POST-INJURY INTERVALS 1

INTRODUCTION

Accurate dating of injuries has been an area of considerable research and debate. The body's response to trauma is diverse and is affected by innumerable variables. A review of the literature will reveal a considerable variation in the time periods associated with injury development and appearance and that there is variation in rates of wound healing in different sites of the same individual. How much force caused the contusion? How deep is it? What is the underlying tissue – is it bone (like the skull or ribs), or is it elastic (such as the abdomen)? What was the nutritional status of the victim and would this be likely to affect their rate of healing? Would the decedent's natural disease state(s) affect the way they heal such that it may be faster or, more likely, slower than in the general population? These are all issues that need to be considered to interpret the age of traumatic lesions, and still we are often left with a more realistic binary decision between "acute" and "remote." It is imperative that you not permit yourself to get "painted in" to an age for a contusion or abrasion. These are best handled in windows of time, posited with the caveat that the vagaries of biology preclude a more precise time factor. Similar issues are encountered with dating subdural hematomas or cerebral contusions. In this chapter there are numerous photographic examples of injuries at many different time points. We have also included a number of tables, reviewed and collected from the existing literature for quick reference.

CONTUSION DATING

Skin

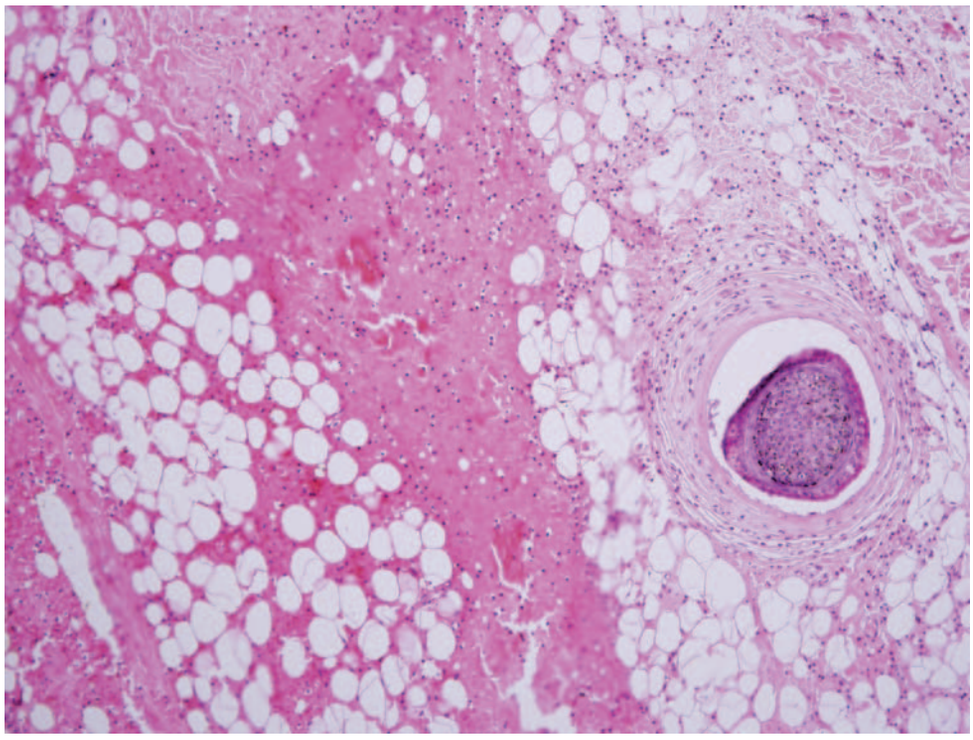

Figure 1.1. Acute contusion (4–12 hours). Acute hemorrhage with marked neutrophilic infiltration.

