

Electron Microscopic Findings of Human Auricular Appendages

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Summary. Auricular appendages were obtained from 20 patients ranging in age from 4 to 64 years with chronic heart diseases at the time of operation and observed with an electron microscope. Myo-endocardial fibrosis was seen in the majority of cases. Proliferation of smooth muscle cells was noticed in the areas of thickened myo-endocardial spaces. These smooth muscle cells played an important role in the production of ground substance, collagen, and elastic fibers in the fibrotic lesions.

Microfibrils and elastic tissue were also observed in close association with the basement membrane of cardiac muscle cells and capillaries.

Cytoplasmic filaments were observed both dispersed and aggregated in bundles in the endothelial cells on the thickened endocardium.

Normal and pathological endocardia have been investigated in human heart specimens taken from autopsy materials (Nagayo, 1909; Kochsiek, 1957; Remmele and Haag, 1967). A few studies have also been reported after examination under an electron microscope on the interstitial spaces in normal (Batting and Low, 1961; Lannigan and Zaki, 1966a, b) and pathological (Ben-Ishay *et al.*, 1968) human heart materials obtained during operation.

The following investigation was carried out to observe myo-endocardial fibrosis of the auricular appendages in human biopsy specimens which were obtained by surgical operation.

Material and Methods

Left or right auricular appendages from 20 patients, both sexes, ranging in age from 4 to 64 years with congenital malformations or valvular diseases were obtained at the time of operation (Table 1).

Small pieces of the tissue were fixed in phosphate-buffered 3.6% glutaraldehyde (pH 7.2) at 4° C. They were postfixated with 1% osmium tetroxide. After dehydration in graded alcohol, they were embedded in Araldite. Ultrathin sections were cut on a Reichert ultramicrotome OmU2 and were stained with uranyl acetate and lead citrate. All the sections were examined under an Elmiskop 101 electron microscope at 60 kV.

Observations

Diffuse or circumscribed thickening of the myo-endocardial spaces were generally observed in our materials. Marked proliferation of smooth muscle cells was seen in the thickened endocardium (Fig. 1). Interrupted elastic lamina was formed between the endothelial cells and the underlying smooth muscle cells

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Table 1. Source of biopsy materials

Case No.	Age	Diagnosis
1 (L)	31 ♀	Mitral stenosis
2 (L)	36 ♀	Mitral stenosis
3 (L)	37 ♀	Mitral stenosis
4 (L)	41 ♀	Mitral stenosis
5 (L)	43 ♀	Mitral stenosis
6 (L)	45 ♂	Mitral stenosis
7 (L)	46 ♀	Mitral stenosis
8 (L)	46 ♀	Mitral stenosis
9 (R)	5 ♀	Ventricular septal defect
10 (R)	23 ♂	Ventricular septal defect
11 (R)	4 ♂	Ventricular septal defect Atrial septal defect
12 (R)	8 ♂	Ventricular septal defect Ductus arteriosus
13 (R)	35 ♀	Ventricular septal defect Pulmonary stenosis
14 (R)	36 ♂	Ventricular septal defect Infundibular stenosis
15 (R)	10 ♂	Tetralogy of Fallot
16 (R)	38 ♀	Aortic insufficiency
17 (R)	43 ♂	Sinus venosus defect
18 (R)	43 ♂	Multivalvular vitium
19 (R)	49 ♀	Atrial septal defect
20 (R)	64 ♂	Combined aortic vitium

L = left auricular appendage, R = Right auricular appendage.

(Fig. 1). Desmosomal junctions were observed in the adjacent endothelial cells. Endothelial lining consisted of clear thick endothelial cells and dark flat ones (Fig. 2). The cytoplasmic filaments of 50–100 Å in diameter running either dispersed in whole cytoplasm or aggregated in bundles were seen in both cells. The filaments in the dark cells were more compactly aggregated than that of the clear ones. The dark cells resembled to smooth muscle cells (Fig. 2). Thin cytoplasmic processes often extended toward the underlying smooth muscle cells from the dark cells (Fig. 2).

