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Fine structure and permeability of capillaries in the stria vascularis and spiral ligament of the inner ear of the guinea pig

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Summary. The blood capillaries in the stria vascularis and the spiral ligament of guinea pigs were studied by electron microscopy with freeze-fracture and thin section methods, including tracer experiments with horseradish peroxidase (HRP) and microperoxidase (MP). The endothelial cells of the capillaries of both tissues are connected by tight junctions, and contain about the same number of micropinocytotic vesicles. In cases of intravascular administration before fixation, both of the tracers stained the perivascular space and almost all endothelial vesicles in the stria vascularis. On the other hand, the perivascular space and many vesicles in the spiral ligament were unstained. The endothelial tight junctions in the stria vascularis prevented the penetration of HRP, but sometimes allowed the penetration of MP. Those of the spiral ligament were impermeable to both tracers. In cases of tracer administration after fixation, leakage spots of HRP from capillaries were sparsely located all over the stria vascularis. Transendothelial channels and isolated fenestrae formed by micropinocytotic vesicles were detected. It is concluded that the capillaries of the stria vascularis are similar to the muscle capillaries and to the capillaries of the elasmobranch brain, whereas those in the spiral ligament are similar to the brain capillaries of higher vertebrates.

Key words: Capillary permeability – Stria vascularis – Spiral ligament – Freeze-fracture – Inner ear

The stria vascularis, which is thought to produce the endolymph, is sealed from the endolymph by the zonulae occludentes between marginal cells and from the perilymph by the zonulae occludentes between basal cells (Winther 1971 b; Jahnke 1975 a,b; Reale et al. 1975). Thus the intraepithelial capillaries of the stria vascularis have a closed environment and seem to have different properties than those of the spiral ligament.

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The stria vascularis has been investigated by the conventional thin-section methods (Hinojasa and Rodriguez-Echandia 1966; Kimura and Schuknecht 1970; Kimura and Ota 1974) and also by use of tracers. Horseradish peroxidase (HRP) has been shown to be transported across the endothelium by micropinocytotic vesicles, and has been demonstrated in the basement membrane of the stria capillary (Duvall et al. 1971; Winther 1971 a; Gorgas and Jahnke 1974). Osako and Hilding (1971), on the contrary, reported that HRP is transported through the intercellular junction rather than by micropinocytotic vesicles. Santos-Sacci and Marovitz (1980) found that ferritin was transported by micropinocytotic vesicles, but that little of it is taken up by the basal lamina. Thus there are many opinions about the permeability of the capillaries of the stria vascularis.

The capillaries of the spiral ligament have also been studied by the conventional thin-section methods (Takahashi and Kimura 1970; Kimura and Ota 1974) and by tracer experiments (Duvall et al. 1971; Winther 1971 a; Gorgas and Jahnke 1974). All tracer experiments indicated that HRP does not penetrate the endothelium.

In this communication we compare, on the basis of freeze-fracture and conventional thin sections, the structures of the stria vascularis with those of the spiral ligament. In addition, we compare the tightness of endothelial junctions and the role of active vesicular transport in the two sets of capillaries by use of horseradish peroxidase and microperoxidase (MP) as tracers.

Materials and methods

Fifty guinea pigs, 30 pigmented and 20 albino of both sexes, weighing 170–430 g, and showing positive Preyer reflexes, were anesthetized with chloral hydrate (0.35/kg) injected intraperitoneally, and maintained on artificial respiration. Fixation was effected by the perfusion through the ascending aorta with the following fixatives: 1 % paraformaldehyde and 1 % glutaraldehyde in 0.1 M phosphate buffer (pH 7.3) for observation of the fine structure by conventional thin sections (9 animals) and by freeze-fracture (30 animals); 1 % paraformaldehyde and 1 % glutaraldehyde in 0.1 M cacodylate buffer (pH 7.3) for tracer experiments (11 animals). After the perfusion fixation, the cochlea was dissected out of the skull and immersed in the same fixative for 2 or 3 h. During this time, the bony cochlea was removed and the lateral cochlear wall was dissected in segments.

Thin sections. Tissue segments were postfixed in aqueous 2 % osmium tetroxide for 2 h, then stained en block in aqueous 2 % uranyl acetate for 30 min. The tissues were dehydrated in graded concentrations of ethanol and embedded in a mixture of Epon 812. The thin sections were cut with a ultramicrotome and stained with uranyl acetate and lead citrate. Sections were examined in a Hitachi HU-11A or HU-11DS electron microscope.