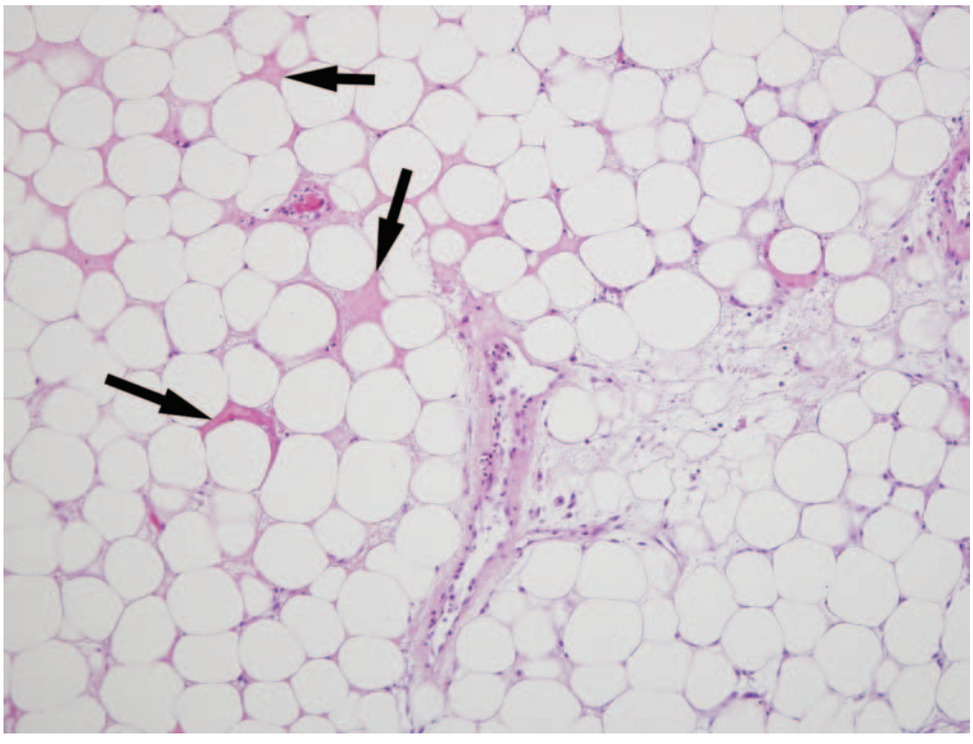

Figure 1.2A. Remote contusion (> 24 hours). Section of subdermal adipose tissue with erythrocyte "laking" (arrows), or loss of erythrocyte borders during close association.

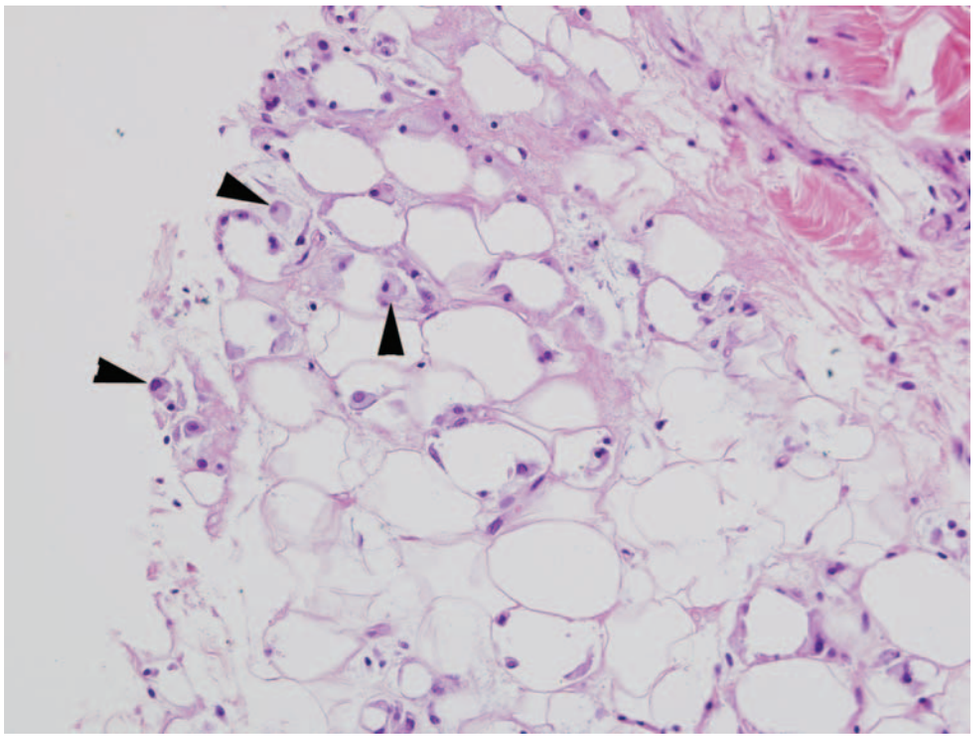

Figure 1.2B. Section of subdermal adipose tissue with numerous foamy macrophages (arrow heads), many of which are visible with stainable iron.

Table 1.1 Contusion ageing.

Time interval	*Histologic appearance*
< 4 hours	- No distinct signs of inflammation - Histological distinction between antemortem and postmortem skin wounds not possible. (Caveat: neutrophilic infiltrates have been reported to appear within 20–30 minutes [1])
4–12 hours	**4 hours**: Some perivascular neutrophils **8–12 hours**: Neutrophils, macrophages, and fibroblasts form a distinct peripheral wound zone. (neutrophils >> macrophages)
12–48 hours	**16–24 hours**: Macrophage infiltrate increases. (macrophages >> neutrophils) **24 hours**: Neutrophils and fibrin deposition at maximum and remain for 2–3 days Cut edge of epidermis shows cytoplasmic processes **24–48 hours**: Epidermis migrates from the edge toward the center of the wound **32 hours**: Necrosis is apparent in central wound zone **48 hours**: Macrophages reach maximum in peripheral wound zone
2–4 days	**2–4 days**: Fibroblasts migrate into wound periphery. Stainable hemosiderin apparent [1][3] **3 days**: Epithelialization of small wounds becomes complete and its stratification is thicker than surrounding epithelium **3–4 days**: Angiogenesis occurs

