PLASTIC SURGERY

VOLUME 8
THE HAND
Part 2

PLASTIC SURGERY

VOLUME 8
THE HAND
Part 2

1990 W.B. SAUNDERS COMPANY

Harcourt Brace Jovanovich, Inc.

Philadelphia • London • Toronto
Montreal • Sydney • Tokyo

W. B. SAUNDERS COMPANY

Harcourt Brace Jovanovich, Inc.

The Curtis Center Independence Square West Philadelphia, PA 19106

Library of Congress Cataloging-in-Publication Data

Plastic surgery.

Contents: v. 1. General principles-v. 2-3. The face-v. 4. Cleft lip & palate and craniofacial anomalies-letc.]

1. Surgery, Plastic. I. McCarthy, Joseph G., 1938-[DNLM: 1. Surgery, Plastic. WO 600 P7122]

RD118.P536 1990 617'.95 87-9809

ISBN 0-7216-1514-7 (set)

Editor: W. B. Saunders Staff Designer: W. B. Saunders Staff Production Manager: Frank Polizzano Manuscript Editor: David Harvey Illustration Coordinator: Lisa Lambert

Indexer: Kathleen Garcia Cover Designer: Ellen Bodner

Volume 1 0-7216-2542-8

Volume 2 0-7216-2543-6

Volume 3 0-7216-2544-4

Volume 4 0-7216-2545-2 Volume 5 0-7216-2546-0

Volume 6 0-7216-2547-9

Volume 7 0-7216-2548-7 Volume 8 0-7216-2549-5

8 Volume Set 0-7216-1514-7

Plastic Surgery

Copyright © 1990 by W. B. Saunders Company.

All rights reserved. No part of this publication may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopy, recording, or any information storage and retrieval system, without permission in writing from the publisher.

Printed in the United States of America.

Last digit is the print number:

Contributors

STEPHAN ARIYAN, M.D.

Professor of Surgery and Chief of Plastic and Reconstructive Surgery, Yale University School of Medicine; Chief of Plastic Surgery, Yale–New Haven Hospital, New Haven; Consultant in Plastic Surgery, Veterans Administration Hospital, West Haven, Connecticut.

PAUL W. BRAND, C.B.E.-M.B.-B.S., F.R.C.S.

Clinical Professor of Orthopaedics and Surgery, Louisiana State University Medical School; Senior Consultant, Gillis W. Long Hansen's Disease Center, Carville, Louisiana.

BENJAMIN E. COHEN, M.D.

Clinical Assistant Professor, Division of Plastic Surgery, Baylor College of Medicine; Academic Chief and Director, Plastic Surgery Residency Program, and Director, Microsurgical Research and Training Laboratory, St. Joseph Hospital, Houston, Texas.

MATTHIAS B. DONELAN, M.D.

Assistant Clinical Professor of Surgery, Harvard Medical School; Chief, Plastic and Reconstructive Surgery, Shriners Burns Institute, Boston; Assistant Surgeon, Massachusetts General Hospital, Boston, Massachusetts.

VINCENT R. HENTZ, M.D.

Associate Professor of Surgery, Stanford University School of Medicine: Chief, Division of Hand and Upper Extremity Surgery, Stanford University Hospital, Palo Alto, California.

LYNN D. KETCHUM, M.D.

Clinical Professor of Surgery, University of Kansas Medical Center, Kansas City; Attending Surgeon, Humana Hospital, Overland Park, Kansas.

J. WILLIAM LITTLER, M.D.

Senior Attending Surgeon, St. Luke's-Roosevelt Hospital Center, New York, New York.

RALPH T. MANKTELOW, M.D.

Professor and Head, Division of Plastic Surgery, University of Toronto Faculty of Medicine; Head of the Division of Plastic Surgery, Toronto General Hospital, Toronto, Ontario, Canada.

IVAN MATEV, M.D.

Professor of Orthopaedic Surgery, The Medical Academy, Head, Department of Upper Extremity Surgery, The Institute of Orthopaedics and Traumatology, Sofia, Bulgaria.

JAMES W. MAY, JR., M.D.

Chief of Plastic and Reconstructive Surgery and Hand Surgery Service, Department of General Surgery, Massachusetts General Hospital; Associate Clinical Professor, Harvard Medical School, Boston, Massachusetts.

ROBERT M. McFARLANE, M.D.

Professor of Surgery and Head, Division of Plastic Surgery, University of Western Ontario Faculty of Medicine; Head, Division of Plastic Surgery, Victoria Hospital, London, Ontario, Canada.

MARY H. McGRATH, M.D.

Professor of Surgery and Chief, Division of Plastic and Reconstructive Surgery, George Washington University School of Medicine and Health Sciences; Chief of Service, University Hospital; Attending Surgeon, Children's Hospital National Medical Center, Washington, D.C.

ERIK MOBERG, M.D., PH.D.

