Human Composite Flexor Tendon Allografts

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IN 1959 one of us reported the results of grafting the entire flexor mechanism of a recently deceased individual into the damaged finger of a 40-year-old patient.[1-3] The graft consisted of an intact digital sheath surrounding both the flexor tendons and the specialized mesentery which connects tendons and sheath (Fig. 1). The theoretical advantage of transplanting the digital flexor mechanism intact is that postoperative fibrous tissue healing should occur only between the recipient bed and the exterior of the transplanted sheath. Because the intricate sheath-mesentery-tendon relationship in such a graft would not be involved or altered by the healing process, gliding function should be maintained. Previously reported studies on the fate of such grafts in experimental animals revealed that donor cells were replaced by recipient cells over a 28-day period.[4] Antigenicity (measured by challenging recipient animals with skin from the same donor and performing complement fixation tests on recipient serum using reconstituted collagen as an antigen) was not significant.[5]

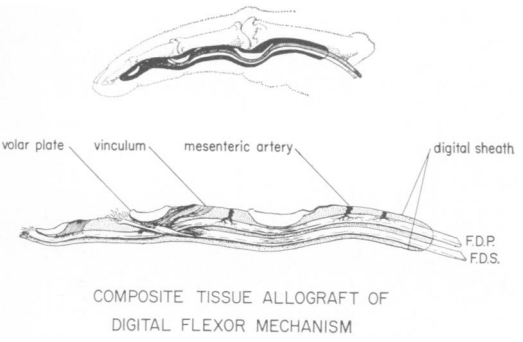

FIG. 1. Schematic diagram of composite tissue allograft of flexor mechanism.

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During the past 10 years, 11 composite flexor tendon allografts have been placed in 10 patients. This paper is a report of the results of these grafts.

Methods

Procuring the Graft. Permission to remove a flexor tendon graft from a cadaver has not been difficult to obtain. The size of a donor finger should be comparable to the recipient finger, although the donor and recipient fingers need not be the same. Age of the donor is immaterial; donors with neoplastic and infectious disease have not been used. Two grafts from jaundiced donors with Laennec's cirrhosis were transplanted without adverse effects.

The cadaver is taken to the operating room and the arm, hand, and fingernails are prepared. The right hand is preferred because the left is generally exposed when the body is viewed. A volar incision extending down the center of the finger and hand is inconspicuous and produces excellent exposure. After reflecting skin flaps laterally, excision of the graft is begun by making incisions in the lateral periosteum of the phalanges. Using only a sharp scalpel, the flexor mechanism is dissected away from the phalanges (Fig. 2). The dorsal aspect of the sheath is thin and cannot be separated from periosteum. At the points of insertion of the profundus and sublimis

tendons, Sharpey's fibers create a special problem. There is a tendency to follow these fibers into the graft unless special precautions are taken to divide them close to the bone. Volar plates are removed with the graft, and both tendons are removed as far proximal as the lower forearm.

After the graft has been removed, it is inspected for small tears in the sheath (Fig. 3) which if found, should be repaired with fine sutures. The graft, wrapped in a gauze sponge moistened with physiologic saline to which penicillin and streptomycin have been added, is placed in a tightly capped sterile bottle, and refrigerated at 4° C. The divided ends of all significant blood vessels in the donor hand should be identified and ligated. An annoying leak of embalming fluid will occur if this step is omitted. The skin incision is closed with a #40 running wire suture.

Transplantation of the Graft. The damaged finger is prepared by opening it along the radial midlateral line or, preferably, by raising volar flaps based on the neurovascular bundles (Fig. 4). The incision is continued into the palm as far proximally as the proximal crease. Damaged flexor tendons and remnants of sheath are excised sharply leaving only the volar plates over the interphalangeal and metacarpophalangeal joints (Fig. 5). Several small periosteal tags are preserved along the sides of the phalanges and at the tip of the distal phalanx to serve as points of attachment for the graft. Tendons are exposed proximally through an oblique incision at the wrist.

