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# Changes of the elastin compartment in the human meniscus

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**Summary.** We report on the elastin, collagen and ground substance compartments in the human meniscus and their interrelationships. Elastin is found in neonates and shows both an arrangement parallel to the collagen fibre and into net-like structures. Branching, caliber inconstancy, rupture and the phenomenon of the "rubber band" are findings within the different forms of the meniscopathy. The function of the elastin compartment cannot be visualised without suggesting "puncta fixa". The morphology of the collagen-elastin-junctions has been described and their possible mode of function is discussed with the help of a model.

**Key words:** Meniscus – Meniscopathy elastin – Elastin compartment

## 1. Introduction

The special functions of the meniscus as an important component of the knee is based on its phylogenetic origin, from which viewpoint the meniscus can be considered to be a special element of the tendon. The actual positioning of the meniscus depends on the posture of the articulation and is determined partly actively (by muscles inserting in the posterior horn) and partly passively (by pressure from the femoral condyle). Every change of position within the articulation is related to an alteration of the form of the meniscus (Fig. 1) possible because of its elastic character (Müller 1982).

The arrangement, flow and imbedment of the collagen network in the ground matrix (which consists mainly of glycoprotein and proteoglycan) have been described in numerous models (outline: Höpker 1984). These models have one thing in common (Clarke 1971): the mechanical stability of the meniscus refers to two main flow directions of the bunches of collagen fibres in longitudinally and transversally arranged fibre groups. To date, these have been described often but the elastin fibres have only reported

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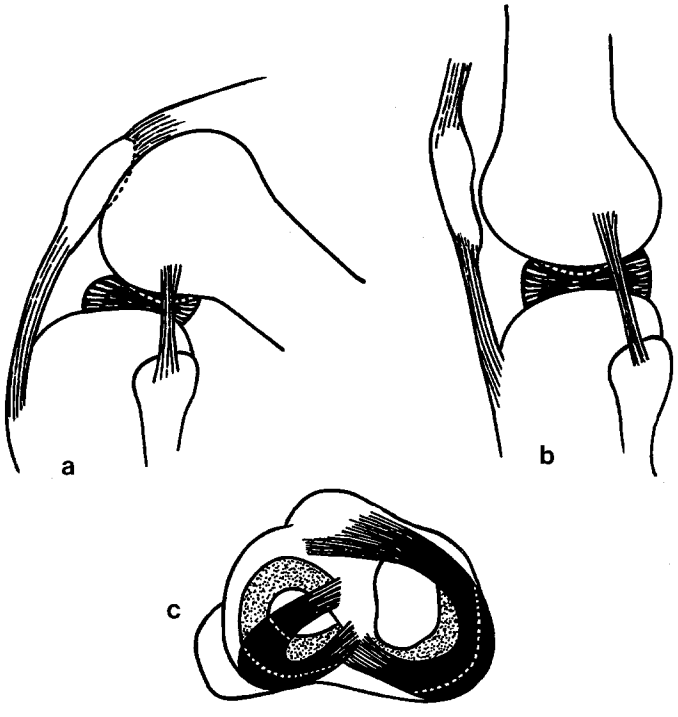



**Fig. 1** Lateral view of the human knee joint during flexion **a** and extension **b.** Alterations of the form of the meniscus in the topview **c** during flexion deformed meniscus (lateral and medial meniscus; black)

once (Raszeja (1938). Their arrangement in the meniscus, their arrangement and their possible starting points are not known.

In physics load dependent deformations of elastic bodies in the region where load/deformation are linearly related (stress-tension-diagram) persist as long as the mechanical stress is reversible. In this proportional area there are no irreversible changes of structure of fibrocartilage to be expected. The phenomenon can only be explained by assuming that the stressable elastin fibres perform this function.

Chronically traumatized menisci (with chronic meniscopathy) show a characteristic macroscopic picture: a brown change in colour of the surface, and a change in sectional area. Several studies have tried to identify lipofuscin pigments, ferruginous and iron-free decomposition products of haemoglobin, but the brown change is still of uncertain origin. It is possible that changes in the elastin network which are increasingly produced in several forms of chronical meniscopathy leads to such a change in colour.

Clinically and morphologically it is clear that cicatrized solitary zones of rupture are rarely the site of a further rupture. Further dissection usually develops in the more distant neighbourhood, not directly in the cicatrized zone (which points to an identical injurous mechanism mediated by the

articulation). The question is again: is the elastin network of the meniscus one of the causes of this phenomenon?

In the human meniscus elastin material can be identified by electron microscopy sporadically (Ghadially 1983) on examination with the light microscope there is no indication of elastin and no data to support a particular localisation.

This study attempts to give an answer to the following questions:

1. Does a defined elastic apparatus in the meniscus exist? If so what functional significance may be attributed to it?
2. What is the relationship and the interaction between the collagen and the elastin network.
3. Is it possibile to differentiate the various forms of the elastin with morphological methods? Is there perhaps a correlation with injuries of the meniscus?
4. Are there – by analogy with the collagen network – different arrangements and directions of elastin fibre flow?
5. Elastic fibres can only work functionally, if they are fixed on both ends in a mechanically stable way (puncta fixa). Is this true for the human meniscus?

## 2. Materials and methods

The patients in this study (men, $n=8$) come from a sports hospital. All patients have done sports throughout several years, all were playing football, some tennis in addition. They are between 30 and 35 years old and are variably overweight.

The indication for operation was always a tear of the meniscus with a serious clinical symptomatology, without any tendency to amelioration. After the meniscotomy the material was divided into three parts:

1. for scanning electron microscopy,
2. for transmission electron microscopy,
3. for light microscopy.

Additionally, some menisci of neonates and sucklings ($n=5$) from autopsies were examined in the same way. The specimens were divided under the stereomicroscope.

*A. Scanning electron microscopy.* Specimens measuring $0.5 \times 0.5 \times 0.5$ mm (about 10 from each patient) were excised from regions situated in the neighbourhood of the grossly evident lesions. The fragments were immediately fixed in 2.5% glutaraldehyde in cacodylate buffer at pH 7.2 for 1 h at 4° C and postfixed for 30 min in 1% osmic acid in the same buffer. After dehydration, the specimens were dried at critical point in $Co_2$ in the Polaron apparatur E-3000 and coated with gold. Then, they were examined with ISM 35 (Jeol, Tokyo; Japan) scanning electron microscope at a 40° angle and 25 kv field emission.

