Interpreting bruises at necropsy

P Vanezis

Abstract
The accurate interpretation of bruising at necropsy is essential to understanding how a victim has been injured and assists the pathologist in a reliable reconstruction of the events leading to death. It is essential not only to assess the mechanism of production of a bruise, taking into account the type of impacting surface and the magnitude of force used, but also to estimate when the injury was caused. An account is given of the various methods used in the examination of bruises, particularly with respect to aging, as well as the factors that may affect their appearance. Differentiation from artefacts resulting from postmortem changes is also discussed in some detail.

Keywords: bruising; necropsy; time of death; cause of death

Factors that influence the development and appearance of bruises
There are many variables that influence the development and absorption of bruises, as well as their appearance and extent of spread, thus adding to the difficulty in their interpretation.

- Bruising occurs more easily where there is loose tissue—for example, over the eyebrow—rather than where the skin is more strongly supported. It also occurs more readily where there is an excess of subcutaneous fat. Because there is a greater skin deposition of fat in women, they tend to bruise more easily than do men.
- The type of surface and force that impacts on the body will have a great effect on the intensity, size, shape, and pattern of the resultant bruising.
- In infants and the elderly bruising tends to occur more easily. In the very young the skin is looser, more delicate, and there is an increased amount of subcutaneous fat. In old people, although there is loss of subcutaneous fat, blood vessels are also more poorly supported and bruises take longer to resolve.
- Skin colouration modifies the appearance of a bruise to the naked eye. It is much easier to observe the extent and colour of bruising in lighter skinned individuals. Thus, it is particularly important to take extra care when examining dark skinned individuals so that any bruising is not overlooked.

Acquired
- Liver disease
- Vitamin K deficiency
- Severe renal failure
- Hypothyroidism
- Disseminated intravascular coagulation from any cause
- Diseases of bone marrow
- Myeloma
- Myeloproliferative disorders
- Myelodysplasia
- Use of antithrombotic medications
- Heparin
- Warfarin
- Aspirin
- Non-steroidal anti-inflammatory drugs
- Thrombocytopenia
- In bone marrow failure
- Autoimmune
- In hypersplenism
- In human immunodeficiency virus infection
- In massive transfusion
- As a result of haemolytic uraemic syndrome
- As a result of thrombotic thrombocytopenic purpura

Congenital
- Von Willebrand’s disease
- Haemophilia

Abstract
The accurate interpretation of bruising at necropsy is essential to understanding how a victim has been injured and assists the pathologist in a reliable reconstruction of the events leading to death. It is essential not only to assess the mechanism of production of a bruise, taking into account the type of impacting surface and the magnitude of force used, but also to estimate when the injury was caused. An account is given of the various methods used in the examination of bruises, particularly with respect to aging, as well as the factors that may affect their appearance. Differentiation from artefacts resulting from postmortem changes is also discussed in some detail.

Keywords: bruising; necropsy; time of death; cause of death
Table 2  The documentation of bruising includes the following

1. Whether the discoloured area seen is in fact a bruise
2. Its causation; that is, nature of the object or surface impacting on skin
3. The force of impact
4. The age of the bruise
5. The site at which it is found and its relevance to causation and appearance
6. Congestion or vascularity of the site
7. Distribution, where there are several bruises
8. Appearance (pattern, size, colour, related injuries—for example, central abrasion)
9. Collective assessment of multiple bruises (and other types of injuries) to reconstruct events—for example, in non-accidental injury in children
10. The presence of natural disease, particularly blood dyscrasias, which may be a primary or contributory factor in its production
11. Constitutional factors of the individual
12. Skin colouration of the individual

Table 3  Assessment of bruising includes consideration of the following

1. Shape: the contour, pattern, and degree of swelling should be described as fully as possible
2. Size: this will depend on the shape of the bruise. However, it is important to give the overall dimensions in terms of at least two measurements—width and length—together with the orientation of each. The size can be compared with its size at any later examination of the injury
3. Colour: a description of the colour of the bruise in simple terms is essential
4. Site: as with any injury it is essential to describe its exact location on the body. This should include a description of the location (for example, lower aspect of left front of chest) and distance from two points of reference (for example, from the midline and below the top of the shoulder)
5. Photography: it is essential to illustrate the description of bruising with good quality photography. A measurement scale should be included within each photograph and, where attempts are made to age bruises by assessing their colour, as depicted in the photographic print, it is useful to include a colour scale
6. In certain circumstances, the use of special photographic techniques using different wavelengths outside the visible spectrum, such as ultraviolet and infrared, may enhance the appearance of a bruise

bleeding disorders (table 1), one must also consider hypertension, cardiovascular degenerative changes, and disorders of collagen and other supporting tissues, which might result in an increase in the amount of blood extravasating from blood vessels.

