Injuries to flexor tendons of the hand in zone 2 are still a difficult problem in hand surgery. The challenge is to restore the gliding mechanism of the tendons. Tendon lacerations in zone 2 have been treated in various ways (Meals, 1985; Schneider and Bush, 1989; Schneider and McEntee, 1986; Steinberg, 1992; Tonkin, 1991; Verdan, 1979; Verdan and Crawford, 1979). Despite increasing knowledge of tendon healing and subsequently better post-operative results, the problem of formation of adhesions between the tendon and its direct surroundings remains. The clinical results of flexor tendon repair are therefore still unpredictable.


The flexor tendon sheath plays an important role in tendon nutrition, especially for the volar part of the tendon, by secreting the synovial fluid. Although data to date are not conclusive regarding repair of the sheath, there are indications that it will lead to fewer adhesions (Leddy, 1993; Lindsay and Thomson, 1960; Lister et al, 1977; Tonkin and Lister, 1986). Tendon sheaths are sometimes restored by an autologous graft, e.g. extensor retinaculum. This has the disadvantage of an extra incision and removal of sound functional tissue.

We examined the possibility of reconstructing the tendon sheath with a collagen membrane. We tested syngeneic parietal peritoneum which consisted mainly of collagenous fibres and fibroblasts. Like peritoneum the synovial membrane is of mesothelial origin and both secrete a lubricant. We also investigated the use of processed porcine collagenous membrane, which has the advantage of avoiding a laparotomy.

**MATERIALS AND METHODS**

Fifty-four female 9-month-old New Zealand white rabbits, weighing about 2.5 kg, were used for the experiments. Syngeneic parietal peritoneum (PP) was retrieved by a median laparotomy. From the inner side of the abdominal wall a small transparent piece was resected and temporarily kept in a sterile 0.9% solution of saline. The xenoplastic bioimplant was a non-tanned processed collagen of porcine origin (PPC). Macroscopically, it has rough and smooth sides. It was retrieved by cleaning raw tissue from the pig through alkaline and acid treatments, and was subsequently dehydrated in acetone. Analysis revealed that it contained 85% collagen, mainly native type I (99%), and 15% elastic fibres. No cellular elements were present. PPC was thicker than normal rabbit tendon sheath and peritoneum (Fig 1). PPC was supplied by Bioplex Medical B.V., Vaals, The Netherlands, a subsidiary of Datascpe Corporation (Montvale, New Jersey, USA).

The experiments were divided into four groups (Table 1) and all performed in the forepaws. Because most animals were operated on both sides, the total number of experiments was 86. The tendons were approached via a longitudinal volar incision. The operations were performed under general anaesthesia and aseptic conditions using an operating microscope. Anaesthesia was induced by intravenously administered pentobarbital 30 mg/kg body weight, and maintained by inhalation of a mixture of nitrous oxide, oxygen and 1% halothane through a ventilation mask. Before the animals were killed, they were anaesthetized with 1.5 ml ketamine hydrochloride 5% and 1.25 ml xylazine hydrochloride 2% intravenously. After macroscopical evaluation and sampling for histology, the animals were resected.
TENDON SHEATH REPAIR

Fig 1 Processed porcine collagen. Elastica Van Gieson stain, 120 ×. Arrow—elastic fibres.

Table 1—Experimental groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Subgroup</th>
<th>Description</th>
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<tr>
<td>1A</td>
<td>(n=7)</td>
<td>Excision of tendon sheath</td>
</tr>
<tr>
<td>1B</td>
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<td>Excision and reconstruction of tendon sheath with PP</td>
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<tr>
<td>1C</td>
<td>(n=9)</td>
<td>Excision and reconstruction of tendon sheath with PPC</td>
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<tr>
<td>2A</td>
<td>(n=6)</td>
<td>Reconstruction of FDS and FDP without resection of tendon sheath</td>
</tr>
<tr>
<td>2B</td>
<td>(n=10)</td>
<td>Reconstruction of FDS and FDP in combination with reconstruction of tendon sheath with PP</td>
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<tr>
<td>2C</td>
<td>(n=23)</td>
<td>Reconstruction of FDS and FDP in combination with reconstruction of tendon sheath with PPC</td>
</tr>
<tr>
<td>3A</td>
<td>(n=6)</td>
<td>Excision of a wedge from FDS and FDP without reconstruction of tendon sheath</td>
</tr>
<tr>
<td>3B</td>
<td>(n=6)</td>
<td>Excision of a wedge from FDS and FDP in combination with reconstruction of tendon sheath with PPC</td>
</tr>
<tr>
<td>4</td>
<td>(n=6)</td>
<td>Subcutaneous implantation of PPC in the interscapular region</td>
</tr>
</tbody>
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FDS = flexor digitorum superficialis.  
FDP = flexor digitorum profundus.

were killed by intravenous administration of 4 ml pentobarbital 6%.