*B. Transmission electron microscopy.* We fixed specimens about $1 \times 1 \times 1$ mm (about 10 from each patient in cacodylat buffered glutaraldehyde (pH 7.2), subsequently the preparations were washed 3 ×; followed by after-fixation in cacodylat buffered osmic tetroxide for 2 h, washing in water and an ascending alcohol series. Afterwards there was dehydration in propyleneoxide; propyleneresin-mixture 3:1 and 1:1 each during 1 h. Vaporization of the rests of propyleneoxide at 48°, imbedment in epone-araldite and polymerization at 60° C was carried out for 16 h.

*Semi-thin-section* ultracut-E-microtome (Reichert-Jung, Nußloch; FRG) double-staining with fuchsine and methylene-blue. Ultra-thin-section (300–800 A): contrasting with uranylacetate and phosphorwolfram acid. Diagnosis with EM 10 (Carl Zeiss, Oberkochen; FRG).

*C. Light Microscopy.* The rest of the material was fixed with formalin (10%) followed by desiccation in an ascending alcohol series and acetone; imbedding in paraplast and sectioning at 3–20 μm. Elastin staining Weigert, was performed with and without staining the cores.

Scanning and transmission electron microscopy were performed at the same time as the light microscopical study. All menisci showed old and new injury. Larger cicatrices as well as complete replacent tissues were not examined.

## 3. Results

The infant meniscus shows a smooth or lightly undulating surface which is formed by closely ordered returning slings of the collagene fibres. By examination with the light microscope a remarkably profuse elastin network is observed. It is reticularly branched (acute angled), the elastin fibres follow the bunches of collagen fibres in a parallel direction.

The infant meniscus lacks the mainly longitudinal direction of collagen fibre flow which is produced with increasing functional loading; all flow directions of the collagen network are found in about the same portions.

Apart from the elastin fibres running in parallel to the collagen fibres there are fibres arranged at a constant angle to the collagen, rarely flowing into a net-like design (Fig. 2).

The arrangement of the elastin is essentially preserved for adult (or more stressed juvenile) menisci (Fig. 3). Elastin fibres are pressed between larger fascicular bundles of collagen fibres. Several phenomena can also be seen:

– The elastin fibres show differences in caliber where the finest fibres arranged in parallel groups are found imposed on the collagen fibres. Differences in caliber within an elastin fibre are not found.
– By means of light microscopy large caliber elastin is identified only along short distances. In the most cases it is found together with a warping phenomenon of the collagen network.
– In areas in which original bundles of collagen fibres are crossing, the elastin fibres seem to transfer to non-parallel collagen fibres. In these wedges the chondrocytes are in close connexion to the elastin.
– With increasing age net-like elastin structures are more and more developed.

While the findings are seen more or less distinctly in uninjured menisci (light microscope), they are found much more frequently in chronical meniscopathy. In these cases several additional alterations can be pointed out which are not generally found in uninjured or only insignificantly harmed menisci. These include:

– Loops and pleating in the elastin (Fig. 4). Such figures are found within the destruction zone, generally they occur, however, more frequently in the direct surrounding mainly uninjured fibrocartilage. The elastin fibre does not follow the direction of the collagen fibres flow; such figures are independent of the arrangement of chondrocytes and vessels. This is named the "phenomenon of the rubber band".
– Often superficially situated sprouts (Fig. 5) of net-like branched fibres are observed. These fibres constant in caliber. They do not show signs

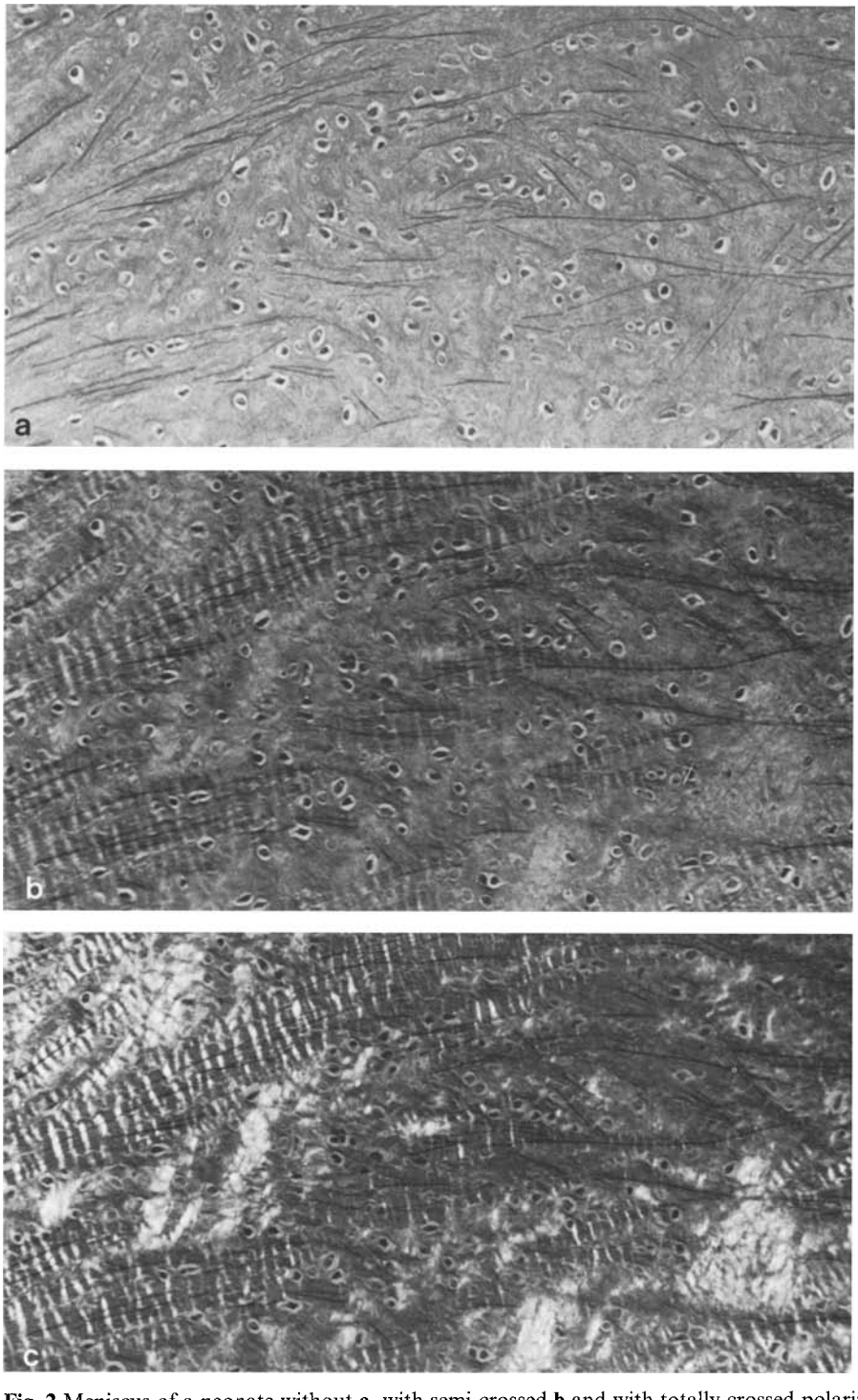
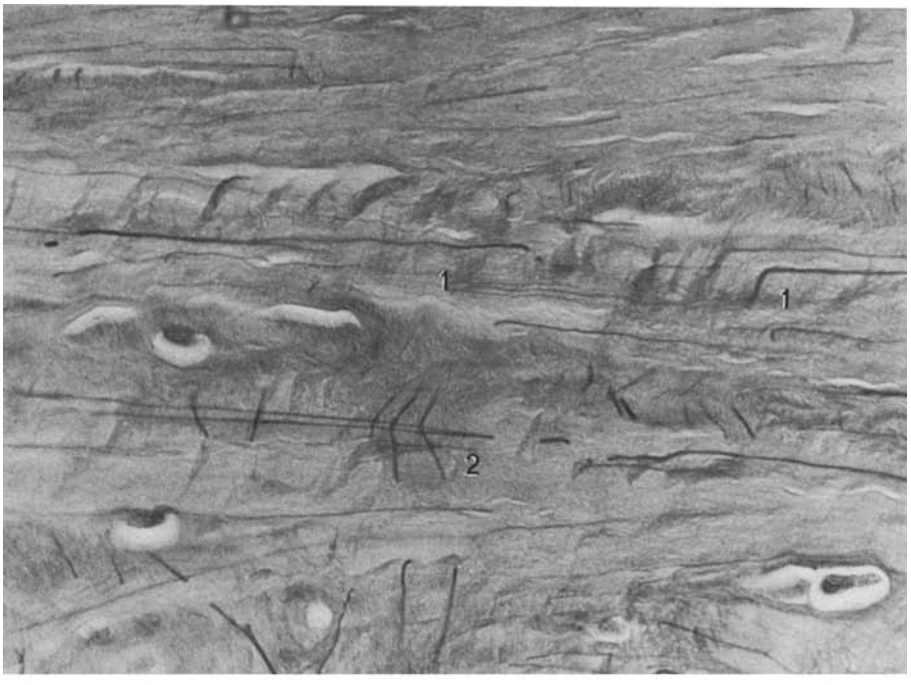