Fig. 1. Cross section of capillary endothelium (*e*) and a part of the marginal cell (*MC*) in the stria vascularis. Two endothelial cells are connected by a tight junction (*arrow*). Several micropinocytotic vesicles, of which some lie free in the cytoplasm, some are open to the abluminal membrane, are observed. Two basement membranes (*bm*) with a narrow perivascular space can be seen. *L* capillary lumen. Bar = 0.1 μm. × 56,200

Fig. 2. One vesicle (*arrow*) appears to be open to both the luminal and abluminal sides in a capillary of the stria vascularis. Another one (*arrowhead*) fuses with the luminal endothelial cell membrane. *L* capillary lumen; *p* pericyte; *e* endothelial cell; *bm* basement membrane. Bar = 0.1 μm. × 64,200

Fig. 3. An isolated fenestra in a capillary of the stria vascularis. Images of surface membranes are overlapping probably due to a slightly greater thickness (*arrow*). *L* capillary lumen; *e* endothelial cell; *p* pericyte; *bm* basement membrane. Bar = 0.1 μm. × 63,000

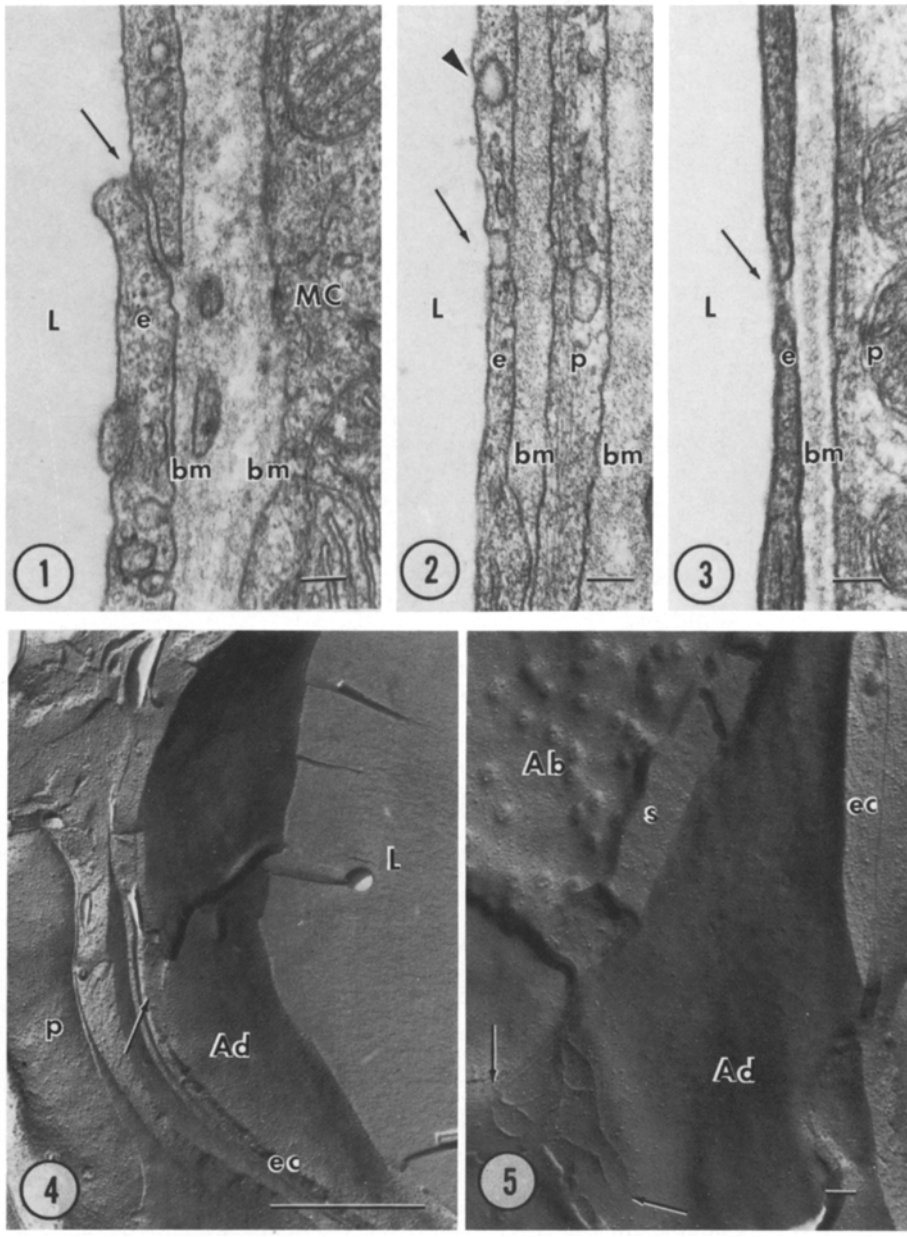


Fig. 4. Freeze-fracture preparation of the adluminal membrane (*Ad*) of the endothelium of a capillary and a pericyte (*p*) in the stria vascularis. A small portion of tight junctional ridges (*arrow*) can be seen on the P-face adluminal membrane which exhibits no vesicular opening in this picture. Several micropinocytotic vesicles are visible in the cross section of the endothelial cytoplasm (*ec*). *L* capillary lumen. Bar = 1 μm. × 20,200

Fig. 5. Capillary endothelial cells of the stria vascularis. The region of the tight junction is fractured. Ridges on the adluminal P-face membrane (*Ad*) are discontinuous (*arrows*). There are few vesicles on this membrane, whereas there are many on the adluminal E-face membrane (*Ab*) of the neighbouring cell; *ec* endothelial cytoplasm; *s* an intercellular space between two endothelial cells. Bar = 0.1 μm. × 38,600

Freeze-fracture. The tissue segments were immersed in 30% glycerol in 0.1 M phosphate buffer. Following infiltration with glycerol, the specimen was rapidly frozen by immersion in Freon-22 cooled by liquid nitrogen. The freeze-fractured replicas were obtained in a Polaron MF 200 apparatus at the stage temperature of $-115°$ C.

