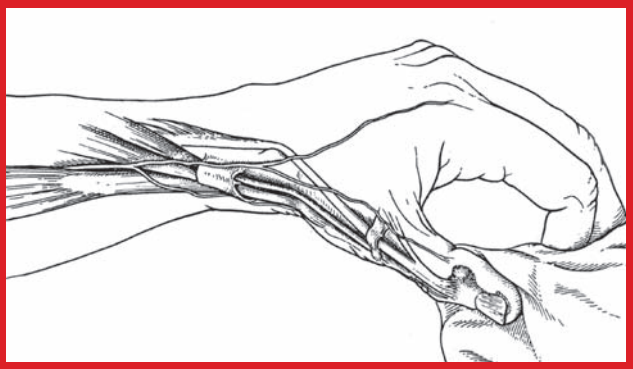
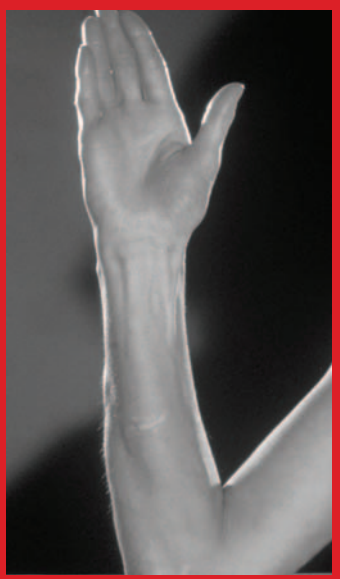
Restoration of Function in Upper Limb Paralyses and Muscular Defects





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Introduction

Paralysis is defined in Dorland's Medical Dictionary[1] as the loss of motor function in a part due to lesions of the neural or muscular mechanism, and also by analogy impairment of sensory function. Such definition, applied to the upper limb paralyses includes a large part of upper extremity pathology. The subject of this book is less ambitions, it intends to include only some nervous and muscular lesions that can be restored by a surgical treatment.

Some of these lesions regress whereas others progress or stabilize. Surgical treatment can only be undertaken if the lesions are stable or only very slowly progressive.

When several functions are lost, surgical reconstructive procedures should respect *a hierarchy of functions* to be restored.

The most important functions in the upper extremity are elbow mechanics, necessary for placing the hand in space, digito-palmar grasp and lateral thumb-finger grip.

Restoration of wrist extension is a fundamental step in the treatment of extensive paralyses of the hand, as this movement provides flexion of the fingers when combined with tenodesis of the digits' flexors. Also, stabilization of the shoulder is essential because without stability at the root of the limb, the precision of movements at its distal extremity becomes problematical.

Other useful functions to be restored, when possible, are thumb opposition and intrinsic finger function. Elbow extension is important for tetraplegics.

Restoration of the volar sensibility of the hand has considerable influence in functional restoration.

Neural paralyses constitute the main cause on functional deficiencies; however, muscular and tendinous lesions, of traumatic, pathological or congenital origin are also frequent and their rehabilitation has similarities with the palliative treatment of the paralyses.

Surgical reconstructive possibilities depend on the extent of deficient functions and the number of active muscles available for tendon transfers. Also muscle transfers or transplantations, tenodeses and arthrodeses have some useful indications in reconstructive surgical treatment.

The techniques to be used are influenced by many factors, namely stabilization, muscle balance and coordination of muscles. As Sterling Bunnell wrote in his classic paper on "Tendon transfers in the hand and forearm":[2] "*As an introduction to tendon transfer, let us first consider a little of anatomy and mechanics ... Before we can grasp, the extensors of the wrist must contract or else the flexors of the fingers will pull the wrist into flexion. Before we can pinch, the long abductor of the thumb has to spring into action or else the carpometacarpal joint will flex ....*"

In this book, according to Bunnell's recommendations, each chapter on restoration of an important function is preceded by a chapter on normal and pathological mechanics.

The more extensive the paralysis, the greater the part of associated nonsurgical treatment. Passive and active mobilization and splinting have an important role in prevention of numerous complications. Joint stiffness, contractures, deformities, trick movements, pain, associated lesions of the skin, vessels and skeleton, lack of patient cooperation, all complicate treatment and considerably worsen the prognosis.

Physiotherapy becomes predominant in the treatment of tetraplegic and spastic patients. In these cases, surgical decisions are made after discussion among the involved neurologists, physiotherapists and surgeons. This collaboration is at its best in specialized treatment centers. Whereas surgical reconstructive treatment is in constant progression for extensive proximal upper limb paralyses, palliative surgery is becoming less necessary for peripheral nerve lesions, because of the better results in nerve repair.

Surgical reconstructive procedures used for restoration of functional deficiencies introduce changes in the body image of the patient. Reintegration by the central nervous system of these alterations is a major problem. Different methods used to facilitate reinsertion are described in several chapters.

I have been very fortunate in that many of my friends and experts from different countries have agreed to contribute to this book and I feel deeply indebted to them for their work.

Our special thanks go also to our publishers, Informa Healthcare and Robert Peden, for their constant help and efficient collaboration since so many years.

Raoul Tubiana

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1 Upper extremity paralysis and muscular deficits

Pierre Rondot

Introduction

Clinical features of neuromuscular disorders

Motor deficits of one or both upper extremities can be divided, according to the abnormalities of the lesions they cause, into three principal categories.

- Clinical syndrome due to muscle diseases, in which the weakness is caused by an alteration of the muscle structure itself or by a dysfunction in the neuromuscular transmission, in which case the muscle no longer responds to the neuronal command.
- Peripheral motor syndrome due to a lesion of the lower motor neuron, which frequently affects the motor neuron itself at the spinal level, or the nerve fiber transmitting the motor signal to the muscle.
- Central motor syndrome is caused by an abnormality at the level of central motor neuron or its connection to the peripheral motor neuron.

Clinical syndrome in muscle disease

Clinical syndrome is characterized by a motor deficit most frequently localized at the root of the extremity, accompanied by an amyotrophy of the territory it affects. The exception to this is Duchenne's pseudohypertrophic muscular dystrophy where the segment first affected, the leg, is characterized by hypertrophy, not due to an increase in number or volume of the muscle fibers, but due to a proliferation of connective tissue. The disorder is not accompanied by fasciculations (apart from in polymyositis cases). The muscular contraction is characterized by an irregular pattern, in particular, at the level of the biceps brachii muscle, where the contraction is stronger. The tendon reflexes are maintained and there are no sensory deficits.

Electromyography produces a rich, polyphasic pattern, increasing as effort increases, until a habitual recruitment is reached. The amplitude and duration of motor unit potentials are decreased. The motor and sensory conduction velocities are normal.

It may be necessary, in complex cases, to perform a biopsy of a muscle in order to observe the non-systematized character of the muscular lesions, the topography of which is diverse. One can find evidence of fatty proliferation during the course of the pathologic process.

Lower motor neuron syndrome

This is the result of a lesion of the lower motor neuron, usually at the level of the spine, or a lesion of the motor root, the plexus, or the supplying nerves. It distinguishes itself from the paralyses caused by abnormalities of the central motor neuron by the characteristics outlined below.

The type of paralysis is flaccid: the muscle does not contract when stimulated, which is the most common myopathic pattern, but allows for differentiation from those syndromes affecting the central motor system. Amyotrophy may be the initial presentation. In certain cases of chronic lower motor neuron syndromes, in particular in the event of a polyradiculoneuritis, there is a hypertrophy, affecting only those muscles affected by a specific neuron, or more generalized hypertrophy can occur when the lesions are more diffuse (Figure 1.1). A certain amount of hypertrophy may exist at the level of the hand, an area sometimes denervated by conditions such as syringomyelia. Initial vasomotor abnormalities, such as a cold extremity, edema, and dry skin, can be found. Tendon reflexes are abolished or diminished. The muscle is the site of fasciculations.

Examination may be helped by using a superficial tape, applying ice on the muscle surface, or stimulating the earlobe as described by André Thomas. Sensory abnormalities may coexist in the area of the paralysis. Electromyographic examination of the paralyzed area shows evidence of biphasic or triphasic potentials of the existing fibrillations. The simple pattern observed during active movement is due to abnormal accelerations of the motor units. In the case of truncal involvement, the motor or sensory conduction velocities may be varied. A muscle biopsy may show evidence of muscle fascicular atrophy.

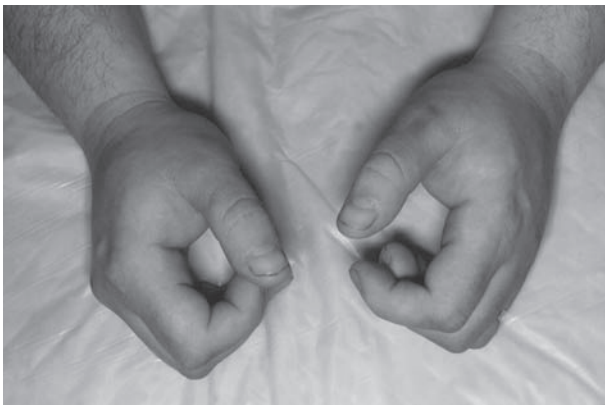

Figure 1.1

Muscular hypertrophy in a case of chronic polyradiculoneuritis, hypertrophy of the interossei muscles.

Figure 1.2

Weakness of the extensors of the third and fourth fingers in a case of muscular dystrophy.

Central motor syndrome

The area of paralysis in central motor syndrome is neither radicular nor truncular. Paralysis of central origin may present areas similar to those of peripheral involvement: paralysis of the interosseous, thenar and hypothenar muscle paralysis, flexor tendon paralysis comparable to median nerve involvement.[1] In these cases, accompanying signs may allow for clinical diagnosis. This central motor involvement is often accompanied by amyotrophy, presenting less in the initial stages as in peripheral neurogenic syndromes, apart from when certain parietal[2] or thalamic[3] lesions exist, which may confuse the diagnosis. These disorders are, however, characterized by increased reflexes in the involved areas and may occur more distally, presenting with a Babinski sign, even when the abnormality involves only the upper extremity. An electromyogram does not show any abnormalities when there are no abnormal movements.

Etiology of the upper extremity paralysis

Disorders of muscle

Muscular dystrophies

Myopathies usually start in childhood and do not usually affect the upper extremity. They may, however, commence at this level and present months or years later in the face and/or lower extremities.

Facioscapulohumeral myopathy is an autosomal dominant disorder. As the name implies, it often starts in childhood at the level of the face, which becomes inexpressive. Eventually the disorder affects the upper extremity and is characterized by amyotrophy, including motor deficits. This remains relatively moderate, as the progression may be slow, sparing certain muscle areas. One can observe very severe muscular defects but with the absence of functional repercussions. The motor weakness is relatively selective for a period of time, predominantly affecting the forearm, in particular the extensors of the third and fourth fingers, which is similar to lead polyneuritis (Figure 1.2).

The diagnosis is usually easy when there is a dominant transmission, where other family members may be affected and the face is involved. Findings on clinical examination are similar to those of myogenic syndrome, as described above. The scapuloperoneal form is a variant of this myopathy. The upper extremity may be affected first, before the muscles of the peroneal compartment, followed by facial abnormalities. In certain cases only the upper extremity is affected. The transmission is also dominant which can help the diagnosis. The therapeutic indications of surgery may be applied in severe forms when rehabilitation has been insufficient, and when retractions are apparent in muscles due to amyotrophy.

Variant scapulohumeral, Erb's, myopathy is particularly interesting because it initially affects the shoulder and then presents in the muscles of the arm and forearm. It progresses rapidly and is accompanied by amyotrophy showing signs similar to myogenic syndrome. Its inheritance is still debated and it may be a variant of facioscapulohumeral myopathy.

Duchenne disorder or pseudohypertrophic paralysis is usually present for years before it affects the upper extremities. The myopathy has an X-linked recessive pattern of inheritance, characterized by a delay in gait and then a weakness of the lower extremities with calf hypertrophy as strength decreases. The process affects the upper extremities and eventually affects the respiratory muscles causing death, usually in the second decade of life.

Becker type myopathy is more moderate and also X-linked recessive, affecting the upper extremities less rapidly and allows for a longer survival. A variant, not linked to the X-chromosome, presenting in women has been reported in North Africa, possessing a dominant inheritance, and its progression is less severe than the Duchenne disorder.

Steinert's disorder or myotonic myopathy can occur at all ages. It can be particularly severe when appearing at

birth, affecting motor function and the mind. It usually starts in adulthood. It has a dominant inheritance and can cause weakness in the upper extremities and show amyotrophy. The pattern may be misleading and is similar to the amyotrophy found in the disorder of Charcot-Marie in the upper or inferior limbs. The diagnosis is very easy: a severe contraction of the muscles of the forearm during a strong handshake, for example, provides evidence of myotonia lasting a few seconds, followed by relaxation of the fingers. Tapping a muscle group with a tendon hammer can provoke this myotonia. Following tapping, a very segmentary muscular contraction is provoked for a few seconds in that specific area. Electromyography may show evidence of this myotonia in characteristic repetitive signals of high frequency lasting for a few seconds. This can occur spontaneously or after insertion of a needle that will register myotonia with amplitude and frequencies that decrease progressively. The functional disorder caused by this myopathy is usually moderate; however, it may be accompanied by other symptoms such as genital deficiency, varying between cases, decreased intellectual capacity, and cardiac rhythm disorders requiring attention which reminds one of the severity of this disorder.

Congenital myopathies are mentioned here but they do not affect the upper extremities alone, independent of their evolution, and they are characterized by hypotonia and the diagnosis is made by the muscular biopsy.

Polymyositis

Polymyositis is difficult to diagnose. The subacute form, which occurs most frequently, may present in the upper extremity, where it usually affects the proximal parts.

Polymyositis is characterized initially by myalgia of variable intensity of no prognostic value. Erythematous eruption may occur, dermatomyositis, muscular weakness, and, sometimes, dysphagia. Fasciculations may be observed on the surface of the affected muscles, which is exceptional for a myogenic disorder. Electromyography may show evidence of these fibrillations. A rich pattern is observed where the amplitude of the potentials is not always homogeneous. By inserting a needle, an opposite pseudomyotonia may be provoked. The diagnosis is established following muscle biopsy, which can show evidence of diffuse lesions in the muscle fibers, surrounded by inflammatory polymorph infiltration. A long course of anti-inflammatory drugs is necessary and cardiac symptoms may complicate the disorder. Cure may be accompanied by retractions of the muscle fiber.

Another subacute form of the chronic dermatomyositis is the so-called myopathy of Nevin. It usually occurs after the menopause and affects the upper extremities where there is usually a progressive muscle weakness of the proximal part. The pain symptoms are much less than in the acute forms. Muscle biopsy does not show infiltration of the subacute forms but does show diffuse lesions of the muscle fiber. In this variant condition, a long period of treatment is also necessary to prevent progression of muscle weakness.

Inclusion myopathies occur in men, in the fifth decade, affecting more distal areas, accompanied by fibrillations. Electromyography patterns demonstrate the diagnostic traces of short motor units of small sizes resembling neurogenic patterns. The muscle biopsy may show evidence of typical vacuoles.

Similar inflammatory processes are observed in the muscles during inflammatory polyarthropathy, polymyalgia rheumatica with or without Horton's syndrome, Gougerot-Sjögren syndrome, systemic lupus erythematosus, sarcoidosis, and sclerodermia. In all of these cases, the muscular disorder is one that is characterized by inflammation and, therefore, does not pose any diagnostic difficulty. Reconstructive surgery is only required in those cases where there is evidence of severe muscular retraction. It is only indicated when the inflammatory processes have been completely resolved.

Muscle function may be affected in certain metabolic disorders that are relatively rare apart from glycogenosis such as the McArdle syndrome, amylo-1,4-glucosidase deficiency (Pompe syndrome). Mitochondrial encephalopathies produce muscular disturbances that are frequent but are not strong enough to produce deformity of the extremity. Diagnosis is established by a muscle biopsy.

Neuromuscular blocks

The clinical signs produced by neuromuscular blocks are usually so severe that the diagnosis of myasthenia can be easily made. Although there is a predilection for the ocular muscles, myasthenia may affect the extremities and mislead diagnosis. For example, in the upper extremity, one can observe a paralysis of the third and fourth extensors whereas extension of the index and little finger is maintained (Figure 1.3).

The diagnosis is usually easy and is characterized by weakness during repetitive effort and improves when pyridostigmine is administered. An increase of antiacetylcholine receptor antibodies is found in the majority of generalized myasthenia patients and in only 50% of patients with purely ocular myasthenia. During supramaximal repetitive stimulation at a basal frequency of 3 Hz, the electromyographic patterns will show a progressive decrement

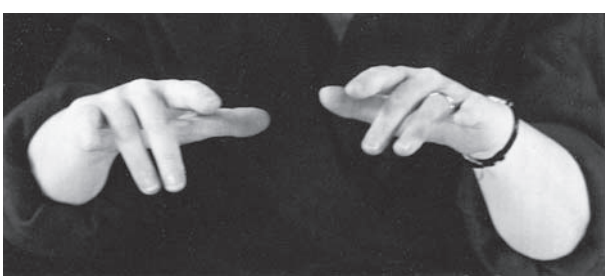

Figure 1.3

Myasthenia gravis. Paralysis of the extensors of the third and fourth fingers as occurs in lead polyneuritis.

of more than 10% in the action potentials, which can be prevented by injecting edrophonium. Eliciting the tapes reflex can also test for muscular fatigue. In the case of fixed myasthenia, which is found more frequently in the ocular muscles than in the extremities, the electromyography results are less demonstrative and the response to anticholinesterase drugs is less evident.

A recently recognized congenital myasthenia syndrome, postsynaptic, is the so-called slow-channel congenital myasthenic syndrome.[4] It is transmitted via an autosomal dominance pattern and occasionally is sporadic. The symptoms start in adolescence, with weakness and amyotrophy of the cervicoscapular, extensor digitorum, and wrist extensor muscles. Ocular disorders, respiratory disorders, and symptoms of the lower extremities are less frequent. Electromyography can show repetitive responses at 3 Hz due to an increased opening time of the ion channels of acetylcholine receptors. This syndrome is resistant to prostigmine, but reacts well to sulfate of quinine.[5]

The paraneoplastic myasthenic syndromes, snake poison intoxications, insect bites, and certain antibiotics, hydantoin myastheniform syndrome present with sufficient symptoms that there is usually no diagnostic difficulty. These disorders are not localized in the upper extremity and do not produce a deformity requiring any reconstructive treatment.

Paralysis due to disorders of the peripheral nerves

Peripheral neurogenic syndromes involving upper limbs provoke polyneuritis when the deficits are bilateral and symmetric, multineuritis when they are asymmetric appearing at different times or in different places, or mononeuritis or truncular syndrome when they are more localized. Palsy is localized more often in the extremities.

Polyneuritis

Polyneuritis is characterized by a bilateral and symmetric involvement of the limbs, predominantly the lower limbs. However, certain types are more often observed in the upper limbs, including lead, arsenic, and mercury polyneuritis. Clinically, polyneuritis of the acute porphyrinuria is similar.

Lead polyneuritis occurs most often after an oral intoxication. It is characterized by a progressive weakness of the finger extensors, after a certain time preventing the extension of the index and little fingers. No doubt this is due to the extension of these two fingers being driven by the extensor digitorum and extensor indicis muscles. The weakness is essentially distal at the beginning. It is not accompanied by sensory disturbance in contrast to most other types of polyneuritis. A few different visceral manifestations are frequent with lead intoxication, lead colic, hypertension.

Electrologic study confirms the neurogenic involvement with anomalies of nerve conduction. The toxic nature is diagnosed by the preceding circumstances and confirmed by the level of lead in the blood. After removing the toxin and treating with chelators, a significant improvement occurs and is followed by complete recovery of muscle function, as muscular retraction was prevented through the paralytic phasis.

Arsenic polyneuritis is now less common. It may be due to criminal actions but can also be as a result of accidental intoxication. Initially it provokes pain in the upper limbs, then tenacious pain in the forearms and hands where the small muscles atrophy. A precocious distal hypesthesia is observed as well as trophic disturbances. When diagnosed at the initial onset, this polyneuritis regresses without sequelae.

Mercury polyneuritis is rarer. Initially mercury attacks the cerebellum, provoking a static and cinetic cerebellar syndrome. Normally the weakness of the upper limbs is feable and is of minor importance compared with the cerebellar syndrome which enables the diagnosis, and is confirmed by the level of mercury in the blood. Treatment with a chelator causes the neuritis to decrease and allows normal life.

Acute idiopathic porphyria is a metabolic disease provoking frequent weakness of the limbs. It is often localized in the extensor muscles of the upper limbs. It is facilitated by physical exercise and certain medications. It provokes a flaccid paralysis, frequently predominant in the extensor digitorum, simulating lead paralysis. Tendon reflexes are abolished in this area, while sensitivity remains intact. There is a danger of respiratory palsy and the patient must be transferred to an emergency center. After this fit, there is no sequelae leading to a reparatory surgery.

Other types of polyneuritis, particularly ethylic neuropathies, are only mentioned because their onset is in the lower limbs and affect the upper limbs only several months or years later.

Certain hereditary neuropathies are clinically close to the previously described syndromes, particularly hereditary motor sensory neuropathy type II. However, unlike the above-mentioned syndromes, they are hereditary, beginning in childhood or at the start of adulthood, and localized most often in the lower limbs (Figure 1.4). Less often, the upper limbs are first involved (Figure 1.5), affecting small muscles of the hand, and then the forearms. The slow evolution explains the frequently observed contrast between an important wasting and the absence of a notable functional disturbance. The tendon reflexes are abolished, sensory disturbances are distal in gloves, sometimes accompanied by deep anesthesia and most often by trophic disturbances such as cyanosis, dishidrosis, and breaking of the nails. When the upper limbs are first affected, the lower limbs are involved later. This extension does not threaten the prognosis. Often a few central neurologic symptoms are associated.

The electromyographic records are neurogenic, a slowing of the conduction velocity is observed in demyelinating forms which are accompanied in the neuromuscular biopsy by an onion bulb squeezing the nerve. In axonal varieties, more often localized to the lower limbs, the amplitude of

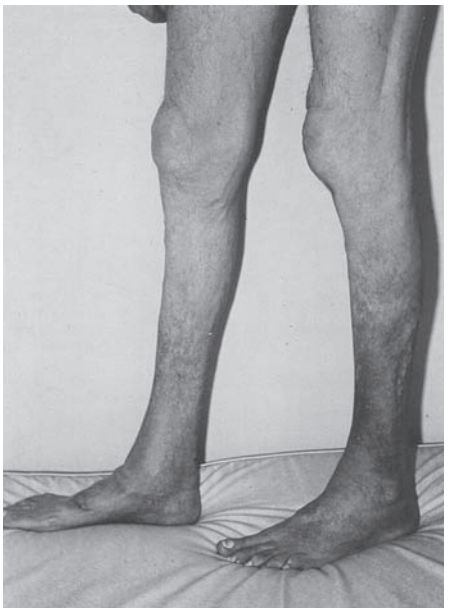

Figure 1.4
Charcot-Marie-Tooth disease. Amyotrophy of the lower limb.

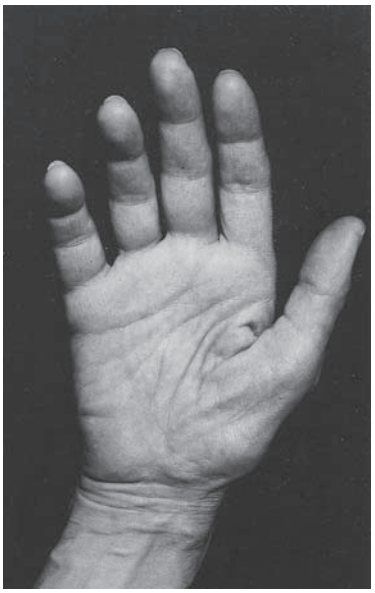

Figure 1.5
Charcot-Marie-Tooth disease. Hand amyotrophy.

the motor units is reduced without any major repercussion on the conduction velocity. The progressive evolution of muscle disturbances can provoke a retraction of muscular fibers which must be prevented by re-education. If retractions are present, it could be useful to have recourse to a reparatory surgery as the muscular function remains for a long time.

There is a scapuloperoneal neuropathy (Davidenkow), which is hereditary and dominant. It begins in childhood in the lower limbs, then spreads to the upper limbs. There are sensory disturbances associated with wasting and tendon areflexia. Electromyographic records are neurogenic, and motor and sensory conduction velocities are strongly affected.

Multineuritis

Multineuritis provokes peripheral, asymmetric, and asynchronous involvement. The upper limbs can be affected either in isolation or in combination with lower limbs. Numerous causes, such as metabolic, immunologic, inflammatory, and neoplasic, have been recognized.

Metabolic or immunological multineuritis Diabetes is one of the most frequent causes of multineuritis, beginning in the sixth decade of life, and complicating old or undiagnosed diabetes. The lower limbs or the oculomotor nerves are more often affected than the upper limbs. The multineuritis causes paralysis accompanied by sensory disturbances. Diabetic treatment yields favorable results.

Amyloid neuropathies are frequently hereditary in Portugal, although they are sometimes sporadic. They can provoke multineuropathies. The nerves are frequently thickened. The sensory changes can be early and prominent. The evolution is slowly progressive. The diagnosis of the hereditary variety is easy since it is dominant. Nerve biopsy is useful for the diagnosis of the sporadic forms. Liver graft results in a prolonged remission.

Multifocal motor neuropathies with conduction block[6] are similar to the previously described metabolic neuropathies. They appear more often in men between the ages of 30 and 50. They are characterized by a progressive and asymmetric exclusively motor deficit, often predominating in the forearms, sometimes with fasciculations, and simulating lateral amyotrophic sclerosis. The latter diagnosis is refuted by electrical stimulation, if proximal conduction blocks are discovered. Frequently they are accompanied by an increase of antiganglioside antibodies (antiGM1 ganglioside antibodies) and temporarily regress as a result of immunoglobulin, chloraminophen, or cyclophosphamide treatment which is added after a few months.

Inflammatory multineuritis Leprosy is very common in Africa, and persists in endemic areas in temperate zones. It manifests itself in one or more nerve trunks, provoking very progressive paralysis, accompanied by wasting and thermic and algesic sensory disturbances in the truncular areas involved. The tendon reflexes are conserved for a long time. The nervous hypertrophy allows diagnosis which is confirmed by nerve biopsy. It is possible to discharge the nerve sheath by surgery.

Sarcoidosis affects the peripheral nervous system less often than the central nervous system. When this complication is observed, it is usually seen as multineuritis. Diagnosis is made by nerve biopsy, the aspects of which are not far from those of leprosy.

Disseminated erythematosus lupus is more often localized to the central nervous system; nevertheless, in a few cases it attacks the peripheral system, provoking multineuritis.

Periarteritis nodosa and variants, Chug and Strauss syndrome, and Wegener's syndrome, most frequently injure the peripheral nervous system, due to a vasculitis, provoking multineuritis, sometimes localized in the upper limbs. Multineuritis in rheumatoid arthritis can be compared to previous multineuritis.

Neoplasic multineuritis Nerve trunks can be injured by neoplasic infiltration, provoking truncular and often painful involvements. After breast carcinoma, such complications can be localized to the brachial plexus. It is difficult to distinguish them from the compression due to the sclerosis of irradiated tissues which encompasses the nerves and provokes nervous symptoms.

The previous process is also observed in the course of leukemia, appearing less frequently since the purification of the hemopathies treatment. Identical complications are due to myeloma.

Dysglobulinemic neuropathies should be included with the previous. They are due to infiltrations along the nerves; however, sometimes a metabolic factor is presumed when an infiltrate is absent. Gammopathy IGH is often associated with neuropathies.

Other types of multineuritis Tomaculous neuropathies are hereditary, autosomic, and dominant. They cause paralysis which occurs after a prolonged compression of superficial nerves; in the upper limb, the ulnar nerve is most often affected. These paralyses usually regress rapidly when the compression disappears.

Serum paralyses are rare since serum has been refined. They follow the injection, beginning with great pain at the point of injection. The paralysis appears later, in the same area as the pain, precociously accompanied by amyotrophy which regresses with the paralysis after a few weeks. Occasional focal pareses persist, particularly in the serratus anterior.

Paralysis of plexus and nerve trunks

The nerve plexus, at the origin of the nerves of the upper limbs, leaves the conjugation holes and passes through the cervicoaxillary canal. The nerves can be squeezed in the outlet, resulting in paralysis of the upper limbs which are neither radicular nor truncular. The most frequent syndrome due to such compression is the cervical rib syndrome.

Nerve entrapment: cervical rib syndrome Cervical ribs are relatively common. Mayfield[7] noted their presence in 0.7% of 30 000 subjects, with a female predominance. They can provoke an entrapment of the last cervical roots (C8–D1) of the brachial plexus which are stretched on the D7 cervical rib, the normal regression of which is incomplete.

Generally the symptoms appear in the fourth decade of life. They can be more precocious or develop later. The anomaly is bilateral but the symptoms are localized on one side, most often, on the right, as a result of playing certain sports such as swimming, golf, etc. Initially the pains are localized in the supraclavicular fossa or in the territory of the ulnar nerve, increased by load carrying, and unimproved by rest. Frequently they are accompanied by paresthesia in the last two fingers. The weakness is moderate while the muscle wasting in the hand is notable (Figure 1.6), the palsy of the interossei muscles enhancing the grooves between the extensor tendons. The adductor pollicis is spared while the abductor and opponens are wasted, their innervation arising from C8 and D1 roots.[8] Thus, the hand amyotrophy of this syndrome differs from that due to ulnar trunk lesions. Vasomotor disturbances sometimes also occur such as cyanosis and cold fingers. Tendon reflexes are preserved.

An eventual compression of the subclavian artery should be looked for by detection of an abnormal subclavian pulse above the clavicle in the supraclavicular fossa and study of the Doppler effect in both arms in the rest position and after Adson's maneuvers (head rotation on the examined side with deep inspiration). Frequently nervous and vascular symptoms are dissociated. Electromyographic examination shows spontaneous fibrillations in the interossei and hypothenar muscles. The conduction velocity in the ulnar and median nerves is normal. There is a marked reduction in the sensory potentials recorded along the ulnar nerve following cutaneous stimulation in the little finger.

Simple X-ray views of the lower cervical spine reveal the bony anomaly. The cervical rib is sometimes fully formed, articulating with the transverse process of the seventh cervical vertebra. In the majority of cases, there is no rib proper but merely an enlarged transverse process of C7 that protrudes over the subjacent costotransverse joint (Figure 1.7). As a rare occurrence, the malformation may arise from C6 or from T1 and the first rib.

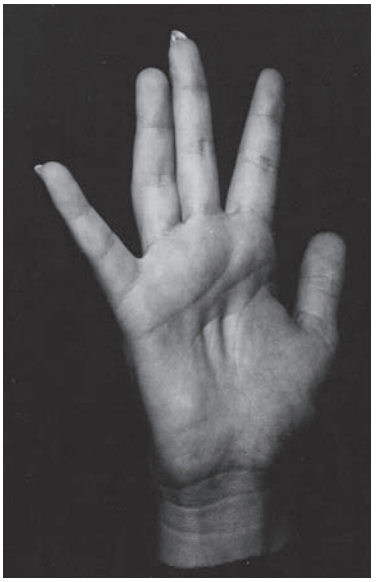

Figure 1.6

Right hand amyotrophy in the course of a cervical rib syndrome (note the involvement of the abductor and opponens pollicis).

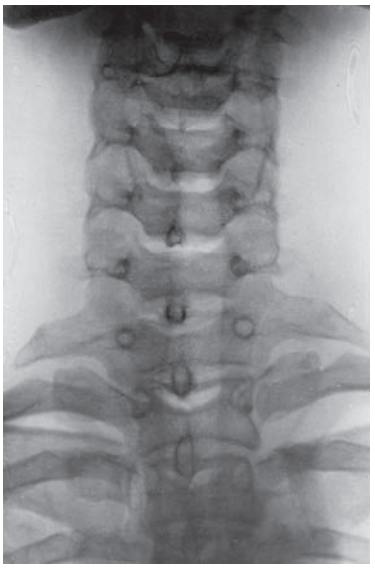

Figure 1.7
Right cervical rib, left apophysomegaly.

Surgery is advisable in the presence of severe sensory symptoms and when the functional loss is progressing. The operation aims to free the roots of the brachial plexus. It does not usually improve motor power or reduce the amyotrophy, but it will halt the progress of complications.

Peripheral entrapment: scalenus anterior syndrome In the absence of a bony malformation, the last cervical roots and the brachial plexus can be compressed during their passage through the scalene triangle. The scalenus anterior is frequently found to be hypertrophied.

The syndrome appears later in life than the cervical rib syndrome. It is more commonly found in women, associated with fatigue and with relaxation of the muscles supporting the shoulder. Amyotrophy, especially of the lateral thenar muscles and sometimes of the interossei, is occasionally seen. It is accompanied by paresthesia in the last two fingers. The neurologic complications are usually less marked than the vascular ones, which include cyanosis of fingers and Raynaud's syndrome. In the intermediate forms, there is intermittent claudication in the upper limb, and repeated efforts lead rapidly to fatigue and to paralysis and cramp-like pains in the forearm.

The vascular insufficiency is easily recognized clinically by reduced or permanently absent pulses, and confirmed by a study of the Doppler effect. Angiography is useful with the arm placed in abduction in localizing the site and mode of compression and in determining the presence of an aneurysm, usually situated in the second part of the subclavian artery.

The decision to operate requires critical scrutiny of the clinical symptoms and radiologic signs. Dividing the scalenus anterior produces significant relief in more than half of patients with severe ischemia. The results are much less spectacular in patients with intermittent compression.

Peripheral entrapment: median compression syndrome The carpal tunnel syndrome was first described by Putnam in 1880 without reporting the cause. In 1909, Ramsey Hunt[9] described 'a neuritis of the motor branch of the median nerve' which he attributed to overuse. In 1913, Marie and Foix[10] reported this syndrome as relating to the volar carpal ligament.

It is the most frequent entrapment in the upper limb leading to operation. It is due to the compression of the median nerve in the osteofibrosis carpal gutter as a result of various causes, including those of traumatic, inflammatory, arthrosic, and edematous origin. According to Tobin,[11] up to 45% of all pregnant women complain of some numbness or tingling of the fingers, with symptoms disappearing in most cases after delivery.

The first symptom is nocturnal paresthesia, numbness, or cramp-like sensation in the second and third fingers, and less often in the fourth finger. The preservation of the little finger is important for differentiating these paresthesias from multineuritis. The sensory disturbances persist during the day; with increasing compression, they may radiate proximally. The hypoesthesia is confined to the median area in the hand. Sometimes it is accompanied by a weakness of abductor and opponens pollicis muscles. Tinel's sign is positive in half of the patients, as demonstrated by tingling paresthesia in the median nerve area by percussion of the nerve proximal to its entrance into the carpal tunnel. Electromyogram shows fibrillations in the thenar muscle (opponens, abductor pollicis). The motor nerve conduction time is abnormal in more than 80% of patients. Measurements of the distal sensory latency are more accurate. Wrist radiography precisely determines the eventual bone lesion, as a fracture, rheumatoid arthritis, chondrocalcinosis, or gout.

Corticoid injection between the palmar tendons is often sufficient to improve symptoms. When this treatment is no longer effective surgery can be carried out in order to enlarge the carpal tunnel with soft tissue release.

Anterior interosseous nerve syndrome was identified by Kiloh and Nevin.[12] It manifests itself as an isolated neuritis of the major branch of the median nerve which has been injured or compressed by various means,[13] including those of tendinous origin of the deep head of the pronator teres, tendinous origin of the flexor superficialis to the long finger, accessory muscle from flexor superficialis, accessory head of the flexor pollicis longus, aberrant radial artery, and enlarged bicipital bursa encroaching on the median nerve. Paralysis attacks the pronator quadratus, the long flexors of the thumb and index and long fingers. The pronator teres remains intact. There are no sensory abnormalities. The hand of the patient has a typical appearance with characteristic disturbance of pinch, with the index finger showing extension of the distal interphalangeal joint and increased flexion of the proximal interphalangeal joint. The thumb reveals hyperextension of the interphalangeal joint and increased flexion of the metacarpophalangeal joint. The site of compression can be

precisely determined by magnetic resonance imaging (MRI), subsequently angiography can be useful. The intervention enables opening of the fibrous compression arch and prevents subsequent worsening.

Supracondyloid process syndrome is an anomaly of the supracondyloid process which is anomalously enlarged and prolonged by Struther's ligament which can compress the median nerve. Rarely motor disturbances are important. They affect the digitorum flexors, the sensory disturbances are localized at the anterior part of the forearm and the first four fingers.

Study of the reduction in conduction velocity precisely defines the site of the nerve compression.

Peripheral entrapment: radial nerve compression Compression radial syndrome can occur in the upper arm, in the region where the nerve traverses a site of physiologic narrowing. The primary site of compression lies in the region of the hiatus of the radial nerve from the lateral intermuscular septum. This compression is generally traumatic. The paralysis affects the supinator longus, and finger and carpi extensors; the triceps muscle is preserved. Sensory disturbances are observed in the posterior part of the forearm and the external part of the dorsum of the hand.

Posterior interosseous nerve syndrome is provoked by an entrapment of the posterior interosseous nerve as it enters the supinator muscles through an inverted arch called 'arcade of Frohse'. The arch is thickened by the repeated movements of certain sports (e.g. tennis) or professions involving pronator-supinator movements. The symptoms are exclusively motor, affecting the extensor digitorum communis, extensor indicis proprius, extensor digiti quinti, extensor carpi ulnaris, abductor pollicis longus, extensor pollicis longus, and extensor pollicis brevis when the syndrome presents a paralysis of all the muscles innervated by the posterior interosseous nerve. Increased pressure on the radius head provokes pain. Treatment aims to incise the compressive arcade.

Peripheral entrapment: ulnar nerve compression Ulnar nerve compression is relatively frequent in the cubital tunnel which is formed by the fascia joining the two heads of the flexor carpi ulnaris muscle, and the floor of this tunnel is formed by the medial aspect of the elbow joint and its medial collateral ligament. The Guyon's canal does not contain any tendon. This compression is observed in certain professions requiring repetitive movements, such as telephonist, locksmith, and rower.

It provokes paresthesia in the internal part of the forelimb and of the hypothenar site of the hand. Later an amyotrophy with weakness of the interossei and of the pollicis adductor muscles occurs; the latter is revealed by Froment's sign (the thumb flexed of the affected side when the two thumbs pinch a sheet of paper, trying to pull outwards) and paralysis of the flexor profundus of the fourth and little fingers. Loss of sensation is observed in the last two fingers.

The lowering of the motor conduction velocity of the ulnar nerve precisely defines the site of compression. The resection of the deep aponeurosis and transposition of the nerve under the epitrochlear muscles are followed by a rapid and complete involvement.

In Guyon's space syndrome, the ulnar nerve can be compressed in Guyon's space owing to various causes, including[14] trauma or microtrauma; vascular disorders such as periarteritis nodosa, aneurysm, angioma, and rheumatoid arthritis; and fractures of the distal radius. Paresthesia and tingling sensations cover the whole ulnar hand area, sometimes radiating to the elbow. There is weakness of the adductor pollicis and interossei muscles, while the flexor carpi ulnaris muscles is preserved.

Electrical study shows a lengthening of the distal latency of the ulnar nerve, without anomaly of the sensory potential.

Corticoid infiltrations can improve the syndrome; acute pain and early muscle atrophy may require surgery in order to treat the cause of compression.

Peripheral entrapment: suprascapular nerve lesion The suprascapular nerve which innervates the infra- and supraspinatus muscles, the abductor and external rotator of the shoulder, respectively, can be compressed either at the supraspinatus notch by the suprascapular ligament,[15] or a little lower at the spinoglenoid notch by the spinoglenoid ligament connecting the scapulae spine to the glenoid cavity.[16] This syndrome manifests itself most often after repetitive and violent movements of the shoulder.[17] Initially this compression provokes permanent or intermittent pains, in the course of certain movements (abduction, antepulsion, or external rotation of the shoulder). The weakness is partial, affecting the abduction and external rotation of the shoulder. It is associated with an amyotrophy of the back of the shoulder. No sensory disruption is observed. Electromyography shows neurogenic records of the involved muscles, the infraspinatus muscle is only involved when the spinoglenoid ligament is implicated. If re-education and corticoid infiltrations loco dolenti are inefficacious, the surgical resection of the ligaments which compress the nerve improves the pain and motor disturbances.

Radicular syndrome of the upper limb

The radicular syndrome of the upper limb can be divided into superior brachial syndrome due to a compression of the C5–C6 roots, middle cervical syndrome involving C7 root, and inferior cervical syndrome localized to the C8–D1 roots.

Motor weakness is of variable amplitude depending upon the cause. Paralysis is flaccid, sometimes accompanied by fasciculations, and tendon reflexes are diminished or abolished in the affected area. There are sensory disturbances when the sensory root is injured at the same time. The causes of these radicular syndromes are very variable, and include infection, trauma, tumor, and compression by vertebral, particularly arthrosic lesions.

Superior brachial syndrome Superior brachial syndrome is characterized by a paralysis both of the shoulder muscles,

rhomboid, teres major, clavicular head of the pectoralis major, deltoid, supra, and infraspinatus, and of the arm muscles, brachii biceps, brachialis and brachioradialis. The paralysis of the latter muscle, associated with the paralysis of the brachii biceps, indicates that this syndrome is not due to a lesion of the radialis nerve. A partial involvement is noted at the level of the coraco-brachialis muscles, brachii triceps, serratus anterior, extensor carpi radialis brevis and longue, pronator teres, and supinator muscles. The sensory disturbance is disseminated from the shoulder to the anterior face of the forelimb until the index, biceps, and styloradial reflexes are abolished or diminished.

Middle brachial syndrome Middle brachial syndrome is due to a lesion of the 7th cervical root. It is characterized by paralysis of the brachii triceps, extensor digitorum, and carpi radialis supinator muscles. The sensory disturbances are located at the external part of the posterior and anterior faces of the forelimb and at the external part of the dorsum of the hand.

Inferior brachial syndrome The involvement of the 8th cervical root is associated with that of the 1st dorsal. The little hand muscles are paralyzed with the flexor carpi radialis, palmaris longus, flexor carpi ulnaris, and the two internal heads of the flexor digitorum profundus muscles. The sensory disturbances affect the internal sides of the arm, forearm, and hand. The digitorum flexor reflex is absent.

Paralysis of nuclear origin

Certain paralyses of the upper limb are due to a lesion of the peripheral motor nucleus.

Acute anterior poliomyelitis Acute anterior poliomyelitis is the most well known type of paralysis of nuclear origin, but it is now rare as a result of vaccination. It does not selectively involve the upper limb but this can be involved either separately, or with other areas. Paralysis is accompanied with myalgias, it spreads in a few hours, rapidly reaching its maximum amplitude. The paralysis is of flaccid type, its extension is variable according the cases, sometimes simulating a truncular paralysis, predominating on certain muscular groups, while sparing others, without systematization. Tendon reflexes are absent, in the involved areas. Trophic disturbances are constant, and include edema and limb coldness without objective sensory disturbances. Spinal fluid has a cellular, lymphocytic reaction, accompanied by an increase in protein. The disturbances regress partially or totally within a few weeks. It is necessary to prevent retraction when the paralysis persists in order to avoid abnormal posture of the limbs or the rachis. If a retraction occurs, it is sometimes necessary to discuss the utility of rehabilitative surgery.

Twenty years after the onset of poliomyelitis, myalgias can sometimes reappear in the area previously involved, even breathing disorders can be observed. This postpolio syndrome requires the same precautions as the first attack.[18]

Spinal amyotrophy The chronic types of lesion of the motor neuron are less frequently observed. The best known is the chronic amyotrophy described by Hirayama (1959–1993).[19] The amyotrophy starts, most often in the young, localized to the distal part of the upper limb, spreading later to the whole upper limb, often remaining unilateral, and stopping after a few years. There is no sensory disturbance but the tendon reflexes are absent in the limb affected. As in the acute form, abnormal posture can occur; however, this is easy to prevent since the evolution is chronic. A lesion of the lower motor neuron in the young adult has been observed at the C5–D1 levels.[20]

A neurogenic variant of the scapuloperoneal amyotrophy was described which provoked a progressive paralysis of the upper limb, accompanied by a paralysis of the peroneal area.

In childhood, Werdnig–Hoffmann disease has a fatal evolution. The paralysis of the upper limbs appears after the progressive involvement of the inferior limbs. Kukelberg–Welander disease has a later age of onset. It attacks first the inferior limbs. In both cases, the lesion is localized to the spinal motor neuron.

Paralysis of peripheral and central motor neurons

Amyotrophic lateral sclerosis Amyotrophic lateral sclerosis is a redoutable disease characterized by lesions of both central and peripheral motor neurons. Frequently, it begins in the upper limbs, which are solely involved for a few months. There is a weakness of the hands, accompanied by a progressive amyotrophy of the small hand muscles, and Aran–Duchenne amyotrophy which spreads outwards to the proximal part of the limb. It is associated with trophic disturbances, coldness, and edema, without objective sensory disturbances. The previous symptoms are suggestive of peripheral neurogenic syndrome, but the tendon reflexes are brisk and are often accompanied by a Babinski sign. Evolution is progressive and untreatable. Usually it does not provoke retraction, probably because agonist and antagonist muscles are involved simultaneously.

Sometimes the motor disturbances are localized to the upper limbs for a few years, predominating in the superior part, and spreading later to the bulbar areas.[21] Tendon reflexes in the upper limbs are absent. As in the common form, there is no sensory disturbance. The amyotrophy of the hand muscle appears at the onset of the disease, as often occurs in amyotrophic lateral sclerosis.

Syringomyelia Syringomyelia is another disease that also begins in the upper limbs, but its prognosis is better. The small hand muscles progressively become amyotrophic (Figure 1.8) but, contrary to the previous disease, syringomyelia is accompanied by sensory disturbances causing pain and warm and coldness sensitivity involving segments, i.e. the upper limbs and the upper part of the trunk. Tendon reflexes are abolished in the upper limbs; however, they are present in the lower limbs where there is a Babinski sign. Surgery is considered when there is an associated basilar impression.

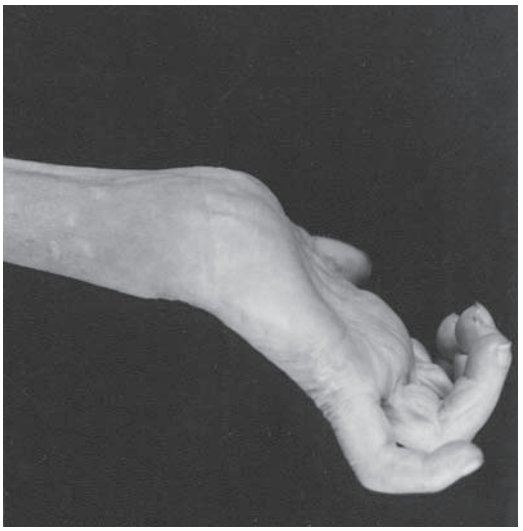

Figure 1.8
Amyotrophy of the hand due to syringomyelia.

Metabolic diseases Certain rare metabolic diseases, such as lipidosis, can provoke a peripheral deficit associated with a motor central impairment in the upper limbs. The first is related to the lesion of the spinal motor neurons, and the second to a pyramidal improvement which generally causes repercussions first on the lower limbs. This is particularly the case in a type of GM2 gangliosidosis.[22] These hereditary, recessive diseases are diagnosed by the level of seric A hexaminidase which is diminished.

Central motor deficits

Central motor defects are provoked by one or several lesions of the central nervous system localized either in the cerebral cortex or in the central motor pathways.

Clinical symptoms

The degree of weakness is variable, it is concealed or aggravated by spasticity as a result of enhancement of the stretch reflex. This causes tendon reflexes to be exaggerated, which appears as a clonus when the foot is passively and sharply flexed dorsally, causing involuntary, rhythmical flexion-extension movements of the foot. In the upper limbs, a similar phenomenon, of involuntary pronosupination movements that occur appear when the supinator reflex is elicited or involuntary flexion-extension movements of the forelimb are released by the biceps reflex, is less often observed. This syndrome, even when localized in the upper limb, can be accompanied by a Babinski sign.

Topographic diagnosis

In central paralysis, all types of distribution can be seen, including hemiplegia involving all the parts equally or most severe in the arm, generally implying the face. It is sometimes more partial, simulating a median or ulnar paralysis. In every case the tendon reflexes are enhanced and are accompanied by a Babinski sign, which is never observed during a peripheral syndrome.

When the upper limb is predominantly or exclusively affected, the cortical lesion is situated in the middle area of the precentral gyrus on the opposite side. If the lesion is situated on the pyramidal tract in the internal capsule, generally hemiplegia affects upper and lower limbs equally. Some fragmentary motor syndromes, eventually localized at the upper limbs, can result from a very small lesion of the internal capsule. Such an eventuality is very rare when the pyramidal tract is constricted under the internal capsule in the brain stem or in the spinal cord.

When the lesion is localized in the spinal cord, the paralysis is situated in the same side. Typically, the paralysis involves both the upper and lower limbs, sparing the face, when there are sensory disturbances Brown-Sequard syndrome is observed. This involves disruption of the deep sensibility on the same side as the paralysis, disruption of the superficial sensibility being situated on the other side. Such a syndrome provides definite evidence of the spinal site of the lesion. Nevertheless, this syndrome is not always complete. Sometimes a spinal lesion paralysis only occurs on the upper limb without sensory trouble. Other clinical signs enable the spinal site of the lesion to be described, a parceller involvement of the pyramidal tract is often due to an inflammatory cause.

Diagnostic problems

Motor disturbances of the upper limb are not always due to paralysis, as akinetic, hypertonic, or dystonic syndromes may also be responsible. During the akinetic syndrome, the limbs do not respond to the motor order while they can be moved by a main impulse without motor deficit. The hypertonic syndrome disturbs motility because the hypertonia of agonist and antagonist muscles hinders or restrains the movements. When an active treatment allows the hypertonia to improve, the movement becomes more easy. The dystonic syndrome, like the akinetic or hypertonic syndromes, does not provoke paralysis. The difficulty in executing movements originates in the sustained contractions of the agonist and antagonist muscles, hindering movements, as occurs in hypertonia. Most often, the dystonia is aggravated by certain postures.

Etiology

Cardiovascular causes Paralysis can have a vascular origin, such as a hemorrhage in a hypertensive patient, a vascular malformation, angioma, aneurysm, ischemia often provoked by an embolism, or vascular thrombosis due to a vascular atheroma. The paralysis can be localized in the upper limb, most often the face is also involved. When the onset is sudden, the paralysis is flaccid during the first few days or weeks. Spasticity is indicated by the tendon reflex

which becomes brisk. The paralysis regresses less or more, it is important to prevent a retraction of the flexor muscles which could necessitate surgery.

The cause of the paralysis is easy to diagnose as a result of the existing anterior vascular problems, MRI allows differentiation between ischemia and bleeding. Extra- and subdural hematomas, spontaneous or traumatic, more often provoke hemiplegia rather than monoplegia, but it is possible sometimes to observe a brachial monoplegia. Cerebral lacunes are frequent in the course of systemic hypertension and diabetes. They can provoke brachial monoplegias when they are situated either in the internal capsule or in the brain stem. Generally, they regress but tend to be recurrent and result in a pseudobulbar paralysis.

Cerebral arteritis is a particular vascular cause which develops with inflammatory symptoms and can provoke monoplegia, affecting the arterioles. The same complication can be determined by periarteritis nodosa. Meningitis, particularly tuberculous meningitis, may present with hemiplegia related to vasculitis.

Infectious or inflammatory causes Brain abscess, localized in the rolandic area, can provoke brachial monoplegia, which is usually rapidly progressive. Diagnosis is easy when it appears during the course of an acute infection. MRI shows a vasogenic edema surrounding the ring lesion of the abscess in the rolandic area. In the adult, the diagnosis can be difficult with a brain metastasis. Encephalitis, cerebral thrombophlebitis can also be accompanied by brachial monoplegia, the diagnosis is facilitated by MRI.

Multiple sclerosis is the main inflammatory cause of paralysis in the adult. Brachial monoplegia is relatively frequent and the diagnosis is difficult when it is the first symptom of the disease. It is necessary to determine any previous, forgotten symptoms, such as a retrobulbar neuritis, a transient ophthalmoplegia, or paresthesia of the limb. Usually the paralysis is accompanied by cerebellar or labyrinthine disturbances. The diagnosis can be confirmed by MRI, which demonstrates the presence of hyperactivity in the white cerebral matter; the examination of the cerebrospinal fluid shows an increase in the protein level and a moderate increase in the number of lymphocytes.

Disseminated lupus erythematosus can also provoke a monobrachial paralysis, which is sometimes accompanied by convulsions. Usually it is not an isolated determination, and cardiac and/or renal complications with a rash of butterfly distribution have preceded the cerebral localization. The cerebral sarcoidosis is usually localized in the basilar area. Pulmonary abnormalities facilitate the diagnosis.

Tumor or trauma causes All the cerebral tumors can provoke monobrachial paralysis, either at their onset, preceded by convulsions, or later when they compress the internal capsula. Evolution is slow; however, it is more rapid when the process is malignant.

Immediate paralysis may result from direct damage to the brain, with or without skull fracture. When the paralysis appears later, but precociously, an extradural hematoma should be suspected, which would be easily visible on a computed tomography (CT) scan; surgical intervention should be discussed. A subdural hematoma may be early or late, sometimes no symptoms are afferent for several weeks after the accident. The motor deficit increases progressively at the same time as the signs of raised intracranial pressure appear. The symptoms may simulate a cerebral tumor. MRI enables the correct diagnosis to be made and indicates the need for surgery.

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2 Surgical classifications of upper limb paralyses

Raoul Tubiana

Upper limb paralyses form a disparate group having multiple etiologies, extents, and prognoses. Classifications of these paralyses are numerous and have different goals. In this chapter, we emphasize classifications which may be used as a guide for surgical restoration of function. Neurologic and etiologic classifications are described in Chapter 1. Knowledge of the causes of the paralyses may have therapeutic consequences which can indicate or contraindicate surgery. Furthermore, the age of the patient is an important consideration for the surgeon. Young individuals have a greater capacity for learning, for normalization of the cortical representational topography, compared with adults.

The Paralytic Disease Committee of the International Federation of Societies for Surgery of the Hand[1] has adopted a classification combining anatomy and origin. It distinguishes lesions of the upper motor neuron, marked by spasticity of the muscles of the paralyzed part with increased tendon reflexes, from those of the lower motor neuron, marked by placidity of the paralyzed muscles (see Chapter 3). In each category there are subcategories for different origins.

Clinically, a prime distinction is made between paralyses limited to one upper extremity and those that affect other parts of the body. Paralyses in one upper limb pose essentially functional problems that are in part compensated by the other upper limb. Paralyses that extend beyond the limit of one upper extremity are of entirely another scope. They are usually secondary to central nervous system lesions such as hemiplegia, from cerebral lesions, or tetraplegia, from spinal cord lesions; they often jeopardize the survival or the autonomy of the patient.

From the evolutionary point of view, one should distinguish progressive paralyses, whether traumatic (compression) or non-traumatic (tumoral, neuromuscular diseases) from regressive paralyses, whether traumatic (neuropraxia, axonotmesis) or non-traumatic (poliomyelitis). Only stable paralyses or paralyses the sequelae of which can be foreseen will benefit from reconstructive surgery. However, certain paralyses caused by a slowly evolving nerve affliction (e.g. Charcot–Marie–Tooth disease) and some infectious paralyses that are stabilized by specific treatment (e.g. leprosy) can also benefit from surgery.

It is also necessary to take contractures, joint stiffness, or instability into consideration for the surgical treatment after upper limb paralyses. Prevention of contractures requires early and continuous physical therapy.

Classification of paralyses according to the level of lesion

Lesions responsible for upper limb paralysis can be of cerebral origin or can occur in the cervical spinal cord, brachial plexus, or peripheral nerves. An abnormal hand posture may be characteristic and establish the diagnosis of a peripheral nerve lesion as with a typical ulnar claw or as with wrist drop and flexion of the metacarpophalangeal joints, which are pathognomonic of radial nerve palsy. Paralysis tends to be more extensive with more proximal lesions. This classification, based on the level of the lesion, can be imprecise because of frequent overlapping of nerve territories, anastomoses, and individual variations.

The level of the lesion dictates, in part, the surgical indications, the method of treatment of which depends on the number of paralyzed muscles and on those muscles that will eventually be used as transfers.

Precise muscle assessment is indispensable. It is necessary to evaluate the paralyzed and non-paralyzed muscles; only muscles graded at 4 or 5 are potential transfers. However, muscle classification is too analytic. Reconstructive surgery of paralyses does not aim to reanimate the paralyzed muscles, but rather to re-establish functional movement. Therefore, in all patients with paralysis of the upper limb, it is necessary to establish a list of functional deficits and necessary functions to restore.

Paralyses of cerebral origin

Upper extremity paralyses of cerebral origin can have diverse causes, the most frequent being trauma, tumor, and, especially in older patients, vascular causes (hypertension, atherosclerosis, aneurysm).

The neurologic deficit depends on the location and extent of the cerebral lesion. These paralyses can be located in only one arm but usually extend beyond the upper extremity to involve half of the body. They are accompanied by a variable degree by spasticity, athetosis, and other extrapyramidal manifestations. Normal electrical reactions and lack of muscle atrophy suggest a cerebral nervous system lesion. Central nervous system lesions can also induce sensory and visual problems, and can result in aphasia, apraxia, and agnosis.

Spinal cord paralyses

Cervical spine paralysis can affect one, two, three, or all four limbs (tetraplegia). As paralysis in the two upper limbs is often asymmetric, each limb must be examined separately. Spinal cord paralyses have diverse etiologies and can be roughly classified into traumatic and non-traumatic spinal cord paralyses. Different classifications have been described for traumatic tetraplegia affecting the hand and wrist, and for poliomyelitis paralyses affecting the shoulder. Saha[2] described five groups of paralyzed and flail shoulders from a critical study of 103 cases.

Traumatic injuries of the cervical cord

When there is an isolated injury to the upper limb, the therapeutic problem is reduced to the quality of function of the affected limb relative to the normal side. In extensive paralysis such as tetraplegia, the problems are displaced by the survival needs imposed by the state of tetraplegia. Because of the massive interruption of neuronal transmission, the dependent functions in all or part of the cord and below the level of the lesion are affected either directly or indirectly. Motor and sensory functions are affected, as are sphincter, genito-sexual, and vasomotor functions, and skin trophicity, and, in high lesions, regulation of blood pressure and respiration (see Chapter 29 on Tetraplegia).

Early pioneers started to use classifications according to the level of the vertebral or medullary lesions, which are imprecise. In effect the disk or bone lesion does not necessarily correspond to the level of the medullary lesion.

The classification of Garrett et al[3] based on paralyzed muscular groups, and Lamb and Landry's classification[4] based on the still-active muscles and the potential transfers, marked the beginning of modern surgical classifications. The actual classifications currently in use are based on the intact lowest functional level. Of course, each level possesses the function of the levels above it.

Zancolli[5] described a classification of tetraplegia divided into four principal types, upper, middle, lower, and irregular, according to the level of the spinal cord lesion, and the function of certain key muscles such as the brachioradialis, wrist extensors, or pronator teres. In 2002, Zancolli individualized midcervical tetraplegia with strong wrist extension.

Sensory function

The area of hand skin where two-point discrimination is preserved, usually does not coincide with the corresponding metameric level in tetraplegic patients. Thus, in midcervical spinal cord lesions, cutaneous sensation may be better preserved more distally than is motor function, due to the splitting of sensory and motor fibers in different tracts. Variation in sensory and motor function between the two hands is common, making it necessary to examine each hand. Moberg[6] stated the differences between one side and the other in half of his patients. In 1978, he included the function of sensation.[7] He proposed two groups: O (oculomotor), in which vision is the only available afferent, and Cu (cutaneous sensibility), in which prehension can be performed without the help of vision.

Most authors now refer to the International Classification first elaborated in Edinburgh in 1978 and modified at the Second International Congress in Giens (France) in 1984 (Table 2.1). This classification includes the forearm and hand only, and incorporates Moberg's sensory component. Motor grouping assumes that all listed muscles are grade 4 (Medical Research Council (MRC)) or better.

