

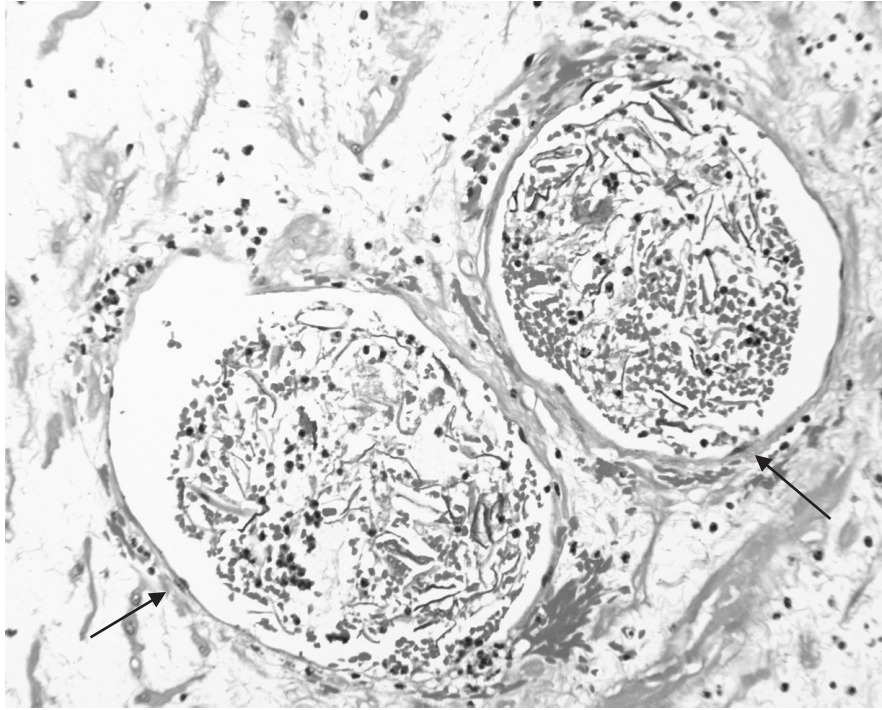


Figure 7 Amniotic debris in venules (arrows) of cervical stroma following a small endocervical tear in labor. Postpartum hemorrhage and disseminated intravascular coagulopathy necessitated hysterectomy (×20)

CERVIX

Important pathologies in the cervix include tears. Small shallow endocervical tears are almost invariably found in the postpartum uterus, and may be present even in those cases where there has been a Cesarean section. Significant and deep tears tend to be lateral in location. These tears may penetrate through to the serosa, with or without hematoma formation, and may extend up into the lower segment or down the cervix into the vagina. Involvement of large uterine arteries should be sought. It is common to find meconium staining of the mucus of the endocervix with fetal distress, and meconium may contaminate the tear. A tear may have severe consequences: an endocervical tear may cause severe blood loss despite a fully contracted uterus. Tears are associated with amniotic fluid embolus or with amniotic infusion and local defibrination. Bleeding can extend into the broad ligament with formation of a large hematoma. Suturing of the tear may not prevent a deep hematoma from

forming and secondary rupture can result in shock, despite cessation of external vaginal hemorrhage.

In the dilated postpartum cervix, edema, hemorrhage and fiber disarray may make it difficult to identify tears on histologic examination. Torn and contracted muscle fibers and torn arteries with fibrin plugs and tense hematomas provide corroboratory evidence of a tear. Histologic sampling should include blocks from above the apex and from below the tear for deep extension and for identification of large torn vessels.