**Fig. 2** Meniscus of a neonate without **a**, with semi-crossed **b** and with totally crossed polarization filter **c**. In the most cases (but not always) the elastin shows a flow parallel to the collagen fibre. Elastica-Kernechtrot ×180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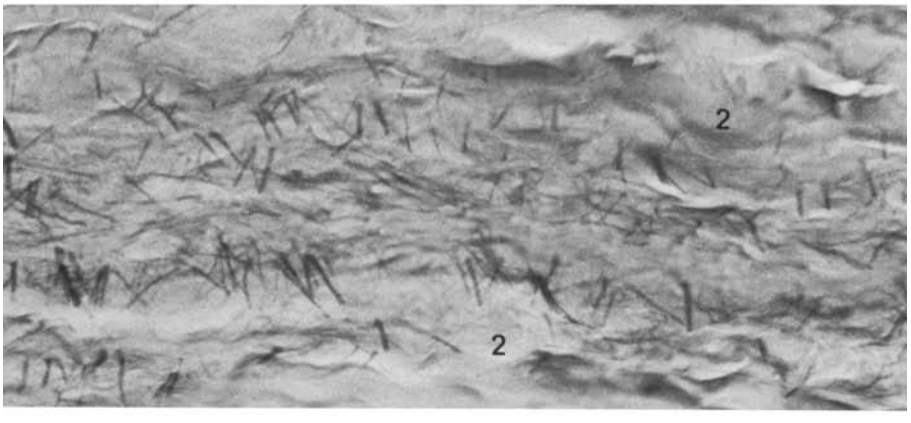
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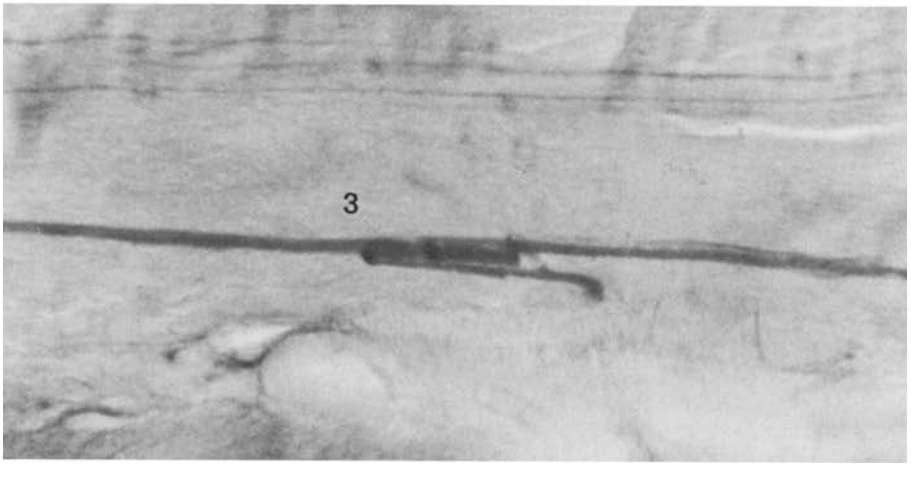
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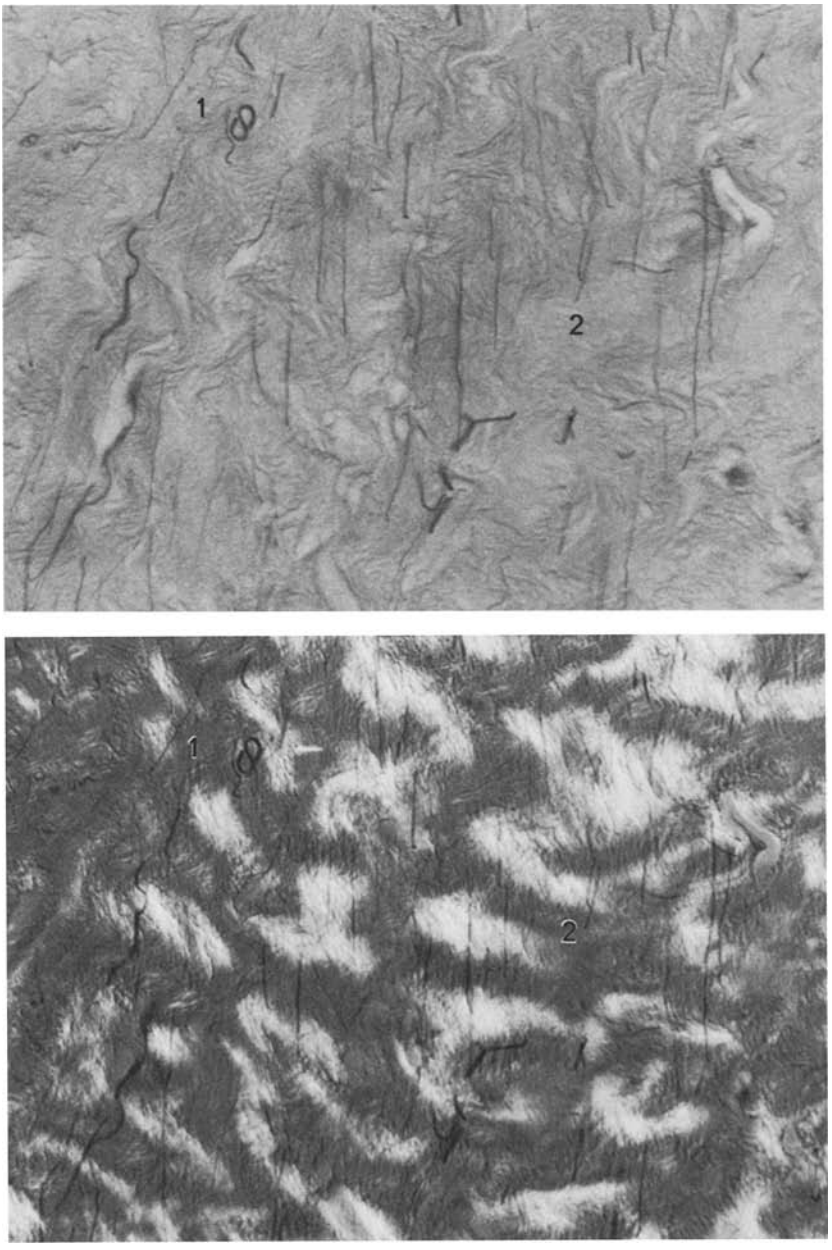