Table 2.1 *International classification for surgery of the hand in tetraplegia. From reference 8*

Sensibility	*Motor characteristics*	*Description of function*
O or Cu group		
0	No muscle below elbow suitable for transfer	Flexion and supination of the elbow
1	BR	
2	ECRL	Extension of the wrist (weak or strong)
3	ECRB*	Extension of the wrist
4	PT	Extension and pronation of the wrist
5	FCR	Flexion of the wrist
6	Finger extensors	Extrinsic extension of the fingers (partial or complete)
7	Thumb extensor	Extrinsic extension of the thumb
8	Partial digital flexors	Extrinsic flexion of the fingers (weak)
9	Lacks only intrinsics	Extrinsic flexion of the fingers
	Exceptions	

*It is not possible to determine strength of ECRB without surgical exposure.

O, oculomotor; Cu, cutaneous; BR, brachioradialis; ECRB, extensor carpi radialis brevis; ECRL, extensor carpi radialis longus; FCR, flexor carpi radialis; PT, pronator teres.

This classification was intended to be a guide to the forearm and hand only, shoulder and elbow function were not included. During the Seventh International Conference in Bologna in 2001, it was decided that elbow function should also be considered but no methods of evaluation were described.[9]

Non-traumatic spinal cord paralyses

Non-traumatic spinal cord paralyses are mainly a result of tumoral causes, mostly metastases or infectious lesions. Among these, poliomyelitis lesions should be emphasized because, although they are decreasing or even disappearing in many countries, poliomyelitis has been a very common endemic disease for centuries and is still present in the tropics and subtropics, affecting growing children.

Poliomyelitis is an acute viral disease, often located in the anterior spinal horn cells. Sharrard[10] has shown the segmental incidence of cell destruction, mainly for the lower limb. Polio affecting the upper limb is not as common.

Brachial plexus paralyses

Traditionally, the classification of brachial plexus paralysis is based on the anatomic root level, and the functional deficit is deduced secondarily. Usually these paralyses are classified in the following five clinical groups: C5/C6, C5/C6/C7, C7/C8/T1, C8/T1, and C5/C6/C7/C8/T1. Functional capabilities should be paramount in the clinical evaluation.

In high lesions (C5/C6 with or without C7 involvement) the shoulder is paralyzed and unstable, and the elbow has no active flexion, but the hand is normal; the differences in clinical forms are based on the evaluation of wrist extensors.

In low lesion paralyses, in which C7 is also variably involved, the differences are based on extension or lack of extension in the fingers. The rare low paralyses (C8/T1) involve finger flexion and intrinsic function.

Adult and children brachial plexus paralyses are dealt with in Chapter 9.

Peripheral nerve paralyses

Lesions at the level of the shoulder, the proximal part of the arm, the distal part of the arm, the proximal third of the forearm, and the distal median and ulnar nerves are considered separately in this section. Several factors justify these distinctions:

- Proximal to the elbow, three or even four peripheral motor nerves can be involved in a single injury.
- With lesions distal to the elbow, the essential functions of the hand, such as opening and wrist extension are always spared. However, associated lesions of the median and ulnar nerves in the proximal third of the forearm seriously affect prehension. For this reason, the junction between the proximal and middle third of the forearm constitutes a frontier between two zones where associated peripheral nerve lesions pose different problems.
- Another factor is the result of nerve repair. Functional recuperation after suture or graft of the median and ulnar nerves distal to this frontier is usually satisfactory, although often incomplete.
- Direct repair of the motor nerves of the shoulder (axillary or suprascapular) also can give satisfactory results because of the proximity of the innervated muscles.
- This is not so at the level of the arm or elbow, where repair of the nerves supplying the hand gives more uncertain results.
- Finally, when secondary reconstruction is necessary, there are many transfers available for distal lesions that do not exist for proximal ones.

Shoulder level lesions

Coene and Narakas[11] have shown that infraclavicular plexus lesions are, in reality, peripheral nerve lesions. The axillary nerve is nearly always involved. To this lesion are added a great number of suprascapular nerve lesions. In other cases, the musculocutaneous and radial nerves are also involved and, less often, the median and ulnar nerves.

The combined mechanism of forced abduction and internal rotation of the limb seems to be the cause of these multiple peripheral nerve lesions. This mechanism in itself is not sufficient explanation. Damage is the result of the existence of anatomic elements that constitute points of fixation for the nerves; stretching or rupture can occur at these points with extreme movement. These points consist of the quadrangular space for the axillary nerve, the suprascapular notch for the suprascapular nerve, the spiral trajectory around the humerus and the posterior intermuscular septum in the distal third of the arm for the radial nerve, and the crossing of the coracobrachial muscle for the musculocutaneous nerve.

Some associations are more frequent including:

- simultaneous lesions of the axillary and suprascapular nerves, which compromise the active movement and stability of the shoulder
- simultaneous lesions of the axillary and musculocutaneous nerves, which paralyze shoulder abduction and elbow flexion
- simultaneous lesions of the axillary, musculocutaneous, and radial nerves, which, when added to the preceding deficits, cause loss of wrist extension and inability to open the digits.

Proximal half of the arm lesions

The mechanism of proximal arm lesions is usually direct trauma to the medial aspect of the arm. The median and

ulnar nerves can be injured together, causing compromise of the essential functions of elbow flexion, finger flexion, and intrinsic movements. The radial nerve is usually spared.

Distal half of the arm lesions

The risk regarding lesions of the distal part of the arm is to have a triple lesion involving the median, ulnar, and radial nerves. In this case, there is no longer any active muscle distal to the elbow, and reconstructive possibilities are very limited. However, it is possible to use active forearm supination to obtain a tenodesis effect in the hand. The situation is different when the median nerve lesion is distally located and associated with a more proximal radial and ulnar nerve lesion. This can be seen in leprosy patients. The flexors of the fingers and wrist allow transfers.[12]

In associated radial and ulnar nerve lesions, which can be seen after a wound to the posterior aspect of the arm or elbow, the goals of reconstructive surgery are restoration of wrist extension, interosseous function, and ring and little finger flexion. In simultaneous lesions of the median and radial nerves, which can be seen in lateral elbow wounds, the goal of reconstructive surgery is restoration of wrist extension, index and long finger flexion, thumb grip, and sensibility of thumb–index finger pinch.

Forearm lesion

In the forearm, three nerves, the radial, median, and ulnar, have their distal branches. The radial nerve division is the most proximal and the radial nerve itself is usually spared, and wrist and finger extension are usually preserved.

In the proximal part of the forearm The median and ulnar nerves provide their branches for the flexors of the wrist and digits. In combined median and ulnar paralyses at this level, all intrinsic muscles of the fingers and thumb are paralyzed. The thumb column is activated only by the extrinsic muscles innervated by the radial nerve. The essential functions to restore are flexion of digits and restoration of thumb–index lateral pinch.

In the distal part of the forearm In case of distal median and ulnar nerve lesions, the essential function to restore is thumb grip.

Whatever the level of the lesions *anesthesia of the palmar aspect of the hand* causes a major functional handicap.

The median and ulnar nerves jointly ensure and anteposition flexion of the thumb. The interdependence of their actions is mirrored in the frequent overlapping of their respective territories. Between the lateral thenar muscles innervated by the median nerve and the adductor pollicis (AP) innervated by the ulnar nerve, the flexor pollicis brevis (FPB) forms a transitional zone. It receives a double innervation and, in many cases, it is the ulnar territory that spills over.

For this reason it appears preferable to classify thenar muscles paralyses according to the muscles involved and not according to the nerve lesion. We have established a classification of paralyses of the thumb into two groups of paralyses, simple paralyses and extensive paralyses (see Table 2.2).

Table 2.2 *Classification of paralyses of the thumb according to their extent*

Simple paralyses
Isolated paralysis of one nerve radial, median, or ulnar
Paralyses of intrinsic muscles of the thumb (without extrinsic muscles paralyses)
paralyses of the lateral thenar muscles
paralyses of the adductor pollicis, associated with partial or predominant ulnar nerve innervation of FPB
paralyses of all the intrinsic muscles of the thumb
Paralyses of intrinsic muscles of the thumb associated with extrinsic muscles of the thumb paralyses
Conservation of active wrist extension
Without active wrist extension

FPB, flexor pollicis brevis.

Palliative treatment of paralyses in the first group, 'simple paralyses,' has the possibility of using more muscles for transfer than in the second group. In most paralyses limited to the intrinsic muscles of the thumb, restoration or reinforcement of a thumb opposition grip is possible. Only lateral thumb–index grip is indicated in the second group of paralyses (see Chapter 17 on restoration of thumb opposition). Active extension of the wrist allows activation of transfer and restoration of grip (see 'Active thumb tenodesis' in Chapter 19 on restoration of thumb–index lateral pinch).

Functional classification of extensive paralyses

Treatment of paralyses takes into account the origin and the level of lesion; however, the functional deficit is the main guide for palliative treatment of extensive paralyses. In 1988, we proposed a functional classification for extensive paralyses of the upper limb[13] based on the functional deficit.

First, we must define what we mean by extensive paralyses. These are severe paralyses that destroy an essential function of the upper limb, which are from proximal to distal:

- stabilization of the shoulder
- elbow function deficit
- extension of the wrist
- sensitive thumb–finger pinch
- digito palmar grasp.

Extensive paralyses were classified into five groups within which origins or lesion levels are different but have a clinical correlation. This classification has been clarified and completed.

Functional deficit is now classified into six groups of extensive paralyses.

Group 1

The first group involves paralyses of the muscles of the shoulder girdle. They differ according to their origin, extent, or prognosis. They may be caused by high level tetraplegia, or proximal brachial plexus injuries (C5–C6), or associated lesions of the musculocutaneous, axillary, and suprascapular nerves. Of course, the clinical similarities are relative, in particular because of lower limb involvement in tetraplegia. However, the main functional goal in the upper limb is restoration of shoulder stability. Stabilization at the root of the limb is necessary for the accuracy of movements in the more distal joints.

In tetraplegia, paralysis of the shoulder muscles indicates high spinal lesions, usually beyond surgical reconstruction.

Group 2

The second group concerns elbow function. Elbow flexion has priority.[14] An upper arm with good shoulder and hand function but with a deficit of elbow flexion is severely impaired. However, deficit of elbow extension is often considered a minor loss, as a result of the compensating action of gravity. However, in tetraplegic patients, elbow extension is of extreme importance in order to be able to get up from a bed or chair, to avoid pressure on the buttocks (pressure sores), and to reach high objects. For patients in a wheelchair, recovery of elbow extension allows transfer from bed to wheelchair and facilitates driving a manual wheelchair. Pronation of the forearm is also essential; however, it can be replaced by abduction and internal rotation of the shoulder.

Schematically, elbow function deficit can be subdivided into:

- deficit of elbow flexion and supination caused by brachial plexus lesions or musculocutaneous nerve lesion
- deficit of elbow pronation or supination caused by brachial plexus lesions or median nerve lesion for pronation and radial nerve lesion for supination
- deficit of elbow extension and pronation caused by brachial plexus lesions or by radial and median nerve lesions.

It should be remembered that:

- Elbow flexion is brought about by the biceps (musculocutaneous nerve C5–C6), brachialis (musculocutaneous and radial nerves C5–C6), brachio radialis (radial nerve C6–C7), and pronator teres (median nerve C6–C7);
- Elbow extension is effected by the triceps (radial nerve C5–C6–C7) and anconeus (radial nerve C6–C7);
- Forearm pronation is effected by the pronator teres (median nerve C6–C7) and pronator quadratus (anterior interosseous nerve (median C7));
- Forearm supination is effected by the biceps (musculocutaneous nerve C5–C6) and supinator (radial nerve C5–C6).

Group 3

The third group includes tetraplegia at the C4–C5 level, inferior brachial plexus paralysis (C7/C8/T1), and lower arm peripheral nerve lesions. Functional deficits at the level of the upper limb are similar and include deficits in forearm pronation, wrist and finger extension, finger flexion, and thumb and intrinsic movements.

The principal reconstructive aims are restoration of wrist extension and thumb pinch.

Group 4

The fourth group involves high and middle brachial plexus lesions (C5/C6/C7). These are clinically similar to associated lesions of the musculocutaneous, axillary, suprascapular, and radial nerves. The functional deficits include a loss of wrist extension, which must be restored first and of finger flexion and thumb and intrinsic movements.

Group 5

The fifth group involves tetraplegia at C6, brachial plexus paralysis at C8/T1, and associated median and ulnar lesions of the proximal forearm. Functional restoration concerns finger flexion and thumb and intrinsic movements.

Group 6

Group 6 consists of C7–C8 tetraplegia, which at the upper limb level is clinically similar to distal ulnar and median nerve lesions. The functional goals are restoration of the intrinsic function of the thumb and fingers.

Sensory deficit

Sensation in the hand is extremely important. There is an indisputable correlation between sensory problems and the

quality of motor recuperation. Two groups can be distinguished based on sensation, those with complete hand anesthesia and those with deficit in sensation in the thumb–index finger pinch.

Complete hand anesthesia

Complete anesthesia can be seen in three types of lesions: high spinal cord lesions (C4), total plexus paralysis (C5/T1), and total associated peripheral nerve lesions of the median, ulnar, and radial nerves.

Very important sensory deficits may exist in distal median-ulnar nerve lesions, with relatively little motor deficit. Some limited return of sensibility is possible, through nerve overlapping or extension of radial nerve territory.

Late median nerve graft repair, 5–6 years after injury cannot restore motor function but can restore some sensibility. Also, various palliative procedures can be used to treat sensory handicap (see Chapters 25 and 26 on sensory palsy).

Deficit of sensation in thumb–index finger pinch

Sensation of thumb–index finger pinch often remains in patients with C5 and C6 tetraplegia, inferior brachial plexus lesions (C7/C8/T1), or peripheral nerve lesions with median nerve sparing.

In a study on the results of 52 extensive and complicated paralyses of the thumb, operated on at the Cochin Hospital in Paris,[15] it is interesting to note that certain patients could use the recovered pinch in spite of pulpar anesthesia of the thumb. Thus, although the return of sensation in the thumb is a considerable advantage, its absence should not be considered a contraindication to attempting to restore pinch, especially a thumb–index lateral pinch in order to benefit from the sensibility in the radial innervated territory on the posterolateral aspect of the proximal phalanx of the index finger (see Chapter 19).

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3 Examination in upper limb nervous paralyses

René Malek and Raoul Tubiana

Paralysis is a complete loss of muscular activity in a specific area. It can result from a destruction of the muscle, or from a lesion of the muscle's nervous command, which is the only subject studied in this chapter.

There are two types of paralyses caused by nervous lesions. The first is peripheral paralysis due to a lesion of the motor neuron which forms with one or more muscular fibers the motor unit. This lesion must necessarily be located between the anterior horn of the spinal cord and the muscle. It can be at the level of the brachial plexus or located on one of the main peripheral nerves of the upper limb. Most of these nerves are at the same time motor and sensitive. Thus, there is also, using an extension of the term, a sensitive paralysis. The second type of paralysis is somewhat different. Central paralysis is due to a lesion proximal to the alpha-motor neuron, and situated in the spinal cord, brain stem, or cortex area. Specific symptoms appear, such as spasticity; however, the associated sensitivity disorders are less systematic than in the nerve trunk lesions.

Nevertheless, the investigation is always conducted in the same way, although different in its results for the two types of paralyses. The investigation must be repeated as often as necessary to appreciate the evolution, spontaneous or after treatment. Arbitrarily we only consider the secondary period after the beginning of the paralysis in this chapter.

Peripheral paralysis

Peripheral paralysis is the most frequent situation encountered by the hand surgeon who has to consider whether restoration of some functions is possible. Upper limb paralyses have a great variety of clinical presentations because of the distribution, extent, and evolution of the paralyses.

Investigation should proceed in successive stages. The investigation has to detail the precise history of the patient including age and the date of onset of the paralysis. It is often possible to identify the origin of the paralysis as traumatic (open wound, fracture, dislocation) or, less frequently, as vascular, tumoral, infectious, or rarely caused by a toxic action. It is essential to be informed about the investigations already done, and the treatment applied to date.

The examination should include two separate parts: first, the motor evaluation and, second, the sensory evaluation. Persistent signs of the paralysis can be associated with signs of recovery. The examination must be analytic to enable correct evaluation of the functional loss and to propose a surgical restoration.

Motor evaluation

When regeneration is not started or not possible, the clinical examination will show the peripheral neurogenous syndrome which is associated with:

- flaccid paralysis
- disappearence of reflexes and an atony of muscles, because the myotatic reflex has lost its afferent and efferent fibers
- atrophy of the belly of the muscle owing to non-utilization, and also to the absence of the trophic action of the nerve
- fibrillations which may be due to local metabolic excitation of the motor plate.

In the secondary period, after some months, are found associated signs of persistent paralysis and signs of regeneration:

- when the nerve is not completely divided (incomplete section) or when it was crushed or stretched
- when the nerve has been sutured after a complete division.

Initially the two possible explanations when some signs of regeneration appear are that a more extended new axonal

penetration has occurred or a larger number of motor plates are in contact with regenerated axons. Fascicular contractions can then be observed which are different from the fibrillations already mentioned, and which are considered as indicators of good prognosis. Accurate assessment of the individual muscles is essential. Muscle power is graded using the Highet scale adopted by the Medical Research Council[1] or one of its variations (Table 3.1).

These assessments are repeated periodically and the results are noted on appropriate forms, such as that shown in Figure 3.1.

Electromyography (EMG) is very helpful, but cannot be undertaken until some weeks after the start of the palsy, leaving time for the Wallerian degeneration to be completed.

- In case of complete section of the nerve EMG shows a total block, without any voluntary activity and with an unexcitability in the correspondent territory.
- When the section is not total, the EMG shows an incomplete block and a diminished conduction of the nerve.
- When regeneration is ongoing the conduction improves with a greater number of motor units and a greater amplitude of the recordings.

If a muscular biopsy were to be performed at this stage it would show a change in the muscle architecture with a new grouping of the atrophic fibers. The study of the nerve

Table 3.1 *Grading of muscle contraction*

M0	No contraction
M1	Flicker (no joint motion)
M2	Contraction with mobility, with gravity eliminated
M3	Contraction against gravity
M4	Contraction with active movement of normal amplitude against gravity and some resistance
M5	Normal power

From the Medical Research Council[1]

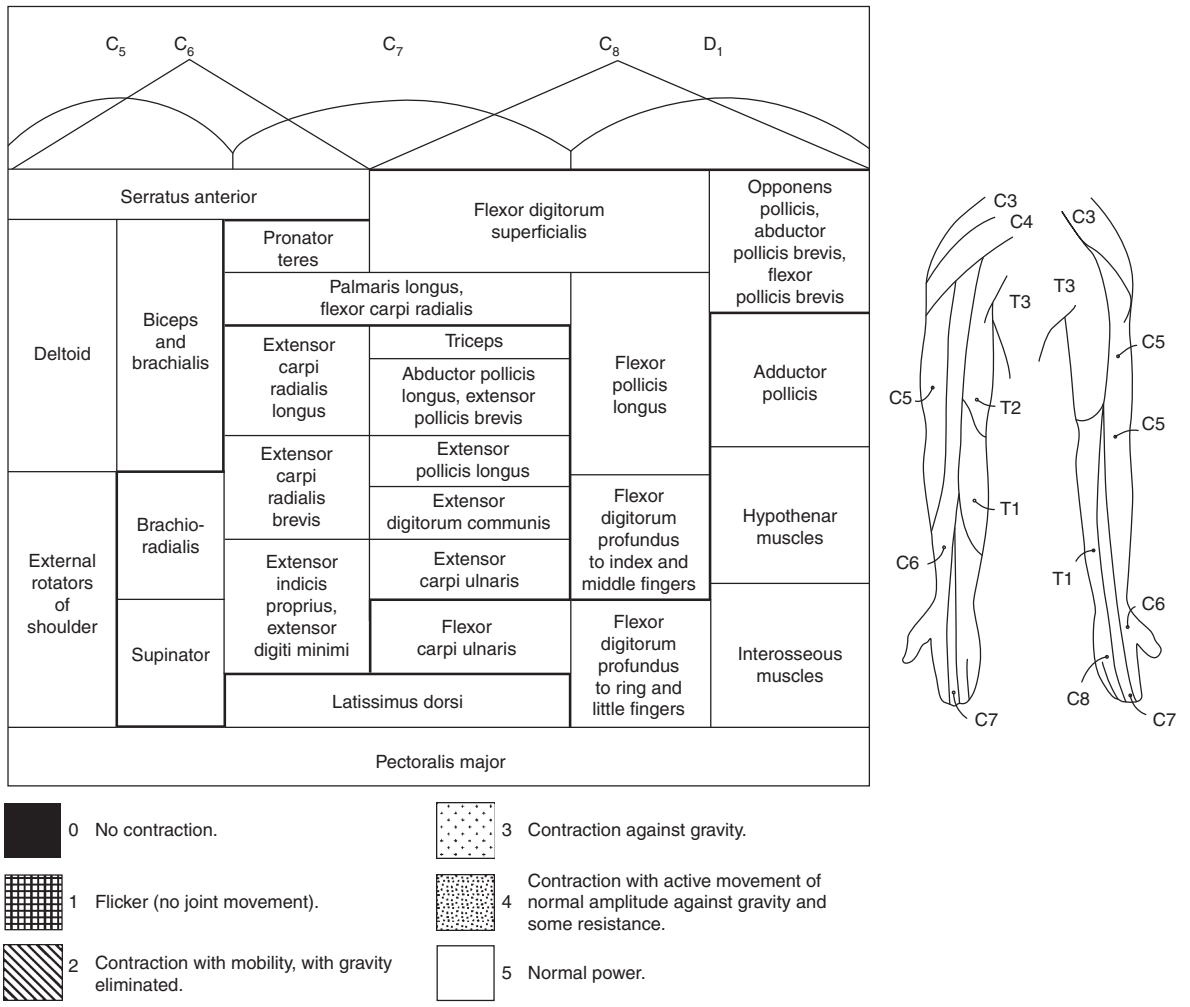

Figure 3.1

A standard form used to record motor function in the upper limb. The shaded boxes correspond to the way to shade the compartments where the muscles are indicated according to the results of the examination. From reference 2.

conduction can be useful to identify the precise level of the lesion, or to locate exactly the blockage under the lesion.

In this motor evaluation it is essential to note the passive joint mobility. Indeed, non-use frequently leads to some degree of contraction (capsula, digital ligaments, interosseous membrane, etc.). In younger patients some osseous deformities can occur due to the muscular imbalance (obsteric palsy).

At the end of the analytic motor assessment a functional evaluation is possible which depends largely on the extent and topography of the lesions. However, the functional loss is not simply the summation of the paralyzed muscles in a well defined territory. It is necessary to distinguish paralyses of nerve trunks from palsies of the brachial plexus.

Nerve trunk paralyses

Here, we simply outline some features of the palsies studied in details in other chapters.

Radial palsy (*C5, C6, C7, C8*) The radial nerve is the nerve of extension. It also provides the sensibility of a large area of the arm, forearm, and hand.

A proximal lesion (which is rare) leads to a loss of extension of the elbow and wrist, and also to a loss of active extension of the first phalanx of the digits, and extension and retroposition of the thumb. There is also a diminution of the force of supination (lack of the supinator) and of elbow flexion (lack of the brachioradialis). The hand is drooping. During prehension, approach and grip are compromised. Anesthesia is located at the lateral and inferior part of the arm, the lateral face of the elbow and the median third of the forearm, the dorsum half of the hand, the dorsal aspect of the first interosseous space and of the thumb, and, finally, the dorsum of the first phalanx of the index (Figure 3.2). The tricipital and styloradial reflexes are absent.

When the lesion is situated below the elbow, the brachioradialis and the styloradial reflex are present.

Median nerve palsy (*C6, C7, C8, D1*) A proximal lesion leads to an impairment of the flexion of the wrist (which can still be flexed by the flexor carpi ulnaris), a palsy of the flexor pollicis brevis and longus, and a paralysis of the flexion of the two distal phalanx of the index (the first being flexed by the intrinsic muscles). Pronation is completely lost, and eventually anteposition and then opposition is impaired.

Anesthesia is located in the palmar zones of the thumb, the index, the medius and the half lateral part of the fourth digit, and the corresponding area of the palm. Also, the dorsal skin of the same digits from the proximal interphalangeal (PIP) joint to the extremity are affected. From the forearm to the hand a complete lesion of the nerve gives the same palsies.

What is peculiar is the frequency of nerve compression at different levels:

- Between the two heads of the pronator teres or under the arch of the flexor digitorum sublimis.

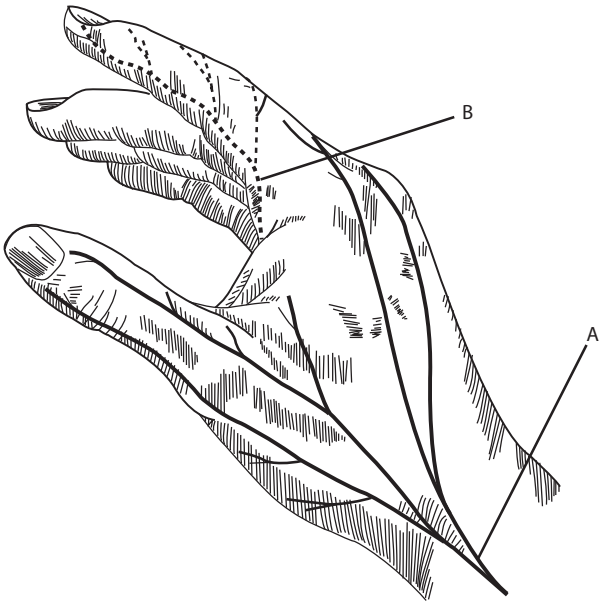

Figure 3.2

Sensitive terminal branch of the radial nerve. (A) Radial sensitive branch and (B) digital collateral nerve from the median nerve.

- More distally compression at the carpal tunnel is very frequent and generates acroparesthesia at night, hypesthesia at the hand, and in severe cases a thenar muscle paralysis. Electrodiagnosis has an essential contribution showing a decreasing of motor and sensitive conductions.

Ulnar nerve (*C8, D1*) The ulnar nerve is globally the nerve of the **grip**. A proximal lesion leads to a paralysis of the interosseous muscles, with loss of abduction and adduction of the digits, an absence of flexion of the metacarpophalangeal (MP) joints of the fourth and fifth digits, a lack of extension of the two fingers, a palsy of hypothenar muscles, a considerable lack of thumb strength, and also a radial deviation during flexion of the wrist because of the defect of the flexor carpi ulnaris. The two medial digits present the classic ulnar claw (Chapter 24). The sensory troubles are localized on the palmar and dorsal aspects of the ulnar half of the hand.

At the level of the elbow the injury of the ulnar nerve gives the same total palsy. Often the lesion is located in the groove behind the epicondylus medialis and electrology gives confirmation.

At the wrist a compression is also possible in Guyon's canal, when intrinsic muscles are involved but the extrinsic muscles are working.

More proximal nerve trunk injuries These less frequent injuries are listed below:

- Axillaris nerve (C5, C6) which determines a paralysis of the deltoideus and an anesthesia of the shoulder;

- Musculocutaneous nerve (C5, C6) with a paralysis of the biceps brachii and of the brachialis, and an anesthesia of the anterior zone of the forearm;
- Suprascapularis and subscapularis nerves (C5 and C6) can also be considered as nerves of the upper limb because of their action on the suprasupinatus and infrasupinatus muscles.

Plexus paralyses

Plexus paralyses (Figure 3.3) are studied in Chapters 9 and 21.

The motor evaluation is much more difficult in plexus paralyses because the nerves share many of the same roots. By examining the lack of action of the muscles one can determine which roots are involved with the palsy. The etiology of these paralyses is generally traumatic (and this includes obstetric palsy). The mechanism and direction of the trauma may indicate which root is more involved.

Radiographic examination (scan or magnetic resonance imaging (MRI)) is particularly helpful.

Sensory evaluation

The topography of the symptoms depends on the location of the causal lesion (Figure 3.4).

Most of the nerves of the upper limb are composite nerves. In a centripetal way, sensitive information goes from the peripheral receptors to the cell situated in the spinal ganglion. All interruptions of the nerve trunk below this ganglion interrupt the sensation. This is not only superficial sensibility in the segments, but includes information carried in the muscles or joints (proprioceptive sensibility).

The difficulty regarding a sensitive assessment lies in the fact that the examiner needs to be aware of the impairments of the patient who has to describe what he is feeling. Moreover, there is no usual instrumental method by which to obtain an objective evaluation. Also, pain can add to the complexity of sensibility assessment. The evaluation of pain warrants a particular subchapter.

In subjective cutaneous sensibility the patient himself can localize an anesthetic zone. He can also describe abnormal

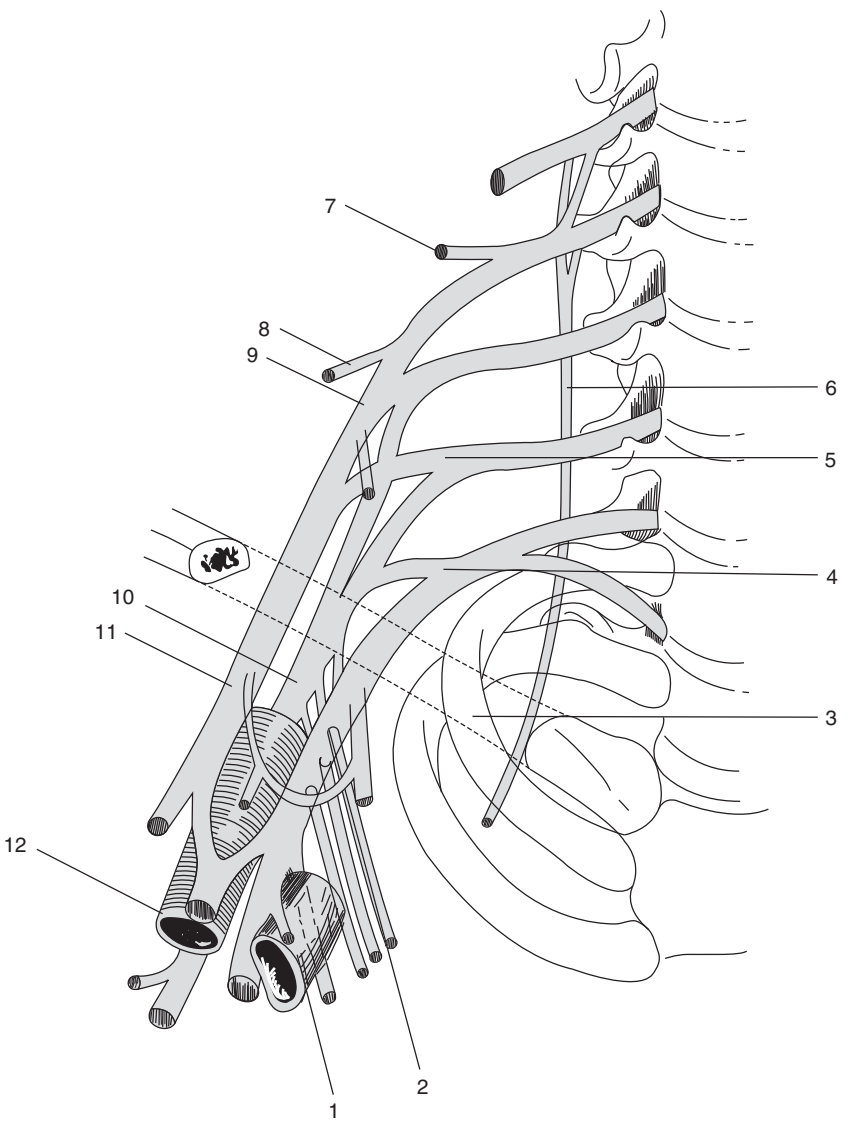

Figure 3.3

Brachial plexus. (1) axillary vein; (2) medial cord; (3) clavicle; (4) lower trunk; (5) middle trunk; (6) long thoracic nerve; (7) dorsal scapular nerve; (8) suprascapular nerve; (9) upper trunk; (10) posterior cord; (11) lateral cord; (12) axillary artery.

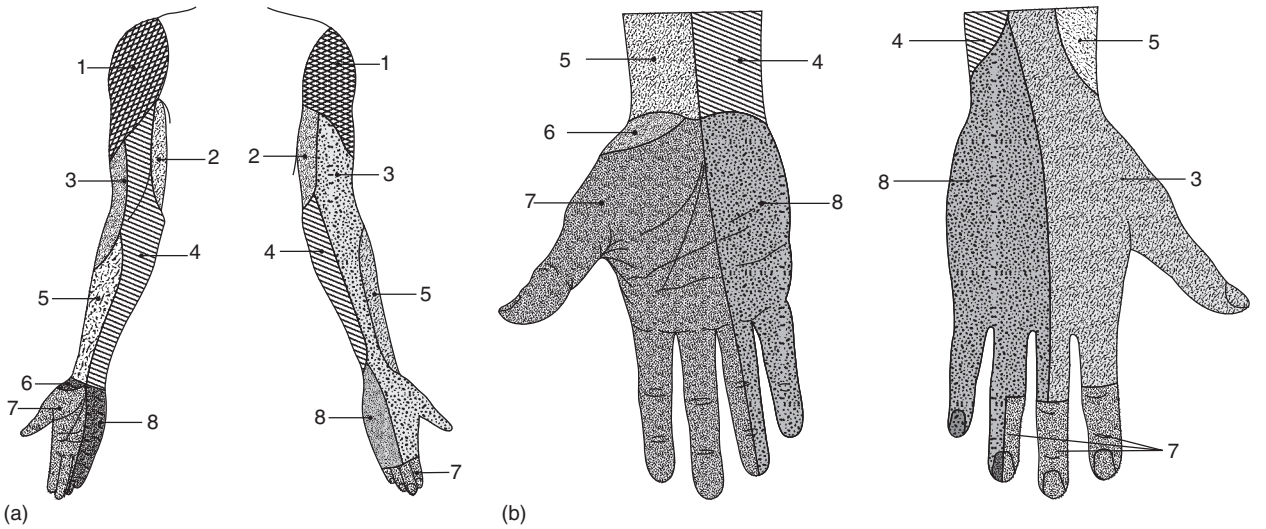

Figure 3.4

(A) Cutaneous sensory distribution of the peripheral nerves of the upper limb. (B) Volar aspect of the hand. (C) Dorsal aspect of the hand: (1) axillary nerve; (2) intercostobrachial nerve; (3) radial nerve; (4) medial cutaneous nerve of forearm and arm; (5) lateral cutaneous nerve of forearm (musculocutaneous nerve); (6) palmar cutaneous branch of radial nerve; (7) median nerve; (8) ulnar nerve. There is a considerable variation in the cutaneous areas supplied by each nerve.

sensations (paresthesia) without pain such as tingling, pins and needles, or a feeling of thick skin. These paresthesia are spontaneous or caused by a light touch. They have no physiologic explanation and occur more frequently in nerve compression.

Objective study of sensitive disruptions should be undertaken always. We investigate to determine whether the superficial information collected by the receptors is transmitted to the centers. The afferent fibers correspond to the dendron of the cell located in the spinal ganglion. In contrast to the motor fibers which lose all action after division of the nerve, if the distal part of the sensitive fibers under this section, are submitted to the Wallerian degeneration, their proximal part remains alive and can transmit nervous influxes, which can be felt by the patient if they had their origin in the periphery of the paralyzed zone. (The same mechanism explains the sensation of a ghost limb in an amputated patient.)

Skin sensibility

In skin sensibility the sense of touch is concerned. We try to determine the sensitive disruptions by the excitation of the receptors. Testing becomes more systematic and documentation more accurate if a grid superimposed on an outline of the hand is used (Figure 3.5). The receptors are situated at the level of the skin or under it (superficial sensibility), or at the level of muscles, tendons, and joints (proprioceptive sensibility).

There are four mechanical receptors in the skin (Figure 3.6). Two receptors have a low threshold (high sensibility, with rapid transmission by myelinic fibers which communicate in sequence because they respond to a stimulus by a rapid, maximal but brief discharge). They are the Meissner's corpuscles located in the glabrous skin between the epidermal papillae, which are responsible for 40% of the sensibility of the hand and are more numerous at the digit extremities; and the multilayered, encapsulated Pacinian bodies. Pacinian bodies have a lower threshold than Meissner's corpuscles and adapt themselves to the stimulus more rapidly. They represent 10–15% of the cutaneous receptors. They intervene to discriminate between superficial textures and mobility, which is an important role. Modern authors insist that over and above the spatial criteria of the study of sensibility (which corresponds to a definite topography), there is another component which is the function of time, indeed the response of the neurons changes as stimulation persists.

The two other receptors have slow adaptation (they are termed 'tonic') and continue to respond as long as the stimulus lasts. They are the Merkel disks and the Ruffini corpuscles, which are located in the epiderm, at the top of the dermal crests. They give the sensation of light pressure, and play a role in discrimination between forms, borders, and rough surfaces of the object.

The skin sensibility is not the same in all parts of the body. A way to evaluate this difference is to use the **two-point discrimination (2PD) test** described by Weber[3] who defined it as the distance between compass points necessary to feel two contacts. The variation of sensibility on the body surface is explained by the difference in the numbers of the receptors. The 2PD test was supposed to be a good objective test to evaluate by successive examinations the recovery of sensibility after treatment of a nerve lesion.[4] The 2PD test does not require any fancy equipment, a paper clip can be enough, and gives immediate information (Figure 3.7). This is the reason for its popularity.

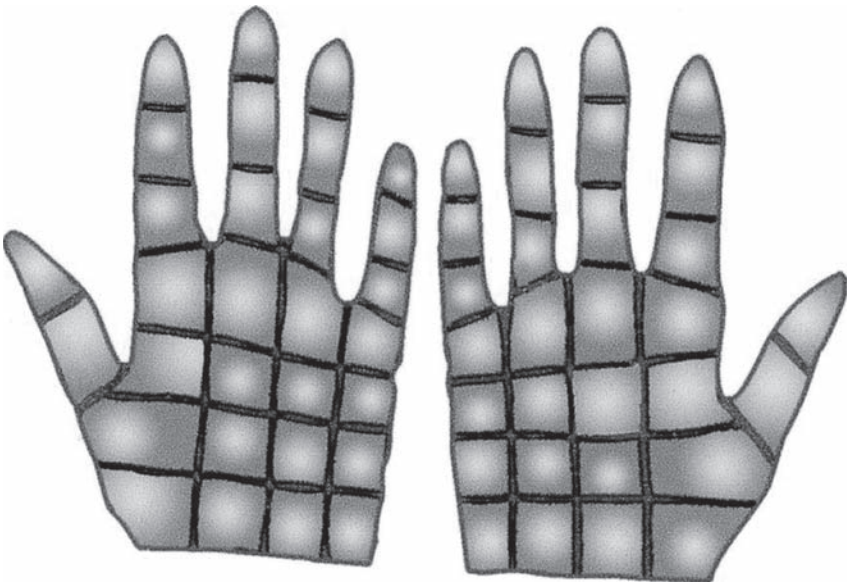

Figure 3.5

Mapping using grid makes the testing more accurate. The results of the mapping can be recorded for serial comparison.

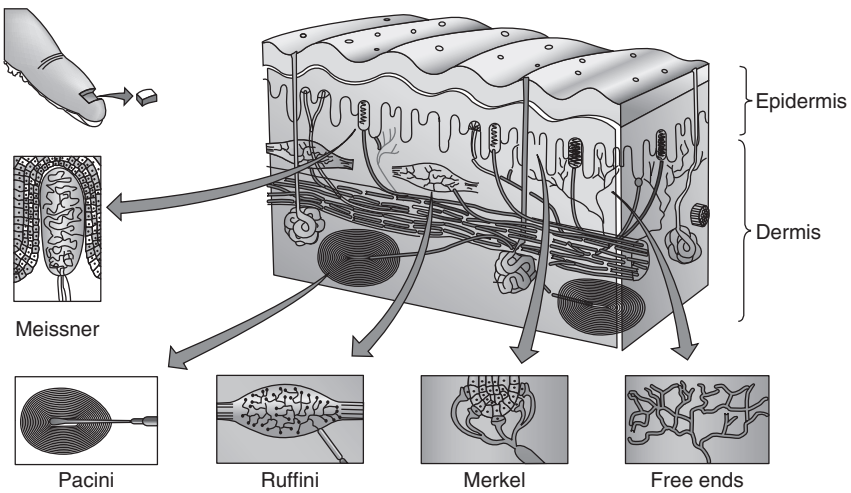

Figure 3.6

Skin receptors.

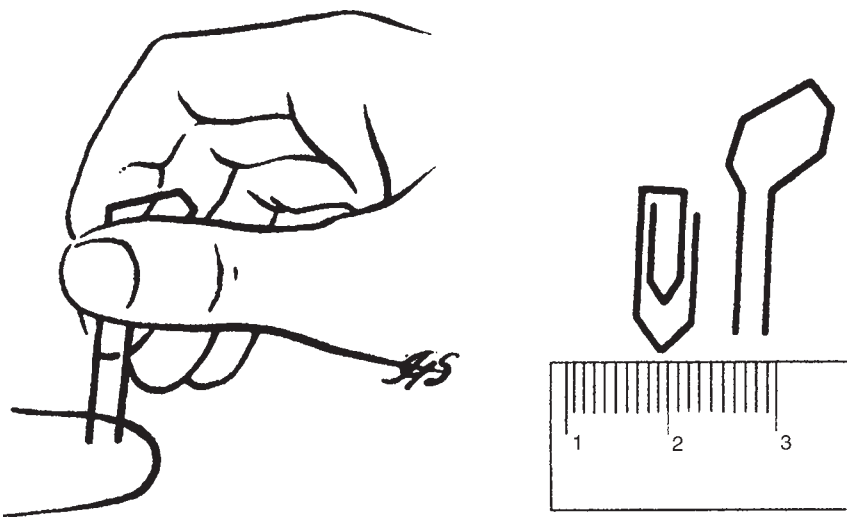

Figure 3.7

A paper clip can be used for the two-point discrimination (2PD) test.

The distance for good utilization of the hand was evaluated to be less than 5 mm in the pads. A 2PD test exceeding 15 mm represents a useless finger.[5] Comparison with the healthy side is also a good feature.

The American Society for Surgery of the Hand (ASSH)[6] adopted the classification of Gelberman et al,[7] which states that less than 6 mm is normal, 6–10 mm is fair, and 11–15 mm is poor (Figure 3.8). The static 2PD test is certainly useful

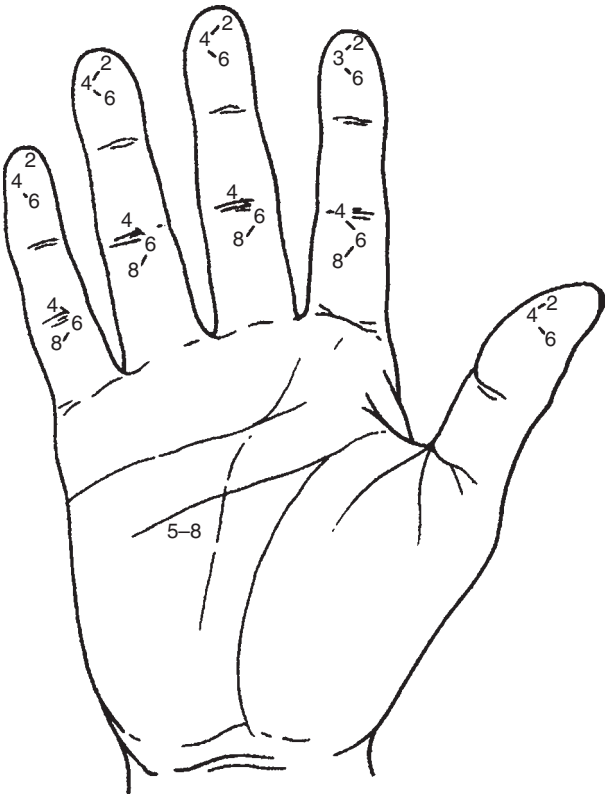

Figure 3.8

Value of discrimination in the 2PD test in millimeters in different zones of palm. The largest figure indicates the average values, the two others the minimum and maximum values. From Moberg.

in the fingertips and can be used as a quick test of normal versus abnormal sensibility. However, the 2PD is open to criticism. Moberg, himself, 40 years after having introduced it wrote a paper with the pessimistic title: 'The unsolved problem: how to test the functional value of hand sensibility.'[8]

Lundborg and Rosén[9] undertook a recent critical evaluation of the 2PD test. First, the consciousness of the patient is always necessary and the ability to communicate orally may be very variable. Moreover, Wynn Parry and Salter[10] have noted that this examination is strictly passive. Other faults are due to the examiner introducing variables in the manner of performing the test, for example, it is necessary that the two needles be pressed at the same time and with the same pressure which is not easy. The higher the pressure, the wider is the area of skin that is deformed and stimulated.[11] Blanching of the skin is used as a control, but it has been shown that blanching can occur at different forces in different areas. The test should be repeated to ensure objectivity. ASSH recommends that seven concordant results should be attained from ten tests. However, the quantity of stimuli and their repetition can also produce modifications of the results, by the mechanism of adaptation (there are two types of dendrons, some with low adaptation and, others with rapid adaptation). A further argument against the test is that the receptors which are sensitive to pressure are mostly the Merkel's disks.

Some authors have tried to improve the 2PD test. Considering that the Weber 2PD test[3] was a static evaluation, Dellon proposed a 'moving' 2PD test[12] which examines the integrity of the fast adapting fiber system, which return earlier and in greater density than the slowly adapting fibers on which constant touch depends. This test consists of moving the two needle points proximally to distally parallel to the long axis of the finger. Testing is stopped at a 2 mm distance between the points, as this represents normal discrimination at the level of the digit extremity. This test does not take into account the spreading of the receptor zones which remained innervated or which are being reinervated in the territory explored.

Other tests of skin sensibility, which can be considered as objective because the passive co-operation of the patient is sufficient, include the ninhydrin test,[13] the wringle test, described by O'Riain[14] who observed that a denervated hand placed in warm water (40°C) for 30 minutes does not wrinkle in the denervated area as would normal skin (this test can be useful for young children unable to respond to other tests), and the nerve conduction study (Chapter 4).

Finally, in the study of the assessment of skin sensibility it should be mentioned that, at the side of the receptors sensitive to pressure, there exists a sensibility to elongation given by the Merkel's disks and the Ruffini corpuscles. This sensibility is peculiar because it is directional.

The Thermic sensibility must be set in the sense of touch but has its own processes. The thermoreceptors are the free ends of dendrons which are forming a plexus between epidermis and dermis. They respond to either cold (myelinic fibers) or warm (amyelinic fibers). Their activity depends simultaneously on the initial temperature, the area of interest, and the speed of change of temperature. The sensation is accompanied by pain when the difference in temperature is important with the two limits of the so-called neutral zone (between 33 and 35°C). This is why thermic sensibility is often studied with nociception. It is not often that the examiner looks for this kind of sensibility in the case of peripheral nerve lesion, but it is important to define it in central palsy.

Proprioceptive sensibility

Proprioceptive sensibility allows the individual to know the position of their body (or parts of it) in the space and also to evaluate the degree of various forces necessary to obtain a desired movement or simply to maintain position. This sense is also obligatory to appreciate the speed, amplitude, and direction of the movement. Paralysis of a nerve trunk interrupts the methods of proprioceptive sensibility.

The receptors here are also mechanoreceptors which are localized in muscles, tendons, and joints. In the muscles the neuromuscular bundles play an essential role in muscular activity (Chapter 5). In the tendons the Golgi's organs are located (see Chapter 5). In the joints there are numerous

encapsulated receptors (Golgi's, Manzoni's, and Ruffini's corpuscles) sharing the same methods of tactile sensibility. It is not easy to determine the disruptions induced by proprioceptive information. The study of reflexes and more particularly the myotatic reflex, can give a good idea of the state of the neurovascular bundles.

The study of the joint position (with angles), and particularly at the level of the digits, with the eyes closed, appeared to be adequate to test proprioception; however, the tension of the skin around these articulations can play a part, related to the receptors in the skin. The use of a diapason to note the bony transmission is not easy. Finally, some proprioceptive data are necessary for equilibrium and posture, but exactly which they are cannot be determined in the proprioceptive confusion.

The motor and sensibility (superficial and deep) evaluation must also take into consideration the problem of **pain** because, in some cases, it can interfere with the restoration of function. Perception of pain has some analogy with the treatment of data collected by receptors under mechanical stimuli. However, it depends on specific factors.

The receptors (nociceptors) are composed of the free endings of some specialized fibers with their origins in the ganglia of the posterior roots of the spinal cord. They respond to stimuli when they reach a high intensity.

Initially, division of a nerve restricts the ability to feel pain in its territory, except if there is excitation of the proximal part of the fibers, spontaneously or after some time, when a fibrous tissue embodies them. Percussion by the examiner can also provoke pain (Tinel's sign) with a sensation of electric discharge which seems to be located in the downstream territory. This sign has a beneficial effect in localization of the end of live fibers, identifying the site of the lesion, and noting the progress of reinnervation. Nerve compression is frequently associated with pain, which can be diffuse proximally or distally.

Pain is difficult to interpret and quote. Apart from the acute pain which corresponds to tissue attrition, the other origins of persistent pain from can be local or central:

- Local, such as by the activation of the nocireceptors by nerve fibers that remain untouched (this activation can occur by humoral, chemical, or vascular transmission) or by the generation of inflammatory tissue, which is often associated with cicatrization.
- Central: we do not describe the central processes of pain but it should be remembered that at each level (spinal cord, thalamus) a bad connection can occur. It is mainly at the level of the cortex (sensitive and frontal associating areas) that pain arises and increases. Furthermore, every pain has a psychologic component, and this is very variable between patients. All these considerations explain the difficulties encountered in the measurement of pain, however, pain measurement is not essential in the context of paralyses. There is no simple instrument that can objectively record how much pain an individual experiences. The more commonly used pain measurement instruments are unidimensional scales and multidimensional scales. Unidimensional scales include:
  - Verbal rating scale (VRS) in which a list of adjectives describes different levels of pain intensity as weak, moderate, severe, or extremely severe;[15]
  - Visual scale in which the patient marks a cross on an horizontal line of 10 cm according to his evaluation;
  - Numerical rating scale (NRS) from 0 to 10 or from 0 to 100 (Figure 3.9). These tests are debatable because they are subjective.

Multidimensional scales which demand multiple responses to the patient allow a more accurate approximation of pain. The McGill pain questionnaire developed by Melzack[16] is most widely utilized. It is also possible to ask the patient to keep a diary of his pain.

In summary, there are no objective methods to measure pain and no single pain measurement instrument can provide all the required information. However, communication with the patient remains essential, as it is often possible to have an opinion based on reality and intensity of a pain described by the patient.

Functional assessment

After the analytic evaluation it is possible to determine the therapeutic indications. If one admits that the paralysis is definitive, its consequences on the life of the patient must be determined. The investigation is necessarily undertaken within a limited space of time and this adds to the difficulties to appreciate exactly what can be proposed to improve function. It is necessary to evaluate the limitations of movements and whether they limit activity. Individuals find adaptations for their movements. However, these adaptations can modify a rapid grip or an intentional use of objects. Moreover, the esthetic aspect or the utility in usual work of such an adapted movement is important to define precisely.

Before any surgical procedure is proposed, it is essential to listen to how the patient feels his disability, and also which improvement he desires. The Highet scheme with graduations from M0 to M5 for motor function and S0 to S5 for sensory function is most often applied. However, as Millesi found, this system is much too crude and does not detect functional loss such as active and passive restriction of movement.[17] Nevertheless, an objective assessment of

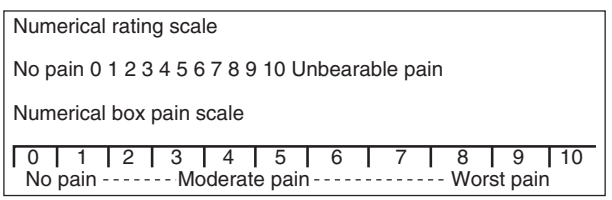

Figure 3.9

Numerical rating scales for pain management.

function is necessary, even if it is necessarily dependent on the subjective views of the patient.

Function of the hand is highly intricate, and as such involves many factors, both anatomic and psychologic. The best way to minimize the influence of all these factors is to use the patient's opposite hand, if it is normal, as a control. Although there is an approximately 5–10% difference between dominant and non-dominant hands, this is a smaller difference than that between patients.

The functional value of a hand depends to a large extent on the force the patient can apply for digito-palmar grip, pinch grip, and key grip. Grip strength is one aspect of function which is amenable to objective and repeatable measurement. However, it is often performed with unfamiliar equipment and measures only a single static reading, which may bear little relationship to the patient's actual function. A fair degree of understanding is often necessary from the patients before they can comply with the test correctly. Measuring the force is completely dependent on the patient's cooperation. A simple compromise would be to measure the force available for performing simple everyday tasks. These tasks have been chosen because they illustrate the standard grip positions of the hand (Table 3.2).

Each test takes a few seconds to perform and with a connection to a microcomputer with analog input a graphic display of force and time can be displayed on the screen and printed on paper for the patient's notes. The Moberg picking-up test assesses general sensibility and tactile gnosis. Moberg[4] established the term 'tactile gnosis' for the specific aspect of functional sensibility representing the interplay between peripheral function of the nerve and the interpretation of sensory impressions in the somatosensory cortex of the brain. Tactile gnosis is the fine sensibility of the finger pulps that permits recognition of what is being touched without the aid of sight. The advantage of the picking-up test is that it combines sensibility with motion, requiring active manipulation and recognition of the object. The patient must pick up nine objects of different sizes and shapes, one at a time, as quickly as possible, and place them in a container. He does this first with eyes open and then with eyes closed (Figure 3.10). The examiner times the patient with a stopwatch and observes the manner of prehension. In order to get an idea of the dexterity the pick-up test is performed on both hands, when possible. Video-recording is now often used for the registration of standardized tasks.

In summary, assessment of hand sensibility and motility requires a series of tests in order to address the following problems.

Which are the deficient functions? The assessment of functional loss is not simply the summation of losses of activity of the paralyzed muscles. Evaluation of the impairment must take into account not only the paralyses but also the frequent anatomic variations, joint stiffness or hyperlaxity, and the existence of pain. Reconstructive possibilities have to take into account the state of the whole limb and also the general condition, both physical and mental, of the patient.

Assessment of the other upper limb is important because it can compensate the deficient functions of the paralyzed limb.

Which movements should be restored? Reconstructive procedures cannot restore all muscle activities. As a movement is physiologically very complex and sets in action almost all the muscles of the organism (Chapter 5) a new distribution of muscles can modify a large number of elements.

Which are the functions to be restored? In extended paralyses, surgery should respect the hierarchy of functions to be restored. This means that the surgeon should select the most necessary movements to be restored for a useful functional limb. These are:

- elbow flexion and forearm prono-supination for placing the hand in the space
- the function of grasping necessitates wrist extension and digital flexion
- thumb adduction allows lateral pinch with the index finger
- if possible thumb opposition and intrinsic finger function
- shoulder stability is important because without stability at the root of the limb the precision of movements at its distal extremity is lost.

Table 3.2

Standard grip position	*Everyday task*
Lateral pinch	Turning a key
Terminal pinch grip	Gripping a pen-shaped object
Tripod grip	Gripping a pen-shaped object
Diagonal grip	Turning a door knob
Span grip	Opening the lid of a coffee jar
Extended grip	Lifting a plate

Figure 3.10

The Moberg picking-up test.

Restoration of volar sensibility has a considerable influence on functional restoration. It is the association of sensibility and motor function that makes the hand an essential organ for gathering information and perfoming tasks.

Which functions is it possible to restore? The reconstructive possibilities depend mainly on the number of available active muscles. Only muscles graded M4 or M5 are transferrable.

When planning a tendon transfer, it is not sufficient to consider the strength of the muscle to be transferred. First, the desired movement must be possible passively. However, there are many other factors to be considered such as the amplitude of the transferred tendon, and also it is necessary to anticipate the consequences of removing the motor muscle and the compensation induced.

Central paralyses

The investigation regarding central paralyses is performed in very different circumstances. The patient is followed by a neurologist who will attempt to identify the etiology. The surgeon must determine which improvements he could bring to the functional situation (only when voluntary command persists) and sometimes if there is a possible amelioration from an esthetic point of view. Indeed central lesions generate muscular imbalances which lead to angular deformities which restrict function and are also unpleasant for personal relationships. Nevertheless, the assessment of such patients requires a good knowledge of the disorders.

The examination is methodically conducted as for a peripheral paralysis and includes a motor assessment and a sensibility assessment. To facilitate these assessments it is useful to remember the organization of the voluntary movement at the level of the cortex. This movement is driven by many cortical territories including the main motor area, premotor area, and supplementary motor area.

The main motor area, in front of the Rolando sulcus, where there is a regional repartition and where the axons which reach directly the spinal cord originate, where they connect with the alpha motor neuron. These two motor neurons form the pyramidal tract which presents the motor decussation under the medulla (Figure 3.11).

The two other motor areas are localized in the adjacent zone of the prefrontal lobe and are in permanent intercellular connection with the area previously quoted. They are the premotor area and the supplementary motor area. Their neurons are connected with the motor neuron (final common way) and by many interneurons between them: the nuclei located at the base of the brain and the cerebellum. These areas appear to be responsible for anticipation, coordination, and establishment of a program for the movement. Moreover, they are connected at each level of the nervous system with sensitive cells.

It is important to bear in mind the coexistence of two pathways: the pyramidal pathway which acts on alpha motor neurons and on the motor neuron bundles, and the extrapyramidal pathway which is usually a moderating system.

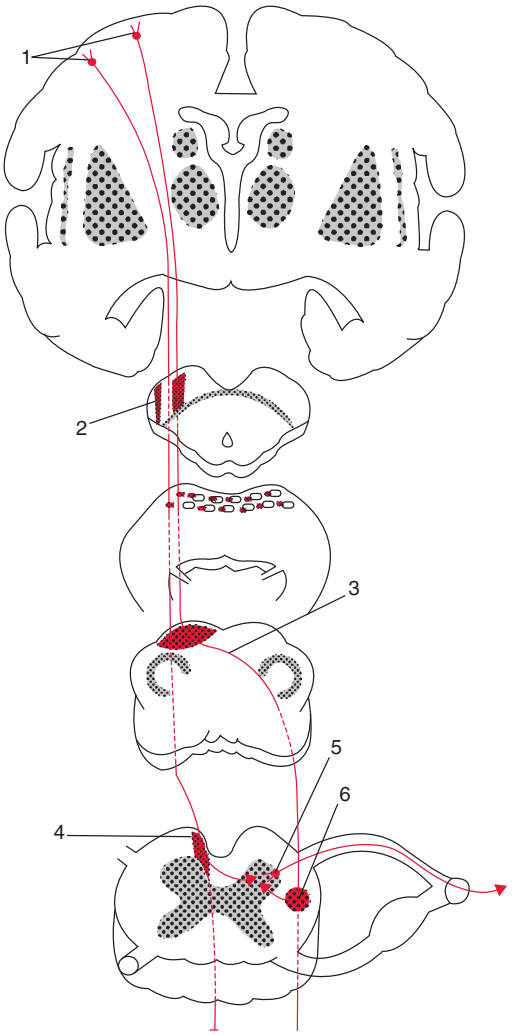

Figure 3.11

The pyramidal motor way: 1, cells of the motor area; 2, tracts in the pons; 3, crossing of the main tract in the bulb; 4, direct tract; 5, motor celle in the anterior horn; 6, crossing. By permission from reference 18.

Only the **pyramidal syndrome** is of interest to the surgeon. It combines motor and reflex disorders. Because of the extent of the causal lesion it is rarely 'pure'. Injury of the motor pathway generates a hemiplegia when unilateral, and a tetraplegia or a paraplegia when it is bilateral. Long after the initial episode is localized, often at the level of the extensors digitorum and the triceps brachialis, the motor deficit can be demonstrated by the assessment of extended arms.

Spastic hypertonia is the main characteristic of this syndrome when imposed movements generate a resistance which increases with the degree of stretch. This resistance can stop suddenly. Spasticity is due to the suppression of the modulating effect of the extrapyramidal system which presents itself associated injury. Reflexes are quick, polykinetic, and diffused.

The Babinski's sign is pathognomonic of the extrapyramidal syndrome: a light scratch of the plantar skin induces a dorsiflexion of the great toe instead of a normal flexion. Its mechanism is unknown.

Finally, syncineses are often observed with movement in the paralyzed area when moving other territories.

The details of the surgical procedures in these central palsies are studied in Chapter 30. Improvement can only be anticipated when there is still voluntary activity of some muscles, then transfers or use of tenodesis action can be considered. Angular deformities can impede the remaining activity. They can be corrected and, better, prevented.