Examination of the uterus histologically following amniotic fluid embolism will show no evidence of intravascular disease in most cases. Very occasionally, there may be fibrin clots adherent to vascular endothelium and, rarely, squames admixed with fibrin have been found in vessels in the body of the uterus. In some cases of postpartum hemorrhage, when there have been no clinical features of amniotic infusion but bleeding and unexpected severe onset of consumptive coagulopathy, histological

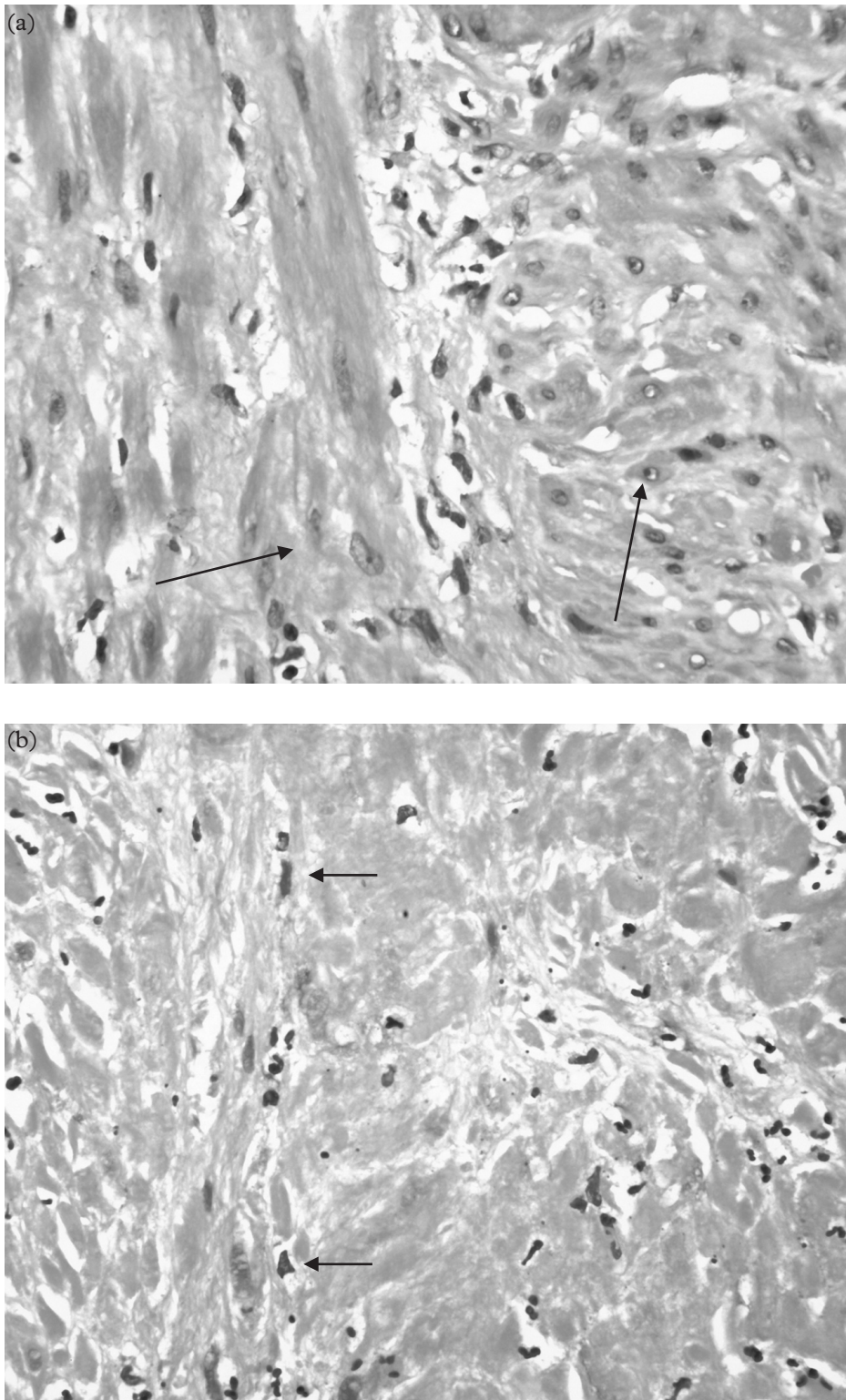


Figure 8 H/E comparison of (a) normal myometrial fibers and (b) myonecrosis in lower uterine segment in hysterectomy specimen for postpartum hemorrhage following Cesarean section (×40). Long arrows, normal viable cell nuclei; short arrows, non-viable necrotic cells

