Virchows Arch [Pathol Anat] (1982) 396:279–289

Virchows Archiv A
© Springer-Verlag 1982

Striae: Morphological Aspects of Connective Tissue

M.Th. Pieraggi,* M. Julian,* M. Delmas, and H. Bouissou

Department of Anatomic Pathology (Prof. H. Bouissou) C.H.U. Rangueil, Chemin du Vallon, F-31054 Toulouse Cedex, France

Summary. The study of 15 abdominal striae in women aged 25 to 57 shows important histological modifications in the skin. The collagen is fragmented and the ground substance is abundant. Fibroblasts are globular, quiescent, and lose all signs of fibrillar secretion. In the light of the recent biochemical data, our results suggest that the striae are the consequence of fibroblastic dysfunction, due to abdominal distension. Comparison with scarred and normal skin indicate that striae are a special entity belonging to the group of connective dystrophies.

Key words. Striae: morphological aspects – Scanning – Transmission electron microscopy

First described by Troisier and Menetrier in 1889, striae, a common disfiguring and innocuous disease, have been studied by physicians. According to our knowledge, only 1 author, Arem, published a morphological study in 1980 (light and scanning electron microscopy) of abdominal striae during lipectomies.

This study specifies the dermal lesions observed in striae unassociated with hormonal causes and compares them with those in ageing and in scars. Non hormonal striae can be interpreted as an original modification of connective tissue.

Material and Methods

The Patients. Striae, scars and normal skin fragments were examined by light and electron microscopy. Fifteen samples of abdominal striae from the Plastic Surgery Department were taken from women between the age of 25 and 57. Seven were post pregnancy striae, four appeared during obesity, four came from obese cases, but continued in the post gravid period. All these patients were operated on for lipectomies. In all cases the age of the striae was over 2 years.

* Chargé de recherche au C.N.R.S.

Offprint requests to: H. Bouissou at the above address

0340-1227/82/0396/0279/$02.20

The biopsies were made perpendicularly to the longitudinal axis of the lesions and concerned the macroscopically injured skin and the adjacent skin not visibly involved.

Normal skin biopsies, (controls) were taken on the same patients, from the inner side of the arms, and allowed a comparison with ageing lesions.

Five abdominal scars 2 years old were also studied.

Not included in our report were the striae occuring in pathological hormonal disease such as Cushing's disease, eliminating a direct action of the corticoids on the fibroblasts the chief element in the formation and the maintenance of the connective tissue.

Methods. Each sample was studied by light and electron microscopy.

Light Microscopy. Collagen tissue was demonstrated by Masson's trichrome blue. The richness of the dermis in elastic fibers was appreciated by Verhoeff's iodin ferric haematoxyline stain. Types I and III collagens were evaluated with polarized light after Sirius red staining, type I collagen appearing in yellow orange, type III and newly made collagen in green.

Scanning Electron Microscopy. The fragments were fixed in glutaraldehyde and osmiun tetroxyde and dehydrated in a graded series of alcohol. Samples were critical point dried and then coated with a 20 nm layer of gold. The fragments were examined in a Jeol. JT 200 scanning electron microscope.

For Transmission Electron Microscopy. 1 mm samples were collected. They were fixed in 2% glutaraldehyde, washed in cacodylate buffer, post fixed in 2% osmium tetroxyde, dehydrated and included in EPON. Slices were made with Reichert OMU 2 microtome, stained with uranyl acetate and lead citrate and observed with an Hitachi HU 11 electron microscope.

Results

In Table 1 the lesions in the striae, the ageing and the scar zones are summarized.

The striae showed narrow atrophic bands more or less parallel and separated by normal skin areas. Their length was variable. In our study, because of

Table 1. On this Table are only summarized, the dermal lesions. In the 3 cases there was not significant epidermic variations

	L.M.		S.E.M.	T.E.M.			
	C.	E.		C.	E.	GS.	F.
Striae	Thin Fragmented	Sparse	Thin-short Stretched	Short Thin Diameter ↘	Sparse	Abundant +++	Globular Quiescent Secretion = 0
Ageing[a] p.D	Fragmented	Dis-appear-ance	Compact Thick	Dislocated	Sparse	Abundant +	Stellate Quiescent Secretion = +
Ageing[a] d.D	Normal	Normal	Compact Thick	Normal	Sparse	Abundant	
Scar	Dense	Sparse	Thick	Thick, Large Compact	Sparse	Little	Stellate Active Secretion = ++

L.M. = light microscopy; S.E.M. = scanning electron microscopy; T.E.M. = transmission electron microscopy; C = collagen; E = elastic fibre; GS = ground substance; F = fibroblast; ↘ = decreased