Professor Emeritus of Hand Surgery and Orthopaedic Surgery, University of Göteborg Medical School, Göteborg, Sweden.

WAYNE A. MORRISON, M.B., B.S., F.R.A.C.S.

Associate, Department of Surgery, University of Melbourne; Assistant Plastic Surgeon and Deputy Director, Microsurgery Research Centre, St. Vincent's Hospital, Melbourne; Plastic Surgeon, Repatriation Hospital, Heidelberg, Melbourne; Consultant Plastic Surgeon, Geelong Hospital, Victoria, Australia.

JAMES F. MURRAY, M.D.

Professor Emeritus, Department of Surgery, University of Toronto Faculty of Medicine; Attending Surgeon, Sunnybrook Medical Center, Toronto, Ontario, Canada.

HENRY W. NEALE, M.D.

Professor of Surgery and Director, Division of Plastic, Reconstructive and Hand Surgery, University of Cincinnati College of Medicine; Attending Surgeon, University Hospital, Children's Hospital Medical Center, and Shriners Burns Hospital, Cincinnati, Ohio.

CHARLES L. PUCKETT, M.D.

Professor and Head, Division of Plastic Surgery, University of Missouri-Columbia School of Medicine; Attending Surgeon, University of Missouri-Columbia Hospital and Clinics, Harry S Truman Memorial Veterans Administration Hospital, Boone Hospital Center and Ellis Fischel State Cancer Hospital, Columbia, Missouri.

ROGER E. SALISBURY, M.D.

Professor of Surgery and Chief of Plastic and Reconstructive Surgery, New York Medical Col-

lege; Director, Burn Center, Westchester Medical Center, Valhalla; Consultant, Plastic and Reconstructive Surgery, Castle Point Veterans Administration Hospital and Glythedale Children's Hospital; Chief of Plastic and Reconstructive Surgery, Metropolitan Hospital Center, New York, New York.

RICHARD J. SMITH, M.D. (deceased)

Clinical Professor of Orthopaedic Surgery, Harvard Medical School, Boston; Director of Hand Surgical Service, Department of Orthopaedic Surgery, Massachusetts General Hospital, Boston, Massachusetts.

JOSEPH UPTON, M.D.

Assistant Professor of Surgery, Harvard Medical School; Active Staff, Division of Plastic Surgery, Department of Surgery, Beth Israel Hospital and Children's Hospital, Boston, Massachusetts.

E. F. SHAW WILGIS, M.D.

Associate Professor of Plastic Surgery and of Orthopaedic Surgery, Johns Hopkins University School of Medicine; Chief, Division of Hand Surgery, Union Memorial Hospital, Baltimore, Maryland.

EDUARDO A. ZANCOLLI, M.D.

Professor of Orthopaedics and Traumatology, Medical School of Buenos Aires; Chief of Orthopaedic Surgery of the Rehabilitation Center of Buenos Aires, Buenos Aires, Argentina.

Contents

	The Hand (Part 2)
114	120
Tendon Transfer Reconstruction for Radial, Ulnar, Median, and Combination Paralyses:	Ischemic Contractures 5033 Eduardo A. Zancolli
Principles and Techniques 4923 Paul W. Brand	121
115	Dupuytren's Disease 5053 Robert M. McFarlane
Functioning Muscle Transfer	
for Reconstruction of the Hand 4966	122
Ralph T. Manktelow	Thumb Soft Tissue
116	Injury Management 5087 Benjamin E. Cohen
Surgery for the Spastic, Stroke,	
and Tetraplegic Hand	123
	Congenital Anomalies of the Thumb 5106 Vincent R. Hentz
117	vincent A. Hentz
Ischemic Conditions of	
the Upper Extremity 4991	124
E. F. Shaw Wilgis	Finger Pollicization
118	for Traumatic Loss
Vasospastic Disorders of the Hand 5012	시작 회사 시간 이 시간 이 경기 전체 기계 개념 생활
Lynn D. Ketchum	125
	Microvascular Great Toe to Hand
119	Transfer for Reconstruction
Lymphedema in the Upper Extremity 5023 Charles L. Puckett	of the Amputated Thumb 5153 James W. May, Jr.

