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Scanning electron microscopy of the tunica albuginea of the corpora cavernosa in normal and impotent subjects

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Abstract The tunica albuginea (TA) of the penis is thought to play a major role in the erection mechanism. It functions by compressing the subalbugineae venulae, which promotes the slower venous flow during erection, and provides a fibrous frame to give an inextensible support for the vessels and nerves. It acts as the inextensible enclosing structure which contains the erectile tissue and gives the erect penis its shape. The functions of the TA result from its structure, consisting for the most part of collagenic and elastic fibers. This study investigated, with the aid of scanning electron microscopy (SEM), the microarchitecture of the TA and the spatial relation of its fibers in ten impotent patients and in six control subjects with normal erectile function. The arrangement of elastic fibers in the TA seems to account for their function, which is to prevent the overstretching of collagenic fibers during maximum intracavernous pressure. In impotent patients, a reduction in the elastic fibers in the TA appears to produce disorders in the arrangement of the collagenic fibers. These alterations in the architecture of the TA in impotent patients can give rise top erection disorders.

Key words Tunica albuginea · Scanning electron microscopy · Elastic fibers · Impotence · Erection

The tunica albuginea (TA) of the penile corpora cavernosa consists mainly of collagenic fiber bundles, with some elastic fibers [3, 4, 8, 9]. The spatial arrangement of these fibers and their relationship to one another allows the TA to extend during erection and to regain its former configuration at rest [3, 8, 9]. The role played by the TA in the maintenance of erection seems most significant, as it promotes the constriction of the subalbugineum venous reticulum and of the veins that drain the TA, and provides an inextensible protective structure to the arteriolae and to the intracavernous nerves [2, 9, 11, 13]. Evaluation by optical microscopy has suggested the TA has a characteristic structure of collagenic fiber ansae linked by elastic fiber bridges [7–9]. The presence of elastic fibers allows the structure to resist an external force, by the exertion of a passive force [3, 8, 9]. Moreover, the elastic fibers could be fundamental in allowing collagenic fiber bundles to resume their undulating arrangement at rest [3, 8, 9]. A reduced concentration of elastic fibers in the TA and in the penile corpora cavernosa has been detected in patients affected by impotence due to several factors [17]. The reduced concentration of elastic fibers in the TA of the penis could account for a disorder of the TA and hence for impotence [10, 11]. The erection deficiency, moreover, seems to increase in severity the greater the reduction in elastic fiber concentration in the TA of the penis [10, 12]. The aim of this study was to explain, with the aid of scanning electron microscopy (SEM), the structural arrangement and any differences in elastic and collagenic fibers in the TA between normal and impotent subjects, and to establish whether these fibers can have a spatial arrangement which could better explain how the TA functions in the erection mechanism.

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Materials and methods

We selected from our clinic ten patients with an average age of 38.1 years (range 23–45 years), who had been affected by impotence for more than 1 year, and who had not suffered from diabetes, endocrinopathies, or metabolic disorders. All patients were subjected to hormonal screening (follicle-stimulating hormone, luteinizing

hormone, testosterone, dihydrotestosterone, estradiol, prolactin), their clinical histories were checked, and they were given a careful physical examination. All patients underwent RigiScan evaluation for three consecutive nights, which was considered normal if there was at least one erection event lasting more than 10 min, with a stiffness of more than 70% at the base and the tip [14, 18]. All patients underwent a surgical biopsy of the TA during penile prosthesis implantation. The biopsies, were all taken from an area of about 10 × 34 m at the base of the penis, along the edge of the incision into the corpus cavernosum. The biopsy specimen was then divided into two, and prepared for SEM and optical microscopy.

Preparation for optical microscopy

The specimen was fixed in 10% buffered formalin and embedded in paraffin, and a 5-μm section was stained with hematoxylin and eosin, and Weigert's stain (for elastic fibers). The elastic fiber count was performed on 10–12 fields (× 40 magnification) on five serial sections (10–12 fields for each section). Normal counting techniques were used, such as mitotic counting or, more specifically, silver staining nucleolar organizer region (AgNOR) counting [6].