[a] Ageing lesions are mainly observed in papillar dermis (pD), deep dermis (d.D)

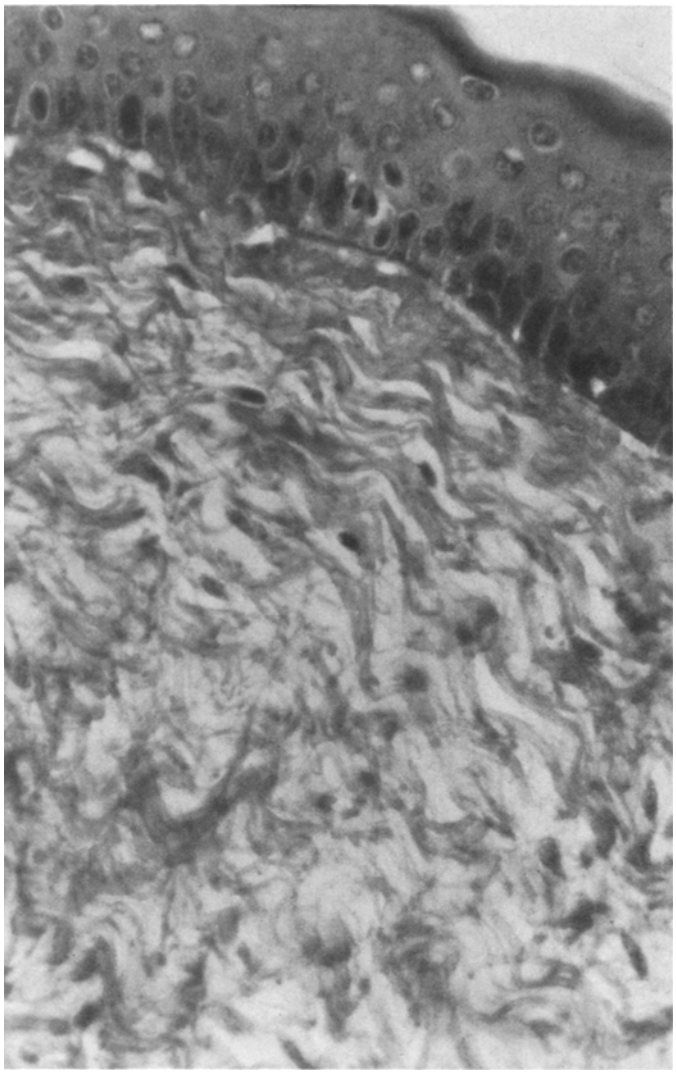

Fig. 1. *Striae.* Histological appearance; the epidermis is flat, the dermal collagen is organized in thin parallel bundles. Masson's blue trichrome stain. ×40

their age, they appeared as whitish depressed furrows, covered with crumpled epidermis.

On light microscopy the striated zone was covered with a thin and generally atrophic epidermis. The whole underlying dermal zone presented major collagen and elastin modifications. In the dermis, the collagen bundles were thin and short often parallel to the epidermis. They were uncoiled, stretched out, fragmented and separated by an abundant ground substance. These lesions appeared mainly in the medium and deep dermis (Fig. 1). The very few elastic fibres were thinned and fragmented. At the junction between healthy and striated

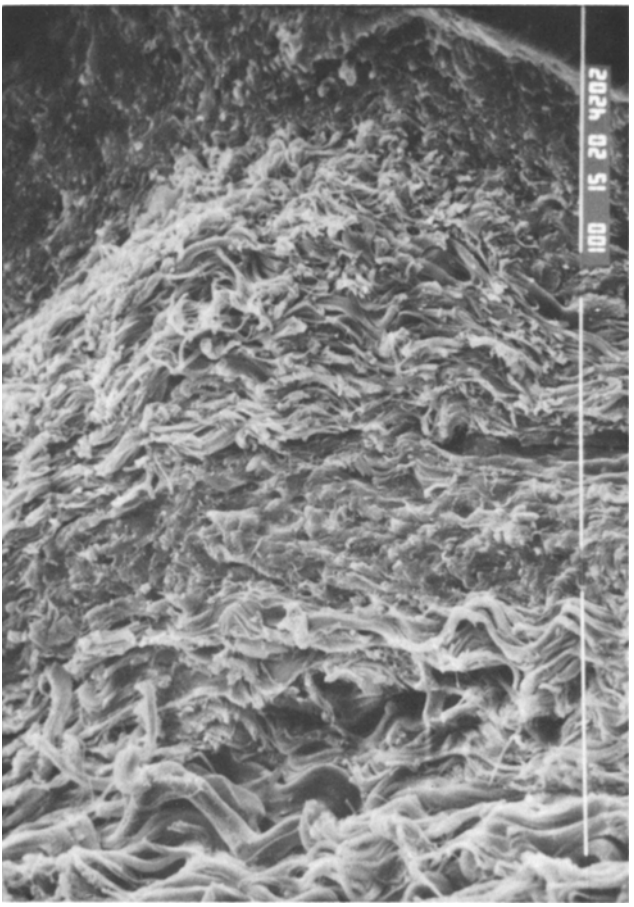

Fig. 2. *Striae*. Scanning electron microscopy. Collagen bundles are thin and stretched. × 350