**Documenting and interpreting bruise characteristics**

All bruises should be documented thoroughly, taking into account their shape, size, and colour (table 2). Photography and occasionally other visual media such as video are an essential part of the documentation process and provide a permanent visual record. However, the assessment of bruising from secondary images is second best to assessment at necropsy.

The nature of the agent causing a particular bruise is central to assessing the circumstances of injury and thus assisting in the reconstruction of events. On occasions, depending on the site of injury and nature of the impacting agent, it may be possible to match a patterned bruise with an object—for example, a heel mark with a corresponding shoe. Other bruises, although not producing a “mirror image” of the offending agent will nevertheless produce characteristics that are typical of the type of impacting agent. For example, round or discoid small bruises can be caused by fingertips, and trampoline bruising can be caused by objects that have a longitudinal cylindrical surface (such as a rod or baseball bat).

A bruise should not be examined in isolation because on many occasions, particularly with show marks or blunt trauma from sticks, iron objects, belts and so on, there may be an accompanying characteristic abrasion or laceration. The size, intensity, and accompanying lacerations or abrasions will be useful pointers in assessing the force of an impact. Nevertheless, it should be appreciated that the extent of bruising will depend on several other factors other than force. These include site of injury (whether over bone or soft tissue), type of agent used, and factors intrinsic to the victim. One must not forget the effect of skin pigmentation on the appearance of bruises and thus their interpretation. It is important in dark skinned individuals to pay particular attention to the subdermal appearance and spread of a bruise.

The distribution and collective assessment of bruises, particularly in reconstructing events, is particularly pertinent to the investigation of the “battered child”. In such circumstances, where there are multiple bruises, possibly of varying ages, it is essential to assess the injuries as a whole to understand the circumstances and manner of causation, rather than to consider each injury in isolation (table 3).

**Dating bruises**

Any investigation into a death of forensic importance will need to establish when a particular trauma event occurred. However, the assessment of the timing of an injury is not a precise science and it is only possible to give an approximate time. Nevertheless, the pathologist should be aware of the various factors and techniques available that can assist him or her to arrive at a reasonable assessment of age, despite the inherent limitations.

For the clinical forensic medical examiner, the assessment of bruises in living subjects is obviously limited to their external gross appearance, including photographs and history given by the victim where available. The pathologist, on the other hand, has more options for an objective assessment, including microscopical examination and closer gross assessment by dissection. In addition, both the pathologist and his/her clinical colleague will need to corroborate or refute accounts given by eye witnesses where appropriate.

An assessment of a bruise to ascertain when it occurred can be made in several different ways, namely:

- Direct gross examination of the victim (external and by dissection).
- Conventional and special photography.
- Conventional histological examination.
- Various histochemical techniques.
- Biochemical methods.
- Objective colour assessment.

**DATING BRUISES AT THE POSTMORTEM EXAMINATION**

A large quantity of information can be obtained from gross examination of the body in the mortuary. For example, differentiating a fresh bruise from an older one is usually not difficult. It is important, however, to bear in
Table 4  Summary of colour change in bruises with time

<table>
<thead>
<tr>
<th>Source</th>
<th>0–24 h</th>
<th>1–3 days</th>
<th>4–7 days</th>
<th>1–2 weeks</th>
<th>Over 2 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Camps (1976)</td>
<td>Red, dusky/purple, black</td>
<td>Dark blue</td>
<td>Dark blue</td>
<td>Green</td>
<td>Yellow</td>
</tr>
<tr>
<td>Glaister (1962)</td>
<td>Dark blue</td>
<td>Dark blue</td>
<td>Green</td>
<td>Yellow</td>
<td>Resolution</td>
</tr>
<tr>
<td>Polson et al (1985)</td>
<td>Red, dark or red/black</td>
<td>Light blue/ red</td>
<td>Yellow (begins)</td>
<td>Yellow</td>
<td>Yellow</td>
</tr>
<tr>
<td>Adelson (1974)</td>
<td>Red/blue, purple</td>
<td>Blue/brown</td>
<td>Yellow/green</td>
<td>Resolution</td>
<td>Resolution</td>
</tr>
</tbody>
</table>

