

## THE AGEING OF BRUISES: A REVIEW AND STUDY OF THE COLOUR CHANGES WITH TIME

N.E.I. LANGLOIS and G.A. GRESHAM

*Department of Pathology, Addenbrooke's Hospital, University of Cambridge (U.K.)*

(Received July 8th, 1990)  
(Revision received April 16th, 1991)  
(Accepted June 16th 1991)

### Summary

This work was inspired by a recent case of child abuse where the question of the age of the bruises on the body was raised. The first part of this paper reviews published work on bruises. It illustrates the paucity of work in this field and the absence of studies of the colour changes in bruises of human skin with time. The second part of this paper consists of our own study of the appearance of bruises. The aim was to identify the colour changes which occur in bruises and over what time-scale, in order to determine whether bruises can be aged by appearance. A total of 369 photographs were obtained of bruises aged <6 h and up to 21 days old, in 89 subjects aged 10–100 years. It was found that the development of a yellow colour was the most significant change (subjects aged <65,  $P < 0.001$ ; subjects aged  $\geq 65$  years,  $P < 0.001$ ). The development of a yellow colour occurred significantly faster in subjects aged <65 years. ( $P < 0.001$ ). The appearance of a blue and purple/black colour was of lesser significance. The appearance of a red colour did not alter significantly with time. From this study it was only possible to conclude that a bruise with a yellow colour was more than 18 h old.

*Key words:* Bruises; Ageing; Skin

### Introduction

“Bruises are a much neglected branch of injuries”. These words were delivered by the late Sir Bernard Spilsbury in an address to the Medico-Legal Society in 1938 [1]. Little has changed since then. Forensic pathologists are aware of the importance of recording details of injuries on a victim's body. As an “expert witness” the forensic pathologist is often asked to age a bruise which has been faithfully recorded. However, it became apparent at a recent Crown Court case of death from child abuse that there are little or no sound data on which such estimates of age can be based. This situation has been encountered by others and Wilson [2] has commented on how vague and varying forensic texts are on the subject of ageing bruises by inspection.

This paper consists of two parts: the first is a review of published work on the ageing of bruises. It covers the aspects of the appearance of the bruise and methods which have been applied to age bruises. No published work on the colour changes in bruises of human skin with time have been found; only some guide-

lines in forensic texts. The issue of whether a bruise can be aged by appearance was found to be controversial. Some work has been performed on other methods of ageing bruises, both in animals and man and this is included in the review. The second part of this paper consists of our own study of changes with time in the appearance of bruises on human skin. This was intended to be preliminary work to identify the colours that occurred over periods of time. The aim was to see if it was possible to age a bruise by appearance if one was presented with a photograph.

### **Part I: A Review of Published Work**

A bruise can be defined as “a hurt or injury to the body by a blunt or heavy instrument causing discolouration, but no laceration of the skin” [1]. Bruises consist of blood escaping from ruptured capillaries and small veins spreading into the surrounding tissue. For the purpose of studying bruises the skin can be considered in three layers:

- (a) a compact and firm outer layer (the epidermis), not easily damaged by crushing;
- (b) a middle, easily deformed layer (the dermis) with a superficial capillary network;
- (c) an inner easily deformed layer rich in capillaries (the subcutaneous tissues).

It is the capillaries of the subcutaneous tissues which make the greatest contribution to bruising. Extravasated blood will spread along any line of cleavage in the tissue producing a discoloured area. Further colour changes take place as haemoglobin breaks down [1,3].

Many factors affect the appearance of bruises, with increasing laxity and loose subcutaneous elements in the tissues there is increasing extravasation of blood. Thus bruises around the eye are more spectacular than those of the palm. Vascular or other tissue overlying bone will also bruise more profusely. Children and elderly people are reported to bruise more easily than young fit adults and increasing age has been shown to be associated with delayed resolution. Some women have been noted to bruise readily. The colour of the skin is important, as bruises are more obvious in fair-skinned people. Diseases such as hypertension and coagulation disorders may affect the extent of bruising [1, 4–8]. Other systemic diseases may have no effect on bruise resolution. Drugs, such as steroids, can alter the rate of bruise dispersion [9]. Previous bruising results in the faster resolution of successive bruises. Increasing mass and velocity of impact with the skin leads to increased area and depth of bruised tissue with prolonged healing time [7,8]. A bruise may appear almost instantaneously or it may take 24–48 h for the extravasated blood to rise to the surface. Thus bruises inflicted ante-mortem can appear after a latent period post-mortem [4,10,11]. Alternatively, the haematoma may not become visible and would require skin stripping to reveal it [1]. It is also recognised that bruising may be significantly reduced or may not occur if pressure is maintained over the area until death has occurred as in some cases of strangulation [1,10] or where death has occurred rapidly [12]. Displacement of the tissue planes and tracking of blood with time