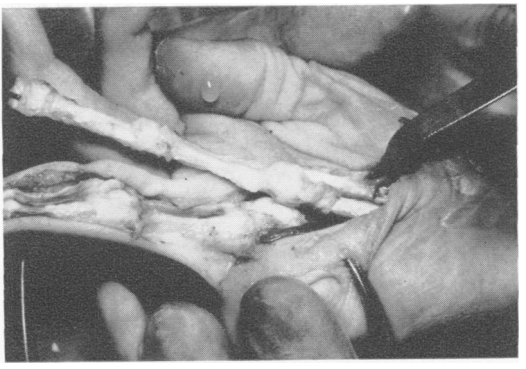

FIG. 2. Excision of composite tissue allograft of digital flexor mechanism. Note volar plates remain attached to graft.

The graft is placed in the prepared digit where it is attached to bone by suturing the graft to the periosteal tags (Fig 6). After the finger has been closed, both tendons are anastomosed by end-to-end buried suture technic. The profundus tendons usually are anastomosed in the palm; the sublimis tendons usually are anastomosed at the wrist. Both tendons can be sutured at the wrist if the suture lines are staggered. Tension must be adjusted carefully so that the finger is in slightly more than normal flexion.

Postoperative Care. The hand is dressed and immobilized with the wrist in approximately 20 degrees of flexion. The reconstructed digit should be almost completely extended at the metacarpophalangeal and distal interphalangeal joints. No more than 15 to 20 degrees flexion should be allowed at the proximal interphalangeal joint. The dressing is removed weekly to put all joints through a passive range of motion; no ac-

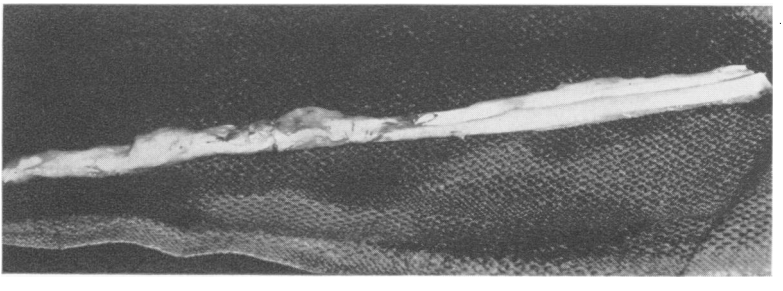

FIG. 3. Composite tissue allograft of digital flexor mechanism.

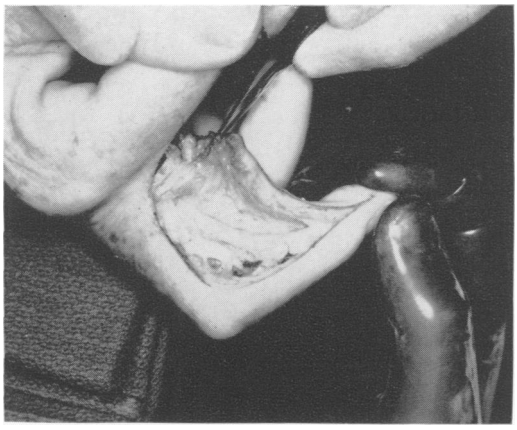

FIG. 4. Exposure of volar cicatrix in contracted long finger. Scar tissue is the result of tenosynovitis.

tive flexion is permitted until the beginning of the fifth week. After the final dressing has been removed, active flexion with the metacarpophalangeal joint held in extension is begun. Passive extension is not permitted until the beginning of the sixth postoperative week. Both active and passive motions may continue to improve for as long as 3 months.

Results

Results of 11 grafts in 10 patients are shown in Table 1. Because the majority of these patients did not have normal passive range of motion preoperatively, results are reported in terms of the ratio of active to passive motion. Grafts were considered successful if restored active motion equaled passive motion. Because existing tendon and sheath remnants in the recipient finger improved passive motion in most fingers, postoperative motion usually was greater than preoperative motion (Fig. 4, 5). Seven grafted fingers recovered active motion equal to passive motion. One patient received two grafts; one was successful, the other restored active motion to only 50% of passive motion. One patient has been operated upon too recently for final evaluation. Two patients did not gain flexion and were classified as failures.