The data are adapted from various authors’ observations, excluding studies from photographs.

mind that the location of a bruise also needs to be taken into account when assessing its age. For example, a deep bruise in thigh muscle might not appear for a day or two after infliction and might have a fresh appearance, whereas a bruise over a bony prominence and where tissue is loose—for example, the eyebrow—will appear very quickly and with accompanying swelling.

Larger bruises remain visible for longer than smaller ones. The development and rate of disappearance of a bruise will also depend on the state of health and age of the person. Bruises take much longer to disappear in the elderly.

GROSS NAKED EYE AND PHOTOGRAPHIC ASSESSMENT

Studies of bruising carried out in living subjects that are based on their appearance in photographs illustrate the difficulties involved in timing such injuries. Langlois and Gresham,1 in their study of 369 photographs, found that the most important change was the development of a yellow colour, which was also found to develop much faster in those under 65 years of age. They concluded that bruises with a yellow colour are more than 18 hours old and that the appearance of the other colours is less important. In a similar study,2 assessing photographs of children who suffered accidental bruising, a yellow colouration was not observed in those bruises that were under one day old.

Over the years, several authors of forensic medicine textbooks have made various observations on colour changes in bruises with time (table 4).3–10 It is clear that the red/blue/purple colour in bruises can persist for days and even longer. The green colour, however, is difficult to interpret because it can reflect a combination of blue and yellow, but is nevertheless seen as an intermediate stage between the initial appearance and the later yellow appearance. One of the interesting findings is the time at which the yellow colour first appears and can be seen with the naked eye. Most authors writing in textbooks and referring mainly to anecdotal evidence of their own personal experience state that the yellow colour does not appear until at least several days and usually one week after injury. One comprehensive study of bruises13 pointed out that deep bruises can take 12–24 hours to appear, and that a brown colour would indicate that the bruise was over 24 hours old. This contrasts with others,1 who did not find a brown colour until the end of the first week.

MICROSCOPICAL CHANGES IN BRUISES

Most of the basic observations used for the timing of bruises have been known for over 100 years. Virchow in 1847 first described pathological pigments in old haemorrhagic areas inside and outside cells as diffuse, granular, and crystalline haemosiderin,12 and which was later identified as bilirubin.13 In 1869 erythrophagocytosis was detected in haemorrhages,14 and in 1888 the iron containing pigment in old haemorrhages was named haemosiderin and differentiated from haematoxidin.15

The earliest change seen microscopically in the development of a bruise is oedema, resulting in fluid exudation within the vicinity of the haemorrhage, together with widening of fibrous septa. This early, non-cellular exudative phase merges into the leucocyte reaction. It should be appreciated that it is not only the white blood cells extravasating with the haemorrhage that are detected, but also the granulocytes that migrate from neighbouring blood vessels. The earliest recorded leucocyte reaction was detected at 20–30 minutes,15 although more recent observations give times in both haemorrhages and wounds varying between one and 24 hours. With regard specifically to subcutaneous haemorrhages, the first influx of polymorphonuclear leucocytes has been noted after about four hours.16 The first leucocytes to migrate become increasingly necrotic after about 15–30 hours and are superseded by phagocytic mononuclear cells (macrophages).

Erythrocytes are seen in macrophages between 15 and 17 hours after infliction of the injury in the skin or brain, whereas the corresponding figure in the human lung is as early as 30 minutes.

Haemosiderin laden macrophages have been seen in the skin and subcutaneous tissue as early as 24–48 hours after the infliction of trauma, and more commonly from four to eight days. In brain, haemosiderin has been seen in macrophages as early as three to four days, but more commonly between five and 15 days. In human subdural haematomas, haemosiderin has been described as occurring after five days, whereas in the human lung haemosiderin is found much earlier (the earliest being at 17 hours).