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Table 1.1 *continued*

Time interval	*Histologic appearance*
4–8 days	**4 days**: New collagen laid down **4–5 days**: Ingrowth of new capillaries, which continues until day 8 **6 days**: Lymphocytes at maximum in peripheral zone **4–8 days**: Copious stainable hemosiderin
8–12 days	- Decrease in number of inflammatory cells, fibroblasts, and capillaries - Increase in the number and size of collagen fibers - Hematoidin becomes apparent
>12 days	- Definite regression of cellular activity in both epidermis and dermis. Vascularity of dermis decreases. Collagen fibers restored and begin to mature and shrink. Epithelium shows definite basement membrane

Table adapted from [2]. Speed of changes are different in different tissues, even in contralateral sites of the same person [3]. Gross and histologic "contusions," or pseudo-contusions, *can* appear after death [1], especially when there is increasing pressure in local vasculature with subsequent rupture and passive extravasation into the surrounding tissues. In these post-mortem pseudo-contusions, there is no inflammatory "vital reaction" seen histologically; however, "the lack of a vital reaction does not imply that the injury occurred postmortem" [1]. Like all things in forensics, these injuries must be correlated with investigatory and gross anatomic findings.

Source:

[1] Langlois, N.E.I., The science behind the quest to determine the age of bruises – a review of the English language literature. *Forensic Sci Med Pathol*, **3** (2007), 241–251.

[2] Raekallio, J., Histologic estimation of the age of injuries. In Perper, J.A., and Wecht, C.H., eds., *Microscopic Diagnosis in Forensic Pathology*. Springfield, IL: Charles C. Thomas, (1980), pp. 3–16.

[3] Vanezis, P.,Interpreting bruises at necropsy. *J Clin Pathol*, **54** (2001), 348–355.

Brain

Subarachnoid hemorrhage dating

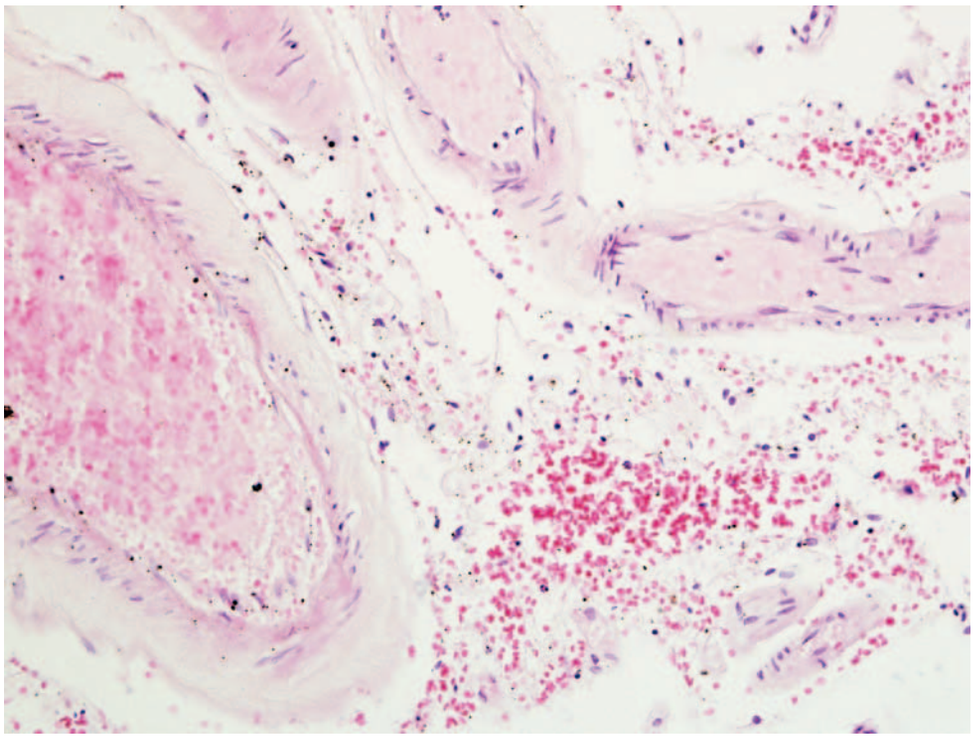

Figure 1.3. Subarachnoid hemorrhage. There is acute hemorrhage in the subarachnoid space. Notice there is no acute inflammatory response and the red blood cell cytoplasmic borders are intact. The age of this lesion is best estimated as less than one hour. After one to four hours neutrophils appear. After four hours the red blood cells begin to lyse.

Table 1.2 Microscopic dating of subarachnoid hemorrhages.

