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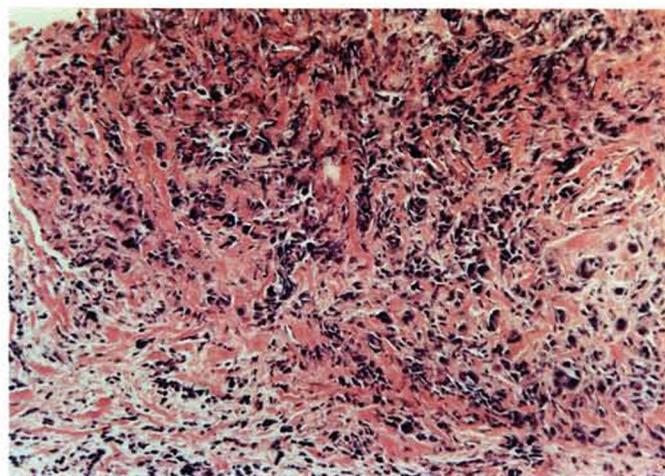
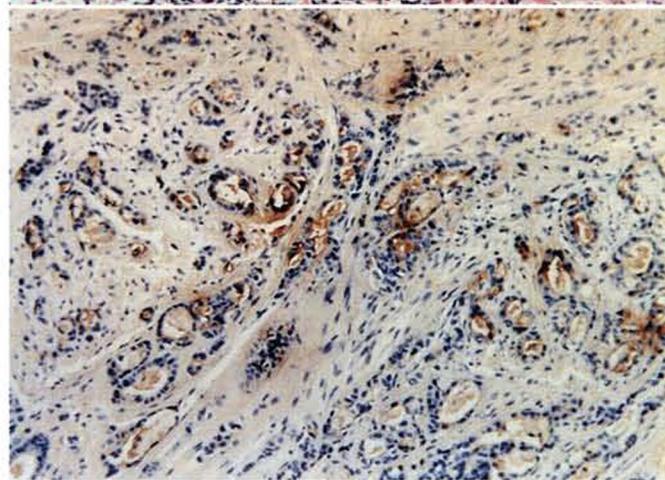
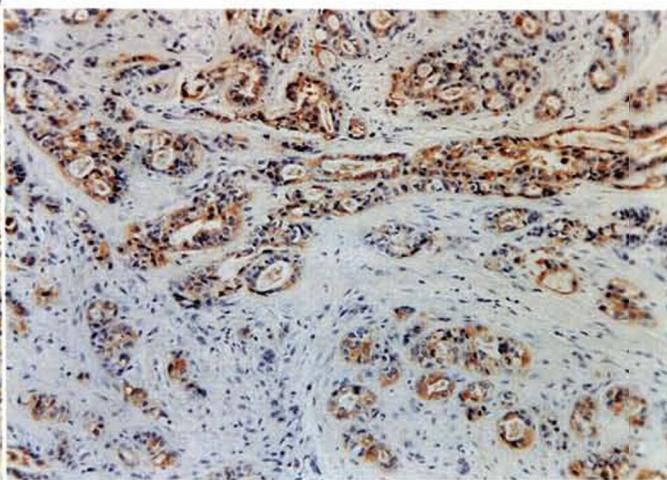


Fig. 1. Representative CT scan shows right adrenal mass. The mass was nonhomogeneous and encapsulated.

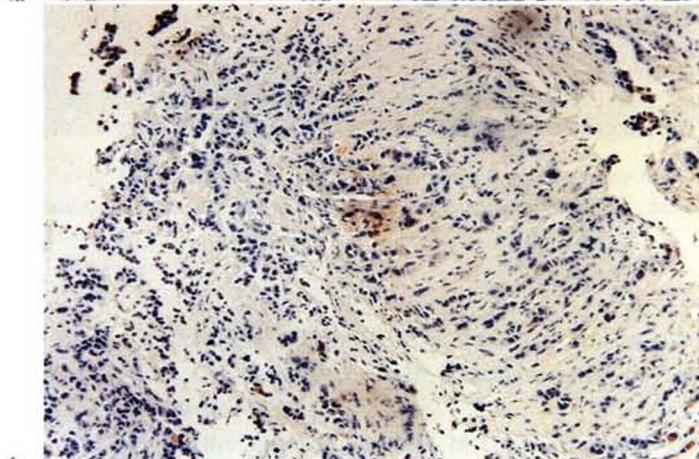
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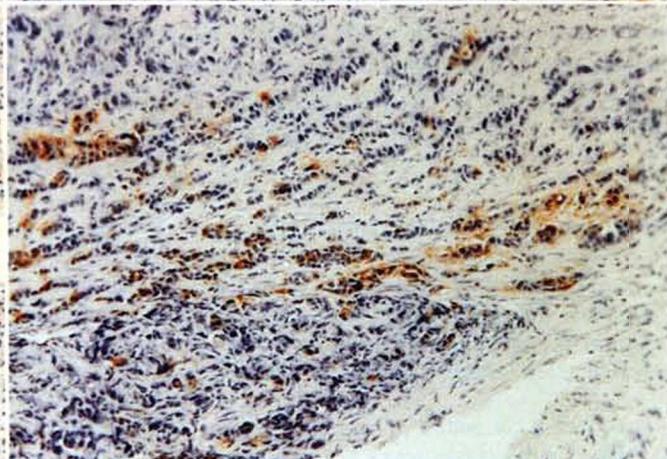
4a



4b



4c



4d

Fig. 4. Both adenocarcinomas of the penis and the prostate demonstrate positive CA19-9 and CEA immunoperoxidase stain. **a** Penis, CA19-9. **b** Penis, CEA. **c** Prostate, CA19-9. **d** Prostate, CEA.