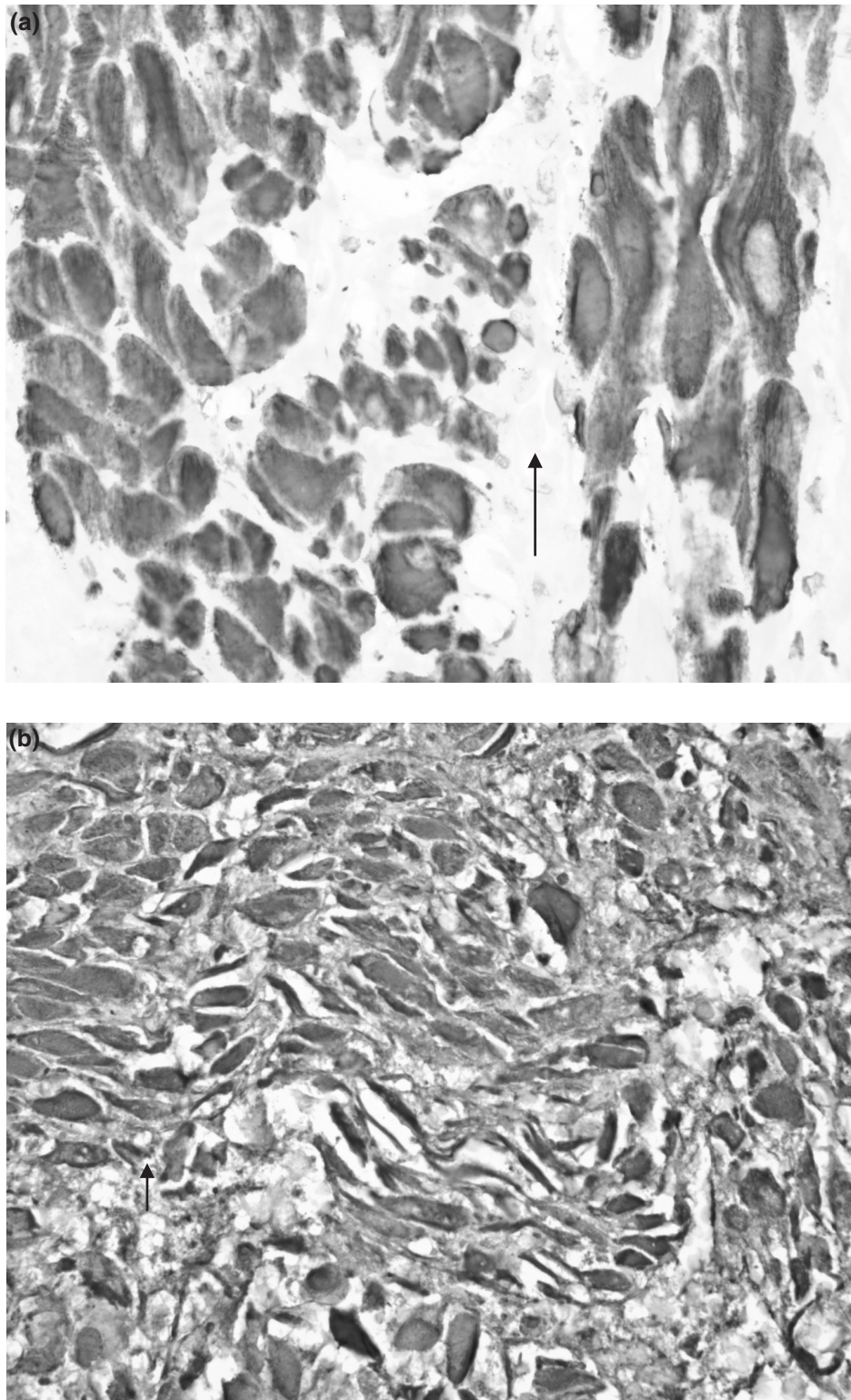


Figure 9 Desmin comparison of same myometrial fibers accentuates the necrosis. (a) Normal; (b) myonecrosis ($\times 40$). Long arrow, normal myometrial cells with intercellular edema; short arrow, dense, compacted necrotic myometrial cells at same magnification