may result in the site of the appearance of the bruise not coinciding with the site of impact [4,13], but it is sometimes possible to identify a particular type of weapon by the character of the bruise [11,12]. Lesions resembling ante-mortem bruises can be produced in neck muscles whilst removing the neck organs at autopsy, or upon the body if sufficient force is applied within a few hours of death [14]. However, less extensive haemorrhage into the interstitial tissue is a feature and it should not be difficult to distinguish ante-mortem bruises from post-mortem bruises and post-mortem lividity [4,10,15]. With so many factors influencing the appearance of a bruise it would not be surprising to find accurate ageing difficult. Methods which have been used to age bruises include visual assessment, biochemical and enzyme histochemical [16], histological [17] and electrophysiological [18].

Raekallio [16] has studied the enzyme histochemistry of wounds and found it to be of use in the early period after wounding before histological changes occur. The changes persist for up to 4 or 5 days post-mortem [19,20]. Free histamine and 5-hydroxytryptamine (5-HT) changes occur early within the first hour [21,22]. Changes in cellular enzyme levels are manifest from 1 to 16 h; however senility, cachexia and severe brain injuries can lead to spurious results [16]. The

TABLE 1  
DIFFERENT AUTHORS' OPINIONS ON THE TIME SEQUENCE OF COLOUR CHANGES IN BRUISES

<i>Source</i>	<i>Colour(s) in bruise</i>	<i>Time</i>
Camps [10,27]	Red	Immediate
	Dusky purple/black	Soon after
	Green	Days 4-5
	Yellow	Days 7-10
	Resolution	Days 14-15
Glaister [4]	Violet	Immediate
	Blue	Day 3
	Green	Days 5-7
	Yellow	Days 8-10
	Resolution	Days 13-18
Polson and Gee [28]	Red/dark red/black	< 24 h
	Greenish tinge	Around day 7
	Yellowing	Around day 14
	Resolution	Up to 30 days
Smith and Fiddes [29]	Red	Immediate
	Purple/Black	Soon after
	Green	Days 4-5
	Yellow	Days 7-10, but small and superficial day 3.
	Resolution	Days 14-15.

effect of age on the rate of enzyme changes has been studied in rats. The enzyme changes occur in the same chronological order, but the older rats took longer to reach the maximum intensity of reaction. The younger rats showed a more intense reaction, but not an earlier reaction [23,24]. Enzymes have been studied specifically in bruises of poultry [25] and guinea pig [26] showing similar results.

There is disagreement as to whether bruises can be aged by appearance. Some authors maintain that it is not possible to age a bruise by appearance [1,12 27], whereas others have published guidelines for judging the ages of bruises [4,10, 28–30] (see Table 1). Although there is a difference of opinion on the exact times there is a consensus that red, blue and purple are early colours, greens appear after 4–7 days and yellow does not appear until at least the seventh day, with the notable exception of Smith and Fiddes [29] who noted yellow in small, superficial bruises by the third day. Moritz [11] has probably published the most comprehensive study of bruises. He noted that if the extravasated blood in a bruise is superficial the red/blue colour may be almost instantaneous, whereas deeper bruising may take 12–24 h to appear. Although he states that it is not possible to estimate accurately the time since injury from the gross appearance of the bruise he does concede that a bruise which appears brown is likely to be more than 24 h old. Bruises have been noted to develop a yellow colouration after death [6] and discolouration can occur if the body is refrigerated [1]. Interestingly, the environmental temperature affected the colour change and rate of healing in bruises of chickens [31]. In experimental bruising of cattle the following sequence of colour changes were found by Hamdy et al. [32]: red colouration from 15 min to 2 days (due to red blood cells and free haemoglobin); green from day 3–4 and yellow and orange from days 4–6 (due to bilirubin). In further work on cattle and rabbits it was found that the sequence of colour changes was consistent, but the rate of change varied with subsequent bruises or in animals of different ages [8]. A study of bruises in calves by McCausland and Dougherty [33] revealed that a yellow colouration appeared in the bruise by 48 h, which is considerably earlier than the above studies.

