FACTORS IN THE ADHERENCE OF FLEXOR TENDON AFTER REPAIR
AN EXPERIMENTAL STUDY IN THE RABBIT

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The effects of splintage, suture and excision of the tendon sheath on the healing of incompletely transected flexor tendons in the rabbit have been evaluated separately and in various combinations. When all procedures were done together, repair was accompanied by dense adhesion formation with little evidence of any healing activity by the tendon cells. The experiments indicated that the adhesions were the result not of any one single factor studied but of all three contributing in varying degrees. Suturing produced the most adhesions but synovial sheath excision and immobilisation also contributed. It is suggested that these factors are also responsible for the adhesions which occur after flexor tendon repair in clinical practice.

The difficulty in obtaining satisfactory results in the repair of divided digital flexor tendons is due largely to the adhesions which develop between the site of suture and the surrounding tissues. Bound down by these adhesions, the tendon can no longer slide freely within its sheath, and function of the finger is seriously impaired.

Adhesion formation is often regarded as an undesirable but necessary part of the tendon healing process. This concept has arisen in consequence of experimental work on tendon healing, and in particular from the observation that the tenocytes within the stumps of a sutured tendon behave in a passive manner and do not participate in reparative activity (Potenza 1962). It has been assumed from this that they lack any potential for repair and that tendon must therefore rely for healing upon the ingrowth of fibroblastic cells from elsewhere. Recent studies into the pattern of healing of incompletely transected tendons do not, however, support this view but indicate that under ideal conditions tendon possesses properties of repair and remodelling which are quite independent of other tissues (Matthews and Richards 1974). It seems that in the pathogenesis of adhesions factors other than the healing characteristics of the tissue must be involved.

The initial injury to the tendon is not in itself the main stimulus for the development of adhesions. When a flexor tendon is severed in the finger, its ends retract and become smoothly rounded over to lie free within the sheath. Adhesions do not form and there is no reaction in the surrounding tissues. It is only when the stumps are apposed surgically that an adhesive response occurs. Could it be that in achieving this apposition factors are introduced which are in some way responsible for the adhesions? The surgical repair of a tendon usually involves the presence of three fresh conditions in addition to the original trauma: the digital sheath is widely opened or excised, sutures are inserted, and the digit is immobilised while healing takes place.

Diagrammatic illustration of the basic experimental procedure. 

**FIG. 1**

**FIG. 2**

The purpose of this study was to determine what influence such factors have on tendon healing and whether they are important in the development of adhesions.
METHODS

The experiments were so designed that the effects of splintage, suture and sheath excision on the regeneration of a traumatized tendon could be assessed, first separately and then in various combinations. In this respect they differ from other studies and provide information which has not previously been available. The operations were based on the model used in our earlier study (Matthews and Richards 1974) but with modifications for the purpose of the investigation. The reasons for choosing this method were two-fold. Firstly, it allowed study of the repair process as it occurs within an intact area of the sheath. Secondly, as the tendon was not quite completely severed, continuity was not totally lost and so healing could be observed either with or without sutures or immobilisation. This facility can be obtained in no other way.

Operative technique of the insertion of the suture. After opening the synovial reflection a hook (on the right of the photograph) is put round the tendon and by pulling proximally, a considerable length of the intrasynovial portion of the profundus is drawn into the wound. The tendon is then cut across transversely, leaving intact only just sufficient to maintain continuity. A Bunnell criss-cross suture of 5/0 braided Dacron is inserted and knotted in tension.

Diagrammatic illustration of the tendon showing the extent to which it has been divided and the position of the suture and knot.

Fully grown New Zealand white rabbits were selected for study. The initial preparation was the same in all the experiments. The rabbits were anaesthetised with intravenous Nembutal, then one of the front paws was shaved and an exsanguinating tourniquet was applied. The skin was then prepared with 1 per cent Hibitane solution in spirit and sterile drapes were applied.

The skin incision was made, not in the digit itself, but in the "palm" area, the flexor tendons then being identified at their point of entry into the sheath and the synovial reflection opened. By fully flexing the middle digit and pulling proximally on the profundus a length of the intrasynovial part of that tendon could be drawn into the wound (Fig. 1). The tendon was next traumatised as distally as possible by cutting across it transversely with a scalpel. The incision stopped just short of complete section, leaving intact only sufficient tendon fibres to maintain continuity. The traction was then released and the traumatised zone of tendon slid back to lie within normal sheath over the distal third of the proximal phalanx (Fig. 2). The skin was closed with fine catgut sutures and an aerosol collodion dressing sprayed on the wound.