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4 Electrodiagnosis in upper extremity paralysis: usefulness and limitations

Angèle Ropert

Introduction

Electrodiagnosis helps to localize and characterize a focal lesion when conducted as an extension to the physical examination in the proper clinical context. According to Dorfman,[1] the pertinent issues in electrodiagnosis of peripheral injuries are localization of the injury, pathophysiology of the lesion, severity of the dysfunction, and the progress of reinnervation. From the literature, it is clear that electrodiagnosis is useful but also has limitations.

This chapter critically reviews electrodiagnostic techniques in the successive stages of peripheral nerve trauma from diagnosis to prognosis with or without nerve repair.

Anatomophysiologic correlations in trauma of peripheral nerves

Electrodiagnosis must always be pertinent to clinical observations. In nerve trauma, physical examination may reveal weakness, numbness, loss of reflexes, a localized provoked pain, or a Tinel's sign. Electrodiagnosis is correlated with these signs. In addition, it often aids the precise determination of the site of the causal lesion. It also helps to identify the exact consequences of trauma on nerve function. Electrophysiologic observations are directly correlated with the histologic consequences of trauma on nerve fibers with certain limitations, e.g. nerve injury damage components of a peripheral nerve. Two classifications are used to describe the consequences of trauma on neural and connective tissue components.

Seddon's classification consists of three levels of injury. The first level is neuropraxia (due to focal demyelinization) which modifies nerve conduction (conduction slowing or conduction block). The second is axonotmesis, which refers to an axonal lesion in which endoneurium and perineurium are intact. Spontaneous recovery can occur with regrowth of axonal sprouts down the endoneurial tubes. This level cannot be recognized immediately after trauma or distinguished from neuropraxia (first level) to which it can be associated or neurotmesis (third level). Diagnosis can only be made after evolution when the full extent of recovery is known. The third level is neurotmesis or complete sectioning of the nerve with loss of continuity of all connective tissue structures. This stage always needs surgical reconstruction.

Sunderland's classification is more detailed about the state of connective tissue structures surrounding the nerve. It helps to understand the prognosis with or without surgery. Sunderland's first degree corresponds to Seddon's neuropraxia with complete recovery during a short time (3–8 weeks) evolution. Sunderland's second degree corresponds to Seddon's axonotmesis. Sunderland's third degree is a subclassification of axonotmesis: loss of axonal continuity and loss of endoneurial tubes, but perineurium and endoneurium remain intact. The recovery of this lesion is variable depending upon how well the axonal sprouts cross the lesion. Surgery may be required. In the fourth degree, axonotmesis is associated with disruption of the endoneurium and perineurium. The epineurium remains intact. Regeneration can occur but in an anarchic way (disorganization of guiding connective tissue elements and intraneural scarring) with little hope of functional recovery. Sunderland's fifth degree injury corresponds to Seddon's neurotmesis: the complete severance of the nerve which requires surgical repair.

Correspondence between neuropathology and electrodiagnostic findings

Correspondence can be established between the pathology and electrophysiology of the neural element. However, electrodiagnosis cannot determine anything about the state

of the connective tissue (epineurium, perineurium and endoneurium).

In neuropraxia (Sunderland's first degree), conduction slowing, conduction block across the site, normal distal evoked responses, and no electromyographic (EMG) denervation occur. In axonotmesis (Sunderland's second, third, and fourth degrees) the same recording occurs, with no response distal to the lesion and EMG denervation potentials. In neurotmesis (Sunderland's fifth degree), no response distal to the lesion and denervation potentials occur. Thus, in the first hours or days after the trauma, electrodiagnosis is not efficient. An aspect of conduction block means a real conduction block, reflecting a pure demyelinative lesion susceptible to spontaneous complete recovery or a localized conduction failure in axonotmesis or neurotmesis which will be followed by Wallerian degeneration.

One month after trauma seems to be an appropriate time for the initial recording because total or partial Wallerian degeneration will have occurred in axonotmesis or neurotmesis. So, differentiation between neuropraxia (with a conduction block) and axonotmesis or neurotmesis can be established.

Two points must be emphasized. First, sometimes, in the same nerve injury, neuropraxia can be associated with a degree of axonotmesis. Conduction block is associated with low distal responses. Second, neurotmesis cannot be distinguished from axonotmesis by electrodiagnosis. Regeneration time must be waited, which is possible with limits in axonotmesis but not in neurotmesis. The pertinent date of the electrophysiologic recording depends on distance between the injury site and localization of possible recording. Generally, a period of not less than 3 months is required.

Electrodiagnostic techniques

The aim of this chapter is not to detail the techniques but rather to indicate their utility in nerve trauma exploration.

Study of motor fibers

Motor conduction velocity

Motor conduction velocity (MCV) needs a target (muscle) to be recorded and at least two points of stimulation (distal and proximal). The MCV is calculated between the two points. Conduction velocities can be obtained from more than two points from many nerves (e.g. at wrist, forearm, arm, and between susclavicular area and axilla for ulnar nerve). This segmentation of motor nerve study is useful to localize a lesion. Some motor nerves or segments are difficult to study. This problem is discussed in the localization of nerve injury chapter.

What is the main contribution of MCV study in nerve trauma evaluation? A localized decrease in MCV or a conduction block means a focal demyelinization in nerve entrapment or trauma. The applications and limits of the technique are discussed below.

Compound motor action potential

Compound motor action potential (CMAP) is measured by the amplitude of the distal potential obtained in the MCV study. The amplitude of CMAP (e.g. thenarian potential for median nerve or hypothenarian potential for ulnar nerve) is directly related to the number and size of muscle fibers in a muscle and indirectly to the number of motor units in the muscle. The amplitude of a CMAP is a rough estimate of the number of motor units.

CMAP is a useful tool to quantify the axonal consequences of a nerve trauma. Its significance is different in acute and chronic nerve lesions. This problem is discussed in the chapter about nerve lesion quantification.

Electromyography

The functional unit of the motor peripheral system is the motor unit. One motor unit[2] is composed of one motor neuron (all cell body and axon) related to a variable number of muscle fibers. So muscle fibers are the target of motor neurons. Their functional state is correlated with strength. All kinds of injuries to motor neurons have consequences on motor function.

EMG is the recording with a needle electrode of the electrical activity of motor units in a muscle. The electrode (concentric needle) records the electrical activity derived from the action potentials of the muscle fibers that are firing singly or in groups near to it. This activity is named motor unit potentials (MUPs). This electrical activity induces the contraction of muscle fibers. Thus, electromyography is a useful tool to study the extent of nerve trauma. Spontaneous and voluntary activity are recorded.

Some prominent facts can be reported about spontaneous activity and voluntary MUPs.

Spontaneous activity (fibrillation, positive sharp waves, and complex repetitive discharges) means the denervation of muscle (in the nerve trauma context). It is recorded in axonotmesis or neurotmesis, but not in neuropraxia. It appears with Wallerian degeneration with a delay depending on the distance between the site of injury on the nerve and the muscle recorded. The delay varies from 1 week (thenar muscles in median section at wrist) to 1 month (hand muscles in severe brachial plexus lesion). It lasts as long as the denervation, disappearing progressively when reinnervation occurs, in correlation with the increase in number of voluntary MUPs.

In case of prolonged denervation, after months of evolution, the absence of fibrillation in a muscle with no voluntary MUPs is an indicator of poor prognosis. It means that complete degeneration of the muscle fibers has occurred with no hope of regeneration. Other spontaneous activity potentials, such as fasciculations, are less useful in traumatology.

Motor unit potentials

In traumatology the following aspects are of interest.

After nerve trauma leading to partial motor axonal loss, the appearance of the MUPs changes with their firing pattern (recruitment) during muscle contraction. These pathologic MUPs are called denervation potentials (polyphasic with a long duration).

Early reinnervated muscle fibers by a growing motor neuron have a specific appearance, they are small, very polyphasic, unstable, and have potentials termed 'nascent potentials'. They indicate direct reinnervation by a motor neuron.

Nascent potentials are different from collateral reinnervation potentials. A spared motor neuron can innervate the contiguous territory of a dead one giving the histologic appearance of 'grouping' which means a larger territory being innervated by a single motor neuron. The electromyographic aspect of this phenomenon is a large potential with a long duration. Thus, it is important to diffentiate these aspects when following a nerve trauma evolution.

Discussion of the goals of electrodiagnosis

Location of nerve injury

Identification of injury localization is easy

When the electrodiagnosis medicine consultant finds a clear conduction block or a decreased conduction velocity localized on the nerve, sometimes the MCV is normal all along the injured nerve, but the sensory conduction velocity is decreased at the site of the injury. This is possible in some early recordings because thick sensory myelinated fibers are more susceptible to entrapment than motor fibers. For example, in a patient complaining of fifth finger hypesthesia with very mild or no weakness in the interosseous muscles and the abductor of the fifth finger, electrodiagnosis can show a decreased sensory conduction velocity at the elbow, despite the fact that the MCV is still normal.

In proximal (above clavicle or radicular) lesions, because direct conduction velocity decrease cannot be recorded (as it is impossible to stimulate on two successive points), motor and conduction velocity studies are useful to exclude a more distal nerve lesion (ulnar nerve versus C8/T1 or lower trunk lesion). EMG is a useful tool in proximal locations, showing denervation in a specific anatomic territory.

Partial or incident nerve lesions occur more often in nerve entrapment than in nerve injury. In some cases, in spite of clinical complaint (usually symptoms of pain with or without paresthesias), motor and sensory conduction velocity recordings are normal. Most nerve fibers are probably still conducting the nerve impulses normally. The electrodiagnosis consultant may use other methods such as the 'inching' motor or sensory methods which try to localize a lesion by progressing centimeter by centimeter along the nerve. This is a sensitive method but has an increased risk of errors compared with classical methods. Conduction velocity comparison with the contralateral upper limb is also useful. Careful investigation of EMG abnormalities characteristic of denervation may be useful (e.g. mild fibrillation and polyphasic potentials with a decreased recruitment only in flexor carpi ulnaris and abductor fifth finger in incomplete ulnar nerve lesion at elbow). Sometimes, when the electrodiagnosis is uncertain, the consultant can propose a delay (1 or 2 months) in order to make a comparative recording.

The most frequent nerve entrapments are located in superficial, easy to record places (e.g. median nerve at wrist, ulnar nerve at elbow). In some less frequent entrapments (median nerve in forearm or ulnar nerve in arm) or other lesions (nerve tumor in forearm or arm) the conduction velocity method is less efficient. Search for EMG denervation in specific muscles is probably efficient. When the nerve injury location is uncertain, the electrodiagnosis consultant should be able to discuss with the surgeon, explain which locations are ruled out, propose a second recording with a delay, and use other methods (e.g. inching with needles). The clinical and electrophysiologic approach can be completed by imagery, specifically when a tumor is suspected.

Nerve lesion quantification and prognosis

After nerve injury, the clinician should be able, after localizing, to determine whether spontaneous recovery is to be expected or surgery is to be planned. Electrodiagnosis has usefulness and limits. Its usefulness includes determination of conduction block, conduction slowing, CMAP, nerve action potential (NAP), and EMG. Acute nerve injury and entrapments must be differentiated.

Acute nerve injury

During the first month a complete conduction block (with no MUPs recorded in muscles) at the injury site which indicates neuropraxia (myelinic lesion leading to spontaneous recovery) cannot be distinguished from a conduction failure (at axonotmesis or neurotmesis site). However, a partial conduction block (with MUPs recorded in muscles) even when initially severe allows a complete recovery to be awaited, which can occur after 4–8 weeks (the time required for remyelinization). A sequential recording (with 2 or 3 week steps) can help to follow the evolution.

The end of the first month is strategic, as by this time, Wallerian degeneration had time to occur in all nerve fibers involved in axonotmesis or neurotmesis. This correlates well with the extension of fibrillation and poor or no MUPs recording. If a clear clinical recovery has occurred,

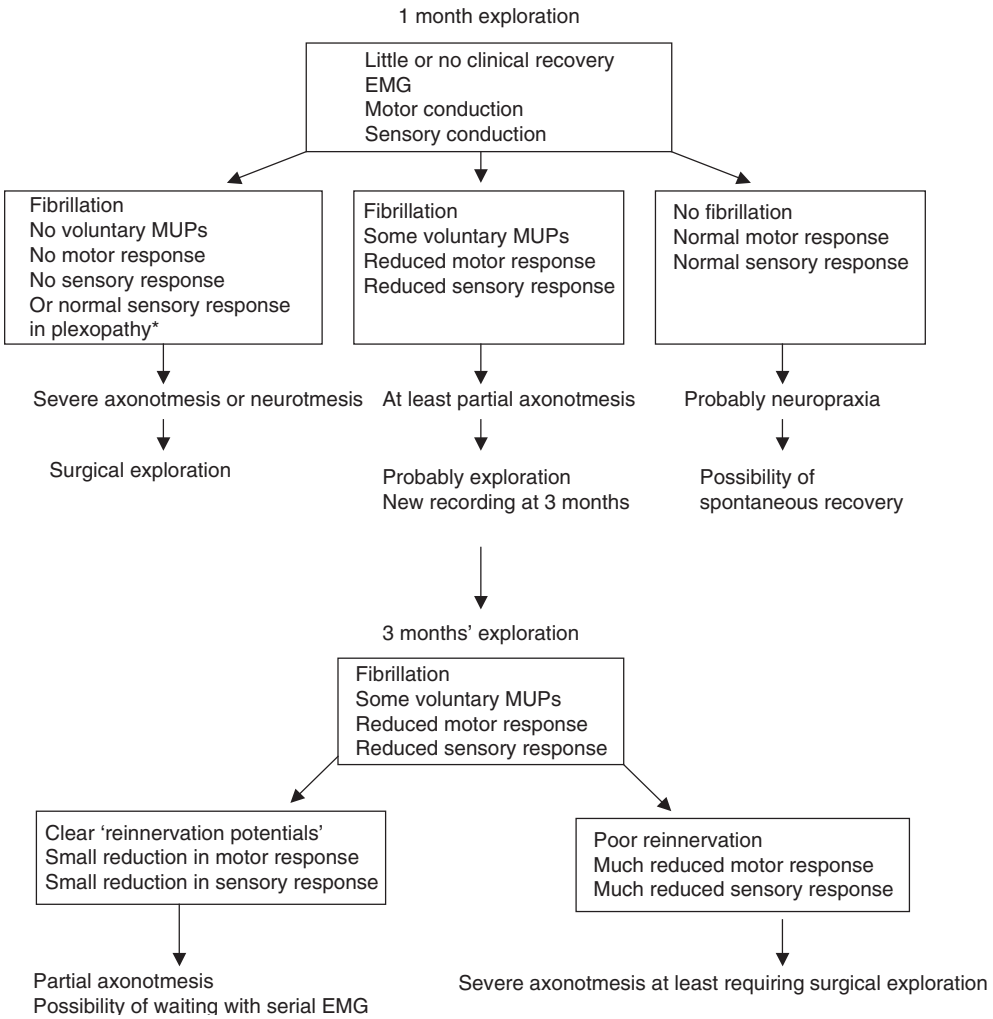

Figure 4.1

Algorithmic approach to assessment and management of traumatic peripheral nerve injuries. *In plexopathy normal sensory response with no motor response mean root avulsion.

with normal CMAP and NAP responses, no fibrillation, and MUPs recording in muscles, neuropraxia is the main pathophysiologic mechanism and spontaneous complete recovery is to be expected. If no or little clinical recovery is seen, electrodiagnosis is a really useful tool, mainly in distinguishing persistent neuropraxia (potentially reversible) from axonotmesis or neurotmesis (see the algorithm, Figure 4.1).

Sometimes, in case of conduction block (neuropraxia), some fibrillation is recorded in muscles. This indicates a certain degree of axonotmesis with Wallerian degeneration. When a poor clinical recovery occurs, with Wallerian degeneration signs (reduction of CMAP and NAP, fibrillation, poor EMG) it is impossible for the electromyographer to distinguish between axonotmesis and partial neurotmesis. Direct nerve visualization (surgery) is pertinent in severe plexic lesions. The particular association of no motor response (no CMAP, no MUPs, and fibrillation) and normal sensory responses (NAPs) should be described. This indicates a lesion between the dorsal root ganglion (DRG) and medulla, and therefore root avulsion. In more distal lesions, the amplitudes of the NAPs are reduced (Wallerian degeneration distal from dorsal root ganglia).

Nerve entrapment

In nerve entrapment (chronic nerve compression), the physiopathology always associates localized demyelinization and distal Wallerian degeneration. So, CMAP, NAP, and EMG demonstrate the degree of axonal loss. A typical axonal loss feature is diminution of CMAP and NAP, diminution of MUP recruitment with or without fibrillation.

It should be noted that nerve entrapment is a chronic situation. Some degree of reinnervation is possible even during Wallerian degeneration. In motor nerve fibers, remodeling of motor units occurs and is termed collateral reinnervation (a spared axon reinnervates denervated muscle fibers). The involved motor units become larger. This is a

method of compensation. Sometimes muscle strength remains relatively normal and CMAP is normal. Only EMG with a needle electrode in the muscle can identify the denervation–reinnervation pattern (large MUPs with long duration).

Prognosis and evolution

The prognosis of a nerve lesion depends on the physiopathology (neuropraxia or axonal loss) and etiology (acute trauma or nerve entrapment). Thus, the history of the patient's disease should be correlated with clinical examination and electrodiagnosis. In traumatic nerve injury, the therapeutic strategy is outlined in the preceding chapter (quantification, the algorithmic approach to assessment, and management of traumatic peripheral nerve injuries).

After the therapeutic decision (with or without surgery) has been made, how should the evolution be followed? After axonotmesis or neurotmesis (with surgery) motor reinnervation can be assessed by the increasing value of the amplitudes of CMAPs and the increasing number of MUPs in muscles on sequential recordings. Sensory reinnervation is assessed by the increase of the amplitudes of NAPs. Conduction velocity is always reduced during reinnervation due to the reduced diameter of reinnervating fibers and reduction of internode length of myelin sheaths. The date of recording of the first possible reinnervation depends, of course, on the length of nerve to be grown.

In neuropraxia, in most cases, after the diagnosis (clinical and neurophysiologic) clinical follow-up is sufficient during the rapid recovery. If electrodiagnosis is repeated, the usual schema is conduction block at site injury with normal CMAPs and NAPs amplitudes, with the number of MUPs being proportional to the degree of conduction block. During the evolution (rapidly, within 6 or 8 weeks) the degree of conduction block decreases in correlation with remyelination. The number of MUPs during voluntary contraction increases (MUPs with normal aspect) and CMAP and NAP are normal.

Applications to the study of upper limb nerves

The techniques described above and the discussion of their limitations, assist the understanding of the way the electrophysiologist studies upper limb nerve trunks.

Further reading

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5 Basic notions of biomechanics

René Malek

It is very noticeable that medical students, and particularly those in orthopedics, are not familiar with physics. This does not apply to biomechanics specialists. Thus it might be useful to go over some basic principles.

Basic principles of physics

Forces

In man, forces exerted by muscle contractions follow Newton's laws of motion. These are three physical laws which relate the forces acting on a body to the motion of the body:

1. A physical body will remain at rest; or continue to move at a constant velocity, unless an unbalanced net force acts upon it.
2. The net force on a body is equal to its mass multiplied by its acceleration.
3. The third law corresponds to action and reaction when a body exerts a force on another, the second applies, on the first, a force of equal value but in the opposite direction.

Study of forces

Forces are represented by vectors, the length of which depends on the intensity. These forces can be applied at a fixed point (linked vectors) or at a mobile point (free vectors). The vectors can be contained in one unique plane. It is possible to obtain the same situation by the projection of vectors located out of the plane, and by drawing perpendicular lines from the extremities of the vector to the plane. It is always possible to have two or more forces applied at one point. With two forces, it is then possible to draw a parallelogram, and one diagonal from the common point of application. This diagonal is called the resultant of the forces, which has a value and a direction quite different to the two initial forces (Figure 5.1). Two resultants can also be studied with a new parallelogram. To construct the parallelogram about the original force, the number of possible components is infinite. The most useful resolution in force components involves the determination of rectangular components. In the force being applied on a bone, for example a phalanx, there will be one component perpendicular to the diaphysis (likely to determine a movement) and one force which will be parallel to the shaft of the bone – this force is called axial. The force applied below a joint has its value multiplied by the lever arm, which is the distance between the axis of the joint and the point where the tendon is inserted. This is called the *principle of moments*. In many cases insertions of the muscles are near the axis of rotation of the joint which is supposed to be moved. This is not a good situation if one considers the force necessary to obtain the movement. However, if the force is necessarily greater, there will be less displacement of the insertions during the movement and this is economical from an energetics point of view.

When two or more forces are applied on a bone and have an opposite direction they constitute a torque of rotation,

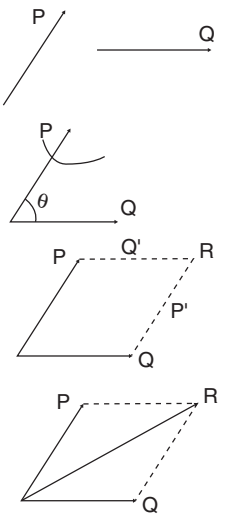

Figure 5.1

Construction of a parallelogram to determine the resultant vector R of two forces P and Q.

which can be clockwise or counterclockwise. The best example of torque in the upper limb is the scapular bone and its tilting movements (see chapter 8).

Center of mass

The center of mass of a volume is a fictitious point which allows us to assimilate a quite complex system for representations and calculations. This is useful when one considers the multiple forces exerted by different muscles on a single bone. Anthropometry can define such centers of mass, if the structures are assumed to be homogeneous.

The center of mass is used in calculating the value of a force exerted on a longitudinal piece of bone. The distance between this center and a point or an axis of rotation on a moving joint is important. The weight of a limb (or part of a limb) is another factor used to evaluate the muscular forces necessary to determine a movement (it is the main one when the movement is 'free' of contact).

In this chapter we consider the permanent activity of all the muscles of the organism, and therefore of a limb, which are classically divided into agonists and antagonists. Movement is determined by the strongest forces at any one moment. In man, the muscles applied near the rotation centers when the displacement is small are favored to limit the energy spent.

Mechanical constraint

Mechanical constraints exist in the musculoskeletal system:

1. In a joint, when the articular surfaces glide over each other there arises a *frictional force*, which is reduced by the properties of the synovial fluid (similar to the action of oil in a wheel).
2. The *pull constraint* is mostly due to the body weight, to external objects handled, and to eventual traction forces exerted on the limb. These pulling forces tend to separate the joint extremities, but they are usually balanced by the intra-articular vacuum, which is generally sufficient to compensate for the weight of the part of limb downstream. Generally, the resistance of the ligaments and the resulting muscle contraction all around the joint intervene, the latter acting in contrast with compressive forces (axial force components of the forces they apply on the bone).
3. The *compression constraint* is also created when the limb is supported by a solid structure, or when, during a movement, the object handled gives a certain resistance. These compression constraints are often surprisingly high.
4. As many mechanical forces intervene, even in the most limited action, to realize the movement wanted and to avoid any 'parasitic' or unnecessary move, it is difficult to describe them precisely.

Many couples of forces are generated.

Muscular physical activity

Each force can be examined using the parallelogram system: it can be considered as the resultant between one vector perpendicular to the main axis of the bone and another vector parallel to this axis. The axial component is, according to the movement, a parasite force, but it is important to maintain the coaptation of the extremities at the level of the joint, and can also be a force that generates a movement of the proximal bone of the joint.

The so-called antagonistic and agonistic muscles simultaneously create axial forces, which must be added. One movement is generated by the difference between the various components perpendicular to the bone, producing a resulting force. To avoid any (unwanted) parasite effect of the axial forces, other muscles are set in action, and so, step by step, it is easy to conclude that all the muscles of a limb are involved and work together. This is why we can say that there is no sector of muscle inactivity. Moreover, when muscles are bi- or pluriarticular, any change at the level of one of the joints causes repercussions in the other parts of the limb.

It is not just muscular activity that is involved in movement of a limb. Indeed, the first step in voluntary handling is called 'approach', which tends to approximate the hand to the object. The positioning and opening of the hand will also depend on the nature of the gesture to be made after the object has been gripped. Thus, during this preliminary step the entire body is involved together with the upper limb (postures).

When contact is established between the hand and the object, cutaneous information, proprioceptive stimuli, and visual observations facilitate adaptation of the entire muscular system.

Some remarks about evaluation of anatomic rotation

In biomechanical studies of a limb or part of a limb, the term *rotation* is often used. However, confusion may sometimes arise in its application, and we shall try to dispel it.

The dictionary gives the following definition:

> Rotation is a transformation of a geometric figure in such a way that all its points describe arcs of angle α around a fixed point 'O'.

The simplest rotation is of a single point in a single plane, represented in Figure 5.2.

If the point is replaced by a segment AB, segment and point of rotation determine a plane. Each point of the segment will rotate at the same angle α (Figure 5.3), and segment AB replaced by the segment A'B'.

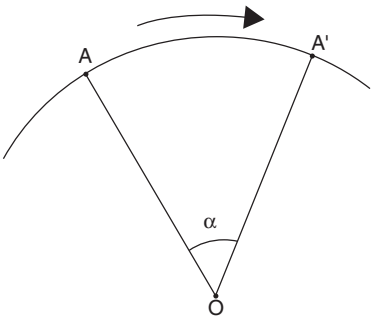

Figure 5.2
Definition of an arc and of the angle at the center of rotation.

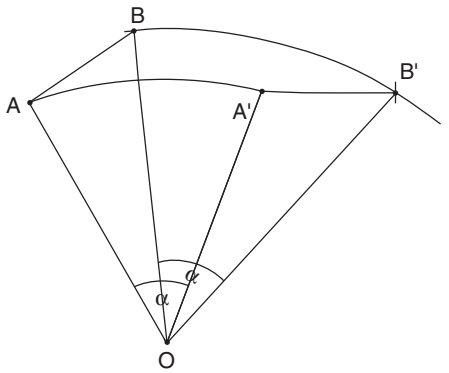

Figure 5.3
The two angles of the rotation A and B are identical, equal to alpha.

When the segment is replaced by a volume, rotation can still occur around the point O. The volume can be assimilated from as many segments as required; each time the segment and point O determine a plane where rotation of the same angle α can be performed. This will determine the new spatial location of the volume.

A more complex possibility is when rotation is made around a linear axis xy. If there is a physical connection between the object and this axis, the displacement which will occur must be evaluated with the perpendicular to the axis drawn from each point of the numerous segments of the volume. For each segment, the angular value of the displacement remains the same.

Application to joint biomechanics

Rotation is commonly evaluated to determine precisely the situation of the distal extremity of the limb (the hand) in the space. The greatest difficulty is to know which center or axis of rotation is used. The anatomic space is always defined with three reference planes: sagittal, frontal, and horizontal:

1. The sagittal plane divides the body into two symmetric parts. In it, or in parallel planes, are determined flexion and extension of an entire limb. It is also according to this plane that we can evaluate lateral and internal rotations when the limb turns around a point, or around an axis, both of which must stay in the reference plane. For this, one marker is necessary, for example the thumb.
2. The frontal plane gives the definitions of abduction and adduction.
3. In the horizontal plane it is again possible to see the degrees of rotation.

The above evaluation can be understood if the limb is considered to comprise a single piece. It is impossible for a clinician to take into account the combined attitudes of segments of the limb. Thus it is essential to define which part of the limb is under consideration, before any evaluation of rotation, and then it is necessary to define, for each measurement, the centers or axes of rotations we use as reference. It is inaccurate to use only the three anatomic planes.

To take some examples: the simplest are those concerning the proximal joints of a limb.

1. At the level of the hip: flexion and extension are commonly evaluated in the sagittal plane (or in parallel planes). However, this method assumes that the mobility of the coxo-femoral joint occurs around a virtual axis of rotation which would be perpendicular to the sagittal plane, and this is not anatomically true. The existence of the femoral neck and the muscle layout next to the superior femoral extremity, together with anteversion of this neck, reveals that the real axis of flexion, which includes the center of the femoral head, is different and is probably close to the neck axis. Flexion of the thigh with regard to the pelvis can correspond to two different possibilities:
   - In the first hypothesis, flexion does not appear in a sagittal plane and this will lead to a lateral rotation: this rotation, as a parasite effect, will be noticeable at the knee or at the foot, which are the reference marks. Many authors call this an *obligatory rotation.* They continue to speak of flexion of the hip but forget to mention that the axis of rotation (and therefore the reference plane) has changed: rotation of the hip occurs around a different axis to the transverse axis previously described.
   - In the second hypothesis, if the sagittal plane remains the reference, flexion occurs without any rotation. The true axis of rotation being oblique and not transverse, it is necessary that some medial rotation occurs simultaneously, to compensate for an obligatory rotation. Some authors call this rotation an *associated rotation.*
2. At the level of the shoulder: we shall put aside the fact that the center of rotation of the humeral head is not

permanent and is replaced by a succession of points located on a 'J-shaped' line. Thus, it is assumed that the movements occur around a single point. Then the displacement of the entire limb can be evaluated according to the three classic anatomic planes. This is usually done by orthopedic specialists:

- The sagittal plane is well defined by elbow flexion of 90°. It is possible to evaluate medial and lateral rotations. Flexion is also called anteposition, and extension is called retroposition.
- It is in the frontal plane that we measure abduction and adduction.
- It is needless to say that combinations of displacements are the rule, and that the position of the limb also depends on the position of the shoulder in space.

3. At the level of the elbow (without considering prosupination, which will be covered later): the only movements possible are flexion and extension. These can be defined according to a transverse axis joining the medial and lateral epiphyses. However, the position of this axis varies, according to the position of the shoulder, which is the only one defined according to the three basic anatomic planes. In flexion and extension of the elbow, the only reference is the transverse axis.
4. The hand and the forearm: these again will be oriented by movements of prosupination and by movements of the wrist:
   - Prosupination comprises around an axis represented by a virtual line joining the radioulnar joints, both superior and inferior.
   - The wrist has its own movements, which are flexion and extension around a radiocarpal axis, and also pendular movements (radial and ulnar inclinations) around another axis perpendicular to the first. These movements may often be combined.
5. Each digital joint add its own complexity with its own mobility and axis: they will not be described here.

So, it can be unfortunate to utilize the same terms (flexion, extension, abduction, adduction, and rotation) when one wants to measure the range of rotational movement of the skeletal parts and joints of the superior limb without precisely defining the centers or axes of rotation.

It is clear that it is very difficult to evaluate the spatial situation of the hand.

We shall conclude by a typical example of this word confusion: the so-called 'Codman's paradox' which is arguable.[1] According to its classic description, there exists a mysterious rotation of the upper limb when this is moved in a particular sequence (Figure 5.4). At the beginning the limb rests at the side of the body, the hand being in a sagittal plane with the palm turned inwards and the thumb located anteriorly. Then the limb is moved in abduction of 180° (in fact there is already a humeral medial rotation of 90° in this position). Next it is flexed in the sagittal plane, during which the palm turns laterally and the thumb is oriented downward. When this movement reaches 180° the limb again lies on the lateral side of the body and seems to have turned laterally through 180° (a lateral rotation of 180° with regard to the initial position).

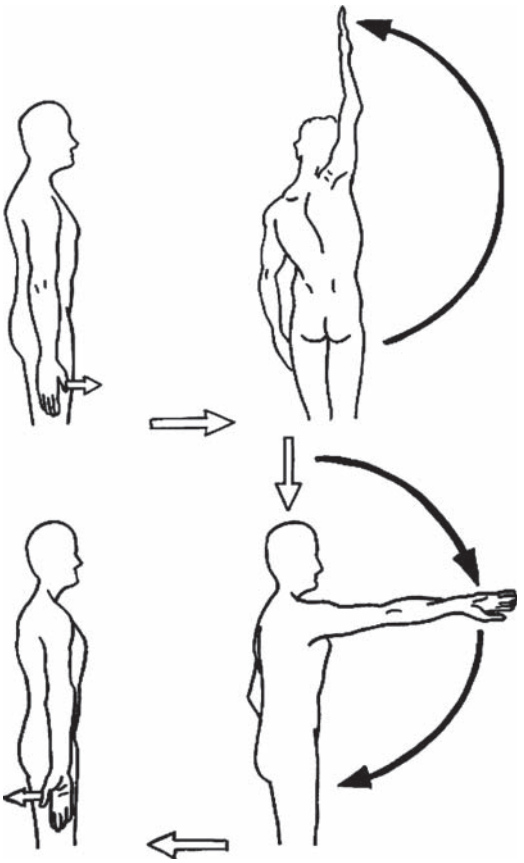

Figure 5.4

Codman's paradox: first a movement of abduction of 180°, followed by a flexion of 180° in a sagittal plane, leading to a palm looking outside with the thumb in posterior position. Adapted from reference 1.

In fact, nothing is paradoxical. The movements described are all rotations around one point (which is at the level of the shoulder) and mathematically the product of these rotations (which are three) is a single rotation equal to the intermediate rotation. Without entering into details of the demonstration, this shows that there is no paradox.

In the following schema (Figure 5.5) in which the elbow is flexed 90°, representing a rotational marker of the humeral bone, it is obvious that it is the flexion of the humerus which turns the hand, and that the lowering of the humerus in the sagittal plane leads the hand in an opposite direction to the initial position.

To conclude it is possible to say:

- that there is *no obligatory rotation* if one refers to the reference axes
- that the *associated rotation* exists if one keeps the anatomic plans of reference

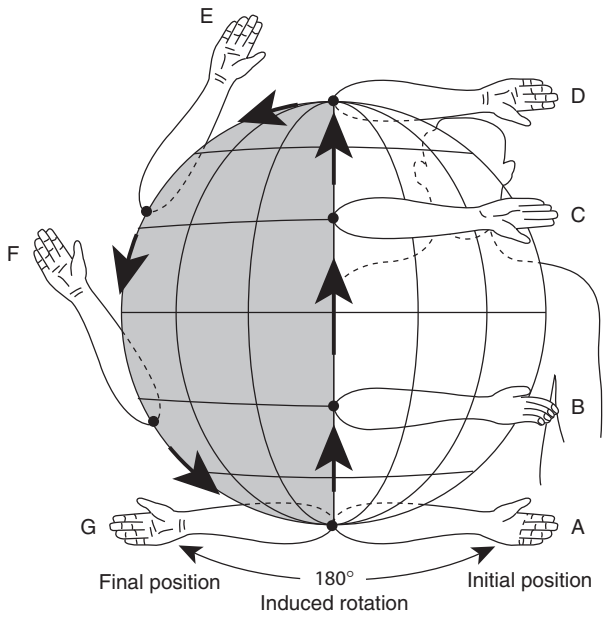

Figure 5.5

Codman's paradox movement sequence. Adapted from reference 1.

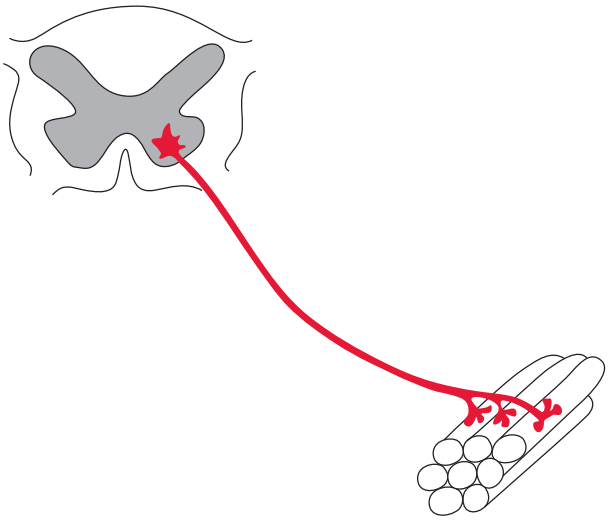

Figure 5.6

The motor unit includes one neuron with its cellular part in the anterior part of the medullary gray zone. Its axon reaches some muscular fibers at the level of the motor plate.

- and lastly that the *Codman's paradox* does not in fact exist. This demonstrates the difficulty in evaluating the spatial situation of part of the upper limb in clinical practice.

Basic notions of neurophysiology

There is nothing more instructive than the study of a scientific field using two approaches from different backgrounds.

Kinesiology encompasses the mechanical evaluation of forces generated by the muscles and applied on mobile bones. Engineers bring their science to the practitioners, by teaching them the lever arms, the moments of forces, and the methods used to study the vectors representing these forces. These elements are essential. However, they are not sufficient to explain the actual mechanism of movement.

It is the *neurophysiologic approach* that adds a priceless contribution to the biomechanical approach, even if there are still some 'gray' areas. Thus, it seems relevant to cover some basics of neurophysiology.

The motor unit

The muscle and the motor nerve cell that innervates it form the motor unit. As the stimuli are permanent and modify the ionic components of the nervous cell, there is constant activity in the muscle, with or without an objective effect.

The motor branches of the nerve cells (axons) are organized via a network extended between nerve centers. These centers are located at different levels of the nervous system, each one being connected with the sensory and sensitivity systems. The first level is the spinal cord, where there is a possiblity of self-regulation owing to reflexes. The higher cell centers, where voluntary movement begins, are located in the brain stem and the cerebral cortex.

This system is quite complex and has some remarkable functional properties. For example, the cerebral motor area, connected to many other areas, has the possibility of anticipation, i.e., it is able to imagine the movement and its results, and to adapt that movement according to visual and to proprioceptive information. Movement realization also supposes the use of patterns, mostly established during infancy.

The motor unit and its constant activity

The muscle as described, anatomically, has an obvious reality. Description of its insertions, of its belly, and of the direction of its fibers seems sufficient to explain the action due to its contraction, usually understood as a narrowing of the two extremities. However, the muscle represents much more than a simple excitable and contractile tissue:

- In fact it is part of a *bioelectric system* composed of a number of motor units (as mentioned above, connecting a motor axon to some muscle fibers) (Figure 5.6). The junction between nerve and muscle is represented by the motor plate, which is a kind of synapse where the nerve impulse is transmitted to the muscle fiber through the secretion of a neurotransmitter called acetylcholine.
- The second point is that the muscle fibers are not identical in one muscle. There are *short* fibers, which contract quickly and rapidly become tired; they are

innervated only by small branches of the motor nerve. These fibers are more numerous in the muscles at the distal extremities of the limb (forearm and hand), where rapid and delicate movements are required. Other fibers are *longer* (some of them are as long as the muscle itself). They contract more slowly and are less quickly tired. They are innervated by larger nerve branches. These fibers are the majority in proximal muscles: their long-lasting contraction and their tendency to resist fatigue explain their role in posture. It is important to know that these two types of fibers are unequally distributed inside the same muscle, and that their total number is quite different according to the muscle type as mentioned.

The motor fibers in the trunk of a single nerve also vary and are independent of each other. There is also a possibility that they are not stimulated at the same time.

Thus, in contrast to what was usually taught to orthopedic students, a muscle does not contract as one homogeneous unit. Therefore the contraction can be modulated inside the muscle volume. Likewise, the muscle can vary the force it generates. This possiblity is also linked to the intensity and the frequency of the nerve stimulus.

The nerve fibers are recruited according to a strict order, which depends on the size of their cellular body: the smallest axons have the lowest threshold (*Hennemann's size principle*). Each motor unit obeys the law of 'all or none', but their numbers vary during the action of one muscle. The orientation and value of the resulting force it generates will consequently vary, and this corresponds to a physiologic movement.

This conception of muscle contraction seems new and fundamental. It allows a better understanding of the variability of muscle action and of its adaptation when its insertions must change, the bones moving in different positions. So, in the same muscle different fibers will intervene according to the direction and value of the force required. In connection with this, it becomes possible to admit that one muscle has more than one distal insertion. For example, an intrinsic muscle of the hand has three means of insertions – one on the first phalanx base, one on the dorsal aponeurosis, and finally one on the lateral band of the extensor tendon. Its action will vary according to the position of the digital joints, and will be applied preferentially on one of these insertions according to the position of the metacarpophalangeal joint and the sliding of the dorsal aponeurosis (Figure 5.7) (see Chapter 14).

There is another very important notion which is not sufficiently regarded.

Muscular work or action is not only a contraction with shortening of the belly and approximation of the proximal and distal insertions. Indeed, there are three different situations:

- In the first one, the more commonly considered, contraction results in a shortening of the muscle. This is known as *isotonic contraction* (Figure 5.8a).

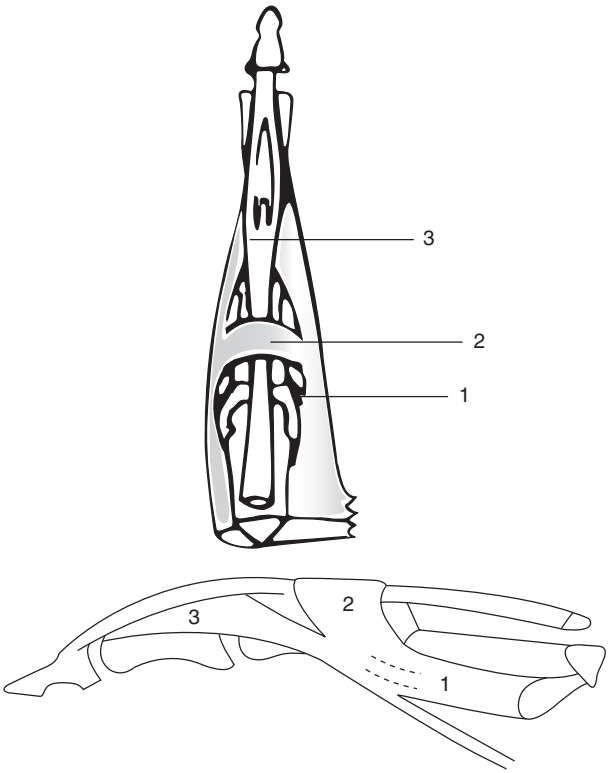

Figure 5.7

The three insertions of an intrinsic muscle: (1) one on the phalanx base; (2) one joining the same on the other side, forming the 'dossière' or wing tendon; (3) the third one on the dosal aponeurosis.

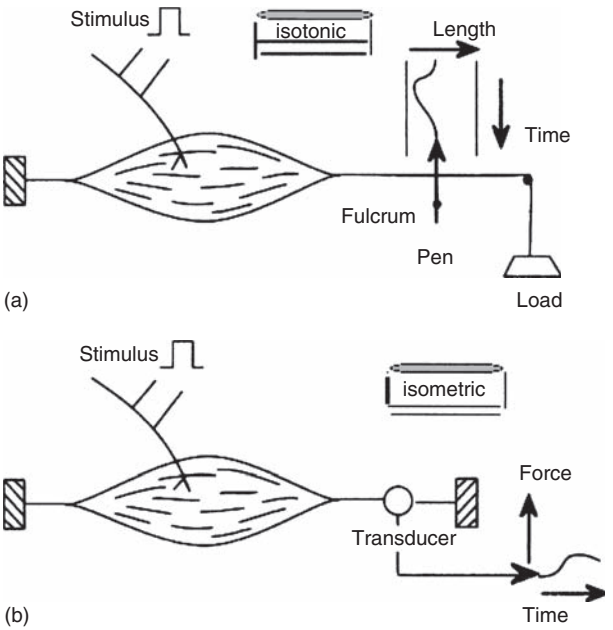

Figure 5.8

Two different types of muscular contraction: (a) isotonic, with shortening of the belly as shown by the pen; (b) isometric, without any shortening of the belly, but exerting forces on the extremities of the muscle.

- The second possibility is called *isometric activity*, in which the length of the muscle remains constant (the two extremities are fixed by the work of many other stabilizing muscles). This situation is explained by the contraction of short fibers inside the muscle, which will develop their tension on the tendon, and on other fibers which will be elongated in the belly. Thus, there is a force exerted by the muscle at the level of its bony insertions, a force which can be measured with a transducer. But there is no movement. This force serves to compensate external forces such as gravity, or forces generated at the same level by the so-called antagonistic muscles, which are obligatory in the same state of isometric activity (Figure 5.8b).
- The third eventuality of muscle action is less well known. It is called *activity with elongation*. The explanation is similar to the preceding; there is a different use of short and long fibers, but the resultant of the forces is in favor of the antagonistic muscles and a movement happens. The fibers which are elongated will contribute to stabilization of the proximal joints, and will serve as a brake, giving fluidity to the movement. So, in this third situation, the muscle is plainly active, but this activity will not appear as a 'contraction'. For that reason it seems better to use the term activity rather than contraction to describe the condition of work of a muscle.

Regulation of muscular activity

Thus, muscle is not only a contractile mass, it is one element of a neuromuscular system. This system is self-regulated at the level of the spinal cord. The neuronal networks are assembled in a similar manner to a cybernetic system, with part of the information getting back to its source to regulate its action.

Tonus

Neurophysiologic studies show that there exists an electric potential due to chemical and physical structures at the level of the membrane limiting an axonal cell. This permanent resting potential is transformed into active potential or influx under the effect of a stimulus, which can have a very large origin. This active potential will proceed along the nerve fiber (the speed of progression varies according to whether there is a myelin sheath around the axon). When the influx arrives at the synapse it causes the release of a chemical neurotransmitter (acetylcholine). Under this action a newly generated influx appears in the nerve cell in contact with it, or in the muscle fibers. Thus the permanent excitability of the nerve induces a muscular excitability which is also permanent.

The number of stimuli, themselves received by more or less important muscular activity, explain that there is more or less a muscular response. In addition, the brain and the central nervous nuclei can initiate this activity. Thus, in a living body, there is no real period of total rest. It is necessary to clarify the term 'tonus'. First, this is variable in the living body, depending on the stimuli. Second, the basic activity exists and a muscle has the capacity of an immediate response. The tonus is the minimum activity always present in all muscular mass. It is possible to compare it to a motor engine, which is permanently running slowly, and which can increase its speed depending on the appropriate stimulus.

Reflexes in the regulation of muscle activity

Self-regulation at the level of the spinal cord is relatively straightforward. Again, an analogy with cybernetics can be made: part of the stimulus is going back to its source and can regulate it (feedback).

There are two systems:

1. The first system is linked purely to the characteristics of the axons. These induce the *reciprocal innervation*: an excitation of some muscles is usually associated with an inhibition of another group of muscles. This can be explained by the fact that the alpha motor axon gives rise to a collateral branch in the spinal cord, which enters into contact with small neurons (Renshaw's cells or inhibitory neurons), which, at the same time, will inhibit axons around the initial one, as well as the axons that innervate the 'antagonistic' muscles (Figure 5.9).
2. The second system is determined by the action of two types of sensorial receptors (muscular proprioceptors): the neuromuscular spindle and the tendinous Golgi's organs.

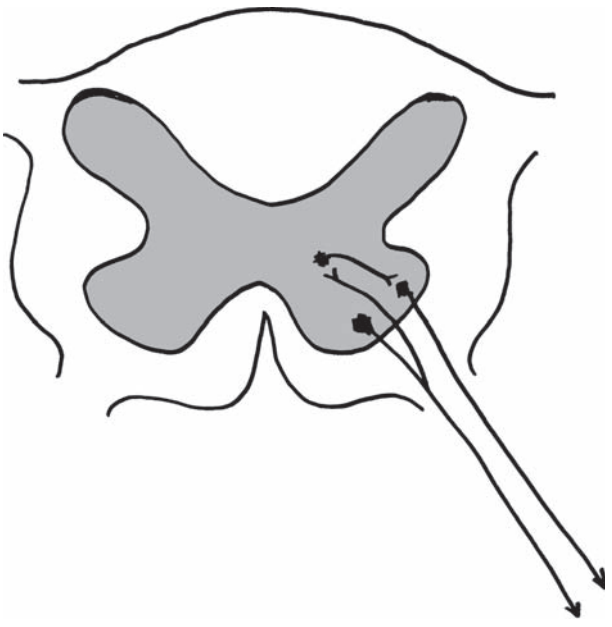

Figure 5.9

Renshaw's cell, which is connected by a recurrent fiber of the alpha motor neuron to an interneuron connected to another alpha motor neuron. This system is inhibitory.

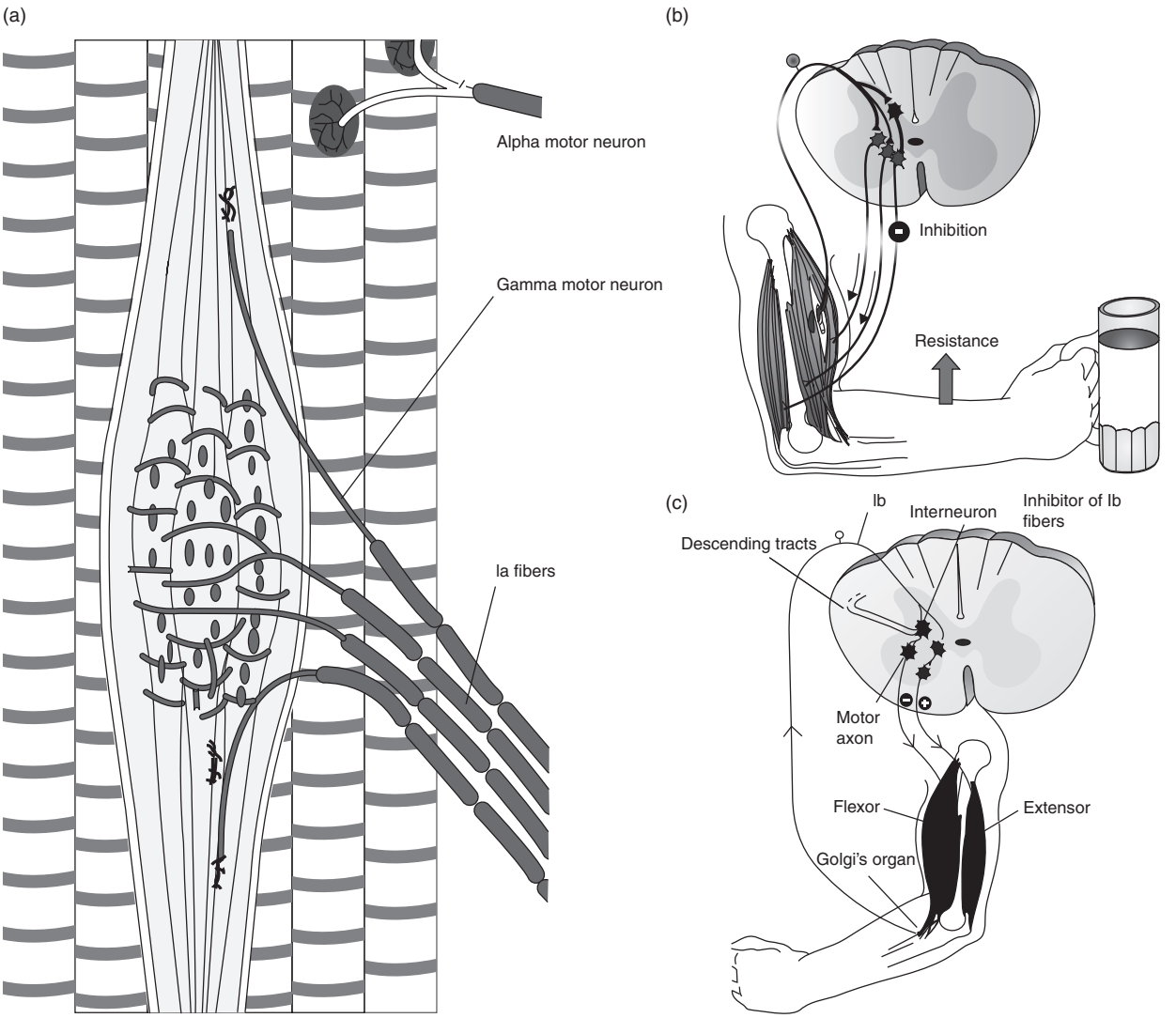

Figure 5.10

(a) The neuromuscular spindle and its two types of fibers (adapted from Purves et al[2]); (b) the myotatic reflex; (c) connections of the Ib fibers from Golgi's organ.

- The neuromuscular spindles are distributed inside the fleshy bodies of the striated muscles. They consist of special muscle fibers which lie on the connective tissue intramuscular septi of the belly and have a special innervation. Rather than describe the exact arrangement of the efferent and afferent nerve fibers of the neuromuscular spindle (Figure 5.10a), it is only necessary to say that they provide information on the length of the muscle and on the speed of variation of this length. The neuromuscular spindle acts as a servo-motor, according to length (retro control). It tends to re-establish the length of a muscle which has been elongated. In practice, lengthening of the spindle (which corresponds to a lengthening of one muscle) creates the myotatic reflex, generating a contraction of this muscle (stretch reflex). Also, the recurrent innervation will have associated effects. This is also necessary for a movement and its adaptation. The myotatic reflex is essential in postural activity (Figure 5.10b).
- Golgi's organs are situated in the aponeurosis surrounding the muscles and the tendons. They are made of collagen fibers and are innervated by sensitive fibers which generate influx when the organs are elongated by muscle contraction. They give information on the variations of forces generated by the muscle on its tendon, and controlling them serves for their protection (Figure 5.10c).

The supra-medullary system is of particular importance due to the multiple connections established between sensory and motor systems at every anatomic level. In the brain stem, connections are situated in the retinaculated nuclei, part in the annular protuberans, part in the medulla oblongata (bulb). The first group receives stimuli from the vestibular nuclei and the cerebellum, and is an

excitatory system which mainly stimulates the muscles which oppose the effects of gravity. The second group receives stimuli from the red nucleus, from the corticospinal motor tract originated in the brain. It has an inhibitory action and it is important to balance the first group and to release some muscles from their role in posture to perform another task. The cerebral command is obviously fundamental, not only to coordinate the different servomechanisms studied, but also in all the activities of the human being.

The posture mechanism is one of the major conditions which can serve as an example of muscle regulation. Visual information is added to that of the muscle proprioceptive system. The importance of these two factors diminishes when internal patterns are established that are utilized almost subconsciously. The vestibular system, with its semicircular canals, is also of prime importance as an organ of dynamic equilibrium.

Posture must be considered not only as the situation of the entire body, but as an attitude at any given moment. This attitude or posture is regulated by mechanisms called 'top-down', because they start in the brain. The brain selects motor nerve excitation and, by a complex mechanism utilizing memory and conscious interpretation (forehead cortex), anticipates and evaluates the effects which will be produced by the planned muscle activity. Thus, posture is not the result of serial reflexes. Selection is possible due to sensory information and anticipation compares the receptor conditions with a preceding model. As Bernstein[3] said, posture is readiness to perform a movement.

Consequently, movement can be considered as a succession of postures. Indeed, movement produces a progressive modification of the balance between muscles on both sides of a joint. This balance is never static. It is continuously modified by the cerebral commands which regulate the activity of the muscles, so that there is no jerk when passing from one position to another.[4]

The concept of agonistic and antagonistic muscles has to be reconsidered. Actually, it no longer corresponds to reality: each muscle in the living body brings it own contribution to the whole. To make a local gesture the resultant of forces generates a mobilization of bones and creates the movement. However, not only the local muscles are active – other muscles spread throughout the body, which are not, at first sight, connected with the movement required, are also more or less active. They are necessary for the general posture of the individual, for the orientation of the limb, for the correction of the effects of gravity, and so on.

Thus adaptation is always necessary, and the use of models or patterns must be modified continually. The choice of a pattern is made automatically as a function of proprioceptive information and of psychic anticipation. The nervous system includes the premotor area and the supplementary motor area of the frontal lobe of the cerebral cortex. The motor patterns are not fixed, and they can probably change and adapt themselves to a mechanical change of the extremity of the limb, and this is important for the surgeon.

Is it possible to have a global interpretation of the physiology of movement?

First, a will emerges in the cerebral cortex to accomplish a gesture with the intention to do something. This will proceed from various stimuli, external or internal. Then a typical pattern, more or less adapted and which can be modified during its application, is chosen, in most conditions, subconsciously. Muscles of the body, which are always capable of a quick response, will create a posture, adapted toward the required movement. It is the image (mental picture) of the movement which creates a kind of motor wave. This wave will excite the motor areas, and proportionally the number of motor units to be activated in each muscle, with the result that the specific joints are moved.

The result is permanently controlled by the different systems already described. There are two interesting consequences to these theoretic considerations. After a trauma which has induced some functional loss, it is notable that a movement normally used for a defined purpose will be modified. However, either spontaneously or with the help of a physiotherapist, the gesture will be done according to the muscular and/or articular remnants in the limb. Another consideration is that, in contrast to the idea that a cerebral pattern is fixed and would be an impediment to rapidly learning to use a modified part of the anatomy – for example due to surgery – the concept of good adaptability of the cortex is conducive to more active surgery in some particular cases (e.g., congenital changes).

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6 Biomechanical research on muscle-tendon transfers: why progress depends on close collaboration between surgeon and bioengineer

Vincent R Hentz, Wendy Murray, M Elise Johanson, Neils Smaby, Katherine Saul Holzbar, Kevin McGill, and Zoya Lateva

Introduction

Loss of hand and upper limb function is a profound debilitating consequence of all paralytic processes, from injury to a single peripheral nerve to cervical-level spinal cord injury. While the functional loss varies according to the magnitude of the injury, clinicians have long recognized that these patients have finite numbers of remaining muscles under voluntary control that can be surgically redeployed to restore useful upper limb function. Therefore, the goal of all surgical procedures has been to optimize the functional outcome by careful selection of patient and procedure so that these scarce resources are not wasted. However, most reconstructive surgeons today make decisions based primarily on training, experience, and enlightened intuition.

There have been relatively few advances in the field of functional muscular reconstruction in the last 25 years. The major exceptions include free muscle transfers and the application of functional neuromuscular stimulation to the paralyzed upper limb. However, unsolved problems still exist, including severe brachial plexus injury and cervical spinal cord injury. For patients who suffer such injuries, there exist few available resources for functional reconstruction. In such cases, the surgeon must choose wisely from a very limited number of options to optimize function. In contrast, for some clinical problems, the opposite circumstance exists. For example, for a low median nerve injury, the dilemma resides in the wide menu of seemingly equally effective choices for some injuries. This becomes a source of confusion. How does even the experienced surgeon choose from so many available procedures to obtain the optimum outcome for the patient with a median nerve injury at the level of the wrist?

What holds back progress? We have identified a number of factors, including the following:

- Most treatments date from 50–100 years ago; essentially all tendon transfers were described long ago.
- Most hand and upper limb surgeons 'cookbook' certain injury patterns. We do what works for us, is easy to do, and is predictable – or because it's the only way we know!
- Few new operative procedures are designed from the 'ground up'. Most are variations on some pre-existing procedure.
- Most concepts and principles are rarely, if ever, challenged, probably because:
  - surgeons believe that we know the important anatomy;
  - surgeons believe that we understand the relevant mechanics of the hand;
  - what we don't fully understand is felt to be unimportant.

What needs to change? Surgeons must learn to apply new knowledge and technology to familiar and presumably solved problems as part of rethinking and redesigning treatment algorithms and procedural choices if we are to improve outcomes of tendon transfers. And to improve

outcomes, we surgeons must join with our basic science and bioengineering colleagues.

Despite the clinician's best efforts to adhere to proven principles of treatment, a review of the functional outcomes of surgery to restore function in the quadriplegic upper limb shows significant variability, from excellent to severely disappointing, even among patients who preoperatively are much alike in terms of the numbers and strengths of upper limb muscles over which they still retain voluntary control. For example, Waters studied the force of pinch following brachioradialis (Br) to flexor pollicis longus (FPL) transfer in 17 hands of 15 quadriplegic patients, and found that while pinch strength averaged 4 lb (1.8 kg) with the elbow flexed and the wrist in a neutral position, the range was from 1.1 to 10 lb (0.5 to 4.5 kg).[1] House, in comparing similar patients preoperatively, found pinch forces varying from 2.3 to 5 kg (average 4.0 kg) following Br-to-FPL transfers.[2] In our own Palo Alto patients, 1 year following surgery, preoperatively similar patients who had undergone Br-to-FPL transfer demonstrated pinch forces averaging 20 N but varying from 5 to 35 N.[3]

These variations in outcome of the same procedure performed in relatively similar patients exemplify the need to understand muscle-tendon transfers at a more fundamental level. For the past 12 years, in our Grasp Research Unit at the Bone and Joint Center (part of the Rehabilitation Research and Development Center, Palo Alto Veterans Affairs Health Care System), surgeons and biomechanical engineers have focused on tendon transfer procedures and on the transferred muscle using computational and anatomic modeling, electrophysiologic analysis, and unique tools to measure force and directional control in an effort to:

- understand how the normal hand produces grasp
- characterize the changes in function brought about by specific paralytic processes
- gain new understanding of the effects of surgery of transferred muscles
- develop new therapeutic strategies
- optimize hand and upper limb function following muscle tendon transfers.