\$3 f. Cold, Che. I a an

126	131
Thumb Reconstruction by the Wrap-Around Technique 5186	Cold, Chemical, and Irradiation Injuries
Wayne A. Morrison	James F. Murray
	132
127	Reconstruction of the
Thumb Reconstruction	Burned Hand and Upper Extremity 5452
by Bone Lengthening	Matthias B. Donelan
100	133
128	Benign and Malignant
Congenital Anomalies of the Hand and Forearm	Soft Tissue Tumors of the Hand 5483 Stephan Ariyan
is may brief ed?	134
129	Benign and Malignant
Acute Care of the Burned Hand 5399	Bone Tumors of the Hand 5510 Richard J. Smith
Roger E. Salisbury	an nan recount neutrin businesses Patrial Umar Misdran,
	135 sayora9 notendred or
130 Electrical Injuries of the Hand	Infections of the Hand
and Upper Extremity5418 Henry W. Neale	
Trong W. Neale	Index
722	- 2005 I nell entre dataurkenness er
Thumb Soft Torse	
rapa, Meningenteri Benjamin Dohin	
	services of the Spanish Street
123	
Congresses Anomore of the Tauria (\$106) Conent A Hear	
124	
Finger Politoitation	
for Trainedictions	
TAMES INC.	
725	Egyme F Williams
Abdrovascular Groat Toe 10 In and	
Franklich für Pacititist UT un	

Tendon Transfer
Reconstruction for
Radial, Ulnar,
Median, and
Combination
Paralyses: Principles
and Techniques

PRINCIPLES

Timing of Tendon Surgery
Tissue Adaptation
Psychologic State of Patient
Recovery of Nerves
Balance of Muscle Strength
Changes After Transfer
Matching of Tendon Excursions
Terminology for Excursion
Changing Excursions and Moment Arms
Management of Passive Soft Tissue Resistance
Surgical Techniques to Minimize Drag

SUGGESTED PATTERNS FOR SPECIFIC NERVE

INJURIES
Radial Nerve Paralysis
Ulnar Nerve Paralysis
Median Nerve Paralysis
High Ulnar and High Median Palsy
Radial and Ulnar Palsy
High Median and Radial
Partial Brachial Plexus Injury and Other Severe
Paralyses

PRINCIPLES

Timing of Tendon Surgery

Time waits for no man. Tissues constantly change and adapt to current patterns of position and stress. The subconscious mind reacts to injury by a restless search for ways to compensate for the disaster. Instincts for survival range over all the options, and react with undue emotion in directions of hope or despair out of all proportion to what would seem a reasonable or sober judgment. All these factors have some effect on the success or failure of reconstructive surgery, and most should encourage us to do what needs to be done as early as possible, so that the patient's own creative efforts may be based on the new pattern of muscle balance, rather than on a situation that will have to be changed again.

The following are some of the factors that should be considered when planning the timing of tendon transfer operations.

Tissue Adaptation

Normal connective tissues and normal skin are constantly responsive to the patterns of mechanical stress imposed on them. Skin is loose at joints to allow normal motion. This loose, excess skin becomes absorbed if motion no longer occurs, as in paralysis. If normal motion is restored by recovery of muscles, the tissues gradually lengthen again. However, if the motion is to be restored by a transferred muscle that lies in a wounded bed, the pattern of scar around the tendon may be determined by the limitation of the reduced joint motion. The muscle may then be unable to overcome the drag of both joint stiffness and tendon scar adhesion.

For this reason, tendon transfers should be done as early as possible. If they have to be delayed for any reason, a program of passive range of motion exercises must be instituted to maintain tissue slack. If joints are already limited in passive range when first seen, any surgery must be delayed until exercises, massage, and splinting have restored the range of motion. This is particularly important if the perpendicular distance between the skin or fascia and the axis of the joint is great enough to result in a strong leverage of restraint. An example of this is the dorsal skin and fascia of the thumb web, which may become contracted after median palsy and is then an overwhelming obstacle to the restoration of pronation and abduction.

Tissue Homeostasis. It is very unwise to perform tendon transfer and tendon grafting procedures while there is any inflammatory state in the tissues through which the tendon must pass. If the wound that caused the nerve injury also involved the local tissues where the tendon must pass, it is better to postpone any elective surgery until the tissues are cool and mobile and without any inflammatory edema.

Psychologic State of Patient

A large part of the success of any muscle balance operation depends on the active cooperation of the patient. A hand injury is a very personal thing. It often involves a person's whole self-image and may induce fear, anger, and despair, or there may be a sort of denial of its reality and significance. After a whole series of rather turbulent mental and psychologic adjustments, the patient finally is able to consider rational and constructive alternatives for the future of his limb, and for its significance to his life and work. It is absolutely vital that the physician and other members of the rehabilitation team keep in touch with the patient and choose the right

moment to begin a discussion of the prospects, and the right moment to intervene with a surgical plan.

Once the patient gets into the hands of a lawyer who works on a contingency fee system, the prospects for recovery are very much reduced. At once it becomes clear to the patient that his best chance for substantial financial compensation, whether from employer or insurance company, is for his residual disability to be severe. The lawyer only has to begin talking in terms of the astronomical figures commonly quoted at the start of a compensation suit, and the patient begins to see his disability as a potential advantage. The subconscious mental conflict makes it very difficult for a patient to put any enthusiasm into the pre- and postoperative discipline that is essential to success.