Tissue preparation for SEM

A portion of the same specimen was stabilized with 2.5% glutaraldehyde in 0.13 M phosphate buffer (pH 7.3) for 2 h at room temperature. The sample was washed in buffer and dehydrated in a graded series of ethanol and critical point dried, using carbon dioxide as the intermediate fluid. Dry tissue blocks were mounted on aluminum stubs using carbon paint. The samples were coated for optical secondary electron emission with a thin gold layer deposited by a sputter-coating unit. All the specimens were observed at 20 kV with a scanning electron microscope Cambridge Mark 3. A second group of six control patients was selected with normal erectile function and an average age of 36 years (range 21–55 years), who were not suffering from endocrinopathies, diabetes, and connective or metabolic disorders. Four of these patients had congenital penile bending, one patient had cancer of the penis, and one was a transsexual patient who had undergone surgical intervention without hormonal therapy. They were all subjected to RigiScan evaluation for three consecutive nights, using the same reference parameters as for the first group [14, 18]. All patients underwent surgical bisopy of the TA and the biospy specimens were prepared for SEM and optical microscopy.

Results

All patients presented a hormonal profile within the normal range. The results of the nocturnal penile tumescence recording (NPTR) of both impotent patients and normal subjects are shown in Table 1. Optical microscopy findings in normal and impotent subject are shown in Fig. 1 and Table 2, SEM observations in normal subjects (Fig. 2a,b) show a significant reticulum of elastic fibers which stretch the ansae of collagenic fibers (Figs. 2c,3a). The collagenic fibers present an undulating appearance, with a nearly uniform periodicity, which appears to be held constant by the elastic fibers (Fig. 2c).

Elastic fibers differentiate themselves from collagenous fibers by their decreased diameter and by the absence of the typical period of the collagenous fibers,

Table 1 Nocturnal penile tumescence recording (Rigiscan) parameters in normal and impotent men (mean ± SD). Differences were assessed using the unpaired Student's *t*-test

	Normal	Impotent
Patients (*n*)	6	10
Age (years)	36 (21–55)	38.1 (23–45)
Frequency[a] (time)	3.6 ± 1	1.5 ± 1
Duration[a] (min)	50.00 ± 15.5	10.6 ± 8.60
Circumference expansion[a]		
Tip	3.6 ± 0.5	1.8 ± 0.7
Base	4.4 ± 1.0	2.01 ± 0.8
Rigidity[a]		
Tip	85 ± 12	52.40 ± 7.0
Base	89 ± 10	55.00 ± 2.9
P value		< 0.0001

[a] Mean ± SD

which is very visible at high magnification (Fig. 3b). In the patients suffering from impotence, a significant reduction in the number of elastic fibers of the TA was observed. The structural arrangement detected in the TA of the normal subjects appeared to be modified in the impotent patients, where the lack of elastic fibers led to a clear alteration in the arrangement of the collagenic fiber ansae, which show a clearly augmented period, up to a complete flattening and the consequent loss of the physiological structural arrangement (Fig. 4a,b).

Discussion

The role played by the TA in the erection mechanism seems most significant. During the first phases of the erection, the TA resists the increase in intracavernous pressure because of its structure, which is very poorly extensible and elastic; moreover, it allows the erect penis to maintain its shape. In particular, the TA exerts compression on the subalbugineum venous reticulum and on the venulae which drain the TA, with a consequent block of blood flow from the corpora cavernosa [2, 15]. This mechanism plays a fundamental role in the maintenance of the erection.

Moreover, the tunica albuginea branches, within the corpus cavernosum, to form a real fibrous frame [8, 9], which acts as an inextensible support for the arteriolae and for the intracavernous nerves. During erection, the overstretching of these neurovascular structures could lead to functional disorders which would appear as an erection deficit [8, 9]. Moreover, the tunica albuginea branches, during the period of maximum intracavernous pressure, to allow the diameter of the corpora cavernosa to be constant in every section of the penis, without going beyond given values. If there was independent and uncontrolled overstretching of the penile sections, the shape of the penis would vary with time, during every erection event, making the organ useless