Tracer experiments. Reagents used as electron-microscopic tracers were horseradish peroxidase (HRP; type II) and microperoxidase (MP; sodium salt from horse heart cytochrome C). The estimated molecular weights and molecular diameters of the tracers are 40,000 daltons, 5nm for HRP and 2,000 daltons, 2 nm for MP.

HRP: Four animals were slowly injected from the left ventricle with 150 mg of HRP dissolved in 1 ml of physiological saline, and then fixed by vascular perfusion 1.5, 3, 4, and 5 min later, respectively. Five animals were first perfused with the fixative for 30 min, and then 150 mg of HRP dissolved in physiological saline was injected into the ascending aorta concominant with the fixative. Perfusion fixation was continued for an additional few minutes. The following process was carried out according to Graham and Karnovsky (1966).

MP: Two animals were fixed by vascular perfusion 4 min after injection of 100 mg MP in the same way as described for HRP. For demonstration of the peroxidase activity of MP, the method of Graham-Karnovsky (1966), as modified by Simionescu et al. (1975), was applied. The composition of the incubation medium was: 0.15% 3, 3-diaminobenzidine tetrahydrochloride, 0.02% H_2O_2 and 0.1 M imidazole in 0.05 M Tris- HCl buffer at pH 8.8. After preincubation in H_2O_2-free medium for 30 min on ice, the segments were incubated in the complete medium for 60 min at room temperature. They were then rinsed, and postfixed in 2% OsO_4 in 0.1 M cacodylate buffer (pH 7.3) for 90 min at room temperature, and finally rapidly dehydrated in a series of ethanol solutions beginning with 90%.

Results

Thin sections. In the stria vascularis, the capillary endothelial cells were thinly attenuated and connected by tight junctions. The endothelial cells were found to be continuous, although single isolated fenestrae were rarely observed (Figs. 1, 3). Some micropinocytotic vesicles were open to the capillary lumen or basement membrane and others lay free in the cytoplasm (Fig. 1). In some instances one vesicle appeared to be open to both the luminal and abluminal sides (Fig. 2). The perivascular space was narrow because of the closely packed cells in the stria epithelium (Fig. 1).

In the spiral ligament, the capillary endothelia were found to be continuous and connected by tight junctions. Some micropinocytotic vesicles, of which some were open to the luminal or abluminal side, some lay free in the cytoplasm, were observed. The number of vesicles was similar in both sets of the tissues. The perivascular space was wide because of the location in loose connective tissue (Fig. 6).

Freeze-fracture. Several preparations yielded information on the distribution of endothelial micropinocytotic vesicles both in the stria vascularis and in the spiral ligament. For example, in one preparation (Fig. 4) the adluminal membrane, together with a bit of fractured abluminal membrane, contained no micropinocytotic vesicles. In other cases the abluminal membrane contained many vesicles, whereas the adluminal did not (Figs. 5, 8), or vice versa. Also, in some cases both adluminal and abluminal membranes contained numerous vesicles (Figs. 7). Generally, vesicles were not diffusely distributed.

The fine structure of the endothelial tight junction appeared as rows of particles representing the fusion of endothelial cell membranes in both sets of capillaries