The reason for the existence of the so-called "ambulance chasing lawyers" is that they know the value of getting their word in first. They want their client to think about money before they begin to admit that they are feeling better. If the physician gets to see the patient first, it is important to establish some basic attitude and goal for recovery. This hand is going to recover; it is going to be a useful hand; he can be proud of it and will be independent. The physician will work with the patient to get reasonable compensation for his injury (perhaps recommend a lawyer who works for an honest fee system), but his real security is his own recovered hands.

The reason the subject of compensation is discussed under the heading of "timing" is that the longer reconstructive surgery is delayed, and the more a patient is forced to remain idle, waiting for nerve regeneration, the more discouraged he becomes. Enforced idleness begets acceptance of idleness, and finally resentment at the prospect of work. It is easy for a surgeon to say "it will take a year for this repaired nerve to grow down to the muscles of the hand, so come back in a year and we will see if you need a tendon transfer." The patient who comes back a year later is a different person, and is less likely to succeed than he would have been a year earlier. Then again there is a real sense in which he may have lost a year of his life. What is a year worth? In some cases it is worth submitting to an operation that is intended only to restore good activity to a hand for a year, after which the old muscles might take over again if they recover. As we

discuss various nerve paralyses, we shall point out that in some situations this policy is reasonable, where no real harm is done by an early transfer, even if the nerve should fully recover later. In weighing the value of such advice, let us recognize that the imponderable factors of hope, dignity, and selfesteem are at least as significant as the ponderable factors of grip strength and range of motion of joints.

Recovery of Nerves

This factor is last on this list in relation to timing because it is the one most commonly used to determine the timing of transfers. Obviously one needs to know whether a muscle is going to remain paralyzed before deciding whether to replace it, and the safest way to know is to wait until recovery actually takes place, or fails to take place when it should have. The trouble is that there are no precise rules about how long to wait, so that if a muscle is not recovering the physician may wait half as long again as the calculated time "just to make sure." In the case of a high ulnar nerve injury, this may mean a two year wait to be sure the intrinsic muscles will not recover.

To avoid unnecessary delays, some assumptions are in order. It is known that recovery is more likely in children and less likely in the elderly. It is known that recovery is most likely following a clean cut, and least likely following a contaminated laceration with loss of length and scarring and infection. Recovery is most likely following a fascicular repair by an experienced surgeon and least likely under emergency conditions by unspecialized surgeons. Muscle reinnervation is most likely when the injury is just proximal to the muscle because the motor fasciculae in the nerve are almost pure motor, and least likely when the injury is far proximal where the axons to any one muscle are widely distributed through the whole nerve. For example, repair of an ulnar nerve injury in the upper arm is likely to result in recovery of the affected forearm muscles, but very unlikely to give significant recovery to the intrinsic muscles of the hand, even after careful repair (Gaul, 1982).

If each of these factors is listed and given a weight for prognosis, there will be some patients in whom it is apparent that good recovery is so unlikely that early or imme-

diate transfer of tendons is justifiable. In addition to this, a careful evaluation of nerve recovery, as by quantitative Tinel's sign (Omer. 1983), should be made at intervals, so that a failure of nerve recovery may be recognized early and corrected promptly, or else compensated for by muscle balance surgery without further waste of time.

Balance of Muscle Strength

Bunnell (1948) used to call tendon transfers "muscle balance operations." This brings out an important principle: we cannot add new strength to a hand after some muscles have become paralyzed; all we can do is to rearrange what is left so that balance is restored. Radial palsy removes about one-third of the summated tension capability of all muscles below the elbow. Therefore, we should not aim to restore the original extensor power of the wrist or fingers by tendon transfer, but perhaps two-thirds of each. In doing so, it would be reasonable to reduce by about onethird the strength of the activities that remained unparalyzed.

In order to plan a new muscle balance for the hand, after injury or disease, we should be able to calculate the effects of the loss of muscles and the probable effects of transferring others. We should also have some idea of the extent to which a muscle can change its mechanical characteristics after transfer to match the requirements of its new situation.

There are only two major variables that define the active capability of any muscle, and one variable to define its passive reaction to stress. The active variables are tension capability and excursion capability. The passive variable is the viscoelastic response to stretch and recoil. Most previous attempts to grade muscle "strength" have not distinguished among these three, and as a result wrong advice has been given about tendon transfers. Most of us were brought up to grade muscle strength on a scale of 1 to 5, where 5 was normal strength and 1 was just an ineffective twitch. We were further told that after a tendon was transferred it would ordinarily function at about one grade lower than it had in its original situation.

This has to be wrong. When a tendon is transferred, there is no reason for any change in any of its basic active characteristics. It

has the same cross sectional area, and its fibers are the same length. It has the same number and quality of sarcomeres. Its blood supply is the same and its nerve supply has not been touched. Why should it be weaker? The answer is that it does not become weaker: it simply has a greater amount of passive drag to overcome before its tension capability can be fully transmitted to the joints where it is inserted. That drag is due mostly to the scar tissue surrounding the tendon and muscle in its new pathway. Once that fact is realized, it becomes obvious that a minor transfer involving the relocation of only the distal centimeter or two of tendon "weakens" a muscle much less than a major change of direction where a tendon and muscle has to be widely freed up and has to negotiate curves and angles, and therefore has to stretch a lot of scar before it can move.