skin, they formed heaps and seemed to be retracted, and even appeared dystrophic. There was no transition between normal and striated skin. The Sirius red stain revealed a great quantity of type I collagen and very few small type III bundles disseminated within the entire dermis. On electron microscopy the dermis appears slack in the scanning machine. The thin, short and stretched out collagen bundles are widely separated from each other and have a wide spread aspect. (Figs. 2 and 3). Using transmission electron microscopy, at low resolution the striated zone is easily recognizable and marked by large empty areas (Fig. 4). The abundant ground substance separated the short thin collagen bundles of irregular thickness. These bundles are composed of a variable number of fibres, sometimes even appearing isolated. Longitudinally, the collagen fibres keep a characteristic structure with a periodicity of about 65 nm. Transversly they have an average diameter ranging from 20 to 40 nm. The fibres are more

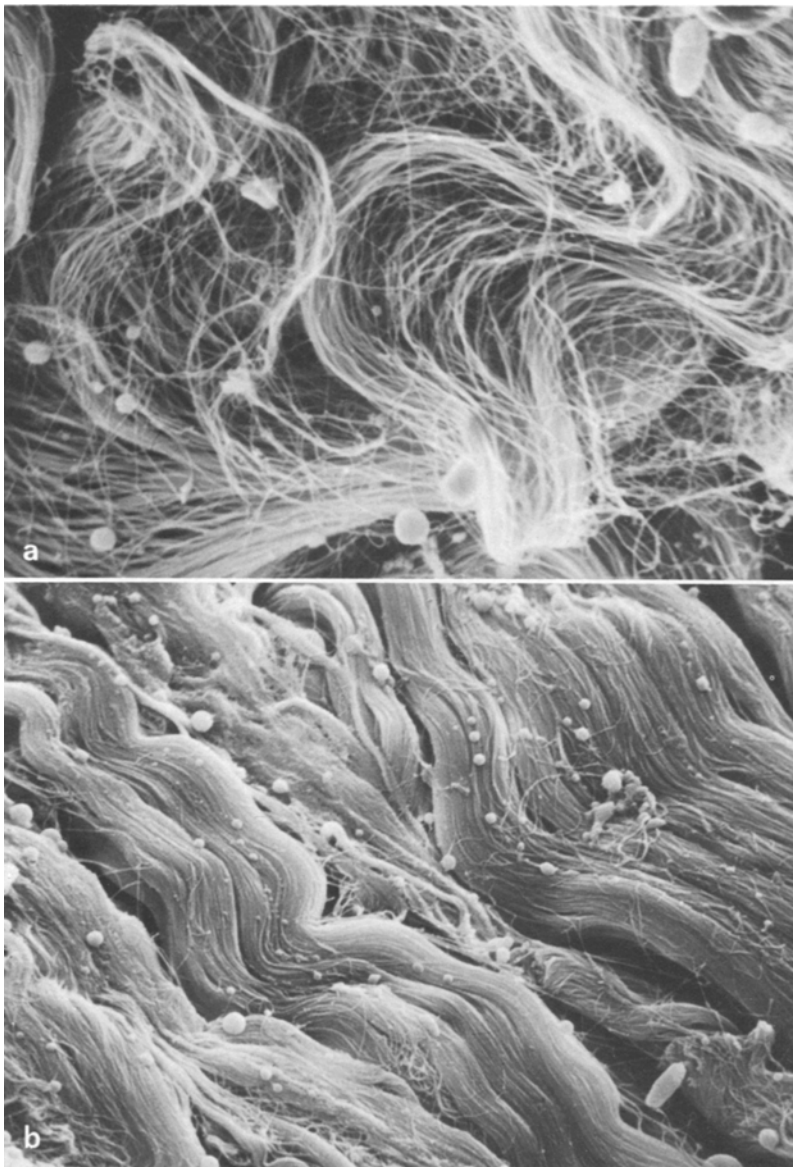

Fig. 3. (**a**) *Striae*. Scanning electron microscopy. Collagen bundles are separated into isolated fibrils. ×1,500. (**b**) *Normal dermis*. Scanning electron microscopy, collagen bundles are thick, parallel and waved. ×1,500

slender than in normal dermis (100 nm) (Fig. 5). The sparse elastic fibres have a normal structure or presented an irregular cross-section along its length. The few fibroblasts seem to be quiescent. They are slightly spread out and become globular (Figs. 6 and 7). Their cytoplasm is not very abundant, very poor in organelle, forming a collar around the nucleus. The rough endoplasmic