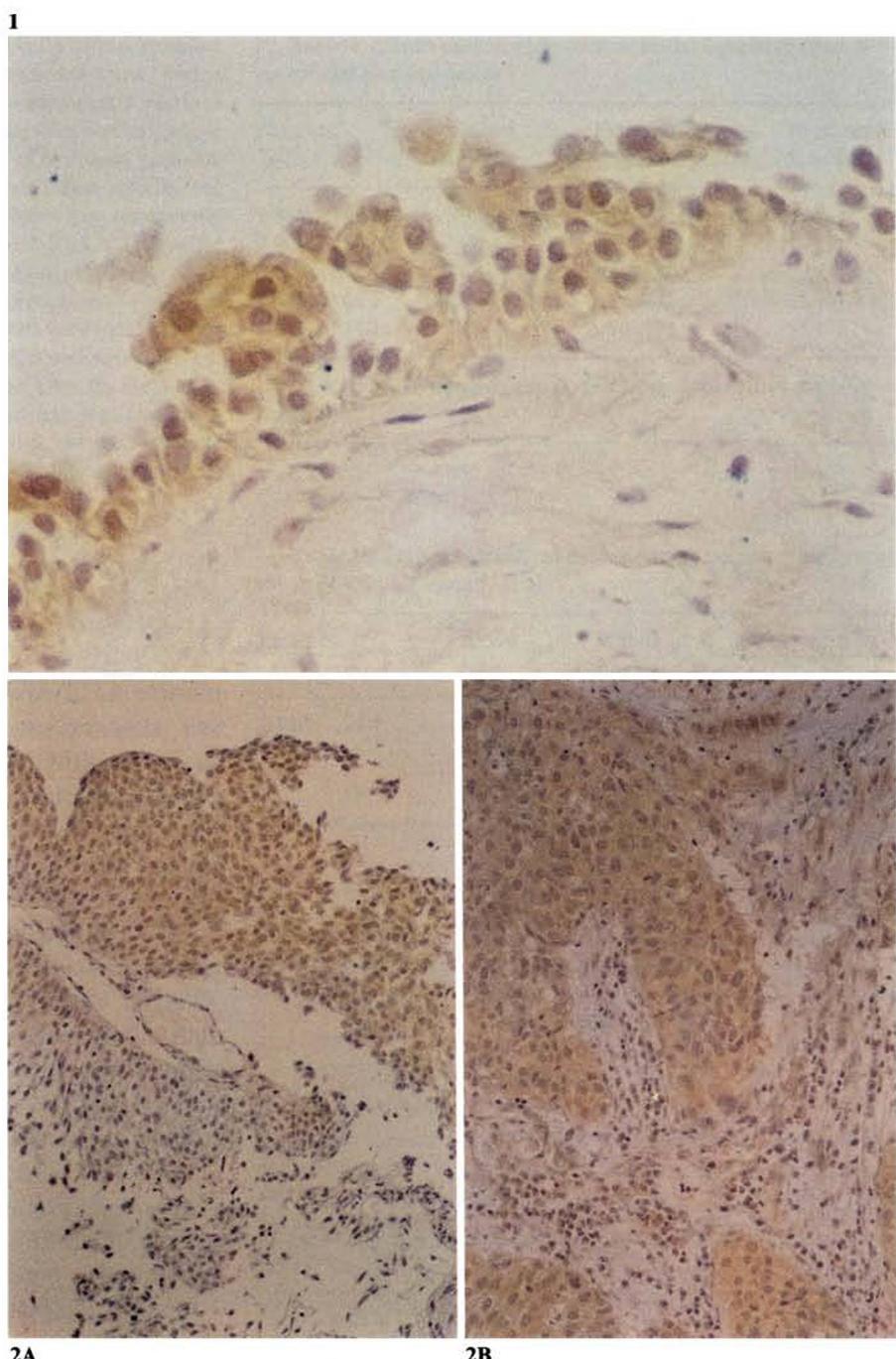
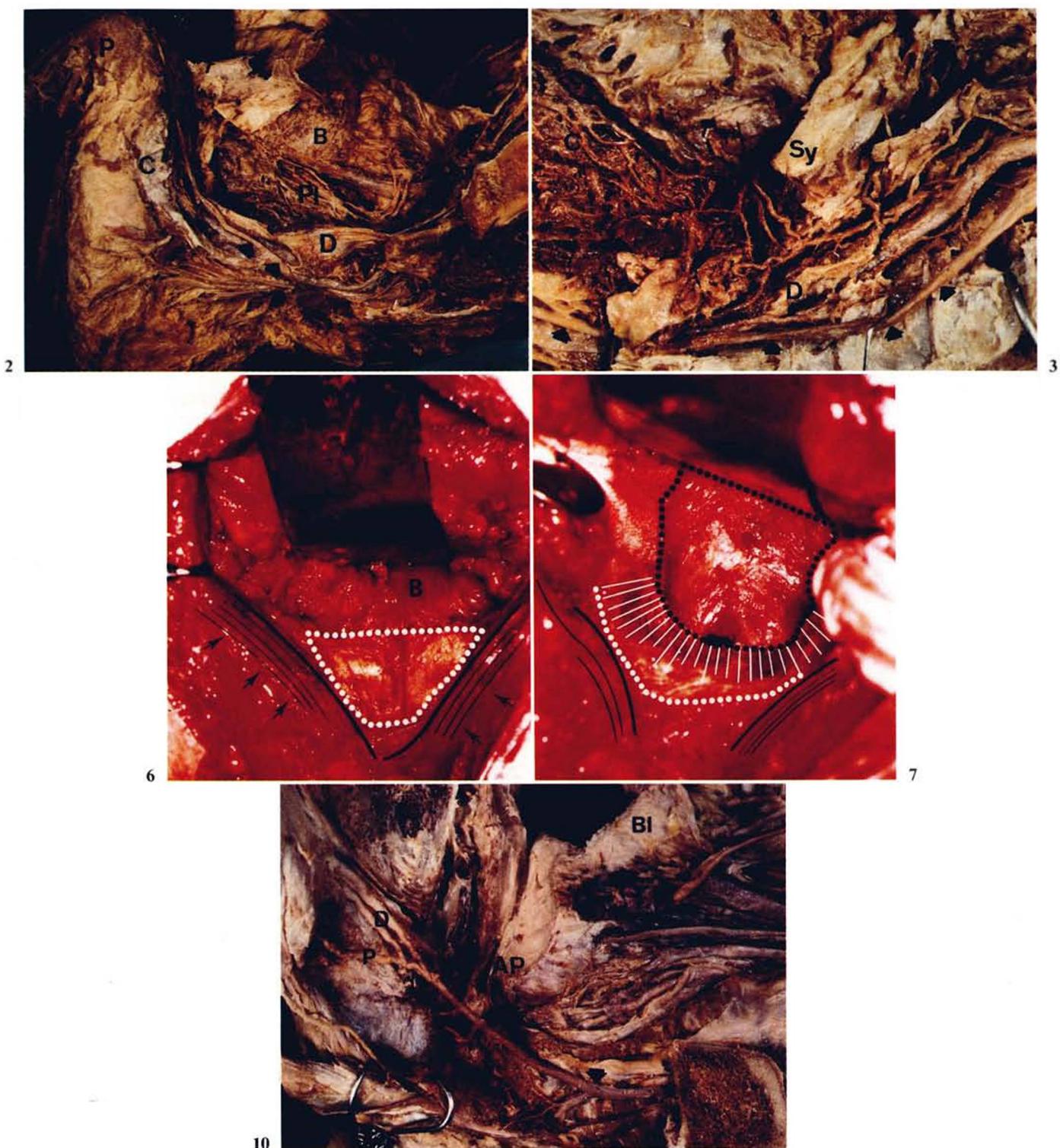