In the myocardial fibrotic lesions, smooth muscle cell proliferation was also noticed especially around capillaries (Fig. 3). Large number of slender cells were also observed in the myocardial fibrotic lesions. Some of them had myofilaments, dense bodies and basement membrane (Fig. 3). Ground substance, collagen and elastic fibers were observed around the smooth muscle cells and the slender cells.

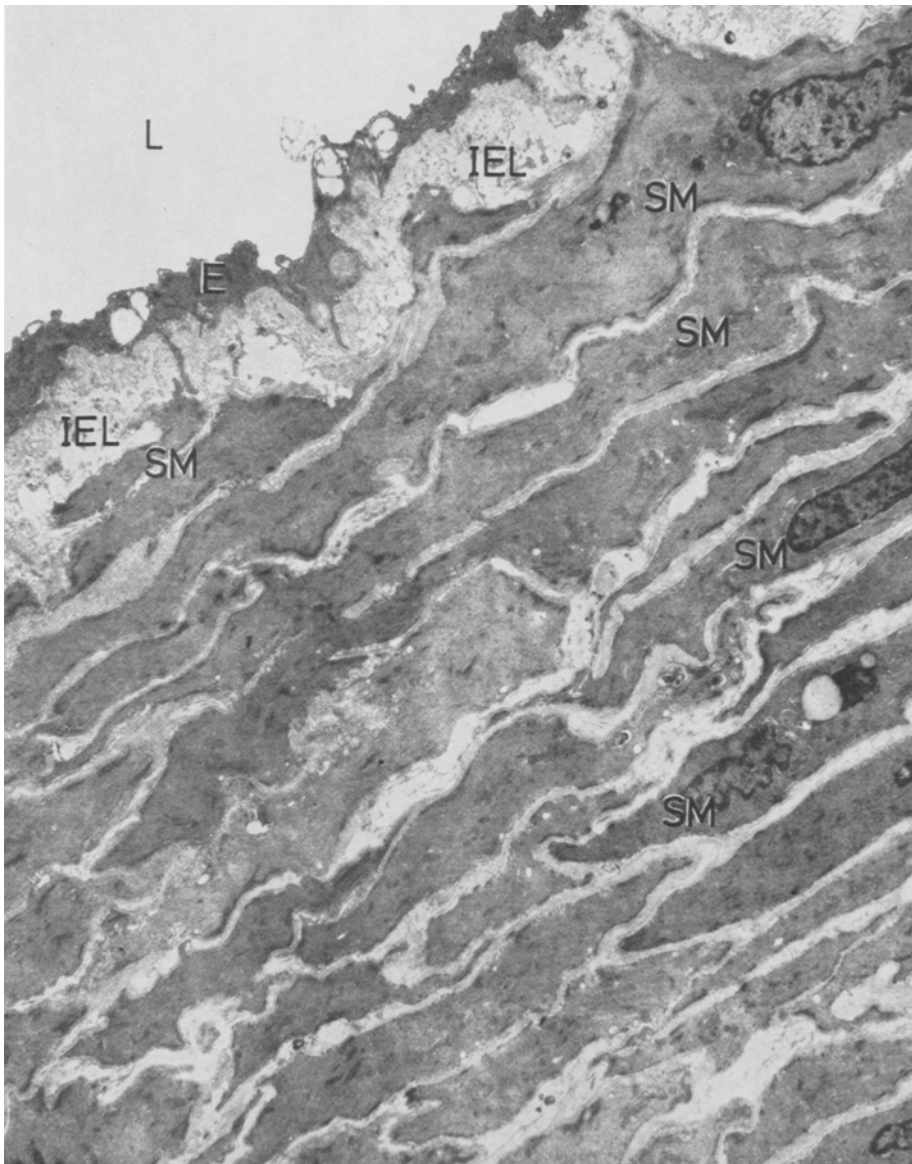


Fig. 1. Endocardium showing proliferation of smooth muscle cells (*SM*). *E* endothelial cell; *IEL* internal elastic lamina; *L* lumen (case 1). $\times 12000$

The basement membrane of capillaries and cardiac muscle cells was very greatly increased in the fibrotic lesions of myocardial spaces. Numerous microfibrils and elastic tissue were in close association with the thickened basement membrane (Fig. 4).