ISOLATED MUSCLE FIBER

When a muscle fiber is considered as an isolated unit, a length-tension curve may be drawn that defines its capability. This curve demonstrates mechanical features that have been well described by Elftman (1966) (Fig. 114-1). When the muscle fiber is in the limb and in equilibrium with all other muscles, it rests more or less midway between its position of full contraction and its position of full passive stretch. In this state it is capable of its maximal contractile force. The tension capability is reduced as it shortens, becoming zero before it reaches one-half its resting length. When the fiber is lengthened its tension capability is also reduced, and it becomes near zero at about 50 per cent longer than its resting length. Thus, the resting length of any muscle fiber is about the same as the length of its maximal possible contraction

from full stretch to full active shortening. This is a useful relationship to remember. since the fiber length can be seen in any fresh cadaver dissection or at any operation when part of a muscle is exposed. If any one fiber of a muscle is traced carefully from its origin to its insertion on tendon, and if the limb is in a neutral position, the length of that fiber is the length through which it might contract under optimal conditions.

Blix (1891, 1893, 1894) extended the basic curve of a muscle fiber to include the lengthtension curve of passive stretch and recoil of the muscle and its connective tissue (Fig. 114-2). The dotted line curve on the diagram represents the tension that is put into a muscle fiber when it is passively pulled out and lengthened. At any point, if the passive stretch is discontinued and if the muscle is stimulated to contract at the same time, the two curves, active and passive, may be added together, and the resultant curve shows the summated tension that pulls on the tendon. At full stretch, most of that high tension is simply passive recoil. Near full contraction the active muscle is on its own, with no help from passive recoil.

MUSCLES IN PAIRS

The contracting muscle fiber is not only without help from passive tension when it is short, but it is also hindered by passive tension. When Blix was drawing his curve he included the tension of the muscle he was studying, but he neglected to include the tensions in the opposing muscle with which the primary muscle is inescapably bound. This is ungrateful in that the tension available to augment the contraction of the stretched muscle has been put into it by the muscle on the other side of the limb that

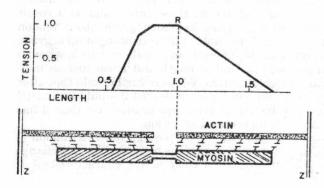


Figure 114-1. Above, Length-tension diagram of a single sarcomere unit. R = Physiologic resting length. Below, Diagram of sarcomere showing a pair of actin and myosin filaments between a pair of Z-plates.

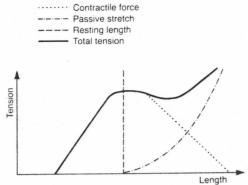


Figure 114-2. Basic concept of Blix curve.

stretches it. It is also unrealistic because not only does the primary muscle benefit by its opposite muscle, but it also unavoidably has to return the favor by putting tension into its helper when the tables are turned. At this point, we had better call the muscles A and B and observe the diagram that dramatizes their interaction. Muscles can only contract. They have no ability to lengthen without help. When B contracts, it lengthens A. In doing so it uses some of its own energy and transfers it to A, so that A receives potential energy that it can expend later, to augment its own contraction from the stretched position. As muscle A shortens further, the roles are reversed and A has to use some of its own energy, in order to stretch muscle B. This limits the tension output of A for effective work. Thus the old Blix curve gives an incomplete idea of the length-tension behavior of a fiber.

The author has proposed a new diagram (Brand, 1985) that includes the two major elements of the Blix curve and adds a third element, which is the passive tension of an opposing muscle that must be stretched when the first muscle contracts (Fig. 114-3). When these three elements are added to give the true output of a muscle fiber, it is obvious that a muscle not only is able to generate tension more effectively in the stretched position because of passive stretch, but is able to generate tension less effectively in the contracted position because of the need to stretch its partner. The final composite diagram gives a more nearly square curve, with a shorter excursion, through which there is a more sustained tension.

The more one studies the reciprocal assistance that muscles constantly give to each

other, the less appropriate it seems to speak of "opposing muscle": they are partners. As in a dance, one retreats while the other advances in harmony, and either one is lost without the other (Fig. 114-4). This is a reminder that in cases of very severe paralvsis, when the few remaining muscles have to be carefully distributed to the most essential functions of the hand, all muscle allocations must be considered in pairs. An isolated muscle is just a twitching lump of flesh until its loval opposition challenges it to usefulness by putting it on the stretch.