Fig. 1. Atypical cell layers showed positive staining. Submucosa, interstitial cells and blood vessels were not stained. Counterstaining was performed with hematoxylin. Case 5. $\times 450$.

Fig. 2. A Upper half layers of the superficial transitional cell carcinoma (G2) was stained with ras-p21. Case 1. $\times 112$. B Cytoplasms of G3 invasive tumor were diffusely stained with ras-p21. Case 6. $\times 112$.



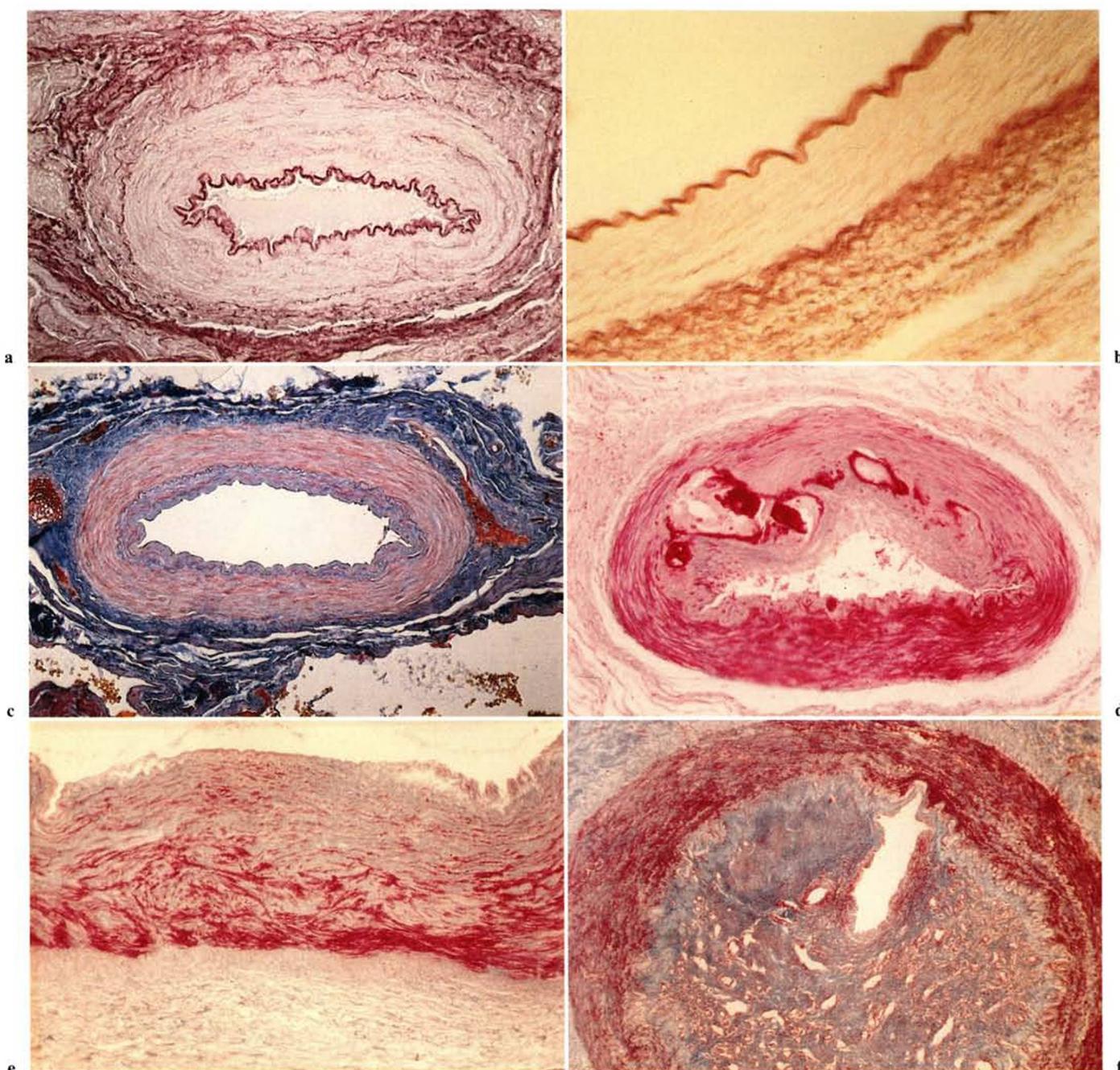


Fig. 2. Penile arteries and their collateral branches. **a–c** Normal arteries. **a** Dorsal artery, 39-year-old. Weigert for elastic tissue. $\times 80$. **b** Dorsal artery, 26-year-old. Weigert for elastic tissue. $\times 120$. **c** Penis dorsal artery, 45-year-old. Ladewig. $\times 80$. **d–f** Three penile arteries, more or less severely altered by sclerosis. **d** Deep artery branch, 22-year-old. Azan. $\times 80$. The lower half of the circumference looks fairly normal. The upper half of the circumference shows quite serious signs of sclerosis and of tissue destruction. Subject with

serious impotence problems. **e** Dorsal artery, 26-year-old. Azan. $\times 140$. At the level of the media, the smooth muscle is greatly reduced, the fibrocollagenous tissue predominates. Subject with quite serious impotence problems. **f** Dorsal artery, 36-year-old. Ladewig. $\times 80$. The arterial lumen is reduced to a narrow slit; very serious sclerotic proliferation of the intima, reproducing the endoarteritis obliterans image. Subject with very serious impotence problems.

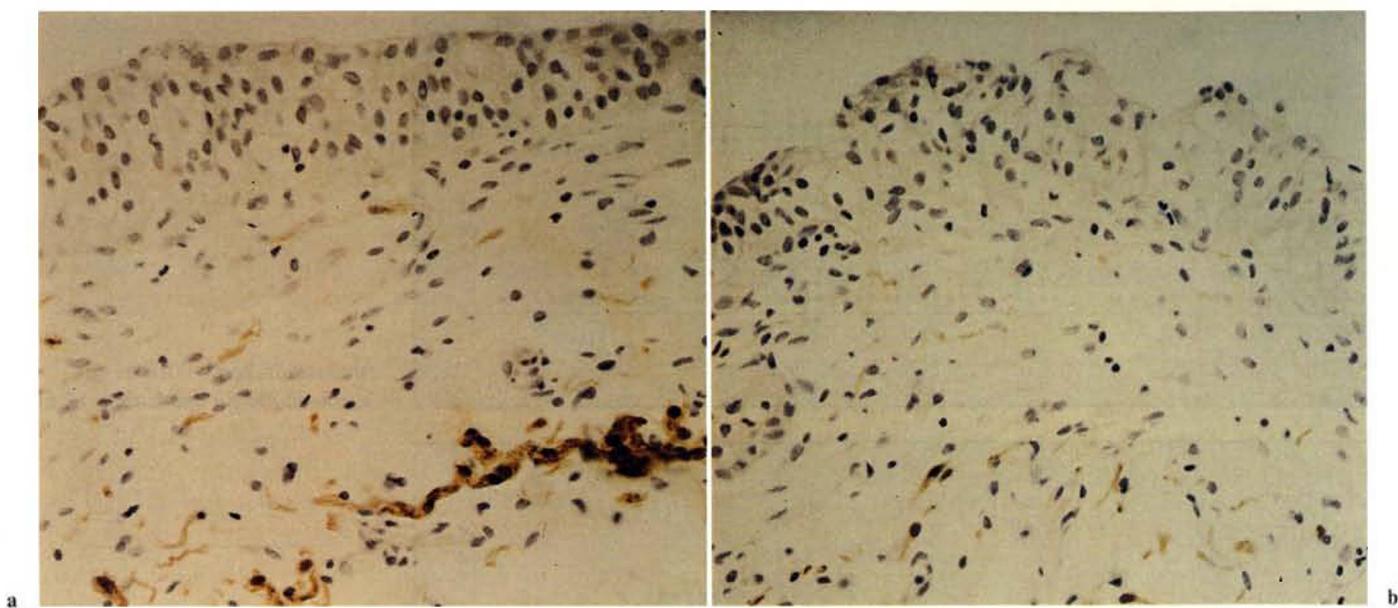


Fig. 1. MS bladder biopsy stained for S100 with increased S100 density (a) and decreased S100 density (b). $\times 400$.

