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Aging of Bitemarks: A Literature Review*

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ABSTRACT: The aging of bitemarks through the subjective interpretation of visual signs has created an area of controversy in the forensic science community. The healing dynamics of these bite wounds has been insufficiently studied and is poorly understood. A review of the literature related to bitemark aging was undertaken in an effort to summarize the currently published information on this misunderstood subject.

KEYWORDS: forensic science, bitemarks, aging, wound healing, bruising, forensic odontology

A recently published review of appellate court decisions related to bitemark analysis has brought into question some of the peripheral or secondary opinions being offered by forensic odontologist's testifying as expert witnesses in these cases (1). Numerous paper presentations, and subsequent discussions, related to bitemark analysis in the Forensic Odontology Section at the Annual Meeting of the American Academy of Forensic Sciences also fed the fires of this controversy. The basis for some of these opinions are said to be unclear in dispute or beyond the expert's area of expertise. One such area concerns the aging of bitemarks. Experts have rendered opinions related to the age of marks based upon visual inspection alone, which is a subjective interpretation of a poorly understood phenomena, wound healing. Yet, some claim the aging of a bitemark to be a logical extension of the forensic odontologist's area of expertise, indeed a required component of any bitemark analysis.

In 1973 the scientific literature was replete with case reports and studies related to bitemarks. However, Harvey bemoaned the fact that the external physical appearance of bitemarks changed with time, and the causative factors precipitating these changes were largely unknown (2). Subsequently, Harvey and his colleagues presented preliminary results relating microscopic findings to the clinical appearance of bitemarks.

A significant number of years has passed since Harvey's attempt to steer the forensic odontology community into an age of scientific discovery and understanding of the vagaries of bitemarks. Unfortunately, little additional research has been done to resolve many of the questions that continue to be raised in the forensic community about bitemarks, especially the reliability of the visual aging of bitemarks, and what are the causative factors related to seemingly illogical developments observed within the pattern injury of a bitemark. Over the years the majority of research related to bitemarks has revolved around bitemark analysis methodologies, comparison techniques, and evidence collection techniques. Additionally, a tremendous amount of time, energy, talent, and financial resources has been spent "reinventing the wheel", that is, continually refining techniques that are already tried-and-true. The reason for this is unclear. All the while, the more fundamental areas for potential research have been largely ignored. As a response to the ongoing debate concerning the aging of bitemarks, a review of the literature was conducted with the hope of distilling the facts related to this controversial subject.

Bite Wounds

Humans are known to have used their dentition as both tools and weapons since the dawn of time. To find historical documentation discussing evidence from the criminal use of teeth (i.e., bitemark evidence), one must move closer to modern civilization, along the time continuum of mankind. Pierce's presentation of the Reverend George Burrough's trial from 1692, seems to be the earliest recorded use of human bitemarks in a legal proceeding (3). The first known publication of a scientific paper discussing a human bite is reported by Harvey to have been a case report of "bite infection" by Skrzeckes in 1874 (4).

Simply stated, a bitemark is a "patterned injury" produced by teeth. This mark may be produced on inanimate objects such as food stuffs, or on the skin of other living creatures. The mark may contain one or more features caused by one tooth or multiple teeth. These features may be described as indentations, abrasions, or discolored areas commonly referred to as bruises or contusions. Skin is an organ of the human body capable of repairing/healing itself when injured. The repair process leaves tell-tale signs at both the macroscopic (visual) and microscopic (histologic, histochemical, and biochemical) levels. In relationship to the life of the organism, this repair process is fleeting. As a result of this rapid loss of evidence, in the case of a bite injury, its collection is important should the causative event be criminal in nature. Because the collection of visual evidence is non-invasive, relatively simple to execute, and easily converted to excellent court exemplars, it is not difficult to comprehend the significant effort devoted to this aspect of bitemark evidence collection. However, what an "expert" does with, and concludes from, this data then becomes the basis for an opinion that could possibly be offered in court. On occasion the weight of the evidence for or against a defendant in court may hinge upon an opinion related to the age of a bitemark, the age relationship of one mark to another, or the age relationship of a mark to the time of assault/death of the victim of the crime. It is the process of aging the mark and the validity of any such conclusions that warrants careful scrutiny.

*The opinions expressed are those of the authors and do not necessarily represent those of the U.S. Army or the Department of Defense.