This chapter focuses on some of the work accomplished by our group on better understanding muscle-tendon transfers.

Rethinking tendon transfers: the tetraplegic patient as the research model

One focus of our research program has been to use biomechanical tools as a framework for conceptualizing, implementing, and evaluating novel rehabilitation procedures for the restoration of grasp after tetraplegia. We have focused on the tetraplegic patient as a model in rethinking tendon transfers because the tetraplegia patient is a unique research subject. Their injury has rendered more simple a previously very complex mechanism. Now only a few muscles retain function, and if one is transferred to perform a new task or function, then the function achieved is solely the outcome of the transferred muscle. The tetraplegic upper extremity is a unique research model offering opportunities to study muscles, biomechanics, and the effect of surgery on physiologic systems – especially tendon transfers, the most common approach to restoring hand function after a cervical spinal cord injury.

Current concepts of muscle-tendon transfers

As commonly performed, the tendon of attachment of a muscle of appropriate strength (the donor muscle), still under voluntary neural control, is detached at some level and the tendon is attached to a new site. Typically, it is woven into the tendon of a weak or paralyzed muscle so that the donor muscle mimics or restores the function of the weak or paralyzed muscle. Surgeons have observed that donor muscles commonly lose at least one grade of strength after tendon transfer.[4] While the reasons for this are not understood, an animal study has shown that muscles may recover as little as one-quarter of their previous working capacity after tenotomy, neurovascular repair, and muscle transplantation, suggesting that surgical intervention can alter a muscle's basic functional capacity.[5] Therefore, a successful transfer requires a donor muscle with adequate strength to perform the desired function. Most importantly, the function gained must far exceed the function lost by redeploying the donor muscle. While tendon transfers do result in functional gains for many individuals with cervical spinal cord injuries, the variability in outcomes and reduction in muscle function postoperatively need to be more fully understood to optimize surgical results for all patients.

The brachioradialis as a research model

One muscle in particular has been extensively studied by us because it is so commonly used as a tendon transfer in quadriplegic patients capable of benefiting from tendon transfer surgery. This muscle, the brachioradialis, is the muscle that most commonly retains or regains suitable strength and neural control in essentially all quadriplegic patients who are candidates for surgical restoration of grasp and pinch. Because it normally functions as an accessory elbow flexor, for the quadriplegic patient who retains strong elbow flexion via more efficient muscles (biceps and brachialis), the brachioradialis is viewed by surgeons as the most expendable muscle for a tendon transfer.[6]

It is, therefore, the most commonly transferred muscle in the quadriplegic patient. Depending on patient needs, it may be transferred to augment the strength of wrist extension (by transfer to the extensor carpi radialis brevis, ECRB) to restore finger extension (by transfer to the common digital extensor tendons), and to restore finger flexion (by transfer to the flexor digitorum profundus tendons). Across centers performing tendon transfers in quadriplegic patients, the most commonly performed transfer is attachment of the tendon of the brachioradialis to the tendon of the flexor pollicis longus to restore active key pinch, termed Br-to-FPL transfer. However, as mentioned above, outcome studies of Br-to-FPL transfers have demonstrated considerable variability, even when muscles of similar preoperative strength were transferred in preoperatively similar patients. While Br-to-FPL tendon transfers do result in functional gains for many individuals with cervical spinal cord injuries, the variability in outcomes and reduction in muscle function postoperatively need to be more fully understood to optimize surgical results for all patients.

Why is there such variability in outcome?

There are many factors that account for variability in pinch force following Br-to-FPL transfer (or, indeed, following any tendon transfer), including:

- surgical and postsurgical alterations in the force-generating capabilities of the brachioradialis muscle – we define these as 'architectural/biomechanical perturbations'
- inability to activate effectively the transferred brachioradialis muscle in its new role as a flexor of the thumb – we define these as 'command/control perturbations'
- events that affect the amplitude or excursion of the transferred tendon (e.g., postoperative scar or adhesions that bind the transferred tendon to surrounding tissues) – we define these as 'force transmission perturbations'.

These factors are further defined in Table 6.1.

Rethinking muscle properties

Muscle architecture/innervation

The most critical determinant of the functional outcome of a tendon transfer is the force-generating capacity of the donor muscle. Clinical studies often cite insufficient postoperative muscle strength as the cause of poor surgical outcomes.[7] A muscle's overall capability to generate force is determined by its architecture, defined as the length, number, and organization of fibers within a muscle.[8] Studies that document muscle architecture in cadaveric specimens, particular the physiologic cross-sectional area, are often used by clinicians to evaluate how well matched a donor muscle is to the force and excursion demands of its new function.[9-11] However, since the majority of these cadavers were of elderly individuals, these studies relating the cross-sectional relationships between upper limb muscles may not accurately reflect the norm. Importantly, using data describing the architecture of non-transferred

Table 6.1 *Factors that could influence hand function after tendon transfer*

Description	*Determined*		
	Pre-	*Intra-*	*Post-*
		Operatively	
Factors that impact force produced by donor muscle			
• Donor muscle architecture	×?		×?
• Motor learning			×
• Surgical tensioning		×	
• Muscle damage: muscle denervation, damage caused intraoperatively	×	×	
Factors that impact force transmission			
• Healing process: formation of scar tissue, adhesions			×
• Other complications: tendon stretch, tendon rupture			×
Factors associated with upper extremity impairment			
• Strength of proximal muscles	×		
• Passive joint properties	×		

muscles to evaluate the appropriateness of the muscle for a tendon transfer presumes that no architectural changes occur following the surgical procedure. However, in vivo studies of muscle function following surgical interventions performed in animals provide data suggesting that basic structural properties of a muscle change postoperatively. For example, some animal data indicate that muscles have the capacity to adapt appropriately, with changes in muscle cross-sectional area (which is proportional to the maximum force) and length (which is proportional to the range of lengths over which force can be produced) matched to new demands for force generation and excursion after surgical alterations.[12] Such data suggest that muscles effectively accommodate to new tasks and innately reorganize their structure to optimize output. In contrast, data from a study involving rabbit hindlimbs demonstrated that when a transferred muscle is attached at longer lengths, as has been documented to occur in human patients,[13] adaptations occur that limit the range of lengths over which the muscle can produce force, which would ultimately degrade the efficacy of the transfer rather than improve it.[10] Whether muscle adaptations optimize or limit postoperative function, our current understanding of surgical outcomes of tendon transfer surgeries does not account for postsurgical changes that may occur in muscle architecture. We need more information regarding the architecture of the commonly transferred muscles and what changes occur in these muscles following surgery.

A muscle's fiber architecture plays an important role in determining its biomechanical action,[8] and can be expected to play an important role in the biomechanical outcome of tendon-transfer surgery. Consider the brachioradialis, the 'workhorse' of tendon transfers in the quadriplegic populations. There is increasing evidence that the brachioradialis muscle is not a simple parallel-fibered muscle as has often been assumed, but rather has a 'series-fibered' architecture. Series-fibered muscles are muscles in which the muscle fibers do not span the entire length of the fascicle. Rather, these muscles are made up of arrays of short, non-spanning fibers arranged in series.[14–16]

This architecture has been found in a number of long, straplike muscles in several animal species. The series-fibered architecture makes it possible for muscles with long overall lengths and excursions to be made up of relatively short muscle fibers.[17,18] Shorter fibers are thought to be more effective than longer ones because the electrical excitation can reach and activate the sarcomeres at the ends of the fiber before the contraction stretches them beyond their optimal length.

Series-fibered muscles have not been widely documented in humans, but several studies suggest that the human brachioradialis may have this architecture. The brachioradialis is a very long muscle, with the longest fascicles of any upper limb muscle.[19] It has been shown, using microdissection and serial cross-sectioning, that some brachioradialis fibers terminate intrafascicularly.[20] It has also been shown, using cholinesterase staining, that the brachioradialis has multiple endplate zones.[21] Multiple endplate zones are needed by serial-fibered muscles in order to supply the fibers at different proximodistal levels.[18,22] We have demonstrated the existence of multiple endplate zones and intrafascicularly terminating fibers in the brachioradialis using an electrophysiologic method. We have also demonstrated that the brachioradialis contains some muscle fibers that are multiply innervated,[23] which is a distinguishing feature of series-fibered muscles.[24]

The way in which the fibers of a series-fibered muscle transmit force to the tendon is complex and incompletely understood. Much of the force of intrafascicularly terminating fibers is transmitted laterally to connective tissue and neighboring muscle fibers.[25,26] The force ultimately transmitted to the tendon is therefore greatly affected by the stiffness or elasticity of the adjacent and in-series fibers. Reconstructions of individual motor units in animal muscles suggest that the fibers of individual motor units are largely confined to a specific proximodistal region.[27,28] Thus, some degree of coordination between the motor units at different levels is required in order to transmit force effectively from tendon to tendon.[29] It is thus to be expected that in a series-fibered muscle, any disruption of the innervation to the fibers at a particular proximodistal level would have a severely debilitating effect on the muscle's ability to produce force. We are concerned that the recommended surgical techniques for mobilizing aggressively the brachioradialis in order to prepare it for tendon transfer may result in denervation of some of the muscle. Paralysis of one of the serially linked bands of a series-fibered muscle would severely weaken the muscle by preventing effective force transfer to the tendon. This would reduce the potential postoperative force-generating capabilities of the muscle in performing its new task. Muscle strength might be preserved if the muscle remodeled from a series-fibered architecture to a single band of fibers with an effective distal tendinous insertion. However, the remodeled muscle would be shorter than the normal muscle, and so its excursion and its ability to produce force in different arm postures would be considerably different than predicted by models based on the normal muscle length.

We are currently studying how the architecture of the transferred brachioradialis muscle differs from the native muscle, and have recently developed a new technique to evaluate aspects of muscle architecture in vivo[23] that involves analyzing motor-unit action potential (MUAP) waveforms recorded during voluntary muscular contractions. The MUAP waveform recorded by a monopolar intramuscular electrode is a temporal record of the electrical events that take place during the motor-unit discharge, beginning with the initiation of the action potential at the endplate and ending with its termination at the muscle/tendon junction.[30] By measuring the latencies of specific waveform features that correspond to these events, it is possible to estimate the location of the endplate and the muscle/tendon junction with respect to the electrode. Moreover, by analyzing multiple MUAPs from the same signal, it is possible to obtain information about the architectural organization of groups of neighboring motor units.

This technique is illustrated in Figure 6.1, which shows results from a typical normal subject. The waveforms in

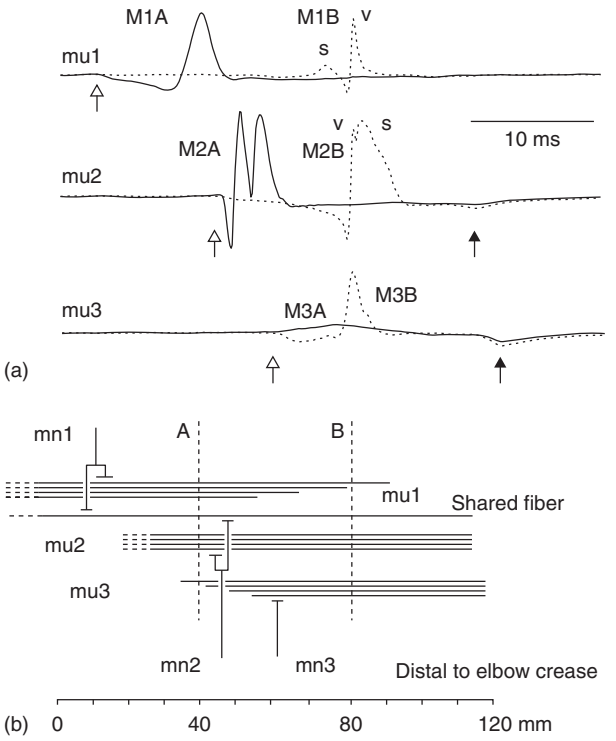

Figure 6.1

Motor-unit architecture of a normal brachioradialis muscle. (a) Motor-unit action potentials (MUAPs) from three motor units recorded at two different sites in the muscle (A and B). M1A is the waveform of the first motor unit at site A, etc. s and v are the stable and volatile components of the MUAPs that shared a multiply innervated fiber. The MUAP onsets are marked by open arrows, and the terminal waves by filled arrows. (b) Architecture of the three motor units determined by analyzing the MUAP waveforms. A and B show the location of the electrodes. mu1 had a proximal endplate (indicated by mn1) and terminated intrafascicularly. mu2 and mu3 had distal endplates and terminated at the distal tendon. The fiber shared by mu1 and mu2 had one endplate in the proximal zone and one in the distal zone.

Figure 6.1(a) show three representative MUAPs of a total of 16 MUAPs identified in the muscle. Figure 6.1(b) illustrates the architecture of these three motor units. The motor units were arranged in two distinct bands. Nine of the motor units were similar to mu1, with their endplates in a proximal band, and with diffuse intrafascicular distal terminations. The other seven motor units were similar to mu2 and mu3, with their endplates in a distal band and with tendinous distal terminations. Two of the motor units shared a multiply innervated fiber, which had an endplate in each band. These results are therefore consistent with a series-fibered architecture.

We are using this technique to test the hypothesis that substantial changes in muscle architecture can take place after tendon-transfer surgery. This involves comparing the presurgical brachioradialis muscle with the same muscle once it has been transferred. The results will reveal whether there is loss of distal muscle after transfer and whether this likely results from injury to delicate intramuscular nerve branches during surgery or from postsurgical remodeling. A better understanding of these processes will be helpful for planning and guiding tendon-transfer surgeries.

Another factor that can affect the force-generating capacity of the donor muscle is muscle damage, including partial denervation resulting from the cervical spinal cord injury, and perioperative damage caused during surgery. Electrophysiologic analysis of muscles before transfer may be a useful tool in predicting the outcomes of muscle-tendon transfers.

Reassessing the role and properties of intercalary joints in tendon transfers

One principal tenet guiding the choice of a muscle to transfer is to choose a muscle of appropriate (i.e., sufficient) strength to perform its new task. As surgeons, we consider muscle strength based on parameters such as manual muscle testing (MRC scale) or on research equating the strength of one muscle to others in the upper limb.[11] However, strength is a manifestation of both muscle and joint properties and not either alone. Therefore, the fiber length/moment arm ratio of a muscle-joint combination must be considered when describing strength. This is particularly important for the Br-to-FPL transfer, where a muscle that normally affects a single joint (the elbow), through transfer to the tendon of the FPL, now influences multiple joints (elbow, wrist, and the three joints of the thumb) and is in turn influenced by the position of these joints across which it now acts.

For the Br-to-FPL transfer, the position of the wrist and elbow on pinch force has been studied previously. Waters[1] found that with the elbow flexed to 90°, the average lateral pinch was 3.9 lb (1.77 kg) with the wrist extended 30°, 4 lb (1.81 kg) with the wrist in the neutral position, and 2.3 lb (1.04 kg) with the wrist flexed 30°. There was a direct correlation between pinch strength and the amount of residual triceps and wrist extensor strength.

We have observed substantial variability in surgical outcomes of brachioradialis tendon transfers that restore wrist extension by transfer to the ECRB (Br-to-ECRB transfer)[31] and active thumb function by transfer to flexor pollicis longus (Br-to-FPL transfer). However, the source of this variability remains unclear. For example, seven of eight limbs evaluated following Br-to-ECRB transfer were able to extend the wrist from the resting position against gravity, but the arc of motion varied among the limbs[32] (Figure 6.2). In addition, postoperative wrist extension strength varied from an MRC grade of 1+ (subject H) to a grade of 4 (subjects A and C). Despite these large differences in outcomes, the subjects whom we evaluated had similar preoperative clinical characteristics. Seven of the individuals were injured at the C5 motor level,

and one was injured at the C6 level. Preoperatively, brachioradialis strength on manual muscle testing was either MRC grade 4 ($n=6$) or grade 5 (i.e., normal function, $n=2$). Preoperative assessments of brachioradialis strength did not distinguish the individuals who achieved at least grade 3 strength (i.e., can achieve full range of motion against gravity) postoperatively from those who did not. Postoperative assessments of elbow extension indicated weak elbow extension across subjects, which could play a role in surgical outcome. The duration of follow-up ranged from 15 months to 76 months across subjects. Differences in outcomes were not correlated with follow-up time.

In order to understand the effect of elbow position on the passive properties and force-generating capacity of the brachioradialis following transfer, we developed a biomechanical model of the Br-to-ECRB tendon transfer[32] that can be used to estimate the maximum isometric moment-generating capacity of the transfer. Using this model, we were able to generate force–length curves representing the operating range of the Br-to-ECRB transfer between full extension of the elbow (0°) and 130° of flexion in three different transfer states. The three curves correspond to a transfer attached at a loose or slack length, one whose tension is 'optimal', and one where the transfer is tensioned tightly (Figures 6.3 and 6.4). The model shows how altering the tension of the transfer and altering the position of the elbow influences each transfer type's force-generating capacity.

In order to simulate the Br-to-ECRB tendon transfer, nominal models of the elbow and wrist were adapted to represent the altered musculoskeletal path of the transferred brachioradialis.[31,33] These nominal models accurately estimate the isometric joint moments produced at both the elbow and the wrist by able-bodied individuals during maximum effort, and were developed based on detailed measurements of muscle architectural parameters[20] and moment arms.[34,35] For the Br-to-ECRB transfer, the differences between the model and the data suggest that muscle parameters that are normally adequate for modeling able-bodied muscles do not reflect the isometric strength of a transferred muscle. In particular, the biomechanical 'ideal'

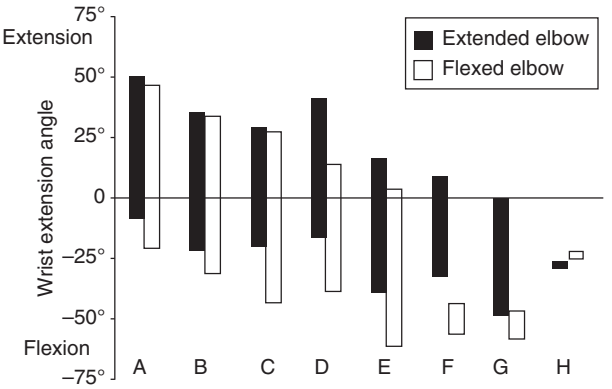

Figure 6.2

The range of motion of the wrist measured in eight wrists with the elbow extended (filled bars) and flexed (open bars). The positive angles indicate extension of the wrist, and the negative numbers indicate flexion of the wrist. Each bar indicates the resting position (bottom) and the most extended position (top) of the wrist against gravity. (Adapted from Murray WM et al. J Bone Joint Surg Am 2002; 84-A: 2203–10[32].)

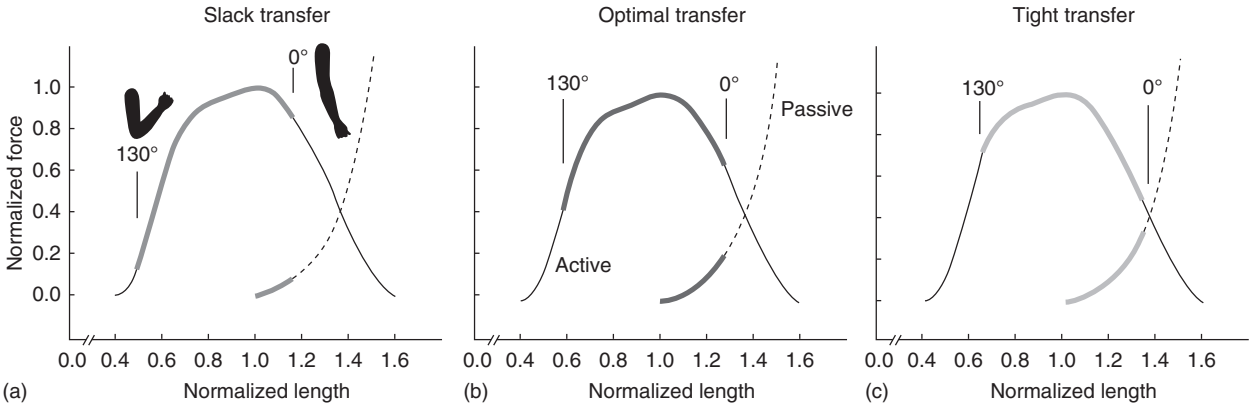

Figure 6.3

The model estimate of the operating range of the brachioradialis to extensor carpi radialis brevis transfer between full extension of the elbow (0°) and 130° of flexion for slack transfer (a), optimal transfer (b), and tight transfer (c), with the wrist in neutral position. The fiber lengths calculated with use of the model were normalized by the optimal fiber length and were superimposed on the active (solid thin line) and the passive (dashed thin line) isometric force–length curves to illustrate how length changes that occur with elbow flexion influence each transfer's force-generating capacity in different joint postures. In this figure, muscle force is normalized to peak isometric force. As indicated here, our model assumes that a muscle develops a passive force when it reaches fiber lengths that are greater than an optimal length. When the muscle is fully activated, the total force produced by the transfer is the sum of the active and passive forces. Note that, for any given joint posture, the slack transfer operates at the shortest fiber length and the tight transfer operates at the longest length. (Adapted from Murray WM et al. J Bone Joint Surg Am 2002; 84-A: 2203–10[32].)

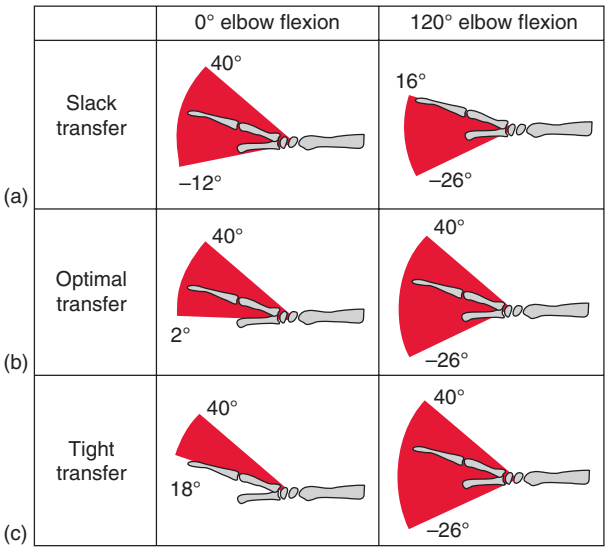

Figure 6.4

Computer simulation of the active range of motion of the wrist against gravity for the slack (a), optimal (b), and tight (c) transfers, with the elbow in two postures. The bottom arc of each shaded region corresponds to the resting position of the wrist against gravity; the top arc corresponds to the most extended position that can be maintained against gravity. (Adapted from Murray WM et al. J Bone Joint Surg Am 2002; 84-A: 2203–10[34].)

implemented in our computer model assumes that the brachioradialis can be maximally activated to provide wrist extension and that the transferred brachioradialis has the same architectural features as before transfer. We have preliminary data suggesting that these assumptions could contribute to the difference between the model and the data.

As mentioned above, we have also observed large differences in outcomes of Br-to-FPL tendon transfers. Specifically, the lateral pinch force produced during maximum effort varied by more than fourfold across a group of preoperatively similar patients. As with the Br-to-ECRB subjects described above, preoperative clinical assessments of brachioradialis strength were at least MRC grade 4. A manual muscle grade of 4 is clinically accepted as an indicator that the donor muscle possesses sufficient strength for an effective clinical outcome.[7,36] Currently, preoperative clinical assessments neither elucidate why nor predict such large differences in outcomes occur following tendon transfer surgery.

Factors that influence activation of the transferred muscle for its new function

Another factor that can contribute to a suboptimal postoperative outcome is the individual's inability to activate the transferred muscle to perform its new function. Studies involving animals and human subjects suggest that the nervous system can synthesize a change in muscle function imposed by surgery. For example, Yumiya et al[37] demonstrated that cats quickly relearned how to place their paw following transfer of a forearm muscle, and also documented changes in electromyograph (EMG) activity during walking that were consistent with the transfer being activated to perform its new function. In human subjects, Waters and colleagues[38] demonstrated that brachioradialis EMG increases with pinch force following transfer of the brachioradialis to the flexor pollicis longus (FPL). The brachioradialis is commonly transferred to the paralyzed FPL to restore active lateral pinch in able-bodied subjects. In these patients, FPL EMG also increases with pinch force. Thus, if the preoperative patient selection criteria are adhered to, we assume that patients will be able to re-educate the transferred muscle to perform its new function and that the postoperative strength of the muscle will be sufficient, even if some strength is lost after surgery.

However, muscles that are not directly involved in the transfer may be paralyzed or severely weakened due to the spinal cord injury and unable to provide sufficient elbow joint stability to allow the patient to fully activate the transferred muscle for its new function. This highlights an important aspect of tendon transfers, that the functional outcome of a tendon-transfer surgery can be affected by impairments in other proximal muscles that are not directly involved in the tendon transfer. For example, following Br-to-FPL transfer, the brachioradialis continues to flex the elbow joint (its native function) when activated to flex the thumb. Thus, the elbow needs to be stabilized during hand function.[39–41] Weakened elbow extensors, a common characteristic of cervical spinal cord injury, may limit the ability to stabilize the elbow in the presence of strong activity of the transferred brachioradialis. Similarly, because the FPL flexes the wrist, the transferred brachioradialis also flexes the wrist. As a result, wrist extension strength is another critical factor that will influence how strongly the transferred brachioradialis can be activated. Thus, the strength of a donor muscle's antagonists and synergists can impact the ultimate outcome of surgery.

We have noticed in our own patients a wide range of pinch force following transfer of brachioradialis muscles of essentially equivalent strength, and have hypothesized that inability to activate fully the brachioradialis following transfer may explain, in part, these differences. In a small number of patients, we placed fine-wire electrodes into the transferred brachioradialis and recorded pinch force with and without elbow stabilization (Figure 6.5).

By first recording the brachioradialis EMG activation as the subject maximally contracted the brachioradialis in resisted elbow flexion, we measured the muscle's activation during its normal function. This activation represents the maximum voluntary contraction (MVC). The patients produced maximum lateral pinch force with and without elbow stabilization. EMG signals recorded during these two lateral pinch activities were normalized to the MVC during isometric elbow flexion.

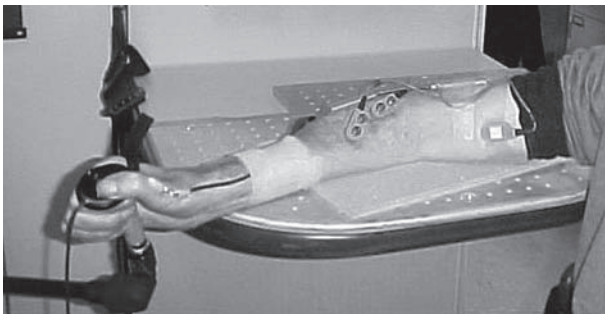

Figure 6.5

Fine-wire electrodes are inserted into the brachioradialis. Surface electrodes record biceps and triceps (in any) activity. The thumb is fastened to a free force sensor to measure pinch force. The elbow may be restrained by the device or left free with the patient being asked to self-stabilize the elbow.

Preliminary measurements in a small number of patients showed that the mean EMG during lateral pinch in the unstabilized elbow condition was 27% of the MVC (range 14–56%). With the elbow stabilized, the EMG increased to 42% of the MVC (range 17–65%). None of the patients was able to activate the brachioradialis to the same level recorded during resisted elbow flexion, even with the elbow stabilized to control for differences in elbow extension strength. The patient's pinch force also increased with the addition of elbow support. Those individuals with weak elbow extension also had the lowest pinch force magnitudes. These preliminary findings suggest that patients with Br-to-FPL tendon transfers have the potential to improve their pinch strength by improving their ability to activate the transferred brachioradialis. In addition, the lower pinch forces measured in the patients with weak elbow extension suggest that the brachioradialis had become weak. Postoperative rehabilitation that includes re-education and strengthening of the transferred muscle as well as the proximal joint musculature needs to be another focus for reassessment of current practice.

Factors that influence force transmission

The third important determinant of the functional outcome of a tendon transfer is the effectiveness with which the transferred muscle is able to transmit force via the tendon. This effectiveness can be impaired by postsurgical scarring or adhesions. This aspect is the most difficult to study in a non-invasive manner. However, newly developed dynamic magnetic resonance imaging (MRI) systems offer the possibility of evaluating postsurgical changes. With these techniques, it may be possible to visualize changes in the transferred muscle, changes in its physiologic cross-sectional areas, muscle lengths, scar adhesions that restrict excursion, etc.

What factors can the surgeon control? Surgical technique

Pre- and postoperative anatomic studies of muscle fiber architecture, pre- and postoperative electrophysiologic studies of muscle activation, and mathematical modeling of tendon transfers promise to address many issues related to suboptimal outcomes for tomorrow's surgeons. However, while many of the effects mentioned above may not be readily controllable by today's surgeons, there is one important variable that is controllable by today's surgeon: how the transfer is tensioned by the surgeon. Inadequate tensioning could influence postoperative strength, joint range of motion, and the integrity of the transfer. Current clinical practice needs a scientific basis to guide intraoperative surgical decision-making for tensioning tendon transfers.

Attachment length or tension of the transfer

The force that a muscle generates varies as a function of its length,[42] and previous studies have characterized the relationship of the length of a muscle (its tension) to its ability to generate force when it contracts (active component) or its ability to generate force as the muscle is increasingly lengthened (passive component). The peak force that a muscle can generate occurs at one specific muscle length along its physiologic length spectrum. Force-generating capacity diminishes as muscle length increases (i.e., the tension of the muscle increases) or decreases (slackening of the muscle resulting in decreased tension). Surgeons understand that the length (tension) of a donor muscle at which the muscle generates its maximum force occurs at what is described as the 'resting length' of the muscle. Surgeons believe that if a tendon transfer restores the normal resting posture of the hand – i.e., if the hand and digits, after the transfer, assume a normal resting posture – then they have tensioned the transfer correctly.

The initial physiologic analysis of muscle force–length relationships was conducted in amphibian muscles. These data have been applied to mammalian muscle without necessarily testing the muscles used for tendon transfers. Work by Fridén et al[43] indicates that different muscles of the upper limb have different force–length relationships, suggesting that it will be necessary to study muscles proposed for tendon transfers in order to know precisely how they should be optimally tensioned when transferred.

Clearly, a donor muscle could be made ineffective if the surgeon positions it at lengths that are too long or too short to generate adequate active force. Intraoperative assessment of muscle sarcomere lengths suggests that surgeons may be choosing donor muscle lengths that are

longer than the optimum length for maximum force production.[13,44] Clinicians traditionally believe that a transferred muscle will adapt by adding sarcomeres in series in this situation. However, this belief is not based on direct observations. Rather, it is inferred from other studies, such as animal models of joint immobilization, where muscles have been shown to add sarcomeres in series when the limb is immobilized in a joint position where the muscle is lengthened.[45] As noted above, an animal model of tendon transfer contradicts the traditional wisdom.[10] The study by Fridén et al demonstrated that choosing a longer length for the donor muscle induces subtraction of sarcomeres in series, decreasing muscle length and muscle excursion.

We recognize that there are other potential factors that influence the postoperative muscle length, including post-tensioning elongation of the tendon-to-tendon juncture. This is known to occur, and if significant elongation occurs during the postoperative healing or rehabilitation and exercise period, then the muscle will become slack. In this case, tension of the transfer measured at the time of surgery would be irrelevant, as it would no longer correlate with the actual ability of the transferred muscle to generate force. For the Br-to-FPL transfer, this variable is probably minimal, compared with other tendon transfers, because there are sufficient lengths of Br and FPL tendons to permit the surgeon to perform a 'tendon-weaving' juncture. Prior studies have shown that this method results in the greatest resistance to elongation at the suture site. In addition, recent studies have indicated that whether or not the elbow is immobilized (this varies among surgeons) following Br-to-FPL transfer has little influence on elongation of the tendon-to-tendon juncture. It is generally accepted that the opposite circumstance, namely postoperative tightening at the juncture, does not occur.

How surgeons currently tension a tendon transfer

Currently, surgeons tension tendon transfers based on one or several factors, all related to experience and education. Their stated goal is to set the tension of the transferred muscle as close as possible to what is termed the normal resting length of the donor muscle. In the case of the Br-to-FPL transfer, other visual clues are used to assist the surgeon in determining if the tension of the transfer is appropriate. For example, following attachment of the tendon of the brachioradialis to the tendon of the FPL, the surgeon will flex and extend the wrist to assess the effect of these positions on the movements of the thumb. As the wrist is extended, the transfer becomes increasingly tense, and the thumb should respond by flexing. As the wrist is flexed, the transfer becomes slack, and the thumb will extend somewhat. Based on experience and teaching, the surgeon will accept the tension as appropriate, or make further adjustments, loosening the transfer if the tension seems excessive, and vice versa. Because the brachioradialis crosses the elbow, some surgeons assess whether the tension is appropriate by flexing and extending the elbow. This also slackens or tightens (respectively) the transfer.

Currently, the most commonly employed technique is based on tactile feedback to the surgeon's hand, often described as how tight or loose the transfer 'feels'. This 'feel' is learned by experience, and is not easily taught or described. The inexact nature of this methodology has long been recognized, and previous investigators have studied more objective techniques.

Prior objective measures of tensioning a transfer: transducers

More objective methods to determine the optimum tension of a tendon transfer have been proposed and studied, but have not been widely adopted. Mendelson et al[46] described placing a turn-buckle transducer on the intact tendon of the muscle to be transferred and measuring the tension of the donor muscle at its 'resting length'. Using the same device, they tried to re-establish this tension following transfer. Whether because such techniques required the presence of engineering specialists in the operating room, or consumed too much intraoperative time, or the studies were not sufficiently persuasive, these techniques are not currently employed by most surgeons performing tendon transfers.

Novel measures: diffraction of laser light as a means of measuring the length of the sarcomere

The anatomic length of a muscle is a fraction of the length of the muscle fibers and the angle at which they are attached to the tendon of the muscle (the pennate angle). The muscle fiber is composed of a series of contractile elements termed sarcomeres aligned in series. The molecular structure of these contractile proteins permits the parallel muscle fibers to act as a diffraction grid. Physiologists studying muscle architecture employed laser light diffraction, and in a theoretic discussion describing the diffraction of laser light by a single fiber of striated muscle, Lieber and colleagues[47] established the theoretic basis for this technology. In a series of studies,[48] they set forth conditions under which grating information (i.e., sarcomere length) can be maximally expressed to yield accurate average sarcomere length values.

Lieber and colleagues[49] also used laser diffraction patterns to study sarcomere changes in a number of in vitro and in vivo studies. These laboratory studies led to the development

of a small, sterilizable device (the laser myometer) that could be used in the operating room to study the sarcomere architecture of normal and abnormal (spastic or contracted) muscles.

Because surgeons performing Br-to-FPL transfers have been educated differently, they perform this standard operative procedure in variable ways. For example, in an informal poll of surgeons who have performed many Br-to-FPL transfers, we found that at least seven different elbow and wrist positions were used in establishing the tension of the transfer.

To assess how surgeons currently tension the brachioradialis muscle during transfer, Lieber and colleagues performed a number of studies. They first characterized the passive force–length relationship of the brachioradialis muscle by attaching a transducer to the detached tendon of the brachioradialis and measuring the change in tension as they relaxed and tensioned the muscle. Using the laser myometer described above, they studied the relationship between sarcomere length and the measured tension as they stretched (tightened) and relaxed (loosened) the transfer. With these data, they could model the passive qualities of this muscle. Recall that it is only the passive properties of the muscle that the surgeon is able to assess at the time of surgery as he or she attempts to set the tension of the transferred muscle at the desirable level. From these data, Lieber and colleagues[44] built a mechanical device that mimicked the brachioradialis muscle. They then asked a large group of surgeons or varying ages and with varying experience in Br-to-FPL transfers to use the modeling device to mimic the tensioning of the brachioradialis that they would choose at surgery. Essentially all surgeons, regardless of age or surgical experience, tensioned the transfer at a level that exceeded the so-called 'physiologic resting length' of this muscle, the stated goal of most tendon-transfer surgeries. This means that surgeons, regardless of experience, tend to set the tension of tendon transfers so that the transferred muscle acts on the descending limb of its force–length curve, rather than at its optimum position. The result, based upon the computational model of the brachioradialis muscle, was that the muscle as tensioned was capable of producing only 27% of its maximum force (Figure 6.6).

These same surgeons were then provided with a graphic readout of the tension, as well as an optimum tension as determined by the data derived from actual measurements of the passive stretch properties of this muscle. With this clue, essentially all surgeons then set the tension of the mimicked transfer at a more slackened length, corresponding to a muscle capable of producing approximately 45% of its maximum force (Table 6.2).

Since 2001, we have acquired significant experience using the laser myometer in performing tendon transfers. We have determined that this device can be used readily by surgeons (as opposed to only the developer) and that data collection – for example, sarcomere measurements of the undisturbed muscle to be transferred followed by repeat measurement of the donor muscle once the transfer has been performed – does not significantly prolong the operative procedure. This or similar devices, if adapted by surgeons, could provide an objective measure of tension of the transferred muscle. In conjunction with more accurate force–length characteristics of commonly transferred muscles, these devices could permit the surgeon to optimize the procedure for a particular patient's needs.

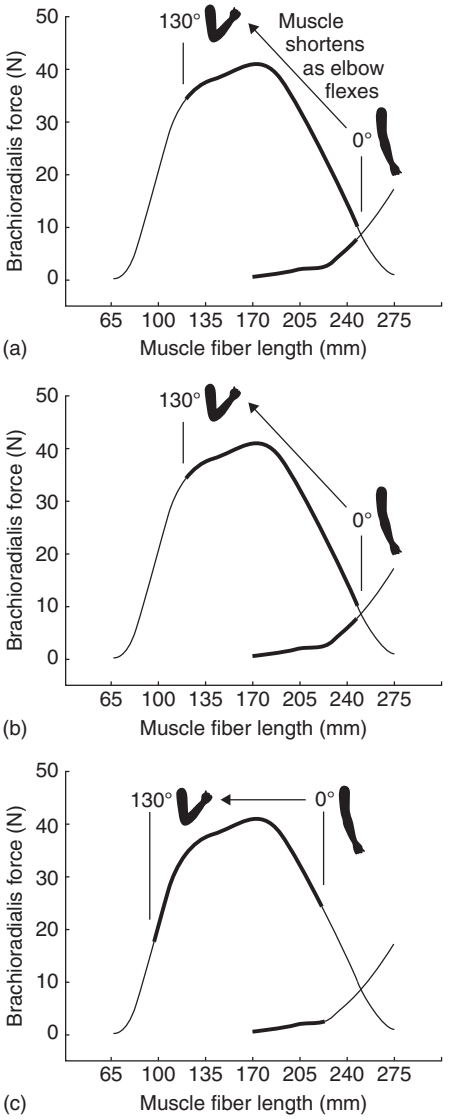

Figure 6.6

Biomechanical simulation of active and passive tension in the brachioradialis muscle as a function of muscle fiber length based on measured sarcomere lengths. A schematic elbow is shown corresponding to specific fiber lengths. (a) Normal brachioradialis range as the elbow is flexed from 0° (full extension) to 130°. (b) Brachioradialis range as the elbow is flexed from 0° to 130° when inserted at a sarcomere length of 3.83 μm, the mean length chosen by the surgeons based only on tactile feedback. (c) Brachioradialis range as the elbow is flexed from 0° to 130° when inserted at a sarcomere length of 3.56 μm, the mean length chosen by the surgeons based on both tactile and visual feedback. (Adapted from Lieber RL et al. J Hand Surg [Am] 2005; 30: 273–82).[44]

Table 6.2 *Brachioradialis muscle force calculated from intraoperative measurements and modeling*

Condition	*Sarcomere length at full extension (μm)*[a]	*Elbow angle of peak active force*	*Muscle force at full extension (N)*	*Mean muscle force from 0° to 130° elbow flexion (N)*
In situ	3.90	94°	Active: 9.7 Passive: 6.9	Active: 27.6 Passive: 2.2
Surgeon's choice (tactile feedback)	3.84	90°	Active: 12.4 Passive: 5.6	Active: 29.2 Passive: 1.7
Surgeon's choice (tactile and visual feedback)[b]	3.56	74°	Active: 24.0 Passive: 1.9	Active: 33.2 Passive: 0.6
Peak force at 130°	4.69	130°	Active: 0.0 Passive: 21.7	Active: 12.3 Passive: 11.1
Peak force at 0°	2.83	0°	Active: 40.5 Passive: 0.0	Active: 27.9 Passive: 0.0

[a]Measured intraoperatively (in situ) or estimated using the surgical simulator or biomechanical model (all others) when the muscle is not active.

[b]This is approximately the sarcomere length that maximizes average muscle force over the range of 0°–130° of elbow flexion.

Adapted from Lieber RL et al. J Hand Surg [Am] 2005; 30: 273–82.

Future rewards from collaborations between surgeons and bioengineers

The majority of tendon transfers are performed on the basis of experience and teaching. Decisions are reinforced by success – yet success has been less than adequately defined, at least in the eyes of the engineer. As more robust and validated mathematical models of the upper limb become available, it will be possible to study the most important parameters of tendon transfers in the upper limb. These tools will allow future surgeons to challenge historically and experientially derived concepts, to refine standard procedures to optimize outcome, and to recognize opportunities for new procedures that may be non-intuitive to current surgeons. Two recent examples from our laboratories will be discussed that highlight these possibilities.

Biomechanical analysis of an established procedure: the Steindler flexor-plasty

As originally described by Steindler, the medial epicondylar origin of the flexor–pronator mass is detached and moved proximally onto the humeral shaft. It is accepted by most surgeons that this transfer must be passively tensioned such that, at the time of surgical tensioning, the transfer 'holds' the elbow flexed against the force of gravity. This means that the transfer is purposefully tightened so that the muscles repositioned by this procedure operate far down the descending limb of their force–length curve. This procedure has been modified by others, including altering the location of the muscle attachment to a site more central than described by Steindler. However, it is unclear how the choice of transfer location affects elbow flexion restoration, especially in light of possible side-effects, including passive effects leading to limitations of elbow, forearm, and wrist motion due to passive restraint imposed by (over)stretched muscles.

A three-dimensional musculoskeletal model of the upper extremity[50] was used to simulate the biomechanical consequences of various surgical alterations. In the case of the Steindler flexor-plasty, analysis of the model showed that elbow flexion strength may potentially be restored to two-thirds of normal; however, passive moments for wrist flexion and for forearm pronation are dramatically increased (to over 6 N m) in elbow extension. The model indicates that active wrist extension strength and forearm supination strength are not adequate to overcome these passive moments. Therefore, the trade-off for increased elbow flexion moment is a restriction on wrist extension and forearm rotation when the elbow is extended. This is what is seen clinically, and our clinical experience validates the model. The model indicates that a small shift inferiorly in the location of the transfer onto the humerus results in essentially the same elbow flexion moment, but almost eliminates the passive wrist flexion and pronation moments with the elbow extended. The model indicates that the location of the transfer is a very sensitive parameter in terms of the passive effects on the wrist and forearm, and suggests that the surgeon does not need to anchor the transfer as far superiorly as has been recommended by

some. Based on these findings, we have begun a clinical study of this issue.

One of the potential outcomes of modeling studies is a better understanding of the effects of varying the technical aspects of even standard tendon transfers by identifying which parameters of a specific procedure are most sensitive to change. For one procedure, this might be the moment at one or another joint; for another, it might be muscle amplitude (e.g., when there is more than one transferable muscle from which to choose). For the Steindler procedure, small alterations in the site of attachment of the flexor–pronator mass result in significant theoretic (by the model) differences in force production and postoperative passive properties. In the future, it may be possible to use evolving methods of assessment (e.g., dynamic MRI or EMG tools) to make a now generic model more 'patient- specific'. Armed with this information, the surgeon will be able to better tailor the procedure to the patient in order to achieve the optimum (for the individual patient) outcome.

Biomechanical analyses resulting in non-intuitive outcomes

We[51] have used a cadaveric/optimization approach to predict and confirm the maximal biomechanically possible fingertip force in the intrinsic palsied hand before and after two currently popular alternative methods of tendon transfer to the volar plate of the metacarpophalangeal (MCP) joint, as described by Zancolli.[52] Both surgeries were also evaluated after release of the A3 pulley – a modification predicted by our computer model of the index finger to increase fingertip force magnitude. We predicted maximal fingertip force by mounting eight fresh cadaveric hands on a frame, placing their index finger in a functional posture (neutral abduction, 45° of flexion at the MCP and proximal interphalangeal joints, and 10° at the distal interphalangeal joint) and pinning the distal phalanx to a 3D dynamometer. We pulled on individual tendons with tensions up to 25% of maximal isometric force of their associated muscle and measured fingertip force and torque output. Using these measurements, we predicted the optimal combination of tendon tensions that maximized palmar force (analogous to pinch force, directed perpendicularly from the midpoint of the distal phalanx, in the plane of finger flexion–extension) for the non-paretic case (all muscles available) and three clinically relevant cases:

(i) intrinsic palsied hand (no intrinsic muscles functioning)
(ii) transfer of the flexor superficialis tendon to the volar plate of the MCP (Zancolli lasso) in the intrinsic palsied hand
(iii) leaving the flexor superficialis intact and transferring a tendon of comparable strength to the volar plate of the MCP in the intrinsic palsied hand.

Lastly, we applied these optimal combinations of tension to the cadaveric tendons and measured fingertip output.

With the A3 pulley intact, the maximal palmar force in cases (i)–(iii) averaged 48%±23% SD (non-paretic=100%; case (iii) (61%±25%) > cases (i) and (ii) (43%±23% and 39%±19%, respectively); $p<0.05$). Releasing the A3 pulley significantly increased the average palmar force in cases (i)–(iii) (73%±42%; $p<0.05$), with no significant differences among them. Thus, releasing the A3 pulley may improve palmar force magnitude when it is necessary to transfer the digit's own flexor superficialis tendon to the volar plate of the MCP to prevent clawing in the intrinsic palsied hand. We now routinely release the A3 pulley as part of the Zancolli lasso procedure.

Conclusion

There is a tendency on the part of hand surgeons to view tendon transfers as a static field. Because the number of transferable upper limb muscles is finite, there is a sense that there is little remaining to be discovered. However, if we hand surgeons collaborate with and adopt the unique analytic tools of our bioengineering colleagues, we have the potential to re-invent the field.

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7 Free functioning muscle transfer

Alain Gilbert and Vittore Costa

In cases of severe muscle paralysis or destruction, there are often few surrounding muscles left for tendon transfers. These are the most common indications for microsurgical muscle transplantation. Muscle transfers have been used for a long time for the coverage of large or infected defects. Although the addition of function in muscle transfer seems logical, it is not easy, as several factors complicate the procedure, such as the presence/absence of a good donor nerve and the tension of the muscle belly.

Tamai et al[1] showed experimentally in dogs that the muscle transplant could remain vital and functioning. Following this, reports of many clinical cases were published.[2–7]

However, more than a quarter-century later, there are very few large series with long follow-up. This may be due to few indications and a certain skepticism regarding this technique.

Muscles used for free functioning transfer

For transplantation of a muscle, several conditions need to be fulfilled:

1. The defect created by removal should be easily filled.
2. It should be a rather long muscle, with, if possible, a tendon on each extremity.
3. It should have only one neurovascular hila, or at least a major one.
4. The excursion of the muscle should be as long as possible, to obtain maximum movement.
5. Its cross-section should be thick enough to produce sufficient force (the maximum tension in mammals is 4 kg/cm^2 of cross-sectional area).

Several muscles have been used.

Gracilis (Figure 7.1)

This muscle is most frequently used. It is long (30–40 cm), with a strong terminal tendon. It is not too bulky, and can be fitted into a limb without additional skin cover. It has a single proximal motor nerve, coming from the obturator nerve, that measures 6–8 cm. The only drawback is its vascular supply: there is a dominant proximal pedicle, but also a secondary pedicle going to the middle part of the muscle and a small distal pedicle. Since this distal pedicle is never necessary, there may sometimes be problems when the middle pedicle is ligated. If, after this ligation, the distal part of the muscle becomes dark (congested), it may be necessary to anastomose the vein from the middle pedicle (Figure 7.2). This has been necessary in 2 cases out of 41.

Pectoralis major

This is a large muscle with a single vascular pedicle; but it has several motor nerves, it is very bulky, and its removal creates a severe defect.

Pectoralis minor

This is small and weak, and has been used only in facial reconstruction. It is difficult to raise, as it is deep to the pectoralis major, but can be isolated on one or two pedicles.

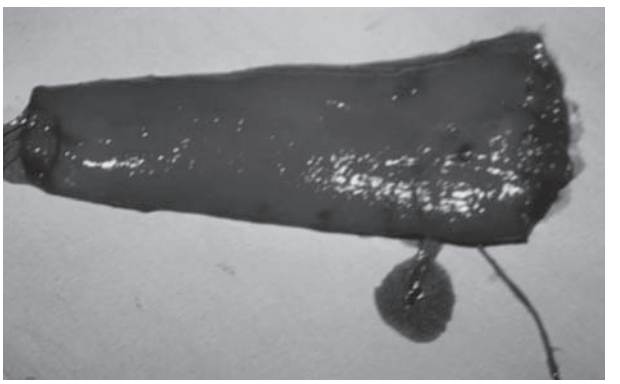


Figure 7.1
The gracilis muscle: the main proximal neurovascular pedicle.

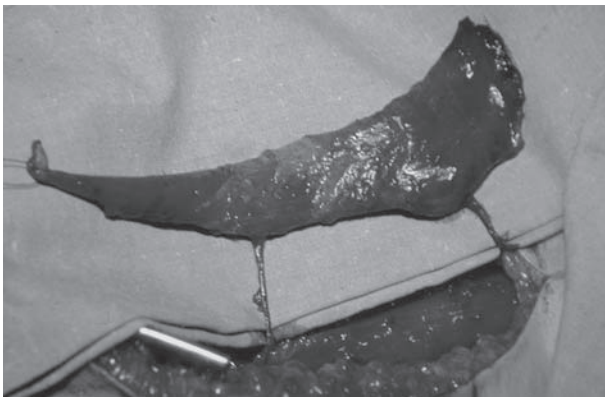


Figure 7.2

In some cases, it is necessary to use the second pedicle.

Latissimus dorsi

This is a very well-known muscle – long, strong, with a single vascular and motor pedicle. Its removal does not create a severe defect. However, some techniques of lengthening the latissimus dorsi with the gluteal aponeurosis allow its transplantation up to the fingers. There are few indications for using a free latissimus dorsi muscle transfer in the upper extremity.

Gastrocnemius

This is a very strong muscle (the strongest in the body), easy to raise, with a proximal neurovascular pedicle. The sural nerve as a vascularized nerve graft (useful in Volkmann's contracture) and/or the overlying skin can be raised at the same time. The defect created is compensated by the soleus. Its main drawbacks are its bulk and very short excursion.

Other muscles that have been used are the rectus femoris (which has several vascular hila), the extensor carpi radialis brevis (too small), and the extensor digitorum brevis from the foot (whose vascularization is very delicate and has been used mainly for the face).

Surgical technique (Figures 7.3 and 7.4)

The surgical technique for gracilis transfer for elbow flexion is as follows. With the patient lying supine, the thigh is prepared, including the pubis area. The muscle can be felt (at least in thin patients) by feeling its contraction during flexion–extension of the knee. This positioning is important if a combined muscle and skin flap is to be used: the area covering the muscle is very narrow, and if the skin incision is not precisely positioned, the vitality of the skin flap may be impaired. Finding the muscle is not always easy, as all the adductor muscles have the same direction and size. However, among these muscles, the gracilis is relatively thin and has a large proximal pedicle.

Once the muscle has been found, dissection is easy. On the medial aspect, the three pedicles can be found:

- The distal pedicle is small and systematically sacrificed.
- The middle pedicle is cut, but with the vessels kept long, in order to be accessible if needed.
- The proximal major pedicle is dissected carefully.

The vascular pedicle is followed until the trunk of the perforator, where the diameter of the artery is 1.5 mm. The nerve has an upward direction towards the inguinal ligament. It originates from the obturator nerve. This motor nerve gives one or two branches to the muscle before the main hila. The existence of these branches that provoke separate contraction of some groups of muscle fibres has led some authors[8] to suture them separately and expect individual contraction to reconstruct a different movement (thumb plus fingers).

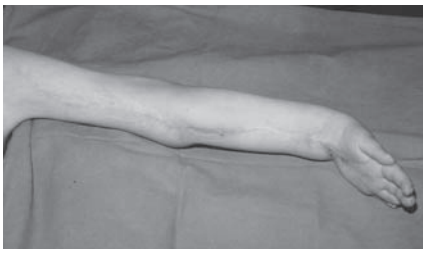
Once the pedicle has been dissected (6–8 cm for vessels, 8–10 cm of nerve), the muscle can be detached; before detaching it, it is important to mark the length with regular stitches, in order to fix it with the exact tension. At this time, it is useful to inject the patient with a muscle relaxant to avoid contraction, after the muscle has been detached. The distal tendon is cut, and, protecting the pedicle, so is the proximal fibrous attachment to the pubis. It is then possible to cut the pedicle and transfer the muscle to the upper extremity.

The muscle is placed in the arm, which is widely exposed. It will be fixed proximally to the coracoid process or to the pectoralis major aponeurosis. The nerve is sutured to the donor nerve, usually the sural nerve placed 1 year before (taken from the pectoralis major nerve on the contralateral side). The size matches well, and there should be no tension. The artery is sutured usually with a branch of the profundus humeral artery; the venous suture connects the vein to a superficial vein. The revascularization of the muscle is assessed, particularly the venous drainage of the distal third. If after 10–15 minutes it is dark and seems congested, the vein from the middle pedicle is sutured. The distal tendon is then fixed with the elbow in acute flexion, using the stitches placed previously to control the tension. It is usually fixed to the biceps tendon. After closure, the elbow is immobilized in flexion for 6 weeks.

Surgical technique for transplantation in the forearm after Volkmann's contracture (Figure 7.5)

There are some differences in the forearm:

- The gracilis is usually too long. It is possible to use another shorter muscle (gastrocnemius or latissimus) or to fix the gracilis higher in the arm.



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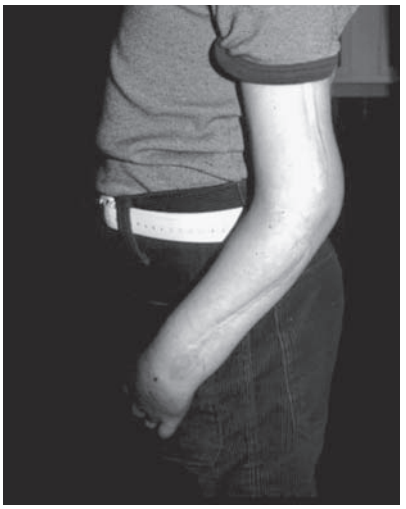

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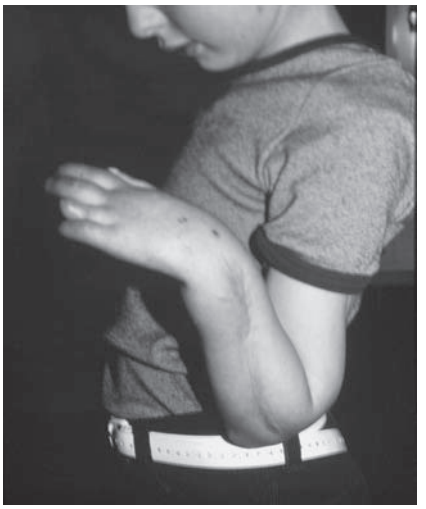
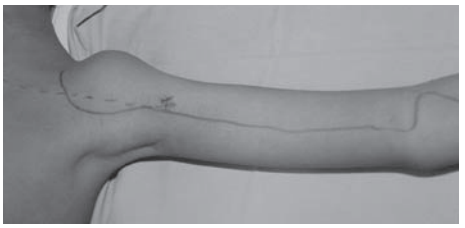


(d)

Figure 7.3

(a) A 6-year-old patient. The arm was previously replanted. The biceps is destroyed. (b) The gracilis is transplanted with neurovascular anastomoses. The nerve from the biceps is used. (c,d) Active elbow flexion after 8 months.



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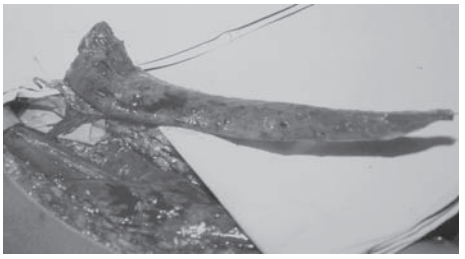

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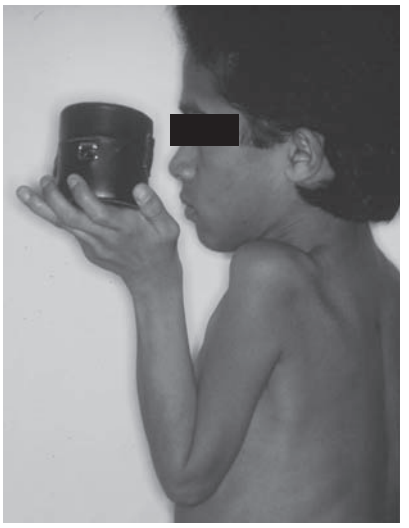


(c)

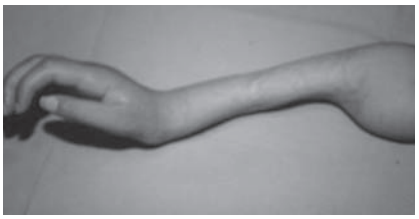
Figure 7.4

(a) Proximal longstanding paralysis of the brachial plexus. (b) Gracilis transplantation on the intercostal nerves. (c) Strong elbow flexion, lifting 2 kg.

- The distal fixation is different. The tendon is sutured to the deep flexors, and a special technique allows simultaneous positioning of the thumb.
- It is usually possible to use a local nerve. The anterior interosseus nerve is often separated at its origin, and can be used.
- A flap may be needed as the forearm is often scarred.

Personal series (Figure 7.6)

Between 1977 and 2007, 54 free muscle transplantations were performed in 53 patients, for various etiologies. Only 31 were carried out following traumatic destruction or paralysis of muscles. One patient had a postoperative



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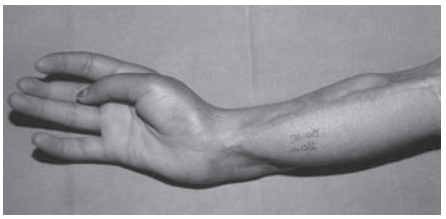

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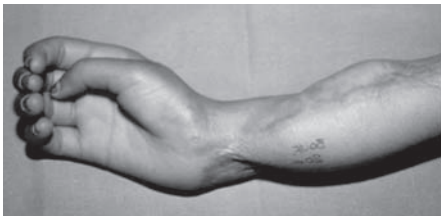
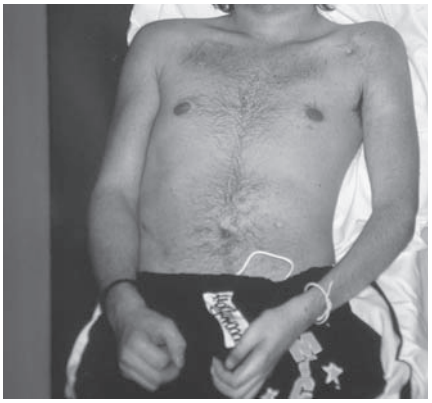


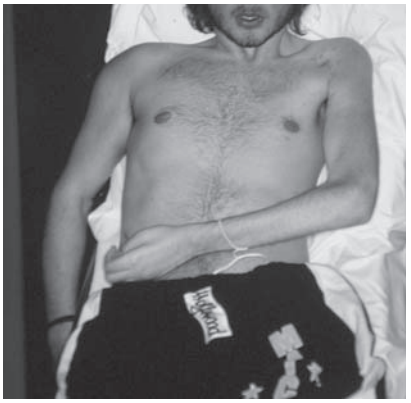
(d)

Figure 7.5

(a) Volkmann's contracture with destruction of the flexor muscles in a 10-year-old child.
(b) Transplantation of the medial gastrocnemius with the sural nerve.
(c, d) Limited flexion–extension.



(a)



(b)

Figure 7.6

(a) A young paraplegic with complete brachial plexus. The gracilis is transferred using cross-chest pectoralis major nerve neurotization.
(b) Elbow flexion after 1 year.

hematoma and necrosis of the muscle; he subsequently had a second gracilis transfer to the arm.