On this basic experiment the following variables were introduced, first separately and then in different permutations: 1) immobilisation, 2) suture, 3) excision of sheath.

Immobilisation—After the wound had been closed, a tightly fitting plaster-of-Paris splint was applied to enclose the whole of the affected limb and to immobilise the digits. The plaster was retained for a total of twenty-four days, after which the limb was left free.

Suture—After the profundus tendon had been cut across, a braided Dacron 5/0 suture was inserted at the same level by a Bunnell criss-cross technique and tied in tension (Figs. 3 and 4).

Excision of sheath—In these experiments the skin incision was made on the volar aspect of the digit and the tendon was exposed by removing a large area of the sheath at the level of the proximal phalanx. After the usual incomplete transaction of the tendon the skin was closed with catgut.

In all the operative procedures, aseptic technique was employed throughout. The tendons were handled as gently as possible and sharp scalpel dissection was used exclusively.

There were seven groups of experiments in the investigation and in each of these ten rabbits were used. A total of seventy operations was thus carried out.

The rabbits were killed at intervals up to twelve weeks. The digit operated upon was first carefully examined with the aid of a dissecting microscope and note made of the state of healing of the tendon, the reaction of the tissues in the vicinity of the injury and the presence or otherwise of adhesions. The tendon was then removed with the surrounding soft tissues and fixed in 10 per cent formol saline. After processing and embedding, serial longitudinal sections were cut for microscopic examination. The sections were stained with haemotoxylin and eosin, James's silver stain and Hale's colloidal iron/PAS stain.

RESULTS

The effect of immobilisation—Healing in all the tendons in this group took place by processes intrinsic to the tendon and without the development of adhesions. On a microscopic level the mode of repair was by local tenoblast proliferation and the elaboration of new collagen fibres. There was no reaction of the synovial sheath and adhesions between it and the tendon were not seen. By the twelfth week the defect had been reconstituted by mature tendon fibres.

Conclusion—Immobilisation in itself has no influence on the healing response of the injured tendon and does not evoke an adhesive reaction in the overlying sheath.

The effect of suture—Repair of the cut in these tendons occurred without adhesions. By the fourth week the whole of the sutured area had become covered by a thin layer of glistening, pearly white repair tissue. The cut itself was gradually obliterated and by the third month the sutured zone appeared as a smooth, mildly fusiform thickening of the tendon which was able to slide freely
within the sheath. In two specimens the end of the Dacron suture had become visible protruding through the surface of the tendon callus (Fig. 5). The thread had become stiffened and appeared to be incorporated in a delicate extension of the repair tissue.

On microscopic examination repair was seen to be brought about by local tendon cell activity (Fig. 6) and there was no reaction in the synovial membrane. Certain microscopic features were regarded as particularly significant. In the early phase after operation the cellularity of the area of tendon enclosed by the suture was diminished and there was disorganisation of the tendon fibres in the same region (Fig. 7). The proliferation of tendon cells and the build-up of tenoblasts appeared delayed in comparison with that in the preceding experiment. Furthermore, once new collagen formation had become established, it became evident that the zone of regeneration was not confined to the site of the original cut but that new fibres were being laid down throughout the area enclosed by the suture (Fig. 8).

Conclusion—Healing of a traumatised tendon can occur without adhesions even in the presence of a suture. It is, however, associated with changes suggestive of reduced vitality in the sutured zone and with extension of the area of regeneration to include the whole of the tendon gripped by the suture.

The effect of excision of the sheath—Healing of these tendons was by tenoblastic activity (Fig. 9) and no lasting adhesions were seen. During the initial post-operative phase, the space between the tendon and the peri-sheath tissues filled with a fibrinous exudate containing mononuclear cells. Within a week the tendon had become surrounded by granulation tissue which did not, however, invade the tendon or contribute to its repair. Coincidentally with the healing of the peri-sheath tissues a new synovial layer developed.

Conclusion—Excision of the area of sheath overlying an injured tendon does not in itself affect the reparative activity of the tendon cells. With the healing of the peri-sheath tissues the synovial layer is rapidly reconstituted and a new gliding pathway formed.

