THE REPAIR POTENTIAL OF DIGITAL FLEXOR TENDONS
An Experimental Study

PHILLIP MATTHEWS and HAROLD RICHARDS, CARDIFF, WALES

Despite advances in knowledge and refinements of technique, the management of flexor tendon injuries within the digital sheath continues to present a formidable challenge to the surgeon. Suture of severed tendons within this area is often followed by the development of dense, unyielding adhesions between the site of repair and the surrounding tissues. These adhesions firmly tether the tendon and effectively block the gliding movement which is so necessary for normal function. All too often the end-result is that the affected finger becomes stiff and useless.

Potenza (1962) studied the process of tendon healing experimentally and concluded that adhesions were an essential part of the repair reaction of injured tendon and that healing could be accomplished only by an ingrowth of granulation tissue from the synovial sheath. Tendon tissue, it was claimed, was peculiar in possessing no power of regeneration and was able to contribute little, if anything, to the repair process.

These views have received wide acceptance and their relevance to the clinical problem is clear and important. If tendon is indeed passive in the matter of its own repair, then no matter how meticulous our techniques or ingenious our method of stump apposition, adhesions must develop before it can unite, and restriction of active flexion is inevitable.

It is difficult to understand why tendon should lack any properties of repair. Although it was long considered that tendons were avascular structures (Kölliker 1850) and virtually "dead" during life (Edwards 1946), there is now ample evidence that this is not the case. Vascular patterns have been regularly demonstrated within tendons (Mayer 1916, Edwards 1946, Brockis 1953, Smith 1965) and evidence that the circulation through these vessels is purposeful has been provided by the demonstration of respiratory activity (Peacock 1959) and of amino acid turnover (Neuberger and Slack 1953) within the collagen of adult tendon. Tendon must, therefore, be regarded as one of the living tissues of the body; so why should it be unique among them in lacking any intrinsic repair potential?

Most experimental studies into tendon repair, including those of Potenza, have involved the healing of tendons which had been sutured and immobilised and in which the synovial sheath had been either excised or extensively damaged. It seems a possibility that the absence of intrinsic repair activity and the healing by adhesions in these studies might have been the product not of the

Fig. 1
The intra-synovial part of the profundus tendon is exposed in the wound. The injured segment of tendon is lying over the tip of the disector. The more slender sublimis tendon has been retracted and pulled distally by the fine hook at the bottom of the photograph.
basic healing process of tendon but of these adverse factors. This suggested that further investigation was necessary into the healing of tendon injuries in isolation from such secondary factors as might exert an adverse influence on tendon repair potential.

**METHOD**

The experimental model was so designed that healing could be studied in the absence of sutures and immobilisation and within a tendon sheath which was, to all intents and purposes, intact. The digital flexor tendon of the rabbit was selected for study.

Fully grown rabbits were anaesthetised with intravenous Nembutal. One of the front paws was shaved, an exsanguinating tourniquet applied, and the skin prepared with Hibitane solution in spirit. All operations were done under strictly aseptic conditions.

A short incision was made through the skin of the flexor surface of the paw, extending proximally from the base of the middle digit. It was deepened to expose the flexor tendons to the middle digit at their point of entry into the digital sheath (Fig. 1). The synovial reflection was opened carefully at this point. The interphalangeal joints were then fully flexed and, by applying firm traction in opposite directions to fine hooks looped around the tendons, it was possible to deliver a considerable part of the intrasynovial part of the profundus tendon into the wound (Fig. 2). A standard injury was then inflicted on the profundus tendon at the point where it was just disappearing from vision into the sheath. Using a sharp scalpel, the tendon was cut transversely for about two-thirds to three-quarters of its cross-section, just sufficient tendon being left intact to maintain continuity (Fig. 3). On releasing the traction and returning the digit to its functional position, the profundus tendon slid back into its sheath, with the injured part coming to lie over the distal third of the proximal phalanx—that is, within the zone which Bunnell termed "no-man's land". By using this indirect method of
exposing the tendon, the integrity of the synovial sheath is preserved to an extent which is not possible if the incision is made in the digit itself.

The skin was closed with a few fine catgut sutures and an aerosol collodion dressing sprayed over the wound. No other dressing was applied and no form of immobilisation was used. The tourniquet was removed and the animal allowed to recover from the anaesthetic.

A total of thirty-six rabbits was used in the experiments. The rabbits were killed, in pairs, at two days, three days, five days, eight days, ten days and then at weekly intervals up to fourteen weeks. Two series of specimens were thus available for study.