The production of bilirubin at the site of bruising has been studied using Fouchet's reagent [33]. In poultry bilirubin was detected by Fouchet's reagent by 14–21 h and was not detected after 4 days [31]. In cattle, however, bilirubin was not detected by Fouchet's reagent before 60 h, but was still present at 5–8 days [8]. When spectrophotometry was applied, bilirubin and biliverdin levels above controls were found at 12 h in poultry [34] and at 2 days in cattle [32]. This would seem to indicate that the use of Fouchet's reagent is not as sensitive in the detection of bilirubin as spectroscopy. It was found that the yellow colour of bruises could be attributed to bilirubin [32].

The histology of bruises in the skin has not been widely studied. One of the few published studies is of bruises in lambs and calves [33] where the following changes were noted:

- (a) Lesions sampled immediately after bruising showed haemorrhage in the subcutaneous tissues and between muscle fibres. Some neutrophils and macrophages were present and a fine network of fibrin was noted.

- (b) In 8 h-old lesions the amount of haemorrhage was greater, there was also more fibrin, neutrophils and macrophages. Some muscle fibres were noted to show degenerative changes.
- (c) At 24 h the amount of haemorrhage and fibrin was unchanged. There was an equal ratio of neutrophils to macrophages some of which contained haemosiderin.
- (d) At 48 h the number of macrophages considerably outnumbered the number of neutrophils. There were heavy deposits of haemosiderin and fibrin nets.

Bruising in the guinea pig intrascapular fat pad has also been studied [26] revealing the emigration of polymorphic neutrophils by 30 min and macrophages within 60 min. Fibroblast proliferation was noted to start by 1–2 h with an increase of ground substance at 2–4 h and an increase in collagen by 8–24 h. New capillaries were found at 24 h. The effect of trauma on muscle has been extensively studied by Allbrook and colleagues [36–38] who have noted intracellular oedema with a loss of definition of myofilaments within a few hours. Polymorphonuclear infiltration beginning at 2–12 h and muscle fibre degeneration occurring by 24 h. Definite signs of muscle fibre regeneration were seen by 4–6 days post-injury.

Statistical analysis has been applied to the histological features in bruises of sheep [16]. Features noted to be of use in the ageing of bruises are:—

- (a) the degree of neutrophil and macrophage exudate, the degree of fibroplasia;
- (b) the amount of haemosiderin in macrophages and the degree of endothelial cell hypertrophy.

Using these features it was found only possible to age bruises as 1–20 h old or 24–72 h old with any degree of confidence.

## **Part II: A Visual Study of Bruises**

### **Materials and Methods**

Photographs were taken of the bruises using a high definition colour daylight film, a single lens reflex camera with a suitable lens for close-up photography and an electronic flash mounted on the camera. Three sources were used: patients attending the accident and emergency department, in-patients and staff. Bruises caused by trauma from accidents were used, but not in severely injured subjects. Only bruises where the cause and age were known were photographed. Included in each photograph was a standard colour chart and a scale to allow accurate colour rendition at processing. When possible bruises were photographed as a sequence from the time of appearance to resolution; however, many photographs were a one-off sample. The photographs were assessed for the presence of particular colours and this data was collated.

### **Results**

A total of 369 photographs were taken of 89 subjects with an age range of

TABLE 2  
 NUMBER OF SAMPLES OBTAINED IN EACH TIME INTERVAL

Time (h)	Number of bruises in each time interval	
	Subjects aged > 65 years	Subjects aged ≥ 65 years
0-6	28	6
7-18	5	8
19-30	34	12
31-42	11	4
43-60	46	21
61-84	40	20
85-108	15	14
109-132	11	13
133-156	7	12
157-228	12	17
229-348	9	21
348-516	0	3

10-100 years over a 5-month period. Some subjects had more than one bruise and some subjects were photographed on more than one occasion. The photographing of bruises was important, as bruises take on different hues depending on the ambient light, but the camera and flash arrangement gave consistent results when tested. The data were separately collated in time intervals for subjects <65 years and those ≥65 years. The number of photographs obtained of bruises in each time interval is shown in Table 2.