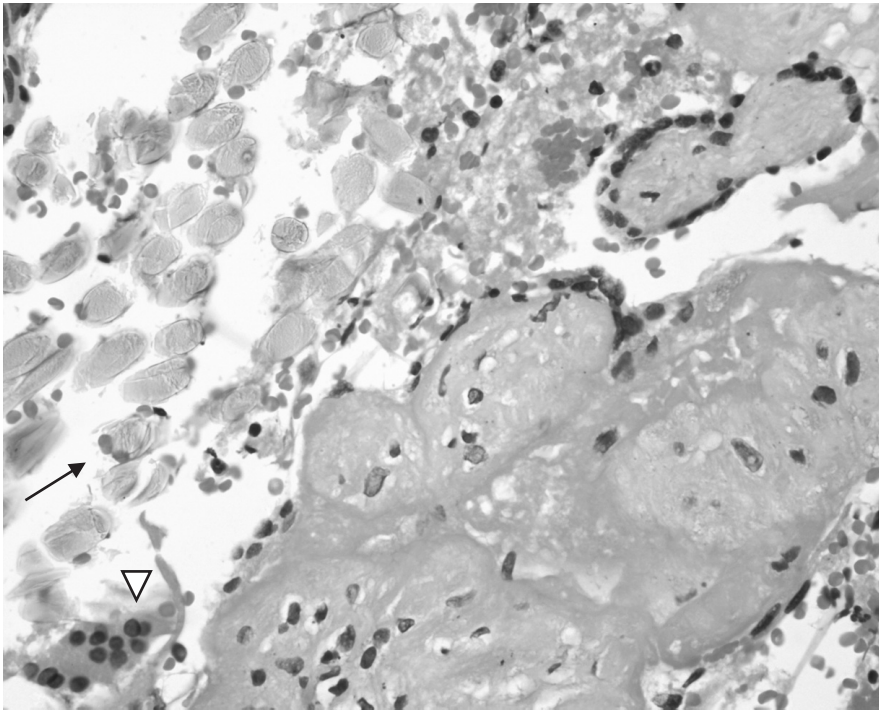


Figure 10 H/E section showing stitch material in uterine curettings following Cesarean section. Arrow, absorbable suture; arrowhead, giant cell reaction to suture material ($\times 40$)

sections of the endocervix will reveal localized areas where amniotic debris fills and expands venules and capillaries. This dramatic appearance is present not just adjacent to the endocervical surface and tears: its presence deeper in the stroma distinguishes it from contamination of the surface mucosa by meconium and amniotic fluid at delivery.

A subgroup of patients have a lesion of local amniotic infusion associated with disseminated intravascular coagulopathy and postpartum hemorrhage without systemic collapse. Squamous cells may be present in only one or two sections taken from around the circumference of the cervix. It is usually on one side. Extensive sampling of the cervix may be required to demonstrate amniotic debris in cases of suspected amniotic fluid embolism². It is possible that ongoing blood loss from a tear in this site may occur before the onset of systemic disseminated intravascular coagulopathy, because local thromboplastin effect alone of the amniotic debris in the wound may inhibit hemostasis.

LOWER UTERINE SEGMENT

Important pathologies here involve implantation on a previous Cesarean section scar, with abnormal adherence or formation of a diverticulum.

A Cesarean section results in chronic changes in the lower uterine segment, including distortion and widening, inflammation, giant cell reaction and adenomyosis³. In some cases, a distinctive V-shaped defect of the anterior wall ('tenting') may be present.

An important cause of weakening of a Cesarean section scar is infection. Postoperative wound infection is not uncommon following Cesarean section, particularly emergency section. Prophylactic antibiotics can modify the extent and rate of infection, as can the quality of closure, the amount of local tissue trauma, the technique used (one- or two-layer), swelling, hematoma and the nature of the organisms infecting the wound. There may be extensive disruption and inflammation in the uterine wall despite a normal healing appearance of the skin wound. Conservative treatment of the wound