![Figure 4](image1.png)

**FIG. 4**—Specimen of tendon three weeks after operation. The divided tendon fibres have retracted away from the site of the initial cut and the resulting gap is filling with a reparative tissue derived from the tendons. Figure 5—Longitudinal section of tendon and sheath at three days. The synovial membrane (1) is normal and the peritendinous space (2) has not been obliterated. The divided tendon fibres (3) have begun to retract and changes in cell density and morphology are visible in the region of the cut. (Haematoxylin and eosin, × 70.)

Specimens from one of the series were first examined under a dissecting microscope and note was made of the state of the traumatised area, the overlying tendon sheath and the presence or otherwise of adhesions. The profundus tendons were then removed and fixed in 10 per cent formol saline. The specimens in the other series were dealt with slightly differently, in that the tendons were fixed and processed en bloc within their sheath. This allowed sections to be cut of the tendon in its sheath and avoided the possibility that loose fine adhesions might be torn across and overlooked.

After processing and embedding, serial longitudinal sections were cut and were stained with haematoxylin and eosin, James's silver stain and Hale's colloidal iron/PAS stain.

**RESULTS**

Formal assessment of function was not attempted because it was thought unlikely that this would provide any useful information. It was noted, however, that within a few days of
operation the rabbits used their paws normally, and as far as could be seen digital movements were not restricted.

Macroscopic appearances—Examination of the specimens showed that at all stages the tendon was healing without the formation of adhesions.

In the earliest specimens examined, the area of tendon injury appeared as a clean cut lying at the level of the neck of the proximal phalanx. Very soon the cut edges of the tendon became swollen and retracted. This retraction of the cut edges away from the site of the original incision progressed until the tenth day, and resulted in an appearance of a narrow strand of intact fibres lying between two prominences formed by the retraction and rucking of divided fibres.

By the tenth day there was a significant change in the gross appearance of the tendon. Over the injured area of tendon a smooth, glistening and semi-transparent membrane had appeared. There was further formation of this new tissue over the next few days, and by the third week it had developed into a firm, jelly-like tissue, adherent to the underlying tendon and spanning the gap between the ends of the cut fibres as a crescentic, translucent veil (Fig. 4). More of this reparative tissue was laid down in the weeks that followed, and the gap gradually filled in from its floor outward. By the eighth to the tenth week the surface of the newly formed tissue was flush with that of the normal tendon and the defect had been obliterated.

From the eighth week on, the site of injury became gradually less easy to define and by the fourteenth week it was impossible to distinguish the injured area of tendon from the normal by naked eye examination.
Throughout the series, adhesions of the tendon to surrounding tissues were completely absent. There was no evidence of increased vascularity either of the tendon or of its sheath. The synovial membrane remained of normal appearance.

**Microscopic appearances**—Within the first three days of injury, the most obvious microscopic feature was the retraction of the divided tendon bundles away from the site of the initial cut. This process of retraction continued for up to ten days from operation, by which time the degree of separation amounted in some specimens to as much as three millimetres. Because of this, it was not generally possible to demonstrate the changes in the whole of the injured area within one microscopic field.

At three days, changes could be seen in the tendon cells in the region of the cut (Fig. 5). There was a moderate increase in the cell density within this zone, and a change from the normal elongated shape of quiescent tendon cells to a marked preponderance of rhomboid basophilic cells with ovoid nuclei. In many places the typical formation of tendon cells in regular rows was lost. The number of these reparative cells, which we may term “tenoblasts”, rapidly increased over the next few days and by the eighth day they had formed a definite layer in the floor of the cut and over the retracted ends of the cut collagen bundles (Fig. 6). By this time also it was possible to observe, in sections prepared with collagen stains, delicate new collagen fibrils in relation to the surfaces of the proliferating tenoblasts (Fig. 7). The orientation of these fibrils was, from their first appearance, more or less in the line of the tendon.

From the eighth day on it was possible to follow a regular sequence in the formation of new tendon tissue. The space between the cut ends of the fibres was gradually filled with
young reparative tissue composed of tenoblasts and maturing collagen fibres (Figs. 8 and 9). The youngest, most recent generation of tenoblasts lay on the superficial surface in contact with the synovial fluid. Adjacent to these cells, and presumably laid down by them, were the most delicate of the new collagen fibrils (Fig. 10). Through the underlying layers, these fine fibrils appeared to thicken or coalesce, and at the same time they acquired a purely longitudinal alignment. In the deepest layers the new collagen had taken on the characteristics of mature tendon fibres, and continuity had been established with the severed bundles.