<1 hour	-	Fresh blood in subarachnoid space
1 to 4 hours	-	Occasional neutrophils seen
	-	Some red blood cells begin to break down
	-	Red blood cells begin to creep down the Virchow–Robin spaces
4 to 12 hours	-	Increased neutrophils
	-	Perivascular lymphocytes
	-	Rare macrophages
12 to 24 hours	-	Hemosiderin and fibrin
	-	Increased numbers of lymphocytes and macrophages
24 to 48 hours	-	Increased neutrophils and macrophages
	-	Definite hemosiderin deposition
Up to 3 days	-	Peak neutrophilic infiltrate
Up to 5 days	-	Laking of red blood cells
	-	Increased lymphocytes
	-	Intense fibrin deposition separating islands of red blood cells
	-	Early collagen formation

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Table 1.2 *continued*

Up to 1 week	-	Hemosiderin-laden macrophages - Neutrophils fade away - Some intact red blood cells remain
Up to 10 days	-	Fibrosis - Breakdown of red blood cells nears completion (this can take up to 20 days)
Up to 2 weeks	-	Continued break down of red blood cells - Macrophages with hematoidin - Increased organization with additional fibrin, collagen and phagocytosis
Up to 4 weeks	-	Rebleeding - Meningeal reactive changes - Variable amounts of mixed inflammatory cells
After 1 month	-	Macrophages and hemosiderin still present sometimes for years

Subdural hematoma dating

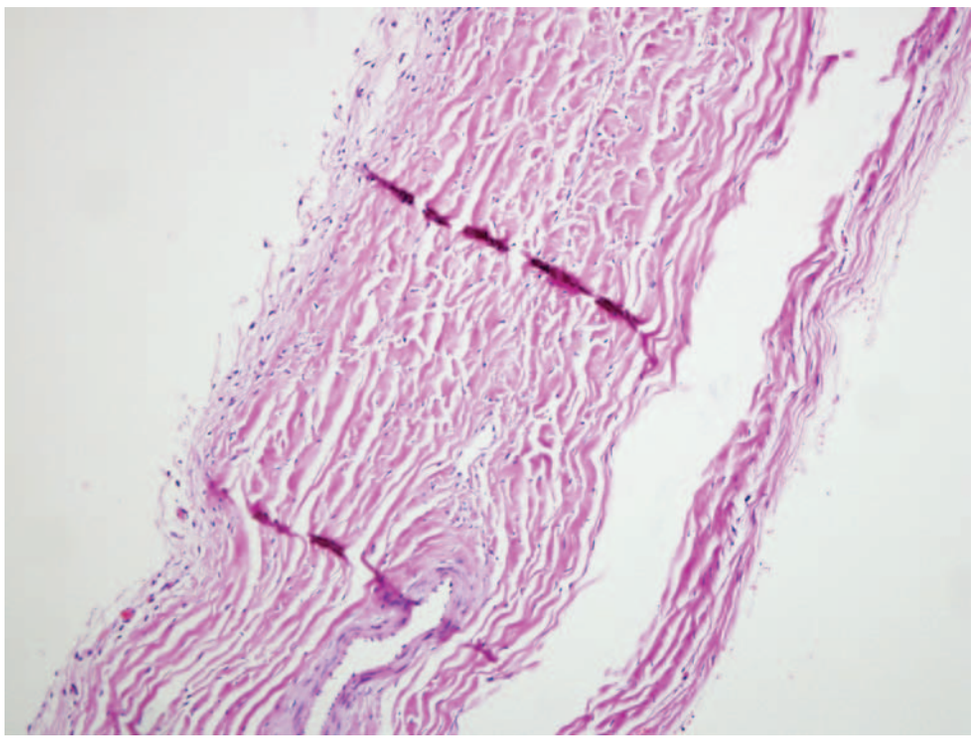

Figure 1.4. Normal baby dura. This section of dura was taken at a site distant from the superior sagittal sinus. If the section is taken too close to the sagittal sinus the intradural blood normally found at that site could be confused with a subdural hemorrhage (compare with Figure 1.5).