In human postmortem material, haematoxidin (bilirubin) has been found in haemorrhages of the skin and subcutaneous tissue from nine days. In haemorrhages of the human brain, haematoxidin has been observed from 10 to 12 days onwards.

The Perl’s Prussian blue reaction has been, and still is, the most useful stain for the detection of ferric iron. However, the presence of haematoxidin is not so regular and its demonstration in paraffin wax embedded sections using the Van Gieson method is seldom...
Interpreting bruises at necropsy

Table 5: Suggested schema for the histological estimation of the time interval from infliction of injury to death in open skin wounds and abrasions

<table>
<thead>
<tr>
<th>Time interval</th>
<th>Histology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 4 hours</td>
<td>No distinct signs of inflammation. Histological distinction between antemortem and postmortem skin wounds not possible</td>
</tr>
<tr>
<td>4–12 hours</td>
<td>4 hours: some polymorph leucocytes perivascularly</td>
</tr>
<tr>
<td></td>
<td>8–12 hours: polymorphs, macrophages, and activated fibroblasts form distinct peripheral wound zone</td>
</tr>
<tr>
<td></td>
<td>Polymorphs more frequent than macrophages (5:1). Imminent necrosis in central zone</td>
</tr>
<tr>
<td></td>
<td>16–24 hours: relative number of macrophages increases, with polymorph to macrophage ratio falling to 0.4:1</td>
</tr>
<tr>
<td></td>
<td>After 16 hours older fibrin stains bright red with Martius scarlet blue, whereas before 16 hours “newer” fibrin stains yellow</td>
</tr>
<tr>
<td></td>
<td>24 hours: the number of polymorphs and amount of fibrin increase to maximum (remain at this value for 2–3 days)</td>
</tr>
<tr>
<td>2–4 days</td>
<td>Cut edge of epidermis shows cytolytic processes</td>
</tr>
<tr>
<td></td>
<td>24–48 hours: epidermis migrates from the incised edge towards the centre of the wound</td>
</tr>
<tr>
<td></td>
<td>At 32 hours and after, necrosis is apparent in the central wound zone</td>
</tr>
<tr>
<td></td>
<td>48 hours: macrophages reach maximum concentration in peripheral zone</td>
</tr>
<tr>
<td>2–4 days</td>
<td>2–4 days: fibroblasts migrate from the nearby connective tissue to the wound periphery</td>
</tr>
<tr>
<td></td>
<td>3 days: epithelialisation of small wounds and abrasions complete; thereafter regenerated epidermis becomes highly stratified and thicker than the normal surrounding epidermis</td>
</tr>
<tr>
<td></td>
<td>3–4 days: capillary buds appear</td>
</tr>
<tr>
<td>4–8 days</td>
<td>4 days: first new collagen fibres seen</td>
</tr>
<tr>
<td></td>
<td>4–5 days: profuse ingrowth of new capillaries; capillaries continue to proliferate until 8th day</td>
</tr>
<tr>
<td></td>
<td>6 days: lymphocytes reach maximum concentration in wound periphery</td>
</tr>
<tr>
<td>8–12 days</td>
<td>Decrease in number of inflammatory cells, fibroblasts, and capillaries; increase in the number and size of collagen fibres</td>
</tr>
<tr>
<td>&gt;12 days</td>
<td>12 days: definite stage of regression of cellular activity in both epithelium and dermis. Vascularity of dermis diminishes. Collagen fibres restored. Epithelium shows stainable basement membrane</td>
</tr>
<tr>
<td></td>
<td>At 14 days fibroplasia reaches its peak. Thereafter there is gradual shrinkage and maturation of connective tissue in the wound</td>
</tr>
</tbody>
</table>

Enzyme histochemical investigations of subcutaneous bruises from routine postmortem material revealed an increase in ATPase activity in the vascular walls after 2.5 hours. Aminopeptidase and esterase activity increased at 4.5 hours and seven hours, respectively, and polymorphonuclear and mononuclear cells appeared within four and nine hours, respectively. Using several histochemical methods (Alcan blue periodic acid Schiff (PAS) and dialysed iron PAS, both controlled by enzyme digestion), after initially decreasing, acid glycosaminoglycans were demonstrated in bruises that were several days old, together with an increase in the cellular elements of the connective tissue.