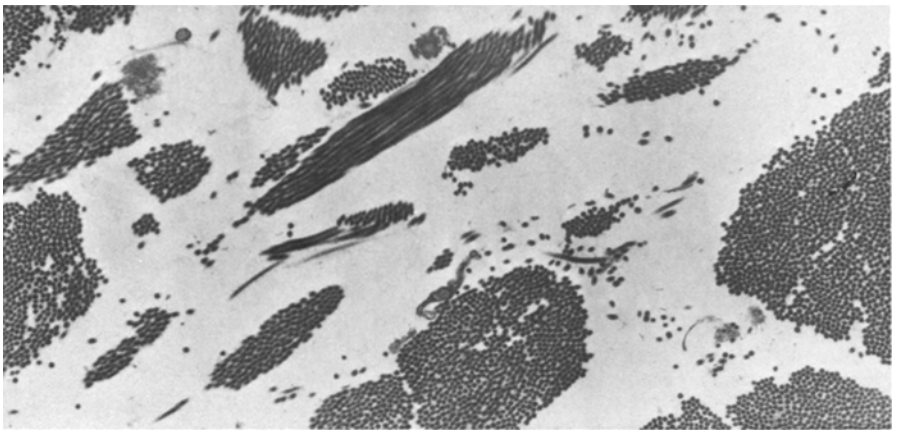

Fig. 4. *Striae.* Electron microscopic aspect. Irregular and thin collagen bundles, separated by large areas of ground substance. Uranyl acetate-lead citrate stain. × 5,000

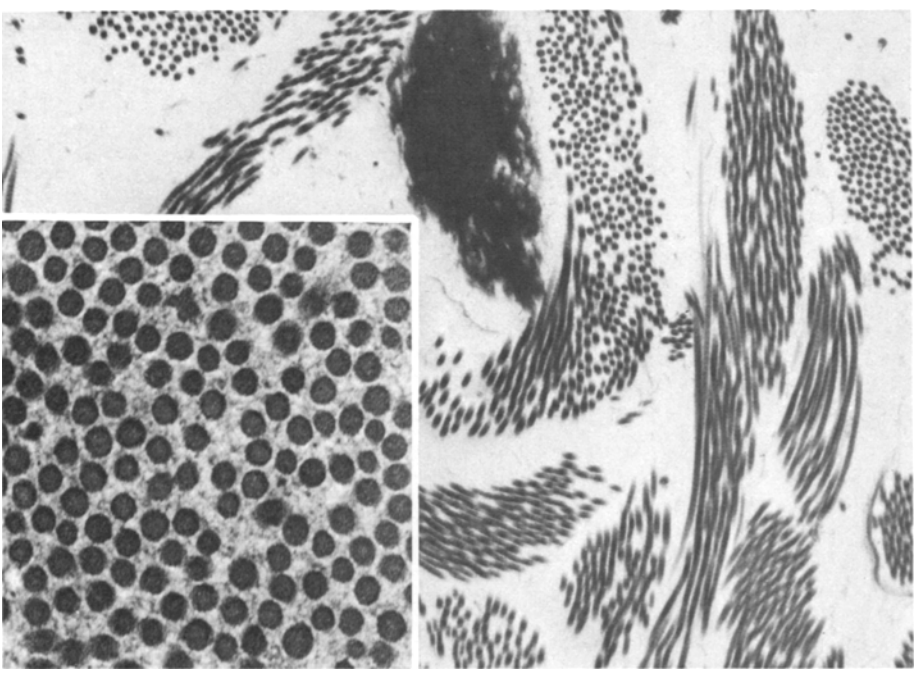

Fig. 5. *Striae.* Electron microscopic aspect. Another view with thin collagen bundles. Elastic fiber with normal appearance. Uranyl acetate-lead citrate stain × 5,000. *Inset.* Cross section of collagen fibers, diameter between $^1/_2$, $^1/_4$ normal. Uranyl acetate-lead citrate stain × 38,000

reticulum, the golgi and vesicular systems are non-existant. A few dense bodies are seen. No fibrillar material is noticed on the side of the plasma membrane. The cells having lost all fibrillar anchorage seem to "float" in the ground substance. Cells with organelles which prove their capacity for secretion are extremely rare (Fig. 7).