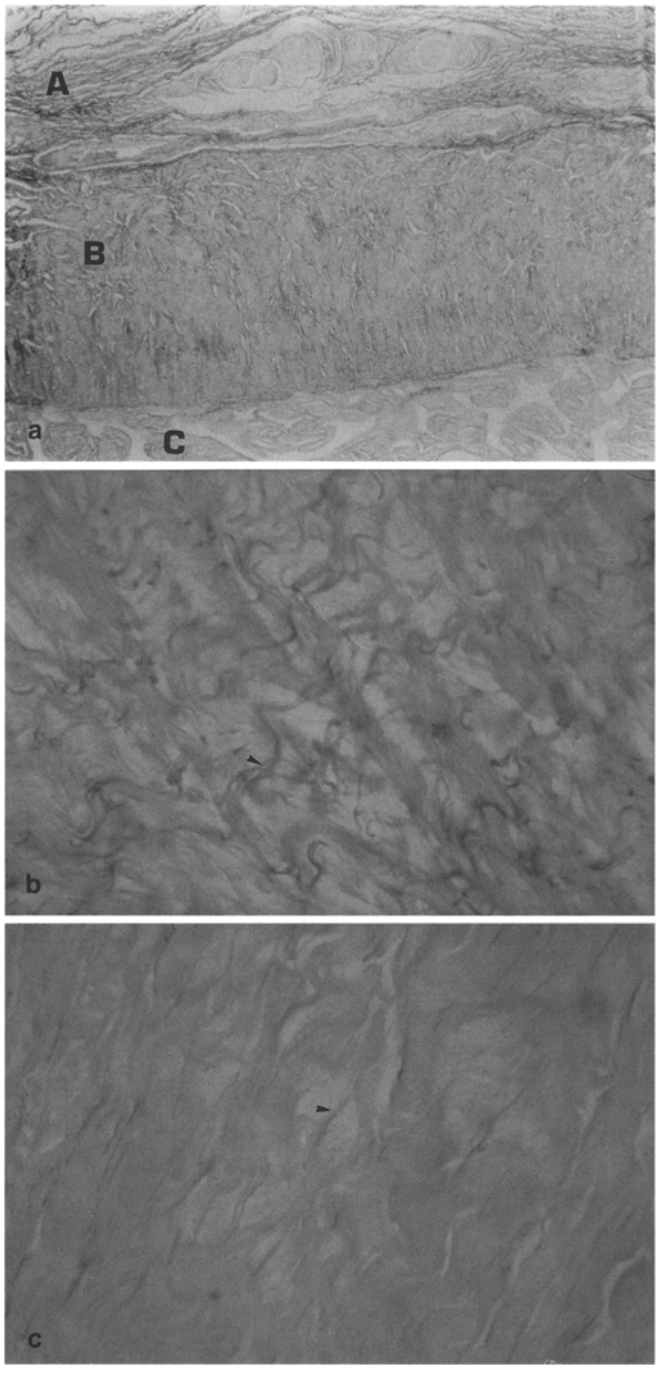

Fig. 1 **a** Light microscopy examination of the TA of a normal subject. Low magnification (× 20) allows easy identification of the TA (*B*) found between the superficial tissue (*A*) and the cavernous tissue (*C*). *Arrow* indicates the superficial venous plexus. **b** Light microscopy examination of the frontal section of the TA in a normal subject. Concentration of elastic is noted. Weigert's stain, × 400 **c** Light microscopy examination of the frontal section of the TA in an impotent patient. An obvious reduction of elastic fibers is noted (*arrow*). Weigert's stain, × 400

for sexual purposes. The TA is able to perform these functions because of its characteristic structure of collagenic fibers, which are spatially and functionally linked with a certain amount of elastic fibers.

Table 2 Quantitative evaluation of elastic fibers (mean ± SD) in the TA of normal and impotent men. Differences were assessed using the unpaired Student's *t*-test

	Impotent patients (× HPF)[a]		Normal men (× HPF)	
Patient No.	1	23	1	130
	2	36.5	2	122
	3	44	2	143.2
	4	36		
	5	54	4	113
	6	31	5	139.6
	7	38	6	149
	8	49		
	9	58		
	10	44		
	P value		0.0001	

[a] High power field

Observations by optical microscopy have led many authors to postulate a bidimensional structure for the TA (Fig. 1b,c) [4, 7–9]. The elastic fibers follow a longitudinal path, linking the collagenic fibers by forming bridges between the ansae of the collagenic bundles [4, 7–9]. SEM observations have led to a larger, tridimensional perspective of the TA structure in normal subjects (Figs. 2c,3a) and in impotent patients (Fig. 4a,c), by allowing new hypotheses on erection pathophysiology.