It is also appropriate in discussing age changes in bruises to consider the morphological changes seen in open skin wounds and abrasions. Histological ageing, which is based on these changes, will generally follow a definite order and can be divided into three stages:

- Inflammatory phase (one to three days after injury): vascular, haemostatic, and cellular response.
- Proliferative phase (up to 10–14 days after injury): epithelial and connective tissue regeneration.
- Reorganisation or remodelling phase (several months after injury).

Table 5 gives a brief timetable of changes suggested by Raekallio, applicable to the histological examination of skin wounds in general.

BIOCHEMICAL METHODS

Biochemical methods are of little practical use in routine forensic practice.

Although measurement of the increase of bilirubin content in haematomas is possible, and seems to correlate with the age of the haematoma (K Laiho at the 8th meeting of the...
Scandinavian Forensic Society, Vedbaek Denmark 1982,21 there are serious methodological difficulties with this approach. Attempts to separate bilirubin from haemoglobin in supernatants of tissue homogenates have been largely unsuccessful and have led to considerable losses of bilirubin. Furthermore, the turbidity of the supernatant is also a problem in spectrophotometric analysis.

Extraction of haem and extractable iron from homogenates is straightforward. However, the concentration of extractable iron alone cannot be used for age estimation because its concentration obviously depends on the amount of haemoglobin present and degraded in the tissue. Total iron concentration minus the control values is at least partly related to the initial concentration of haemoglobin present in the tissue. The ratio of extractable iron in relation to haem content reflects the change between the amount of haem or haemoglobin present in the tissue and the degraded extractable iron, but is influenced more by the amount of blood originally present than the ratio based on the total iron. When the haemorrhage in the tissue is very small and the values of extractable iron, total iron, and haem are nearly the same as in the control tissue, the evaluation of the age of a haematoma by ratio comparison would give unreliable results.

OBJECTIVE COLOUR ASSESSMENT

Dating a bruise from its colouration is problematical for various reasons, as discussed earlier. One of these reasons is that in general forensic practice the colour of a bruise on a cadaver or a living person is assessed by naked eye examination which, by its very nature, is subjective and conditions for assessment are virtually impossible to standardise.

Despite the limitations of relating colour change to date of trauma infliction, it is useful to apply a standardised method for measuring colour and intensity (lightness or luminance) of a bruise.

There are two standard techniques that are used to measure colour and that have been applied to bruising, namely: colorimetry and spectrophotometry.

Measurement of skin colour by colorimetry was initially used22 21 to assess hypostasis in relation to time of death estimation and was subsequently applied to colour changes in bruising (our unpublished results, 1997).24 The colorimeter is designed to work on the same principle as the human eye, which interprets the pigmentation of an image by calculating the amount of light, as well as the proportions of its constituent red, green, and blue colours (fig 1).

It allows an accurate, non-subjective assessment to be made, giving a numerical value based on an internationally recognised colour system: system CIE (Commission Internationale de l’Eclairage) L*a*b*. Thus, any object can be described in terms of its spatial position on a three dimensional axis (fig 2). The vertical axis L describes the luminance of the image, the horizontal a* value describes the position of the red–green axis, and the b* value describes the position on the yellow–blue axis.

The technique is easy to perform, fast, gives reproducible results, and is non-invasive. The test surface is lit by a standard illuminant produced by a xenon flash lamp, which approximates to the same conditions as daylight, and the light is directed to three measuring filters for the three standard tristimulus values X, Y, and Z. A complicated series of formulae is required to convert the raw data into the L*a*b* format, which is the conventional form for communicating the results.

Using this technique, data recently presented (our unpublished results, 1997) were in broad agreement with naked eye observations in relation to colour changes. It was noted in this study of 93 live subjects examined in a casualty department that, although there was great variability in colour change, the results followed the general trend seen in other studies. Furthermore, in common with the observations of two of these a yellow colour was not seen until towards the end of the first day.