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The Visual Aging of Bruises

If the injury produced by a bite is sufficient, and healing is allowed to proceed, a diffuse discolored area, or bruise, may be the result with time. During the process of healing, the bruise will go through several definitive color changes before fading from perception by the human eye. Dorland's Medical Dictionary defines a bruise as "a superficial injury produced without laceration"(5). Commonly, in the definition of bruise or contusion, there is no discussion of coloration. DiMaio and DiMaio describe this entity as "an area of hemorrhage into soft tissue due to the rupture of blood vessels caused by blunt trauma" (6).

The use of the appearance of bruises for the purpose of aging a wound has been declared invalid by numerous investigators during this century. Just as frequently however, expert witnesses continue to resurrect the technique and present their observations as valid "expert opinions" concerning the age of bruises. As early as 1939, Spillsbury invalidated the use of appearance as a means of aging bruises (7). In 1957 Robertson and Mansfield declared unreliable the previously published criteria being applied to distinguish between antemortem and postmortem bruises (8). In 1993, DiMaio and DiMaio advised caution in the visual aging of bruises through the subjective interpretations of their color (6).

Although there is little agreement concerning the aging of bruises by appearance, that has not prevented guidelines and commentaries concerning the color changes observed in healing bruises of human skin from being published in the literature. Some of these guidelines are compared in Table 1.

A review of the literature found numerous animal studies related to bruising in poultry, cattle, and other commercially raised species, but only one scientific publication related to the color changes in the healing bruises of humans. In an excellent study, Langlois and Gresham (10) recorded the following conclusions:

1. The colors of bruises are dynamic, as colors which are present one day may disappear the next, only to return at a later time.
2. In the same subject with two separate bruises on the same part of the anatomy, identical etiology, and identical age, they may display different coloration and that coloration may change at different times.
3. Not all bruises produce a yellow coloration prior to resolution.
4. Color of the victims skin, or natural pigmentation, is important. That is, if the victims skin has a yellowish coloration one may not perceive a yellowish color within the bruise. Additionally, if the victim is very dark skinned bruising might not be seen at all.
5. If a bruise has a yellow color it is "very likely" to be greater than 18 hours old. The converse is not true that if a bruise has no yellow it is likely less than 18 hours old.

TABLE 1—*Comparative findings of color changes in bruises*.*

Time	Camps	Glaister	Polson and Gee	Smith and Fiddes
Immediate (0–24 H)	R	V	R	R
Soner After (24–48 H)	P/B			P/B
Day 3		B		
Days 4–5	G			G
Days 5–7		G	G	
Days 7–10	Y		G	Y
Days 8–10		Y		
Days 13–18		RES		
Days 14–15	RES		Y	RES
Up to day 30			RES	

NOTE—R = red; V = violet; P/B = purple/black; B = blue; G = green; Y = yellow; RES = resolution.
*After Bowers (9) & Langlois and Gresham (10).

Based on these findings, Langlois and Gresham concluded that "it would seem unlikely that a bruise could be reliably aged from appearance alone."

All authors tend to agree that the unpredictability in the appearance of bruises is due to a great number of variables affecting the complex biologic events occurring within the injury. Below is a list of the published variables affecting the appearance of bruises:

1. The structure and vascularity of the tissue injured. For example, bruising in the loose and highly vascular tissues around the eyes is much more pronounced than skin in areas such as the palm of the hand or the soles of the feet.
2. Vascular tissue over bone bruises more.
3. Children and the elderly bruise more "easily" than other generations, due to loose delicate skin in the former and loss of subcutaneous supportive tissue in the latter.
4. Metabolic rate.
5. Women bruise more easily than men, especially obese women.
6. The victims state of health can have an effect. Hypertension, coagulation disorders, and liver dysfunction may alter the extent of bruising.
7. Medications such as aspirin can increase bleeding. Steroids can alter the dispersion rate of bruising. It has been speculated that the stress undergone by the victim may result in an endogenous release of corticosteroids, thereby delaying the healing of a bruise (11).
8. The normal color of the skin (i.e., pigmentation) may affect the observation, or missed observation of a bruise.
9. Mass and velocity of the impact may have an influence on the depth and surface of injury, as well as the rate of healing.
10. Deep subcutaneous injury can prolong bleeding time.
11. Previous bruising at the same site may affect subsequent bruising by increasing the rate of resolution.
12. Body temperature may effect the amount of bruising. The temperature is different on an appendage versus that over the core.
13. A bruise may appear at the instant of injury, or take as long as 48 hours to appear. The time of appearance is related to the time required for the extravasated blood the reach the surface. This lag time will allow antemortem bruises to appear postmortem. However, the hematoma may never become visible and only a tissue specimen would reveal its presence.
14. Rapidity of death after injury may affect the presence or absence of bruising.
15. Bruising may occur at a site different than that of the initial injury due to the movement of extravasated blood through the tissue layers.
16. Bruises appearing as antemortem can be created postmortem.
17. Environmental conditions may affect bruising.