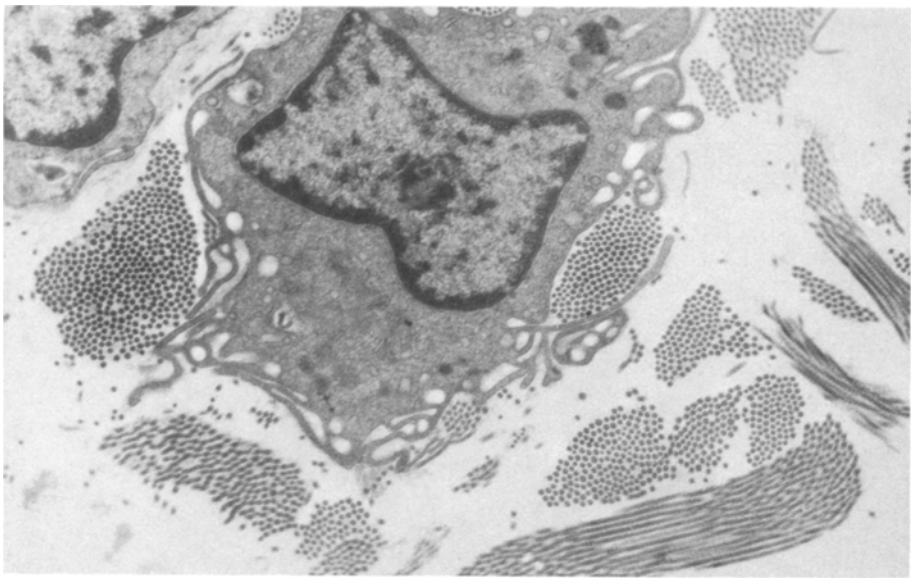

Fig. 6. *Striae.* Electron microscopic aspect of one fibroblast. Round cell without extension. Little cytoplasm. Uranyl acetate-lead citrate stain. ×15,000

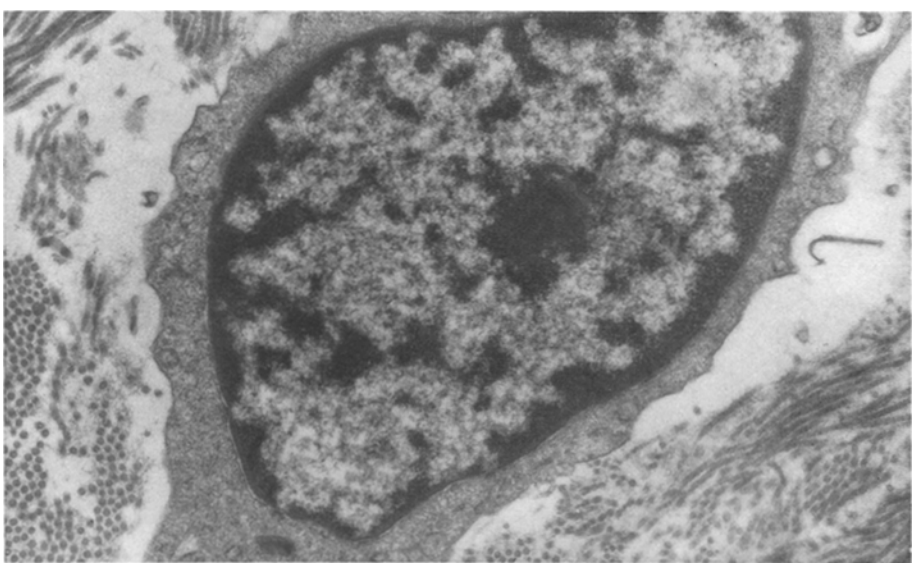

Fig. 7. *Striae.* Electron microscopic aspect of another fibroblast. Thin cytoplasmic band containing rare organelles. Uranyl acetate-lead citrate. ×32,000

Changes in the normal skin are as follows, with ageing the epidermis keeps normal or becomes thin. Ageing lesions are mainly situated in the papillary dermis. They are marked by a progressive disappearance of the arborescent elastic material and the collagen looses its bundled aspect and becomes granular. Depending on the degree of ageing, the skin is classed according to three different