**Fig. 4** Elastin with inconstancy of caliber arranged diagonally-longitudinally to the collagen with the phenomenon of the "rubber band" (*1*). Elastin fibres do not flow in parallel but are acute-angled. Elastica staining × 300, with and without polarization

**Fig. 3** Preserved (light microscopically uninjured) parts of the meniscus. Ruptured elastin fibres (*1*) showing a flow parallel to the collagen fibre. Organization of an elastin net (*2*) with transversal-spiral arrangement. Clinched caliber inconstant elastin (*3*), flow parallel to the collagen fibre. Elastica-Kernechtrot × 230 (*1, 2*); × 300 (*2*); × 470 (*3*)

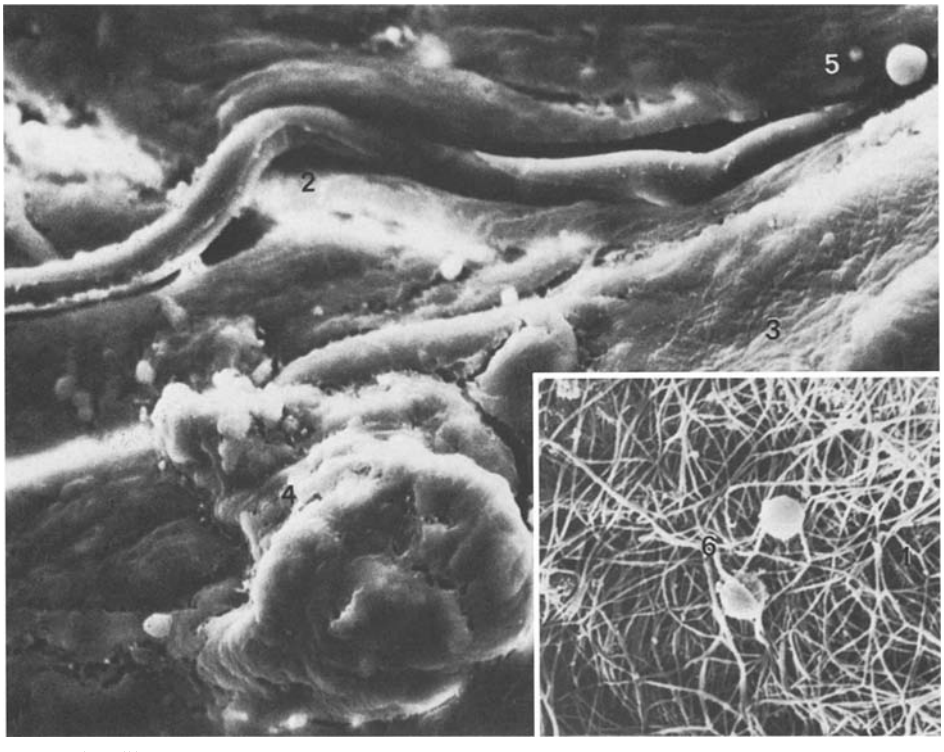


**Fig. 5** Clew-like agglomerated elastin imposed on the surface of the meniscus within a chronical meniscopathy (*1*). Multiple connexions and branchings, lipid droplets (*6*). Singular elastin fibre (*2*) in ruptured bunches of collagen (*3, 4*). Surface partly preserved (*3*). Lipid droplets in various dimensions (*5, 6*). ×800 (*1, 6*); ×7,100 (*2, 5*)

of splinters, agglutination, up-setting or fraymes, which occurs in damaged collagen.

– Superficial stumps of cracked amorphous fibrillar structures (Fig. 6) are sometimes identified imbedded in a damaged matrix of collagen fibres. These stumps are assumed to consist of ruptured elastin fibres.