Twenty-four cases were followed up for a maximum of 12.4 years and a minimum of 11 months (average 4.7 years). The etiologies of these were traumatic brachial plexus (n = 36), obstetric brachial plexus (n = 7), and Volkmann's contracture (n = 11). The gracilis was used most often (41 times), but we used also gatrocnemius (three times) and the extensor carpi radialis brevis (once) in the forearm. There were 30 reconstructions of elbow flexion,

12 reconstructions of finger flexion, and 2 extensor reconstructions. The criteria used for assessment were joint ROM and MMT, and the modified scale for end-result evaluation.[9,10]

The results showed that 31 patients had muscle function >M3 and 13 had <M3. The results are easier to assess in the arm for an elbow reconstruction secondary to paralysis than in a forearm after Volkmann's contracture. In the latter case, the associated nerve paralysis, intrinsic wasting, and stiffness will have a deleterious effect on the result, even with excellent muscle contraction.

For elbow flexion reconstruction, the donor nerve used was the upper pectoralis nerve from the contralateral side (23 times) and the sternomastoid nerve from the same side (twice). Both of the latter had poor results with weak muscle contraction and no function. The contralateral pectoralis nerve was used in two stages. In the first stage, the upper nerve was isolated and sutured to the end of the sural nerve. The nerve was passed subcutaneously, anterior to the chest, to the upper arm. At this level, a small incision allowed the nerve end to be found and marked with a metal clip. After 3 weeks of immobilization, the progression of nerve regeneration was followed with the Tinel sign. When tapping the nerve over the chest, the patient felt a slight tingling over the thorax on the donor site. Usually, after 1 year, the regeneration was considered complete, and the second stage of muscle transplantation was possible.

For reconstruction of finger flexion or extension, the anterior interosseus nerve was used in seven cases, and a contralateral graft from the pectoralis nerve was used in two.

Discussion

Since the first reports of clinical muscle transplantation in 1976, a few series have been published. In 1988, Manktelow,[9] using mostly the gracilis, reported the results of his first 12 transfers for the forearm. Ten of these cases had good results. Akasaka et al[11] in 1991 showed that in 17 cases of rectus femoris transplantation for elbow flexion, 8 of the 11 cases assessed had resulting function >M3. In wrist extension surgery, they had performed 29 transfers in conjunction with elbow flexion. They found that 9 cases had regained M3 function after 1 year. Chuang et al[12] stated that in a series of 17 patients, using intercostal nerve transfers, 7 had good results (>M3).

Grotting et al[13] used the gracilis and tensor fascia lata in 12 patients, with satisfactory results (M4) in 11 cases. Berger and Brenner[14] used a free latissimus dorsi (8 times) for elbow reconstruction after brachial plexus paralysis. They found an average of 1–2 kg of strength.

Doi et al[15] suggested using free muscle transfers for the combined reconstruction of two functions; i.e., elbow flexion and finger flexion, or elbow flexion and wrist extension. They operated on 46 patients (58 muscles), of whom 31 had had post-traumatic loss. The donor nerves were the accessory nerve or intercostal nerves. They claimed that with a double-muscle transfer or a double-function, single-muscle transfer, the results are good, allowing useful function in completely paralyzed patients. Ercetin and Akinci[16] showed that in transplanting gracilis muscle for Volkmann's contracture, they could obtain consistently good results.

Although these series are few in number, they all demonstrate the feasibility of vascularized muscle transfer. The results vary from 40% to 70%. Useful results were achieved depending on various factors, such as:

- A good donor nerve is necessary. In cases of brachial plexus paralysis, several extraplexal neurotizations (sternomastoid nerve, intercostal nerves, or contralateral C7 or pectoralis nerve) have been used. These nerves cannot bring axonal influx of the same quality as an anterior interosseus nerve, or a musculocutaneous nerve.
- Sensation in the hand is very important, not for recovery of the motor nerve but for its use.
- Associated nerve lesions can impair the result. In finger flexor reconstruction, the addition of ulnar nerve paralysis, with claw and lack of thumb adduction, will not allow good function.
- Provided that these problems can be avoided, the procedure is reliable and can give some very good results.

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8 Biomechanics of the shoulder

René Malek

The shoulder is the anatomic region which binds the upper limb to the trunk. It is formed by three bones: the scapula, the clavicle, and the upper extremity of the humerus, which constitute the scapular girdle. These bones are connected by three joints, only one of which is articulated with the trunk – the sternocleidal joint – the other two being the acromioclavicular joint and the glenohumeral joint, which is often called the shoulder joint.

The muscles of the shoulder are very numerous. They can be divided into three anatomic groups:

- the first one includes the muscles joining the head and the neck to the scapular blade
- the second unites the scapula to the other bones of the scapular girdle
- the third one includes all the muscles going from the scapular girdle to the humerus.

Each muscle has an action described by the anatomists which involves the narrowing of their two extremities by contraction of the fibers, but this does not correspond to the reality of a very complex biomechanics. In fact, all the muscles are in action in each movement of the upper limb, some to obtain the movement wanted and some to maintain stability and avoid a 'parasite' motion. The radial disposition of the muscles explains the formation of couples of forces which become difficult to quantitate.

Anatomy

Joints

The sternoclavicular joint

The sternoclavicular is a 'saddle' joint similar to the trapeziometacarpal joint of the thumb. It can move in all directions: up, down, forward, and backward, with some rotational possibilities. For certain authors it evokes a Cardan's shaft, but in fact only the clavicle is the mobile element.

The two surfaces in contact are not of the same size and therefore a meniscus exists to improve their adaptation. This meniscus divides the articular cavity into two parts, and by its elasticity gives the possibility of rotation instead of a solid attachment by the capsula and ligaments (Figure 8.1).

The superior ligament is strong and is reinforced by the strong interclavicular ligament. Another ligament also exists, which is of note because it is located far from the joint. This costoclavicular ligament, conjointly with the subclavius muscle, holds the clavicle firmly to the first rib as a 'shroud', nearly displaced from the center of mobility. In spite of these bonds, the amplitude of movement of the clavicle is important. Measured at its lateral extremity it can reach 10 cm upwards and also forwards, and 3 cm backwards until the bone comes into contact with the first rib (Figure 8.2). This mobility is essential to allow movements of the scapula.

The acromioclavicular joint

Less mobile than the preceding joint, this joint also plays a far from negligible role. It is an arthrodia (allowing only

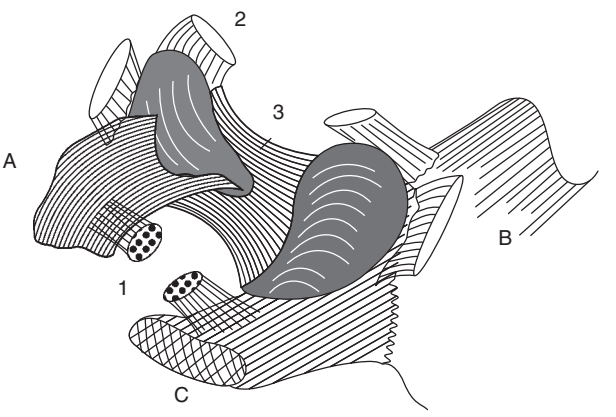

Figure 8.1

The sternoclavicular joint: A, clavicle, B, sternum, C, first rib; 1, the costoclavicular ligament; 2, the superior ligament; 3, the posterior ligament.

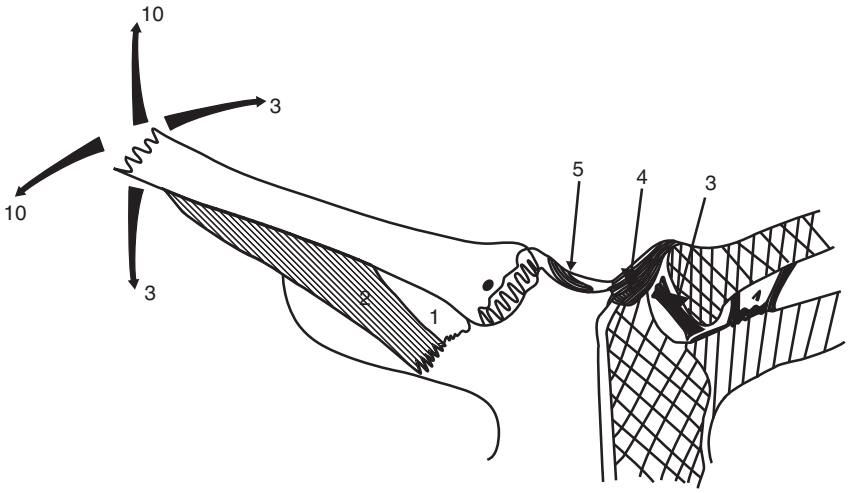

Figure 8.2

The sternoclavicular joint: 1, the costoclavicular ligament; 2, the subclavicular muscle; 3, the meniscus of the joint; 4, the sternoclavicular ligament; 5, the interclavicular ligament. On the left are the amplitudes of the movements of lateral extremity of the clavicle, measured in cm. Adapted from Kapandji.[1]

gliding motion) and the surfaces in contact are almost flat. The clavicle seems to lay on the acromion through a fibrocartilage. There is a strong superior ligament, but the bones are maintained by a number of ligaments located far from the joint which arise from the coracoid process: the conoid and the trapezoid ligaments. The conoid ligament is triangular with an inferior summit, and arises from the angle of the coracoid. The trapezoid ligament is quadrangular, forms a dihedral angle with the former, and arises from the medial border of the horizontal part of the coracoid process. These two ligaments insert on the inferior side of the clavicle. Their orientation restrains the 'rocking' movements of the scapula, but allows movement along two axes – vertical and horizontal. They also restrain the movements of the clavicle downwards. A third ligament can be added, which unites the medial border of the coracoid (in front of the trapezoid) and the medial border of the clavicle. This is Caldani's 'cocked-hat' ligament (Figure 8.3).

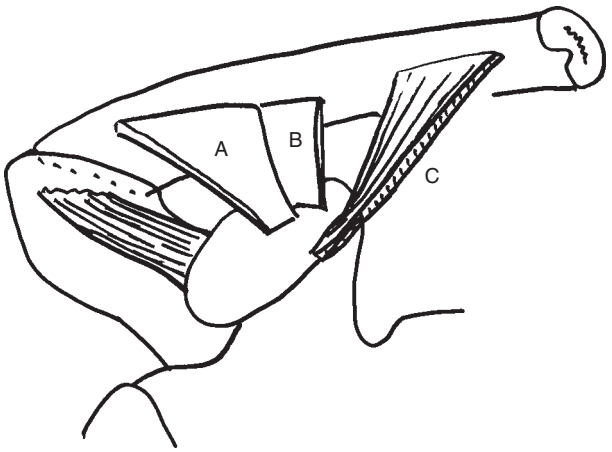

Figure 8.3

The coracoclavicular ligaments: A, trapezoid ligament; B, conoid ligament; C, Caldani's ligament.

Movements of the acromioclavicular joint, in spite of their short amplitude, have a physiologic importance. The clavicle and the scapula form an angle opened inwards. The summit of this angle (normally 60°) is situated at the acomioclavicular joint. The two bones resemble the needles of a compass. When the shoulder is elevated the value of the angle diminishes. In contrast, it increases when the shoulder is in a low position. This mobility allows a good, permanent application of the scapula on the ribs, and is only possible owing to the gliding movements of the acromioclavicular joint (Figure 8.4). On the other hand, as the upper part of the thoracic wall is round while an acute angle exists between the scapula and clavicle, the summit of this angle is kept apart from the ribs. This maintains a space behind the clavicle, allowing the passage of nerves and vessels on their way to the upper limb.

Finally, the proximal muscles of the limb have their insertions on the clavicle and on the scapula independent of the thorax.

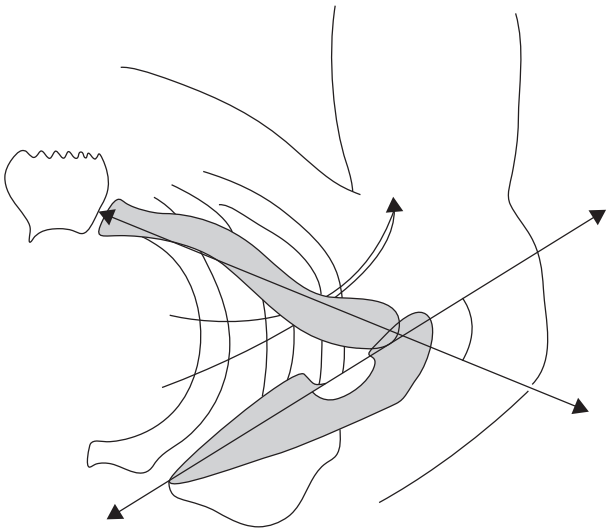

Figure 8.4

The variations of the angle between the clavicle and the scapula are possible owing to the mobility of the acromioclavicular joint.

The scapula

These two small joints that have been described are essential for the mobility of the scapula. This bone has a triangular

form, with an inferior summit, and can be considered as an intramuscular bone serving for the insertions of many muscles of the shoulder girdle which cover its two faces. Indeed, it enters into contact with the thoracic wall only by its medial border. It can glide on it owing to the existence of loose cellular tissue between the ribs and the anterior side of the serratus lateralis muscle, forming the thoraco-serratus space. The scapula is applied on the posterior and lateral faces of the thorax by the contraction of many other muscles.

Movements of the scapula are important and depend on the two articulations studied previously. At rest the scapula blade forms an angle of 30° with the anatomic frontal plane and the glenoid surface faces anteriorly. The medial border of the scapula is located at 5 or 6 cm from the spinal line, but this distance can increase to 15 cm by a sliding movement. With respect to the thoracic wall, the bone lies between the 2nd and the 7th ribs. It can move up and down within 10 to 12 cm (Figure 8.5).

Finally, it can rotate along an axis which is perpendicular to its own plane (pendulum movement) and the range is 60° on each side of a vertical line. This movement leads to a modification of the orientation of the glene, which looks downwards or upwards as well.

The gleno-humeral (or shoulder) joint

The scapulohumeral joint is considered as the most important joint of the scapular girdle. It is a ball and socket joint uniting the humeral head to the glene of the scapula. The movements of this articulation are difficult to distinguish from the movements of the other joints which are always associated with them. Many structural constraints limit the range of mobility, which remains very broad in all directions.

The joint surfaces The humeral head corresponds to one-third of a sphere. It is slightly flattened from front to back. The glene of the scapula which articulates with it is smaller, but adaptation is given by the fibrocartilage on its periphery. In spite of this the fit is not perfect and explains the frequency of dislocation.

Movements of the shoulder have three degrees of freedom (in all the spatial planes) thus there is no single center point. Moreover, the humeral head does not have a permanent position, and thus there are many axes or centers of motion. Some authors maintain that all these centers are located in a small sphere inside the head itself. However, this is irrelevant.

The humeral head is covered by the acromion and is kept apart from it by the suprasupinatus muscle, which, surrounded by a serous sheath, passes in a tunnel formed by the superior pole of the glene and the root of the acromion at its inferior part, and which is closed on the top by the acromion itself and by the acromiocoracoid ligament.

The glene is grooved at the superior and lateral angle of the scapula blade. It is concave in the two directions, but the radius of this concavity is superior to the radius of the convexity of the head, which is much larger.

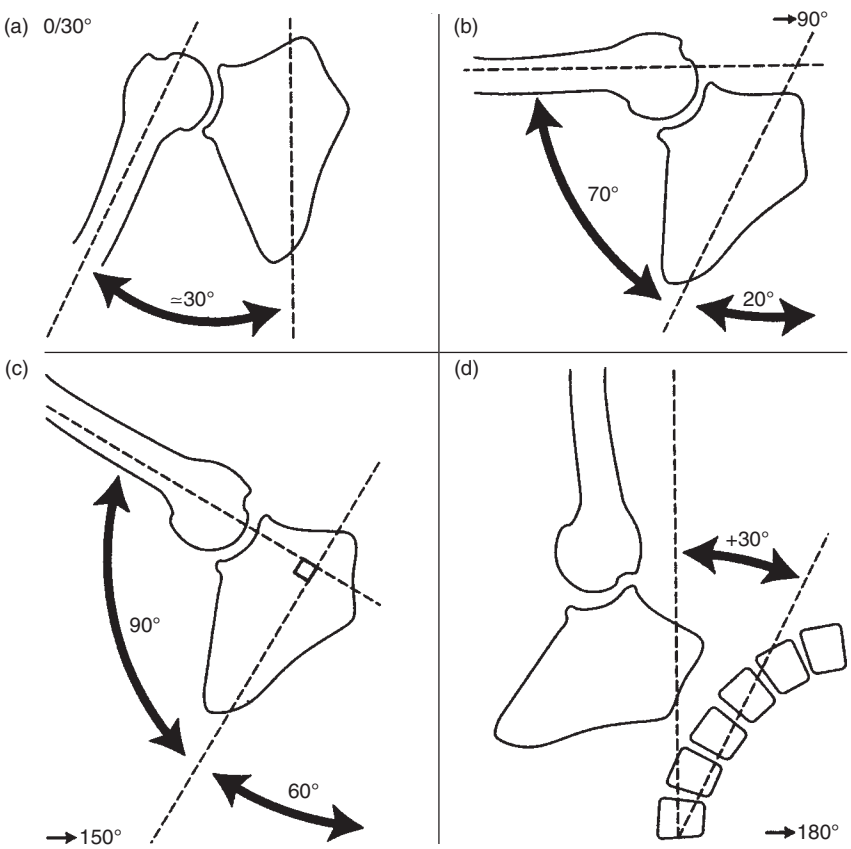

Figure 8.5

The abduction movements of the shoulder: (a) the first 30° are obtained in the scapulohumeral joint; (b) up to 90° of abduction are obtained in the scapulohumeral joint, and up to 20° in the scapulothoracic joint; (c) up to 150° more in the scapulothoracic joint (the last 60° of which is enabled by the mobility of the scapula); (d) more than 150° of abduction obtained by vertebral inclination. Adapted from Delamarche et al.[2]

The surrounding borders are significant: the anterior border presents the glenoid notch. At its superior pole is the supraglenoidal tubercle, from which arises the long biceps tendon. At its inferior pole is the infraglenoidal tubercle, corresponding to the insertion of the tendon of the long head of the triceps brachii muscle.

The fibrocartilage (glenoid labrum) tends to adapt the two surfaces of the head of the humerus and the glene of the scapula. Its transversal section is triangular and the cartilage which covers its surface is in continuity with the glenoid cartilage only at its inferior part. At the upper part it looks like a true meniscus, and it crosses the anterior notch of the glene border. It is reinforced by some fibers from the adjacent tendons of the biceps and the triceps muscles.

The restraining structures Coaptation of the joint surfaces is obtained with the following factors:

- Not by the capsula, which is remarkably loose and lax, larger than the cartilage zone, and extends from the base of the coracoid process to the anatomic neck of the humerus, where it presents some recurrent fibers called the 'frenula capsulae'. The capsula is reinforced by the adjacent muscles, but often presents gaps.
- The passive ligaments are (Figure 8.6):
  - the three glenohumeral ligaments, which are anterior, superior, and inferior, representing a letter 'Z', from the border of the glene and the fibrocartilage to the lesser tubercle and the surgical neck of the humerus
  - the coracohumeral ligament, which is more solid, and arises from the borders of the horizontal and vertical parts of the coracoid and divides to end on the two tubercles managing the entrance of the tendon of the long biceps
  - it is noticeable that there is no posterior ligament.
- Coaptation is mainly maintained by the muscles all around the joint. They are so-called 'active' ligaments.
- Lastly there exists an important factor of restraint, which is a vacuum inside the joint. This vacuum helps to compensate for the weight of the upper limb, which exerts a tensile stress on the joint.

Muscles

A great number of muscles exists at the level of the scapular girdle and they are all necessary to give proximal stability and to obtain movement of the upper limb. Some of them counter gravitational forces, others oppose the axial forces exerted on the bones, leaving to the resultant forces the possibility of movement.

It is essential to remember that all the muscles around one joint are active simultaneously, along one of three patterns of activity, i.e. isotonic, isometric, or with elongation. The particular disposition of the shoulder muscles moreover explains the existence of torque forces between most of them. As the movement changes the situation of the bones, the forces applied on them must change continuously.

To facilitate the understanding of shoulder muscle physiology it seems necessary to study successively three groups:

1. muscles which involve the head, the neck, and the scapular girdle

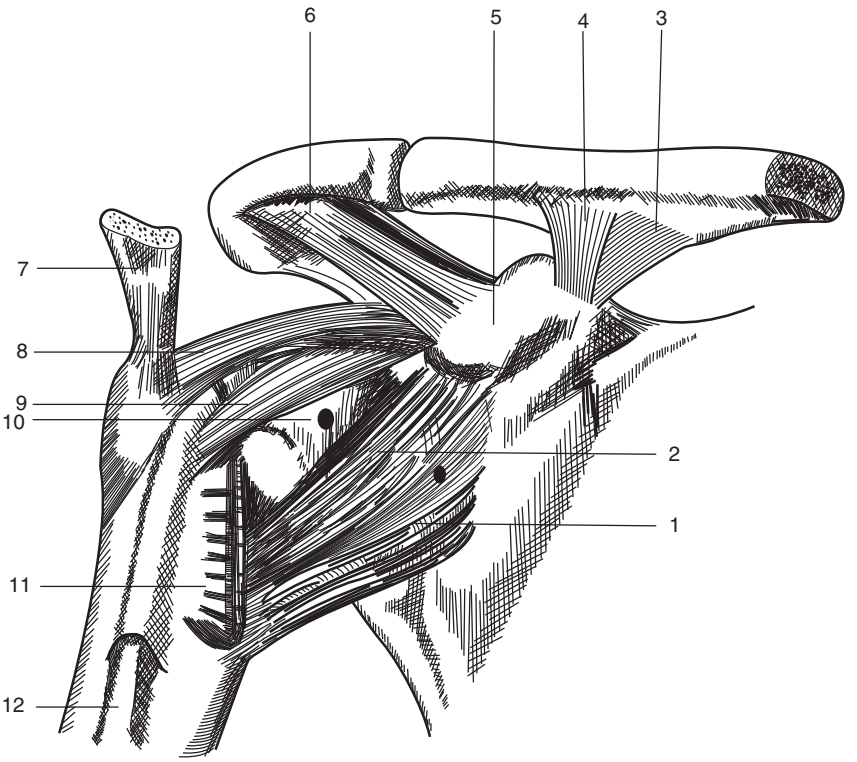

Figure 8.6

Anterior view of the shoulder and ligament complex: 1, inferior glenohumeral ligament; 2, medial glenohumeral ligament; 3, coronoid ligament; 4, trapezoid ligament; 5, coracoid process; 6, coracoacromial ligament; 7, supraspinatus; 8, coracohumeral ligament; 9, superior glenohumeral ligament; 10, humeral head; 11, subscapularis; 12, long head of biceps. The two black points show the dead spaces. Adapted from Tubiana and Camadio.[3]

2. muscles inserted on the scapula, the clavicle, and the thorax (ribs and spine)
3. finally, muscles joining the scapular girdle and the humerus.

Muscles involving the head, neck, and scapular girdle

Backwards there are two muscles:

- The *superior tract of the trapezius*, which arises from the superior line of the occipital bone (third medial) and from the cervical ligament (from the external occipital to the processus spinalis of the 7th vertebra). This tract ends on the lateral third of the posterior border of the clavicle and elevates the clavicle and the scapula.
- The *levator scapularis* which is a long, thin muscle arising from the upper-medial angle of the scapula blade and ending, by tendinous bundles, on the transverse processes of the first four cervical vertebrae. It also elevates the shoulder (Figure 8.7A).
  The two muscles incline the neck on their side when the arm is fixed.

Forwards the *sterno-cleido-mastoideus* has two heads inserted on the clavicle:

- one arises from the medial third of the superior face of the clavicle by muscular fibers and inserts on the two lateral thirds of the occipital superior line
- the other arises from the clavicle behind the former, and ends on the mastoid process.

They are distinct from the strong sternal tendon of the muscle and have a role in the elevation of the clavicle.

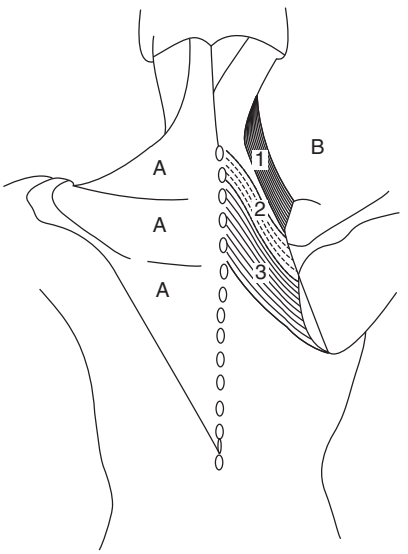

Figure 8.7

A, The trapezium and its three heads, upper, intermediate, and inferior. B, The deep layer with: 1, the levator scapula; 2, the rhomboideus minor; 3, the rhomboideus major.

Muscles inserted on the scapula, the clavicle, and the thorax (ribs and sternum)

These are divided into two groups: deep and superficial. They all have the same function: to move the scapula, which is the mobile area of their insertions, or, in contrast, to fix the bone to allow movements at the lower part of the limb.

The deep group *Anteriorly and laterally* this includes three muscles forming a radiant layer which is almost continuous around the medial border of the scapula. These are as follows.

- Toward the medial line the *great rhomboideus* arises from the 2nd, 3rd, 4th, and 5th spinal processes, and from the supraspinal ligament. It ends on a tendinous arch extending from the triangular area of the scapular spine to the inferior angle of the scapula
- In a higher location the *little rhomboideus* arises from the inferior part of the septum nucchae and from the spinal processes of C7 and D1. Its fibers go obliquely to insert on the spinal border of the scapula, under the triangular area (Figure 8.7B).
- On the spinal border of the scapula arises the *serratus anterior*, the fibers of which, like a fan with as many bellies and tendons, reach the first 10 ribs. Its penniform aspect is characteristic and allows each belly to contract separately. It is usual to describe three parts of this large muscle, with somewhat different functions:
  - a superior one arising in the upper and inner angle of the scapula and ending on the second, and sometimes the first ribs
  - an intermediate part arising from the spinal border, extended to the 2nd, 3rd, and 4th ribs
  - and the inferior part, arising from the medial and inferior area of the anterior surface of the scapula and ending on the 5th to 10th ribs.

  Its fibers intercross with one of the oblique descendens muscles (Figure 8.8a). The thoraco-serratic space arises at the anterior surface of this muscle, which can contract freely (Figure 8.8b). Each of the three portions forms torque forces with the muscles described on the spinal border of the scapula.

This border can glide in the two transversal directions and also up and down. As already said, there is also a possibility of swinging movements around an axis perpendicular to the body of the bone. All these movements change the orientation of the humeral glene, and consequently the range of the limb movements.

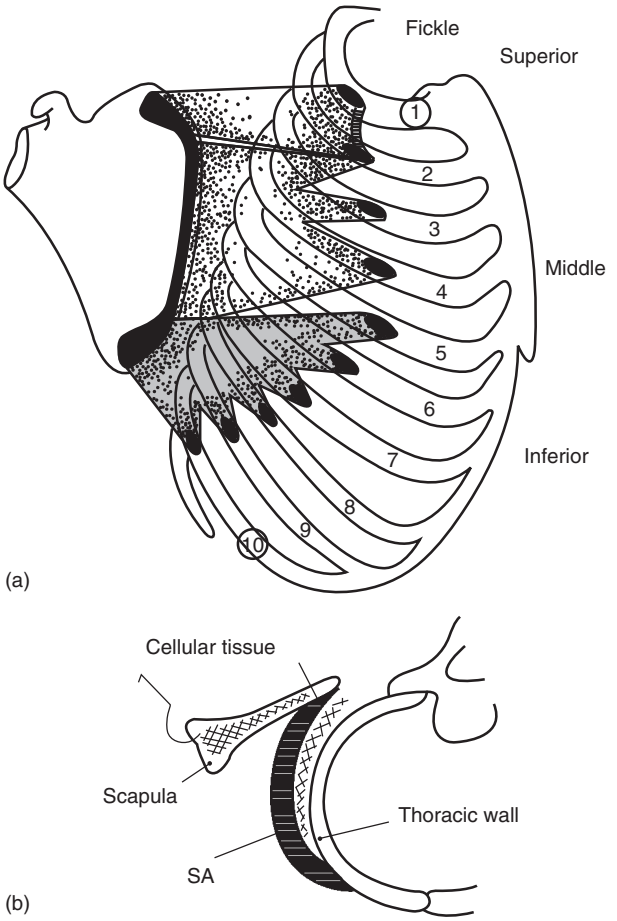

Figure 8.8

The serratus anterior, (a) and its three heads superior, middle, and inferior; (b) the thoraco-serratic space. SA, serratus anterior. Adapted from Brizon and Castaing.[4]

Anteriorly there are two deep muscles:

- *pectoralis minor*, extending from the coracoid process to the 3rd, 4th, and 5th ribs; when it acts on the coracoid this muscle lowers the scapular girdle and brings it forward
- the *subclavius muscle*, arising from the inferior surface of the clavicle and ending on the first rib; it lowers the clavicle and then the scapular girdle.

The superficial layer This is only represented by the *trapezium* muscle and, more precisely, by its intermediate and inferior heads, which arise from the spinal apophyses of the 10 or 12 dorsal vertebrae, and from the ligaments joining them. The upper fibers have a triangular disposition, the intermediate are transverse from the acromion to the lateral portion of the spine of the scapula, and the inferior fibers are oblique, ascending and ending on a tendinous lamella which glides on the triangular area at the beginning of the spine and, passing under the former intermediate fibers, ends on the inferior part of the lateral border of the spine. Evidently the three parts of the trapezium do not have the same action: they can elevate or lower the scapula. The trapezium is not essential for the movement of 'shrugging' the shoulder: the levator scapulae is the most important muscle for this action.

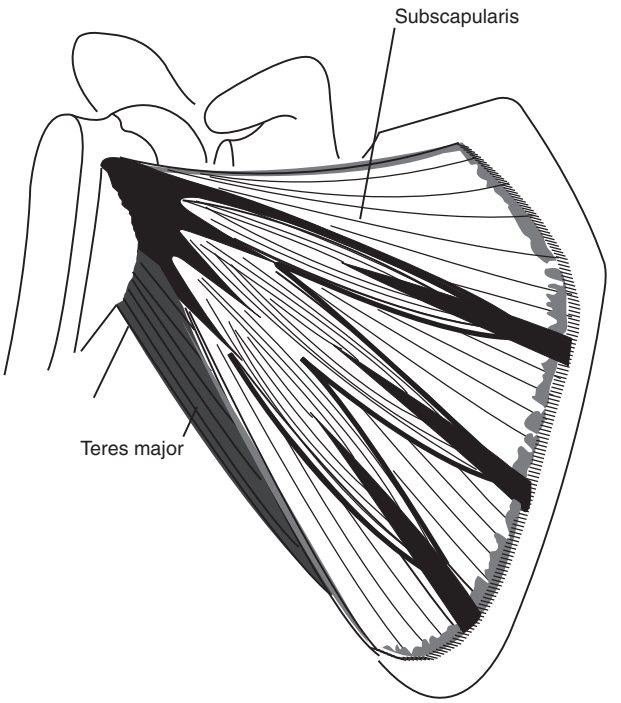

Figure 8.9

The subscapularis muscle and its penniform anatomy. Adapted from Brizon and Castaing.[4]

Muscles uniting the scapular girdle and humerus

These are also divided into a deep layer and a superficial layer.

The deep layer This can itself be separated into two differents parts: one in which the bellies arise from the scapula, and the other where fibers arise from the humerus:

Group 1

Anteriorly is the *subscapularis* muscle, which has a penniform aspect, with as many insertions as the numerous crests of the anterior face of the scapula. It then inserts on the lesser tubercle of the humerus. Its action is variable because it can be an abductor or an adductor, depending on the position of the humerus, and also on the situation of the little tubercle with regard to the center of movement of the humeral head (Figure 8.9).

Posteriorly, up to the spine and arising from the supraspinatus fossae, is the *supraspinatus* muscle, which passes into the tunnel already described and ends in the higher impression of the great tubercle of the humerus. Its tendon is intimately adherent to the capsula of the shoulder joint.

Down the spine are:

- The *infraspinatus muscle*, also of a triangular form, which arises by muscular fibers from the medial two-thirds of the infraspinal fossae and by tendinous fibers from the ridges of the fossae. It ends in the middle impression of the great tubercle.
- The *teres minor* is a narrow muscle arising from the dorsal surface of the axillary border of the scapula and from the aponeurosis, which separates it from infrasupinatus and teres major. It inserts on the inferior impression of the great tubercle, and on the shaft of the humerus below.
- The *teres major*, on the contrary, is thick and somewhat flattened. It arises from the oval area of the dorsal surface of the inferior angle of the scapula. Obliquely the fibers end in a flat tendon inserted into the crest presented on the lesser tubercle lying behind the tendon of the latissimus dorsi.
- The *latissimus dorsi* can be described with all the preceeding muscles becauses it sometimes has insertions on the scapula (Figure 8.10).

The 'shoulder cap' (or rotator muscles) is formed by the muscles which are in contact with the head of the humerus, i.e., the suprasupinatus, infrasupinatus, teres minor, and subscapularis muscles. Their four tendons are partially fused and adhere to the capsula. This is why they are said to be 'active ligaments' of the joint. Indeed, they play an essential role because they represent opposition to the dislocating forces generated by the deltoideus and the muscles of the arm. The shoulder cap is separated from the acromial vault by a serous sheath (subacromial and subdeltoideus).

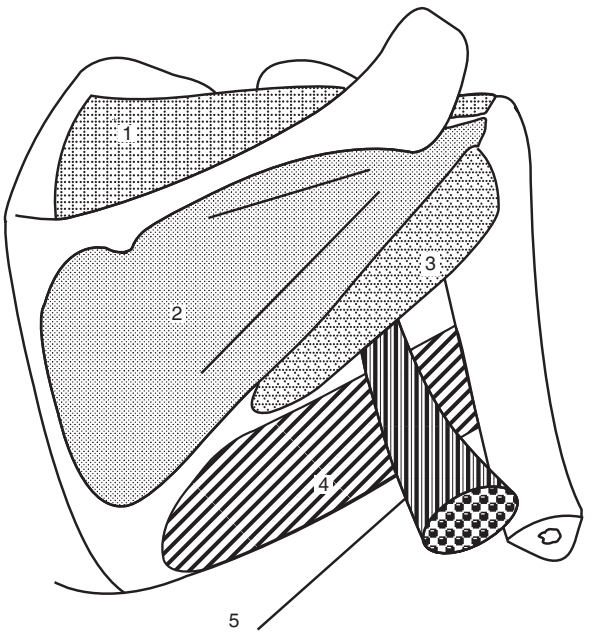

Figure 8.10

Muscles uniting the scapular girdle and the humerus (deep layer): 1, the supraspinatus; 2, the infraspinatus; 3, the teres minor; 4, the teres major; 5, the latissimus dorsi.

Group 2
Two muscles which have their insertions on the humerus and their tendons ending on locations between the anterior and the posterior part of group 1. They are:

- The *long head of the biceps brachii*, which passes through the intertubercularis groove and, turning at a right angle, ends at the superior pole of the glene. Usually it is in this portion, inside the capsula but surrounded by a mesotendon given by the synovial membrane, kept out of the joint cavity.
- The tendon of the *long head of the triceps*, which is extra-articular and ends at the inferior part of the glene.

The superficial layer This is represented by two muscles:

- The *pectoralis major* arises around the girdle, from the clavicle, from the sternum, and from the upper part of the sheath of the rectus abdominis muscle. It is divided into three parts, superior, intermediate, and inferior, which unite in a strong tendon ending on the lateral ridge of the bicipital groove. It is mainly an adductor of the arm (Figure 8.11).
- The deltoideus is the muscle which gives, by its volume, the outline of the shoulder. It also has a wide origin and three heads:
  (a) the clavicular head arises from the lateral third of the border of the clavicle
  (b) the acromial head inserted on the inferior border of the apophysis, and
  (c) the scapular head which arises from two-thirds of the lateral part of the spine of the T scapula.

  The deltoideus is formed by multiple bundles, giving it a penniform aspect, which corresponds to the variability of its action. The three parts unite in a thick tendon which is inserted into the deltoid prominence on the middle of the lateral side of the shaft of the humerus. There it gives an expansion to the deep fascia of the arm. The intermediate head is unique in texture, with vertical tendinous intersections in the acromial part, bipenniform fibers, and again intersections reaching the tendon. Schematically, the deltoideus acts as an abductor of the shoulder and also produces anterior or posterior movement (Figure 8.12).

There is an aponeurotic continuity between the trapezium, deltoideus, and pectoralis major, and this holds all the structures of the shoulder.

Biomechanics

After this recall of the anatomy of the shoulder, showing the extraordinary complexity of the muscle disposition, a consideration of the dynamic physiology will follow.

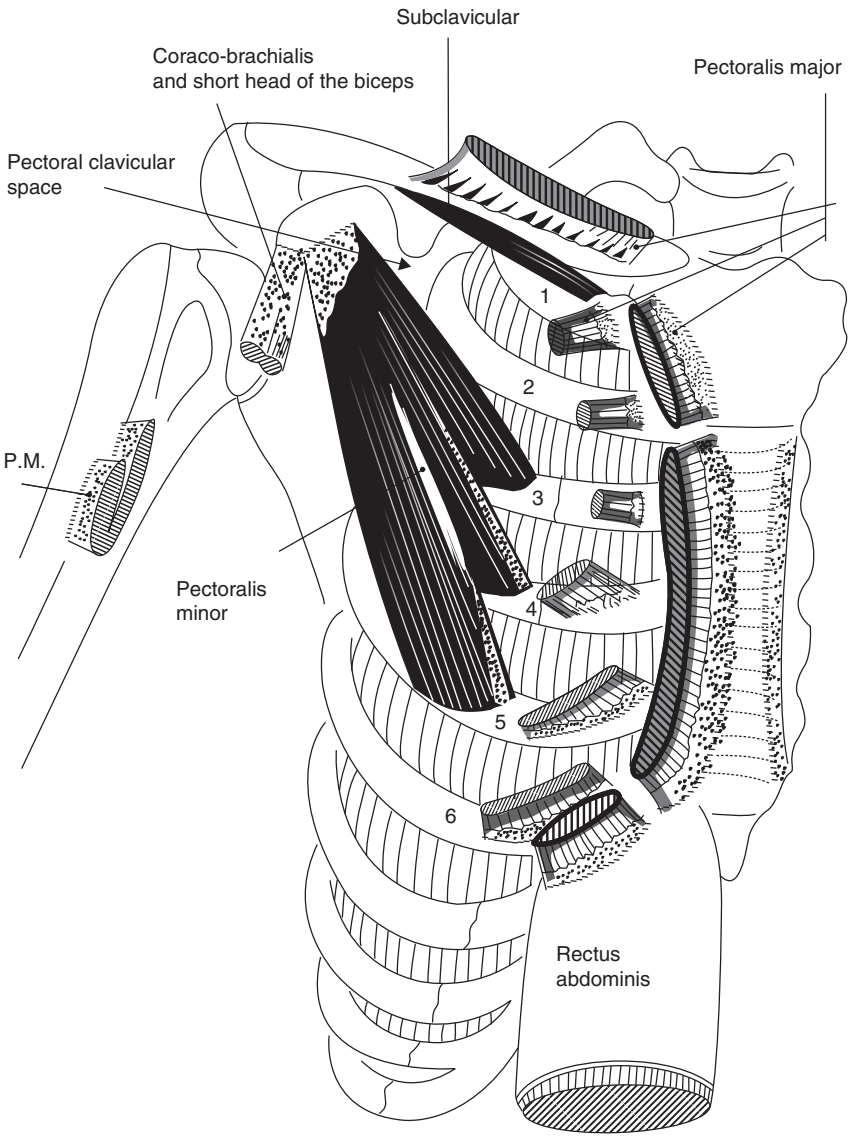

Figure 8.11

Insertions of the pectoralis major (PM), which covers the pectoralis minor. Adapted from Brizon and Castaing.[4]

Once again, it is necessary to stress the fact that all the scapular movements are linked, that all the muscles are more or less active, and that all the bones move together.

The final purpose of the scapular girdle system is to position the upper limb in space. The glenohumeral is the more mobile joint. Owing to the mobility of the bones, most of the muscles do not have fixed insertions, except the one attached to the spine and the skull. Then the range of mobility is eminently variable and it is quite impossible to determine axes or centers of any displacement.

It has been already shown that the action of one muscle can change with the position of the bone on which it is inserted, sometimes in a completely reverse direction. Torque forces realized by two or more muscles are not studied in the anatomic classical definition of actions.

Finally, in the same muscle predominant active fibers (see Chapter 5) can give a variable direction of a force generated by the muscle, and there is also an influence of the external resistance to the movement, which can radically change the resultant of the forces.

A live biomechanic study is therefore very difficult, or even impossible. Nevertheless, a clear understanding of the movements needed to reach a defined position can be obtained.

Usually movements of the shoulder are described in relation to the anatomic frontal plane (abduction and adduction) and to the sagittal plane (anteposition or retroposition), and, when the arm is maintained vertical with the elbow into contact with the thorax, in terms of rotation (lateral or medial).

A passive amplitude allows a good appreciation of the glenohumeral joint status. An active range of movement is more difficult to describe, and it can be quite different from the passive appreciation, because it appears in one very specific situation at the level of the shoulder called the 'functional arthrodesis'. Indeed, when one or more of the muscles around the glenohumeral joint is failing the balance is lost, and the patient tries to perform the movement with the scapula, and to by-pass the shoulder.

For muscle testing in particular, some points should be made about abduction, rotation, adduction, and retroposition.

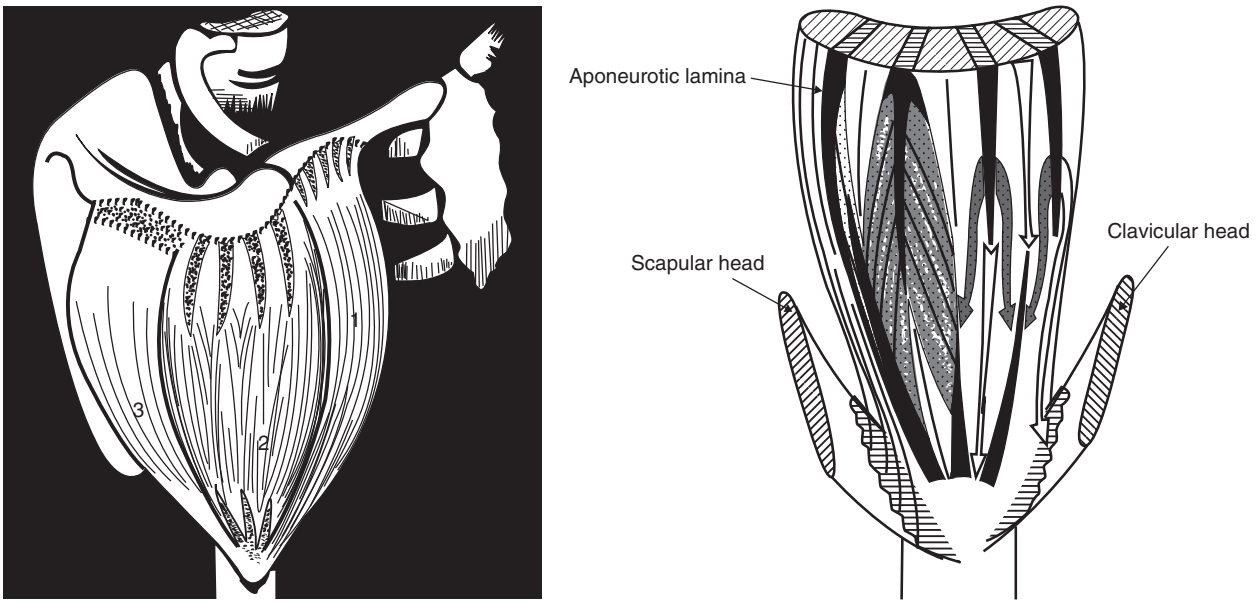

Figure 8.12

The deltoideus. (a) The three heads. 1, anterior, clavicular; 2, middle or acromial; 3, posterior or scapular. (b) The penniform aspect of the fibers of the acromial head. Adapted from Brizon and Castaing.[4]

During *abduction*, the deltoideus, the suprasupinatus, the trapezium, and the serratus major are involved. Many works have attempted to define the respective role of the deltoideus and the supraspinatus at the beginning of abduction. The latter is usually called 'the starter'. This role must be explained. The two muscles are always working together and the action of the deltoideus prevails. However, when the arm is vertical the intermediate head of the deltoideus generates a very strong axial force which can elevate the head of the bone (Figure 8.13). Dislocation of the joint is avoided not only by the resistance of the capsula and of the superior tissues, but by the force exerted by the supraspinatus which, horizontally, maintains the head in place, associated with the tendon of the long head of the biceps. These muscles combat the axial force of the deltoideus and their torque is an essential couple for abduction.

It is thus easy to understand why abduction is difficult when the supraspinatus is disabled (cap rupture or plexic palsy). Paralaysis of the deltoideus (lesion of the axillary nerve), on the contrary, leaves a possibility of abduction, given by the cap muscles, but with a diminished force. The imbalance leads to a functional arthrodesis.

Globally, maximum passive abduction reaches 180°, which can be divided into:

- the first 30° in the glenohumeral joint
- the following 60°, twice more (40°) in the glenohumeral, and then in the scapulothoracic (20°)

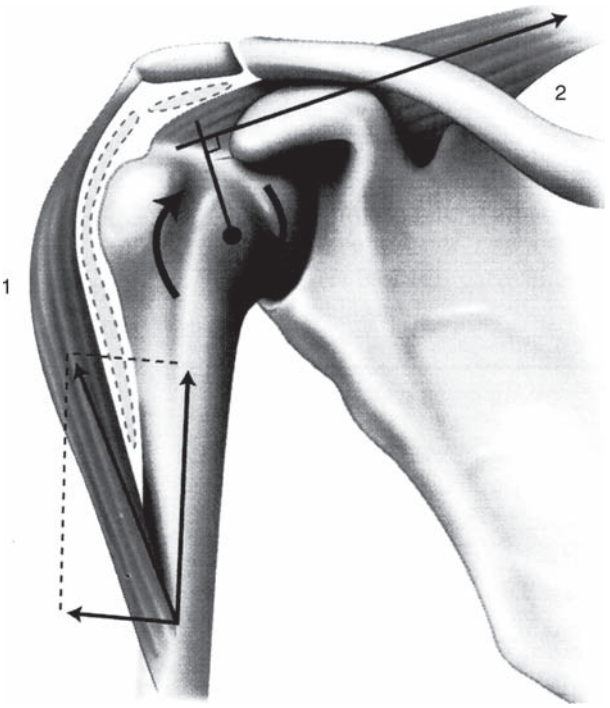

Figure 8.13

The role of the supraspinatus in abduction of the scapula. It avoids the upper displacement of the head due to the axial component of the force exerted by the deltoideus. Part 1 is the deltoid muscle and part 2 is the suprasupinatus muscle. Reproduced with permission from Delamarche et al.[2]

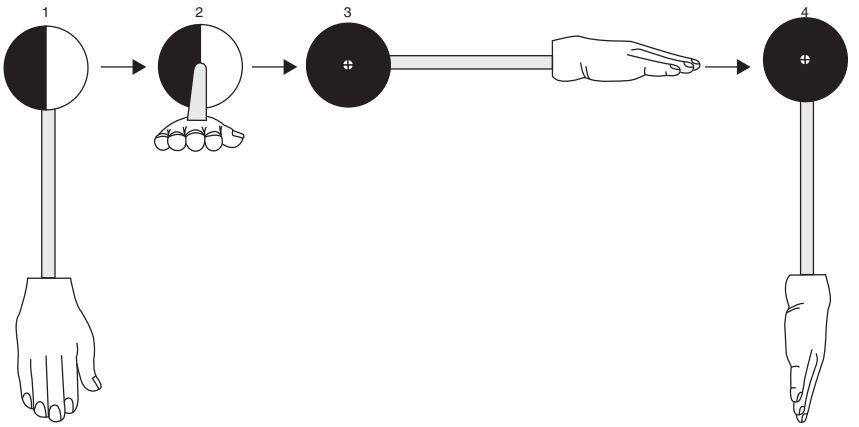

Figure 8.14
The Codman paradox. Adapted from Delamarche et al.[2]

- then, from 90° to 150° the situation is reversed: 40° in the scapulothoracic and 20° which follows in the glenohumeral joint
- finally another 30° are obtained with a vertebral inclination.

Many authors evoke a possiblity of *rotation* which is obligatory and linked to the other movements. Such a rotation exists in biomechanics when the axis of movement does not correspond to the usual axis of mobility of the joint. For example, a movement around the axis of the cervical part of the femur leads to a rotation in regard to the flexion of the hip considered with a transversal axis through the head in the frontal plane. When a movement can be broken down into two spatial planes, then a rotation can occur. This explains the well-known Codman paradox: anteflexion of the limb with a rotation of 180° of the hand, followed by an adduction of the scapula of 90° and afterwards by a retroposition of 90° puts the hand in a position of 90° (Figure 8.14).

However, at the level of the shoulder, and particularly at the glenohumeral joint, there is no axis of movement but centers of mobility, and the obligatory rotation does not appear obligatory.

Nevertheless, there are unexpected rotational movements induced by muscular actions of the numerous muscles involved. For example, when the scapula moves forward it can be seen that the orientation of the glene is modified, and if the movement wanted by the individual does not need any rotation he has to correct the rotation induced by the scapula.

Medial rotation is set by the teres major, the subscapularis, and the serratus major. However, the movement of the scapula can obtain the same result.

Adduction is produced by all the muscles which have insertions on the humerus. The forces they generate, by the parallelogram study, have an axial component which

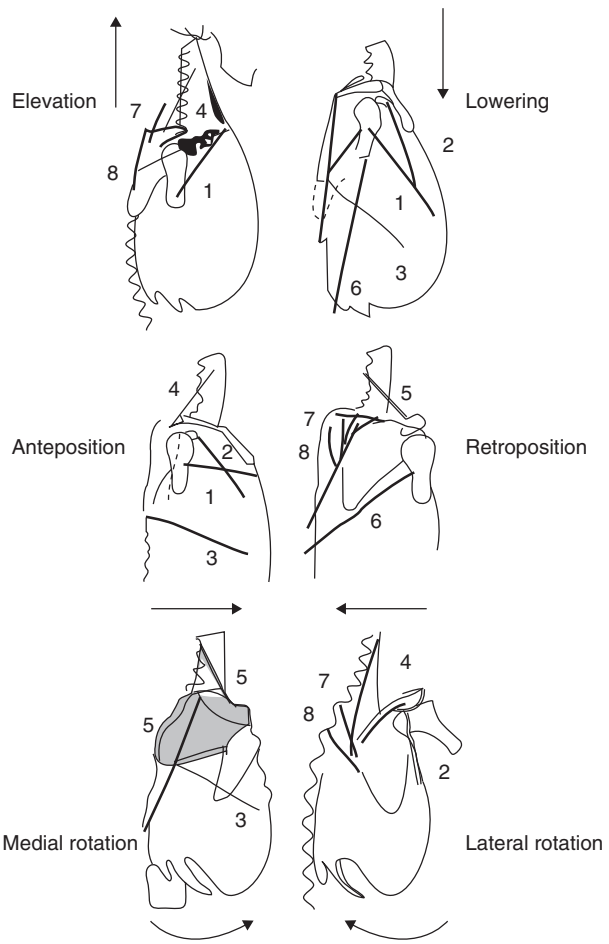

Figure 8.15
The muscles mainly involved in the movements of the scapular girdle: 1, pectoralis major; 2, pectoralis minor; 3, serratus major; 4, levator scapulae; 5, trapezium; 6, latissimus dorsi; 7, rhomboideus minor; 8, rhomboideus major.
Adapted from Steindler.[5]

increases when adduction becomes predominant, and the result is to lower the limb and increase the force of gravity (in a free movement). The adductor muscles have a significant role, acting as a brake on the forces of the abductor and, particularly, the deltoideus. This shows once again the necessity of simultaneous activity of the shoulder muscles. The existence of many torque forces further complicates the understanding of biomechanics.

Finally, *retroposition* of the limb is obtained by retroposition of the scapula by the muscles inserted on the spine, and also by the contraction of the posterior muscles inserted between the scapula and humerus.

In conclusion, it should be repeated that many muscles are working together during all the movements of the upper limb. In Figure 8.15, the drawings based on the work of Steindler[3] show the main associations and torque effects in specific movements.

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9 The shoulder: physiology and motors

Alex Muset and PL Raimondi

The five joints that make up the scapular girdle, or shoulder joint complex, participate in the movement of raising the arm (Figure 9.1). Under physiologic conditions, the sternoclavicular joint is the pivotal point of the movements of raising and rotating the entire complex. Its action is complemented by the movement of the acromioclavicular joint. The active/passive balance of the sternoclavicular joint is at 40° of elevation and 40° of axial rotation. The passive balance of the acromioclavicular joint is at 20° of elevation, although only a few degrees of rotation allow this.[1]

Thoracoscapular syssarcosis, defined as a false joint, is a sliding space with a bicameral structure; the two chambers are subdivided by the serratus anterior muscle. The internal part, the so-called thoracic serratus, is found on the costal wall and the serratus anterior muscle, while the external part, the shoulder blade serratus, is found between the serratus anterior muscle and the subscapular.[2] From a theoretic point of view, since there are no joint surfaces or static stabilizers intrinsic to hydrarthrosis, it behaves as a system of unlimited mobility (Figure 9.2).

The connection of the superior extreme of the appendicular skeletal system with the axial skeletal system is made via the clavicle, as a result of which the sternoclavicular and acromioclavicular joints and the costoclavicular and thoracoclavicular ligaments are integrated as part of the system. As a consequence of this integration, there is automatic limitation of the movements of the thoracoscapular system. In fact, the degree of participation of this complex in the complete elevation of the arm is 60°, with a possibility of axial rotation of 40°. Furthermore, in the normal articulating function of the shoulder, the clavicle acts as a supporting cam that constitutes an element of mechanical advantage in a system seen as intrinsically unstable, the mobility and stability of which is assured by muscular function. In conclusion, the clavicle, through the sternoclavicular and acromioclavicular joints, acts as an element of union between the appendicular and axial skeletons, with a biomechanical aspect:[1,3]

- First, it acts as the pivotal point in the sternoclavicular joint, enabling muscular motorizing and stabilizing action in the movement of raising the arm.
- Second, the active and passive articular tilt of the joints that is found in their extremes limits the mobility of the thoracoscapular area.

Paradoxically, the dual role that the musculature plays in this articular complex, assuring three-dimensional rotational stability through motorization, allows it to supplement the function carried out by the clavicular system. The coexistence of clavicular pseudoarthrosis and lesions of the thoracoclavicular ligaments in the presence of normal muscular function does not impose a limitation on the complete elevation of the superior extreme.

The above situation raises the question of how muscles lose function in the medium and long term, coinciding with degenerative changes associated with the fifth decade of life. During the elevation of the arm purely in the scapular plane, or with a few degrees of anteversion with respect to this plane, in which flexo-abduction movements take place, there is an automatic external rotation of the head of the humerus.

In the frontal plane, or the pure coronal plane, at approximately 90° of abduction, a collision occurs between the greater tuberosity of the humerus and the acromion, such that continued elevation is rendered impossible. The external rotation of the humeral head, which occurs at the level of

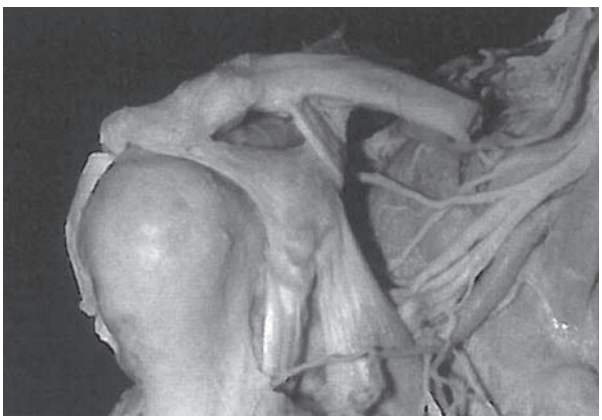


Figure 9.1
Glenohumeral structure.

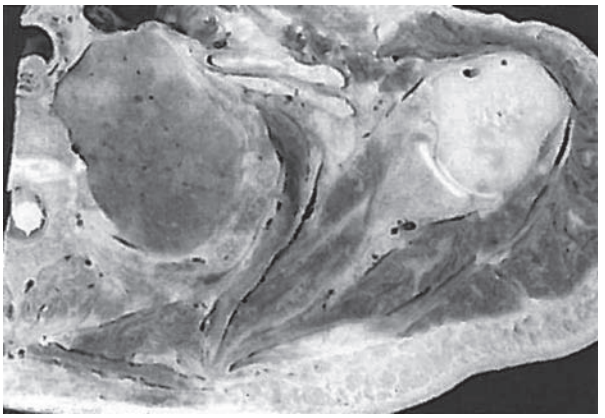


Figure 9.2
Thoracoscapular space.

the glenohumeral joint, pushes this tuberosity back, thereby allowing the movement to continue. In this plane, the scapula is raised and tilts, later positioning itself on the thoracic cage as it is pulled toward the spine by contraction of the superior and medial trapezius muscles and the rhomboid. If we maintain the scapula in this plane, the progression of the movement of elevation is forced and unnatural. It is possible that the dominance of the rhomboid muscles counteracts the action of the serratus superior muscle, which inhibits the balancing rotational movement. As no complete balancing movement is produced, the acromion is not subsequently projected, and, despite external rotation, further movement is difficult. In addition, the posterior location of the scapula impedes the anterior migration of the inferior scapular angle, and even though we may passively force external glenohumeral rotation, we cannot produce axial rotation of the scapula, and thus complete elevation of the arm is prevented.

In the scapular plane itself, or with a few degrees of anteversion, the thoracoscapular joint is seen to be free, limited only by the articular tilt of the sternoclavicular and acromioclavicular joints. If we carry out a lifting movement, at 60° of scapular elevation we have to add in scapular axial rotation, with a maximum of 40°, which will produce the motorized scapular tilting movement with the inferior fascicle of the serratus anterior muscle.

The presence of this tilting movement implies in turn a posterior projection of the acromion, a twisting of the glenoid fossa in the axial direction, and a displacement of the scapula in an upwards, forwards, and outwards direction. The elevation of the acromion affects the function of the deltoid, which compensates progressively by shortening its muscle fibers during the raising of the arm. In the opposite case, the lack of acromial mobility leads to complete cutting off of the deltoid muscle upon arriving at the maximum 90°, thereby terminating its function.[4] Biomechanical studies have demonstrated that the external glenohumeral rotation produced in flexo-abduction movements is conditioned by muscular motorization and by the morphology of the subacromial vault. Studies investigating the morphologic differences of the vault have allowed several rotational patterns to be defined: premature, linear, and late. In these studies scapular tilting movement was eliminated, given that the shoulder blade remained attached to a trial support. Thus, the acromion constitutes a limit and the subacromial vault forces an external rotational movement to be made, allowing for elevation up to 110–120°. Even in this situation, the degree of external rotation averages 10–14°. We may conclude, then, that we can raise the arm with a minimal true glenohumeral rotation.

If, in addition, we factor in the 40° of axial rotation that the scapula is capable of in its tilting movement, we can see that the humeral stabilization in neutral external rotation or at 10° of external rotation is sufficient for the scapular movement, in its glenoid reorientation, to project the arm to its zenith when the movement is carried out at the scapular level or with a few degrees of anteversion to this level.

The destructuring of the glenohumeral joint, when this allows for external active-passive neutral rotation (or approximately 10°), enables elevation of the arm. At the coronal level, with 90° abduction and flexion of the elbow, the forearm is positioned parallel to the ground. At the scapular level, or with slight anteversion, the complementary scapular tilting movement and the axial rotation that it brings about complement the external glenohumeral rotation, enabling projection of the hand behind the neck.