The main colours noted and subjected to analysis were blue, red, yellow and purple/black. A green colour was observed in some bruises, but in many cases it was difficult to tell whether this was a true colour or a mixture of blue and yellow. For the same reasons orange and brown, which were also observed, were also not counted. The frequency of occurrence of each colour was ascertained within each time interval for the two age groups and for the two age groups combined. Graphs showing the percentage of photographs in each time interval with the colour yellow (Fig. 1), red (Fig. 2), blue (Fig. 3) and purple/black (Fig. 4) are shown for all the photographs collected for both age groups. It can be seen that yellow is not present in bruises in the first two time intervals (0-6 h and 7-18 h); thereafter it is observed with increasing frequency in bruises up to the 157-288 h time interval. Red is commonly present in bruises of all ages. Purple/black and blue are less commonly seen in bruises and their frequency of occurrence appears to decrease with time.

The data were analysed separately and statistically using logistic regression for the bruises in subjects aged <65 and those aged ≥65, to see if a clearer pattern would emerge. For both age groups the increase in frequency of yellow was highly significant ( $P < 0.001$ ). The rate of increase was faster for subjects aged

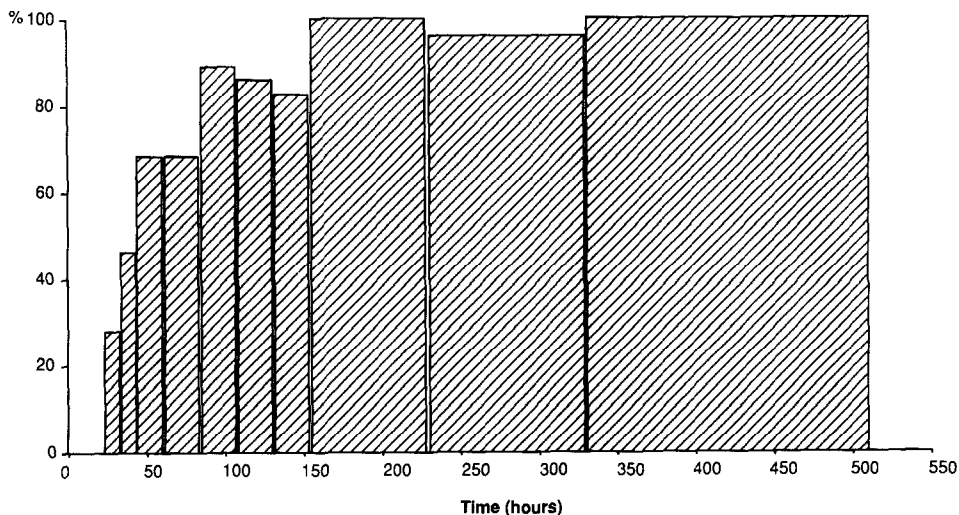


Fig. 1. Percent of samples in each time interval showing yellow colour.

< 65 years than for those aged  $\geq 65$  ( $P < 0.001$ ). For subjects aged < 65 years the decrease in frequency of purple/black was significant ( $P < 0.01$ ) and for subjects aged  $\geq 65$  the decrease in blue was significant ( $P < 0.025$ ). No other changes showed a significant dependence on time.