The caliber inconstancy is an important characteristic of the elastin network in cases of chronical meniscopathy (Fig. 7). Loops and pleatings were not found in connexion with uninjured menisci.

In the light microscopy coiled elastin fibres are seen in the area of old acaryote scars (Fig. 7). In new dissections they can be pursued up to the surface, lying totally free. With the scanning electron microscope fibrillar elements are found which correspond well to this (Figs. 5, 6). The fibres present a round cross-section (they are partly split), transversally flowing superficial structures (resembling the banding pattern of the collagen fibres) are not observed. Frequently filiform impositions of a non-identifiable material are found without a recognizable banding pattern (Fig. 5).


**Fig. 6** Totally destroyed surface of the meniscus within a chronical meniscopathy. Few preserved returning slings (*4*) besides deep tunnel-like phenomena (*1*). Rests of ruptured fascicles (*3*). Splintered (perhaps: elastin) fibres reaching out of the tunnel-like defects; smooth, structureless ruptured surfaces (*2*). × 800

Elastin equivalences are not only found in solitary but also in cluelike agglomerated bunches (Fig. 5). In these areas it seems as if the ground matrix and the collagen network are completely destroyed and only the agglomerated elastin network remains.

By means of transmission electron microscopy destructive phenomena in the collagen network, in the form of solitary pleatings and "necrosis"-like unilateral fibrillar deficiencies (Fig. 8) are observed. Moreover there are plenty of longitudinal splinters with a complete dissolution of the fibre, with osmiophilic granular debris remaining sometimes showing an apparent banding pattern. On top the collagen fibres are torsived under tension in a longitudinal direction (Fig. 9), in the cross-section numerous differences in caliber are demonstrable.

Elastin is found in several varieties (Fig. 9). In cross-section small osmiophilic zones (in longitudinal section formed as stripes) are differentiable. The exterior circumference is formed by a coatshaped zone of finest fibrillar structures. These findings are not constant as they depend on the caliber and the optical density of the elastin section. The elastin areas are not

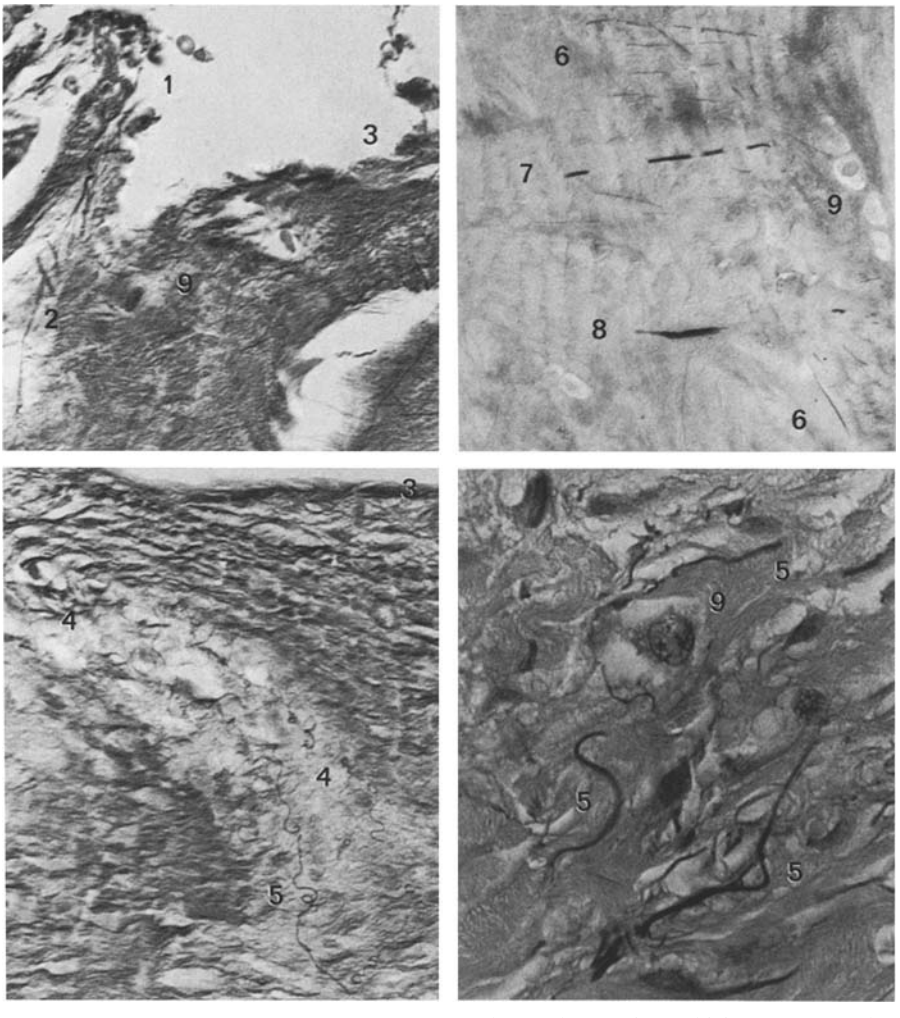


**Fig. 7** Superficial alterations within chronical meniscopathy (section's thickness: 20 μ). Elastin fibres reach directly up to the destroyed surface (*1*), they can be persued to the oedematous zones of warping in the deeper segments (*2*). Partly preserved partly destroyed surface (*3*). Completely cured scar (poor-cored) (*4*) with a multiply torsaded spiralicly coiled elastin fibre (*5*; section's thickness: 20 μ); elastin with inconstancy of caliber (*6–8*), chondrocyte core vacuolarely alterated with an oedematous court of warping (section's thickness: 5 μ). Elastica staining × 90–105

precisely confined, a banding pattern or regularly arranged zones of networking was not demonstrated.