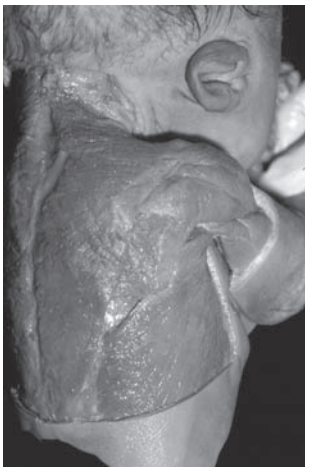
The external rotational movement carried out in the glenohumeral joint must be examined with the elbow flexed and pinned to the body, along with innervation of the brachial biceps and brachial plexus anterior muscles. During the movement of raising the arm at the scapular level, or in slight anteversion, the external glenohumeral rotational movement is complemented by the scapular tilt, thereby eliminating acromial shock and reorienting the fascia of the glenoid joint.

The rotator cuffs constitute the intrinsic muscular system for glenohumeral motorization and stabilization.[5,6] The coordinated action of the subscapularis, supraspinatus, infraspinatus, and teres minor muscles not only acts as a reconversion element for the sideways and rotating glenohumeral movement, but also enables complete elevation of the arm. Thus, this functional complex may be considered to be autonomous in carrying out internal rotational movements, external rotational movements, and elevation of the arm.

The intervention of the infraspinatus muscle in the process of raising the superior extremity marks the inception of the external rotational movement. Along with the active action of glenohumeral extrarotation, we need to associate the passive action of automatic extrarotation induced by the morphology of the subacromial vault when carrying out a flexo-abduction movement. This may potentially affect the chronology of the external rotation, from both a passive and an active point of view. The false subacromial joint (subacromial sack and/or subdeltoid) is functionally linked to the movements of the glenohumeral joint during the process of elevation of the superior extremity.

The scapular tilting movement complements glenohumeral extrarotation, such that achieving a neutral external rotation, or one of 10–15°, is sufficient to carry out complete elevation of the superior extremity.

(a)



(b)

Figure 9.3

Muscle bundles for upward and downward movements. (a) Angular – trap superior; (b) gravity – trap inferior.

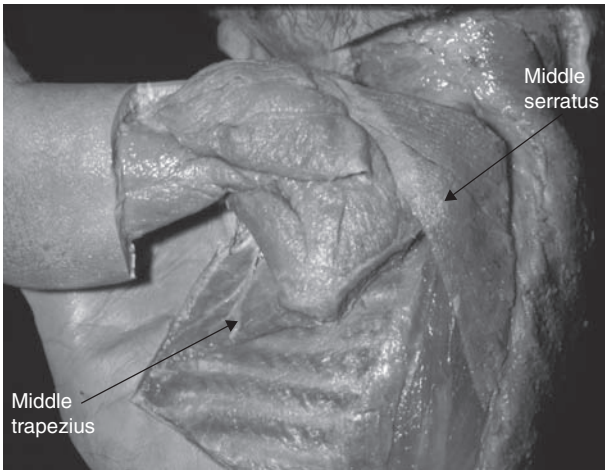


Figure 9.4

Muscle bundle for protraction and retraction movements.

Upon examination of the function of the infraspinatus muscle, it becomes clear that with a function of M3, the patient may carry out complete external rotation with the elbow pinned to the body, and this force is sufficient to maintain the forearm parallel to the ground at 90° of abduction in the coronal plane. The false thoracoscapular joint is the supporting point and the main motor of the spatial projection movements of the hand. The intrinsic stability of this sliding surface is non-existent, given that both its stability and its function depend exclusively on the trunk and scapular muscles.

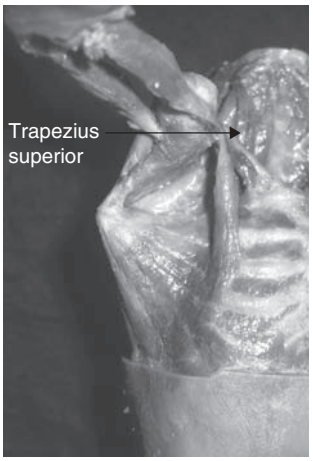
Anatomic and clinical studies allow for definition of the thoracoscapular motorizing entities – the so-called muscle bundles (Figures 9.3–9.6).[3] Muscle bundles are coordinated functional units that are made up of two antagonistic muscles for specific scapular movements. Four scapula muscle bundles have been defined:

1. That made up of the superior part of the trapezius muscle and the pectoralis minor. This allows the scapula to perform an oblique upward and backward movement, or oblique descent in the opposite direction. The nerves involved in this action are the accessory nerve (XI cranial nerve) and fibers from C7–C8.
2. That made up of the scapula elevator muscle and the inferior part of the trapezius muscle. This allows cranial–caudal displacement of the scapula. The nerves involved in this action are the accessory nerve (XI cranial nerve) and the dorsal nerve of the scapula (fibers from C5).
3. That made up of the medial part of the trapezius muscle and the superior and medial parts of the serratus anterior muscle. This allows for protraction and retraction of the scapula. The nerves involved in this action are the accessory nerve (XI cranial nerve) and the long thoracic nerve (Bell's nerve).
4. That made up of the rhomboid muscle and the inferior part of the serratus anterior muscle. This allows for scapular tilting movement. The nerves involved in this action are the long thoracic nerve (Bell's nerve) and the dorsal nerve of the scapula.

The perfect coordination of these muscle groups assures the sliding of the scapula across the thorax, as well as its stabilization. The movements are not simple ones, as they involve articulations within the functional complex of the shoulder. The demands imposed upon this musculature increase with any movements of the glenohumeral joint, multiplied by the load displaced with the hand.

The functional objective of the sliding system of the scapula is to place its superexternal, or articular angle, in the maximally advantageous mechanical position for enabling the movements that are carried out in the glenohumeral joint. Paralysis of any of these muscles, either in isolation or in the context of a more generalized condition, such as an injury to the brachial plexus, hampers projection of the glenoid fossa and therefore hampers the global function of the articulation complex of the shoulder.

From the starting point of an analytic study of the morphology of muscular innervation, the existence of compensatory functional thoracoscapular systems is of considerable clinical interest and relevance, in that they open the door



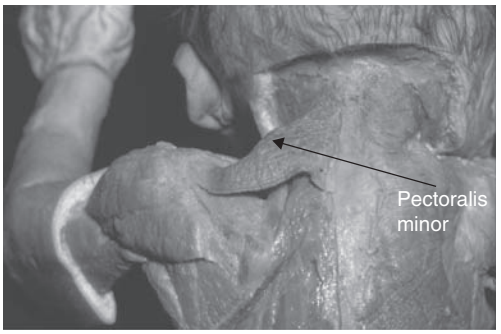


Figure 9.5

Muscle bundle for upward and oblique downwards movements.

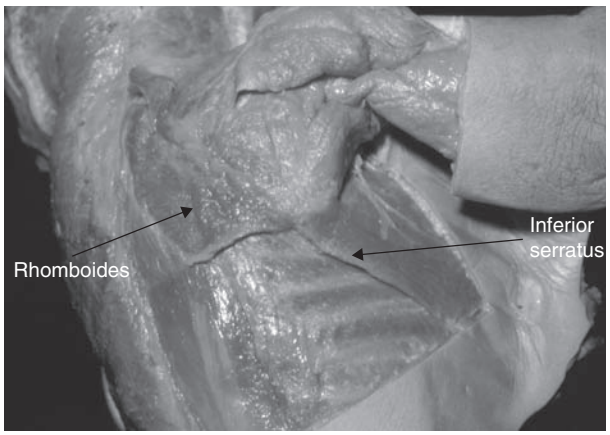


Figure 9.6

Muscle bundle for tilt scapular movement.

○ Upwards	Angular – upper trapezius
○ Downwards	Inferior trapezius – gravity
○ Retraction	Middle trapezius – rhomboid
○ Protraction	Middle serratus – pectoral

Figure 9.7

Thoraco-scapular compensation systems.

to a therapeutic clinical classification of thoracoscapular paralysis.

The angular muscle of the scapula and the superior part of the trapezius muscle are complementary in the movement of cranial displacement of the scapula, each representing distinct innervations (dorsal nerves of the scapula and accessory, respectively). Inferior displacement of the scapula is actively assured by the contraction of the inferior part of the trapezius muscle and by the action of the force of gravity. Participating in scapular retraction are the medial fascicle of the trapezius muscle and the rhomboid muscle. This action, then, is complemented by two muscles that receive distinct innervations.

Scapular protraction is brought about by the contraction of the medial fascicle of the serratus anterior muscle and the pectoralis minor muscle. In this case, it occurs as a result of the voluntary dynamic fixing of the glenohumeral joint owing to the co-contraction of the intrinsic glenohumeral muscles. Innervation is distinct for the two muscular systems: the long thoracic nerve (Bell's nerve) and the handle of the pectorals (where there is a variable participation of the distinct roots of the brachial plexus).

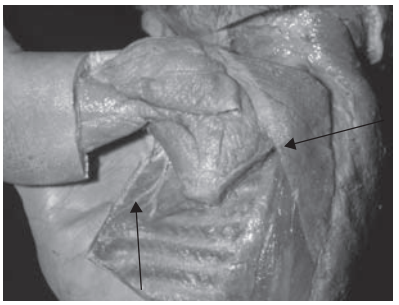
For scapular tilt movement there is no compensatory system, in that the action of the inferior part of the serratus muscle is not complemented or duplicated by another muscle or muscle group (Figures 9.7 and 9.8).

Paralysis of the trapezius muscle

Injury to the accessory nerve (spinal, XI cranial nerve) frequently occurs where it passes through the postsuperior zone of the cervical triangle. The resulting denervation that this produces in the trapezius muscle has significant anatomic and functional repercussions for the thoracoscapular joint. The role that this joint plays in the functional complex of the shoulder explains the clinical repercussions that are seen in the movements undertaken in the projection of the superior member.

In the movement of raising the arm, muscle coordination among the five joints that make up the functional complex of the shoulder translates into preservation of a scapular-humeral rhythm. This notion, introduced and developed by Codman, presupposes the intrinsic complexity of this articular group. Later authors have recognized and explored this idea through studies of joint function, defining static and dynamic functions of the various muscles that motorize the shoulder complex. The concept of muscle bundles, put forward by Benninghoff, places the trapezius muscle in a basic role.

The trapezius muscle is divided functionally into three parts, each of which participates in three of the four basic functional motorization bundles of the thoracoscapular joint. Paralysis of the descending superior fascicle of the trapezius muscle causes scapular displacement in the caudal



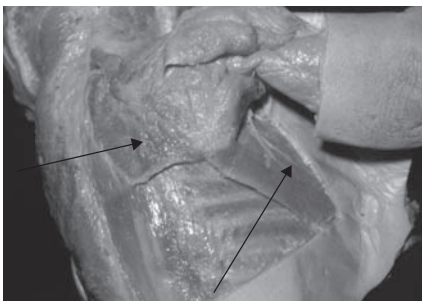


Figure 9.8
Trapezius and rhomboid complexes. Arrows indicate serratus muscles.

direction, partially taken over by the elevator muscle of the scapula, as well as by the lowering of the superexternal angle of the shoulder blade. Paralysis of the medial (transverse) and inferior (ascending) fascicles is responsible for the instability of the medial edge and the lateral displacement of the scapula.[3]

Along these lines, the disorganization of three of the four muscle bundles has important clinical implications. The loss of control of the scapular rhythm brings with it a lack of coordination of the intrinsic and extrinsic movements of the glenohumeral and thoracoscapular joints, seriously affecting the elevation of the extremity. The lowering of the superexternal scapular angle leads to inappropriate orientation of the glenoid cavity in respect to the humeral head, forcing the rotator cuff, the deltoids, and the trunk muscles to act in a physiologically incorrect manner.

Patients report posterior periscapular pain, muscle fatigue in the shoulder area, and the inability to raise their arms, with the appearance of an overhanging shoulder blade when they attempt abduction, owing to the instability of the scapular spinal edge. The acromial fall is the main etiologic factor in the subacromial impingement syndromes and rotator cuff injuries following lesion of the accessory nerve. In deeply rooted situations, the clinical symptoms of the patients are exacerbated by the appearance of thoracic outlet syndromes, radiculitis from neurologic traction, and irreversible contractures of the periscapular and cervical musculature. Upon arrival at the doctor's office, patients present with the inability to raise their arms, limited on average to 95°. The rest of the articular tilt of the shoulder, both active and passive, is generally complete.

Neurophysiologic explorations show the presence of potential for denervation in the trapezius muscle without voluntary potentials, or a reduced number of voluntary potentials in the superior portion and denervation potentials in the medial and inferior fascicles.

Since the beginning of the twentieth century a number of procedures for reconstructing the function of the trapezius muscle have been described, most notably those of Henry, Dewar and Harris, and Eden and Lange. Henry developed a technique of passive spinal-scapular tenodesis, the results of which have not proven consistent. Dewar and Harris associated with this technique acromial transposition of the angular muscle of the scapula. In an attempt at anatomic reconstruction of the fascicles of the trapezius muscle, Eden and Langers proposed transposition of the rhomboid muscle associated with the elevator muscle of the scapula.

Currently, microsurgery allows us to try anatomic reconstruction of the lesion, which is why, with an eye to the sequelae and the difficulties in correction that may arise at a later date, microsurgical repair should be given priority. We advise microsurgical treatment for all these patients, and they should also be informed of the possibility of carrying out palliative muscular transposition in the event that reinnervation proves deficient.

The superficial trajectory of the accessory nerve through the posterior cervical triangle places it in a position of risk in terms of possible lesion. Iatrogenic lesions are one of the most important etiologic factors in isolated lesions of this nerve. Thus, the minor surgical procedures carried out in this topographic region, or radical surgery of the neck for proliferating malignant processes, are high-risk procedures. Current surgical techniques used in oncology offer block resection with preservation of the integrity of the accessory nerve. Although the trapezius nerve receives supplementary innervation from branches of the cervical plexus (C2–C4), this has not been demonstrated to be clinically sufficient to compensate for lesion of the accessory nerve in the innervation of its superior part (descending).

Generally, surgical exposure of the accessory nerve poses no more difficulty than that of building up to the sternocleidomastoid muscle and, in the reverse direction, locating the closest neuroma. Nevertheless, there is a certain difficulty in the dissection of the distal extreme, since the only reference point is on the deep face of the trapezius muscle.

Some authors propose doing the microsurgical reconstruction by means of direct suture. However, our casuistry has always dictated that there be free grafting of the sural nerve, in spite of the mobilization of proximal and distal nerve endings.

Paralysis of the superior trapezius

The paralysis that affects the superior part (descending) of the trapezius muscle involves the loss of elevation of the superexternal angle of the scapula, the articular angle, in both directions: its pure elevation in conjunction with the angular muscle of the shoulder blade, and its oblique elevation upwards and backwards. The inferior movement of the superexternal angle and the impossibility of its projecting upwards and forwards penalize the glenohumeral abduction. Thus, the glenoid cavity and the subacromial

vault end up facing downwards. The acromion acts as a brake on the elevating movement, stopping the contractile movement of the deltoid muscle and impeding the possibility of external automatic rotation. The position acquired by the glenoid cavity does not assure correct positioning of the humerus–scapula axis, leading to intrinsic instability and inferior glenohumeral subluxation of the humeral head when adductive movement is attempted.

The action represented by the force of gravity on the scapula necessitates the functional compensation of the angular muscle of the shoulder blade. Its action in the initial periods of paralysis of the trapezius muscle allows it to maintain 50% of the scapular travel distance in the cranial direction. With the passage of time, patients with this type of paralysis complain of contracture of the cervical paravertebral muscles and the occipitofrontal cephalic muscles, owing to the muscular compression of the cervical nerves (major occipital nerve – Arnold's nerve).

The continuous activity required of the angular muscle of the shoulder blade in these patients produces a muscular hypertrophy, which may be observed clinically after 1 to 2 years of evolution of the lesion. This hypertrophy may also be observed in nuclear magnetic resonance studies if the appropriate plane is found.

The best indication of transposition of the angular muscle of the shoulder blade pro-trapezius superior is established in chronic processes – those in which the muscle has hypertrophied due to compensatory overload. In adult patients it is possible to effect this transfer incorporating a muscularly inserted bone pad fixed acromially with two screws. In young patients, in whom the insertion is cartilaginous, the transposition requires bone suturing (Figure 9.9).

The transposition of the latissimus dorsi pro-trapezius muscle may be carried out via pedicle, across the secondary trunks at the deltopectoral level, placing the muscle in a position that is practically anatomic from the spine to the acromion. As to the innervation, that of the muscle itself may be maintained, or a neurotization of it close to the accessory nerve may be carried out in iatrogenic or trauma cases. The advantage of reinnervation is the physiologic automatism of function (Figure 9.10).

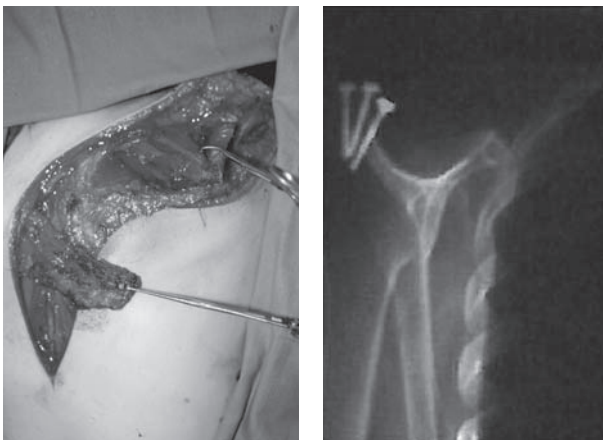


Figure 9.9
Levator scapula and contralateral trapezius transfer.

Paralysis of the medial and inferior trapezius

Paralysis of the medial (transverse) and inferior (ascending) fascicles of the trapezius muscle implies the loss of the projection of the scapula toward the midline. The pure retractive movement of the scapula is carried out by the medial fascicle of the trapezius muscle. The oblique-descending direction of the rhomboid complex and its insertion at the level of the inferior angle of the scapula control the approach of this angle to the spine; this movement may be defined as part of the scapular tilt. Nevertheless, to a certain extent it complements and superimposes its action upon the trapezius, participating in the movement of retraction. In contrast, the direction and insertion of the fibers of the trapezius muscle cannot replace the movements of the scapular tilt in the direction of the spine. Thus, each of these muscular entities has a defined function, but the rhomboid complex, unlike the trapezius, can sometimes partially supplant the movement that brings the internal edge of the scapula to the spine, thereby complementing the action of the medial fascicle of the trapezius muscle.

The scapular traction that brings about the medial part of the serratus anterior muscle in a protractive movement joins the action of the inferior fascicle, by means of the movement of the axillary tilt, distancing the spinal edge of the scapula from the column, without the ability of active return. Therefore, we may note that the combined paralysis of the trapezius and rhomboid muscles is what completely destabilizes the scapula–spine border. In the presence of the serratus anterior, the protraction that this muscle confers on the scapula–spine border through its medial fascicle, and on the inferior angle through its inferior fascicle, brings about a maximum lateral displacement and a marked posterior protrusion of this bone edge. The added paralysis of the elevator muscle of the shoulder blade produces a maximum caudal movement and complete dysfunction. In these cases, the control of the inferior scapula angle is unidirectional, in the axillary direction of the scapular tilt, without the capacity of active return in the direction of the spine.

In theory, the paralysis of the rhomboid muscle would be offset by the medial fascicle of the trapezius muscle. The direction of the fibers of the rhomboid muscle, major and minor, is cranial–caudal, and from inside out, which provides in part a supporting action. From the functional point of view, the superior trapezius supplements this action during active movements. During periods of rest, in the bipedestation position, the trapezius muscle is electrically inactive, so that the superior extreme is maintained by static elements of suspension.

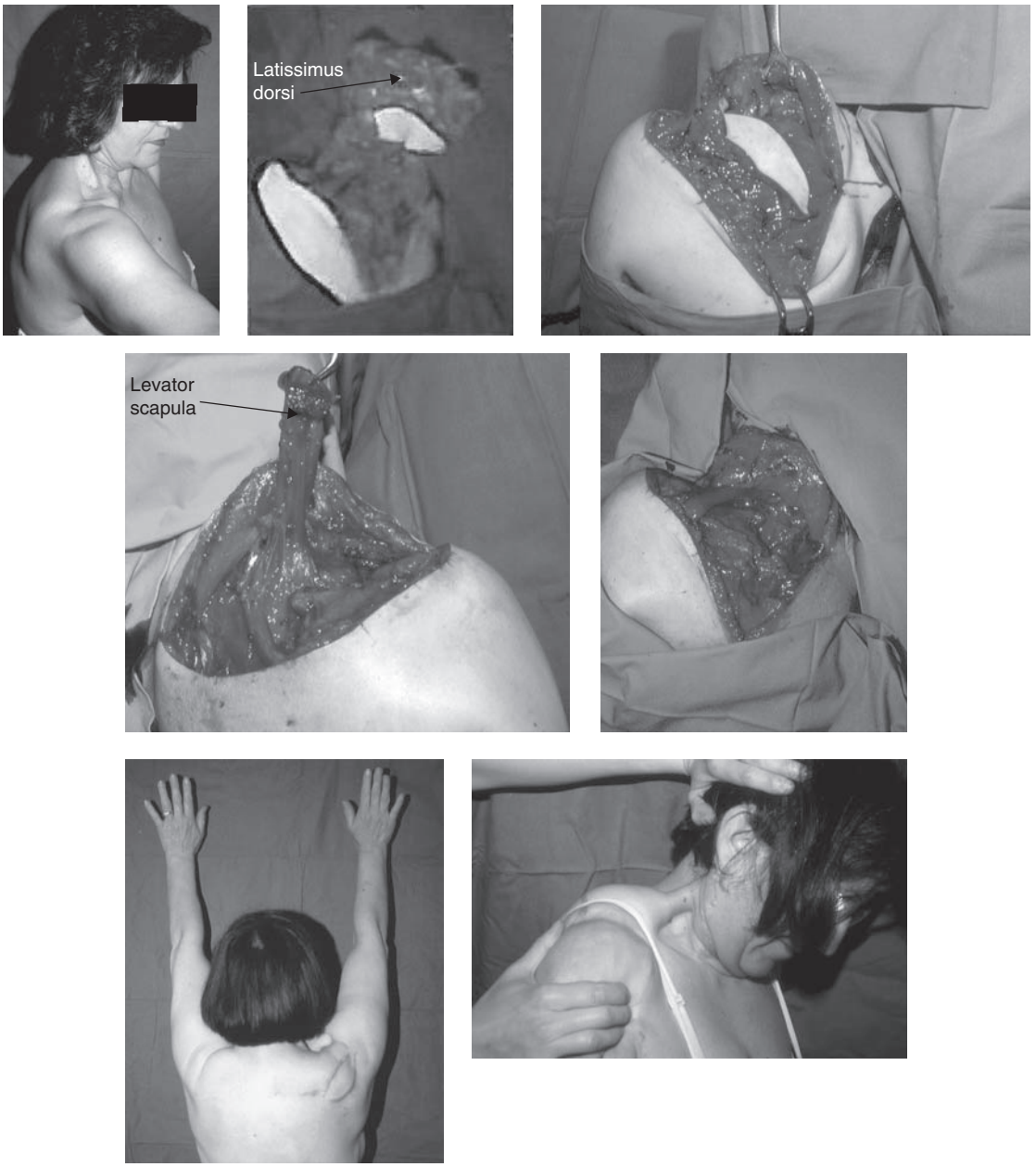


Figure 9.10

Transposition of latissimus dorsi pro-trapezius muscle for trapezius palsy.

The mechanical requests to which substituting muscles are subjected frequently give rise to the appearance of contractures and cervical algia as a symptom of claudication, resulting in the short term in weakness when it comes to carrying out minimal tasks with the affected extremity. The end result is fatigue and displacement of the scapula in the lateral and caudal direction, with insufficiency in scapular rotation and articular orientation accompanied by protrusion of the spinal–scapular border, even though the inferior angle is held stable.

In some cases of reinnervation of the suprascapular nerve with the accessory nerve, good recovery of the supraspinatus muscle is observed, with signs of denervation of the infraspinatus muscle. If the patient also presents paralysis of the axillar nerve, which has not been reconstructed, there will be signs of complete denervation of the round muscle minor and, therefore, a complete deficit of the external rotators. In these cases the reinnervation of the brachial biceps muscle accompanied by effective pectoralis major muscle will give rise to partial

flexion movement of the shoulder and elbow flexion in intrarotation.

The destabilization of the medial rhomboid-trapezius muscle complex impedes the scapular retraction movement, and, to some extent, control of the external passive rotation that may be provided to these patients with a humeral derotative osteotomy. In theory, the scapular tilt movement is more important than protraction in the movements of elevation and projection of the hand in useful angles. In reality, the important part of the movement is that which orients the articular face upwards, forwards, and outwards, and this depends on the projection of the inferior angle in the axillary direction, which is the task of the inferior fascicle of the serratus anterior muscle.

The spinal return movement of the scapular tilt is, in part, a product of gravity, although this is not the whole story. However, scapular retraction in elemental shoulders may complement external rotation, as occurs in the movements that are effected following scapular-humeral arthrodesis.

From a functional point of view, concerning serious paralysis of the bracchi plexus, the retraction movement via stabilization of the medial scapular edge is a point in favor of palliative reconstruction of the external glenohumeral rotation, in that it moves the hand away from the midbody line.

Technically, the transposition of the medial and inferior fascicles of the contralateral trapezius muscle permits reinsertion at various levels on the medial edge of the destablized scapula. Reinsertion at the level of the inferior angle makes a priority of reconstruction of the scapular tilt in the direction of the spine. In fact this movement does not enable reorientation of the glenoid cavity in the movements of arm elevation, but quite the reverse if there is no synchronization with the activity of the serratus anterior muscle. Furthermore, this movement is not essential; the most useful arrangement is that which complements the actions of the superior fascicle of the trapezius muscle with the inferior fascicle of the serratus anterior muscle, which needs to be active. Nevertheless, the problem in this is that in carrying out these movements, the serratus anterior muscle drags the scapula and situates it laterally, placing an excessive load on the other structures, and on the return movement the scapular retraction has no gravitational substitute (Figures 9.11 and 9.12).

The reinsertion of the medial and inferior fascicles of the contralateral trapezius muscle at the scapular midpoint restabilizes the medial edge of the scapula and does not interfere in the useful movements of the tilt. Thus the retraction is reconstructed without interfering in the reorientation of the glenoid cavity in the glenohumeral flexo-abduction movements.

In all of these situations, the instability involves the protrusion, to a greater or lesser extent, of the spinal edge of the scapula, presenting as a sign of winged shoulder blade. Given this sign it is advisable to carry out appropriate differential diagnosis of the cause of the instability.

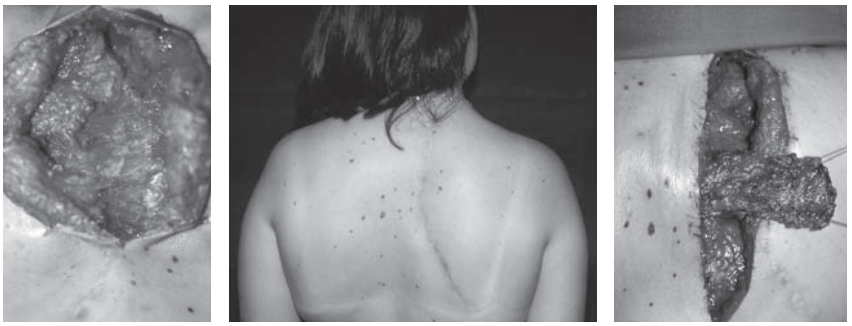


Figure 9.11
Medial instability.

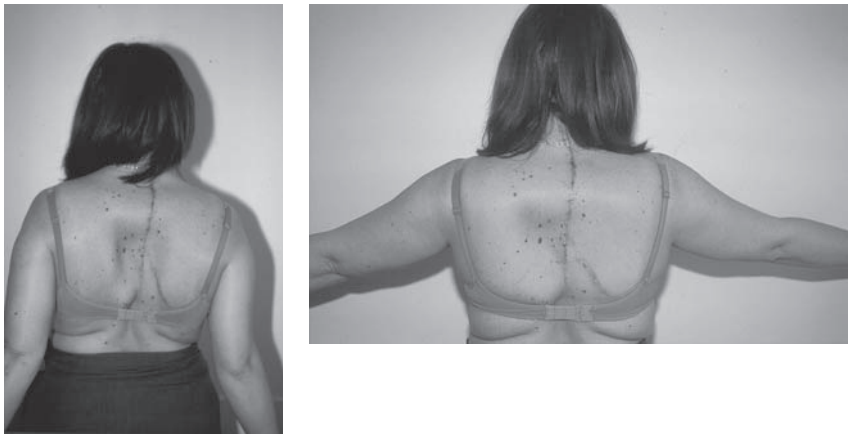


Figure 9.12
Scapular retraction and lateral translation control.

In addition to paralysis we need to add secondary winged shoulder blade to the retractile processes of the glenohumeral joint. It is frequent in the area of obstetric paralysis, translating into direct or inverse Putti signs, depending on whether the contracture is intrarotational or extrarotational, respectively. The deformities brought about by this kind of pathology have been viewed as exclusive to children. This is the case with true joints, but at the thoracoscapular level secondary esthetic glenohumeral deformities do not have an effect on real function, so that biomechanically speaking, the physiopathologic and clinico-therapeutic considerations are as applicable to both traumatic and idiopathic paralysis in the adult as they are to the obstetrics of the child.

Conclusion

The function of the thoracoscapular joint remains acceptable in those patients for whom the accessory nerve has been used as an extraplexual neurotization in microsurgical repair of the bracchi plexus in paralysis of the medial and inferior fascicles of the trapezius muscle. The superior fascicle of the trapezius muscle and the angular muscle of the shoulder blade maintain the scapular suspension, while the rhomboid complex fills in as a priority for the medial fascicle of the trapezius muscle.

The association of paralysis of the medial and inferior fascicles of the trapezius muscle with the rhomboid complex destabilizes the medial scapular edge, with the disappearance of the movement of retraction. Although it may be for iatrogenic reasons, in the paravertebral posterior channels to the spine or the plexus, the use of the accessory nerve in patients with C5–C6 avulsion, without innervation from C4, or in cases of serious trauma with association of a lesion of the proximate segment of the accessory nerve and the plexus superior, the resulting deficit requires repair.

Along these lines, the use of the dorsal nerve of the scapula as a source for a neurotization in conjunction with the use of the accessory nerve is not advisable. The microsurgical reconstruction of the two nerves must be considered if what we wish to achieve is a good functional result with glenohumeral reinnervation.

When microsurgical reconstruction is not feasible, palliative stabilizing surgery from the secondary medial scapular edge to the paralysis of the medial and inferior fascicles of the trapezius muscle and the rhomboid may be carried out by transposition of the medial and inferior sections of the contralateral trapezius muscle. The reinsertion of the muscular transfer is applied at the level of the upper half of the scapular edge, in order to add to the retractive movement a complementary rotational vector, and cranial projection of the acromion in an agonistic form to the action of the superior fascicle of the trapezius muscle and of the inferior fascicle of the serratus anterior muscle. For those cases in which it coexists with paralysis of the rhomboid complex, it is feasible to reconstruct the medial and inferior fascicles of the trapezius muscle by means of a transposition of those fascicles from the contralateral side.

Correct thoracoscapular stabilization is an indispensable condition for proper glenohumeral function. Microsurgical and/or palliative glenohumeral reconstruction must be based on appropriate motorization between the axial skeleton and the appendicular. Minimal motorization of a rotator cuff, which at the glenohumeral level may be self-sufficient, will be severely penalized if, in the reconstruction, we destabilize the thoracoscapular joint. We must, therefore, examine, in the various paralytic lesions, the global reconstructive possibilities of the superior member, especially its motorization with respect to the thorax, if we do not wish to slam the door on the proximal reconstructive process.

The two nerves receiving priority in the reconstruction or preservation of this function are the following:

1. The accessory nerve, responsible for the functioning of the trapezius muscle. The effectiveness of extraplexual neurotization between this nerve and the suprascapular has led to its routine use, even in the presence of conserved proximal axonal capital, to reinnervate the rotator cuff. For the cases in which the rhomboid complex is innervated, the reduction of the medial and inferior fascicles of the trapezius muscle is offset.
2. The dorsal nerve of the scapula, responsible for the functioning of the angular muscles of the shoulder blade and the rhomboids, major and minor. The angular muscle can, in addition, receive branches from the cervical plexus, becoming generally innervated. Complete lesions of the nerve lead to paralysis of the rhomboid complex. Radicular lesions in the zone affect C5, and in many cases C4. In the presence of a sound trapezius muscle, the nerve's function is palliated to an acceptable degree by the muscle.

The muscular elements that we must have available in order to consider palliative surgery of the thoracoscapular joint are:

- the angular muscle of the shoulder blade as an alternative to the superior fascicle of the trapezius muscle
- the medial and inferior sections of the contralateral trapezius muscle, to make up for the homonymous fascicles of the other trapezius muscle and the rhomboid complex.

Paralysis of the serratus

The serratus anterior muscle receives its innervation from the long thoracic nerve (Bell's nerve), the origin of which is variable. Although generally found in the roots of C5–C6–C7, with some frequency it has a component of C4,

and, rarely, of the roots of C8. Its radicular origin, very close to the foramen level, may facilitate clinical diagnosis of the radicular avulsion, as the muscle remains functional in cases of radicular rupture of extraforamenal lesions, and paralyzed, except in its superior part, in cases of avulsion.

The nerve runs first between the fibers of the medial scalene muscle, before reaching the axillary cavity, adhering to the costal wall at the level of the medial axillary line, and branching to the various fascicles of the serratus anterior muscle. The muscle originates in the scapular–spinal border, reaching its superinternal angle, from where it projects along the lateral wall of the thorax. It inserts into the lateral face of the ribcage, at approximately the level of the anterior axillary line. The superior fascicles receive innervation from branches coming from the cervical plexus, while the medial and inferior fascicles only receive fibers from Bell's nerve.[3,7,8]

Functionally, the superior part of the serratus anterior muscle maintains the scapula in suspension. The medial part is responsible for the movements of protraction, and the inferior part controls the movements of scapular tilting, in the axillary direction, by means of traction of the inferior angle of the shoulder blade.[4,5]

The serratus anterior muscle may be considered as one of the key elements in the thoracoscapular joint. Extending from the spinal border to the anterior-lateral zone of the costal wall, it lies between the shoulder blade, with the subscapular muscle, and the thoracic wall, thus delimiting two sliding spaces: one internal and defined by the thorax and the serratus, and the other external and marked by the shoulder blade and the serratus.[1]

From a morphologic point of view, these two spaces are virtual, for sliding, but they do not contain the elements that define an articulation of the diarthrotic type. Nonetheless, from an anatomic-functional point of view, this dual level of sliding space facilitates the complete mobility of the shoulder blade on the costal wall, giving rise to the characterization of 'false thoracoscapular joint'.[1]

This joint forms part of the functional articular complex of the shoulder, and it has a dual biomechanical function. On the one hand it constitutes the true nexus of the dynamic union of the axial skeleton and the appendicular skeleton, through the clavicle, while on the other hand it projects, in a situation of maximal mechanical advantage, the superexternal scapular articular angle, thereby optimizing the glenohumeral and subacromial function and, consequently, providing ideal positioning of the arm for optimal three-dimensional projection of the hand.

The anatomic and functional evaluation that we are able to make in patients affected by flaccid thoracoscapular paralysis allows us to probe more deeply into the two-sided study: control of thoracoscapular motorization and the functional deficit and residual instability. A clear etiologic and physiopathologic definition of the problem is indispensable for the establishment of an appropriate therapeutic strategy.

Typically with paralysis of the serratus anterior muscle, a flexion movement of the shoulder carried out on the sagittal plane sees the inferior scapular angle projecting posteriorly, and the spinal edge of the shoulder blade emerges as evidence thereof. In consequence, the superexternal or articular angle does not project adequately, and this maintains the acromion low and the glenoid cavity oriented in the inferior, anterior direction.[9] What actually occurs is a loss of scapulohumeral rhythm in the movement of elevation, which delays the aperture of the scapulohumeral angle, despite the correct functioning of the intrinsic and extrinsic glenohumeral musculature.[10,11]

The destabilization of the inferior scapular angle and its posterior protrusion imply automatic acromial descent. At the level of the biomechanics of the deltoid the repercussion is decisive. The ascent of the acromion, which is produced progressively under normal conditions during elevation and which occurs by the movement of scapular protraction and tilt, coordinated with the glenohumeral movement through the scapulohumeral rhythm, involves constant traction of the deltoid fibers, compensating for the natural contraction generated in the deltoids when abduction is carried out. In this manner the muscle regulates itself and does not spend its contractile capacity in the first phases of abduction, thereby progressively optimizing its action. The glenoid cavity does not orient itself outwardly, forwardly, and upwardly, as occurs under physiologic conditions, so it conditions an increase in the intrinsic glenohumeral instability and as a consequence, an overload of the intrinsic and extrinsic motorization structures in an attempt to maintain the glenohumeral aperture in unfavorable conditions.[1,4,5,12] To the impossibility of completing the elevation in flexion we need to add the premature shock of the tubercle major (troquiter) with the acromion, modifying the natural biomechanics of the subacromial joint. Furthermore, in the absence of the scapular tilting movement, the acromion does not project posteriorly, which translates into a subacromial overload during the movements, pure or otherwise, of external rotation that will then be wholly glenohumeral.

The anatomic and functional study of the thoracoscapular motorization shows the incapacity for physiologic suppletion on the part of the serratus anterior muscle in the axillary tilting movements and protraction. This is in contrast with the capacity for suppletion that exists between the medial fascicle of the trapezius muscle and the rhomboid complex in movements of opposing bundles.

The clinical condition resulting from isolated paralysis of the serratus anterior muscle must be corrected surgically. The scapular stabilization test is carried out by coapting the scapula to the thorax when the examiner places one hand on the spinal–scapular border and the other on the sternum, in so doing pressing the shoulder blade to the costal wall during the active elevation of the extremity. The patient will note the immediate disappearance of pain and, as well, the normal ability to execute movement with the arm.

Paralysis of the serratus anterior muscle has multiple etiologies. A review of the bibliography and the casuistry will show the relationships between its occurrence and demanding athletic performance, acute trauma and direct impact at the level of the shoulder and the upper extremity,

viral diseases, plexual neuritis, and iatrogenic causes produced during decompression of the thoracic outlet or lesion of the brachial plexus, inter alia.[8,13–16]

Patients complain of prescapular posterior pain, muscle fatigue of the shoulder, and frontal elevation deficit, with the appearance of winged shoulder blade upon abduction. They usually present weakness on anterior elevation on the sagittal plane, with a limitation of between 80° and 120° (average 95°), although the passive articular balance of the shoulder is generally complete.

The majority of cases of isolated paralysis of the serratus anterior muscle resolve spontaneously within 6–9 months. In recent studies it has been noted that approximately 25–30% of patients continue to show winged shoulder blade, periscapular pain, and muscle fatigue. In those cases with neurologic demonstration of lesion of the long thoracic nerve, those with partial regeneration, and those with persistent clinical signs after a prolonged period of observation, surgical reconstruction is the inevitable indication.[15–17]

The methods used for scapular stabilization are varied. They include scapulothoracic arthrodesis, passive tenodesis to the ribs, muscular transfers (pectoralis minor, teres major, rhomboids, and/or shoulder blade angle), all with varied results.[13,14,18–21] The transfer of the sternal part of the pectoralis major muscle is the most widely accepted technique owing to the orientation of the muscle, parallel with the direction of the fibers of the serratus anterior muscle, once it is transferred. Its capacity for excursion and the strength of the muscle are additional arguments in its favor. When carrying out the scapular stabilization test, the examiner can ascertain the significant force that this transfer will provide in elevation movements, especially when made against resistance.[9,16,21] In our experience, the transfer of the external fascicle of the pectoralis major muscle meets the necessary requirements, both of direction of muscle fiber and of the force that is developed.

The operation is carried out with the patient in a supine position, with a deltopectoral incision, or one following the anterior axillary edge parallel to the edge of the humeral insertion tendon of the pectoralis major muscle. This is followed by isolation of the sternal fascicle of this muscle, removing it from the humeral diaphysis. With this technique the clavicular fascicle of the pectoralis major muscle is preserved. The deltopectoral incision is extended along the medial axillary line to the inferior angle of the scapula, or a second, vertical incision is made at the level of the medial axillary line, corresponding to the inferior scapular angle.

The tendon of the pectoralis major muscle may be extended, if needed, with a fascia lata graft (three fingers across in width) which should be folded over itself and turned to form a strong, resistant corkscrew. This technical detail avoids many of the dehiscences described in the literature, and which we have also found in our series.[2,3,22]

The prolongation of the tendon of the pectoralis major muscle is made in order to attach it to the inferior angle of the scapula, through an orifice equidistant at 1 cm from the axillary and spinal edges. A double passage of the plasty is made so that the tendon of the pectoralis major muscle itself may be re-anchored. Ideal placement of the inferior angle of the scapula is achieved by raising the arm to its zenith on the scapular plane; in realizing complete abduction we passively force, through the automatic external glenohumeral rotation, the tilting scapular movement in the axillary direction. In this manner the inferior angle of the shoulder blade assumes its optimal functional position – the same position it arrives at in the complete elevation of the arm on the scapular plane under physiologic conditions. This maneuver enables control of the tension on the pectoralis major muscle, avoiding another of the frequent complications, namely myolysis. During the movement in open air, or in the tunneling of the inferior fascicle, we have to preserve the sensitive branch that connects intercostally, as well as control the nerve pedicle of the pectoralis muscle itself, as it comes from the secondary trunk and the thoracodorsal pedicle (Figures 9.13–9.15).

Postoperative immobilization consists of a Gilchrist-type bandage for 6 weeks. After this time, rehabilitation of the passive articular tilt of the shoulder begins, with pendular movements up to 90° and assisted elevations in the supine position. At 2 months, rehabilitation of the active articular tilt begins, and it is consolidated at 3 months postoperatively.

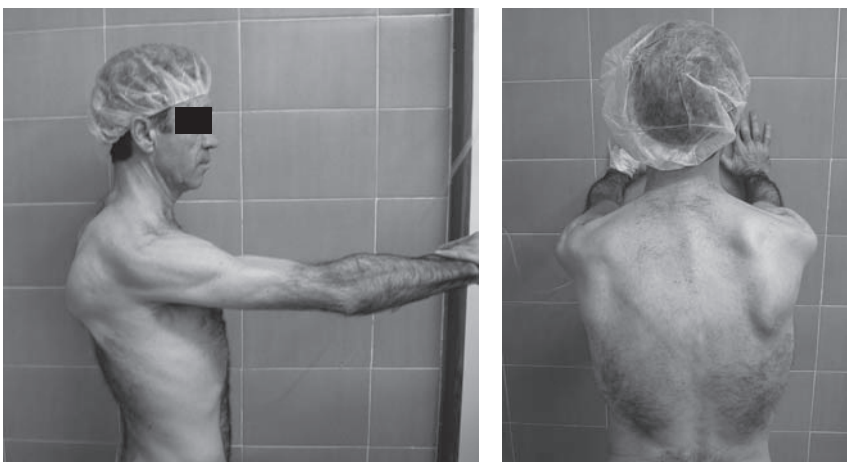


Figure 9.13
Serratus palsy.

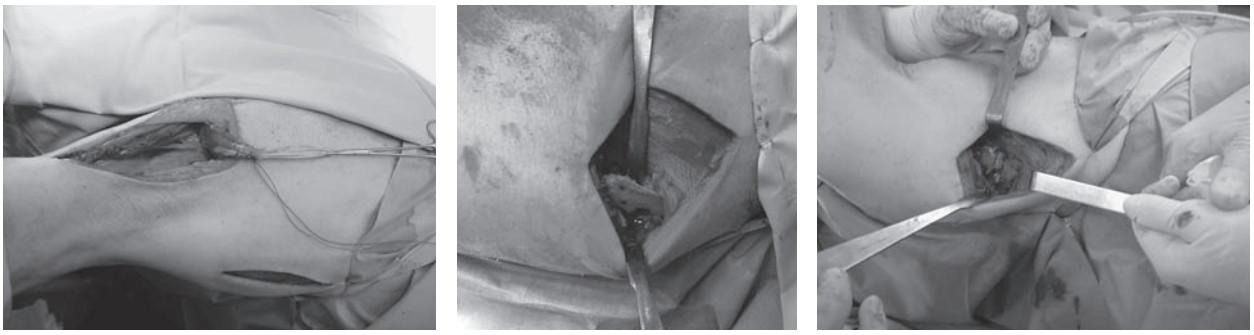


Figure 9.14
Sternal pectoral fascicule pro inferior scapular edge.

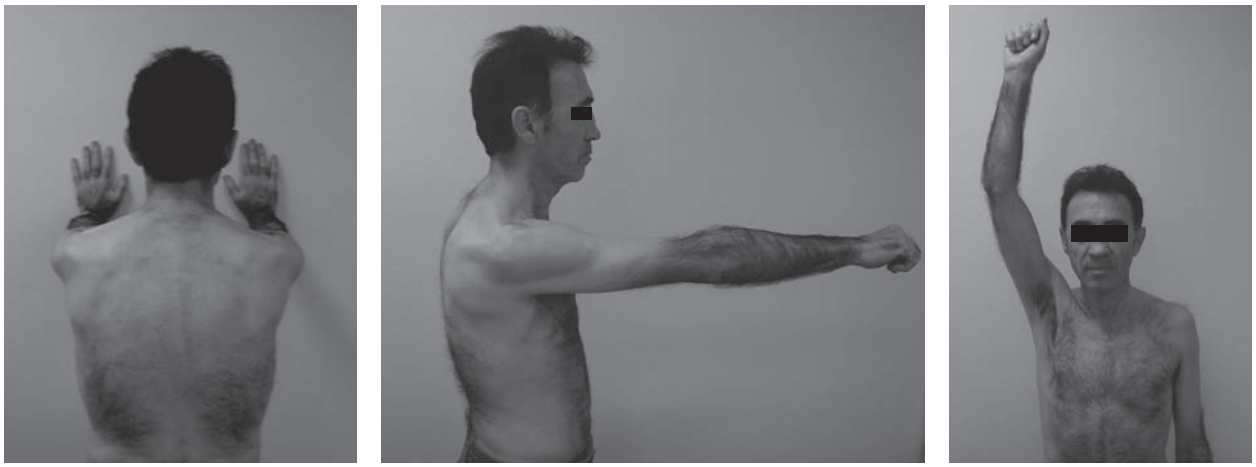


Figure 9.15
Post treatment.

The surgical result is ad integrum restoration of the anterior flexion of the shoulder, eliminating the posterior protrusion of the inferior angle of the scapula/spine edge, with significant improvements in periscapular pain and the sensation of muscle fatigue. Postoperative pain and the sensation of axillary tension persist for 4 to 6 months, with results consolidated at 9 to 12 months postoperatively. The cosmetic aspect of the surgery in cases of double incision is excellent, with no mammary asymmetry in women, and scarring hidden at the axillary level.

After the palliative transfer, with the raising of the arm the muscle may palpitate subcutaneously, clinically confirming its contraction throughout the entire movement of the arm. The interposition of an autologous graft, between the tendon of the pectoralis major muscle and the scapular angle, has been necessary in some cases. In our series it was carried out with the fascia lata entubed, in a corkscrew form, to provide additional resistance. As noted in the bibliography, the obvious consideration is that of interposing a non-vascular structure when it is necessary to try to connect the tendon of the pectoralis muscle with the shoulder blade. In some cases this has not been possible because of evidence of excessive tension at the level of the pectoralis major muscle, or its nerve pedicle; relaxation at the level of the suture was required.

The cosmetic dimension in the pectoral area is good, as the transfer is carried out with two incisions. Nonetheless, in the donor zone of the muscle appreciable scarring remains.

Conclusions

In isolated paralysis of the serratus anterior muscle, the transfer of the sternal fascicle of the pectoralis major muscle, reinforced with an autologous graft when necessary, has been shown to be an effective technique in the correction of winged shoulder blade and in the resolution of the periscapular pain and muscle fatigue that it brings on.

It is a technique of choice in the direct coaptation of the tendon of the pectoralis major muscle at the inferior angle of the scapula, although it may not always be possible, due to muscular tension and/or nerve pedicle. We feel that the use of an autologous graft, as reinforcement between the tendon of the pectoralis major muscle and the scapular

angle, is a technical possibility that is both of utility and effective.

The use of fascia lata as a properly entubed graft, based on the results of our series, has been shown to be technically trustworthy. The main shortcoming of this is the scarring defect that occurs at the inferior level of the extremity.

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10 Biomechanics of the elbow and pronosupination

*René Malek**

Movements of the shoulder girdle, and particularly of the ulnohumeral joint, are necessary to let an individual begin to move his hand closer to an object he wants to take. But it is at the level of the elbow joint that he can adjust the appropriate distance: he does it in a simple manner by modifying the angle between his arm and his forearm, which represent the two branches of a compass. This adaptation is possible owing to the flexion and extension mobility of the elbow.

In addition the hand can be oriented in rotation by means of a special movement (pronosupination) which happens at the level of the elbow. It takes place in the radio-humeral and the superior radioulnar joints. The inferior radioulnar joint is implicated, as well as a particular morphology of the two forearm bones. This rotational movement is combined with the movements of the shoulder (not only rotation of the scapulo-humeral joint).

Thus, it is clear that all these movements are simultaneous and related to each other; nevertheless, it is easier to study them separately.

Flexion–extension movements of the elbow

These two movements involve the radiohumeral and the ulnarohumeral joints sharing the same joint cavity (Figure 10.1).

The joint surfaces

The *lower humeral extremity* (Figure 10.2) presents three articular zones: the trochlea, the humeral capitulum, and the groove between the trochlea and the capitatum. This extremity is globally flattened (hence its name of 'palette'). The two borders of the humeral shaft diverge, like the fork on a bicycle, to clasp the trochlea and the capitatum. They terminate by two processes: the medial and the lateral epicondyles, which represent the bolts of the fork.

The transversal axis of the palette is located forwards and this permits a geater amplitude of flexion of the forearm on the arm.

Figure 10.1

The three joints of the elbow sharing the same articular cavity: ulnohumeral, radiohumeral, and superior radioulnar. Adapted from Delamarche et al.[1]

*with V Gasiunas for the translation in English

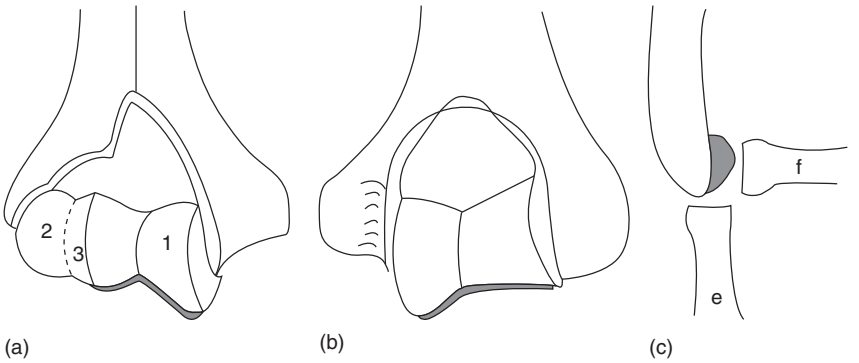

Figure 10.2

The lower humeral extremity. (a) The three parts of the articular surface: 1, the trochlea; 2, the capitellum; 3, the condylo-trachlear groove. (b) The posterior view. (c) The profile view showing that the capitellum is developed anteriorly. (e) Extension; (f) flexion.

- *The trochlea* is larger in the posterior direction, and presents two slopes and a groove which correspond to helicoid arch. It is surmounted in front by the coronoid fossa and behind by the olecranoid fossa. These two fossae are separated by a thin lamella of bone, that is sometimes dehiscent.
- *The capitulum* (capitellum) of the humerus corresponds to a spherical part which is angled forwards and downwards. Its vertical axis is greater than its transversal axis. In a posterior view the cartilage surface is not seen.
- Between the two preceding structures is *the condylo-trochlear groove*, which is bevel-edged laterally.

The *superior ulnar extremity* (Figure 10.3a) is the main element of flexion. It presents two processes: the olecranon behind the humeral palette and the coronoid process below it. The anterior surface of the olecranon and the superior surface of the coronoid form the great sigmoid cavity, which is articulated with the trochlea. This cavity is divided by a vertical crest corresponding to the groove of the trochlea.

On the radial side of the coronoid process is located the lesser sigmoid cavity, which is articulated with the radial head. It is quadrangular with a side of 1 cm, and is concave from front to back. It is separated from the large cavity by a blunt crest covered by cartilage.

The *superior extremity of the radius* (Figure 10.3b), or, more precisely, the radial head which overhangs the neck and the bicipital tuberosity, presents at its superior aspect a fovea which is articulated with the capitulum. The cartilage is in continuity with the one of the circumference of the head, which is higher anteriorly and medially. The junction with the fovea is bevel-edged and joined to the condylo-humeral groove.

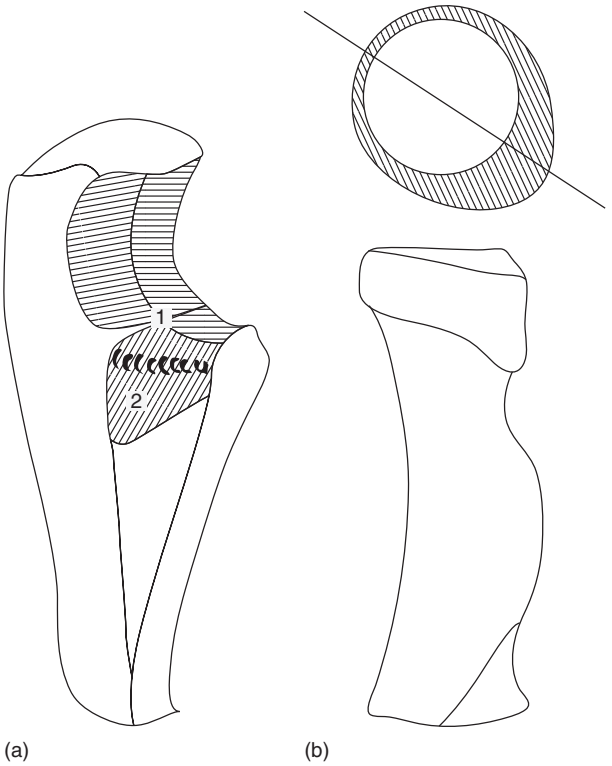

Figure 10.3

(a) The superior ulnar extremity and the two sigmoid cavities. (b) The superior extremity of the radius: its cartilage and axis.

The restraining structures

- The *capsula* is rather loose. Its insertion delimits the elbow joint and follows the borders of the fossae of the palette, which are intra-articular, and also include the extremities of the two forearm bones.
- A *posterior ligament* (Figure 10.4) exists, which is very thin but reinforced by a transversal band crossing the olecranial fossa, and under which there is a layer of fat. This ligament is covered by the triceps brachii tendon and the anconeus muscle.
- An *anterior ligament* extends from the ulnar epicondyle to the radial head, but is very weak.
- The side ligaments are much stronger: the *medial* (Figure 10.5) has three bundles; the middle one – triangular – extends from the coronoid process to the inner border of the ulna. The *posterior* tract (called Bardinet's ligament) is transversal from the medial epicondyle to the olecranon. It forms the medial wall of the epitrochleo-olecranial groove.
- The *lateral* ligament (Figure 10.6a) is also formed by three bundles. The anterior and middle ones surround the radial head, reinforcing the annular ligament, and are inserted on the vertical borders of the lesser sigmoid cavity.

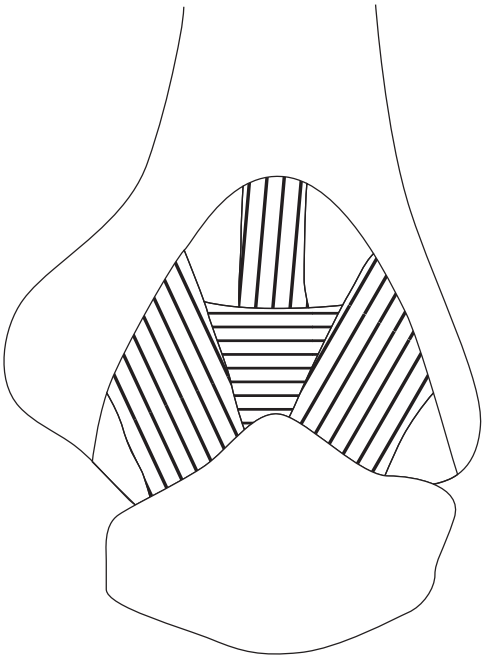

Figure 10.4
The posterior ligaments of the elbow joint.

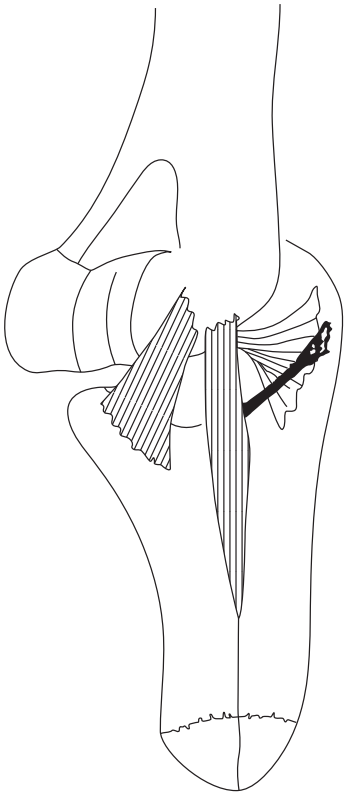

Figure 10.5
The ulnar collateral ligament and its three parts: anterior, middle, and posterior (Bardinet's ligament).

- The ligaments of the radiohumeral joint are also ligaments of the elbow:
  - the *annular ligament* is a strong band of transversal fibers, which encircles the radial head; it is inserted on the two borders of the lesser sigmoid cavity
  - under the latter, the Denucé's ligament, or *quadrate ligament*, is inserted on the inferior border of the cavity and ends on the neck of the radius. It is not very tight, to permit sufficient mobility of the superior extremity during pronosupination (Figure 10.6b).

The muscles

These include the muscles of the arm as well as the muscles of the forearm. The particular feature of all the muscles of the elbow is that, at least for some of their bundles, they are bi- or pluriarticular. Those which get their bellies at the arm and are also inserted on the scapular girdle have an action on its joints. Those which get their bellies in the forearm also act on the wrist and digits. This support the idea that *all* the muscles of the upper limb are involved in any movement of any part of it.

The study of the insertions of the muscles will be followed by an explanation of their action as described by the anatomists. However, this does not correspond to the biomechanic reality, which will be discussed later.

Flexor muscles

The flexor muscles can be divided into two groups. *Group 1 muscles* have their bellies at the level of the arm (Figure 10.7):

- The *brachialis*, which arises from the two sides medially and laterally of the shaft of the humerus, under the insertions of the deltoideus and from the intermuscular septa (further from the medial). The fibers cover the front of the elbow joint and are extended by a thick tendon inserted on the coronoid process. It presents an aponeurotic expansion which covers laterally the muscles of the radial epicondyle.
- The *biceps brachii* has two heads: the short portion arises from the apex of the coracoid process (coraco-brachialis) and the long head arises from the supraglenoid tuberosity at the upper margin of the glenoid cavity, in continuity with the glenoidal labrum. As has been described in a preceding chapter, this cylindrical tendon goes through the joint without entering the joint cavity (Chapter 8). The two bellies join each other and the unique belly forms a flattened tendon which is twisted and inserts in the posterior part of the tuberosity of the radius. A serous bursa separates it from the anterior part of the tuberosity. This tendon gives, on its medial side, a broad aponeurosis expansion (lacertus fibrosus), which covers the muscles arising from the medial epicondyle.

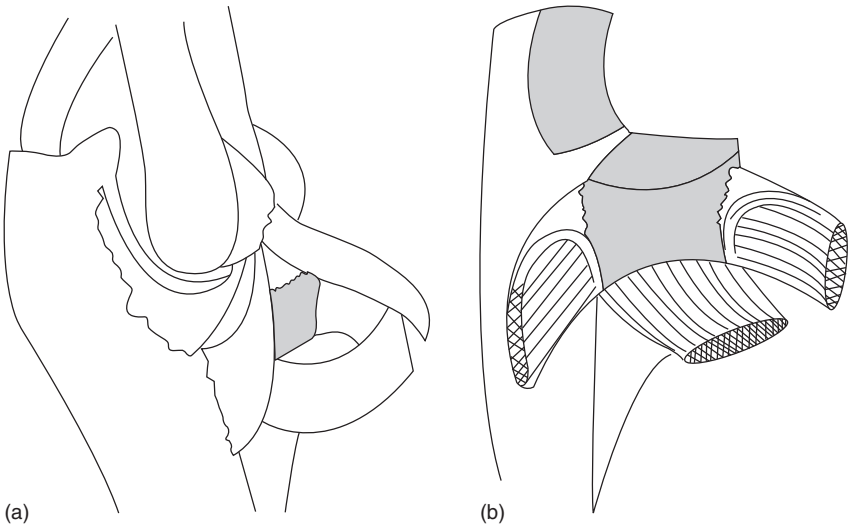

Figure 10.6

(a) The radial collateral ligament and the annular ligament. (b) The quadrate ligament and the annular ligament.