18. Interpretation of color within the bruise is subjective, and the observer's ability to discriminate color may be impaired (e.g., color blindness).
19. Ambient light may affect the appearance of the bruise (i.e., the temperature of the light utilized at the time of observation).

With the information available today, and discussed above, it would seem wise for an expert, about to voice an opinion concerning the age of a bruise, to consider the advice of DiMaio and DiMaio when they conclude one would be well advised to simply qualify the age of a bruise as "recent or old" (6).

Microscopic Aging Techniques

If bruise pattern injuries cannot be reliably aged visually, are there any techniques available to the forensic expert that will enable a wound to be accurately aged? Other methods that have been investigated and utilized to age such injuries include histologic, histochemical, biochemical, and most recently immunologic studies.

Histologic Aging Techniques

Robertson and Mansfield wrote that the consistent microscopic aging of bruises is not possible (8). They concluded that the "microscopic aging of a bruise inflicted up to several hours before death may not make possible its distinction from one caused several hours after death while the body is still warm". There is no known study to dispute these findings, and one recently published forensic pathology textbook cites this paper when discussing the microscopic aging of bruises (6).

In contrast, the aging of laceration and abrasion injuries is well documented and widely accepted. This can be important in the aging of bite marks, as frequently abrasions are seen as features within such marks. Robertson and Hodge (11) have published what is considered one of the most authoritative and logical methodologies for the analysis and aging of abrasions:

Stage I—Scab Formation:

0–4 Hours: serum, red cells, and fibrin are deposited on the abrasion (these indicators are not used for aging, but do indicate survival following injury).

2–6 Hours: infiltration of polymorphonuclear (PMN) cells perivascularly.

By 8 Hours: the bed of the scab is marked by a zone of PMN cells underlying the area of epithelial injury.

By 12 Hours: there are three layers present:

1. Superficial: surface zone of fibrin and red cells (or crushed epithelium in the case of impact abrasion).
2. Middle: a zone of PMN cells.
3. Deepest: a layer of damaged abnormally staining collagen.

12–18 Hours: the third zone becomes infiltrated by PMN cells.

Stage II—Epithelial Regeneration:

Begins at surviving hair follicles and at the edges. Epithelial growth may appear as early as 30 hours in superficial "scrapes," and is clearly visible by 72 hours in most abrasions.

Stage III—Subepidermal Granulation:

This stage occurs after epidermal covering of the abrasion, and is usually prominent at 5–8 days. Perivascular infiltration and chronic inflammatory cells are prominent. From 9–12 days the overlying epithelium becomes progressively hyperplastic with keratin formation, and new collagen fibers begin to appear.

Stage IV—Regression:

At about 12 days the epithelium is remodeled and becoming thinner, even atrophic, and collagen fibers are now prominent.

Millington studied the histologic load-related changes in the staining of collagen (mentioned in Stage I above) as a result of bitemarks (12). He found that these changes remained in postmortem tissue without "appreciable diminution." Storage of the tissue for up to six months still produced favorable results. In living tissue the reaction to load had completely disappeared within 21 days. Millington, however, concluded such evidence "itself is not substantive but could be useful correlative data." Raekallio states that cellular infiltration is the most reliable histologic aging criterion, but that the lag phase, from time of injury until reliable histologic indicators are in place, is generally too long to be useful in forensic cases (13).

Enzyme Histochemical Methods

Histochemical methods can be utilized to narrow the time since injury from an 8 h window to a 1 h window. The enzyme studies performed by Raekallio (13), and their appearance from time of injury are as follows:

1. ATP-ase: as early as 1 h after injury.
2. Esterases: also from 1 h after injury.
3. Aminopeptidase: at 2 h after injury.
4. Acid Phosphatase: at 4 h after injury.
5. Alkaline Phosphatase: at 8 h after injury.

Raekallio feels the consecutive appearance of positive vital reactions, demonstrable by these various methods of enzyme histochemistry, allows one to construct a biologic time-table which may be useful as a "rough estimate" (emphasis added) of the age of the wound.