In the TA of normal subjects, collagenic fiber bundles, which have a close texture, present an undulating appreance which resembles hair waves (Fig. 2c). The period of the waves of the collagenic fibers is maintained by a dense net of elastic fibers, which link one collagenic wave to another by forming an actual biological link (Fig. 2c), the function of which could be the prevention of overstretching of collagenic waves during erection and allowing the collagenic fibers regain a resting condition as the intracavernous pressure decreases during penile detumescence. This arrangement of elastic fibers would indicate the TA and corpora cavernosa have elastic properties [3, 16].

The TA can resist an overstretching of 600–700 mmHg; beyond this figure, the elastic fibers may rupture, and the structure of the TA is irreparably altered, with the consequent loss of its elastic properties [3]. This situation, then, would be similar to that in impotent patients, who were found to have a strong reduction in elastic fibers in the TA alone and in the corpora cavernosa [17]. This reduction seems to increase as erection function deteriorates, and it is independent of the recognized etiological factor [11, 12, 17]. Furthermore, reduced penile extensibility has been detected in impotent patients [5].

SEM observations of the TA of patients affected by impotence due to several factors showed an almost complete absence of elastic fibers among the collagenic fiber waves, with a disorientation of the latter and some loss of their undulating appearance, assuming a flatter appearance.