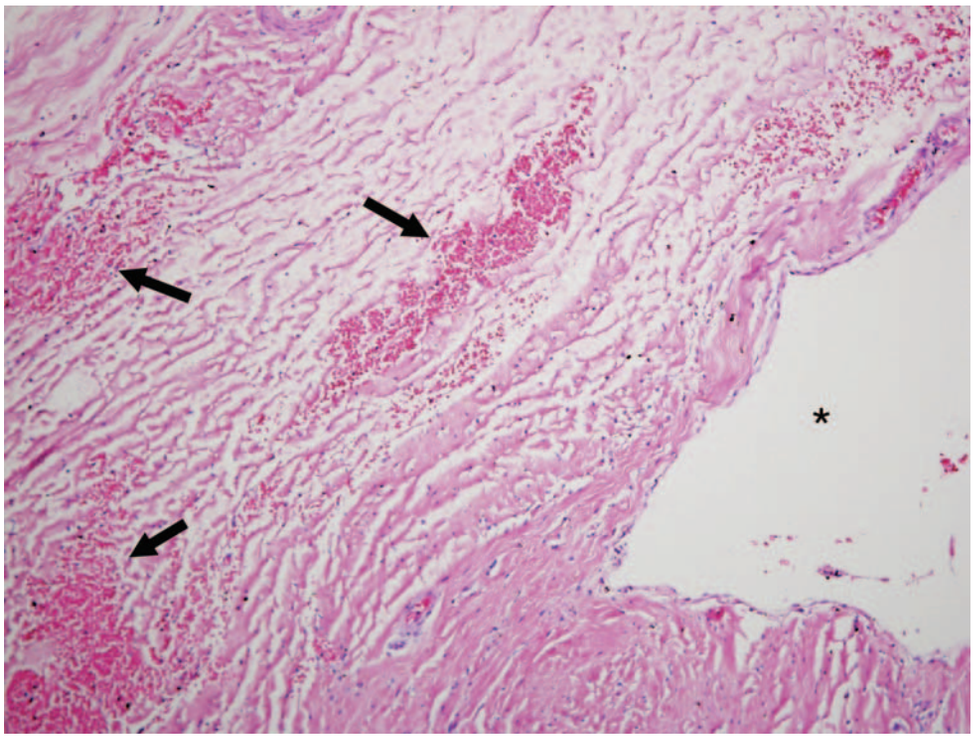

Figure 1.5. Normal baby dura. This section is taken close to the superior sagittal sinus (asterisk). Note the foci of acute hemorrhage (arrows) (compare with Figure 1.4).

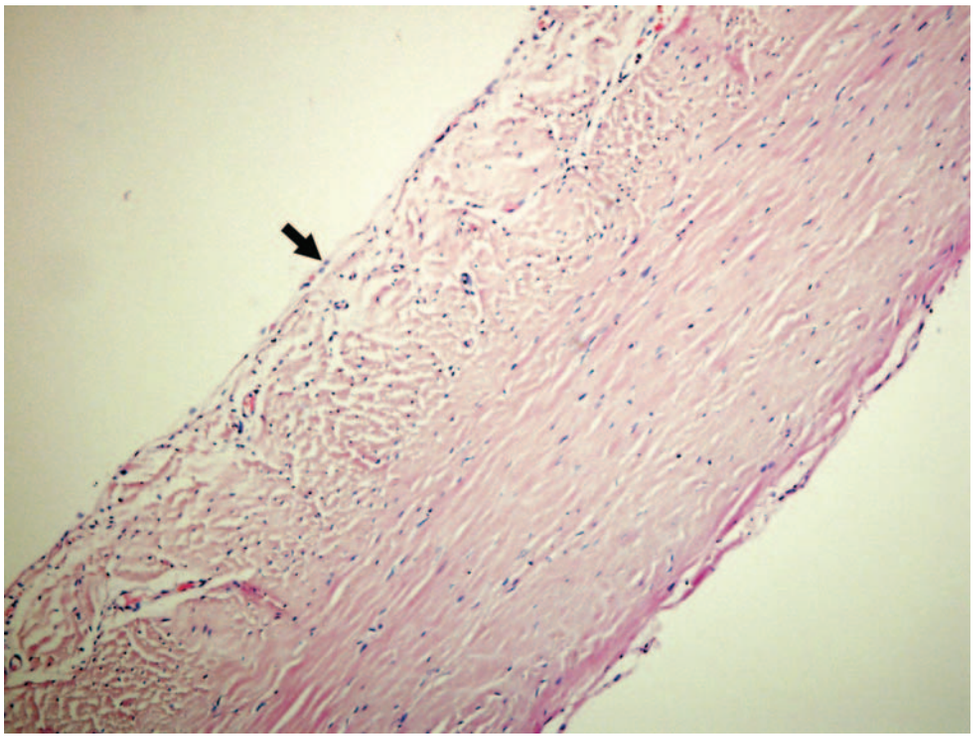

Figure 1.6. Normal adult dura. The meningeal artery designates the periosteal side of the dura (not seen in this section). The dura is comprised of dense fibrotic tissue with sparse spindle-shaped cells. There are small capillaries on the inside of the dura. The thin single-cell layer represents the dura border cells (arrow) that are in contact with the arachnoid barrier cells. This region is the origin of subdural hemorrhages.