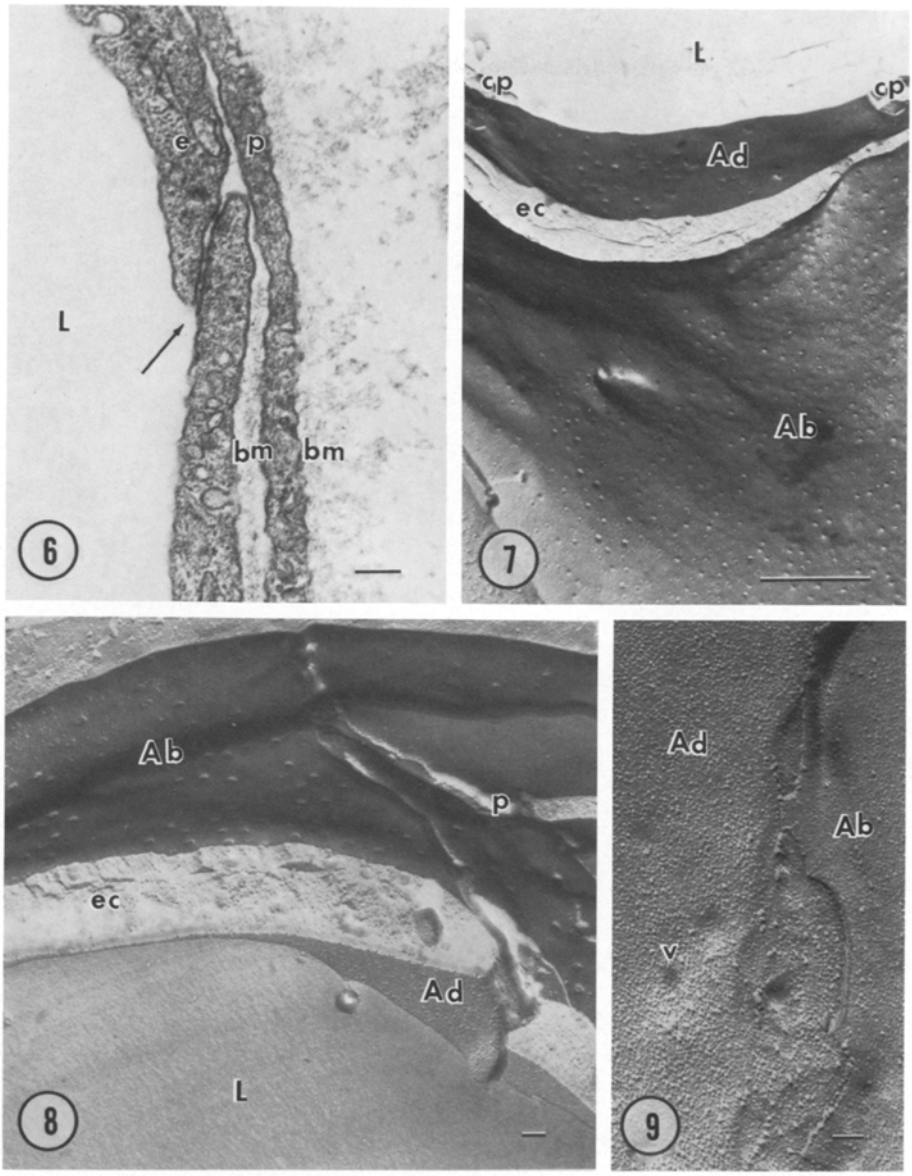
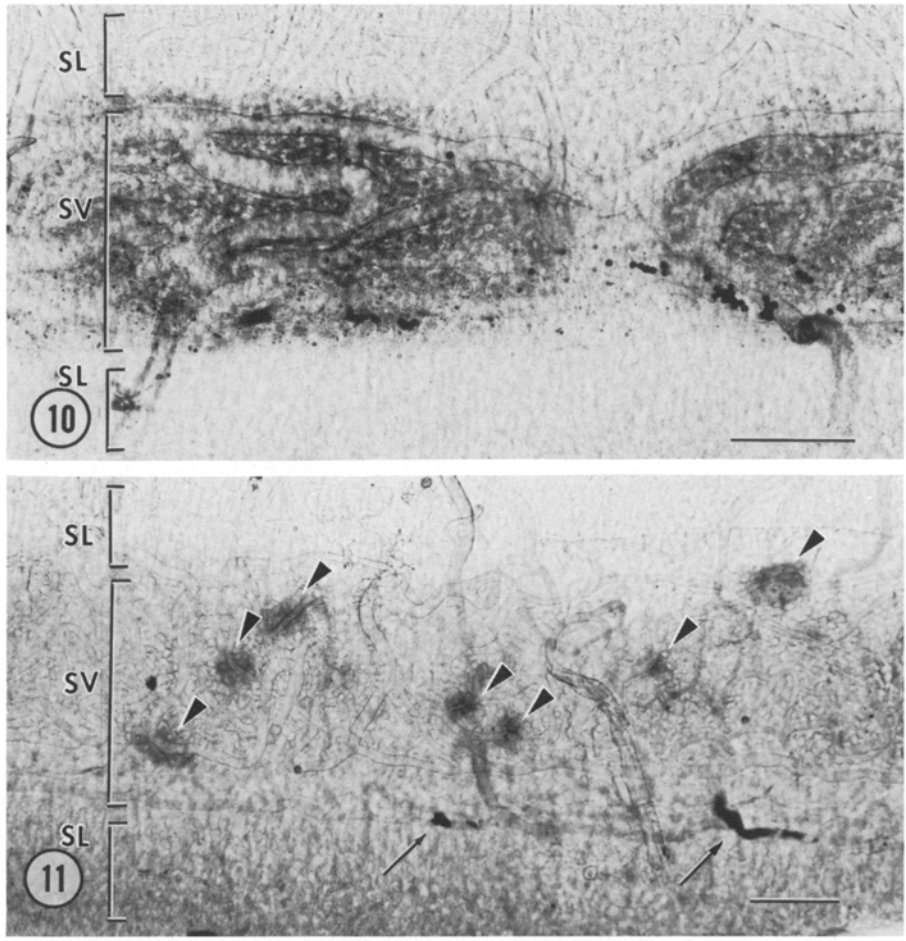