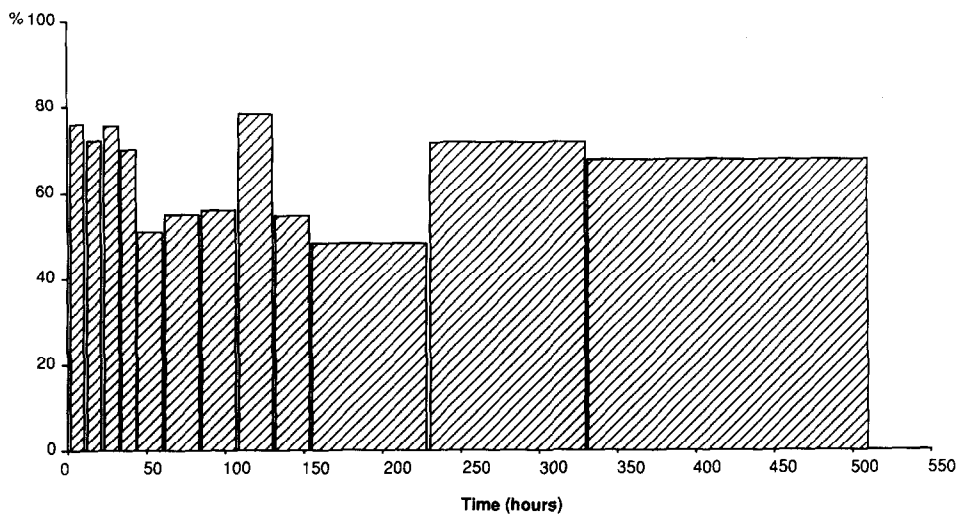


Fig. 2. Percent of samples in each time interval showing red colour.

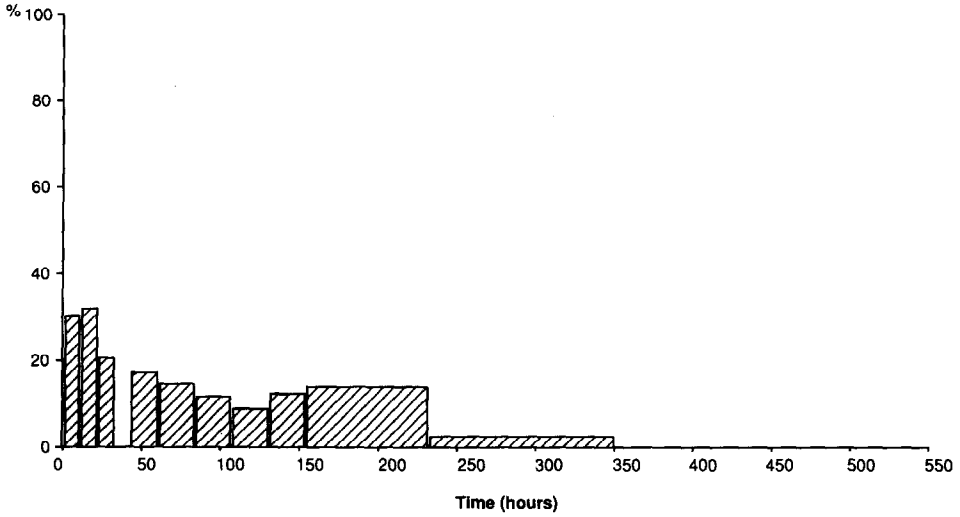


Fig. 3. Percent of samples in each time interval showing blue colour.

Some features that are not apparent from the data are important and should be noted. Firstly, the colours in bruises are dynamic, as colours which are present one day may disappear the next, only to reappear at a later date. Secondly, even in the same subject where two separate bruises have occurred on the same part of the anatomy and are identical in aetiology and age these need not display identical colours, nor undergo changes of colour at the equivalent rate. Thirdly,

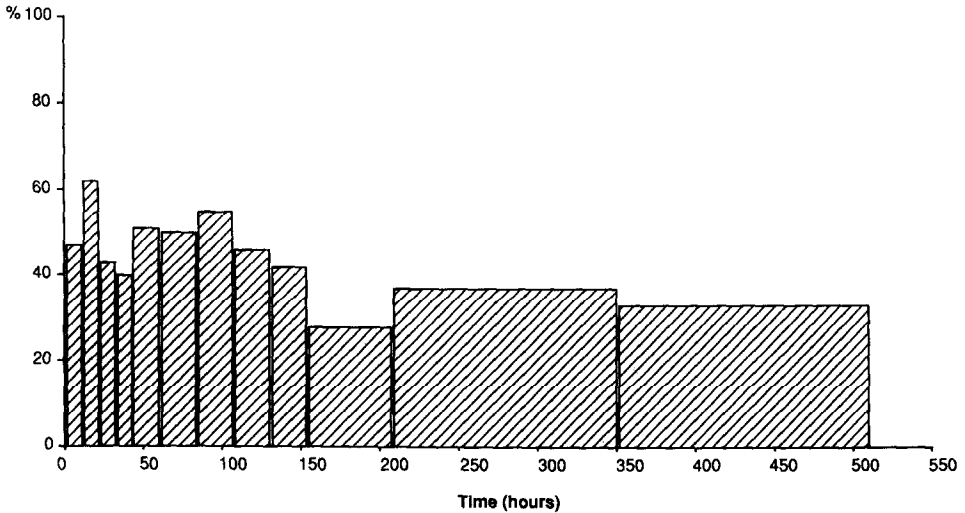


Fig. 4. Percent of samples in each time interval with purple/black colour.