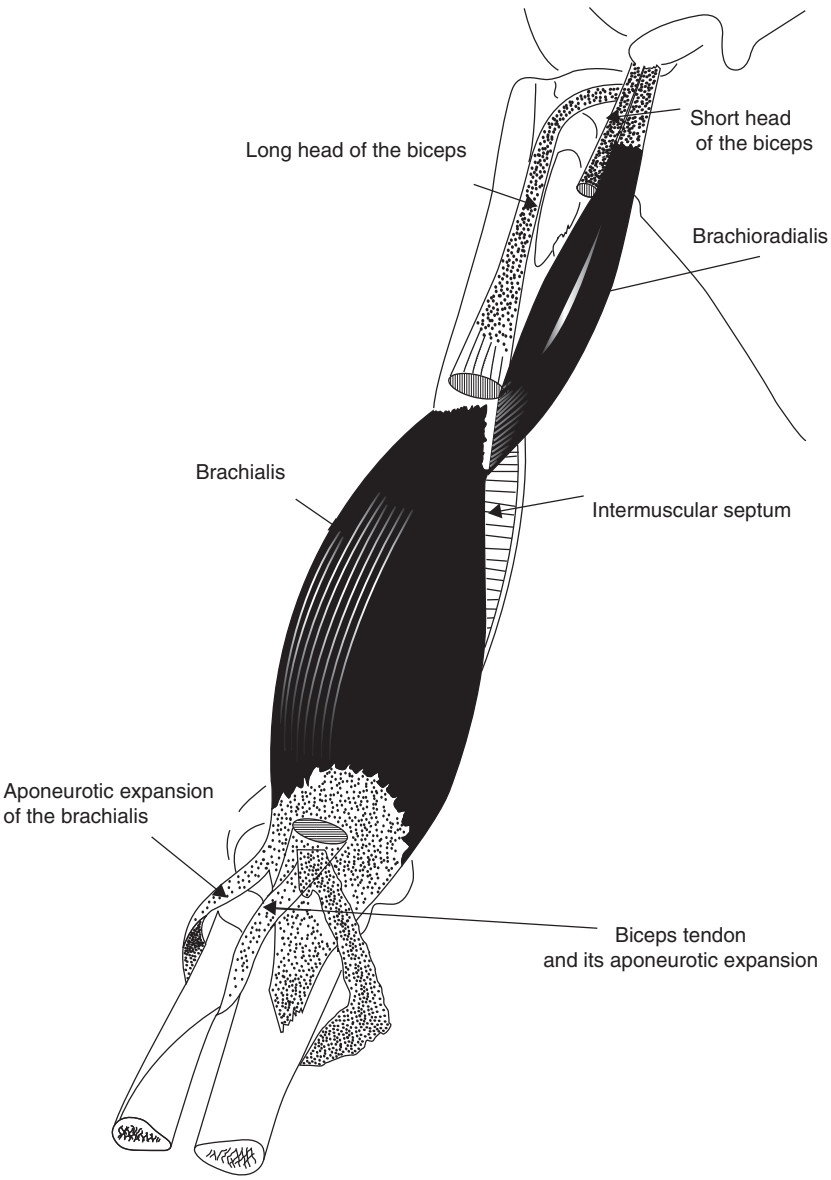

Figure 10.7

Group 1 of the flexors: the brachioradialis and biceps brachii muscles, showing the particular route of the tendon of the long head of the biceps. Adapted from Brizon and Castaing.[2]

The two muscles are flexors of the forearm on the arm. The biceps has a supination action, when at the starting point the forearm is in pronation. The role of their two expansions is not known. It helps to keep the bellies of the forearm muscles in contact with the skeleton, thus avoiding the formation of a 'bow string' position as the elbow flexes.

Group 2 muscles arise in the forearm (Figure 10.8):

- The *brachio-radialis* muscle is located at the radial side. Its belly is close to the elbow joint. It arises from the lateral border of the humerus and from the lateral intermuscular septum. It is flattened and covers the two extensors carpi radialis. Its tendon ends at the base of the radial styloid process, forming the lateral border of the pulse groove. As a uniarticular muscle, it is essentially a flexor of the forearm. It acts as a supinator only when, at the begining of motion, the forearm is in extreme pronation.
- The *muscles of the medial epicondyle* are the pronator teres, the flexor superficialis (Figure 10.8b), the flexor carpi radialis, the palmaris longus, and the flexor carpi ulnaris. Arising near the transversal axis of flexion of the elbow, their forces do not have great leverage, thus their role as flexors of the elbow is minor. However, when flexion increases, their action can be more effective. In the case of flexor palsy (lesion of the musculocutaneous nerve) Steindler[3] proposed fixing the insertions of the medial epicondyle muscles more proximally, to give them a more important effect on flexion.

All these muscles have a role in pronosupination, not only the pronator teres, which is the pronator, but also the other muscles which go to the carpus or digits. They will be studied in other chapters.

Extensor muscles

Only one (triceps) has its belly at the level of the arm; the others are muscles of the wrist and of the digits.

The *triceps brachii* (Figure 10.9) has three heads joined at the inferior part. The long head is inserted at the inferior pole of the glene of the scapula, the vastus lateralis arises from the entire length of the humeral shaft above the radial groove, and the vastus medialis arises from the shaft beneath this sulcus and from the medial intermuscularis septum. The three heads end on a tendon which is inserted on the posterior part of the superior surface of the olecranon. Classically, the unique action of the triceps is to extend the forearm.

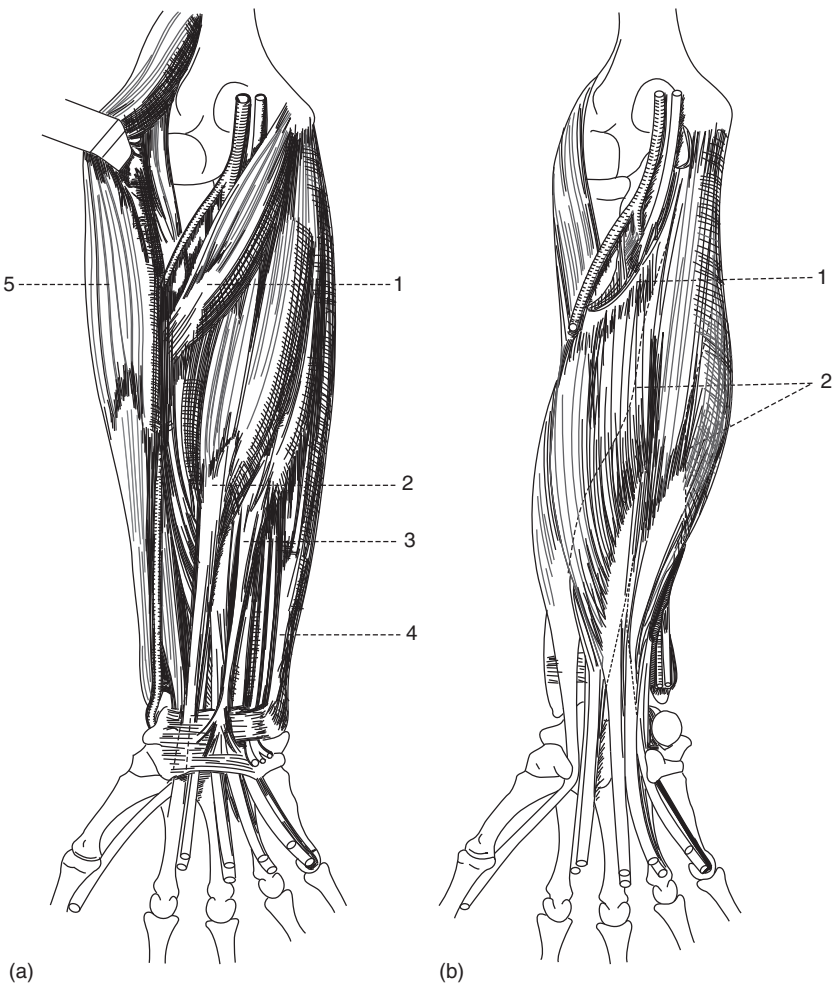

Figure 10.8

Group 2 of the flexors includes the brachioradialis and the muscles inserted on the medial epicondyle. (a) Superficial muscles. (1) pronator teres; (2) flexor carpi radialis; (3) palmaris longus; (4) flexor carpi ulnaris; (5) brachioradialis. (b) The flexor superficialis is deeper. (1) The fibrous arch of the flexor digitorum superficialis; (2) the deep digastric fascia from which the tendon of the index and ring fingers arises. Adapted from Brizon and Castaing.[2]

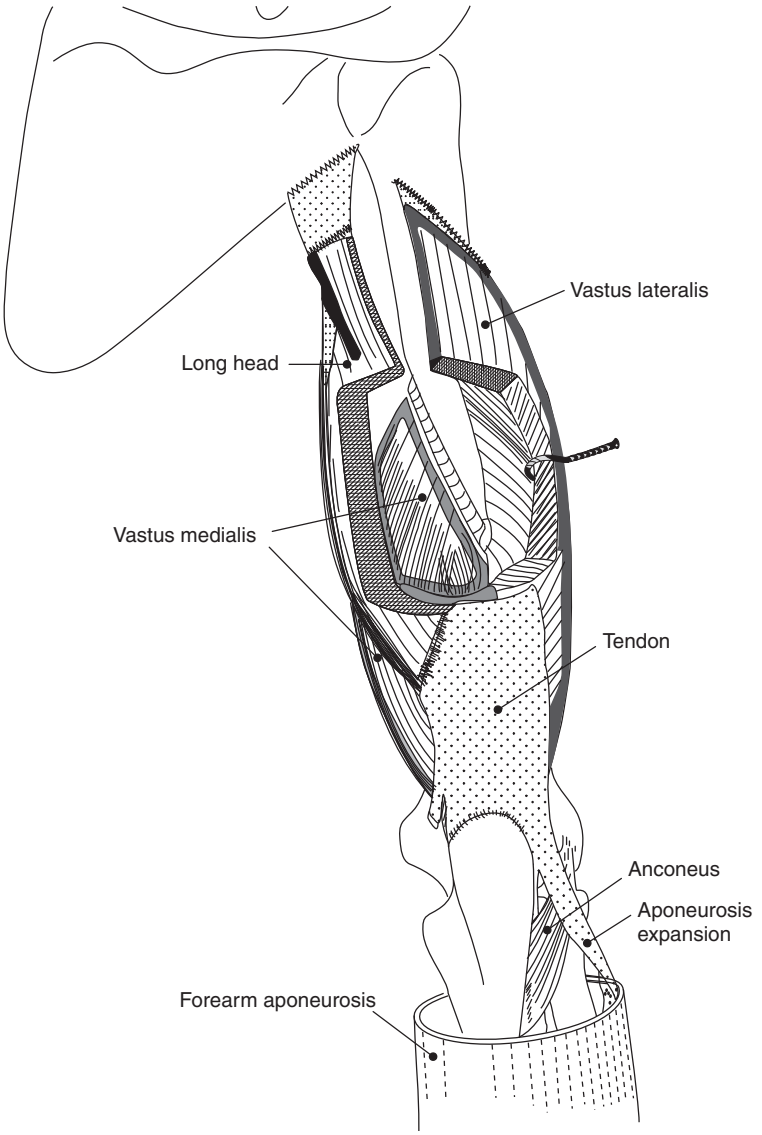

Figure 10.9

Extensors of the elbow, the triceps brachii, and its three bundles. Adapted from Brizon and Castaing.[2]

- The muscles of the forearm (Figure 10.10) are inserted on the lateral epicondyle and extend to the wrist and the digits. Once again, their insertions are too close to the transversal axis of flexion–extension of the elbow, so that the resultant forces generated are too weak to obtain extension. They are, in order from the lateral to medial direction, the extensor digitorum communis, the extensor digiti quinti, and the anconeus muscle. The last one is the unique monoarticular muscle which has an extensor action. In fact it has merely the role of a stabilizing device, the same as the supinator brevis. This will be discussed in the next section.

Pronosupination

Pronosupination is an essential movement of the elbow. It also involves the inferior radioulnar joint.

Mechanical conditions

For pronosupination some mechanical conditions are necessary. The *superior radioulnar joint* has already been described. It must be remembered that the *ulnar deviation* (which is physiologic) and that the helicoid movement of the ulna around the trochlea, leads to a displacement of the radial head in front of the capitulum. This movement is possible because of the special disposition of the lateral ligaments and the looseness of the annular and the quadrate ligaments.

In pediatric orthopedics, painful pronation occurs frequently, and is a consequence of excessive traction on the extended forearm, which leads the radial head to 'wedge' in the annular ligament. Treatment is easy and consists of mobilizing the forearm in supination associated with flexion of the elbow.

The *inferior radioulnar joint* (Figure 10.11) is a sort of satellite of the precedent. It has only one free movement, i.e. rotation, which occurs between two cylindric surfaces: the

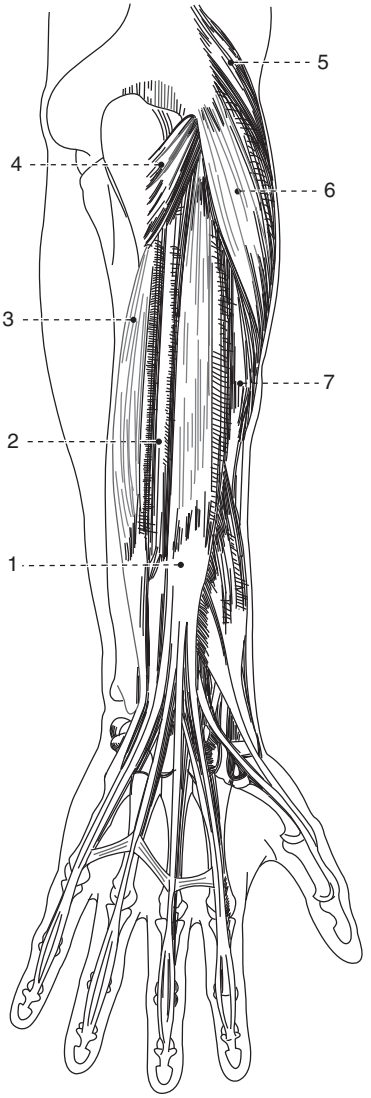

Figure 10.10

Superficial muscles of the dorsal forearm: *posterior* 1, the extensor digitorum communis; 2, the extensor digiti minimi proprius; 3, the extensor carpi ulnaris; 4, the anconeus; *lateral* 5, the brachioradialis; 6, the extensor carpi radialis longus; 7, the extensor carpi radialis brevis. Adapted from Tubiana.[4]

first one is vertical at the inferior extremity of the radius and located between the two branches of the lateral border of the bone. It is concave from front to back, and higher in the middle. The second involves two-thirds of the inferior extremity of the ulna (ulnar head) and presents two cartilagenous facets: one lateral and convex from front to back, higher in the middle, and the other surface inferior and slightly convex regarding the triangular ligament of the wrist. At the junction of these two parts the basis of the ulnar styloid is located. Between the styloid and the inferior surface, posteriorly and medially, is the site where the triangular ligament is inserted. These three zones are aligned on an oblique line, which corresponds to the circumference of the ulnar head to the greater zone articulated with the radius.

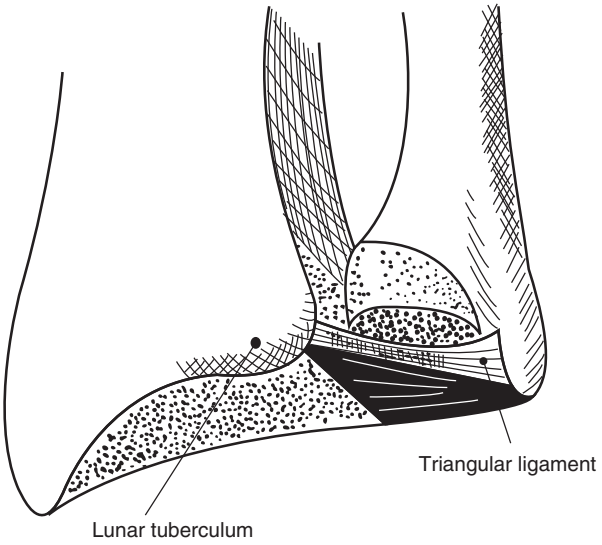

Figure 10.11

The inferior radioulnar joint. Adapted from Tubiana.[4]

The restraining structures of the inferor radioulnar joint are:

- A capsula reinforced forwards and backwards by the radioulnar ligaments, both anterior and posterior.
- The triangular ligament, which is a horizontal fibrocartilaginous structure intermediate between the ulnar head and the carpus. The lateral insertion is at the level of the inferior border of the sigmoid cavity of the radius.

The *two bones of the forearm* have a special form:

- the *radius neck* is oblique downwards and inwards up to the bicipital tuberosity
- the *rotation axis* of the two radioulnar joints is unique and corresponds to the axis of the neck of the radius
- the *radial shaft*, which has a prismatic section, presents a curve on the medial side, called the pronation curvature, allowing the crossing of the two bones of the forearm, in the best conditions, in extreme pronation this curve becomes posterior and the radius 'steps' over the ulna. The ulnar diaphysis presents a double curve, figuring 'S', with a superior concavity on the ulnar side, and an inferior one on the lateral side.

The radius and ulna are held together by *the interosseous membrane*, which extends between the inner border of the radius and the lateral border of the ulna. Its texture is somewhat complex, but it is very well adapted to movements of the radius, which is the mobile bone. The interosseous membrane (Figure 10.12) comprises two layers of ligamentous fibers: the anterior has fibers obliquely inwards and downwards, the posterior layer has fibers conversely oriented. It is reinforced by the Weitbrecht ligament, which extends from the coronoid process to the bicipital tuberosity.

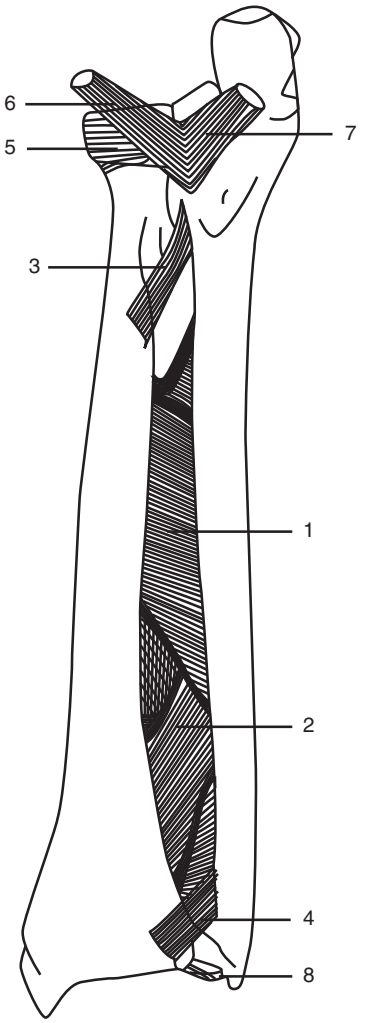

Figure 10.12

The interosseous membrane: 1, anterior layer; 2, posterior layer; 3, The Weibrecht ligament; 4, anterior ligament of the inferior radioulnar joint; 5, annular ligament; 6 and 7, anterior ligament of the elbow; 8, triangular ligament. Adapted from Kapandji.[5]

The superior border of the membrane forms an arch, above which the anterior and posterior regions of the forearm join each other: this route is taken by the interosseous and posterior neurovascular bundles. In general, the interosseus membrane impedes the descent of the radius in relationship with the ulna, but allows the movements of the radius.

Movements of pronosupination can be evaluated along a measurement which must be precisely determined

- According to Merle d'Aubigné,[6] the reference plane is given by the anatomic position of the forearm, i.e. the hand facing anteriorly with its medial border on the side of the body, with the elbow in complete extension. Then supination is equal to 0° and there is a possiblity of pronation of about 160°. Clinically, the movement of pronosupination is recorded, for example from 60° to 120°.
- For the majority of authors, pronation and supination are quoted on two sides of a reference plane given by the following position: the elbow is flexed at 90°, the arm is at the side of the body, and the hand is vertical with the thumb oriented upward (position 0). Thus, with an outwards rotation of the hand and the forearm, there is theoretically a possible supination of 90°, and an inwards rotation gives a possible 90° of pronation.

This explains the difficulty of evaluating the movements, and it is even more complicated when other displacements or stiffness of the different joints of the limb co-exist.

Muscles of pronosupination

A priori, only four muscles have adequate insertions and orientations of their fibers and can be considered to have a major role: two are supinators – the biceps and the supinator brevis – and two are pronators – the pronator teres and the pronator quadratus. In fact, all the muscles of the forearm exert an action in one or other direction, and this renders the biomechanical study more complex.

The supinators

The supinator brevis (Figure 10.13a) This is a flattened muscle that turns around the lateral surface of the elbow. It is divided into two heads, superficial and deep:

- The superficial head arises partially at the top of the radial epicondyle, by a tendon which reinforces the lateral ligament of the joint, and also from the posterior crest of the lesser sigmoid cavity of the ulna. Its fibers roll around the head of the radius and insert on the oblique part of the anterior border of the bone.
- The deep head has the same origin, but the ends of the fibers arising from the epicondyle insert on the neck of the radius up to the bicipital tuberosity; the fibers coming from the ulna insert laterally and below the tuberosity.

The two bellies are separated by a cellular interstice, where the posterior branch of the radial nerve passes. It enters there under the superior border of the superficial head of the muscle, which resembles an arch. This anatomy is important surgically because the nerve's branch can be compressed at this site.

By definition, the supinator brevis produces a rotation–supination of the forearm, and then of the hand. It seems logical to think that the horizontal fibers which turn around the radial head play the main role. They act as a couple with the tendon of the biceps. The forces exerted by the muscle on the radius have a dorsal component which is counterbalanced by the pronator teres and all the muscles of the anterior part of the forearm, which have a pronation action. The fibers which arise from the lateral epicondyle have yet another role, which is to maintain the narrow

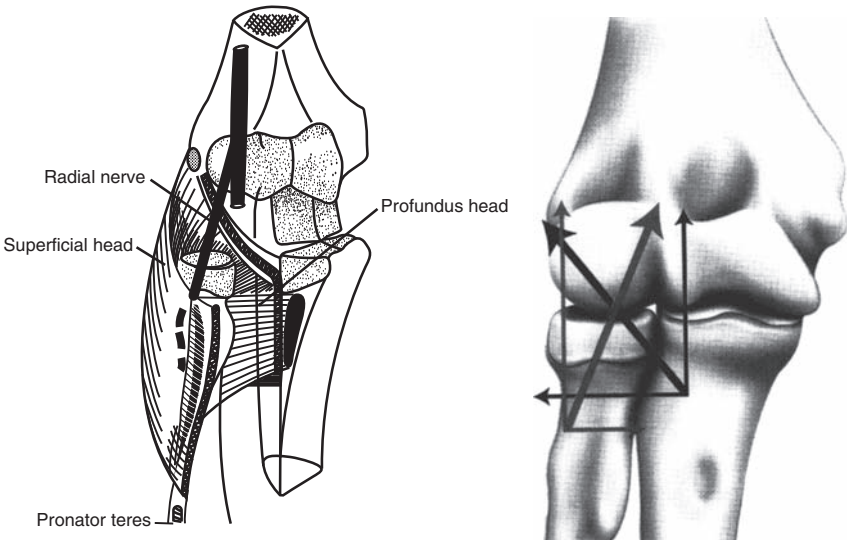

Figure 10.13

(a) The supinator brevis and its two heads with the route of the posterior radial branch. Adapted from Brizon and Castaing.[2] (b) The forces exerted by the supinator brevis apply the head of the radius on the capitellum. They are balanced by the posterior forces exerted by the anconeus. Adapted from Delamarche et al.[1]

contact between the radial head and the capitellum, and also to maintain the radius head in the lesser sigmoid cavity. This role is well illustrated in Figure 10.13b, in which the forces are analyzed by the parallelogram method. The resultant force of the two types, arising from the horizontal and vertical fibers, has an oblique direction, crossed by the fibers of the anconeus muscle.

This disposition is favorable to maintain permanent contact of the head of the radius and the capitellum in every position of flexion of the elbow.

The biceps brachii This has already been described. Its supinator action is dependent on the initial situation of the two forearm bones. Also, and by bringing into play the forces of the extensor muscles, this supination effect can be dissociated from the action of flexion. Inversely, it can only be a flexor of the elbow for the same reasons of antagonistic forces. The lever arm of the biceps is not very important, although it slightly increases during flexion. However, the expense of energy is less in this anatomic situation.

The other muscles with a supination action These are:

- The brachioradialis, which is a supinator only when, at the beginning of movement, the forearm is in full pronation, and only up to the neutral position.
- Muscles of the deep layer of the posterior region of the forearm (Figure 10.14), which, having an oblique direction because they are oriented towards the thumb, certainly have a role – blocking the radius and the interosseous space in full supination – but it is difficult to quantify this role. The contraction of these muscles can be felt when the position is accentuated.

Motor innervation of these muscles is provided by the musculocutaneous and radial nerves.

The pronators

The pronator teres (Figure 10.15) This is a short and thick muscle with two heads:

- The first arises from the medial epicondyle and the neighboring intermuscular septum. It is the most medial muscle of the group of muscles inserted on the medial epicondyle.
- The other arises from the coronoid process outside the brachialis. Between these two bellies there is an interstice, where the median nerve passes, and which can be compressed in some pathologic conditions (Volkmann's contracture, for example).

The united fibers insert on a flat tendon which passes under the lateral muscles and inserts on the middle part of the lateral surface of the radius. This muscle is a pronator and, in addition, it has a flexor action on the elbow due to its epicondyle head.

The pronator quadratus This is located on the inferior quarter of the forearm. It has insertions on one fixed element (represented by the ulna) by muscular fibers, and on the other, mobile by an aponeurotic lamina on the inferior quarter of the anterior face of the radius and on the lateral border of the bone. This disposition is well adapted to pronation, which can reach 90°.

Other muscles with a pronation action Because of the oblique direction of their fibers, all the medial epicondyle muscles also have a pronation action in addition to their flexion action. They are multi-articular muscles and their effect on the wrist is important.

It is noticeable that the pronation action is well illustrated in the radial club hand, although the radius and the pronator muscles are absent.

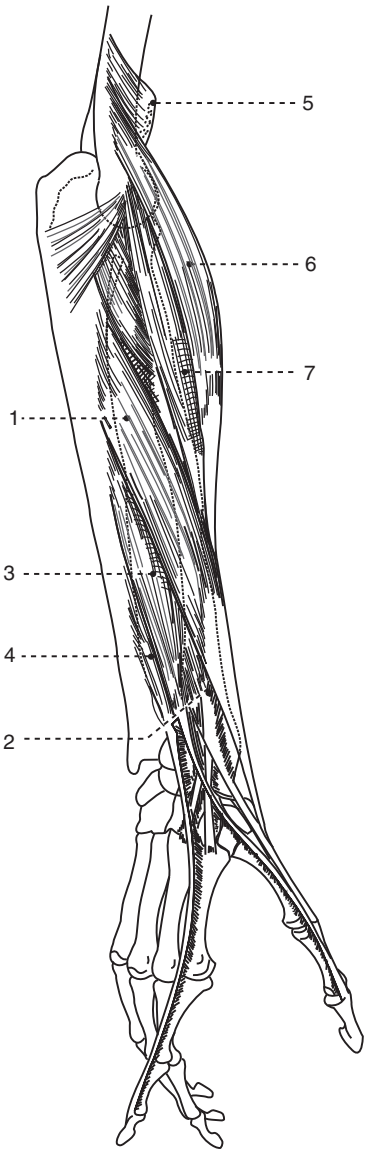

Figure 10.14

Deep muscles of the dorsal forearm are supinators. *Posterior*; (1) abductor pollicis longus; (2) extensor pollicis brevis; (3) extensor pollicis longus; (4) extensor indicis proprius. *Lateral*; (5) brachioradialis; (6) extensor carpi radialis longus; (7) extensor carpi radialis brevis. Adapted from Tubiana.[4]

With the exception of the brachioradialis, which is innervated by the radial nerve, all the pronators are supplied by the median nerve.

Conclusion

To conclude this study of the biomechanics of the elbow and pronosupination function, it seems useful to repeat that, from a dynamic point of view, all the muscles work

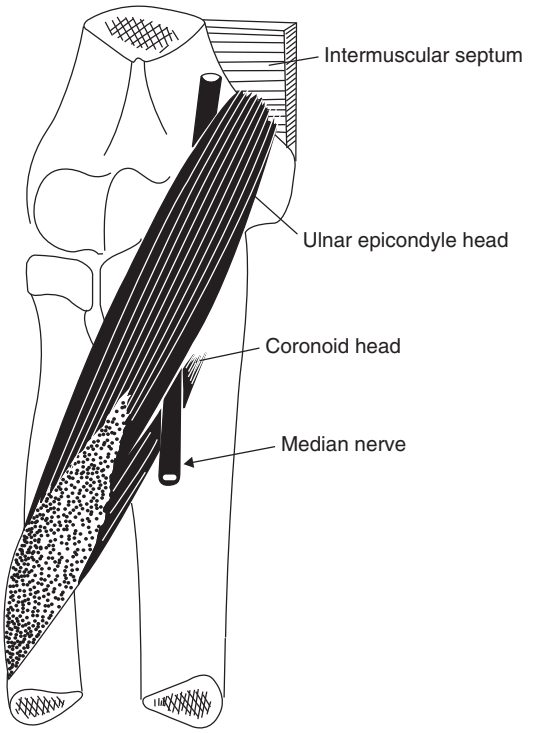

Figure 10.15

Pronator teres. Adapted from Brizon and Castaing.[2]

simultaneously and together. The muscles involved are not only the elbow or forearm muscles, but the entire musculature of the limb and of the whole organism.

This is an important notion when the surgeon wants to improve the function of a limb in which a destruction or palsy has disturbed the physiologic balance. Only resultant forces act in a movement, at the same time for the displacement of the bones, and also for the stabilization of the proximal or distal parts of the limb during object handling.

The surgeon must bear these physiologic facts in mind. In particular, when a tendon transfer is proposed, it is not sufficient to evoke the problem of antagonistic muscles, neither to take into consideration the strength or the course of the muscle to be used, but it should be paramount to determine the effect of the newly created global muscular situation. And this is very hard, if indeed possible.

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11 The paralyzed elbow

11A Flexion and extension

Alain Gilbert

Paralyses of the elbow can occur after brachial plexus injuries, direct peripheral nerve lesions, or muscle destruction. After a brachial plexus injury, the paralysis may be due to insufficient spontaneous recovery or failure of the repair. Extension paralysis is more common as, unlike flexion, it is not usually addressed as a priority. It is considered as less limiting than the flexion deficit – however, most patients find this defect a handicap. Flexion can be produced by the biceps, the brachialis, or, under some circumstances, the brachioradialis. For extension, the only active muscle is the triceps. An active life is possible, although uncomfortable, without a triceps, but the function of the whole upper extremity is impaired by the lack of elbow flexion. This situation will lead to a systematic surgical attempt to restore flexion.

Tendon transfers for elbow flexion

A variety of muscles around the shoulder and upper extremity can be used for elbow flexion. The difficulty arises when the paralysis is extensive and many regional muscles are paralyzed or weak, as in brachial plexus injuries. The volume, length, and shape of the muscle will be decisive factors.

After using all these muscles, our experience, which is similar to most authors, shows that the two best procedures are:

- transfer of the latissimus dorsi and
- Steindler procedures.

Other techniques may give good results but have some disadvantages:

- the triceps gives a strong flexion but the loss of extension is not benign
- pectoralis major is a strong muscle, but too short to reach the biceps tendon
- pectoralis minor needs extensive dissection and it is thin and not strong enough to completely replace a biceps, it can, however, be used to reinforce a weak biceps
- other muscles (sternocleidomastoid, abdominal muscles) have been used, but cannot be considered as standard procedures.

Latissimus dorsi transfer

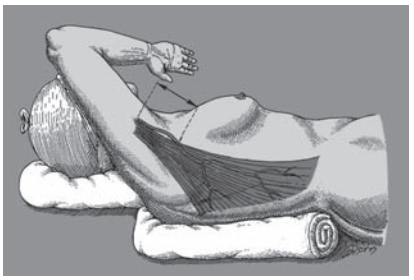
The latissimus dorsi is a large, well-innervated muscle. It is often spared in mid or lower plexus injuries and represents one of the best choices for elbow flexion reconstruction. Its anatomy is well known, but two points must be stressed:

- Over the entire muscle, skin arterial branches allow a skin flap to be raised, which may be very useful. Nevertheless, the density of skin branches is more important on the anterior border.
- Very proximally, the neurovascular bundle divides into an anterior and a posterior branch, which in fact creates two separate bellies of the muscle. These bellies can be raised independently (Figure 11.1).

Incision

Starting from the axilla, the muscle inserts on the spine and the posterior iliac crest. The incision can follow this direction or can travel anteriorly towards the axillary midline, in order to avoid ugly dorsal scars. One has to remember that the anterior border is the thickest part of the muscle. The flap, when needed, is elliptic, in a longitudinal direction, and has to be included in the incision.

For elbow flexion reconstruction, a complete latissimus dorsi muscle is too bulky to enter the anterior arm and replace the biceps. In these circumstances the muscle is either transferred completely, with a flap to augment the arm space, or it is only partially transferred. In paralytic



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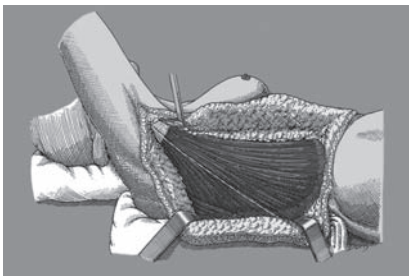

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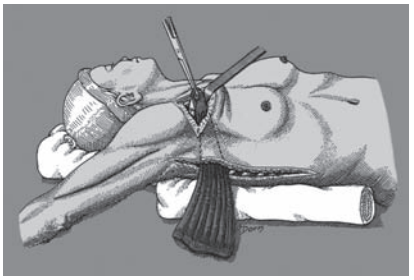

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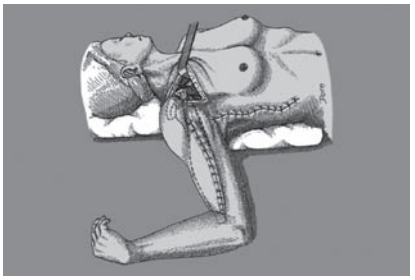

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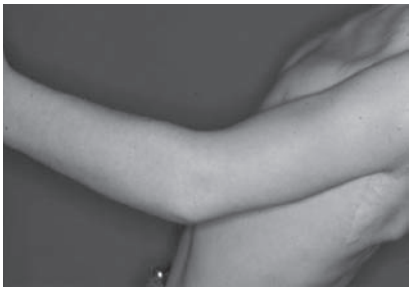


(e)

(f)

Figure 11.1

(a) The vascular pedicle is about 10–12 cm long. (b) The artery divides in two, creating two independent muscles. (c) The muscle is freed at each extremity. (d) When the muscle is large, it is necessary to use a skin flap. (e and f) Result after latissimus dorsi transfer.

cases, the recovered muscle never regains its original bulk and can be transferred in totality.

Flap elevation

After skin incision, the muscle appears immediately. The fascia should be preserved, in order to facilitate gliding in the future. The anterior border is found first and followed downwards and upwards. When approaching the axilla, care must be exercised close to the vessels. The posterior border is then found and elevated. The insertion to the tip of the scapula is sectioned. When the muscle is freed from its deep attachments, the distal limit is decided by measuring the distance between the coracoid process and the elbow. The muscle is cut, with good hemostasis. It is then lifted retrogradely. Once the muscle is raised, the neurovascular bundle is visible, and must be treated with care. It will be dissected up to the axilla. Some descending branches of the artery are ligated to provide more freedom. The tendon of the muscle is separated from the teres major tendon. This separation must be done on the posterior aspect, where the tendons are separated. The tendon is followed to the humerus and sectioned close to the bone. The muscle is then free on its neurovascular pedicle.

Preparing the insert

On the anterior aspect of the arm a longitudinal incision over the biceps will give access to the remnants of this muscle. Its fascia is opened and the dissection proceeds to the distal tendon. A small incision is made over the coracoid process and its tip is freed. A tunnel is prepared from the posterior axilla to the anterior opening and the muscle is passed slowly, avoiding rotation, kinking, or tension on the pedicle. When the muscle is placed on the anterior part of the arm, the tendon is passed subcutaneously to the coracoid process and held with a temporary suture.

Insertion

The distal sutures are done first. The difficulty is that the distal part of the muscle is purely muscular, which hinders

the suture with the biceps tendon. The muscle is usually sutured with a long, side-to-side suture to the biceps tendon. In some instances, where the latissimus looks weak and in order to give a better arm lever, the suture can be done more distally and directly on the ulna. A hole is drilled in the ulna, 4–5 cm from the elbow, and the distal latissimus is fixed with transosseous sutures. Once the distal fixation has been done, the skin over the arm and elbow is closed, as the dorsal skin. The elbow is flexed at 120° and the proximal tendon can be fixed with proper tension on the coracoid process, using intraosseous fixation. It must be insured that the tension on the pedicle is not excessive. Immobilization of the elbow at 120° of flexion is maintained for 6 weeks, and then physiotherapy is started.

Steindler procedure

The principle is to move the insertion of the medial forearm muscles proximally on the medial epicondyle.[1–3] Elbow flexion is obtained when the wrist and finger flexors contract. To obtain a good flexion it is mandatory to extend the wrist, or at least to resist flexion. Paralysis of wrist extension is a contraindication of this operation.

Incision and dissection (Figure 11.2)

Incision is made on the medial aspect of the elbow, extending to the forearm and the arm. The ulnar nerve is dissected free and followed to its entrance into the flexor carpi ulnaris (FCU). The posterior belly of the muscle is lifted from the ulna. Then the whole bulk of the muscles inserting on the medial epicondyle, comprising the FCU, the flexor carpi radialis longus (FCRL), and the flexor digitorum superficialis (FDS), is freed to their insertions. A piece of bone is cut with a saw from the epicondyle, holding all the muscular insertions. In children, it is not necessary to take bone but only periosteum. The distal dissection of the muscles must go far enough to allow good mobilization of the bony–muscular flap, especially the deep insertions. The anterior aspect of the lower humerus is approached medially, lifting the biceps and coraco-brachial muscles. The median nerve is protected anteriorly. The piece of bone with the muscular insertions is moved proximally as high as possible on the humeral shaft.

Fixation

The bone is fixed with a 3.5 mm screw on the anterior humerus at 80° of flexion of the elbow. In children, the periosteal slip is fixed with an anchor. After fixation is secured, the ulnar nerve should be left free and anterior to the epicondyle. The skin is closed and the elbow immobilized in a splint with 110° flexion for 6 weeks. After removal of the splint, rehabilitation is done with another extension splint of the wrist.

Triceps transfer

In severe lesions of C5–C6 roots of the brachial plexus, the biceps, deltoid, external rotators, and often latissimus dorsi are paralyzed. The triceps transfer may be a solution.[4] The operation is straightforward, and usually quite effective, but it has the disadvantage of suppressing active elbow extension, which is useful for stabilization on a table and for prehension.

Incisions and elevation (Figure 11.3)

The patient lies on his back, with the shoulder and upper extremity prepared. For the posterior dissection the arm is placed on the thorax, and on an arm rest in the second stage of the procedure. A vertical incision is made in the posterior direction. The triceps appears beneath the skin. The skin is retracted, and the ulnar nerve is isolated and protected. The distal tendon is cut close to its insertion on the olecranon, then the muscle is lifted from distal to proximal. There are many attachments, and hemostasis must be precise as bleeding is extensive. At the union of the middle and lower third of the humerus, the radial nerve is dissected and protected. Due to the large number of vascular and nervous branches entering the muscle, it is extremely dificult to detach the muscle further than the midshaft.

Insertion and fixation

The lateral skin of the arm is elevated and the muscle is passed anteriorly. The posterior skin is closed and the triceps tendon is fixed anteriorly to the biceps tendon in flexion of the elbow, using a strong No 1 non-resorbable suture. The anterior skin is closed on a drain and the elbow immobilized at 120° of flexion for 6 weeks.

Pectoralis major transfer

A curved incision is made, extending from the 7th sternocostal joint upwards and laterally towards the lateral half of the clavicle, 2 cm below it.[5] The incision is then continued to the deltopectoral groove as far as the insertion of the tendon of the pectoralis major (PM) to the humerus. The entire muscle is exposed, together with the margin of the acromion (Figure 11.4); it is then totally detached from the clavicular and sternocostal insertions. The muscle is detached distally, together with a strip of anterior rectus abdominis fascia, approximately 8–10 cm long and 4 cm wide. Then the humeral tendon is completely divided.[6,7]

Care must be taken throughout the operation to avoid stretching or damaging the neurovascular pedicles, as both are maintained in place. The muscle (the distal part of which has been rolled into a tube) is now rotated 90° on its pedicles and the distal part of the muscle with its new prolonged tendon is passed through a subcutaneous tunnel

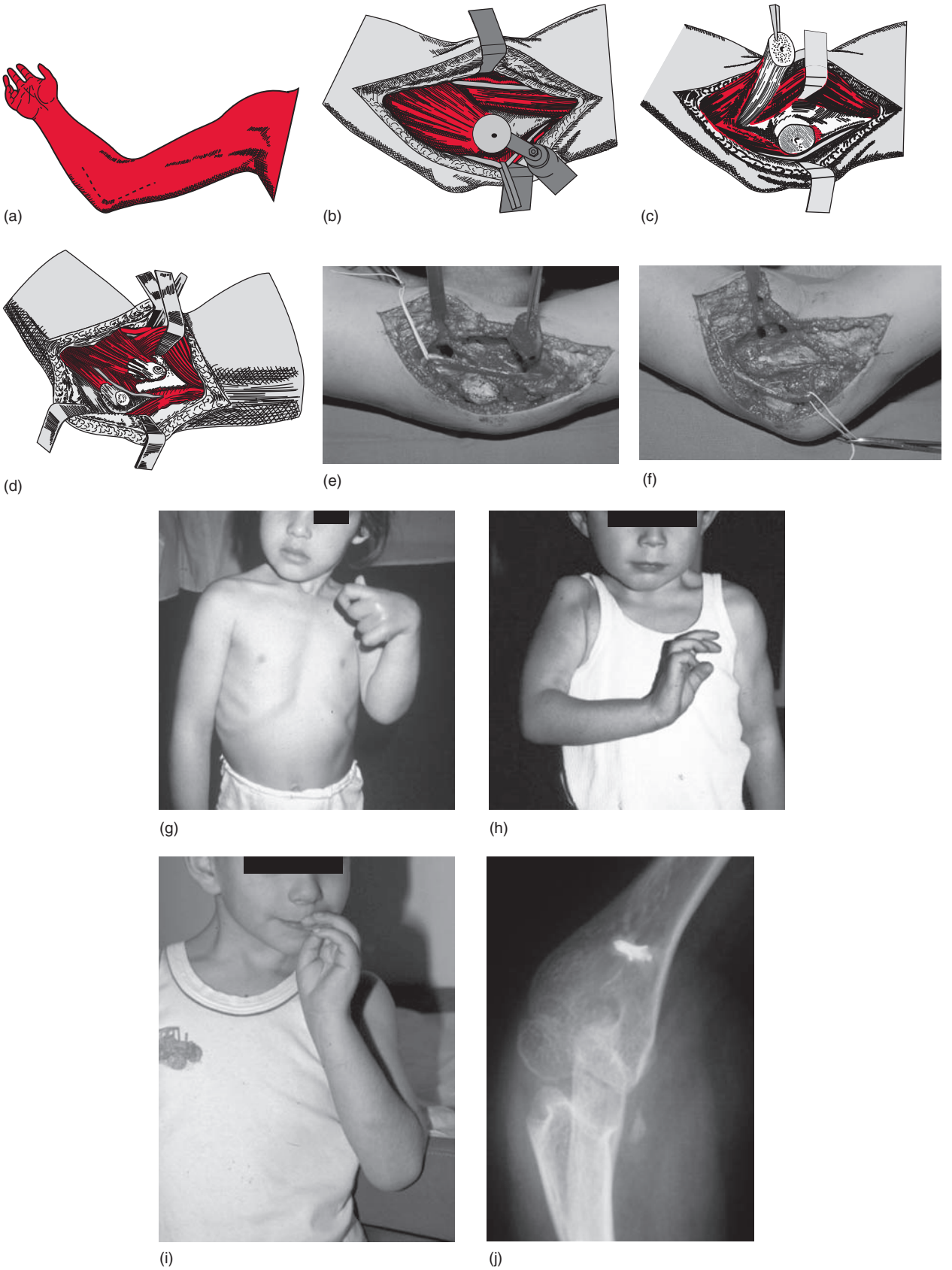
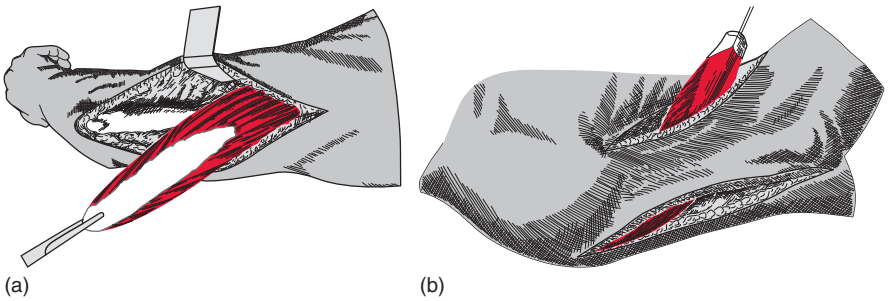


Figure 11.2

(a) The medial incision on the condyle. (b) The bony insertion of the muscles is cut with a saw. (c) The pedicled bone is lifted. (d) The bone and muscles are fixed on the anterior humerus with a screw. (e) The ulnar nerve has passed anterior to the medial condyle. (f) After fixation, the nerve is free. (g) Result: good elbow flexion but poor wrist control. (h and i) Result: good elbow flexion and good wrist extension. (j) In children, fixation is done with an anchor.



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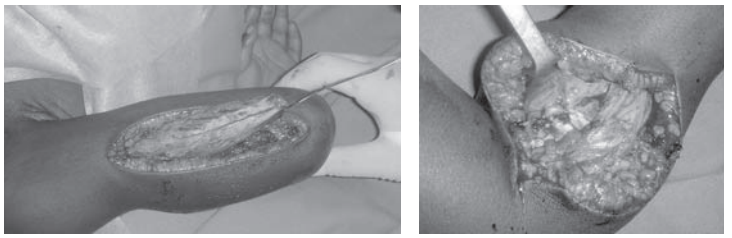

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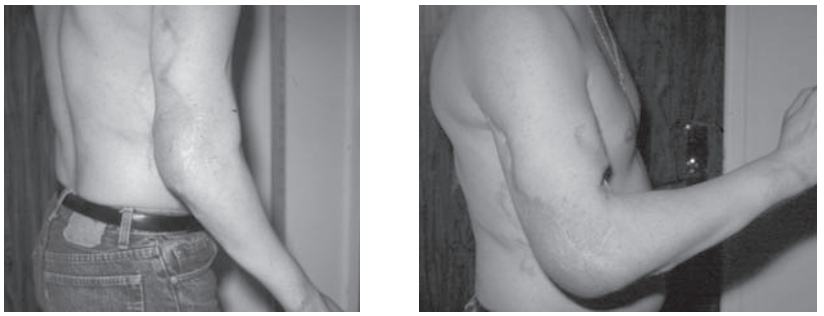
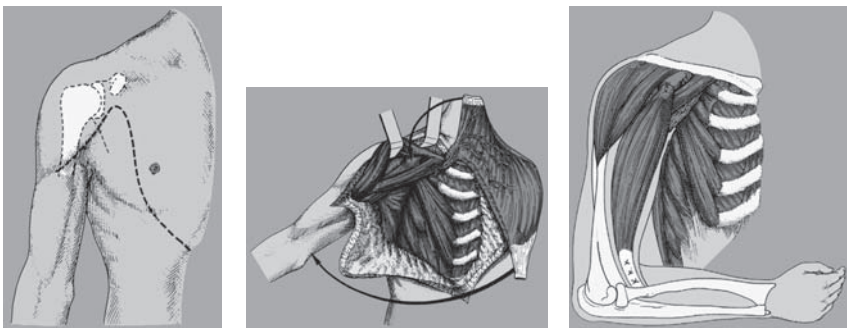


(e) (f)

Figure 11.3

(a) The triceps is sectioned and lifted. (b) It is passed laterally to the anterior compartment. (c) Dissection of the triceps. (d) Distal suture to the biceps tendon. (e and f) Result after latissimus dorsi transfer to replace the triceps.



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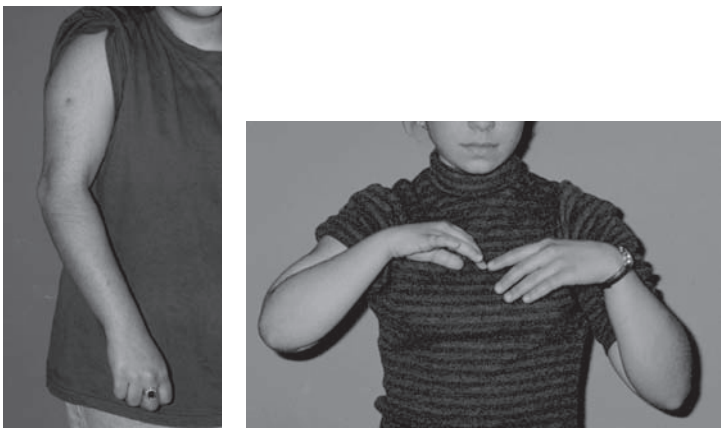


(d) (e)

Figure 11.4

(a) The incision for pectoralis major transfer. (b) The muscle is widely dissected. (c) The muscle is fixed on the biceps tendon directly or with a graft. (d and e) Result after pectoralis major transfer.

to insert to the distal tendon of the biceps at the elbow. The proximal tendon is then fixed under relative tension by means of non-resorbable sutures to the anterior border of the acromion. After careful hemostasis, suction drains are applied and skin closure is performed. Thoracobrachial immobilization, with the elbow flexed at 50° in plaster, will be maintained for 6 weeks.

Dautry et al[8] (1977) described a modification of the technique. The lateral third of the PM is left in place and the cranial tendon is fixed to the coracoid instead of the acromion.

A similar technique has been described by Tsai et al.[9] A variation comprises the utilization of both PM and minor muscles, with the aim of reducing the possible damage to both neurovascular bundles, which are located laterally to the middle portion of the clavicle, and at the same time to reinforce the strength of the PM by adding the action of pectoralis minor, the coracoid insertion of which is maintained intact. In this particular technique a wide strip of fascia attached to the coracoid (actually a part of the coracobrachialis) is woven into the PM insertion. The distal insertion is not very different from the one described above. This variation seems logical as it provides a stronger elbow flexion, assuring better protection of the neurovascular bundles.[10]

Pectoralis minor transfer

Pectoralis minor is a small muscle, and is usually not strong enough for full elbow flexion of a paralyzed muscle. In cases with a weak elbow flexion, the pectoralis minor muscle may help in strengthening the flexion. The use of pectoralis minor leaves no functional defect, but often an ugly scar. It is difficult to assess preoperatively: the examination involves pressing on the elbow with the fingers placed under the PM.

Incision and raising of the muscle (Figure 11.5)

The thorax, axilla, and upper extremity are prepared. On the thorax, the incision is done at the lateral border of the PM. The PM is reflected and the distal insertion on the ribs appears. The pectoralis minor inserts distally on the 3rd, 4th, and 5th ribs. The muscle is desinserted and dissected up to its insertion on the coracoid process, without interfering with the deep neurovascular bundle (which is often multiple).

Placing and fixing the transfer

The skin of the arm is incised to the biceps muscle. The proximal arm and axilla are undermined and the muscle passed subcutaneously. It lies on the biceps muscle but is too short to go to the biceps tendon. There are two options:

- either prolongation of the muscle and its periosteal insertions with a graft (tensor fascia lata) to the biceps tendon or, as preferred
- direct suture to the biceps fascia, using long, non-resorbable sutures extending some way on the biceps.

This suture is done in elbow flexion. The skin is sutured and the elbow immobilized for 6 weeks in flexion.

Vascularized gracilis transfer

In very severe cases, when no regional muscles are available, it may be necessary to bring a muscle from another part of the body. This vascularized transplantation implies microvascular sutures of the vessels and the nerve. Although several donor muscles can be used, the gracilis muscle is the preferred one for this indication as its shape and length are adequate. The neurovascular hila is unique, and the vascular pedicle is long (6–8 cm) and large (1–1.5 cm) enough.[11]

The choice of the recipient nerve is very important. It may be possible in cases of muscle destruction or limited paralyses to find a local recipient nerve, but most of the time this nerve has to be found in the brachial plexus. Several possibilities exist: the accessory spinal, intercostals, and nerves from the other side – C7 or, our favorite, the superior branch to the pectoralis muscle. When this nerve is relatively far away the procedure is done in two stages (Figure 11.6): in the first stage a nerve graft is inserted between the pectoralis branch and the opposite shoulder. One year later, the gracilis muscle is harvested and replaces the biceps (see Chapter 7).

Indications and results

The indications depend on the etiology and the severity of the defect.

Traumatic destruction of the muscles All techniques are possible; the simplest and most effective is the latissimus transfer, although it is not always easy to place the muscle and give it adequate tension. The most elegant is the gracilis transfer, as it almost exactly replaces the biceps and the musculocutaneous nerve can be used. The problem lies in the use of microsurgery and its potential complications.

Brachial plexus injury The indications depend on the extent of the paralysis. In a case with limited sequelae, when the shoulder is good, the latissimus can be used on the condition that it has a good recovery. If not, the Steindler procedure will give an excellent result. If the shoulder is not good enough, and especially if weak external rotation necessitates a latissimus transfer, the Steindler is the best procedure. When the paralysis is extensive, and the forearm paralyzed or very weak, or when there is no wrist extension, difficulties arise, especially if there is no latissimus. The choice is then limited between the triceps and the PM. In the worst situation (complete paralysis), the gracilis two-stage transfer is the only solution.

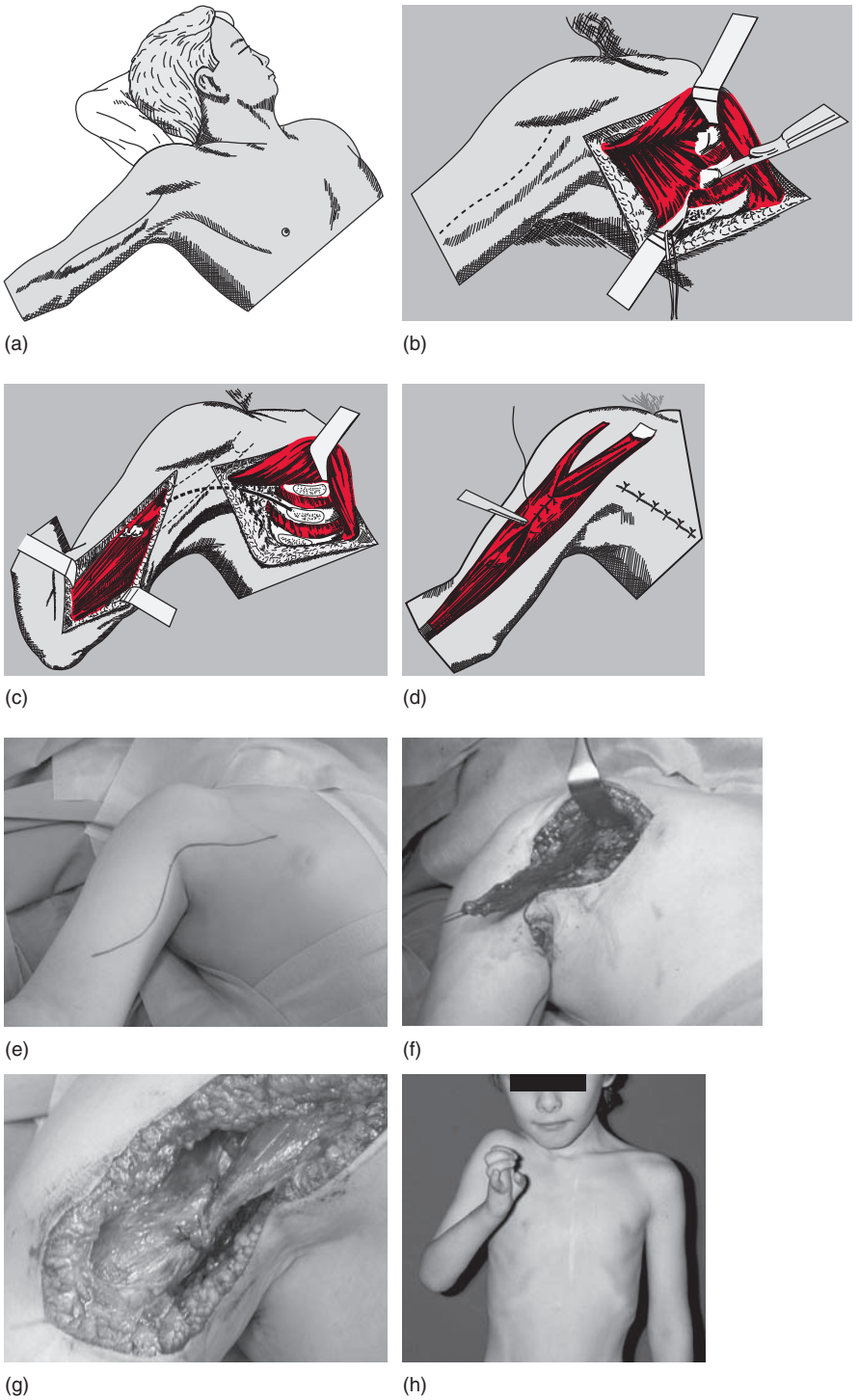


Figure 11.5

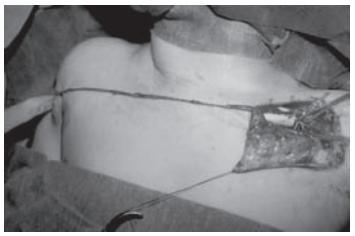
(a) Incision for pectoralis minor transfer. (b) Dissection of the muscle. (c) Fixation on the biceps. (e–g) The technique. (h) Result.

Paralysis of extension

Every effort should be made to avoid extension paralysis: secondary surgery is difficult and disappointing and justifies the new approaches of direct neurotization of the triceps motor nerves. As mentioned previously, the lack of elbow extension is not as incapacitating as flexion deficit, but most patients feel they need it. When, after spontaneous recovery or despite a nerve repair, there is no elbow extension, the possibilities are limited. If the latissimus dorsi is good, and it is not needed for elbow flexion or shoulder transfer, it can be used if the patient is very demanding, and if he understands the pros and cons of the procedure.