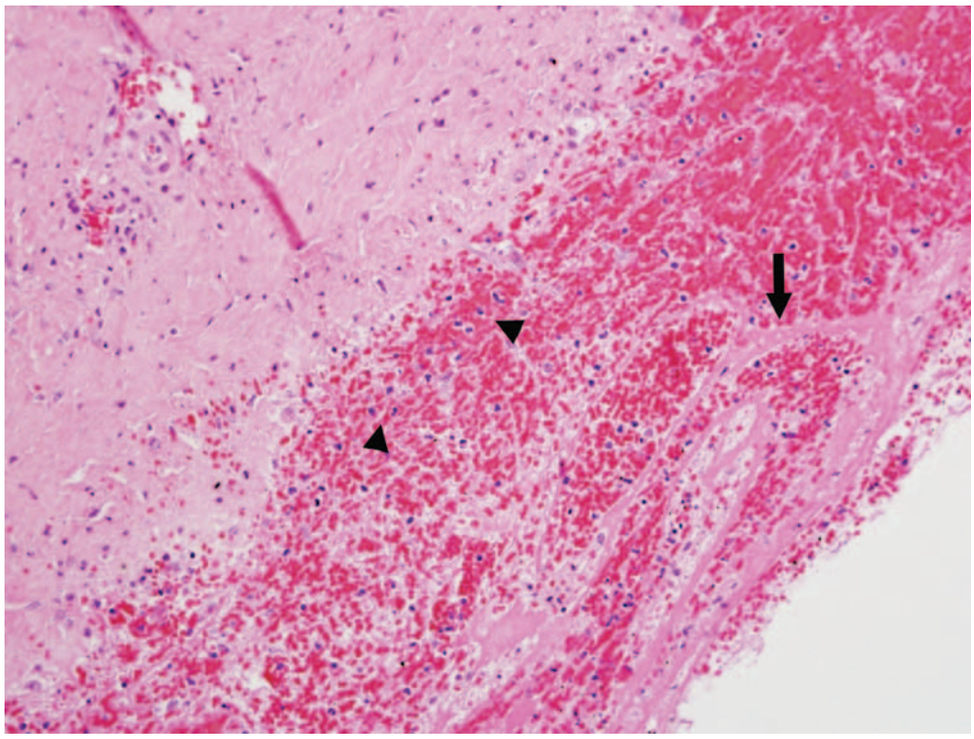

Figure 1.7. Acute subdural hemorrhage: 24 to 48 hours. There is acute hemorrhage with fibrin infiltration (arrow). A number of neutrophils are seen and are greater in number close to the dura–hemorrhage interface (arrowheads). Notice the red blood cells are intact.

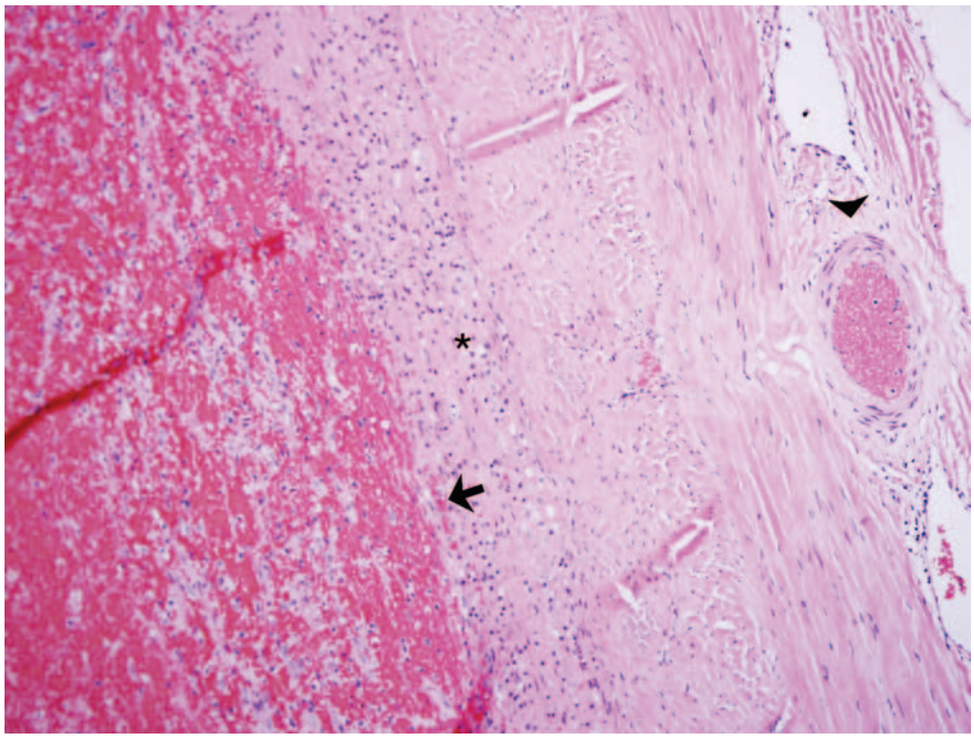

Figure 1.8A. Subdural hematoma: 3 to 4 days. The meningeal artery designates the periosteal side of the dura (arrow head). There is a fibroblast layer forming at the clot–dura interface that is beginning to creep into the hemorrhage (asterisk). Notice the small- caliber blood vessels characteristic of neovascularization (arrow). The red blood cells have begun to lose their distinct cytoplasmic borders as they begin to degenerate. A higher magnification of this same region can be seen in Figure 1.8B.