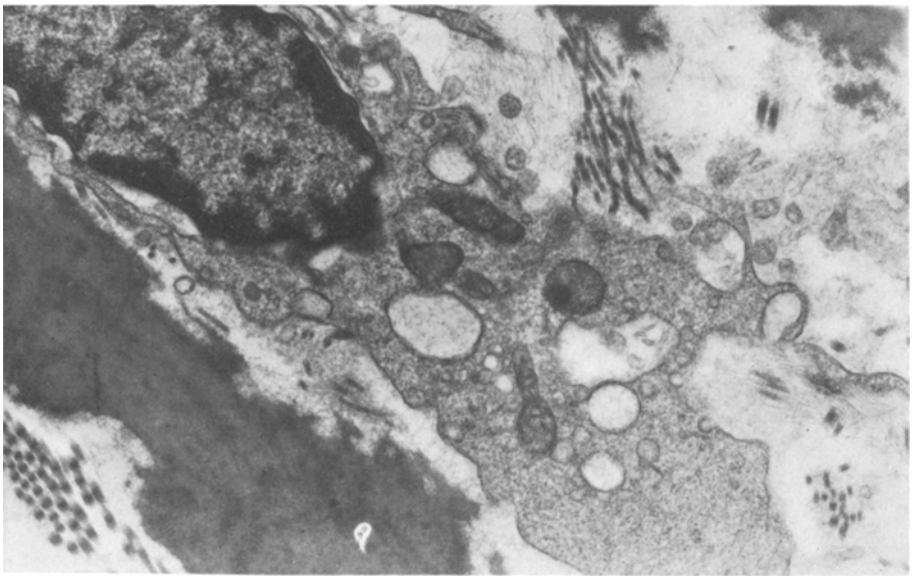

Fig. 8. *Normal cutaneous ageing.* Fibroblast with some cytoplasmic organelles and microfibrils in connection with plasma membrane. Uranyl acetate-lead citrate stain. × 12,000

types or groups: types 0, I and II. In type 0 (normal skin) the elastic fibres of the papillary network are numerous and perpendicular to the epidermis. In type I, the elastic fibers disappear from place to place. In type II, (aged skin) no elastic fibres are seen in the papillary dermis. In the median and deep dermis, the numerous elastic fibres form a plexiform network. The collagen conserves a dense and compact fibrillary aspect. Sirius red staining shows a large quantity of type I collagen, with small masses of type III collagen from place to place.

With scanning electron microscopy, the dermis of normal skin is compact, collagen bundles are thick, wavy and pressed together. With transmission electron microscopy the major lesion is seen in the papillary dermis. In type II skin, the fragmented collagen forms short and thin bundles, separated by large zones of ground substance. The fibroblasts become quiescent with dense bodies and with a depletion of the rough endoplasmic reticulum. In the medium dermis, the collagen bundles are thick, dense, and separated by very little ground substance. Elastic fibres and fibroblastic extensions are situated between the bundles. The fibroblasts have starlike form. The cells still conserve their secretory characteristics with peripheral microfibrils and have a well developed endoplasmic reticulum indicating their fibrillary secretory activity (Fig. 8).

In scar zones, by light microscopy the dermis of a scar over 2 years old is formed by densely pressed collagen bundles, sometimes forming whirling bundles. Elastic fibres are very rare. The Sirius Red stain shows plenty of type I collagen and small heaps of type III collagen.

With scanning electron microscopy, dermis collagen bundles are wavy, thick, large and packed together.