Colour change can also be measured using spectrophotometry. This technique is used in many branches of science and its main use is to identify substances by their colour properties. Spectrophotometry works on the principle that different substances absorb, reflect, or emit light in different ways. These changes in light intensity, relative to its wavelength after it
has interacted with the sample, can be measured. To summarise the process, a spectrophotometer emits light from a lamp on to a test sample. The sample absorbs some of the light, with the rest passing through. In the spectrophotometer, a diffraction grating disperses the light from the sample by wavelength and the spectrum formed is observed with a detector. Reflectance spectrophotometry (light measured on the same side of the sample as the source of light and used on opaque samples such as solids) is now commonly used in dermatology. It was first used to analyse skin colour and has since been developed to measure colour changes in skin under various circumstances (such as the application of corticosteroids). The technique has also been applied to measure colour changes in subcutaneous bruises over time. These authors performed measurements between wavelengths 450 and 700 nm (visible wavelength 360/380–750/780 nm) and found the best results to be between 540 and 580 nm. Although they were not able to produce unambiguous results on the ageing of bruising, the authors concluded that the technique could be used as an objective non-invasive technique for bruise analysis. However, a recent study using both excised postmortem samples and live subjects found spectrophotometry to be of limited use because of the large number of variables involved.

Differentiation from artefacts and other postmortem appearances

Clearly, it is essential to establish whether an area that is discoloured and has the appearance of a bruise is in fact a bruise. This is generally straightforward in a non-putrefied cadaver, although even in such cases there are occasions when confusion may arise.

“PSEUDO BRUISING”

The extravasation of blood into the tissues after death, for whatever reason, in certain circumstances can lead to misinterpretation. I have avoided using the word bruising in relation to postmortem events because the forensic understanding of the word implies an antemortem phenomenon. The phrase “pseudo bruise” is perhaps preferable and could be applied to postmortem discoulourations that resemble true bruises.

This problem is of more than academic interest and, as most forensic pathologists will confirm, the distinction between bruising and pseudo bruising can sometimes be difficult if not impossible, both at necropsy and even after routine histological and immunohistochemical examination.

At this point, it is appropriate to consider whether blunt trauma delivered after death and causing tissue disruption can result in extravasation of blood with an appearance indistinguishable from bruising produced before death. It has been reported in one study that the production of postmortem “bruises” required the application of considerable violence, with the resulting bruise almost always being small or wholly disproportionate to the force delivered. Interestingly, however, they found that a blow with a wooden mallet delivered after death to the occiput (moderate force was used so as not to fracture the skull) can cause blood extravasation involving the full thickness of the scalp up to one inch (2.54 cm) in diameter. Bearing in mind that the scalp is rich in blood vessels and that cadavers are stored in the supine position, this might not be an unusual phenomenon, and therefore such bruising requires careful interpretation.

HYPOSTASIS AND CONGESTION

Postmortem lividity or hypostasis, which results from pooling of blood after death as a result of gravity, gives a red/purple appearance to the areas where the blood is pooled. Usually there is no difficulty differentiating hypostasis from bruising, except where the hypostasis has a patchy appearance, or bruising is on the back (cadavers stored in a mortuary in a supine position are usually found with a substantial proportion of lividity on the back). The situation can be compounded by congestive changes to the body, particularly where congestion is seen in association with deaths involving some kind of trauma.

Cases involving a mechanical asphyxial mode of death, such as manual strangulation or postural (positional) asphyxia, might show areas of congestion and genuine bruises, the sizes of which are larger than would be expected because of the increased volume of blood in surrounding vessels, which will escape and contribute to the bruised area. In addition, congested areas may show postmortem ecchymoses that resemble petechial haemorrhages. These are often seen within areas of hypostasis.

Although it is recommended that a careful dissection of the involved area should be carried out to assess whether, as in the case of a bruise, the blood has escaped from blood vessels into the surrounding tissue, occasionally (particularly with pronounced congestion) this can still be problematical. It is sometimes useful to move the cadaver into another position to allow drainage of pooled blood to a secondary position; true bruising will remain in the same position.