Biochemical Studies

Although enzyme histochemistry is approximately eight times more accurate than conventional histology, there is still a 1 h period for which more accurate determinations would be extremely valuable to forensic scientists. This is where biochemical studies of free histamine and serotonin (5-Hydroxytryptamine or 5-HT) are useful.

Raekallio presented the following conclusions concerning biochemical studies of wounds:

1. There is no increase in 5-HT or free histamine in postmortem wounds.
2. There is no significant degradation of 5-HT or free histamine demonstrable several days after death.
3. A distinct increase in measured levels of these two compounds (i.e., at least a two-fold increase in 5-HT and 1.5-fold increase in free histamine) indicates the antemortem origin of a wound.
4. An increase in 5-HT is a better indicator than free histamine. Most importantly, biochemical studies show measurable quantities of these two compounds within minutes of injury, and definitive results within one hour.

In a non-forensic scientific paper investigating the role prostacyclin (a vasodilator and an inhibitor of platelet aggregation) in bruising in children, it was suggested that "prostacyclin formed at the injured vessel surface collects within the first few seconds after injury inside the tissue space at the site of the bruise and, by influencing the formation of the platelet/fibrin plug and/or the leakage of blood from the vessels, plays a significant role in modifying the development of bruising" (14). Perhaps further study of these results from a forensic perspective are in order.

Immunological Studies

Occasionally, it may be necessary to distinguish an antemortem bruise from postmortem infiltration of haemoglobin, or postmortem lividity. One study used a component of the erythrocytic membranes, glycophorin A, to verify a discoloration as an antemortem bruise (15). These researchers found glycophorin A in all antemortem bruise specimens taken from 4 h to 10 days postmortem, even if the body showed severe putrefaction. No glycophorin A was found in any postmortem produced discolorations.

As with any forensic evidence, the more studies one can conduct, or tests one can run, the more reliable the conclusions. Therefore, the age determination of an injury will be more accurate if several of the methods discussed above are used independently.

Conclusion

In a recent publication, the author discusses three appellate court decisions related to the aging of bitemarks by three board certified forensic odontologists (1). In one, a Tennessee appellate court was told that a bitemark had occurred "at or about" the time of the victims death. At trial in Mississippi, another expert testified some of the bitemarks on the murder victim had been inflicted "while the victim was alive", and others, had not. Finally, an Illinois appellate court heard the forensic dentist's opinion that separate bitemarks on a murder victim were "approximately the same age and were inflicted with the same time frame." Of major concern in all three of these cases, the author continues, was an absence of commentary by the appellate court concerning how the forensic dentists arrived at their conclusions.

While the bitemarks themselves in these three cases may have offered indisputable evidence relating the accused to the victim's wounds, the controversy raised, by offering what appears to be unsubstantiated opinions regarding the age of the marks, leaves the remainder of the forensic community with a negative impression of the whole process of bitemark analysis.

As can be seen from the small number of studies either directly or indirectly related to the aging of bitemarks, forensic experts have very little scientific research to support opinions regarding the aging of bruises either visually or microscopically. Tissue specimens can be aged, but collecting the specimen eliminates further use of the mark. Harvey speculates that unless/until "it could be shown that histochemical studies were of more value than the photographic record in the description of the mark, then it is unlikely that such tests could become routine" (2). The same can undoubtedly be said for histologic and biochemical studies.

More than 20 years ago Harvey stated that "we are ignorant of the conditions during normal biting, and results of simulation studies and conclusions drawn from them must be regarded with some caution" (2). Since that statement was made, little has changed in our understanding of the complex and seemingly mysterious processes that occur within a bitemark over time, and subsequently alter that mark in the eyes of the observer. Interestingly, Harvey also found that even under carefully controlled conditions with a trained researcher attempting to apply multiple identical bites to an anesthetized "victim", no two bitemarks were alike. Harvey concluded each mark was "quite different" from the others.

In 1982 Levine wrote an excellent overview of bitemark evidence and analysis (16). Within the body of his writing there is a brief discussion concerning the resection of bite wounds for aging by the pathologist. Levine then discusses the use of good color photographs for the aging of an injury, and concludes the discussion by saying that forensic odontologist's and others "may be competent to render opinions as to the age of tissue injuries." It would seem today that the spirit of Levine's intent has been violated and some individuals may have taken this single comment as a license to become "experts" in aging bitemarks, thus overstepping the bounds of their true abilities and bringing about the controversy that rages today.

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