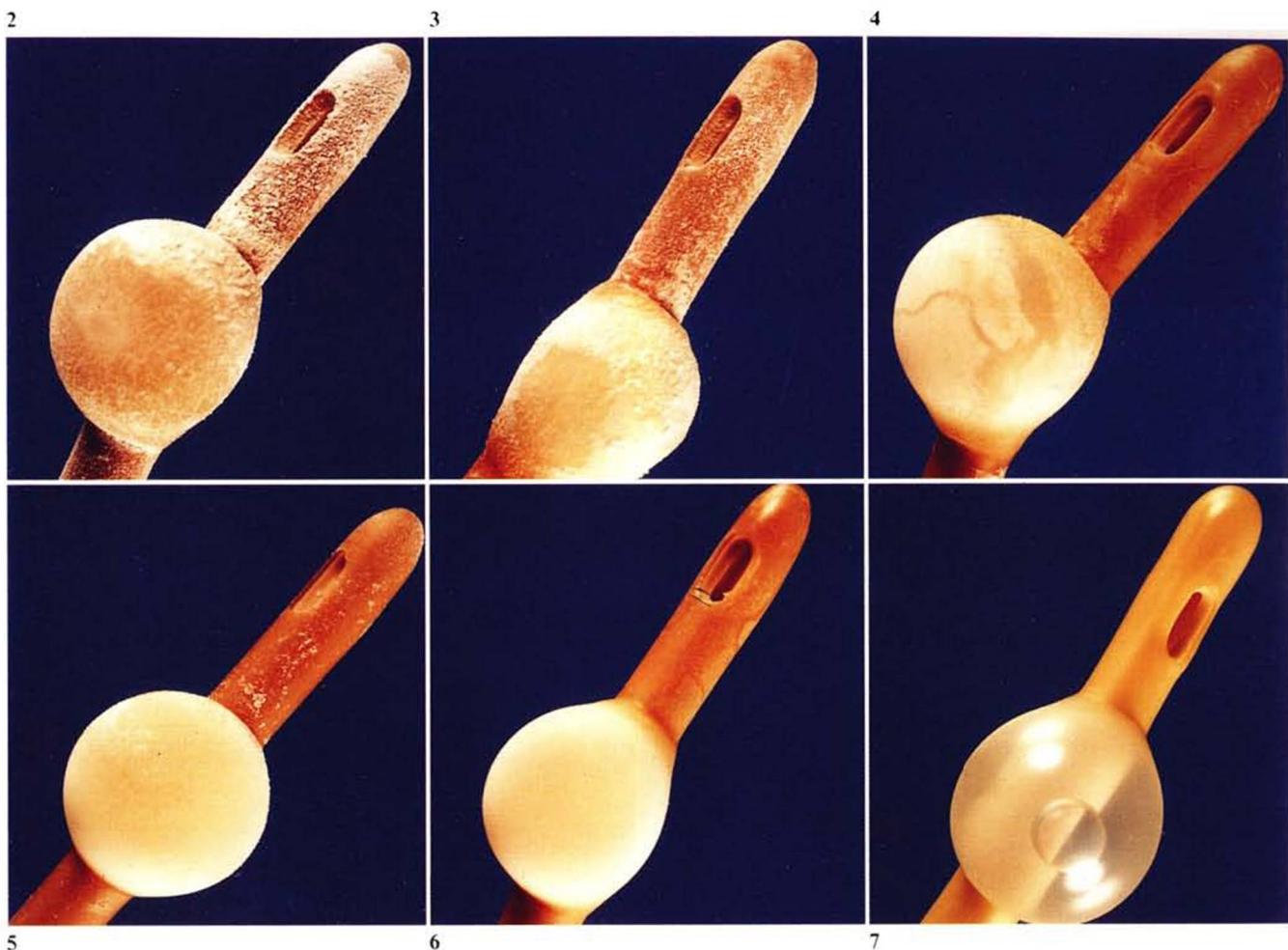


Fig. 2. Major incrustation after 7 days of irrigation with urine (circulation 1).

Fig. 3. Major incrustation after 7 days of irrigation with urine and daily irrigation with NaCl solution (circulation 2).

Fig. 4. Gelatinous coat after dissolution of the major incrustations (blank experiment analogous to fig. 3) at the end of the 7-day cycle after irrigation twice with citrate solution (Suby G, circulation 3).

Fig. 5. Daily irrigation with citrate solution (total 6 times). On the 7th day (end of the experiment), no irrigation, slight crystal deposits (circulation 4).

Fig. 6. Daily irrigation with citrate solution (total of 7 times, i.e. also at the end of experiment). No crystals detectable (circulation 5).

Fig. 7. Untreated silicon-coated latex catheter.