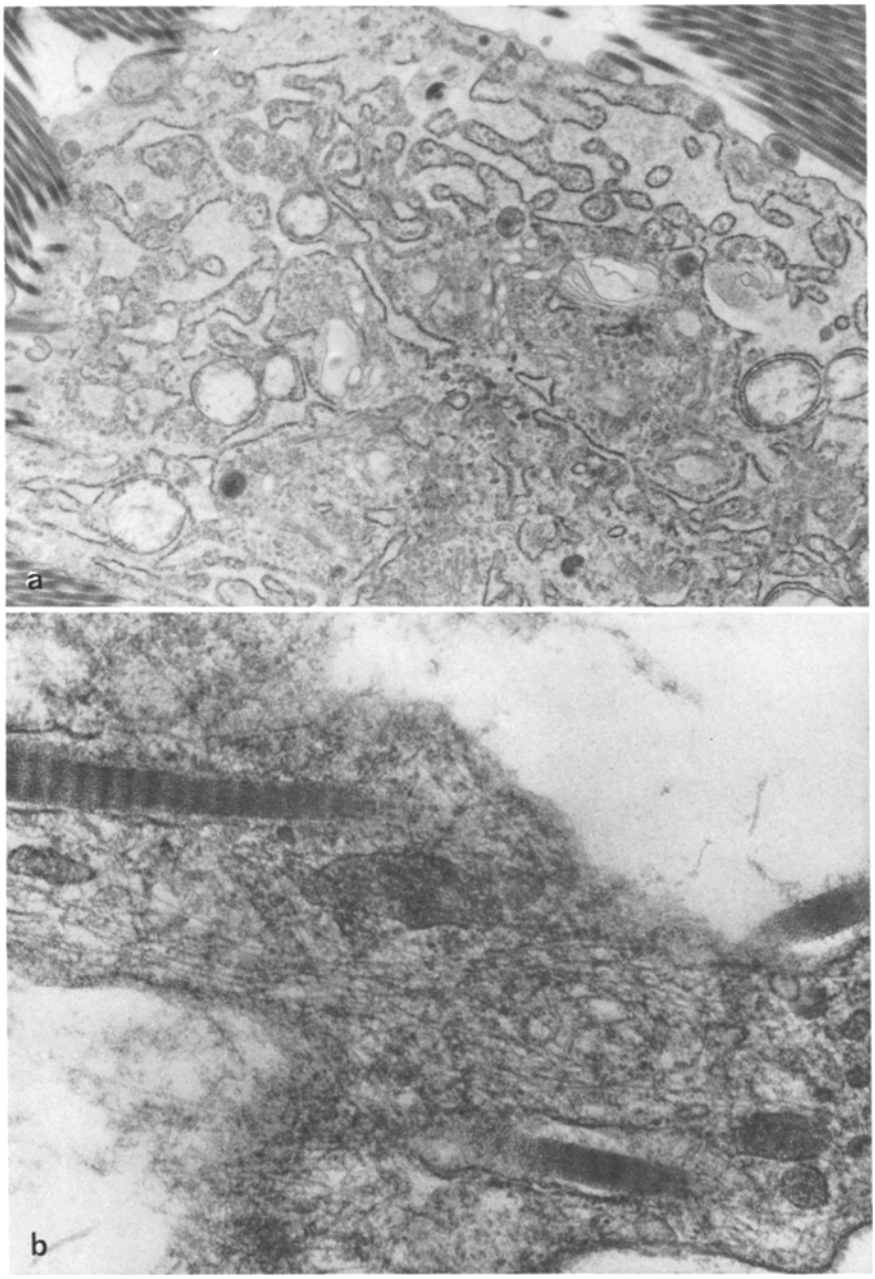

Fig. 9. a *Cutaneous scar.* Active fibroblast containing numerous organelles. Endoplasmic reticulum very well developed. Uranyl acetate-lead citrate stain. ×14,000. **b** *Cutaneous scar.* Fragment of fibroblast, into the cytoplasm there are typical collagen fibrils. Uranyl acetate-lead citrate stain. ×32,000

Using transmission microscopy the collagen is seen to be made of large bundles with a normal structure. There is little ground substance. The fibroblasts are active, their cytoplasm contains a lot of organelles, the endoplasmic reticulum is very developed. On the external side of the plasma membrane, there are many microfibrils, proving evidence of fibrillar secretion. Also, a number of collagen fibrils are present within vesicular spaces the cytoplasm (Fig. 9) which is related to synthetic and not lystic activity, acid phosphatase assay being negative.

Discussion

Examination of striated skin has shown collagen modifications and fibroblastic lesions, which were entirely different to cutaneous scar and skin ageing. Though considered to be a scar by Arem and Kischer (1980) striae are the consequence of dermal lesions by stretching, and differ profoundly from the connective modifications observed during the scarring process. In scars, histological signs of active fibrillogenesis are seen particularly in the presence of intracytoplasmic collagen. This is not a sign of collagenolysis (Rose 1980); (Svoboda et al. 1976), but rather of a greater synthesis of collagen. These intracytoplasmic fibrils are seen when too many collagen precursors are formed, and in this case it is a complementary pathway in normal collagen synthesis (Garrone 1975 and 1978); and (Trelstad and Hayashi 1979); (Trelstad 1981).

Such features have never been observed in the fibroblasts of striae, even if there is collagen synthesis, as reported by Arem and Kischer (1980); but their biochemical assays were only made on one sample and this synthesis had no histologically recognisable image. The fibroblasts remain quiescent, and do not contain secretive organelles.

The striae cannot be considered to be a localized ageing lesion. The site of the lesion is different: in the superficial dermis in ageing, in the entire dermis and especially the medium and deep dermis for the striae. The histological aspects of ageing (Bouissou et al. 1973 and 1976) are quite different from those of striae. In an early or normally aged skin, collagen is less dislocated or fragmented than in striae, and the ground substance is abundant but does not form large inhabited zones. Fibroblasts conserve their stellate form and have some signs of secretion (microfibrils in contact with the plasmic membrane). Such appearances are very seldom seen in the striae, where the fibroblasts are globular and have lost their abilities for fibrillary secretion. The absence of secretion is also shown by the similarity between aged and striated skin after Sirius red staining. However, in scars there is a greater quantity of type III collagen.