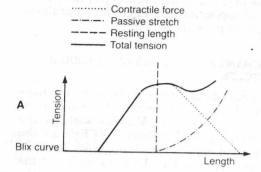
MECHANICAL QUALITIES OF INDIVIDUAL MUSCLES

It is easy to be deceived about the mechanical qualities of a muscle by looking only at its gross appearance. Muscles such as the extensor carpi radialis brevis (ECRB) and the flexor carpi ulnaris (FCU) both look fusiform and seem to be about the same bulk, yet the former has 50 per cent longer fibers while the latter is capable of 50 per cent more tension (Table 114-1) (Brand, Beach, and Thompson, 1981). The author has prepared tables of all muscles below the elbow and has recorded figures for each one, giving tension capability and potential excursion as well as total mass in proportion to all the other muscles. These figures are approximations based on averages from a number of cadaver arms. The graph of these numbers in Figure 114-5 (Brand, Beach, and Thompson, 1981) provides a useful way of comparing the potential performance of various muscles that may be used for transfer. At surgery a quick way to judge muscle cross sectional area (or tension capability) is to observe tendon diameter. Elliott and Crawford (1965a,b) have shown that the cross sectional area of tendons is not exactly proportional to the cross sectional area of the muscle fibers whose tension they transmit, but they are a useful guide. Thus, an inherently weak muscle always has a slender tendon. This applies only to the state of the muscle in health. A recently paralyzed muscle retains a thick tendon for a long time after its muscle fibers have wasted away.

Changes After Transfer

In deciding which muscle to use as transfer, a surgeon may wonder whether a muscle with Text continued on page 4932

Length—Tension Curve



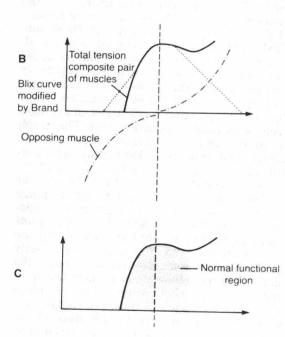
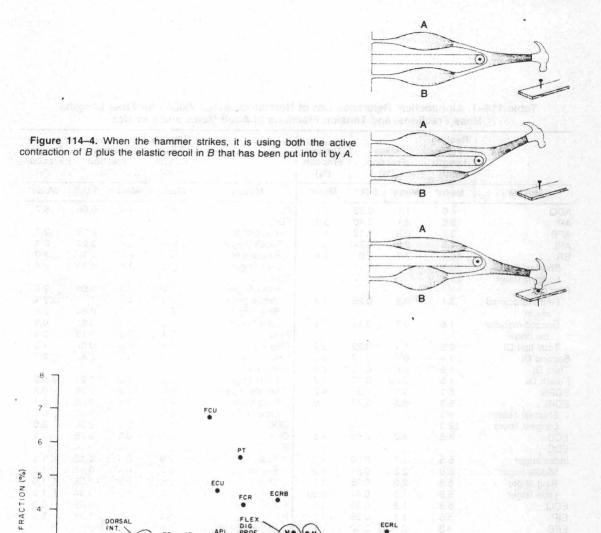


Figure 114–3. A, Blix curve, integrating the active contraction and elastic recoil. B, Brand curve integrating the above with the elastic curve of the opposing muscle subtracted from the active output of the primary muscle. C, Final approximate shape of muscle curve in situ in intact limb. (Note: The curve is more nearly square in shape compared with the isolated sarcomere in Figure 114–1. Also note the excursion is shorter than the potential excursion as judged from Figure 114–1.)



ECRL

10

12

FIFY

Figure 114-5. Using values from Table 114-1, this diagram shows the relationship between fiber length and tension producing capability. Muscle groups of similar function are circled together for clarity. ADQ = abductor digiti quinti; AP adductor pollicis; APB = abductor pollicis brevis; APL = abductor pollicis longus; BR = brachioradialis; Dorsal Int. dorsal interosseous (first, second, third, fourth); ECRB = extensor carpi radialis brevis; ECRL = extensor carpi radialis longus; ECU = extensor carpi ulnaris; Ext. Dig. Comm. = extensor digiti communis (index, middle, ring, little); EDQ = extensor digiti quinti; EIP = extensor indicis proprius; EPB = extensor pollicis brevis; EPL = extensor pollicis longus; FCR = flexor carpi radialis; FCU = flexor carpi ulnaris; Flex. Dig. Prof. = flexor digitorum profundus; FDQ = flexor digitorum quadratus; Flex. Dig. Sup. = flexor digitorum superficialis; FPB = flexor pollicis brevis; FPL = flexor pollicis longus: L = lumbricals (index, middle, ring, little); ODQ = opponens digiti quinti; OP = opponens pollicis; Palmar. Int. = palmar interosseous (first, second, third); PL = palmaris longus; PQ = pronator quadratus; PT = pronator teres.

MEAN RESTING FIBER LENGTH (cm)

DIG.