not all bruises develop a yellow colouration before they resolve. This is true for both young and old subjects. Fourthly, the colour of the skin is important, as in subjects with a yellowish skin it was difficult to determine if a yellow colour was present in the bruise. It was very difficult to discern the colours in a bruise in a dark-skinned subject and therefore this study was limited to caucasians.

## Conclusion

The appearance of a yellow colouration is a highly significant colour change in a bruise ( $P < 0.001$ ). No bruises in the time intervals 0–6 and 7–18 h showed a yellow colour. Thus if a bruise has a yellow colour it is very likely to be more than 18 h old; unfortunately it is not possible to assign a probability value to a study of this kind. Older subjects showed a slower development of a yellow colour ( $P < 0.001$ ). The colours red, blue and purple/black could occur anytime from within 1 h of bruising to resolution (up to 21 days in this study). The appearance of red colouration has no bearing on the age as there is no significant change. Although blue and purple/black colouration may show a significant decrease in appearance with time, their small overall change and variability render them unlikely to be useful for ageing. It must also be noted that bruises of identical age and aetiology on a subject may not show the same colours or undergo changes at an equal rate. Therefore, we conclude that a bruise which shows a yellow colour must be more than 18 h old, but the converse (that a bruise without a yellow colour is less than 18 h old) is not necessarily true. However, it must be noted that bruises may not develop a visible yellow colour until much later than after 18 h.

## Discussion

Our study of the colour changes in bruises focuses on the definite appearance or absence of colour. The data consist of a series of samples collected over a 5-month period, most of which are one-off, but some are series from patients and staff. It was difficult to obtain a full series of photographs of bruises from appearance to resolution, as often the bruise was not discovered early enough or the subject left before the bruise resolved. It was also difficult to find bruises where the cause and exact age were known. We did not use subjects with major trauma as the systemic effects may have had an effect on bruise healing. We wanted to determine if it would be possible to assign an age to a bruise that had been photographed and presented as evidence in a court case. As no reference to a previous study of this kind had been found this was intended as a preliminary study. We feel our review and findings are of importance to those involved in the medico-legal field.

The development of a yellow colour in bruises is the most significant change, however, when (and if) it occurs is variable. We could detect no visible yellow colour in bruises in the time intervals 0–6 and 7–18 h. The yellow colouration in bruises has been attributed to bilirubin [32] which is derived from the metabolism of haemoglobin [39]. It has been shown that the degradation of haemoglobin does

not occur in the skin [40]. The development of a yellow colour in bruises as early as the 19–30 h interval was initially surprising. However, other studies have reported a yellow colour in haematomas of 24 h old [9, 41]. Moritz [11] has noted uptake of red blood cells by phagocytes within a few hours of bruising and by 24 h that they react positively with Prussian Blue stain for iron. Thus it is likely that the bilirubin in a bruise is produced locally in phagocytes and the delay in appearance of a yellow colour may be due to the time required for sufficient amount of pigment to be produced locally to be visible. Presumably, in bruises where a yellow colour did not occur there was insufficient local bilirubin to give a visible colour. The red colour in bruises has been attributed to free hemoglobin [32]. The blue and purple/black is thought to be due to blood reflecting light at different depths in the skin. A decrease in red, blue and purple/black would be expected with time, but as we only recorded the presence or absence of these colours with time, this trend did not become so apparent. The fact that colours were found to be dynamic, that they may come and go with time, was assumed to be due to blood changing its position within the layers of the skin and its degradation. Other colours noted in the bruises photographed included green, brown and orange. A green colour has been noted in experimental bruises and has been attributed to biliverdin [35]. However, in our study it was noted that it was often difficult to tell if the observed green colour was true colour or was a mixture of yellow and blue. Therefore, green was not recorded as an independent colour. Similarly, a brown colour could be due to methemoglobin [42], but could also be a mixture of blue, red and yellow. Orange could be found where red and yellow coincided.

