to 5.5. The fall in plasma pH is significant because it is thought to activate fibrinolytic enzymes, which in turn prevent the blood from clotting, resulting in an increase in fluidity of the blood.

It is this activation of fibrinolytic enzymes due to the fall in blood pH, which is primarily responsible for the lack of blood clotting following death. The rapidity in which fibrinolysis process takes place in turn determines the severity of postmortem bleeding. Since there are a number of variables, which affect the genesis of the fibrinolytic process, it behooves the Forensic Pathologist to be cautious in their determination of how much bleeding took place before death and how much occurred after death. Lastly, the statement, "For there to be bleeding the heart must still be pumping," as a foundation for the quantity of blood in the various body cavities at autopsy, is simply not true, for it discounts **postmortem bleeding**.