EIF

EPB

4

FDQ

3

4

2

0

0

TENSION 3 DORSAL

2

Table 114–1. Alphabetical Reference List of Normal-Expected Values for Fiber Lengths, Mass Fractions, and Tension Fractions in Adult Males and Females

Muscle	Resting Fiber Length (cm)	Mass Fraction (%)		Tension Fraction (%)	de president in Service La treace service in	Resting Fiber Length (cm)	Mass Fraction (%)		Tension Fraction (%)
	Mean*	Mean*	S.D.*	Mean*	Muscle	Mean*	Mean*	S.D.*	Mean*
	4.0	1.1	0.23	1.4	FCU	4.2	5.6	0.66	6.7
ADQ	3.6	2.1	0.40	3.0	FDP				
AP	3.7	0.8	0.18	1.1	Index finger	6.6	3.5	0.76	2.7
APB		2.8	0.10	3.1	Middle finger	6.6	4.4	0.94	3.4
APL	4.6	7.7	2.0	2.4	Ring finger	6.8	4.1	1.1	3.0
BR	16.1	1.1	2.0	2.4	Little finger	6.2	3.4	0.93	2.8
Shortest fibers	10.9				FDS				
Longest fibers	21.3				Index finger	7.2	2.9	0.64	2.0
First DI		7	0.05	4.0	Middle finger	7.0	4.7	1.1	3.4
First metacarpal	3.1	0.8	0.25	1.3		7.3	3.0	0.84	2.0
origin					Ring finger	7.0	1.3	0.81	0.9
Second metacar-	1.6	0.6	0.11	1.9	Little finger	3.4	0.3	0.10	0.4
pal origin					FDQ		0.9	0.10	1.3
Total first DI	2.5	1.4	0.29	3.2	FPB	3.6	3.2	0.42	2.7
Second DI	1.4	0.7	0.17	2.5	FPL	5.9	3.2	0.42	2.7
Third DI	1.5	0.60	0.19	2.0	Lumbrical		0.0	0.00	0.2
Fourth DI	1.5	0.50	0.13	1.7	Index finger	5.5	0.2	0.08	0.2
ECRB	6.1	5.1	1.3	4.2	Middle finger	6.6	0.2	0.06	
ECRL	9.3	6.5	0.77	3.5	Ring finger	6.0	0.1	0.06	0.1
Shortest fibers	6.3				Little finger	4.9	0.1	0.05	0.1
Longest fibers	12.3				ODQ	1.5†	0.6	0.20	2.0
ECU	4.5	4.0	0.52	4.5	OP	2.4†	0.9	0.26	1.9
EDC	1.0				PI				
	5.5	1.1	0.20	1.0	First	1.5	0.4	0.12	1.3
Index finger	6.0	2.2	0.51	1.9	Second	1.7	0.4	0.11	1.2
Middle finger	5.8	2.0	0.35	1.7	Third	1.5	0.3	0:08	1.0
Ring finger	5.8	1.0	0.33	0.90	PL	5.0	1.2	0.34	1.2
Little finger			0.35	1.0	PQ	3.0†	1.8	0.32	3.0
EDQ	5.9	1.2	0.36	1.0	PT	5.1	5.6	1.24	5.5
EIP	5.5	1.1			Superficial fibers	6.5			
EPB	4.3	0.70	0.32		Deep fibers	3.7			
EPL	5.7	1.5	0.48	1.3	Supinator	2.7†	3.8	0.95	7.1
FCR	5.2	4.2	0.87	4.1	1 Supmator	2./1	0.0	0.00	

^{*}Data from 15 hands determined the mean and standard deviation of the mass fraction for each muscle. Mass and fiber length measurements from the last five of these hands were used to calculate tension fractions.

Figures for mass (or volume) of muscles are calculated as percentages of the total mass (or volume) of all muscles below the elbow. Figures for tension fraction also are percentages of the total of all muscles below the elbow.

From Brand, P. W., Beach, R. B., and Thompson, D. E.: Relative tension and potential excursion of muscles in the forearm and hand. J. Hand Surg., 6:209, 1981.

[†]The fibers of these four muscles cross the joint axis with wide variation of fiber length. The figures quoted here for the mean fiber length of these four muscles are more visual estimates than mathematical averages. The mass fraction is accurate, but the tension fraction for these four muscles is only as true as the fiber length. Mean fiber lengths are included for the shortest and longest fibers of BR, ECRL, and PT because of the large range of fiber lengths. Values are included for the two segments of the first DI as well as total values. The data were not normalized for skeletal size differences.