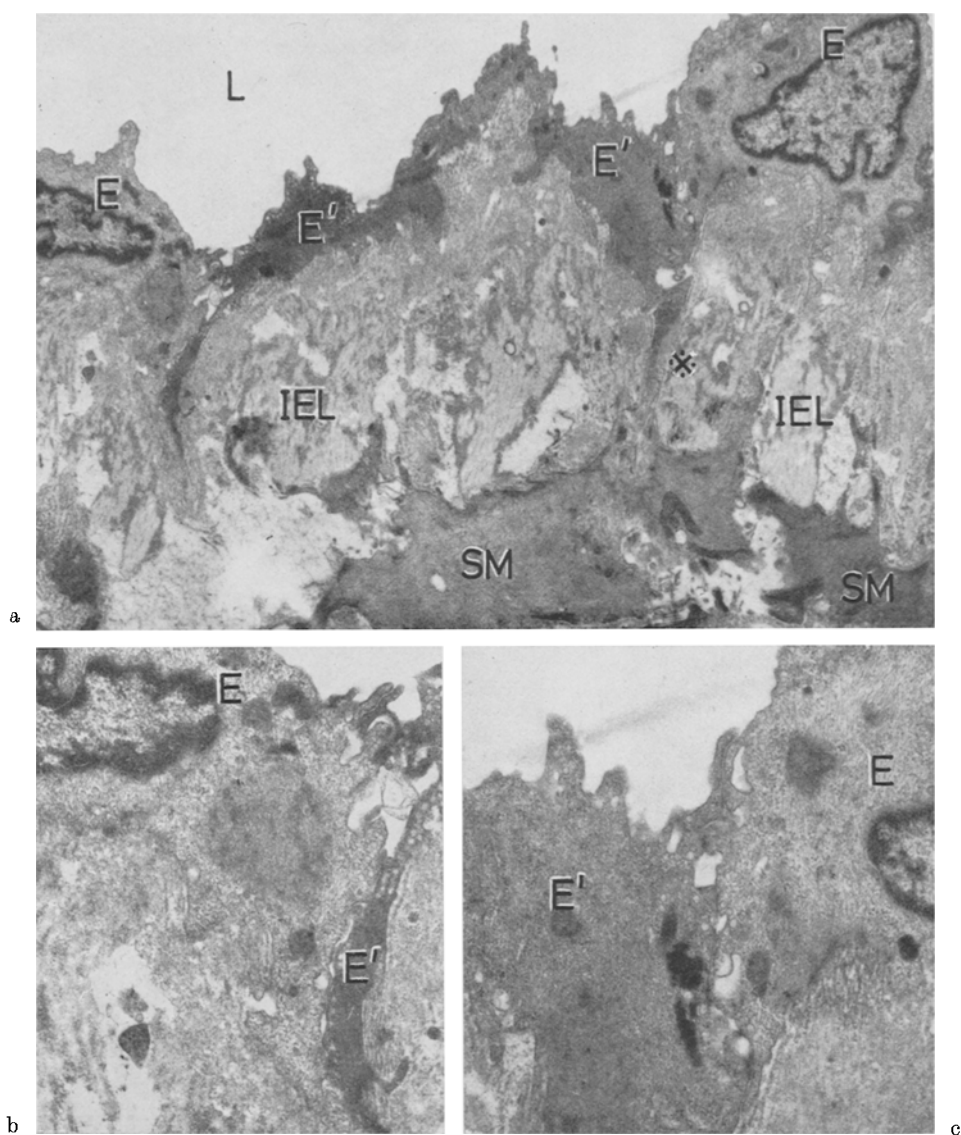


Fig. 2. a Clear endothelial cells (*E*) and dark endothelial cells (*E'*) in thickened endocardium. Note the myo-endothelial junction (*). *L* lumen; *SM* smooth muscle cells; *IEL* internal elastic lamina (case 1). $\times 9000$. b and c High magnification of the endothelial cells seen in Fig. 2a. Note the cytoplasmic filaments in the clear cells (*E*) and dark cells (*E'*). b $\times 21000$, c $\times 22500$