By the twelfth to the fourteenth weeks, the defect in the tendon had been completely replaced with mature tendon fibres, and the microscopic appearances suggested that full tensile strength had been restored.

At no stage during the repair process was there any indication that the reparative tissue had originated other than from the tendon itself. Adhesions between the tendon and its sheath were never seen, and the synovial membrane appeared normal throughout.

**DISCUSSION**

These experiments have shown that, under ideal conditions, the digital flexor tendons are not inert but possess the ability to repair areas of injury without delay and without the ingrowth of adhesions. In all the specimens studied, the tendon itself appeared to be the sole agent in bringing about reconstitution of the defect, and the tendon cells exhibited marked reparative activity. Repair was effected firstly by the transformation of resting tenocytes into an active tenoblast form, followed by the proliferation of these cells and the production of new collagen.

These findings are at variance with the views of Potenza (1962) on tendon healing. Studying the pattern of healing of completely divided profundus tendons, he found that the tendon cells played no active role in the repair process and that union was effected entirely...
by the cellular activity of the sheath. He concluded from this that tendon tissue is inherently passive in the matter of its own repair, the tendon cells lacking the ability to behave as fibroblasts, perhaps because they have reached a functional end stage in mature tendon.

It is possible to reconcile these apparently conflicting findings if the experimental conditions are taken into account. In Potenza's experiments, since the tendons had been completely severed, the need to maintain constant apposition of the stumps necessitated the insertion of tendon sutures, further damage to the synovial sheath and prolonged splintage of the operated limb. It follows that the process observed was not simply the response of tendon tissue to injury, but also its reaction to these secondary factors. It is known that at least one of these, tendon suture, is able to excite adhesion formation even in uninjured tendon (Lindsay and Thomson 1960). It seems wrong, therefore, to infer that, because a cut tendon held together in this way heals by adhesions, this is its natural mode of repair and the only one of which it is capable. We suggest that the reason why divided tendons have been shown to heal by adhesions rather than by tenoblast activity is not so much the fault of the tissue as of adverse conditions created by the surgical repair.

The failure of tendon stumps to participate in healing may, we believe, have a nutritional basis. The response of any tissue to injury is dependent upon its state of nutrition. In the case of digital flexor tendons there are two possible sources of nourishment, namely the blood flow in the intratendinous vessels and the diffusion of metabolites through the synovial fluid. Both these sources are at risk when a tendon is joined by conventional suture techniques. All tendons appear to possess blood vessels but the rate of flow through them is extremely low (White, Ter-Pogossian and Stein 1964). This, together with the arrangement of the vessels in a dense tissue with a minimum of areolar packing, must render the circulation vulnerable to pressure. The existence of areas of ischaemia within the portions of tendon gripped by sutures has, in fact, been demonstrated (Bergljung 1968), and it is thought that they are caused by compression of the intratendinous arterioles. The role of the synovial fluid in tendon nutrition is far less clearly defined. Although fluid is regarded as being produced primarily to facilitate gliding of the tendon within its sheath, evidence has accumulated that it also has a nutritive function (Potenza 1963, 1964). If this is so, it is understandable that the damage to the synovial sheath incurred in suturing a tendon might interfere with the normal exchange of metabolites across the synovial pathway. It is, we suggest, possible that impairment of tendon nutrition resulting from factors involved in maintaining stump apposition may reduce the vitality of the tendon cells to a point at which, even if they can survive, they are no longer able to participate in repair. The relative importance of these factors is the subject of continuing investigation.

Although we have been able to demonstrate that tendon is a tissue which possesses a potential for repair, it remains to be seen whether it will ever be possible in clinical practice to achieve union of a divided tendon in the absence of adhesions. It may well prove difficult to devise a method of holding the tendon ends together which does not at the same time impair tenoblast response and provoke an adhesive reaction in the surrounding tissues.

SUMMARY

1. Tendon possesses an active potential for repair and remodelling.
2. Large defects made in the flexor tendons of rabbits showed tenoblastic activity and repair without the formation of adhesions.
3. The failure to show this intrinsic ability for repair in previous studies may have been influenced by adverse factors introduced in order to hold the cut tendon ends together.

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REFERENCES