Table 114-2. Normal Values Abstracted From Table 114-1 and Listed in Order of Magnitude for Mean Fiber Lengths, Mass Fractions, and Tension Fractions for Adults

BR ECRL FDS (ring finger) FDS (index finger) FDS (little finger) FDS (middle finger) FDP (ring finger) FDP (index finger)	16.1 9.3 7.3 7.2 7.0 7.0 6.8 6.6	BR ECRL FCU PT ECRB FDS (middle finger) FDP (middle finger)	7.7 6.5 5.6 5.6 5.1	Supinator* FCU PT* ECU	7.1 6.7 5.5
ECRL FDS (ring finger) FDS (index finger) FDS (little finger) FDS (middle finger) FDP (ring finger)	9.3 7.3 7.2 7.0 7.0 6.8	ECRL FCU PT ECRB FDS (middle finger)	5.6 5.6	PT* ECU	
FDS (ring finger) FDS (index finger) FDS (little finger) FDS (middle finger) FDP (ring finger)	7.3 7.2 7.0 7.0 6.8	FCU PT ECRB FDS (middle finger)	5.6 5.6	ECU	5 5
FDS (index finger) FDS (little finger) FDS (middle finger) FDP (ring finger)	7.2 7.0 7.0 6.8	PT ECRB FDS (middle finger)	5.6		0.0
FDS (little finger) FDS (middle finger) FDP (ring finger)	7.0 7.0 6.8	ECRB FDS (middle finger)			4.5
FDS (middle finger) FDP (ring finger)	7.0 6.8	FDS (middle finger)		ECRB	4.2
FDP (ring finger)	6.8		4.7	FCR	4.1
			4.4	ECRL	3.5
-DP (index finder)	0.0	FCR	4.2	FDP (middle finger)	3.4
	0.0		4.1	FDS (middle finger)	3.4
FDP (middle finger)	6.6	FDP (ring finger)	4.0	First DI	3.2
Lumbrical (middle finger)	6.6	ECU GOTOS TOTALIS		APL	3.1
FDP (little finger)	6.2	Supinator	3.8		3.0
ECRB	6.1	FDP (index finger)	3.5	AP	
EDC (middle finger)	6.0	FDP (little finger)	3.4	FDP (ring finger)	3.0
Lumbrical (ring finger)	6.0	FPL	3.2	PQ*	3.0
EDC (little finger)	5.9	FDS (ring finger)	3.0	FDP (little finger)	2.8
EDQ	5.9	FDS (index finger)	2.9	FDP (index finger)	2.7
FPL	5.9	APL TO THE TOTAL	2.8	FPL to display	2.7
EDC (ring finger)	5.8	EDC (middle finger)	2.2	Second DI	2.5
EPL	5.7	AP	2.1	BR	2.4
EDC (index finger)	5.5	EDC (ring finger)	2.0	Third DI	2.0
EIP	5.5	do na PQ nettoria ile utat	1.8	FDS (index finger)	2.0
Lumbrical (index finger)	5.5	vinces EPL coment of this d	1.5	FDS (ring finger)	2.0
FCR	5.2	First DI	1.4	ODQ*	2.0
PT	5.1	FDS (little finger)	1.3	EDC (middle finger)	1.9
PLE de tring on a vi	5.0	EDQ (Intile Intiger)	1.2	OP*	1.9
The second Particular research and the second	4.9	anobrel lo ales . In note	1.2	Fourth DI	1.7
Lumbrical (little finger)		ADQ	91.171704	EDC (ring finger)	1.7
APL all during and file	4.6	EDC (index finger)	1.1	ADQ	1.4
ECU	4.5	EIP	1.1	EPL	1.3
EPB	4.3		1.0	FPB	1.3
FCU	4.2	EDC (little finger)	0.9	First PI	1.3
ADQ	4.0	OP OP	0.9	Second PI	1.2
APB	3.7	number FPB resolution and sta		PL	1.2
AP	3.6	e fee APB harton blacket	0.9		1.1
FPB	3.6	Second DI	0.7	APB	
FDQ	3.4	EPB	0.7	EDC (index finger)	1.0
PQ	3.0	Third DI	0.6	EDQ	1.0
Supinator	2.7	ODQ	0.6	or di EIPI to Macobio see a	1.0
First DI	2.5	Fourth DI	0.5	Third Pl	1.0
OP	2.4	First PI	0.4	EDC (little finger)	0.9
Second PI	1.7	Second PI	0.4	FDS (little finger)	0.9
Third DI	1.5	FDQ	0.3	EPB	8.0
Fourth DI	1.5	Third PI	0.3	FDQ	0.4
ODQ	1.5	Lumbrical (index finger)	0.2	Lumbrical (index finger)	0.2
First PI	1.5	Lumbrical (middle finger)	0.2	Lumbrical (middle finger)	0.2
	1.5	Lumbrical (ring finger)	0.1	Lumbrical (ring finger)	0.1
Third PI Second DI	1.4	Lumbrical (little finger)	0.1	Lumbrical (little finger)	0.1

*See † footnote for Table 114-1.

From Brand, P. W., Beach, R. B., and Thompson, D. E.: Relative tension and potential excursion of muscles in the forearm and hand. J. Hand Surg., 6:209, 1981.