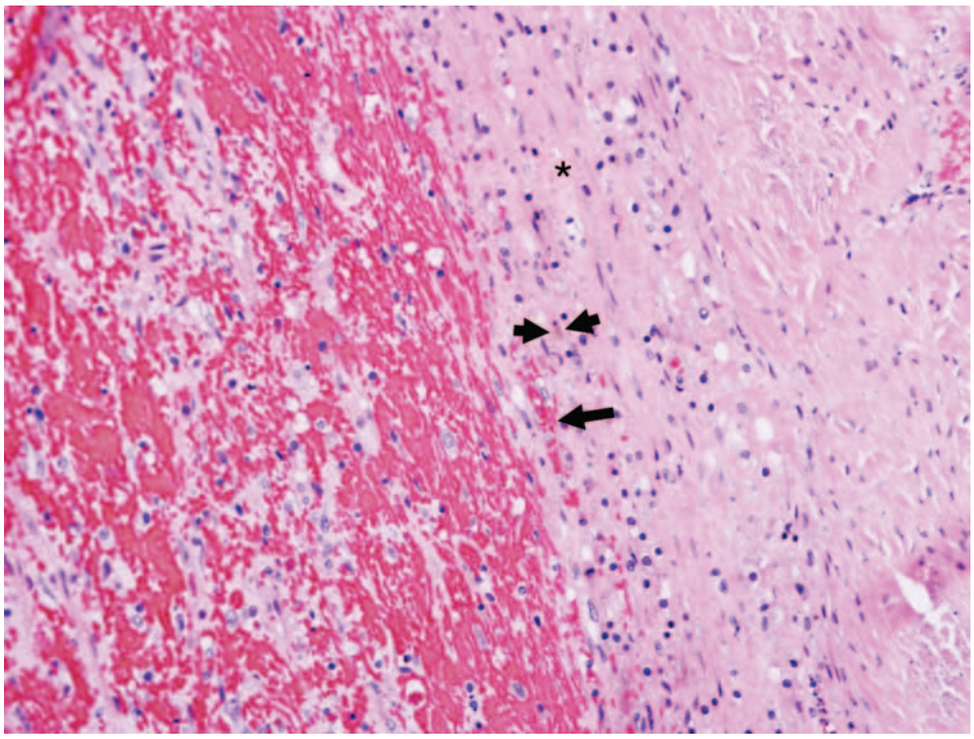

Figure 1.8B. At this level of magnification hemosiderin can be seen (double arrow). There is a fibroblast layer forming at the clot–dural interface that is beginning to creep into the hemorrhage (asterisk). Notice the small-caliber blood vessels characteristic of neovascularization (arrow).

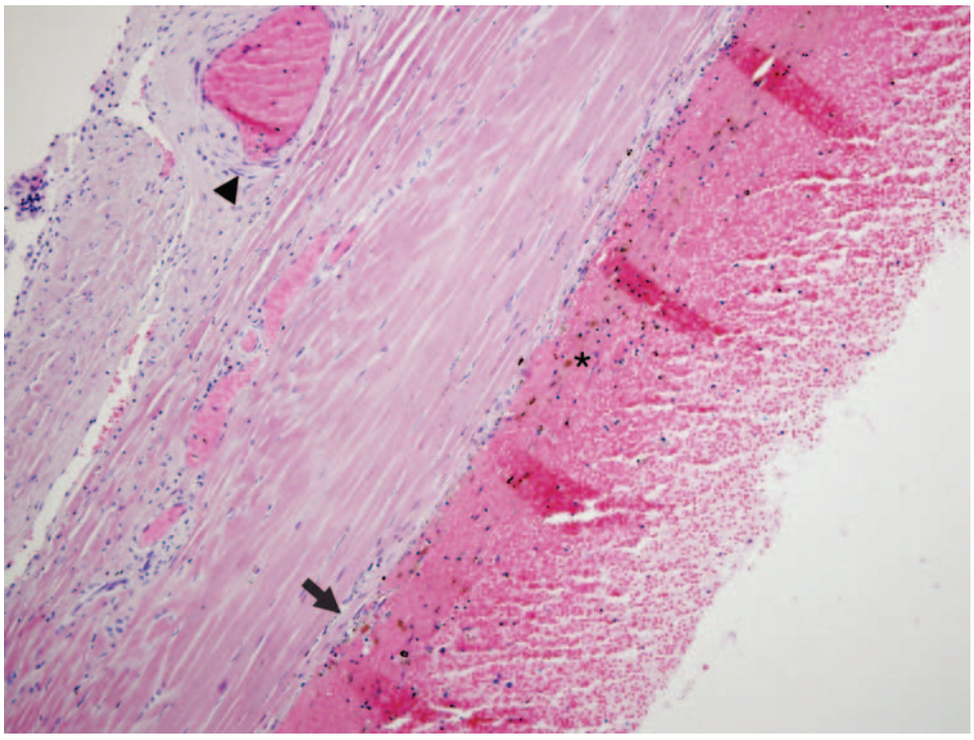

Figure 1.9. Subdural hematoma: 3 to 4 days. The meningeal artery designates the periosteal side (arrow head). There is acute hemorrhage on the subdural side of the dura. There is an early proliferation of fibroblasts, which is forming a layer approximately two cells thick (arrow). Notice the scant fibroblastic infiltrate into the area of hemorrhage (also seen under the arrow). Also, the red blood cells are intact and there is rare hemosiderin deposition (asterisk).