Combined effect of splintage and suture—In this group healing occurred mainly by tendon cell proliferation. Adhesions were noted, however, in five of the ten specimens, the earliest being apparent at ten days. The adhesions were mild to moderate in degree and so localised that complete obliteration of the synovial space did not occur (Fig. 10). On discarding splintage the adhesions

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**FIG. 5**
The effect of suture on the regeneration of tendon. Healing tendon at six weeks. The zone of injury and suture is swollen and its surface is covered with a smooth layer of glistening white repair tissue. There are no adhesions. In two specimens, one of which is shown here, the end of the Dacron suture had become visible and protruded through the surface of the tendon callus. The thread had become stiffened and appeared to be incorporated in a delicate extension of the repair tissue.

**FIG. 6**
The effect of suture in the regeneration of tendon. Longitudinal section through tendon at four weeks. Marked tenoblast repair activity can be seen. There are no adhesions. (Haematoxylin and eosin, ×43.)
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began to disperse and in the later specimens had completely disappeared.

Conclusion—When a traumatised tendon is sutured and then immobilised but its sheath left intact, it heals partly by processes intrinsic to the tendon but the repair is augmented by adhesions. The adhesions are relatively mild and are absorbed once splintage is discarded.

Combined effect of suture and sheath excision—Although some tenoblast repair activity was present in all the specimens in this group, adhesions were also encountered, especially in the earlier stages. During the initial phase after operation, the zone of trauma became enveloped in soft grey fibrinous clot. Later, loosely arranged adhesions formed but with time these either absorbed completely or appeared as fine strands of fibrous tissue extended in the direction of gliding and with adequate length to allow a normal excursion of the tendon (Fig. 11).

Conclusion—Excision of the synovial sheath over a sutured area of traumatised tendon results in an adhesive response which contributes to the repair of the tendon defect. The adhesions tend either to absorb or to remodel in such a way as to allow gliding movement to take place.

**Fig. 7**
The effect of suture on the regeneration of tendon. Longitudinal section through tendon and sheath at five days. The cut in the tendon is on the extreme left of the photograph and suture material can be readily seen within the adjacent portion of the tendon. Note the marked reduction in the cellularity of the region of tendon enclosed by the suture. (Haematoxylin and eosin, × 33.)

**Fig. 8**
The effect of suture on the regeneration of tendon. Longitudinal section through tendon and sheath at eight weeks. The defect in the tendon has been reconstituted by fine newly formed tendon fibres. Note that the zone of regeneration is extensive and includes the whole of the area enclosed by the sutures. There are no adhesions. (James silver stain, × 31.)
Combined effect of sheath excision and splintage—Within a few days of operation the traumatised area of tendon had become surrounded by blood clot and fibrinous exudate. Granulation tissue arising from the peri-sheath tissues formed around the tendon but the adhesions were light and filmy. They did not appear to play a dominant role in the healing of the tendon. By the third week the adhesions began to resolve and when splintage was discarded they completely disappeared. The area of regeneration of tendon was confined to the initial injury. A new synovial sheath was formed and by the twelfth week anatomical and functional normality had been restored.

**Conclusion**—Immobilisation and removal of the sheath were introduced simultaneously. All tendons in the group healed with marked adhesion formation. The sutured area of tendon became embedded in a granulation tissue which invaded both the region of the cut and the suture tracks (Fig. 12). Tendon cell activity was sparse and reconstitution of the defect was accomplished mainly by the ingrowth of fibroelastic repair tissue derived from the peri-sheath layers. This gradually matured into a dense

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**FIG. 9**
Healing after incomplete tendon transection and excision of the overlying sheath. Longitudinal section through the injured tendon and soft tissues at ten days. Early repair activity is visible within the tendon itself and is occurring independently of the changes in the peri-sheath tissues. (Haematoxylin and eosin. × 52.)

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**FIG. 10**—The effect of splintage and suture on tendon regeneration in the presence of an intact sheath. Healing tendon at three weeks: a few adhesions can be seen but they are mild and localised so that complete obliteration of the synovial space has not occurred.

**FIG. 11**—The effect of suturing an injured tendon and excising the overlying sheath. No immobilisation. Healing tendon at eight weeks. The cut in the tendon is completely healed and there is generalised swelling of the sutured area. A solitary adhesion is visible; it is thin and elongated and compatible with a useful range of movement.
scar tissue that persisted even when splintage had been discarded (Fig. 13). At twelve weeks the tendon was still bound down by tough restrictive adhesions and no significant sliding movement was possible.

**Conclusion**—The presence of sutures, sheath excision and splintage results in a profuse and lasting adhesive response and to the suppression of the intrinsic repair reaction of the injured tendon.