Fig. 6. Cross section of capillary endothelium (*e*) connected by tight junction (*arrow*) and pericyte (*p*) in the spiral ligament. Several micropinocytotic vesicles, of which some are open to the adluminal or abluminal membrane, whereas some lie free in the cytoplasm, are observed. The vascular basement membrane (*bm*) faces a wide interstitial space. *L* capillary lumen. Bar = 0.1 μm. × 59,000

Fig. 7. Freeze-fracture preparation showing capillary endothelium in the spiral ligament with a wide abluminal membrane (*Ab*) and a part of adluminal membrane (*Ad*). Micropinocytotic vesicles are abundant on both side membranes. Two cell processes (*cp*) of the neighboring endothelial cells are seen in cross sections; *ec* endothelial cytoplasm; *L* capillary lumen. Bar = 1 μm. × 14,800

Fig. 8. Blood capillary of the spiral ligament. The abluminal membrane (*Ab*) carries many micropinocytotic vesicles, while the adluminal membrane (*Ad*) has none. *p* Cross section of pericyte; *ec* endothelial cytoplasm; *L* capillary lumen. Bar = 0.1 μm. × 30,100

Fig. 9. Endothelial cells of a blood capillary of the spiral ligament. The region of the tight junction is fractured. Intramembranous particles at the junctional region are larger than the other P-face particles. They do not form typical ridges, but occur sparsely on the membrane elevations. Openings of micropincytotic vesicles (*v*) are present on the adluminal membrane (*Ad* P-vace). *Ab* abluminal membrane (E-face). Bar = 0.1 μm. × 40,700



Figs. 10–18. Microscopic demonstration of benzidine reaction after intracardiac injection of tracers. The reaction product in the vascular lumen is often washed out by the perfused fixatives

Fig. 10. Light micrographs of lateral cochlear wall including the stria vascularis (*SV*) and the spiral ligament (*SL*). The tissue was fixed 5 min after injection of HRP. The stria vascularis was diffusely stained with the tracer, whereas the ligament was unstained. Bar = 100 μm. × 170

Fig. 11. In this picture HRP was injected after 30 min perfusion fixation. Leaky segments of capillaries in the stria vascularis (*SV*) are easily recognized (*arrowheads*). No leakage from capillaries in the spiral ligament (*SL*) is observed. *Arrows* indicate peroxidatic activity of erythrocytes. Bar = 100 μm. × 120

Fig. 12. Blood capillary of the stria vascularis. HRP was injected 5 min before fixation. The tracer stains basement membranes of the endothelium (*e*) and the pericyte (*p*). Many micropinocytotic vesicles, both on the endothelial luminal and abluminal membranes as well as those lying free in the cytoplasm, are stained. The luminal half of the interendothelial junctions is unstained (*arrows*). *L* capillary lumen. Bar = 1 μm. × 18,000

Fig. 13. A capillary of the leaky segment in the stria vascularis. HRP was injected 30 min after perfusion. (*a*) The perivascular space are stained with HRP. Many labeled micropinocytotic vesicles can be seen (*frame*). (*b*) Higher magnification of the framed region. Several labeled vesicles (*arrowheads*) are visible in the endothelial cytoplasm dispersed from luminal side to abluminal side. *L* capillary lumen, *MC* marginal cell; *IC* intermediate cell; *BC* basal cell; *E* endolymph; *bm* basement membrane. (a) Bar = 1 μm. × 7300 (b) Bar = 0.1 μm. × 40,000