Discussion

Areas of myo-endocardial fibrotic lesions were observed with an electron microscope in the human heart biopsy materials obtained from cases of chronic heart diseases (Table 1). In our materials, slight myo-endocardial fibrosis was

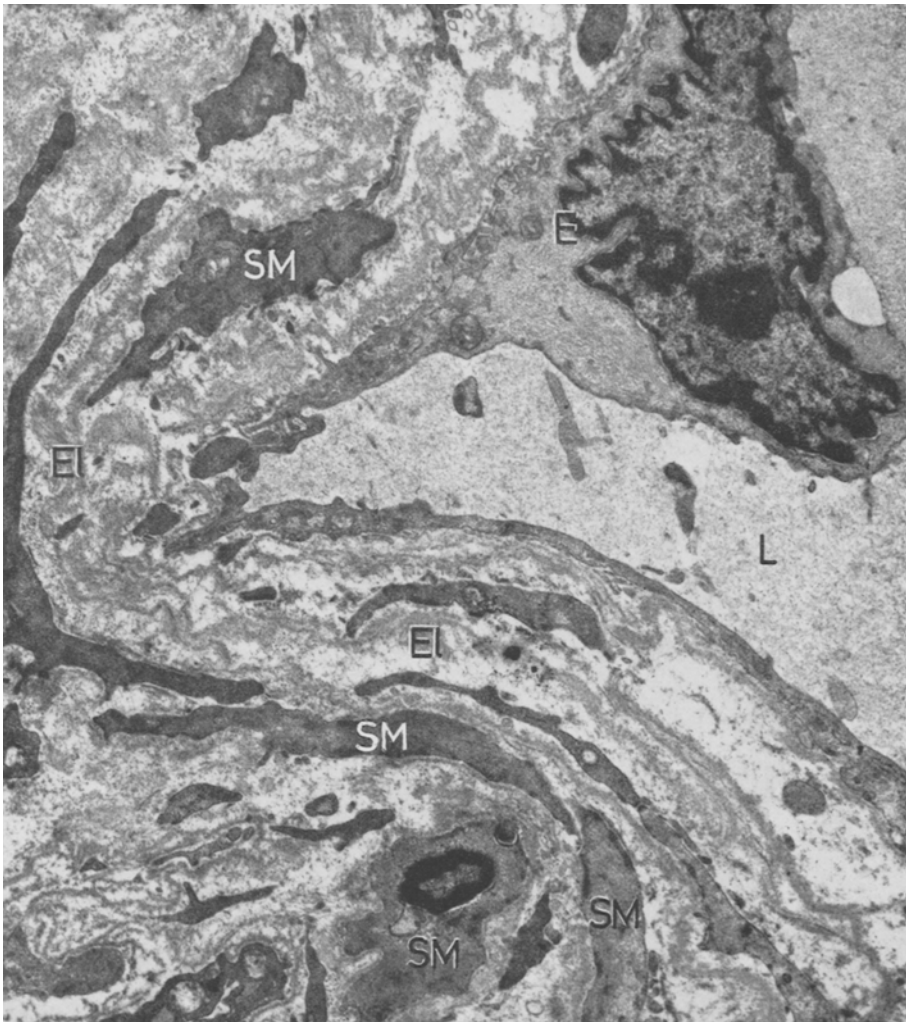


Fig. 3. Slender cells proliferated around capillary show characteristics of smooth muscle cells (SM). Fine filaments are seen in the endothelial cell (E), El elastic fibers, L lumen (case 1).
× 12800

observed in almost all cases. Among them severer lesion was seen in the cases of mitral stenosis.