**DISCUSSION**

Although adhesion formation after flexor tendon repair is usually regarded as an integral part of the healing process of the tissue, these results (summarised in Table I) show that it can be readily accounted for by certain external factors introduced by the surgical repair. The combined influence of suture, sheath excision and splintage on an injured tendon was sufficient to produce a marked adhesive reaction and to suppress the intrinsic repair response of the tendon cells. Of the factors studied, suture appeared to have the most adverse effect on the tendon and produced changes suggestive of reduced viability and wide extension of the zone of regeneration. Nevertheless, the most dense adhesions occurred when all three factors were present and it seems, therefore, that excision of the sheath and immobilisation do play significant complimentary roles in the process.
The mode of action of these factors is uncertain but it is possible that it is based on interference with the nutrition of the tendon. It is recognised that in other

### Table 1
#### Results Summarised; the Influence of Iatrogenic Factors on the Healing of Injured Digital Flexor Tendon

<table>
<thead>
<tr>
<th>No adverse factors</th>
<th>No adhesions</th>
<th>Healing in the absence of immobilisation, suture and splintage</th>
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<tr>
<td>Individual factors</td>
<td></td>
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<tr>
<td>Immobilisation</td>
<td>Suture</td>
<td>Healing entirely by tendon cell proliferation</td>
</tr>
<tr>
<td>No adhesions</td>
<td>No adhesions</td>
<td>(Matthews and Richards 1974)</td>
</tr>
<tr>
<td>Healing entirely</td>
<td>Reduced viability of tendon enclosed by the suture and wide extension of zone of regeneration</td>
<td></td>
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<tr>
<td>by tendon cell proliferation</td>
<td>Sheath excision</td>
<td>Healing by tendon cell proliferation. Rapid regeneration of new synovial layer</td>
</tr>
<tr>
<td>Factors acting in pairs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Immobilisation + suture</td>
<td>Suture + sheath excision</td>
<td>Regeneration of new synovial layer</td>
</tr>
<tr>
<td>Mild to moderate transient adhesions</td>
<td>Mild to moderate adhesions, tending either to absorb or elongate</td>
<td>Immobilisation + sheath excision Light, filmy, transient adhesions</td>
</tr>
<tr>
<td>Reduced viability of sutured tendon with extensive regeneration</td>
<td>Dense, restrictive, persistent adhesions. Suppression of intrinsic tenoblast repair activity</td>
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<td>All factors acting simultaneously</td>
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parts of the body ischaemia from strangulation of tissue is a potent stimulus to adhesion formation (Ellis 1962); and because areas of ischaemia also occur within the parts of a tendon gripping by a suture (Bergljung 1968) it could be that a similar mechanism applies in this tissue as well. The effect of sheath excision may, perhaps, have a similar basis. There is evidence to suggest that although flexor tendons possess blood vessels, the synovial fluid is able to provide an additional pathway of nutrition through diffusion of metabolites across the synovial space (Potenza 1963, 1964). If this is so, interference with this pathway by excision of the sheath could only aggravate the ill effects on the tendon of deprivation of its blood supply by suture.

How relevant is this laboratory research to the clinical situation? Although there are limitations to the information which may be gleaned from animal experiments, it has to be borne in mind that most previous conclusions on the healing of tendon have also arisen from this type of study. There is no prima facie evidence to indicate that the properties of rabbit tendon differ in any essential from those of other mammalian species; so it is reasonable to suggest that the same factors which we have studied are also responsible for the adhesions which complicate clinical tendon repair.

In clinical practice, partial divisions of flexor tendons are only occasionally seen and they do not constitute a major problem of management. How far then can we relate our findings to the much more formidable problem of the completely severed tendon? There is certainly no reason to believe that the influence of the factors we have studied would be any the less in this case. Indeed, if we suspect suture tension is important, one would expect the effect to be even more pronounced. Given this conclusion, the clinical goal set for the repair of tendons should be the maintenance of stump apposition during healing while at the same time avoiding those factors we have incriminated in the formation of adhesions. It is difficult to see how, in practice, this could be achieved, and the problem of adherence of flexor tendons after repair may well prove insoluble.

We wish to thank Professor Brian McKibbin for his encouragement and advice and for allowing us to use the facilities of the Orthopaedic Research Laboratories. We are indebted to Mrs Heather Ralisi for her assistance with the histological preparations, to Miss Cheryl Peake, Mr Peter Blake and Mr Peter Langham for the illustrations and to Miss Jacqueline Marriott for secretarial help.

This work was supported by a grant from the Clinical Research Committee of the Welsh Office.

### References