POSTMORTEM INJURIES

All pathologists are familiar with the typical appearance of postmortem injuries, which have a tendency to a yellowish brown “bloodless” appearance and lack vital reaction. To the naked eye the bruising accompanying injuries (such as lacerations) demonstrate the appearance of the extravasation of blood (red/purple/blue when fresh) and other changes reflecting tissue reaction to injury, and which are dependent on the interval between the infliction of trauma and death. However, in many instances, where there is congestion of the cadaver, sufficient blood escapes from vessels damaged after death to give an appearance of bruising that is indistinguishable from a fresh injury occurring shortly before death.

The postmortem dissection procedure will also produce artefactual bruising that is indistinguishable from true bruising. This is
particularly problematical when pronounced congestion is present or where the area in question is very vascular. The pathologist carrying out a second necropsy must be aware of this and not over interpret findings. Furthermore, postmortem dissection will also facilitate the migration of genuine bruising. Against this background, true bruises will be modified after death as discussed in the next section.

A particular situation that can lead to misinterpretation concerns the examination of the neck structures in asphyxial deaths involving compression—for example, manual strangulation. In such cases it is important to bear in mind that the neck structures will probably be congested. The assessment of bruising and its distribution is of paramount importance to the pathologist in his/her examination. As stated above, bleeding from vessels cut or pulled away from other structures occurs as part of the postmortem dissection and evisceration, and results in collections of blood in soft tissues and organs that might be thought erroneously to be true bruises. With regard to the neck, even with careful dissection of the anterior structures away from the cervical vertebral column, considerable extravasation of blood can still occur. Such artefactual production of bruising has been described as the Prinsloo-Gordon effect. These authors recommend that a bloodless field be produced in the neck region before examination and removal of the anterior structures. This is achieved by opening the skull as well as the chest and abdomen to allow free flow of blood from these areas before dissection of the neck.

The differentiation between haemorrhage that has occurred before or after death might be impossible where injuries are inflicted just before, during the agonal phase, or within a short time after death.

Clotted haemorrhages were originally thought to occur only during life, but it has been shown that coagulation in blood can occur as late as six hours after death. Furthermore, it has also been found that the standard histological staining methods for fibrin are not conclusive and that the absence of fibrin does not mean that the haemorrhage occurred after death. Even with immunohistochemical studies, the same author could not distinguish postmortem from antemortem fibrin with certainty. In contrast, however, well preserved fibrin networks found at necropsy performed two to three days after death pointed to an antemortem or agonal haemorrhage. This is because a large proportion of a postmortem subcutaneous haemorrhage undergoes fibrinolysis within one day of its production.

**RESUSCITATION INJURIES AND HANDLING AFTER DEATH**

Another area of practical difficulty for the pathologist is the differentiation of bruises from marks caused by resuscitation. Because in most instances resuscitation occurs around the time of death with some degree of maintenance of a circulation, or at least intermittent forced movement of blood within vessels, there is every possibility of producing extravasation of blood to the tissues that is indistinguishable from true bruising. Therefore, care should be taken in the examination of injuries in areas of the body where resuscitation has taken place. These areas include the face, neck, and chest. Frequently, the question arises in cases involving pressure to the face and neck, especially manual strangulation, where fingertip type bruising, as well as other marks such as abrasions caused by fingernails, are commonly found. The problem is compounded in areas where there is intense congestion.

**DECOMPOSITION**

With the increasing postmortem interval, bruises become more diffuse and are frequently accentuated in intensity as a result of the degradation products of haemoglobin. Indeed, bruises can appear a day or two after the postmortem examination that were not visible at the first necropsy, or those that were seen initially can appear more pronounced. Finger-tip bruises indicative of grip marks are a particularly good example of this phenomenon. With the onset of putrefaction, the body becomes discoloured and bruises become modified in their appearance, making their accurate assessment difficult. Immunological methods have demonstrated the usefulness of glycoprotein A, a constituent of red blood cell membranes, as a marker to differentiate between true bruising and putrefactive discoloration. Although haemoglobin pigments readily filter through blood vessels, erythrocyte membranes do so less easily because of their molecular size. Therefore, bruises will contain a greater amount of erythrocyte membrane material than areas of discoloration resulting from putrefactive change. However, glycoprotein A cannot help to differentiate between antemortem and postmortem injury because extravasated blood from vessels includes erythrocytes, regardless of whether the damage occurred before or after death.