The first patient operated upon was started on active motion 3 weeks following operation. Three days later her finger suddenly became flail. Exploration revealed that separation had occurred at the profundus tendon junction. Examination of the sheath and tendon relationships revealed that the graft was intact and that there were no apparent adhesions between tendons and sheath. The dehiscence was repaired, and 4 weeks later active motion was started which ultimately was equal to passive motion.

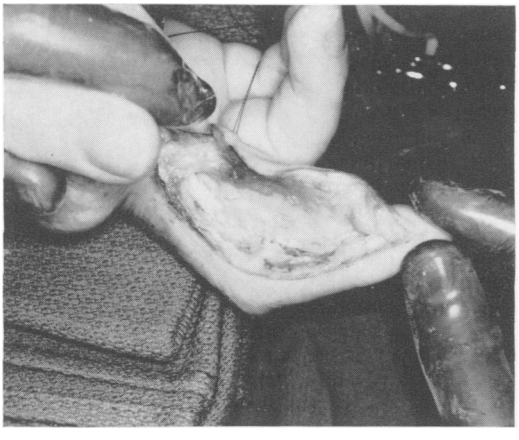

FIG. 5. Same patient as in Figure 4. Note partial correction of flexion contracture following excision of scarred tendon and sheath.

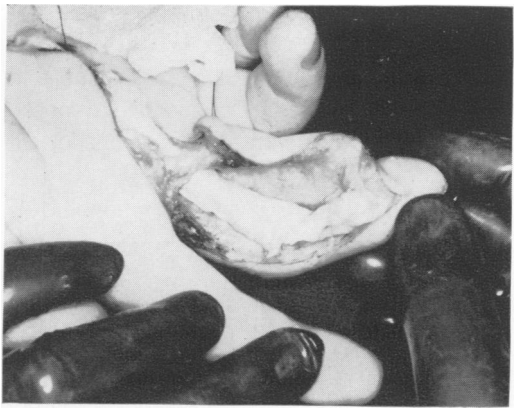

FIG. 6. Same patient as in Figure 4. Composite tissue allograft in prepared digit. Note periosteal tags attached to lateral periosteum of middle phalanx.

TABLE 1. *Results of Composite Tissue Allografts of Finger Flexor Mechanism. North Carolina Memorial Hospital 1957–1967*

NCMH No.	Age/Sex	Date	Digit	Classification	Result
06-43-04	47F	1957	Index	Tenosynovitis	Active = Passive
07-35-99	25M	1958	Long	Congenital absence	Active = Passive
08-94-76	28M	1959	Small	Autograft failure	Failure-Flicker of motion
07-90-57	17M	1959	Index	Primary repair failure	Failure-Flexion contracture
03-36-16	47M	1961	Ring	Tenosynovitis	Active = Passive
11-29-72	47M	1962	Index	Soft tissue avulsion	Active = 50% of passive
			Long	Soft tissue avulsion	Active = Passive
11-35-75	14M	1962	Ring	Tenosynovitis	Active = Passive
13-36-30	14F	1964	Small	Autograft failure	Active = Passive
19-36-62	35M	1966	Long	Tenosynovitis	Active = Passive
19-26-52	19M	1967	Ring	Autograft failure	Too recent to evaluate

Discussion

Following uncomplicated lacerations of digital flexor tendons, it is usually possible to restore useful flexion by performing a conventional tendon autograft. When soft tissue damage has been extensive or complications such as infection have destroyed normal tendon-sheath relationships, autografts have been uniformly disappointing. It is in these cases that the time and difficulty involved in obtaining composite tissue allografts have appeared justified. Although satisfactory restoration of flexion was obtained in only 70% of patients receiving allografts, these patients were a select group in whom autografts already had been unsuccessful or in whom autografts had not been attempted because of impossible local conditions. Composite tissue tendon grafts restored function in a substantial number of patients in whom the salvage rate by conventional autografting would have been near zero. At this stage in our evaluation of composite tissue allografts, the main indication for their use appears to be failure of a conventional autograft or in patients in whom complications in surrounding tissues make autografting impossible (Fig. 7, 8, 9).