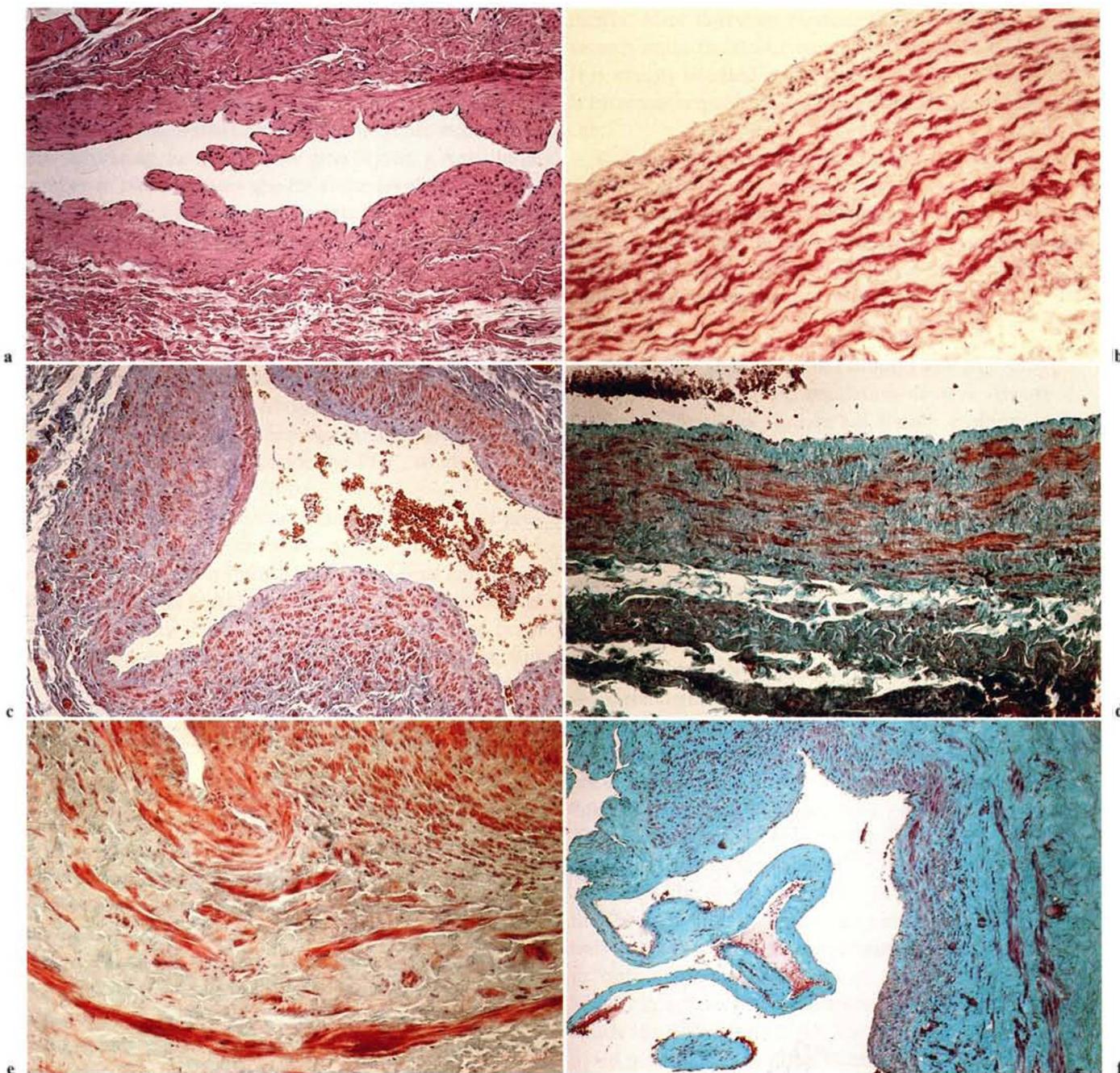


Fig. 3. Penile veins and their collateral branches. **a-c** Normal penile veins with a fairly large muscle quantity. **a** Superficial dorsal vein, 47-year-old in the venous lumen one valve may be seen. HE. $\times 80$. **b** Deep dorsal vein, 24-year-old. Weigert. $\times 120$. **c** Superficial dorsal vein, 37-year-old. Ladewig. $\times 80$. **d-f** Penile veins with different degrees of sclerosis. **d** Superficial dorsal vein, 26-year-old.

The media is rather sclerotic. No impotence problems. Goldner. $\times 70$. **e** Superficial dorsal vein, 34-year-old. The media is strongly sclerotic. Serious impotence problems. Goldner. $\times 80$. **f** Superficial dorsal vein, 33-year-old. The venous wall as well as the flap of the venous valve visible in the lumen are completely sclerotic: very serious impotence problems. Goldner. $\times 80$.