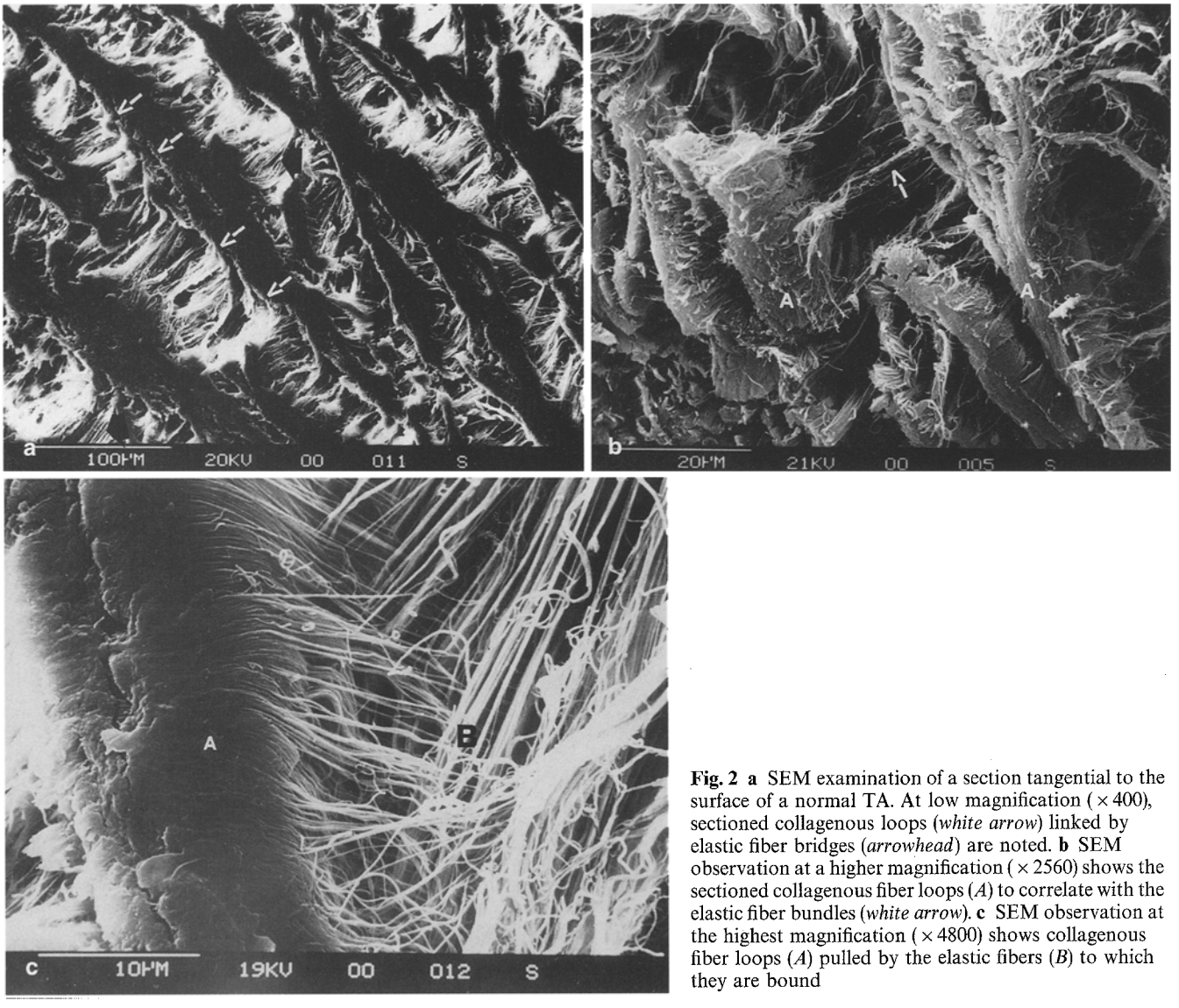

Fig. 2 **a** SEM examination of a section tangential to the surface of a normal TA. At low magnification (× 400), sectioned collagenous loops (*white arrow*) linked by elastic fiber bridges (*arrowhead*) are noted. **b** SEM observation at a higher magnification (× 2560) shows the sectioned collagenous fiber loops (*A*) to correlate with the elastic fiber bundles (*white arrow*). **c** SEM observation at the highest magnification (× 4800) shows collagenous fiber loops (*A*) pulled by the elastic fibers (*B*) to which they are bound

Such a TA, hence, presents a reduced elasticity which will lead to a reduced or to a totally absent function. By analyzing individually the several functional hypotheses ascribed so far to the TA, we can see how reduced elasticity can alter its individual functions and lead to impotence.

Compressive function of the TA on the subalbugineae venulae and on the venulae that drain the TA [2, 15]. A less elastic TA will resist poorly the increase in intracavernous pressure during the erection; in this way the subalbugineum venous compression could be totally or partially absent, with venous leakage and hence impotence. On the other hand, the arterious inflow cloud partially counterbalance [1] the absence of venous compression exerted by the TA, with an increase in the intracavernous pressure, leading to a greater stretching of the TA. This counterbalancing mechanism could be a subjective response, and could also occur after the intracavernous administration of vasoactive drugs. This would explain why some patients with moderate venous leakage respond to intracavernous treatment with vasoactive drugs. When, instead, a simultaneous disorder of the arterious inflow is present, the counterbalance mechanism does not work, leading to impotence which does not respond to intracavernous treatment.

Function of the fibrous frame of the corpora cavernosa [8, 9]. A reduced amount or the absence of elastic fibers in the TA of the penis and in tunica albuginea branches produces a stretching of collagenic fiber ansae. The consequence is that the TA and its branches will be more yielding, when the intracavernous pressure increases during erection. Thus, the nerves and intracavernous arteriolae will lose their inextensible support and will be subjected to a structural overstretching, even if limited to a few microns. This could give rise