This work still leaves many questions unanswered. We still know little about bruising in children where differences in subcutaneous fat thickness and metabolic rates may affect bruising. As older subjects do show a slower development of yellow we would expect to see a difference in the rate of change in children. We also do not know whether major trauma affects the rate of colour change especially if the subject suffers from septicaemia, intravascular coagulation or the release of proteolytic enzymes from pancreatic injury. A larger study of serial photographs in subjects of different age groups analysed by Bayesian methods would be useful in answering the question whether bruises can or cannot be aged reliably by appearance.

There are a number of variables that are known to be involved in the production and healing of bruises: the effect of force, location on the body, age of the subject, previous bruising, blood pressure and even temperature (which is different at the periphery compared to the core) [7,8,31]. Given these variables and our observation that bruises of identical age and aetiology on the same part of the body need not show the same colours nor undergo changes at an identical rate, it would seem unlikely that a bruise could be reliably aged from appearance alone.

### **Acknowledgements**

We are indebted to Dr P.M.E. Altham of the Statistical Laboratory, Universi-

ty of Cambridge for the statistical analysis. We would also like to thank all those who allowed us to photograph their bruises at the Queen Elizabeth Hospital, King's Lynn. This work was supported by a Wellcome Trust small project grant (reference 033147/Z/91/Z/1.5/LHS/SRD).

## References

- 1 B. Spilsbury, The medico-legal significance of bruises. *Med.-Leg. Crim. Rev.*, 7 (1939) 215–227.
- 2 E.F. Wilson, Estimation of the age of cutaneous contusions in child abuse. *Pediatrics*, 60 (1977) 750–752.
- 3 K. Simpson and B. Knight, *Forensic Medicine*, Edward Arnold, London, 1985, pp. 54–56.
- 4 J. Glaister, *Medical Jurisprudence and Toxicology*, E. & S. Livingstone, Edinburgh, 1973, pp. 242–246.
- 5 F.E. Camps, Interpretation of wounds. *Br. Med. J.*, 2 (1952) 770–772.
- 6 S.H. Burgess and J.E. Hilton, *The New Police Surgeon*, Hutchinson Benham, London, 1978, pp. 170–171.
- 7 M.K. Hamdy, K.N. May and J.J. Powers, Some physical and physiological factors affecting poultry bruises. *Poultry Sci.*, 40 (1961) 790–795.
- 8 M.K. Hamdy, L.E. Kunkle, M.S. Rheins and F.E. Deatherage, Bruised tissue III: some factors affecting experimental bruises. *J. Anim. Sci.*, 16 (1957) 496–501.
- 9 R. Lovell, G.B.D. Scott, B. Hudson and J.A. Osbourne, The effects of cortisone and adrenocorticotrophic hormone on dispersion of bruises in the skin. *Br. J. Exp. Pathol.*, 34 (1953) 535–541.
- 10 F.E. Camps and J.M. Cameron, *Practical Forensic Medicine*, 2nd edn., Hutchinson, London, 1971, pp. 104, 167–169.
- 11 A.R. Moritz, *The Pathology of Trauma*, Henry Kimpton, London, 1942, pp. 21–34.
- 12 I. Gordon, H.A. Shapiro and S.D. Berson, *Forensic Medicine. A Guide to Principles*, Churchill Livingstone, London, 1988, pp. 223–229.
- 13 K. Simpson, *Forensic Medicine*, Edward Arnold, London, 1979, p. 10.
- 14 I. Prinsloo and I. Gordon, Post-mortem dissection artefacts of the neck and their differentiation from ante-mortem bruises, *S. Afr. Med. J.*, 25 (1951) 358–361.
- 15 I. Robertson, Ante-mortem and post-mortem bruises of the skin. *J. Forensic Med.*, 4 (1957) 2–10.
- 16 J. Raekallio, Determination of the age of wounds by histochemical and biochemical methods. *Forensic Sci.*, 1 (1972) 1–16.
- 17 R.N. Thornton and R.D. Jolly, The objective interpretation of histopathological data: an application to the ageing of ovine bruises. *Forensic Sci. Int.*, 31 (1986) 225–239.
- 18 C.V. Ananiev, V.V. Semyonov and A.V. Finlkovsky, Estimation of the age in bruises in living persons by multidimensional statistical analysis. *Sud.-Med. Ekspert.*, 27 (1984) 7–10.
- 19 J. Raekallio, Estimation of time in forensic biology and pathology. *Am. J. Forensic Med. Pathol.*, 1 (1980) 213–218.
- 20 J. Raekallio, Estimation of age of injuries by histochemical and biochemical methods. *Z. Rechtsmed.*, 73 (1972) 83–102.
- 21 J. Raekallio and P.-L. Makinen, Serotonin content as vital reaction. *Zacchia*, 44 (1969) 587–594.
- 22 J. Raekallio and P.-L. Makinen, Serotonin and histamine contents as vital reactions. *Zacchia*, 45 (1970) 403–414.
- 23 J. Raekallio and P.-L. Makinen, The effect of ageing on enzyme histochemical vital reaction. *Z. Rechtsmed.*, 75 (1974) 105–111.
- 24 J. Raekallio, The effect of ageing on enzymes in wound healing. A histochemical and biochemical study. *Gerontology*, 21 (1976) 31–35.
- 25 W.E. Brown and M.K. Hamdy, Some biological changes in traumatised poultry muscle. *Proc. Soc. Exp. Biol. Med.*, 119 (1965) 783–788.
- 26 J. Hirvonen, Histochemical studies in vital reaction and traumatic fat necrosis in the interscapular adipose tissue of adult guinea pigs. *Ann. Acad. Sci. Fenn., Suppl.*, 136 (1968).

