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EDITORIAL

Bruises: Is it a case of "the more we know, the less we understand?"

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In this issue of the journal the possibility that heme oxygenase activity is responsible for the delay in the appearance of the yellow color in bruises has been investigated [1]. Although bruises may have been a "very much neglected branch of injuries" [2] for many years, recent investigations, including the current paper [1] have revealed some unexpected and highly relevant findings. Bruises in the skin and subcutaneous tissues occur when there has been extravasation of blood following the application of blunt force. Generally the requirements for their formation are injury, compromise of the integrity of vessel walls, and blood pressure [3]. The most useful bruises in a forensic context are those that have a patterned appearance reflecting the nature of the impacting object. Questions that are commonly asked in court regarding bruises involve their age, the amount of force required for their production, and whether they occurred before or after death. Although it was once considered possible to provide answers to all of these queries, recent studies have shown that forensic assessments, particularly of the age of these injuries, may be less reliable than was previously considered.

For example, it was once generally accepted that bruises could be dated from their macroscopic appearance and standard text books gave quite precise times for this, with bruises appearing red, purple, and swollen in the first 2 days, green from 4 to 7 days, yellow from 7 to 10 days, with resolution between 14 and 30 days. Of note, even then there was disagreement among the texts in the exact timeline for these color changes [4–7]. The paper by Langlois and Gresham in 1990 debunked this certainty, and demonstrated that the only reliable comment that could be made was that yellow bruises were likely to be older than 18 h [8]. It should be noted, however, that this study did rely on photographs of bruises, which may present additional complication in interpretation (see below). The reasons for the great variation in individual bruising responses to blunt trauma are varied and include differences in the nature of the impacting object and forces, age of the victim, variations in subcutaneous tissue thickness, variability in bleeding tendencies, the effects of underlying disease states, the influence of medications (both pharmaceutical and herbal) [9], and background skin pigmentation. Other issues concern considerable inter-observer variability in the perception of yellow in bruises [10] and in the identification of specific colors [11], i.e. one observer's brown may be another observer's yellow, and the tendency for single bruises from one episode of trauma to develop different colors in different areas at the same time. All of this tells us that dating of bruises macroscopically is, at best, highly inaccurate.

Photographing bruises introduces further variables as a bruise can be made to disappear if a picture is over-exposed; in addition, an apparent "bruise" can be created if shadows occur from under-exposure. For this reason photographs should not be relied upon for the assessment of the age of bruises. Ideally bruises should be photographed in color at autopsy in suspicious cases, both before and after incision (the latter to confirm the presence of subcutaneous hemorrhage). Photography in black and white using infra-red or alternative light source illumination may provide additional information, and trace wound patterns may be detected by use of an alternative light source (although alternative light source [polilight®] illumination with digital image analysis does not assist in determining age).

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Microscopy was once regarded as a much more accurate way of dating a bruise as it was thought that neutrophils marginated within vessels between 30 min and 4 h after injury, and then infiltrated into the adjacent perivascular soft tissues between 4 and 12 h [12]. While there has been considerable variation in this proposed orderly sequence, it has been stated that the absence of neutrophils indicates a post-infliction interval of less than 15 h [13]. The demonstration of sections of bruises in three children aged 3, 11, and 27 months who had bruises of at least 24 h duration confirmed by medical evaluation (30, 44, and 79 h before death), with extravasated red blood cells but no leukocyte infiltration or other cellular reaction, however, was not in keeping with this assertion [14]. While this may be due to differences in individual responses to trauma, subsequent studies on human and animal tissues have suggested an additional alternative possibility: i.e. that the inflammatory reaction to trauma may be triggered by direct damage to tissues rather than by extravasated erythrocytes. In support of this contention, it has been noted that trauma (compression) of fat can have pro-inflammatory effects [15].

A bruise can, therefore, be thought of as a "target" with the central area having damaged tissues that leak chemotactic factors that incite an inflammatory infiltrate, compared to the less reactive peripheral areas where there are intact, relatively-inert erythrocytes. Thus, the site of sampling of the bruise for microscopy may be crucial, as tissue in areas of extravasated blood peripheral to the impact may not show the same type or intensity of inflammation that may be found in areas where direct tissue damage has occurred [16, 17].

The roles of other parameters such as immunohistochemistry, hyperspectral imaging, and magnetic resonance imaging (MRI) in helping to date bruises are yet to be established [18–21].

An issue that may arise in court when there has been lethal blunt trauma to internal organs, but no bruising to the skin, concerns the degree of force involved. It is suggested that less severe force must have been applied than has been proposed. Unfortunately this underestimates the resiliency and elasticity of skin and subcutaneous tissues that enables them to avoid injury while allowing significant forces to be transmitted to underlying soft tissues, bones, and organs. This was clearly documented by Casper in 1860 and demonstrates that bruises do not always accompany lethal blunt force injuries [22–24]. In other cases trauma may have resulted in subcutaneous hemorrhage that is not externally visible due to the limited ability of light to penetrate skin. For this reason bruises may not be suspected until the skin has been reflected [25].

A final issue that arises is the possibility of post mortem bruising. Although blood pressure is generally required to force erythrocytes from the vascular compartment into adjacent tissues it is possible that post mortem bruising can occur in congested or abnormal areas, and that decomposition (with potential pressure effects from gas combined with degradation of tissue) may be a contributing factor. It has been shown that a body in the head down position may develop tissue congestion that simulates bruises [26]; however, Saukko and Knight [12] observe that if force is involved, rather than just body position, it must be significant as the bleeding is by passive oozing rather than by active extravasation. Certainly all of the post mortem handling of bodies in forensic morgues does not cause bruising of ankles and wrists.

In conclusion, our investigations of bruises in recent years have led to less dogmatism when it comes to trying to date these injuries. Bruising of the skin may not be present despite significant internal injuries from blunt trauma, and although post mortem artefacts may simulate bruises, these are rare.

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