Striae are actually a singular dermal lesion characterized by special modifications of fibroblasts and connective tissue. Elton and Pinkus (1966); Mauss (1972) think that the mechanical "stretching" is the triggering element, occuring in individuals having a genetic predisposition. This hypothesis can be supported by biochemical statements and by our morphological results. The connective tissue is an entity, with interdependency between fibroblasts and interstitial tissue [Robert (1980); Amblard and Zambelli (1980)]. In the skin, connective tissue stability can be broken by special physical conditions. Longacre (1976);

Larson et al. (1976) studying hypertrophic scars, showed that increased pressure or varying tensions could lead to a decrease in fibroblast secretion, modifying the interstitial structure.

In the striae we can observe skin stretching by abdominal tensions resulting from obesity or pregnancy, fibroblastic and interstitial histological modification, and biochemical alterations in the incorporation of molecular precursors (mainly glycosylated) within the frame of the polymeric fibres (Gautray et al. 1980).

Thus these 3 complementary facts (physical, morphological and biochemical) enables us to consider *striae to be a consequence of the disfunction of the fibroblast induced by abdominal distension and occuring in a predisposed subjects.*

The striae is a special entity, distinct from the scarring process, and from localized ageing. They belong to the large group of connective tissue dystrophies where genetic and acquired factors intervene.

Acknowledgements. The technical assistance of Mrs Foulquier F., Galy A., and Py Y. is gratefully acknowledged.

References

Amblard P, Zambelli P (1980) Precis de physiologie cutanée. Meynadier F (ed). De la Porte Verte, Paris

Arem AJ, Kischer CW (1980) Analysis of Striae. Plast Reconstruct Surg 65:22–29

Bouissou H, Pieraggi MTh, Julian M, Douste-Blazy L (1973) Cutaneous Ageing. In: Robert L (ed) Frontiers of matrix biology. Vol 1 S. Karger, Basel

Bouissou H, Pieraggi MTh, Julian M, Douste-Blazy L (1976) Simultaneous degradation of elastin in Dermis and in aorta. In: Robert L (ed) Frontiers of matrix biology. Vol 3. S. Karger, Basel, p 242

Elton RF, Pinkus H (1966) Striae in normal men. Arch Dermatol 93:33–39

Garrone R (1975) Nature, genese et fonctions des formations conjonctives chez les spongiaires. Thèse Sciences, Lyon N° 7548

Garrone R (1978) Phylogenesis of connective tissue. In: Robert L (ed) Frontiers of matrix of biology. Vol 5. S. Karger, Basel

Gautray JP, Guingamp M, Miskulin M, Tixier JM, Robert AM, Robert D (1980) Modifications de la biosynthèse des macromolécules de la matrice intercellulaire du derme dans les vergetures mise en évidence par une méthode de biopsie et d'incorporation des précurseurs radioactifs. J Agrégés 13:464–473

Junqueira LCU, Cosser Melli W, Bretani RR (1978) Differential staining of collagen type I, II and III by sirius red and polarization microscopy. Arch Histol Jpn 41:267–274

Junqueira LCU, Bignolas G, Bretani RR (1979) Picro sirius staining plus polarization microscopy a specific method for collagen detection in tissue sections. Histochem J 11:447–455

Larson DL, Willis B, Linares H, Shetlar MR, Kischer CW (1976) The ultrastructure of collagen. Longacre JJ (ed). Charles Thomas Publisher, Springfield, Ill, USA

Longacre JJ (1976) The ultrastructure of collagen. Longacre JJ (ed). Charles Thomas Publisher, Springfield, Ill, USA

Mauss HJ (1972) Striaebildung und Varizenhäufigkeit bei Frauen. Geburtsh Frauenheilk 32:990–993

Robert L (1980) Precis de physiologie cutanée (F. Meynadier) Ed. de la Porte Verte, Paris

Rose GG, Yajima T, Maham CJ (1980) Human gingival fibroblast cell lines in vitro: electron microscopic studies of collagenolysis. J Periodontal Res 15:53–70

Svoboda ELA, Melcher AH, Brunette DM (1979) Stereological study of collagen phagocytosis by cultured periodontal ligament fibroblasts. Time course and effect of deficient culture medium. J Ultrastruct Res 68:195–208

Trelstad R (1981) Le collagene. La Recherche 12:312–321

Trelstad RL, Hayashi K (1979) Tendon collagen fibrillogenesis. Intracellular subassemblies and cell surface changes associated with growth. Develop Biol 71:228–242

Troisier E, Menetrier P (1889) Histologie des vergetures. Ann Gynecol 31:206–208

Accepted April 13, 1982