Analysis of the three autografts which did not restore active motion equal to passive motion is incomplete because in only one patient were we able to dissect the hand and establish why gliding failed. In this patient, adhesions were not found

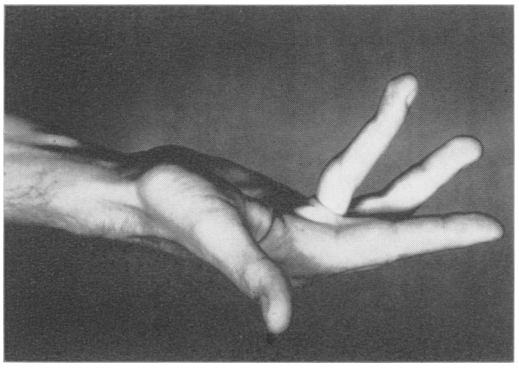

FIG. 7. Preoperative view of patient with absence of digital flexor tendons and sheath secondary to laceration and tenosynovitis at 6 months of age. Patient attempting to flex affected finger.

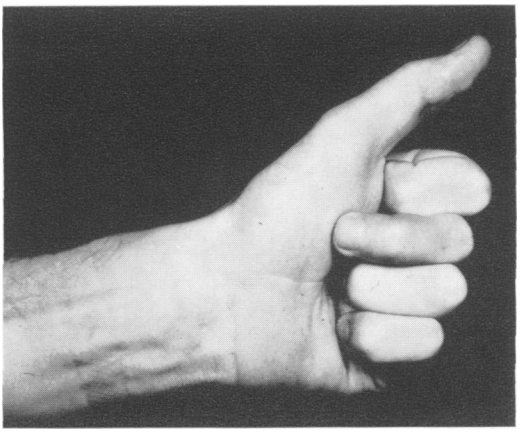

FIG. 8. Postoperative view of patient in Figure 7. Note flexion of proximal and distal interphalangeal joints.

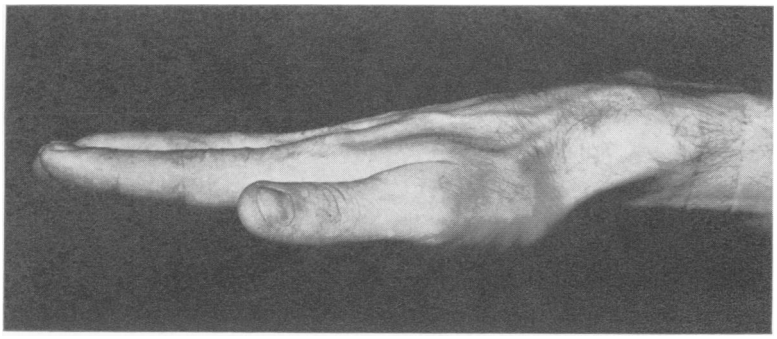

FIG. 9. Same patient as in Figures 7 and 8. Note complete extension of long finger.

between the transplanted sheath and tendon; normal gliding between tendons within the sheath was present. Failure of the transplant to function was due entirely to the formation of dense, restricting, collagenous adhesions along the exposed portions of both tendons in the palm and wrist. Failure of these adhesions to undergo remodeling of a type permitting gliding function represents a biological variant of the healing mechanism which is familiar to restorative surgeons. We can only hypothesize that failure of the graft to glide in another patient and failure of a third graft to develop more than 50% of the passive range of motion were the results of similar biological variants.