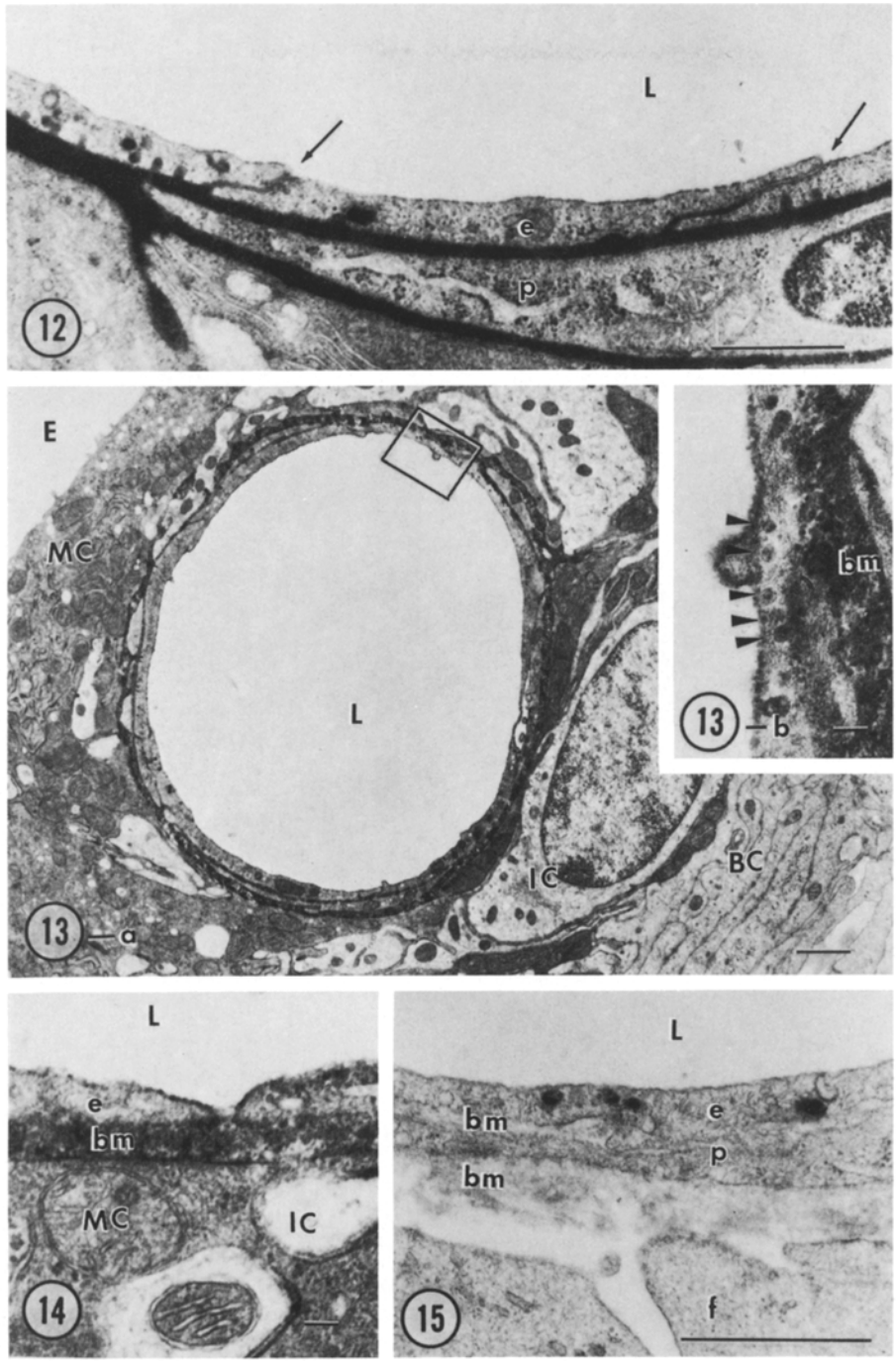


Fig. 14. HRP, 30 min after fixation. An isolated fenestra was decorated with reaction product in the stria vascularis capillary. *L* capillary lumen; *e* endothelial cell; *bm* basement membrane; *MC* marginal cell; *IC* intermediate cell. Bar = 0.1 μm. × 45,7000

Fig. 15. Blood capillary of the spiral ligament. HRP was injected 5 min before fixation. The tracer stains neither basement membrane (*bm*) of the endothelium (*e*) nor the pericyte (*p*). Some vesicles are stained with reaction products, whereas others are unstained. *L* capillary lumen; *f* fibrocyte. Bar = 1 μm. × 26,300