It has been reported that fibroblasts are the main cellular element in areas of myocardial fibrosis (Ben-Ishay *et al.*, 1968). The slender cells investigated have been considered to be mesenchymal cells or intermyocardial cells of an undifferentiated type of fibroblast in normal heart myocardium (Batting and Low, 1961; Lannigan and Zaki, 1966b). Some of the slender cells in our materials showed characteristics of smooth muscle cells. Proliferated smooth muscle cells

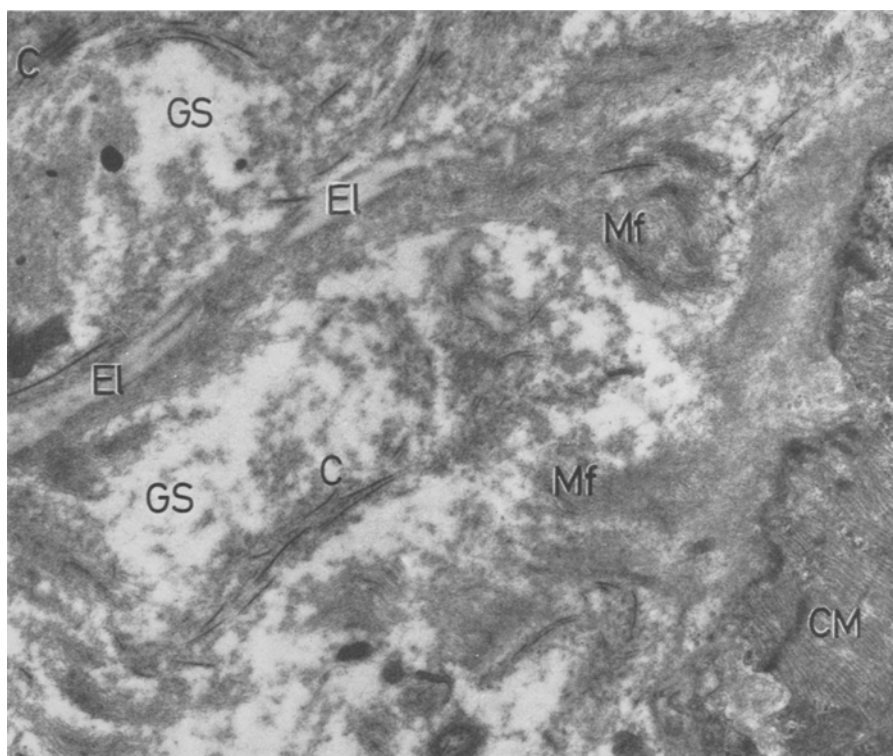


Fig. 4. Microfibrils (*Mf*) and elastic fibers (*El*) are in close association with the thickened basement membrane of a cardiac muscle cell (*CM*). *C* collagen fibers, *GS* ground substance (case 2). $\times 16200$

in the myo-endocardial spaces produced ground substance, collagen and elastic fibers.

The endocardium can be compared with either intima of vessels (Bargmann, 1963) or whole vessels (Becker, 1964). The proliferation of smooth muscle cells in the endocardium is thus considered to be comparable with smooth muscle cells in thickened intima of vessels. Microfibrils seen around the smooth muscle cells are considered to be the precursor of collagen and elastic fibers (Rhodin, 1962). Thin microfibrils are also considered to be synthesized at the outside of the basement membrane of capillaries and cardiac muscle cells (Ben-Ishay *et al.*, 1968).

Two types of endothelial cells were observed in the areas of thickened endocardium (Fig. 2). Clear cells were in general thick and had loosely arranged cytoplasmic filaments. Dark cells were often flat and had more myofibril-like structure ("myoendothelial cells", Hama, 1960). Two types of endothelial cells were also reported in the capillaries of rat heart (Bullon, 1971). Distinct dense bands arranged at intervals in the endothelial cells (Röhlich and Oláh, 1967) were not observed in our materials, but the author would consider that the filaments in the cytoplasm of the endothelial cells exist as contractile in function. It is considered that

the dark endothelial cells play an important role in transmitting the information of "contraction" to the underlying smooth muscle cells in the myo-endothelial junctions.

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