Collagen fibrils and elastin are often connected to one another (Figs. 8–9). Clinging to the elastin there are partly parallel flowing fibrillar collagen fibres, some singular, some grouped with occasional acutely angled branches. With large, matured elastin fibres this observation can be made

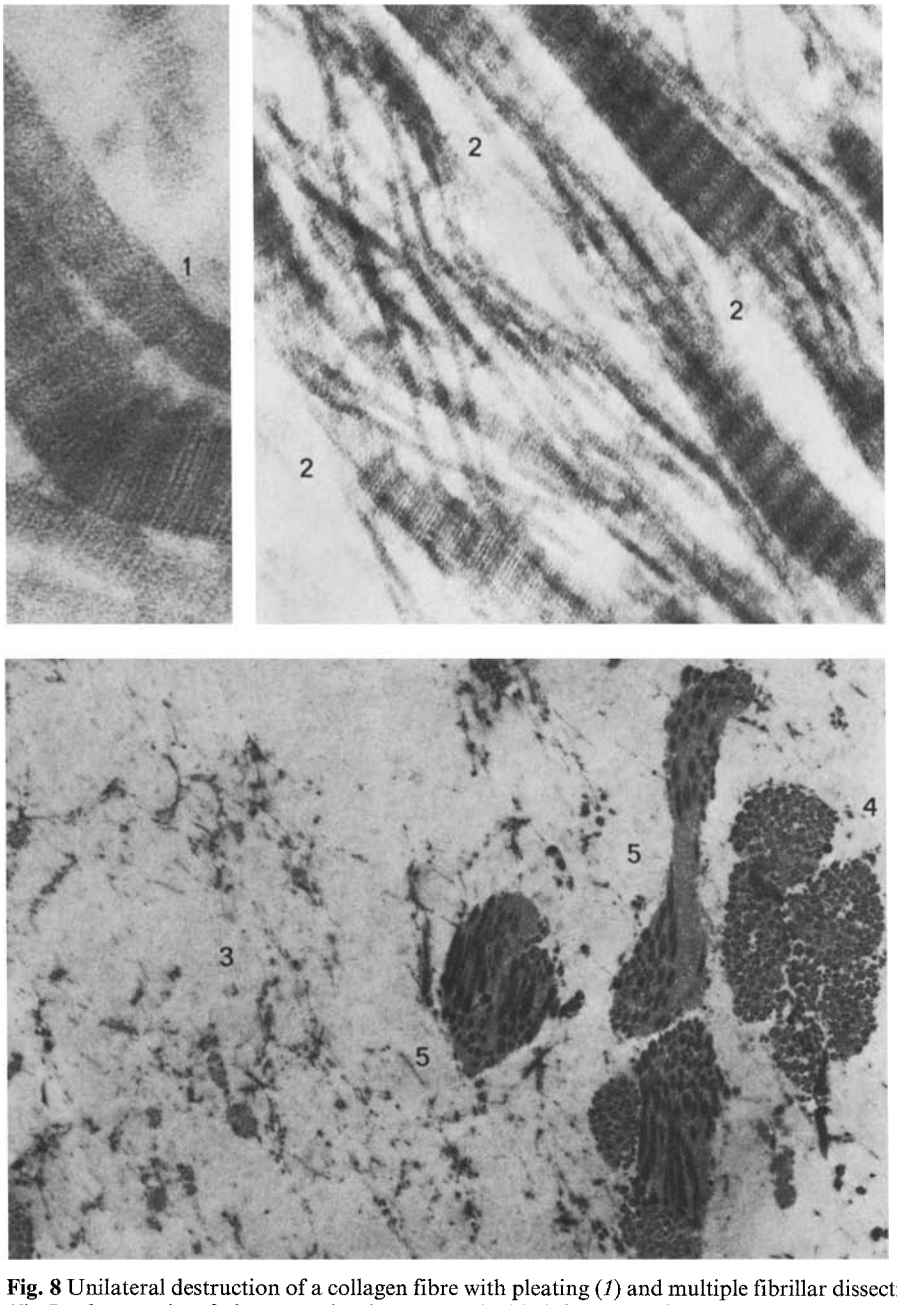


**Fig. 8** Unilateral destruction of a collagen fibre with pleating (*1*) and multiple fibrillar dissection (*2*). In the matrix of the ground substance embedded focusses of elastin with transversally striping clearly demonstrable (*4, 5*). ×133,300 (*1*); ×84,000 (*2*); ×14,600 (*3–5*)