In group 1 the tendon sheath was circumferentially excised over a distance of about 1 cm between the two annular pulleys. In subgroups 1B and 1C the excised sheath was replaced by PP or PPC respectively. The reconstruction of the tendon sheaths was done with 10/0 polyglactin after wrapping the deep and superficial flexor tendons with either PP or PPC. In group 2 both tendons were cut transversely and then repaired by a modified Kessler-Mason stitch for the deep flexor tendon and a mattress suture for the superficial tendon using 7/0 polypropylene. In group 2A the tendon sheath was closed without a suture. In the subgroups 2B and 2C it was replaced by PP or PPC respectively. In group 3 a wedge of approximately 20% of the diameter was excised from both tendons to simulate a partial lesion of the tendon (3A). Tendon sheath was reconstructed with PPC in the subgroup 3B. In group 4 a patch of PPC was implanted subcutaneously in the interscapular region, to investigate the behaviour of the material in a mechanically unloaded region and the reaction of the host on a physiological non-functional graft. The patches measured approximately 1 × 1 centimetre and were secured by 3/0 polypropylene. In all cases the skin was closed with interrupted sutures of 3/0 polyglycolic acid sutures. None of the forepaws was immobilized.

The results were evaluated after 7 weeks and 3 months. Tissue was fixed by immersion in 3.8% formaldehyde. Specimens were embedded in paraffin or polymethylmethacrylate, sectioned in a plane perpendicular or parallel to the long axis of the tendon, and stained with Hematoxylin-Azaphloxin (HA) or Hematoxylin-Eosin (HE), and Elastica-Van Gieson (EG) dyes.

RESULTS

Except in two animals, all wounds healed normally. One animal had a wound dehiscence after 1 day that was repaired and healed without further complications.
Another had a purulent wound infection in one paw, with uncomplicated healing of the other. Gap formation at the tendon repair site occurred in seven cases in group 2 (one in group 2B and six in group 2C). Total dehiscence of the tendon repair was seen in two rabbits (one each in groups 2B and 2C) and led to the formation of adhesions between the site of repair and the surroundings.

**Group 1A**
Circular excision of the sheath led to gross adhesions 7 weeks post-operatively. Microscopically, there was a diffuse proliferation of fibroblasts and collagen fibres that adhered to the tendons (Fig 2). There was no sign of formation of a neo-sheath and the collagen fibres had a random orientation, as in scar tissue.

**Group 1B**
Reconstruction of the tendon sheath with PP led to the formation of a neo-sheath after 7 weeks. Macroscopically, the neo-sheath had a glassy appearance through which the tendons were visible. The tendons could be moved easily within it. Formation of the neo-sheath was easily recognized microscopically. It had a cellular lining and was thicker than the normal tendon sheath. The tendons had a normal appearance without necrosis. There were no adhesions (Fig 3). In the neo-sheath blood vessels were identified clearly. Sparse macrophages and lymphocytes were visible, mainly around the polyglactin suture material.

**Group 1C**
Remnants of the elastic fibres of the PPC were seen in the Elastica-Van Gieson stained sections after 7 weeks and less after 3 months. The elastic fibres were located on the outer side of the neo-sheath (Figs 4 and 5). No calcification was seen at the site of the grafts.

**Group 2A**
In this group, in which the tendon sheath was excised, tendon healing was unremarkable 3 months post-operatively. The specimens showed a normal tendon sheath without signs of adhesions. There was some local proliferation of synovial-like tissue. Some leucocytes were seen in the subintima. A proliferation of collagenous fibres was seen, which was well shown in the EG stain, at the site of tendon repair. Remnants of the suture material, which were surrounded by lymphocytes, were identifiable (Figs 6 and 7).