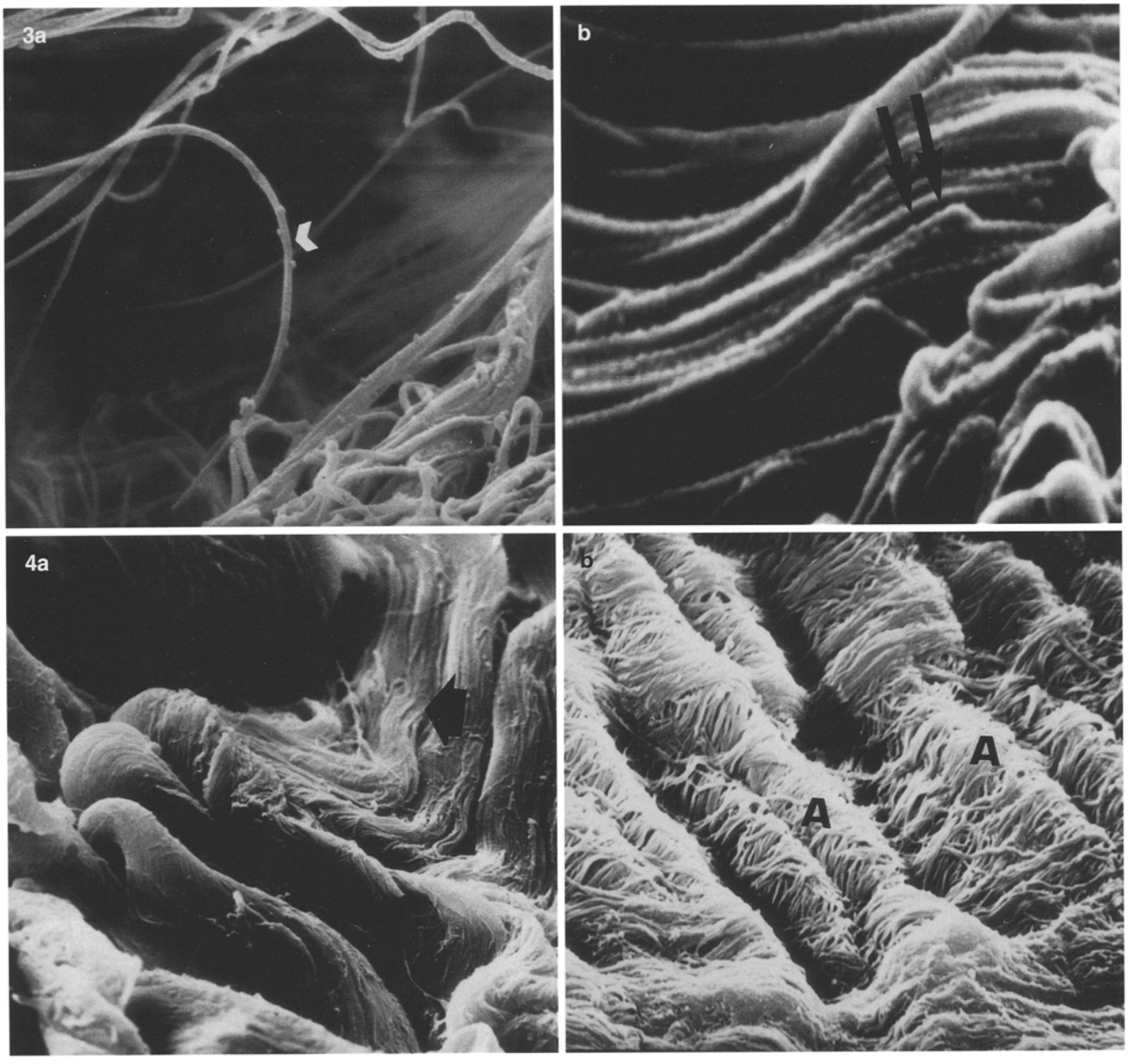

Fig. 3 a Micrograph shows an elastic fiber at very high magnification (*white arrow*) and many collagenous fibers (*black arrows*) (× 25 800). **b** High magnification of collagenous fibers show their characteristic period. The thickness of collagenous fibers results of 50 nm

Fig. 4 a Observation at × 4800 shows collagenous fiber loops (*arrow*) that appear to be more relaxed in the absence of elastic fiber. **b** Observation at × 10 600 shows the collagenous fiber (*A*) bundles without elastic fiber

to hemodynamic disorders and to alterations of the intracavernous neurotransmission [2].

TA as a container (enclosing structure) of the erection tissue. The cavernous tissue of both the corpora cavernosa is delimited by a wall formed by the TA, the inextensibility of which allows the penis to maintain a steady shape during the erection and during sexual intercourse. By likening the two corpora cavernosa to two cylinders containing fluid under pressure, a reduced resistance in the cylinder wall can under pressure produce an increase of the cylinder radius, with a consequent inner pressure drop (La Place's law). Thus, if the TA loses its elastic capacity, it can resist less than before the dilation of the corpora cavernosa during the erection, and a very slight increase of the corpora cavernosa radius, with a consequent pressure drop and impotence, will occur.

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