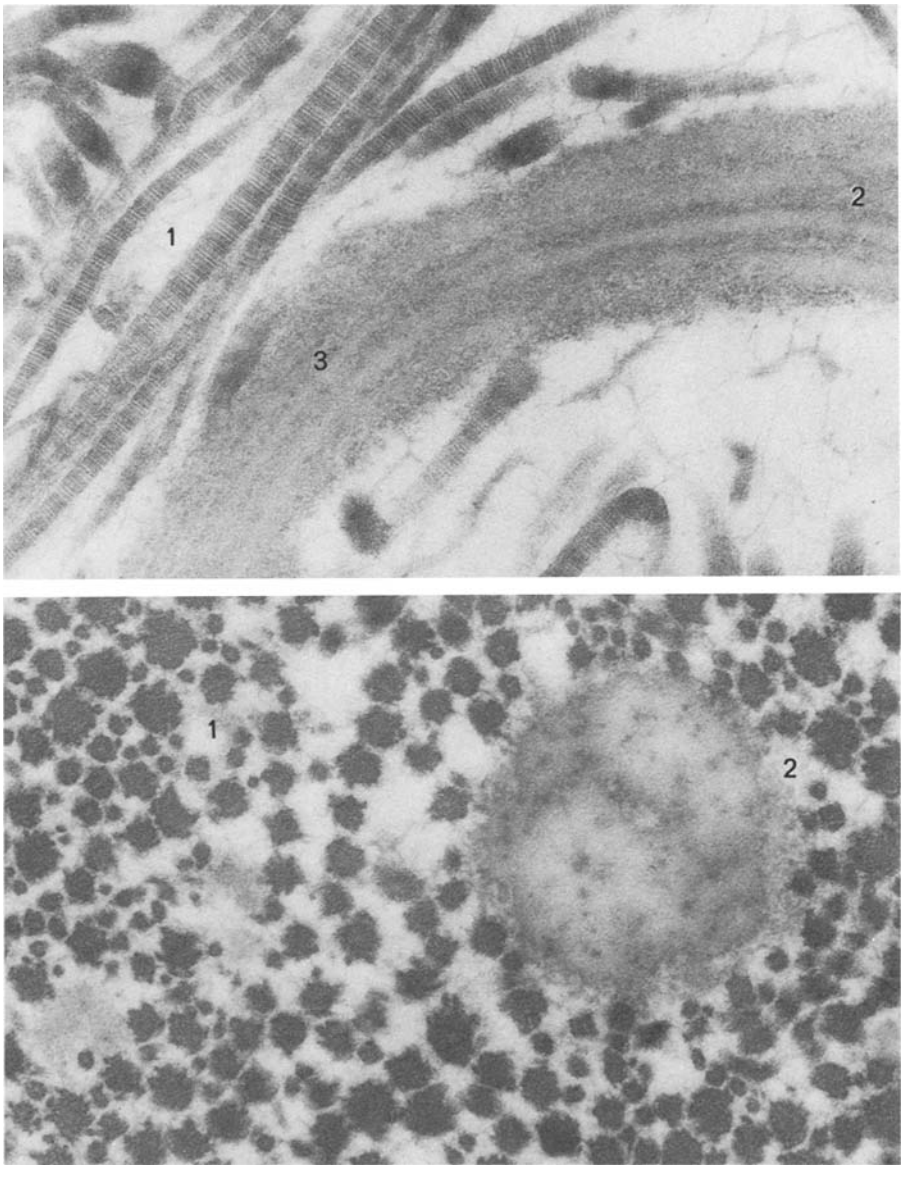


**Fig. 9** Longitudinally and transversally cut elastin (*1*) in preserved but clearly distortioned collagen (*2*). The elastin is marked by a finest-fibrillar surface and longitudinal structures. Variously bridge-like tight connexions between collagen and elastin (*3*). × 71,400 (*1–3*)

quite frequently. Distinctly bordered fibrillar structures are also cross within the elastin demonstrable in longitudinal and cross-sections. In other areas they enclose the elastin in form of palisades. Smaller or larger bundles of collagen fibrils are also found. In all cases we found elongated arrangent, a characteristic cross-section, rhythmical banding (even within the elastin)

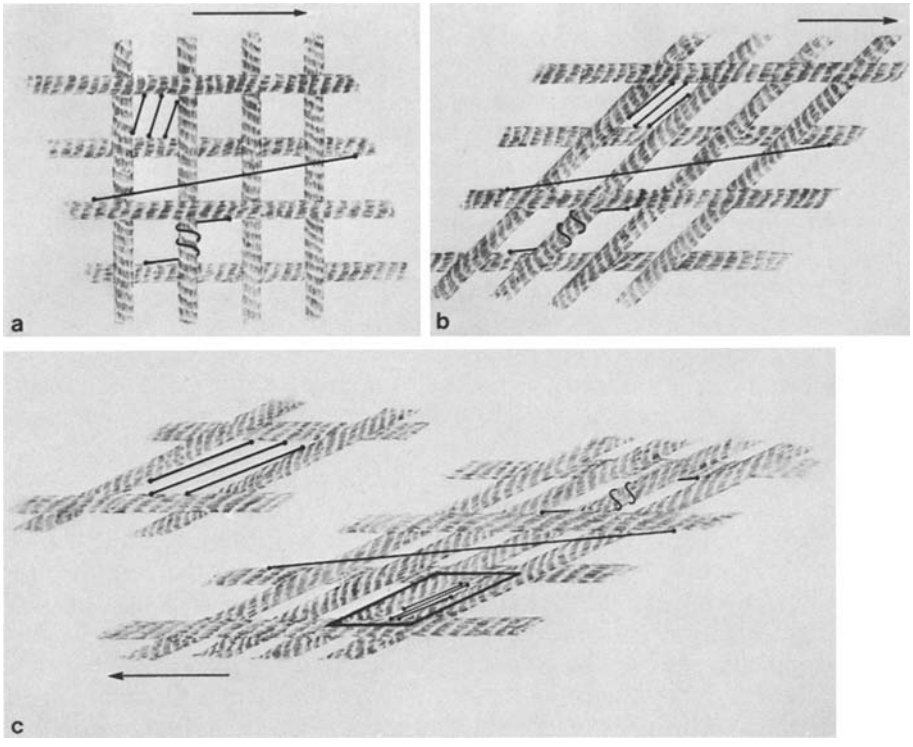


**Fig. 10** Scissors-like grating model of the collagen compartment in the human meniscus. The model considers the deformation of the meniscus during movements of the knee joint. The diagonally flowing collagen fibres (longitudinal and transversal fibres) are shifted in opposite directions (*a, b, c*), the angle of flow of the collagen changes. This leads to an alteration of the distances between the points of insertion of the elastin within the collagen, so the elastin is tightened. Besides short elastin fibres directly inserting in the neighbour collagen fibre longer stripping fascicle-fibres could also be imagined. The occurence of clinging phenomena may be envisaged

in the longitudinal section – these fibrils can clearly be identified as collagen fibrils.

Additionally, there are bridge-like connexions which are exceptionally fine and have a transverse pattern, however, for the most part, they are amorphous in structure.

The net-like connexion between the collagen fibrils and the elastin is assumed to be especially marked with the poorly-structured elastin. Networks of larger and elder fibres with collagen are not observed so often, more frequently there are ruptures and splinters.

## 4. Discussion

### *A. Fine structure of the elastin*

According to our knowledge the human meniscus consists of collagen type I (outline: Höpker 1984). The elastin shows a fibrillar substructure (Dryll

et al. 1981) which is particularly distinguished in the border zone and seems to correlate with the differences of light intensity in longitudinal and transverse sections (Cleary and Cliff 1978; Cleary et al. 1981). The researches of Cleary et al. (1981) suggest the elastin associated microfibrils established a network for the organization and imposition of precursor elastin. The actions of lysyloxidase and cross-linking-enzymes converts the precursor elastin to amorphous elastin (Robert et al. 1984). The limited total surface of the fibrillar ground structure leads to an increase of the amorphous component of the elastin with time and is the reason for the expansion in caliber of the elastin fibril (Banfield and Brindley 1963; Robins 1982). This phenomenon seems to have nothing to do with the actual genesis of elastin (Cleary et al. 1981; Oakes and Batty 1982; Bressan et al. 1983). Perhaps the newly produced elastin may impinge on the collagen fibrils, a thesis which could explain our electron microscopical findings (Ichimura and Hashimoto 1982).

The sections of the destroyed menisci were not taken out of zones which were not scarred when examined with the stereo-microscope. Therefore substitutional but not scarred meniscus is presented (Refior and Fischer 1974). This is an important condition for the interpretation of the interfibrillar elastin-collagen-connexions. Collagen fibrils readily produced and healing with cicatrices is found in almost every organ. Healing processes are accompanied by an increase of the elastic network, it is therefore, possible that genesis of elastin takes place in the scar tissue. This can be demonstrated in our findings: with growing age chronical meniscopathy is more readily demonstrated.

Other interfibrillar elastin-collagen-connexions have not been found. If it is necessary to have "puncta fixa" for the elastin system, these connexions are really corresponding to this thesis.