Dehiscence occurred in the first patient and was the result of failure to realize that allografted tendons do not regain structural stability, particularly lateral cohesiveness, as quickly as autografts. At least one additional week of immobilization is required before active motion is started. By allowing only passive interphalangeal joint motion for 4 weeks after an allograft restoration, we have since prevented dehiscence. Because no gross or histologic evidence of longitudinal fiber disruption was found, we believe that alterations in the ground substance of the graft occur and must be corrected before structural stability is regained.

No significant clinical evidence of antigenicity in the grafted patients was apparent. Two patients who received grafts in the ring or small fingers developed enlarged, tender epitrochlear lymph nodes two weeks later, but the nodes subsided within 10 days and no sequelae were noted. Previous studies of collagen antigenicity suggest that, although soluble collagen may be slightly antigenic in some species, insoluble collagen is relatively non-antigenic.[6] It seems likely that the dose of antigen from the relatively small numbers of transplanted cells is too small to be of clinical significance. The important question of the fate of endothelial cells in the transplant has not been investigated, although in experimental preparations they remain intact histologically.

Summary

Entire flexor tendon mechanisms can be grafted successfully in human fingers. Grafts retain their specialized architecture and gliding function.

Indications for composite tissue allografts have been failure of a conventional autograft or complications in surrounding tissues which make autografting impossible.

Eleven composite grafts have been performed in 10 patients. Seven fingers had postoperative active motion equal to passive motion and have been classified as successful; three grafts have been classified as failures; one is too recent to evaluate.

No significant antigenicity has been encountered.

References

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Discussion

Dr. Gordon H. Grant (Victoria, Canada): Dr. Creech, Members and Guests of the Association: Thank you very much for the privilege of the floor for a brief and unprepared discussion.

I have been enormously stimulated—and I am sure you have been—by the exciting suggestions that Dr. Peacock has brought to us here today. He has been doing this for us over the years in the American Society for Surgery of the Hand and, if we have begun to make some forward progress in the more refined physiologic areas of hand surgery, he certainly has been among those who have been greatly responsible.

I have been stimulated by this presentation, perhaps, for a number of reasons. One is, of course, that the incidence of trauma persists and continues to mount on this continent. In the past we have had to settle for results in the repair of, or substitution for, normal tendons that we simply would not consider satisfactory in other areas of surgery.

We have heard something today that points the way to a much more effective substitution in the future, when immunosuppression becomes more of a clinical fact. This is exciting enough if one considers only the repair of trauma but there is that vast army of rheumatoid sufferers that we perhaps can begin to view with more optimism than was possible in the past.

There are between 7 and 8 million rheumatoid sufferers in the United States and Canada. In a great many of these the dominant hand is attacked; in most of these the all-important metacarpophalangeal complex is affected. As a result the function of the hand is reduced to a mere parody of what hand function ought to be.

Now if we can begin to think of a better immunosuppressive day, we can also begin to contemplate a total substitution, say, of a complete metacarpophalangeal complex, together with a complete flexor unit. When that day arrives we should be able to come up with something a good deal less primitive than what we have to settle for today.

Dr. Dwight C. McGoon (Rochester, Minn.): I beg the indulgence of the Association and Dr. Peacock for speaking on a subject in which I have no experience.

In other areas of the body where replacement of a purely mechanical device or structure is involved, there is a great controversy now as to whether homografts or prosthetic material should be used.

I simply would like to ask the authors if in this area a prosthesis would have any benefit.

Dr. Erle E. Peacock, Jr. (Closing): Dr. McGoon, the problem here is very similar to one which you do have experience in, and that is a mechanical situation in which, when you find something really inert enough, in the end that's what gets it. In the end it's so inert that it can't be replaced, and it undergoes a great deal of mechanical damage through the years, so that it simply doesn't last long enough.

There has been considerable work done on a replacement with a synthetic material, and the two things that have licked it so far have been: 1) Trying to find a way to join it to the proximal muscle without creating a lot of scar tissue that will restrict motion; and 2) Finding something that would last for another 35 or 40 years with the continual tremendous mechanical stimulation that it's subjected to.

Dr. Grant, thank you very much for your generous remarks and for developing our thesis further.