**Group 2B**
Macroscopically, the autograft was not identifiable as a distinct entity from the remnants of the sheath 3 months post-operatively. It had a glassy appearance and underneath the neo-sheath the tendons were visible and could be moved easily.

Microscopically, the neo-sheath was recognized easily and lined by synovial-like cells. The neo-sheath contained small quantities of slim, elongated elastic fibres (Figs 8 and 9). Three months post-operatively giant
cells were seen, grouped around the polypropylene suture materials. Some cells contained bi-refrangent material, resembling the suture material. Suture material was seen in tendons, surrounded by lymphocytes and giant cells. The tendon repair site was bridged by a cell-rich tissue containing fibroblasts, some leucocytes and collagen fibres. Around the suture materials some lymphocytes and macrophages were seen. In the neo-sheath capillaries were plentiful.

**Group 2C**

Three months after reconstruction of the sheath with PPC, a neo-sheath was formed without signs of
adhesions. The neo-sheath was capillary-rich and lined by synovial-like cells. At the site of the tendon repair fibroblasts with longitudinally oriented collagen fibres were seen. Remnants of the suture material were surrounded by lymphocytes and giant cells. In the Elastica-Van Gieson stained sections, remnants of the PPC could be identified as coarse, elastic fibres. These elastic fibres were located in the periphery of the neo-sheath. Beside these elastic fibres, some thin and elongated elastic fibres were seen (Figs 10 and 11). There was no calcification.

Fig 5 Three months after reconstruction of tendon sheath with PPC (group 1C). EG stain, 120 x. Triangle—tendon; arrow—neo-sheath.

Fig 6 Three months after tendon repair (group 2A). HA stain, 120 x. Triangle—tendon; arrow—remnants of suture material.
Group 3A

Excision of wedges in the tendons and closure of the sheath resulted in uneventful tendon healing without adhesions 3 months post-operatively. Cell-rich connective tissue with proliferative collagenous fibres was seen at the site of the excised wedge. The sheath was microscopically normal and contained fine elastic fibres, which were quite distinct from the elastic fibres of PPC (Figs 12 and 13).
Fig 9  Three months after tendon repair and reconstruction of tendon sheath with peritoneum (group 2B). EG stain, 120×. Triangle—tendon; arrow—neo-sheath.

Fig 10  Three months after tendon repair and reconstruction of tendon sheath with peritoneum (group 2C). HE stain, 120×. Triangle—tendon; arrow—neo-sheath.

**Group 3B**

Three months post-operatively a neo-sheath had formed, which was confirmed microscopically. It consisted of young connective tissue with synovial-like cells lining the inner side. Locally, an accumulation of coarse elastic fibres which resembled those of PPC was seen in one specimen at the insertion of the tendon. No signs of calcifications were seen in the neo-sheath. The appearance of the flexor tendons was normal without adhesions (Figs 14 and 15). Cell-rich connective tissue was seen at the site of the wedge excision.
Fig. 11 Three months after tendon repair and reconstruction of tendon sheath with PPC (group 2C). EG stain, 120×. Triangle—tendon; arrow—neo-sheath.

Fig. 12 Three months after excision of a wedge in both tendons and closure of tendon sheath (group 3A). HA stain, 120×. Triangle—tendon; arrow—local reaction at excision of wedge.

**Group 4**

Three months after implantation no remnants of the PPC could be identified. The sites where the patches had been implanted could only be recognized by the suture materials. In the histological specimens no remnants of PPC were seen, not even elastic fibres. The specimens were dominated by fatty vacuolar tissue with
Fig 13 Three months after excision of a wedge in both tendons and closure of tendon sheath (group 3A). EG stain, 120 x. Triangle—tendon; arrow—local reaction at excision of wedge.

Fig 14 Three months after excision of a wedge in both tendons and reconstruction of sheath with PPC (group 3B). HE stain, 120 x. Triangle—tendon; arrow—neo-sheath.

suture material. Remnants of the suture material were surrounded by macrophages. Bi-refringent material was visible in some macrophages, suggesting phagocytosis of the suture material (Fig 16). There was no signs of calcification at the site of the grafts.