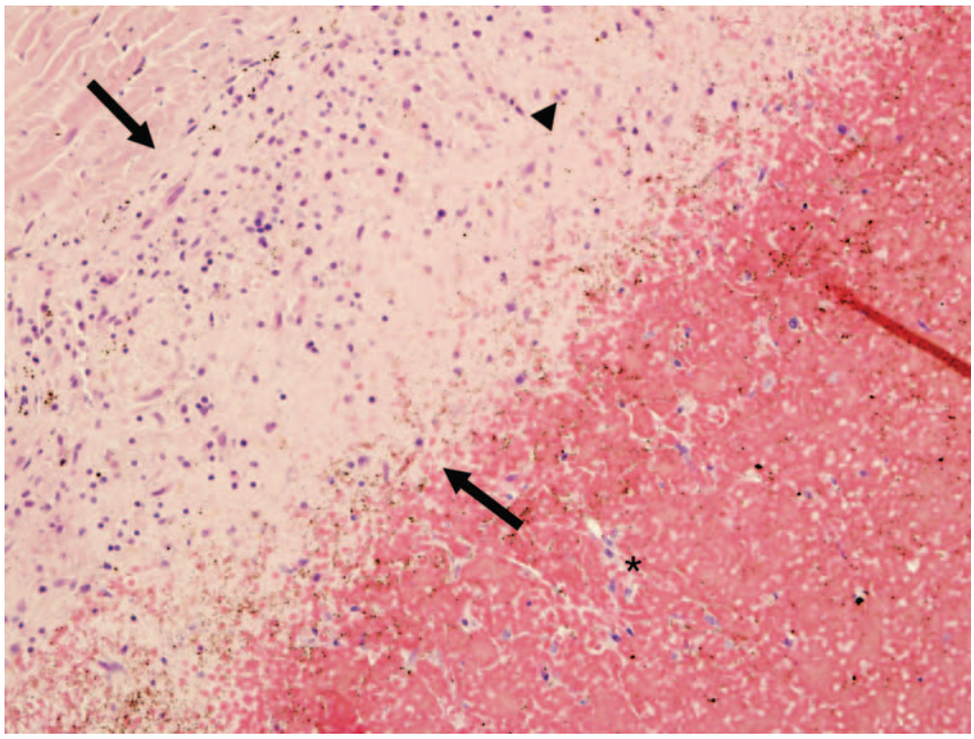

Figure 1.10. Subdural hematoma: 7 days. The fibroblast layer is very thick (between arrows), greater than ten cells in thickness, and there is some migration into the clot (asterisk). Macrophages are present and some contain hemosiderin (arrowhead). The red blood cells have become pale and have lost their distinct cellular borders.

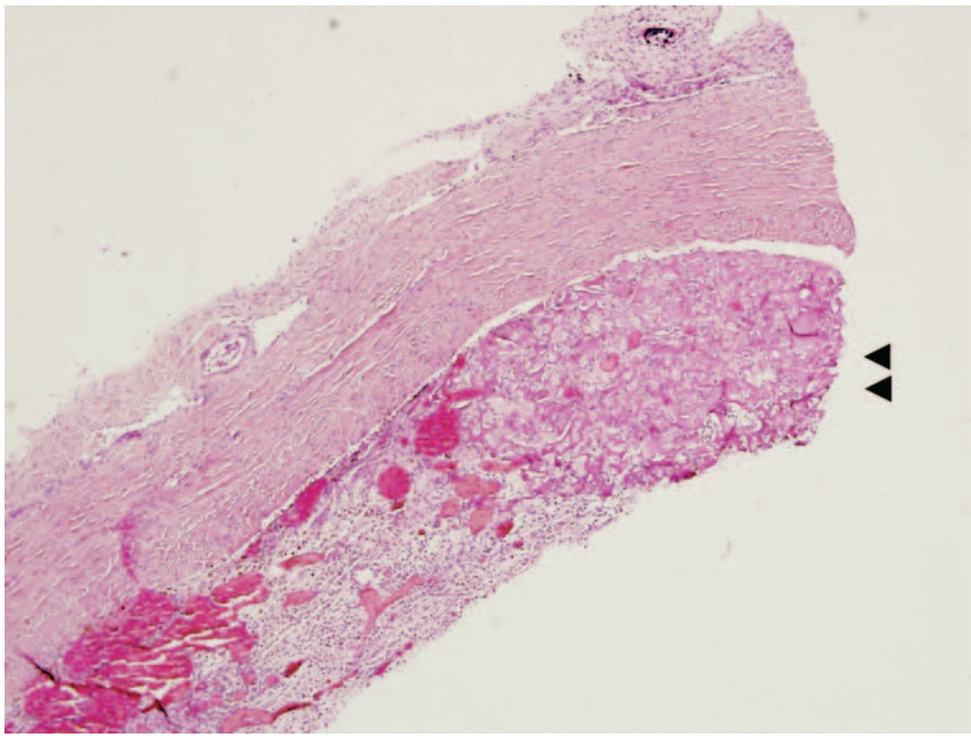

Figure 1.11A. Subdural hematoma: 2 to 3 weeks. The thickness of the neomembrane is close to that of the native dura. Also note the fragment of DuraGel®present at one end of the section (double arrowhead).