- 27 J. Raekallio, Timing of the Wound. In C.G. Tedeschi, W.G. Eckert and L.G. Tedeschi, *Forensic Medicine, Vol. 1*, W.B. Saunders, Philadelphia, PA, 1977, pp. 22–28.
- 28 F.E. Camps, *Gradwohl's Legal Medicine, 2nd edn.*, John Wright, Bristol, 1976, p. 265.
- 29 C.J. Polson and D.J. Gee, *The Essentials Of Forensic Medicine*, Pergamon Press, Oxford, 1973, p. 101.
- 30 S. Smith and F.S. Fiddes, *Forensic Medicine*, Churchill, London, 1955, pp. 110–111.
- 31 M.K. Hamdy, K.N. May, W.P. Flanagan and J.J. Powers, Determination of the age of bruises in chicken broilers. *Poultry Sci.*, 40 (1961) 787–789.
- 32 M.K. Hamdy, F.E. Deatheraye and G.Y. Shinowara, Bruised tissue I: biochemical changes resulting from blunt injury. *Proc. Soc. Exp. Biol. Med.*, 95 (1957) 255–258.
- 33 I.P. McCausland and R. Dougherty, Histological ageing of bruises in lambs and calves. *Aust. Vet. J.*, 54 (1978) 525–528.
- 34 H.N. Nauman, Studies on bile pigments. *Biochem. J.*, 30 (1936) 762–764.
- 35 M.K. Hamdy, K.N. May and J.J. Powers, Some experimental and physical changes occurring in experimentally inflicted poultry bruises. *Proc. Soc. Exp. Biol. Med.*, 108 (1961) 185–188.
- 36 D. Allbrook, W. de C. Baker and W.H. Kirkaldy-Willis, Muscle regeneration in experimental animals and in man. *J. Bone Jt. Surg.*, 48b (1966) 153–169.
- 37 D. Allbrook, An electron microscopic study of regenerating skeletal muscle. *J. Anat.*, 96 (1962) 137–152.
- 38 J.C.T. Church, R.F.X. Noronha and D.B. Allbrook, Satellite cells and skeletal muscle regeneration. *Br. J. Surg.*, 53 (1966) 638–642.
- 39 R. Tenhunen, The enzymatic degradation of haemoglobin. *Semin. Hematol.*, 9 (1972) 19–29.
- 40 R. Lemberg and R.A. Wyndham, Reduction of biliverdin to bilirubin in tissues. *Biochem. J.*, 30 (1936) 1147–1170.
- 41 R. Muir and J.S.F. Niven, The local formation of blood pigments. *J. Pathol.*, 41 (1933) 183–197.
- 42 J.B. Miale, *Laboratory Medicine Hematology*, C.V. Mosby, St. Louis, MO, 1982, pp. 461–464.