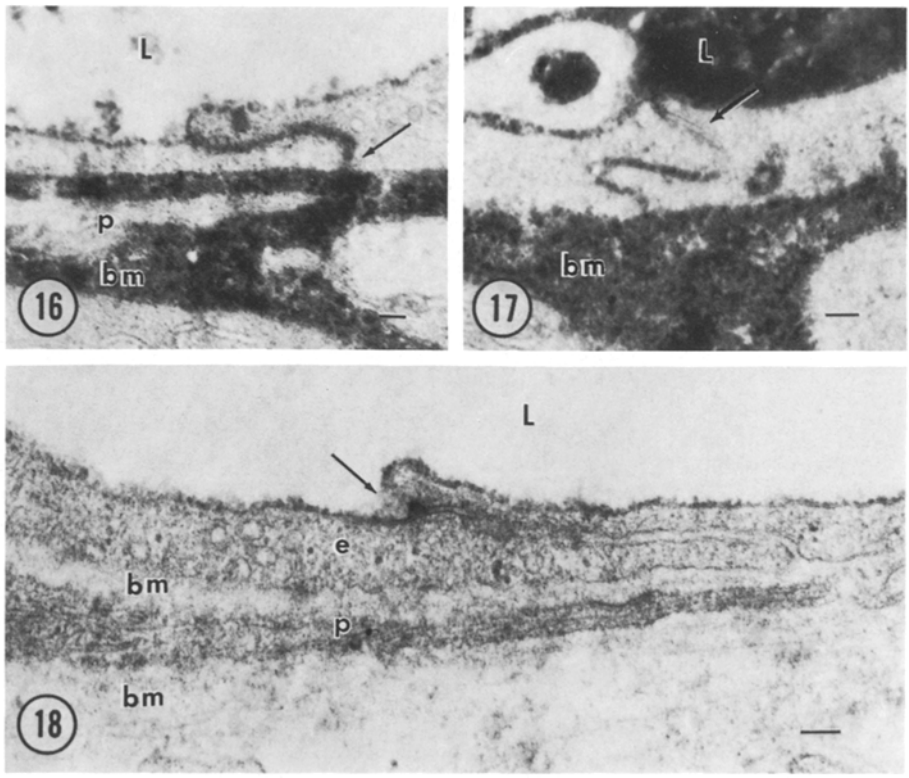


Fig. 16. A blood capillary of the stria vascularis. MP was injected 4 min before fixation. Basement membranes (*bm*) are densely stained. The interendothelial cleft is stained throughout the full length except for fusion points (*arrow*). *p* Pericyte; *L* capillary lumen. Bar = 0.1 μm. × 37,500

Fig. 17. Capillary in the stria vascularis with MP in the lumen (*L*). About half of the interendothelial cleft from the basal surface is stained whereas the other half is unstained (*arrow*). *bm* Basement membrane. Bar = 0.1 μm. × 40,800

Fig. 18. Capillary of the spiral ligament after injection with MP. The tracer is stopped at the apical part of the endothelial tight junction (*arrow*). No vesicles containing MP can be observed in this figure. Basement membranes (*bm*) are free of tracers. *L* capillary lumen; *e* endothelial cell; *p* pericyte. Bar = 0.1 μm. × 50,800

(Figs. 4, 5, 9). Some appeared as discontinuous ridges (Fig. 5) and others as atypical ridges (Fig. 9) on P-faces. The particle density of the endothelial cell membrane was generally high on the P-face and moderate on the E-face.

Tracer experiments

HRP. By light microscopy, the diffuse leakage of intravascular HRP injected before fixation was found in the stria vascularis, whereas no leakage was observed in the parts of the spiral ligament adjacent both to the scala vestibuli and to the scala tympani, as well as in the part lateral to the stria vascularis (Fig. 10). However, the

regions of spotty leakage of intravascular HRP injected after perfusion fixation were sparsely located in the stria vascularis. The capillary walls in the spotty flecks were more densely stained with reaction product than those of non-leaky segments. No leakage was found in the spiral ligament (Fig. 11).

By electron microscopy, HRP was detected at the basement membranes of the endothelium and of the pericyte of capillaries in the stria vascularis at 1.5, 3, 4 and 5 min after injection (Fig. 12). When HRP was injected after perfusion, the tracer was present at the perivascular basement membranes in the leaky segments of capillaries in the stria vascularis (Figs. 13, 14), whereas it was absent in the non-leaky segments. In cases in which the basement membrane was stained, the basal half of the interendothelial cleft was also stained probably due to entrance of HRP from the basal side. The luminal half of the cleft remained unstained. Capillary endothelium contained many micropinocytotic vesicles filled with reaction products (Fig. 12). In the segment that leaked HRP after perfusion fixation, there was a region in which many vesicles extended across the endothelium from the luminal surface to the basal surface (Fig. 13). Single isolated fenestrae labeled with reaction product were observed, although they were relatively rare (Fig. 14).

In the spiral ligament, no tracer was found at the perivascular basement membranes of the spiral ligament in all cases. Some vesicles were stained with the reaction product, but others remained unstained (Fig. 15). The interendothelial cleft was unstained throughout its full length (cf. Fig. 18).

MP. In the stria vascularis the basement membrane was densely stained, and many labeled micropinocytotic vesicles were observed as was the case with HRP experiment. In contrast to HRP, MP labeled some junctional regions of the cleft, but not the entire cleft (Figs. 17, 18).

In the spiral ligament the findings with MP were entirely the same as those with HRP.

Discussion

In our freeze-fracture preparations and conventional thin sections the endothelial of both sets of capillaries are continuous, with the endothelial cells being connected by tight junctions. Although freeze-fracture study did not demonstrate examples of continuous tight junctional ridges in the capillaries of the spiral ligament, the structure of tight junctions in both sets of capillaries appeared to be the same (cf. Figs. 1 and 5; 6 and 9). The distribution of micropinocytotic vesicles in both sets of capillaries varies. The vesicles, however, tend to be more abundunt at the basal surface in accordance with previous studies (Kimura and Ota 1974). Brain capillaries also have this tendency (Tani 1977). The frequency of micropinocytotic vesicles in both sets of capillaries is about the same. Vesicles are more numerous in both sets of the capillaries than in brain capillaries, and less numerous than in muscle capillaries. It may be that the capillaries of both the stria vascularis and spiral ligament are intermediate in fine structure between the brain and muscle capillaries.