With morphological methods an exact differentiation between the precursor elastin structures (oxytalan and elastin fibres) is not possible (Stadler and Orfanos 1978; Ghadially 1982). Precursor fibrils are assumed to accept colour in the elastin staining but lack a banding pattern demonstrated by light or electron microscopy. Due to their substructure however confusion with collagen fibres is impossible. The following differences from collagen are remarkable:

1. Elastin fibres do not seem to fray out like collagen fibres they rupture abruptly. With the scanning electron microscope splintering and splitting are not seen.

2. In serious chronical meniscopathy even the elastin – the last remaining defined structure of the meniscus – seems to be destroyed. Often agglomerations of elastin are seen imposed on the natural surface. In light microscopical slides the elastin fibres can be persued even up to the already amorphous zone of necrosis. Only the surrounding tissue indicates newly produced or older elastin.

3. Thus small coreless scars have plenty of elastin. Obviously the phenomena of upsetting and warping are cumulative. These are verified in the flow of the elastin fibres. In new granulation tissue small, stump-like, non-reticular fibres of elastin are found.

4. By means of electron microscopy an almost regular continuous fibrillar structure of the elastin can be demonstrated in the coat areas of the fibres, they seem to correlate with optical dense spots (in the cross-section) and stripes (in the longitudinal section). In contrast to the opinion of Ghadially (1982) we think that this variability of the optical density does not refer to artefacts resulting from staining or fixation.

5. In spite of the multiplicity of the manifestations it is not safe to talk about a process of decay. The differences in structure are obviously age dependant processes, however, it is still unclear which findings should be interpreted as "normal" and which as "pathological" (the same problem is formulated for other organs by Ghadially 1982). The points of interest are characterized by fibrillar dissection, optical dense zones in the cross-section arranged street-like with partly cystic areas and formations of pleating, angles and loops.

Effects within the elastin are clearly initiated by a complex process of destruction and warping of the collagen network.

### *B. Interfibrillar connexions*

Morphological connexions between the elastin and the collagen fibres are demonstrable. They include small bridges sometimes presenting a vague banding pattern but more frequently an amorphous structure like the elastin. Aditionally, there are large amorphously structured areas with partly parallel, partly acute-angled elastin. In longitudinal or diagonal-longitudinal cutting planes the elastin areas seem to dive into the picture plane and seem to disappear again – this may demonstrate the contraction phase of the elastin and the undulating flow of the collagen. This could be also an indicator for the mechanical stable connexion (without any phenomena of sliding and shifting) between the elastin and the collagen.

Elastin is found in forms of fibres of different calibers. No previous bundles of fibres nor zones of connexions within the elastin have been observed by transmission electron microscopy.

The three-dimensional relation between the collagen and the elastin is an obviously unassociated parallel flow, a loose imposition of singular collagen fibrils on the elastin as well as imbedding of complete fibrillar complexes.

Whereas collagen fibres show phenomena of destruction (pleating, lateral fibrillar deficiencies, dissolution of the fibrillar network), the elastin fibrils seem to be intact. By means of transmission electron microscopy only a few zones tending to an increasing confluence are demonstrable. Fukuda and Ferrans (1984) have demonstrated (with the help of antielastase-antibodies) that there is only an insignificant super-imposed layer of elastin in the microfibrillar exterior zones whereas the amount of elastin increases in direction of the central amorphous segments of the fibre (Cleary et al. 1981; Oakes and Batty 1982; in-vitro-researches: Madsen 1983).

Interfibrillar junctions between elastin and collagen have been described by Dryll et al. (1981) in relapsing polychondritis. The authors interprete the appearance of fibrillar structure as destruction of the fibre due to elastase. In our findings fusion of elastin and collagen is often found within

segments of the meniscus in which singular (not connected) collagen and elastin fibres occur rarely. Interfibrillar junctions are found either singularly or agglomerated in groups in the ground matrix.

### *C. The elastin as a compartment of the meniscus*

The elastin of the meniscus is considered to be a compartment distinct from collagen and ground matrix. Collagen is the most obvious and quantitatively important compartment of the meniscus. The interaction between collagen and the proteoglycan compartment refers mostly to the extracellular fibrillar genesis of the collagen (Junqueira and Montes 1983). The fine structure as well as the conditions of the production and the network of the elastin are assumed to depend only slightly on the ground matrix (Urry 1983; Eyre 1984).

Proper function of an elastin system depends on "puncta fixa". In the meniscus these can be only represented in the collagen compartment – the proteoglycan compartment is not mechanically qualified for this purpose. The zones of junctions have got to be mechanically stressable and have to connect the two fibrillar compartments in a definitive topological order.

Our observations correspond to these reflections, rarely we found crooked or undulated collagen fibres whose flow might refer to the function of an elastin fibre located in the close neighbourhood. We do not consider such alterations of form of the collagen as a morphological structure for the functional stressing of the meniscus (Fig. 1). On the contrary, we observed a scissors-like sliding of the collagen which tightens with increasing alteration of the form the elastin compartment. The insertion points of the elastin have a special importance: the insertion angle determines effective leverage and therewith the conditions of the stress/tension diagram of the meniscus (Fig. 10). It is not quite sure for the meniscus, whether the shortest mechanical junction lines are chosen or jumping positions for the insertion region of the elastin fibres. Our morphological findings point to both possibilities. Finally also clinging to the collagen by the elastin may be conceivable, especially within or in the close neighbourhood of the region of insertion.

The findings of wrinkled, agglomerated and pleated elastin fibres lead to the thesis that the functional interrelation of the elastin compartment has been destroyed in the junction zones by the consequences of the meniscopathy:

– by primary or direct destruction, mechanically or enzymatically mediated, the elastin is no longer fixed, it "wrinkles";
– the bundles of collagen fibres slide; the elastin compartment is overmaximally stretched; it ruptures and "contracts";
– by secondary destruction with a break in continuity, after destruction within the collagen compartment.

## *5. Outlook*

There is an elastin network in the human meniscus. Because of its precise, topological arrangement in relationship to the collagen network it is correct

to talk about an elastin compartment, distinct from the proteoglycan compartment and the collagen compartment. Elastin shows the same morphological characteristics as already described for other organs with identical differences in dimension, fibrillar substructure and the density of the network and the constancy in caliber. The elastin-collagen-connexion of the meniscus seems to be realized in a special way. The connexions show a specific topology corresponding to the elastic function of the meniscus related to the mechanical stressing. Destruction of the elastin compartment correlates directly with the seriousness of the meniscopathy. The macroscopically seen brown change in colour of the menisci a chronic meniscopathy correlates with the increase of elastin.

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