DISCUSSION

The flexor tendon sheath is essential for tendon nutrition. Resection of it is therefore detrimental to tendon healing and increases the chance of adhesions.
We found that circumferential resection of the sheath led to adhesions. Replacement of the excised sheath by either an autologous or a xenogenic graft resulted in the formation of a neo-sheath. The graft was placed completely around the tendons. A half-circumferential type of reconstruction, i.e. only on the volar side, could be sufficient, since the dorsal aspect of the fibro-osseous canal is not injured in all tendon lacerations. The neo-sheath was thicker than normal tendon sheath, possibly due to the original thickness of the material. A thinner
graft would perhaps produce a neo-sheath similar in thickness to the normal tendon sheath. The combination of the reconstruction of the sheath and a tendon repair did not affect tendon healing. Adhesions formed only when tendon gapping occurred and the graft did not prevent the formation of adhesions. The collagenous and elastic fibres of PPC were well tolerated by the host and were eventually replaced by host's tissue as shown by vascular ingrowth, which mainly occurred at the rough side of the graft. This is in accordance with the results of Ruijgrok (1993).

Both types of graft probably acted as a scaffold for the migration of cells from the adjacent native sheath and the deposition of collagenous fibres (“creeping substitution”). Remodelling is an important aspect in the process of substitution. Physiological stimuli are important for remodelling. In their absence, the graft will be resorbed at a faster rate, as in group 4. The grafts probably also acted as a barrier to the invasion of fibroblasts of non-tendinous origin to the site of the repair. Tendon repair is known to occur without the formation of adhesions if the fibroblasts of the epitenon and endotenon survive and proliferate. In cases of partial tendon lesions (group 3), replacement of the sheath by PPC did not interfere with restoration of the gliding function. The wedge was filled by fibroblasts and collagenous fibres and there were no adhesions.

The fact that PPC was not tanned may have contributed to the results. Tanning chemicals can induce an inflammatory reaction and are cytotoxic, thus interfering with normal wound healing. Tanning increases the number of cross-link bonds of the collagenous fibres and is important if the collagenous membrane is subjected to high mechanical forces. Since mechanical strength is not important for reconstruction of the tendon sheath, such reinforcement of the membrane was not required. An advantage of PPC is that it is “ready for use” at any time. The chemical properties of the collagenous fibres are not changed by sterilization with ethylene-oxide. It can be packed sterilely in any required size. Use of PPC has an advantage over peritoneum or other autologous grafts, in that it avoids an extra incision.

Our findings are partly in contrast to those of Lindsay and Thomson (1960), who found the formation of a neo-sheath with only filmy adhesions. Their experiments differed from ours in that they did not perform a circumferential excision of the sheath, but left the dorsal part intact. Our results are comparable to those of Peterson et al (1990) and Tang et al (1990; 1993). Excision of tendon sheath led to more adhesions than closure or reconstruction with a graft. In clinical situations Tang et al (1993) obtained 86% good to excellent results in delayed primary repair with sheath reconstructions using the extensor retinaculum of the wrist.

Unfortunately, reconstructions of the tendons were followed by a relatively large number of dehiscences, probably due to the incompatibility in the size of the suture material and the thickness of the tendon. Thinner suture material did not possess the necessary strength. The relatively high incidence of gap formation could be attributed to the absence of an epitenon (peripheral) stitch, which is essential to prevent it (Wade et al, 1986). We did not immobilize the rabbits post-operatively which compromises the functional outcome of tendon repair, and does not prevent gap formation or dehiscence of the suture (Gelberman et al, 1983).

We believe that it is feasible to maintain the gliding function of the flexor tendons in rabbits, even after tendon repair, by restoring the tendon sheath with autologous or xenogenic collagenous implants. Collagen has a low antigenicity and will be resorbed ultimately by the host (Simpson, 1983; Tang et al, 1990; Timpl, 1984). There is perhaps an indication to use the xenogenic graft in humans in situations where sheath reconstruction is impossible because of major damage. Whether this will be as successful has to be investigated. Another indication may be the reconstruction of the pulley system. The pulleys, especially the A2 and A4 pulleys, are essential for normal flexion of the finger (Kleinert et al, 1981). The material to reconstruct the pulley must be strong enough to withstand the flexing forces of the tendon but should not lead to adhesions. A collagenous membrane is superior to other implants such as expanded-polytetrafluoroethylene, because it will ultimately be completely remodelled and resorbed (Hanff et al, 1991).

References


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