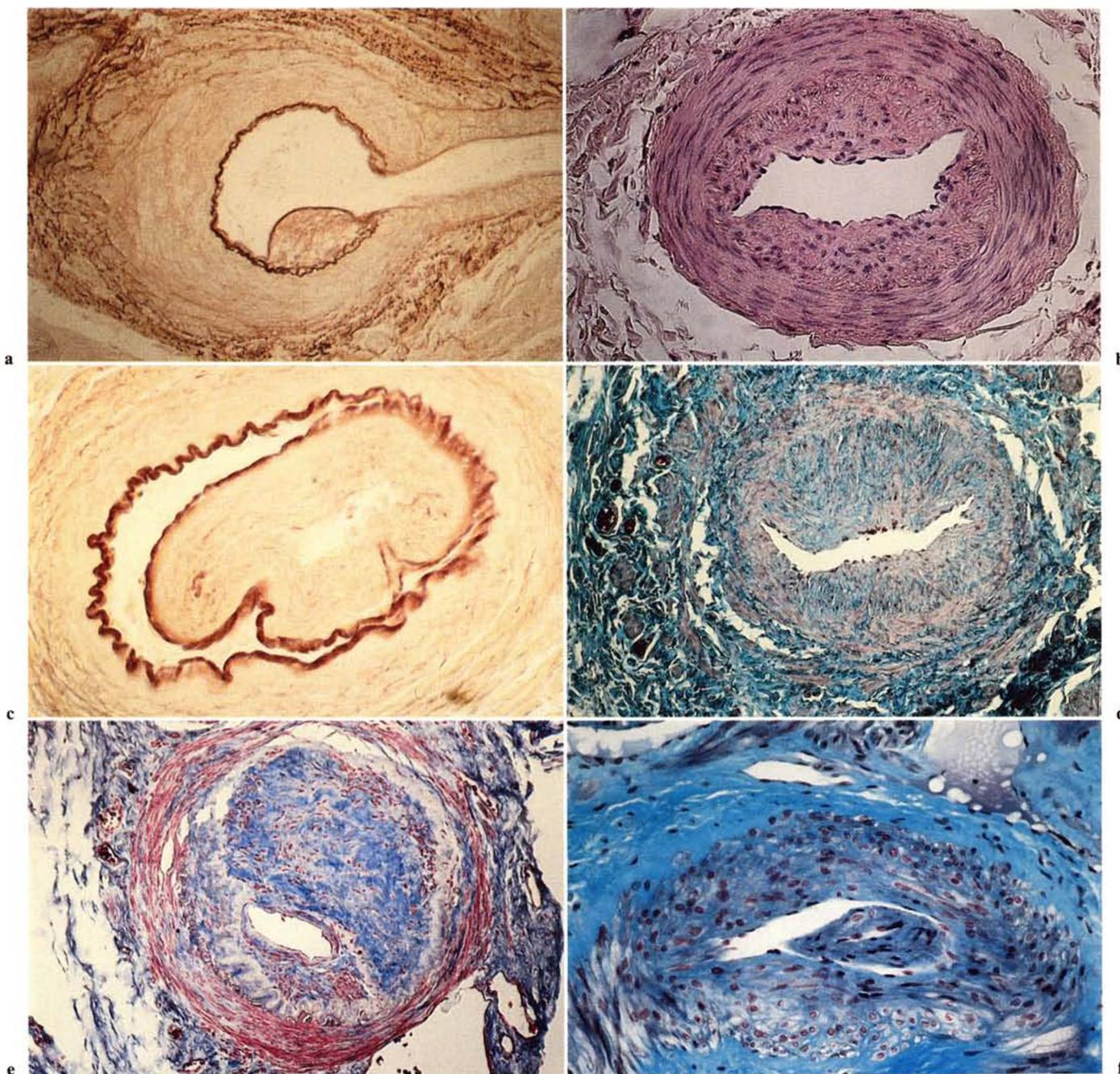
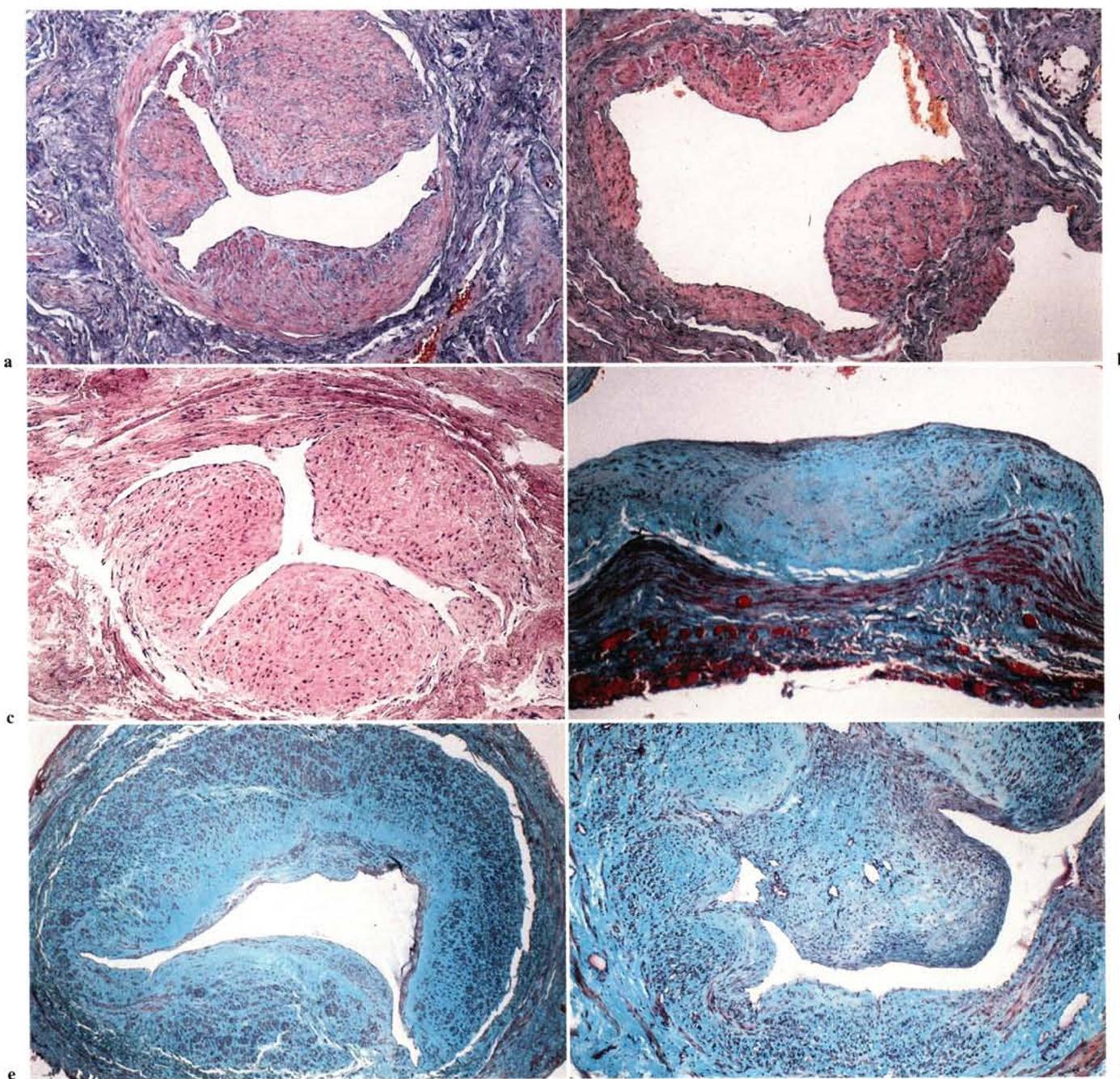


Fig. 4. Penile arterial regulation mechanisms. a-c The cushions look completely normal and the subjects do not show any impotence problems. d-f Cushions of the corresponding type are shown, but affected by sclerosis in subjects affected by more or less serious impotence problems. a Intima cushion, deep artery branch, 36-year-old. Elastic fibre staining. $\times 230$. b Closing mechanisms, made up of longitudinal muscle tissue, disposed in two cushions of the intima: the muscle tissue is relaxed, the arterial lumen is wide open, 55-year-old. HE. $\times 240$. c Polypoid cushion seemingly free in the lumen, in a subject not affected by impotence problems.

Deep artery branch, 38-year-old. Elastic fibre staining. $\times 240$. d Intima cushions in a deep artery branch. The cushions are sclerotic; subject affected by serious impotence problems, 36-year-old. Goldner. $\times 180$. e Same type of cushion as in b. The arteriole wall is affected by serious sclerosis; the cushion is not functional. Moderate impotence problems, 52-year-old. Ladewig. $\times 240$. f Same type of cushion as in c. The arteriole wall as well as the polypoid cushion are affected by a quite serious sclerosis. The cushion is not functional. Moderate impotence problems: 47-year-old. Goldner. $\times 240$.