Our tracer study, in which the tracer was injected before fixation, showed that HRP was observed in the basement membrane of the stria vascularis, but not in that of the spiral ligament as in previous studies (Duvall et al. 1971; Winther 1971 a;

Gorgas and Jahnke 1974). This was also the case with MP which we first used on both sets of the capillaries. The tight junctions between capillary endothelial cells in the stria vascularis prevented the penetration of HRP, but sometimes allowed the penetration of MP. On the other hand, those of the spiral ligament were impermeable to both of the tracers. Therefore, the tight junctions of capillaries of the stria vascularis seem to be leaky as compared with those of the spiral ligament. Micropinocytotic vesicles in the capillaries of the stria vascularis were stained with HRP and MP, whereas most vesicles in capillaries of the spiral ligament were unstained. This observation leads to the idea that the numbers of micropinocytotic vesicles in both sets of capillaries are about the same, but that most vesicles of the stria-vascularis capillaries take up tracer from the lumen, whereas few vesicles of the spiral ligament capillaries do so. The capillaries of the stria vascularis have a high rate of vesicular transport as compared with those of the spiral ligament.

The tracer experiment, in which HRP was injected after fixation, showed that leakage spots of HRP were sparsely located all over the stria vascularis, and that there was a region in which many labeled vesicles were concentrated in the endothelium. Since the fixative was perfused for 30 min, it is likely that the active vesicular transport was stopped. On the other hand, since the tissue was already fixed, it seems unlikely that was a side effect caused by release of histamine induced by HRP (Cotran and Karnovsky 1967). The findings suggest the occurence of transendothelial channels formed by vesicles in the capillary endothelium of the stria vascularis.

It is generally believed that there is no fenestration in the inner ear capillaries except for those in the cochlear plexus (Kimura and Ota 1974; Jahnke and Gorgas 1974; Jahnke 1980). However, our investigation suggests the existence of the fenestrae in capillaries of the stria vascularis, although they are unlike typical fenestrated capillaries such as those of intestine. It was proposed that there is a close relation between the fenestrae and the plasmalemmal vesicle, and that the plasmalemmal vesicle transformed through several steps into the usual fenestra with a single-layered diaphragm (Palade et al. 1979). Fenestrae observed in the present study were individually isolated and very infrequently encountered. We suggest that fenestrae observed in this study are transformed from micropinocytotic vesicles, and that they are not stable structures.

It therefore seems that transendothelial channels and isolated fenestrae formed by vesicles contribute to the high permeability of the stria vascularis capillary, in addition to the active vesicular transport which is generally accepted.

Many tracer experiments in brain and muscle capillaries have been reported. In brain capillaries in which HRP does not penetrate the intercellular junctions between the endothelial cells labeled micropinocytotic vesicles are sparse (Reese and Karnovsky 1967; Deurs 1977); this is also the case even with MP (Deurs and Amotop 1978).

In tracer experiments with muscle capillaries, Karnovsky (1967) and Williams and Wissig (1975) reported that HRP is transported through the cleft between the endothelial cells rather than by micropinocytotic vesicles. Simionescu et al. (1975 a), on the contrary, reported that MP passes through transendothelial channels rather than through interendothelial clefts. Simionescu et al. (1975 b) also reported that endothelial cells in omental or intestinal arterioles and capillaries

have completely tight junctions, whereas those in postcapillary venules have incomplete tight junctions. Wissig and Williams (1978), however, revealed that MP is transported through endothelial clefts, but not through transendothelial channels. Simionescu et al. (1978) reported further that MP fully permeates the imcomplete tight junctions of postcapillary venules in the muscle and HRP permeated 50 % of them. Thus, as mentioned above, there are two explanations of the permeability of muscle capillaries. Karnovsky (1967) and Wissig (1979) concluded that the clefts between the capillary endothelial cells, rather than micropinocytotic vesicles or transendothelial channels, are the main pathway from the lumen to the basal lamina. On the other hand, Simionescu et al. (1975 a) and Palade (1979) showed that the tracer is transported by micropinocytotic vesicles or transendothelial channels rather than through the interendothelial cleft, exept in postcapillary venules.

From the point of the permeability of brain and muscle capillaries as mentioned above, we conclude that the capillary in the stria vascularis is similar to the muscle capillary, whereas that in the spiral ligament is similar to the brain capillary.

Hashimoto (1972) reported that HRP is transported across the endothelium by chains of vesicles or channels in the capillary of the shark brain which has a narrow perivascular space like that of the stria vascularis. In the shark brain perivascular astrocytotic processes have long, tight contacts. The capillary is closely packed by these astrocytes which are thought to serve as a barrier. In the guinea pig, in which the stria vascularis is sealed towards the endolymph by tight junction between the marginal cells and towards the perilymph by tight junctions between the basal cells, the intraepithelial capillaries are permeable to HRP. It may, therefore, be said that the capillaries of the stria vascularis are similar to those of the shark brain, whereas the capillaries of the spiral ligament are similar to that of the higher vertebrate brain. Moreover, from a viewpoint of the capillaries and the surrounding tissue, the blood-endolymph barrier may resemble the blood-brain barrier of elasmobranchs, while the blood-perilymph barrier may resemble the blood-brain barrier of vertebrates higher than the teleosts.

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