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Fig. 5. Penile venous regulation mechanisms. **a-c** Completely normal and functional. **d-f** Affected by sclerosis: they are no longer functional. **a** Superficial dorsal vein, three functional cushions made up of longitudinal muscle tissue in the intima, 34-year-old. Ladewig. $\times 120$. **b** Deep dorsal vein branch, two functional cushions rich in muscle tissue, 55-year-old. Ladewig. $\times 180$. **c** Deep dorsal vein branch, three functional cushions of the intima rich in muscle tissue, functional, 60-year-old. HE. $\times 120$. **d** One intima

cushion along the superficial dorsal vein, completely sclerotic. Impotence problems, 44-year-old. Goldner. $\times 120$. **e** Deep dorsal vein. The venous wall is deficient in muscle tissue, the intima cushions are almost completely sclerotic. Subject affected by venous leakage, 53-year-old. Goldner. $\times 150$. **f** Superficial dorsal vein. The venous wall as well as the voluminous cushion of the intima are completely sclerotic. Total impotence, 46-year-old. Goldner. $\times 120$.

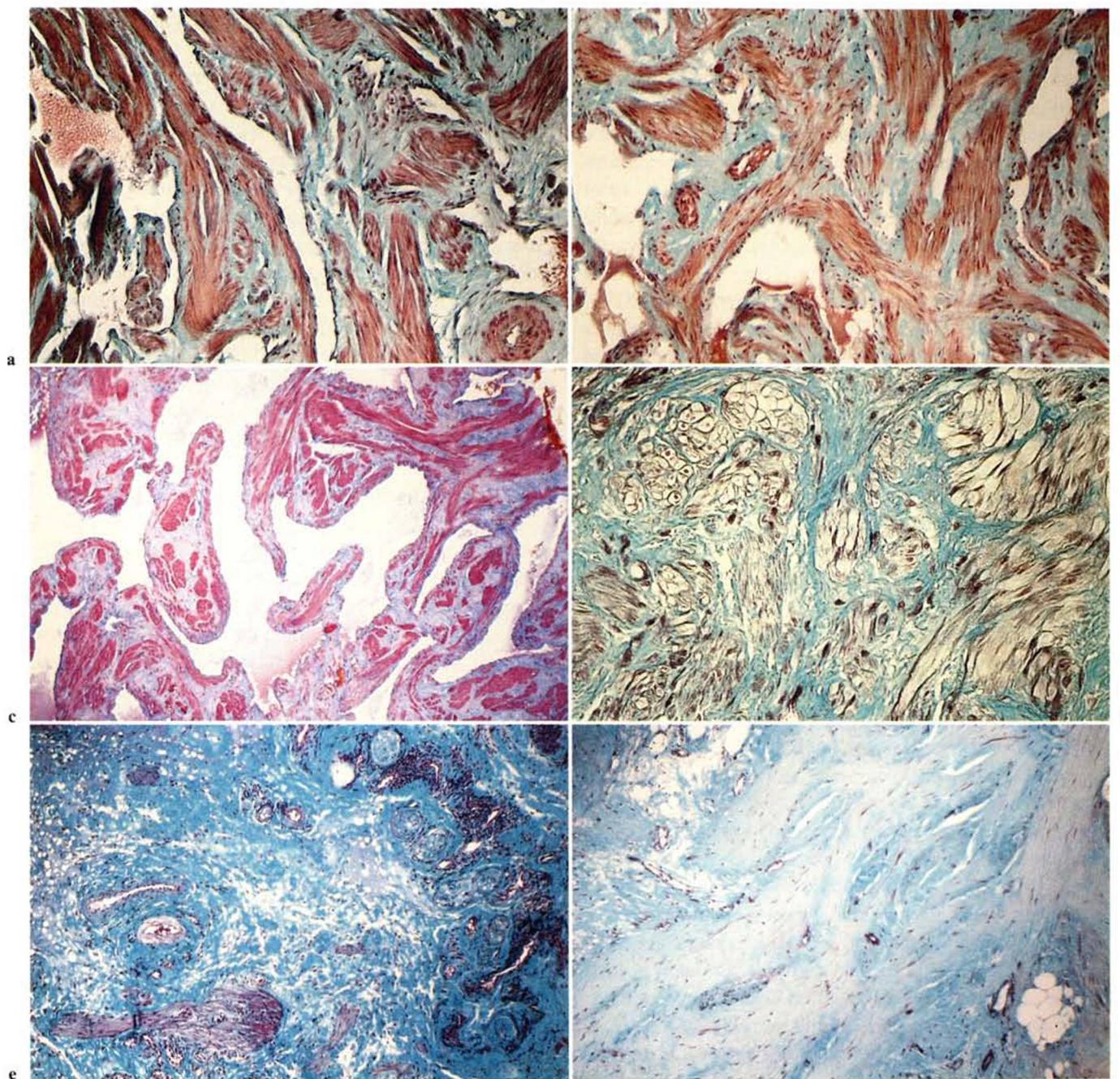


Fig. 6. Trabeculae of the PCC. **a–c** Normal. **d–f** More or less seriously altered. **a, b** Images of more or less equal trabeculae, with equivalent quantity of smooth muscle tissue and of collagenous tissue. No impotence problems. Goldner. $\times 120$. **a** 47-year-old. **b** 37-year-old. **c** Intracavernous trabeculae, where the quantity of muscle tissue appears larger than that of the collagenous tissue. No impotence problems. 44-year-old. Ladewig. $\times 120$. **d** This image is very important because it shows the earliest manifestations of deterioration which affect the trabecula muscle tissue with the increase in

age: the smooth muscle cells are highly hypertrophic, 48-year-old. Goldner. $\times 120$. **e** The PCC morphology is no longer recognizable, the normal trabecular structure is no longer visible. The PCC is formed almost completely by collagenous tissue inside which there still exist sparse smooth muscle cells unable to contract. Impotence, 23-year-old. Goldner. $\times 120$. **f** The PCC morphology is no longer recognizable. The trabecular structure is no longer visible. A marked cartilaginous metaplasia may be noticed, which well explains the total impotence of the subject, 53-year-old. Goldner. $\times 120$.