Latissimus dorsi transfer for elbow extension

The patient is installed in the lateral position. The muscle is raised as described previously; in most cases, due to the tightness of the skin or scarring in the posterior part of the arm, it will be necessary to transfer the muscle with a skin



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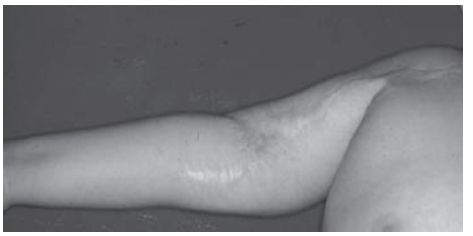

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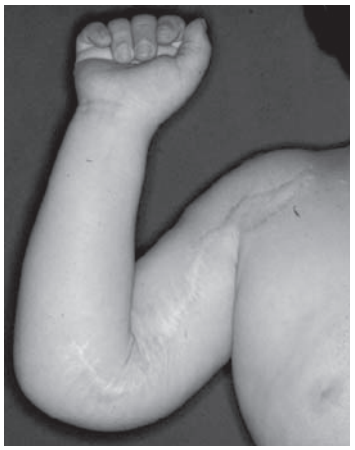

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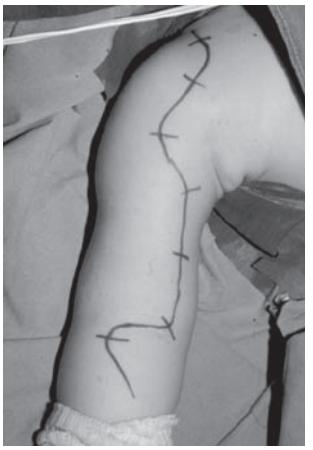

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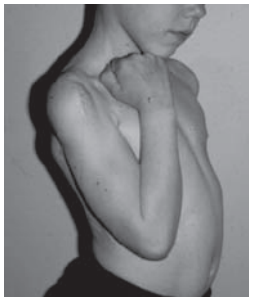

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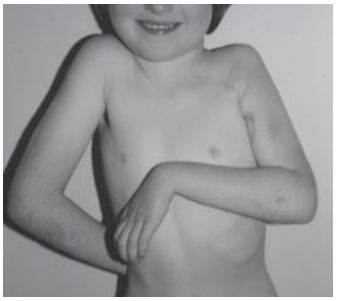

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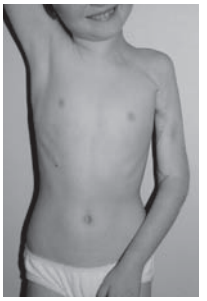
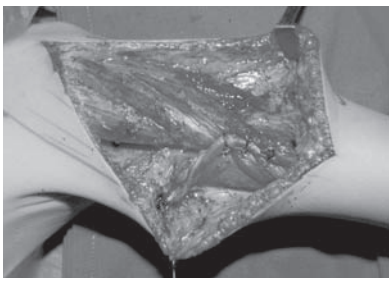


(g)

Figure 11.6

(a) The transthoracic nerve graft. (b and c) Result after gracilis transfer. (d and e) Another good result. (f and g) The patient needs to contract the opposite pectoralis in order to flex the elbow.

(a)



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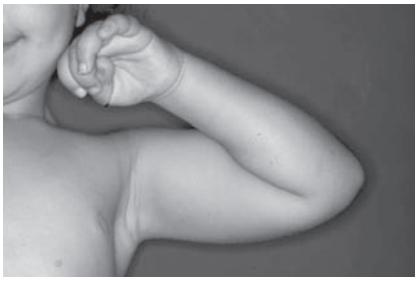

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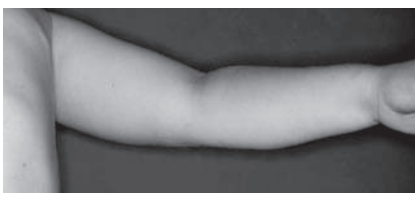

(d)

Figure 11.7

(a and b) Transfer of teres major onto the triceps. (c and d) Results of teres transfer.

island. The muscle retains its humeral attachment and is placed under the posterior skin of the arm, if possible, or with the skin island. Distal fixation is secured in elbow extension, with sufficient tension either on the distal remnants of the triceps or directly on the olecranon. The arm is immobilized in extension with a splint for 6 weeks, before rehabilitation and flexion.

When the latissimus dorsi cannot be used the choice is reduced and the triceps may be reanimated using the posterior deltoid (see Chapter 29); however in severe paralysis the deltoid is rarely of good quality. In these cases, the teres major can be used.

Transfer of teres major to the triceps (Figure 11.7)

With the parient in the lateral position, an incision is made over the proximal triceps and the lateral scapula. The tendons of teres major and triceps are freed, and sectioned from their bony insertions. The two tendons are sutured to each other in 90° abduction and in complete elbow extension. Immobilization is maintained in that position for one month, then physiotherapy is started.

The results of these operations are difficult to assess as the number of these procedures is limited and no large series have been published.[1,3,10,12,13] In our experience, the latissimus transfer will regularly give good results, although often incomplete and weak extension; the teres transfer has given disappointing results as, when active, extension is only possible in abduction. In adduction, the transfer is relaxed and not active.

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11B Forearm supination contracture in children: surgical correction and influence in the upper extremity

Christian A Allende and Alain Gilbert

Introduction

In brachial plexus lesions muscle imbalance may result, with active supinator muscles and paralyzed pronator muscles, taking the hand into a 'begging' position, with poor function and esthetics. This is generally associated with wrist hyperextension and ulnar deviation (due to the paralysis of its volar flexors and the partial activity of the dorsi flexor tendons, and because, with supination, gravity increases wrist hyperextension and ulnar deviation of the wrist, accentuated by the frequent presence of an active extensor carpi ulnaris, usually of greater power than the radial extensors of the wrist). The position assumed by the fingers is influenced by the position of the wrist and forearm – they show great weakness and paralysis and are usually flexed at the interphalangeal joints and extended at the metacarpal phalangeal joints (MP); the thumb is generally drawn in adduction. Flexion contracture of the elbow is frequently associated, due to the imbalance between a paralytic triceps and an active biceps.

Supination deformity tends to be progressive, and although it is initially susceptible to complete passive pronation, it evolves to an attitude more or less fixed in supination (secondary mainly to retraction of the interosseous membrane (IOM) and dorsal ligaments of the distal radio-ulnar joint (DRUJ), shortening of the biceps and short supinator muscle, and increasing radius curvature); followed by progressive bone deformity, with or without associated radial head and/or DRUJ dislocation or subluxation.

Supination attitude is particularly frequent after obstetrical brachial plexus palsy (OBPP) type C7–C8–D1, and patients with this deformity are not motivated to use the affected upper extremity because they have a functional deficit not correlated with their real sensory and muscular conditions.

Forearm supination deformity cannot be evaluated or treated without being included in the neurologic context of the whole upper extremity affected by the OBPP.[1] These surgical procedures should be done only in patients with active wrist extension or when wrist extension can be restored through a tendon transfer at the same or previous surgical intervention; otherwise we transform the supination deformity into a radial palsy posture.[2]

The objective of this chapter is to evaluate the indications and compare the results obtained with radius osteotomy and soft-tissue procedures for the treatment of forearm supination deformity in obstetric brachial plexus palsy (OBPP); the forearm position at rest, and shoulder, elbow, and hand function were evaluated.

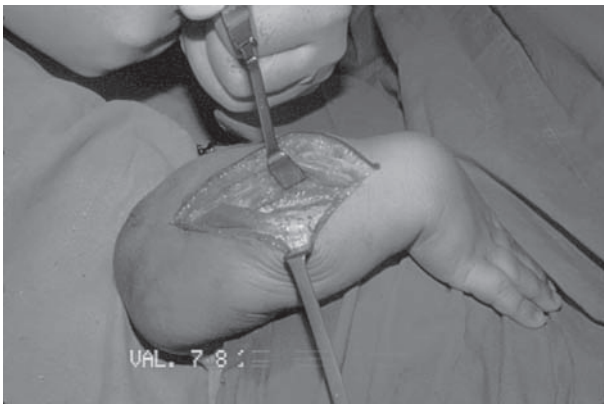
Materials and methods

Sixty-six patients who had undergone a radius osteotomy or soft-tissue procedures between 1984 and 1999, to improve a persistent supination attitude at rest secondary to OBPP, were retrospectively evaluated.

The selection of the surgical procedure depended mainly on the pathology of the involved soft tissues, the condition of the proximal and distal radio-ulnar joints, triceps function, deformities of the shoulder and elbow, and hand function. When forearm pronation could be completely restored passively, biceps rerouting alone was done; in forearms with mild or moderate soft-tissue retraction and no associated joint dislocation, biceps rerouting (with IOM release if necessary) was done; and in fixed deformities with associated bone deformity, a radius osteotomy was done. In general, a severe shoulder deformity should be corrected first; a mild or moderate flexion contracture of the elbow is not a contraindication to forearm surgery in supination deformity.

Radius osteotomy was done in 44 patients, their average age was 79.9 months, 23 were male and 21 female, the right forearm was affected in 24 and the left one in 18; 2 patients had an associated DRUJ dislocation and 3 had an

associated radial head dislocation. Other previous operations on the same limb averaged 1.4, and 21 patients had an associated procedure at the same surgical intervention to improve wrist and/or hand function.



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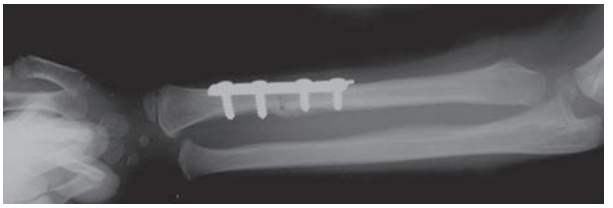


(b)

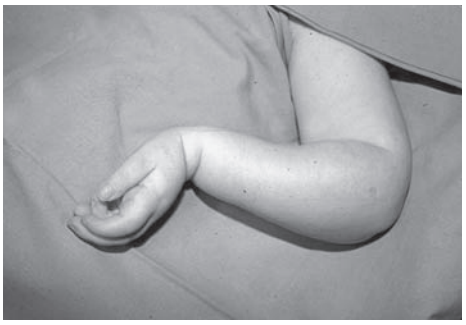
Figure 11.8

Radius osteotomy: (a) approach and level of osteotomy; (b) postoperative X-ray.

Twenty-two patients had soft-tissue procedures done, their average age was 57.4 months, 15 were male and 7 female, the right forearm was affected in 14 and the left one in 8; 1 patient had an associated DRUJ dislocation and there were no radial head dislocations in this group of patients. Other previous operations on the same limb averaged 1.2, and 9 patients had an associated procedure at the same surgical intervention to improve wrist and/or hand function. One patient was treated by a radius osteotomy and biceps rerouting at the same surgical intervention.

The osteotomies were done at the union of the middle and distal thirds of the radius, through an anterior Henry approach, always associated with release of the IOM through the same approach; the forearm was maintained in pronation by rotating the distal fragment of the radius by forced pronation of the hand and wrist and was stabilized with a 1/3 tubular plate and protected with a long arm cast in full pronation for the first 6 postoperative weeks (Figure 11.8). The intraoperative pronation obtained averaged 24.3°.

The soft-tissue procedures done were: biceps rerouting in 10 cases and biceps rerouting + IOM release in 12 cases. One patient had biceps rerouting + radius osteotomy done at the same surgical intervention. IOM release was done as described by Putti,[3] through a dorsal forearm approach and by detachment of its ulnar insertion. Biceps rerouting was carried out following the technique initially described by Schottstaedt et al[1] and Grilli,[4] and modified by Zancolli.[5] In this method a long Z-plasty is used to lengthen the biceps tendon and allow its distal segment to be rerouted around the neck of the radius mediolaterally before the two cut ends are attached together; the tendon ends are sutured side by side, at a length which will maintain pronation and at the same time allow extension of the elbow (Figure 11.9).



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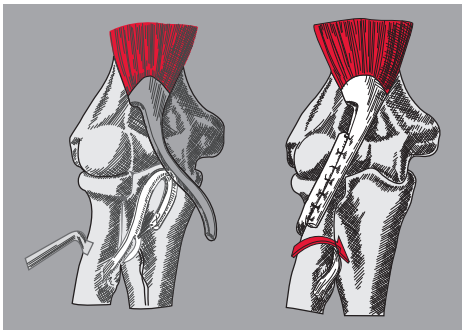

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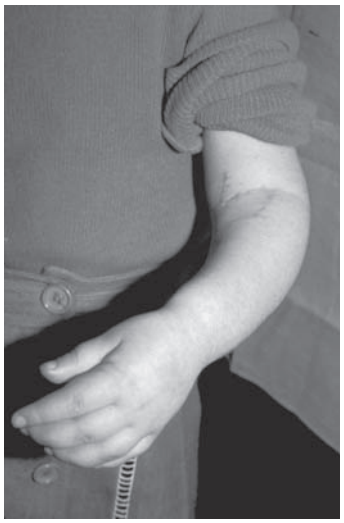

(c)

Figure 11.9

(a) Preoperative supination deformity; (b) Zancolli's biceps rerouting technique; (c) result 2.5 years postoperatively.

Results

Results were evaluated after a follow-up that averaged 64.3 months for the group of patients who had a radius osteotomy and 72.57 months for the group treated by soft-tissue procedures.

Pronosupination was evaluated by comparing: (1) preoperative degrees of supination at rest with intraoperative pronation obtained, (2) intraoperative pronation obtained with position of the forearm at rest at last control, (3) preoperative degrees of supination at rest with the position of the forearm at rest at last control (Table 11.1). The range of pronosupination was evaluated from 0° to 180°, where 0° equals full supination, 90° equals neutral forearm rotation, and 180° equals full pronation. In the osteotomy group there was a statistically significant difference in the three pairs of values compared, while in the soft-tissue procedure group there was no statistically significant difference between the intraoperative pronation obtained and the forearm position at rest at last control. When both groups were compared with each other, the osteotomy group showed a significantly worse preoperative supination deformity than that of the group that was subjected to soft-tissue procedures; although the pronation obtained intraoperatively was similar in both groups, the pronation at rest at last control was significantly worse in the osteotomy group.

Shoulder function was evaluated comparing abduction and external rotation preoperatively and at last control; there were no statistically significant differences in either group or between the two groups of patients (Table 11.2).

Elbow extension at last follow-up was significantly affected in both groups, compared to its preoperative status; in the osteotomy group elbow flexion was also significantly reduced (Table 11.3). There was no statistically significant difference when both groups were compared with regard to preoperative and postoperative elbow flexion and extension values.

Wrist and finger extension were improved in both groups, but this improvement was only statistically significant for finger extension (Table 11.4).

Table 11.1 *Pronosupination results*

Preoperation	*Intraoperation*	*Last control*	
Radius osteotomy			
31.25 (20.43)	114.28 (15.4)		$p<0.001^{*}$
	114.28 (15.4)	91.71 (23.26)	$p<0.001^{*}$
31.25 (20.43)		91.71 (23.26)	$p<0.001^{*}$
Soft-tissue procedures			
43.25 (22.8)	116.53 (12.48)		$p<0.001^{*}$
	116.53 (12.48)	107.95 (22.4)	$p<0.05^{**}$
43.25 (22.8)		107.95 (22.4)	$p<0.001^{*}$

*Significant; **non-significant.

Table 11.2 *Shoulder function*

	Preoperation	*Last control*	
Radius osteotomy			
Abduction	85.71 (28.3)	81.36 (27.1)	$p<0.05^{**}$
External rotation	1.58 (0.76)	1.73 (0.61)	$p<0.05^{**}$
Soft-tissue procedures			
Abduction	83.82 (34.79)	85.6 (42.8)	$p<0.05^{**}$
External rotation	1.43 (0.89)	1.62 (0.88)	$p<0.05^{**}$

**Non-significant.

Table 11.3 *Elbow function*

	Preoperation	*Last control*	
Radius osteotomy			
Extension	−22.05 (24.03)	−36.07 (20.56)	$p<0.001^{*}$
Flexion	131.76 (14.45)	124.46 (12.7)	$p<0.05^{*}$
Soft-tissue procedures			
Extension	−14.3 (13.27)	−30.8 (23.6)	$p<0.001^{*}$
Flexion	127.5 (19.14)	126.4 (11.14)	$p<0.05^{**}$

*Significant; ** non-significant.

Table 11.4 *Wrist and finger extension*

	Preoperation	*Last control*	
Radius osteotomy			
Wrist extension	1.71 (1.007)	2 (0.95)	$p<0.05^{**}$
Finger extension	1.18 (1.16)	2.11 (0.9)	$p<0.05^{*}$
Soft-tissue procedures			
Wrist extension	1.73 (1.27)	2.13 (0.83)	$p<0.05^{**}$
Finger extension	1.12 (0.83)	1.91 (0.66)	$p<0.05^{*}$

*Significant; **non-significant.

Complications were 2 delayed unions, 1 non-union (successfully treated by changing the plate), and 9 recurrences; all recurrences were secondary to a radius osteotomy and they were reoperated (6 had a new radius osteotomy done and in the remaining 3 cases biceps rerouting + IOM release were done).

Discussion

The results obtained show that early diagnosis and treatment of the supination attitude is effective and prevents it from becoming a fixed deformity, allowing a much better

prognostic, with a more useful hand and some active pronosupination in selected cases.

Shoulder function was not affected by any of these interventions. Elbow extension was decreased at last control after both surgical procedures, probably due to progressive biceps contracture rather than the procedures themselves. Improvement in wrist and finger extension was mainly due to better biomechanical conditions and a more physiologic positioning of the hand. The group of patients that had an osteotomy done had a worse preoperative deformity, and although the intraoperative correction of the deformity obtained was similar in both groups, there was a greater tendency of the osteotomy group to recurrences. All recurrences were secondary to radius osteotomies, probably because some passive movement was still present at the time of the osteotomy and allowed the active supinator muscles to restore the deformity; if biceps rerouting was associated at the same surgical intervention, recurrences would probably be avoided. Radius osteotomy produced a more important 'blocking' of rotatory movement in the forearm, but this did not seem to significantly impair the functional result; active and passive range of motion was not measured in these patients.

When present, the subluxation or dislocation of the radial head must be approached at the same surgical intervention. If reducible, the radial head is positioned appropriately and a capsuloplasty is done; however, continuous traction from the active biceps is frequently related to recurrences. If, instead, the radial head is irreducible (due to the bone deformity), the biceps tendon can be transposed to the ulna, in order to avoid further traction on the radial head.

Conservative treatment does not stop the progression of the disease. Various surgical techniques for reconstruction of this deformity are described in the literature; a high rate of recurrences has been shown with most of these techniques, and few significant patient series have been published. In 1923, Steindler[6] reported successful correction of forearm supination contracture in 2 of 3 patients, by section of the supinator and biceps muscles. Blount,[7] in 1940, concluded that the soft-tissue operations alone were ineffective, and proposed osteoclasis as the treatment of choice in forearm supination deformity, recommending overcorrection of the deformity because of its tendency to gradual recurrence. Burman,[8] in 1956, advocated the importance of correcting the shoulder component of the deformity first. Schottstaedt et al,[1] in 1958, and Grilli,[4] in 1959, suggested treatment by reversing the biceps function by transferring it to the site of the radial tuberosity opposite to its normal insertion. Zaoussis,[9] in 1963, proposed proximal radius osteotomy as an alternative to osteoclasis. Zancolli,[5,10] in 1967, evaluated 14 patients (8 after OBPP), treated by IOM release as described by Putti[8] and a modification of the biceps rerouting technique described by Grilli,[4] with the best functional results obtained in the cases of quadriplegia and poliomyelitis, while after obstetric palsy only a few degrees of active motion were obtained. Owings, et al[11] in 1971, evaluated 15 biceps rerouting operations in brachial plexus injuries, with 7 good results, 5 satisfactory, and 3 lost to follow-up; there were no recurrences or serious loss of motion, hand function and appearance improved, and lack of supination power was a common finding. Manske, et al[12] in 1980, described an association of biceps rerouting and osteoclasis of the radius and ulna in 2 patients with significant improvement. Seringe and Dubousset,[13] in 1977, described the three successive stages of the pathology. Lipskeir and Weizenbluth,[14] in 1993, reported no recurrences after double osteotomy of the forearm bones in 10 patients, recommending it over single bone osteotomy.

Based on our results and the results previously published, we recommend:

1. Biceps rerouting in patients with complete preoperative passive pronosupination and no joint dislocation.
2. Biceps rerouting + IOM release when there is good but not complete preoperative and/or intraoperative passive pronosupination and no joint dislocation.
3. Radius osteotomy in patients with fixed deformities, bone deformities, and minimum range of motion.
4. Radius osteotomy + biceps transfer to the ulna or brachialis tendon, in patients with associated irreducible radial head dislocation.
5. Radius osteotomy + biceps rerouting, in children with severe deformities and some passive motion, in which IOM release alone is not enough to allow an adequate passive pronosupination.

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12 Biomechanics of the wrist

René Malek

In classical medical books it is usual to divide the anatomic study of the superior limb into three parts:

- the forearm
- the wrist and
- the hand.

This division is justified by the different bones and their joints, but it is somewhat artificial concerning the functional aspect. Indeed, there is only one physiologic unit: hand muscles, which are extrinsic, arise at the level of the elbow or in the forearm. They are pluri-articular muscles, crossing several joints, which have their motion necessarily linked with each other.

In prehension, the first step is for the limb to approach the object to be handled, and it depends on movements of the scapular girdle and the elbow. Before the hand makes contact, it is positioned three-dimensionally by pronosupination of the forearm and wrist movements (flexion, extension, and ulnar and radial inclinations).

The limits of the wrist are still debated: a few authors concede a proximal limit by a horizontal plane located at the level of the inferior border of the pronator quadratus muscle. Distally, the limit is given by another horizontal plane which passes the superior borders of the pisiform and the tubercle of the scaphoid bone. Thus, the radiocarpal joint is deemed to be the unique joint of the wrist.

For most authors, however, particularly physiologists and those interested in biomechanics, the limits are quite different: the anatomic region of the wrist includes the carpus in its totality (Figure 12.1). There are two rows of carpal bones:

- the proximal row, including scaphoid, lunate, and triquetrum; the pisiform is separate
- the distal row, with the trapezium, trapezoid, capitate, and the hamate bones.

Then the wrist comprises three groups of joints:

- radiocarpal
- mediocarpal
- carpometacarpal.

Anatomy

It is artificial to study these joints separately because they have a simultaneous functional mechanics. The arrangement of the complex ligaments is such that they are shared by all the joints. However, the joint surfaces can be described individually.

Radiocarpal joint

On the *radial side*, the joint articular surface of the radius has a double obliquity: 15 to 20° in the anteroposterior view and 12 to 15° in the side view (Figure 12.2). It presents a cartilaginous surface divided into two parts, corresponding to the scaphoid and the semilunar bone. The posterior lip of this surface and the radial styloid thus have a buttressing effect. Because of the obliquity of the joint surface both forward and downward, there is a normal tendency of the carpus to subluxate anteriorly, and this is opposed by the restraining structures.

A second superior joint surface is in continuity with the preceding one, and corresponds to the inferior side of the triangular ligament which is extended horizontally from the ulna (where it is inserted at the base of the styloid process) to the inferior border of the radial glenoid cavity of the inferior radioulnar joint (Figure 12.3).

On the *carpal side* under an almost continuous cartilaginous layer (it is sometimes called the carpal condyle) the joint surface is composed of (from the lateral to the medial side) the surfaces of the scaphoid, lunate, and triquetrum. The scaphoid presents its anterior tubercle just under its cartilage surface. Its long axis is inclined by 45° to the long axis of the radius. The lunate is larger in the forward direction, and presents two anterior and posterior tubercles. In a neutral position, its superior surface straddles the surfaces of the radius and the triangular ligament. The triquetrum is not in contact with the ulnar head, being separated from it by the triangular ligament. The pisiform, which is joined to the triquetrum, is not a part of the radiocarpal joint. It can be considered as a sesamoid in the flexor carpi ulnaris tendon.

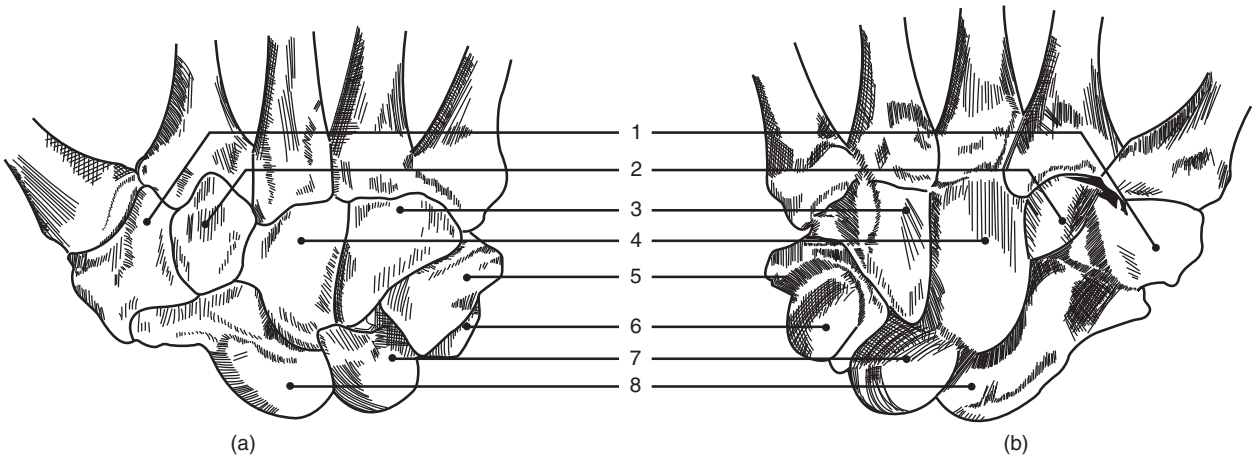

Figure 12.1

The wrist includes the entire carpus and the three joints: radiocarpal, mediocarpal, and carpometacarpal. The carpal bones. (a) Dorsal view. (1) Trapezium; (2) trapezoid; (3) hamate; (4) capitate; (5) triquetrum; (6) pisiform; (7) lunate; (8) scaphoid. (b) Palmar view. Adapted from Tubiana et al.[1]

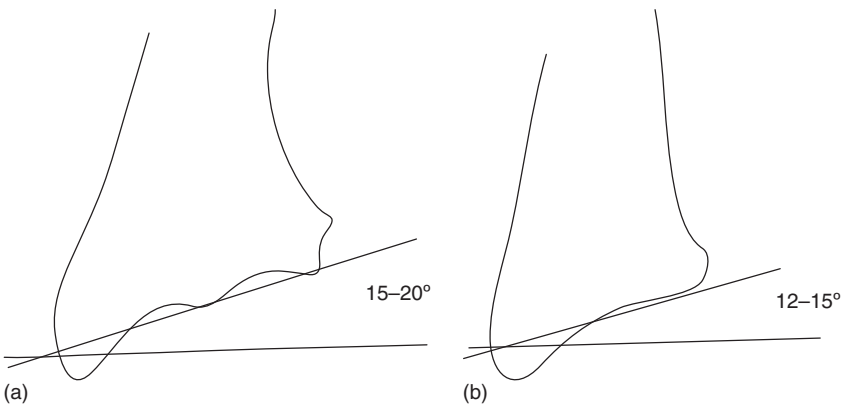

Figure 12.2

The double obliquity of the radial surface: (a) frontal view; (b) profile view.

The inferior radioulnar joint is studied separately from the radiocarpal joint, but the ligaments are very intertwined, and sometimes they share the same synovial cavity.

Mediocarpal joint (Figure 12.4)

The joint surfaces are, from the lateral to the medial side, the inferior surface of the scaphoid, which is joined to the trapezium and the trapezoid; the head of the capitate, in contact with the scaphoid under the articulation between the scaphoid and lunate; the inferior surface of the lunate; for a short distance the joint between the lunate and the triquetrum; and lastly the articulation between the triquetrum and the hamatum. The triquetrum does not enter into contact with the capitate.

Carpometacarpal joints

Arbitrarily, the trapeziometacarpal joint is studied with the thumb. The bases of the metacarpals are embedded in the bones of the second row of the carpus, and they are also in contact with each other. All these joints are poorly mobile, since they are linked by solid interosseous ligaments, but they share the same articular cavity (Figure 12.5). Only the fourth and, particularly, the fifth carpo-metacarpo-phalangeal joints have some mobility. For the fourth joint this is only in flexion and extension (barely 10°). The fifth joint also has more important flexion–extension movements. The obliquity of the joint with the hamulus renders the transverse axis of movement very inclined with regard to the axis of the hand. The flexion movement tends to give the digit a kind of opposition, because this inclination favors contact with the thumb and helps to accentuate the hollow of the palm. Nevertheless, this movement is restricted by the small size of the joint surfaces, by the stretch of the ligaments, and also by the intermetacarpal ligament, which is located at the level of the metacarpo-phalangeal joints.[2]

Ligaments of the wrist

General arrangement

The carpus, and therefore the hand, is joined to the forearm by the radiocarpal joint. However, all its other joints are

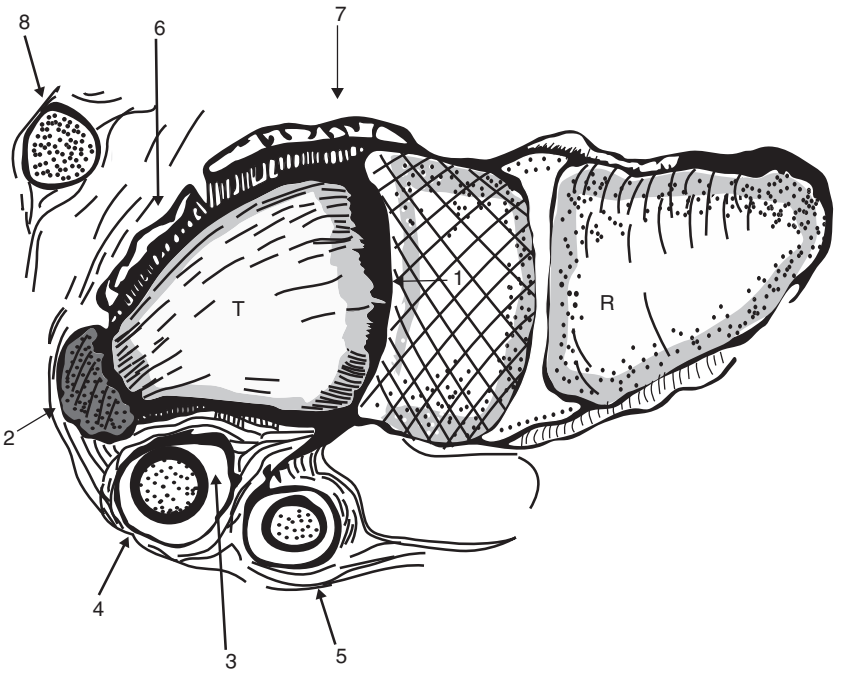

Figure 12.3

Upper joint surfaces of the radiocarpal joint (adapted from reference 1). T, inferior surface of the triangular ligament inserted on (1) the inferior border of the sigmoid surface of the radius. R, the divided surface of the radius. (2) the ulnar styloid; (3) the posterior reinforcements of the triangular ligament, joined with the dorsal muscular fibrous sheaths (4 and 5). (6) The anterior border of the triangular ligament is fixed on the ulnocarpal ligament, and (7) on the ulnar part of the radio-lunaro-triquetral ligament. (8) The tendon of the flexor carpa ulnaris. Adapted from Zancolli.[3]

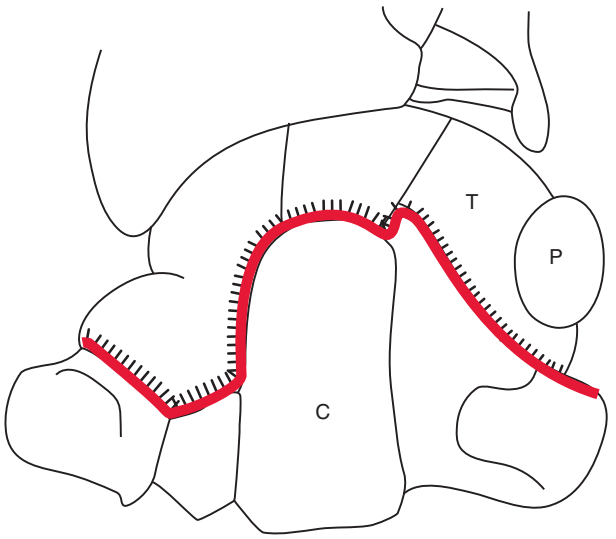

Figure 12.4

The mediocarpal joint: the capitate (C) does not enter into contact with the triquetrum (T), and the pisiform (P) is a separate bone.

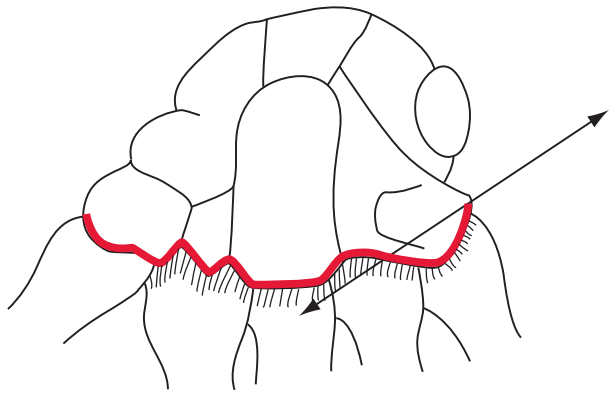

Figure 12.5

The carpometacarpal joint. The axis of flexion of the fifth digit facilitates opposition.

linked, so that their ligaments must be studied together. The main purpose of the wrist ligaments is mobility, rather than stability, in contrast to requirements in the lower limb. The small bones of the carpus are connected by interosseous ligaments, which are able to transmit sagittal displacements from one bone to the next. The movement of the bones of the proximal row are less than has been claimed by many authors, and the second row is even more rigid.

The ligaments of the carpus are numerous, and their arrangement corresponds to the different movements required: they are all reinforcement of the capsulae which are not very tight. (Figure 12.6). The ligaments cannot be very strong on the volar and dorsal aspects of the radiocarpal and the mediocarpal joints in order not to restrict flexion and extension, which are important. However, the numerous tendons crossing the two faces of the wrist play the true role of ligaments and give stability to the joints. The axial forces they create are sufficient to oppose the forces of traction generated by the weight of the handled object. To allow easy flexion and extension, the anterior and posterior ligaments are horizontal or oblique, with different fasciculi, and act like laths of a rolling shutter. The axis of these movements is almost horizontal.

The hand is suspended from the forearm bones by two collateral ligaments, ulnar and radial, which are not very strong but which have the role of limiting the inclination movements of the hand. They are also oriented in such a

way as to oppose the dislocating forces due to the inclination of the radial surface, as already mentioned. The axis of these movements is anteroposterior. There is only one central ligament which can also intervene: the vertical radioscapholunate ligament, which is very loose but carries vessels to the two small bones.

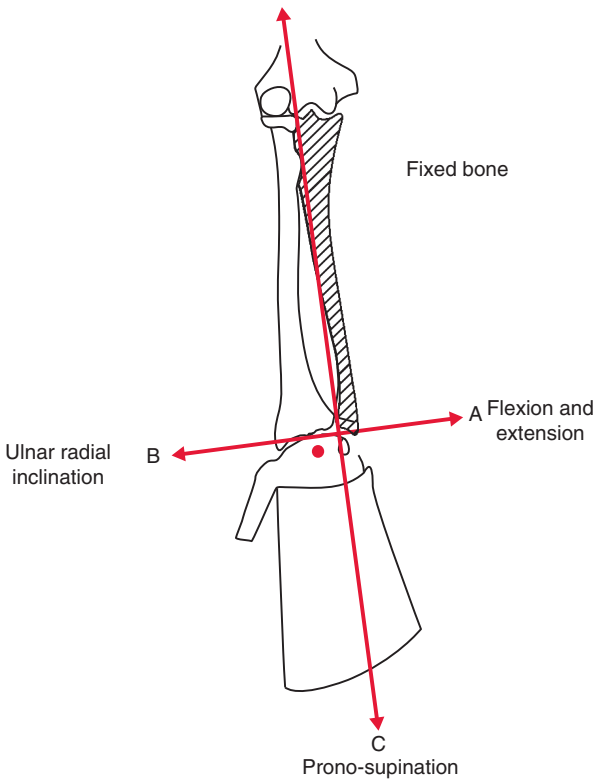

Figure 12.6

The three axes of mobility of the hand and radial ray of the forearm: A, flexion and extension of the wrist; B, radial and ulnar inclination of the hand; C, prono-supination.

The movements of pronation and supination (relevant not only to the carpus and the hand, but also to the radial part of the forearm) occur around the ulnar head. The rotational movement needs mobility at the wrist of the structures joining the carpus and the radius to the ulnar head. These include the anterior and posterior ligaments of the distal radioulnar joint and mainly the triangular fibrocartilage complex. As already noted, the pronosupination movements also need the mobility of the interosseous membrane and good mobility of the proximal radioulnar joint. Their axis is the olique line joining the two radioulnar axes of motion, it can be prolonged by the ulnar border of the hand.

Description

The interosseous ligaments (Figure 12.7) The bones of the proximal row – scaphoid, lunate, and triquetrum – are connected by strong ligaments inserted on the proximal part of each bone. There are no intrinsic ligaments between the capitate and the lunate, but there exist on the two sides of this bone: the scaphoid is connected to the trapezium and to the trapezoid, and it is also attached to the capitate. At the ulnar side, the triquetrum is attached to the capitate by a volar oblique ligament, which facilitates the ulnar sliding of the triquetrum on the inclined surface of the hamate.

As has been noted, the pisiform is not a bone of either the carpometacarpal or the mediocarpal joint. Considered as a sesamoid of the flexor carpi ulnaris tendon, it has the advantage to project that tendon in an anterior position owing to the triquetrum, and maintains its action on the ulnar side of the hand whatever the position of the carpus (during flexion–extension or pronosupination).

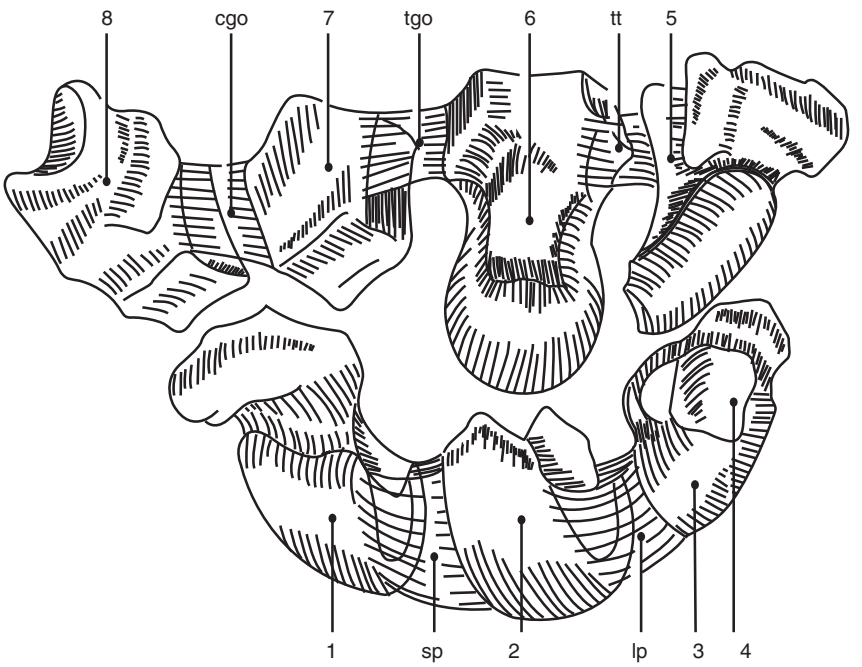

Figure 12.7

The interosseous ligaments and the two rows of the carpus. Reproduced with permission. Horizontal classification of the carpus. Proximal row: (1) scaphoid; (2) lunate; (3) triquetrum, which articulates with the pisiform (4) anteriorly. These bones are held together by interosseous ligaments (sp, scapholunate; lp, lunotriquetral). Distal row: (5) hamate; (6) capitate; (7) trapezoid; (8) trapezium. These bones are linked by three interosseous ligaments (tt, trapezio-trapezoid; tgo, trapezoid–capitate; cgo, capitate–hamate). Adapted from Tubiana et al.[1]

The pisiform is connected to all the surrounding bones, and two ligaments form two arches under which passes the ulnar vascular bundle.

The anterior and posterior ligaments Anterior ligaments:

1. The volar radiocarpal ligament has three parts:
   - The radioscapholunate ligament which can be considered as a buttress for the carpus and opposes its tendency to slide on the oblique radial surface (Figure 12.8).
   - The stylocapitate bundle which crosses the waist of the scaphoid (according to Verdan,[4] it presses on the scaphoid during pronosupination), and which is the only ligament joining the radius to the distal row of the wrist. Some fibers from this part insert on the scaphoid.
   - The radiotriquetral bundle, which is large and quadrilateral (Figure 12.9).
2. The V-shaped ligament, uniting:
   - superiorly, the capitate to the scaphoid and to the triquetrum and the pisiform (V-ligament leaves without insertion into the lunate); this ulnar part is elongated in the ulnar instability or VISI involving the mediocarpal joint
   - and inferiorly to the trapezium and to the bases of the metacarpal bones (Figure 12.9).

Dorsal ligaments are weaker than the palmar ones. They are also mainly horizontal; the triquetral sling is an oblique band which inserts into the trapezium and the trapezoid; the posterior band arises from the radius and inserts on the triquetrum (Figure 12.10).

Volar and dorsal ligaments arising from the radial styloid and inserted on the triquetrum act as a chin strap and oppose the sliding effect of forces on the oblique surface of the radius (Kuhlmann sling)[5] (Figure 12.11).

The collateral ligaments These are inserted in the proximal on a very narrow zone at the extremity of the radial styloid or the ulnar styloid. This arrangement is favorable for flexion and extension movements. The two ligaments are divided in their inferior parts, which end on the scaphoid tubercle on the radial side and on the pisiform and the triquetrum on the ulnar side.

Ligaments of the distal radioulnar joint (DRUJ) These are also ligaments of the wrist. During pronosupination they evidently maintain the contact between the radius, and then the carpus, with the head of the ulna. They are the anterior and posterior ligaments of the DRUJ and mainly the triangular fibrocartilage (Figure 12.12), which is placed transversally beneath the head of the ulna, binding the lower ends of the ulna and radius firmly together. Its periphery is thicker than its center, which is occasionally perforated. It is attached by its apex to the depression between the styloid process and the head of the ulna. This triangular disposition is beneficial to the rotation of the radius. Moreover, the styloid is posterior and located on an oblique line which corresponds to the highest zone of the articular surface of the head of the ulna.

The borders of the triangular ligament are reinforced by ligaments of the wrist, the radiotriquetral and the meniscus ligaments, described by Taleisnik,[6] which have a common origin with the triangular fibrocartilage from the dorsoulnar corner of the radius. Then the meniscus swings around the ulnar border of the wrist, to insert on the triquetrum (Figure 12.13).

These ligaments, together with the ulnar colateral ligament, are assembled in the 'triangular fibrocartilage complex' which is also associated with the ulnar ligaments borne on the radius. This complex is inevitably twisted during pronation or supination. The tension created during pronation is balanced by the sliding of the triquetrum on the superior surface of the hamate.

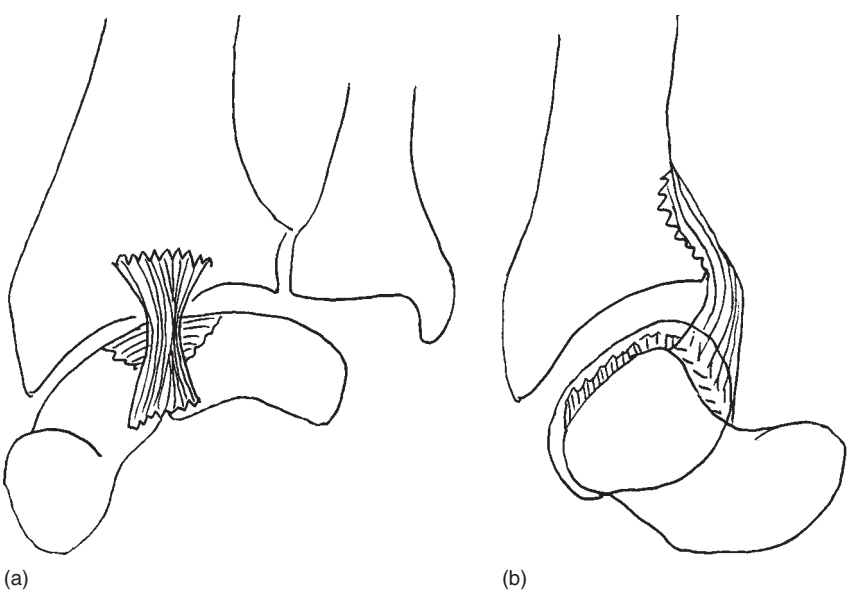

Figure 12.8
The radioscapholunate ligament: (a) front view; (b) profile view.

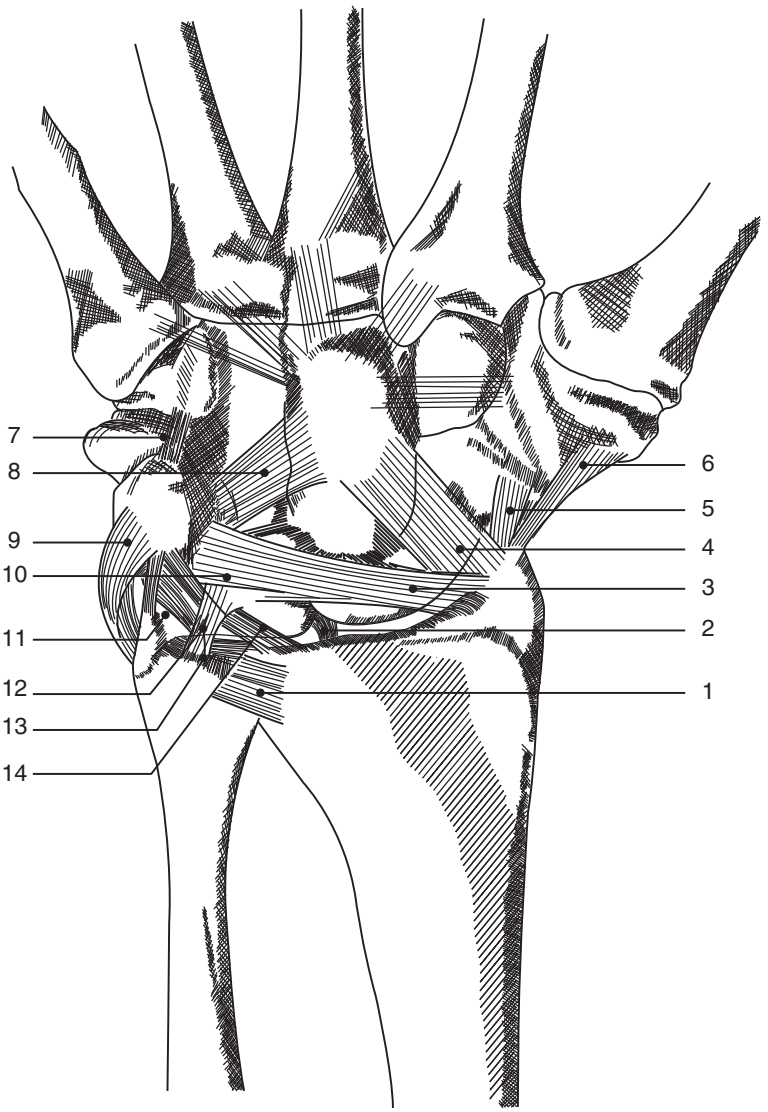

Figure 12.9

Volar ligaments of the wrist. (1) Palmar ligament of distal radioulnar joint; (2) radioscapholunate ligament; (3) stylolunate-triquetral bundle of the volar radiocarpal ligament – there are weak points in the anterior capsule between these bundles; (4) stylocapitate bundle of volar radiocarpal ligament – this thick ligament, which crosses the waist of the scaphoid, is the only ligamentous structure linking the radius to the distal carpal row; (5) styloscaphoid bundle of volar radiocarpal ligament; (6) radial collateral ligament; (7) palmar triquetrocapitate ligament – medial branch of volar ligamentous V, the other branch being formed by the volar radiocapitate ligament; (8) pisohamate ligament; (9) extensor retinaculum; (10) ulnar collateral ligament; (11) radiotriquetral meniscus; (12) radioulnar triangular fibrocartilage; (13) ulnotriquetral band of palmar ulnocarpal ligament; (14) palmar radiotriquetral ligament. Adapted from Tubiana et al.[1]

The volar radiocarpal ligament This is also a structure of the wrist (see Chapter 14). It consists of two parts:

- the carpi-volar ligament, which arises from the tendon of the flexor carpi ulnaris and extends the superficial aponeurosis of the forearm in front of the radiocarpal joint and which surrounds the tendon of the flexor carpi radialis
- the anterior annular ligament or palmar retinaculum, which is at the level of the mediocarpal and carpometacarpal joints (Figure 12.14).

It has a deep layer, extending from the scaphoid tubercle and the trapezium tubercle to, on the ulnar side, the triquetrum, and the capsula of the joint between the pisiform and the triquetrum, and the ligament between the pisiform and the unciform process. It is almost 1 cm thick. This annular ligament transforms the hollow of the carpus in the carpal tunnel. It presents a sagittal septum which inserts on the trapezium and the trapezoid and helps to form the sliding tunnel for the flexor carpi radialis. In addition, it has a superficial layer which arises from the surrounding aponeurotic elements, and which inserts medially on the tendon of the flexor carpi ulnaris, forming the anterior wall of the Guyon's canal.

Architectural and dynamic study of the wrist

The anatomic description of the wrist already gives an idea of the complexity of the biomechanical study of this region. The carpus is not made in one piece as, had once been believed, before the X-ray study. Also, its malleability and adaptability are not best described by compring it to a bag of balls. Each little bone has its own significance, and can change its orientation according to the different wrist positions required. However, there are many constraints, particularly architectural modalities, which will be studied first.

Architecture of the wrist

The predominant feature of the carpus is its concavity, which increases from the first to the second row. This concavity is evidently due to the anatomic arrangement of the bones. It also corresponds to the tuberosities of two radial bones (scaphoid and trapezium) and to the existence of the pisiform and the unciform process of the hamate on the ulnar side. This hollow is maintained by ligaments, and particularly by the palmar retinaculum (an annular ligament of the carpus) which, like a bridge, closes the carpal tunnel anteriorly (Figure 12.15).

Regarding the extremities of the two bones of the forearm, the concavity of the carpus projects the ulnar head backwards so that it is palpable and visible at the posterior side of the wrist. The carpal canal and the hollow of the carpus protect the elements passing through, and facilitate the convergence of tendons passing from the forearm to the fingers, as well as their reorientation beneath the anterior annular ligament, which corresponds to a pulley (for radial and medial inclinations, as for flexion–extension movements). Finally, the transverse arch of the carpus is the starting point for the layout of the digits, which also present a transverse arch that is necessary for a good grip.

The bones of the wrist enter a system of intercalary bones, on which there are no muscular insertions (except on the pisiform for the flexor carpi ulnaris, but this is a mediate insertion, and on the anterior annular ligament for the intrinsic muscles of the two eminences). The carpus is thus subject to many forces generated by the different muscles, and the entire bony system must be stabilized in order to obtain precise movements at the level of the digits.

At the level of the carpus there are some well-defined functional units (Figure 12.16):

1. A couple between the lunate and the scaphoid which is articulated with the head of the capitate and which turns around it. Moreover, this couple can present a possible torsion by minimal movements between the two bones (with a limitation due to the radiocapholunate ligament). This functional couple works in conjunction with the trapezium and with the thumb column.
2. There exists a central pivot represented by the capitate associated with the trapezoid and the hamate. The capitate resembles a tow bar or tenon. This pivot allows flexion and extension of the preceeding couple, which, as previously mentioned, can also turn on the head of the capitate.
3. A couple between the triquetrum and the hamate which has some mobility around the central pivot and can also be deformed by sliding of the triquetrum upon the hamatum.

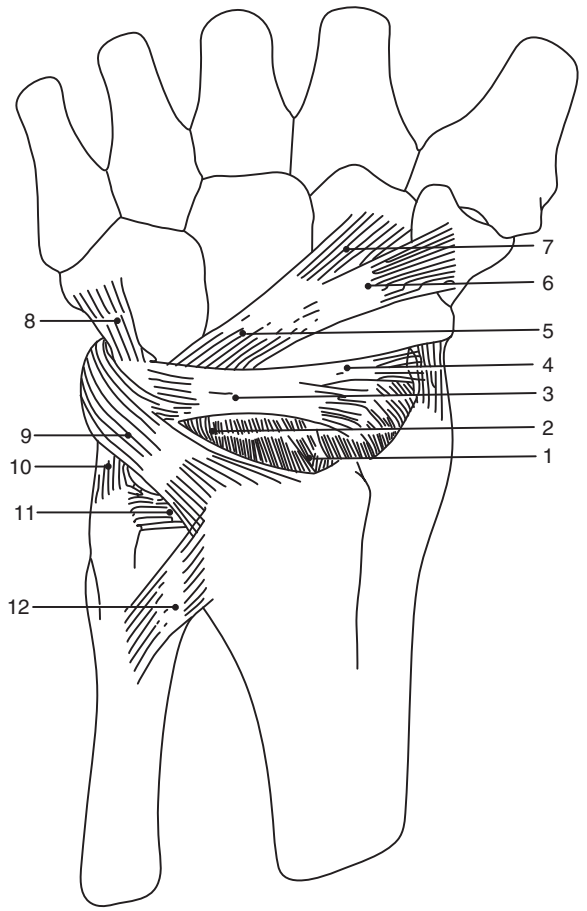

Figure 12.10

The dorsal ligaments: the posterior radioscapholunate ligament (1), the posterior radiotriquetral ligament (2), the horizontal band (3) of the triquetral sling, which gives off an expansion (4) toward the radial collateral ligament, the oblique band (5), which inserts into the trapezium (6) and the trapezoid (7), the posterior triquetrohamate ligament (8), the posterior band (9), the ulnar collateral ligament (10), the triangular ligament (11), and the posterior radioulnar ligament (12). Adapted from Tubiana et al.[1]

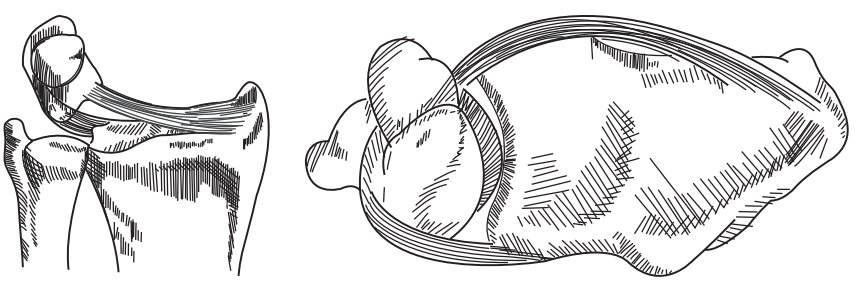

Figure 12.11

The Kuhlmann sling formed by the volar and dorsal ligaments arising from the radial styloid. Adapted from Tubiana et al.[1]

This ulnar section of the carpus presents a vertical axis which extends the medial part of the forearm to the ulnar fingers. It can also support the pisiform, which represents a sort of trestle for the flexor carpi ulnaris inserted at its superior pole. This arrangement has the advantage of preserving the lever arm of the tendon of this muscle during wrist movements.

Mobility of the wrist

Pronation and supination are the rotational movements of the wrist around the fixed element, which is the inferior extremity of the ulna. In addition, there are movements of flexion and extension, as well as radial and ulnar movements. The combination of all these movements determines the circumduction, which can be defined as movements inside a cone-shaped volume.

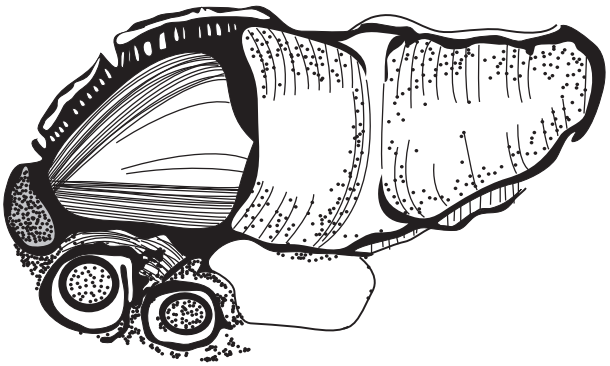

Figure 12.12

The triangular ligament. Adapted from Zancolli.[3]

Measurement of the movement amplitude of the wrist is clinically straightforward: the starting reference is the anatomic plane and normal flexion reachs 85°. According to Fisk this movement occurs initially in the mediocarpal joint (first half) and thereafter in the radiocarpal joint.[7] These positions are modified in carpal instablities due to ligament lesions. Extension (also sometimes called dorsal flexion) also has a maximum of 85°. This time, still for Fisk, the initial two-thirds occurs in the radiocarpal joint, and thereafter in the mediocarpal joint. It can be observed by lateral X-rays that the lunate presents a forward displacement during extension, and, in contrast, presents a normal situation in flexion (Figure 12.21).

Radial inclination in the wrist is about 15°, while ulnar inclination is a more significant 45°.

However, these numbers are not reliable because they depend on the position of flexion or extension, and also on the position of prono-supination. As mentioned above, the hand can be moved in a conical volume. The summit is at the center of the wrist and the angle of revolution of this cone is around 80°. Relationships exist between the different movements: flexion is concomitant with ulnar inclination, and extension with radial inclination.

Muscles of the wrist and biomechanics

Almost all the muscles of the forearm are found at the level of the wrist. They can be divided into two groups:

1. The muscles are inserted on the distinctive ligamentous structures of the carpus or on the bases of the metacarpal bones (they act on the entire carpus because of the relatively stiff joints between the carpal and the metacarpal bones). Flexor carpi ulnaris is the only muscle to insert

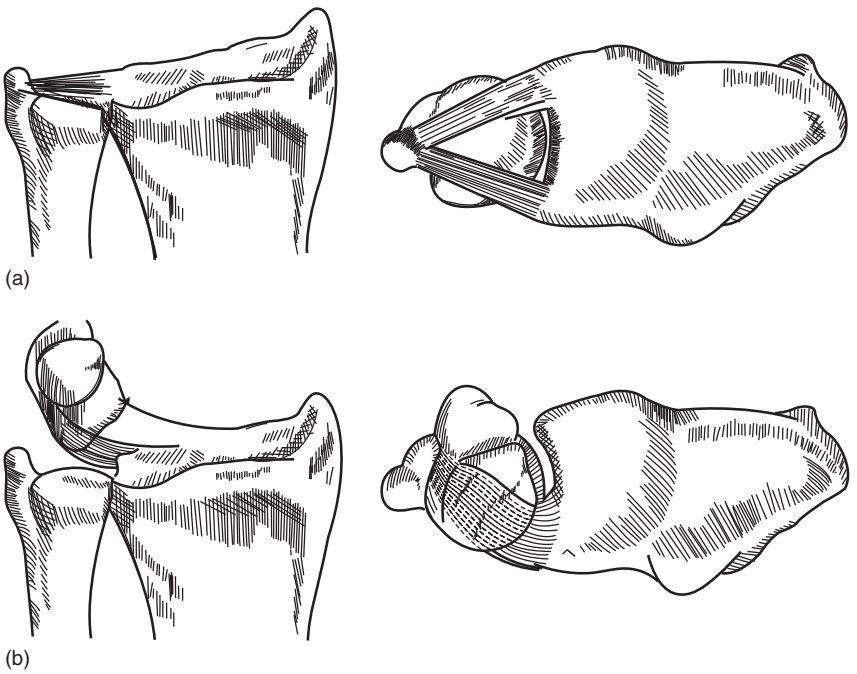

Figure 12.13

The ulnar fibrous structures.
(a) Triangular fibrocartilage ligament.
(b) Taleisnik's meniscus. Adapted from Tubiana et al.[1]

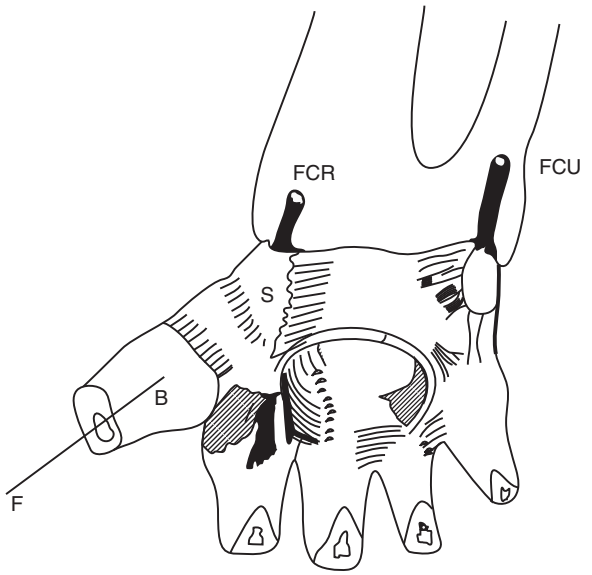

Figure 12.14

The palmar retinaculum and the carpal tunnel, with the special route of the flexor carpi radialis (FCR) and the GUYON's canal. Flexor carpi ulnaris (FCU); long flexor of the thumb (F); basis of the first metacarpal (B); scaphoid bone (S).

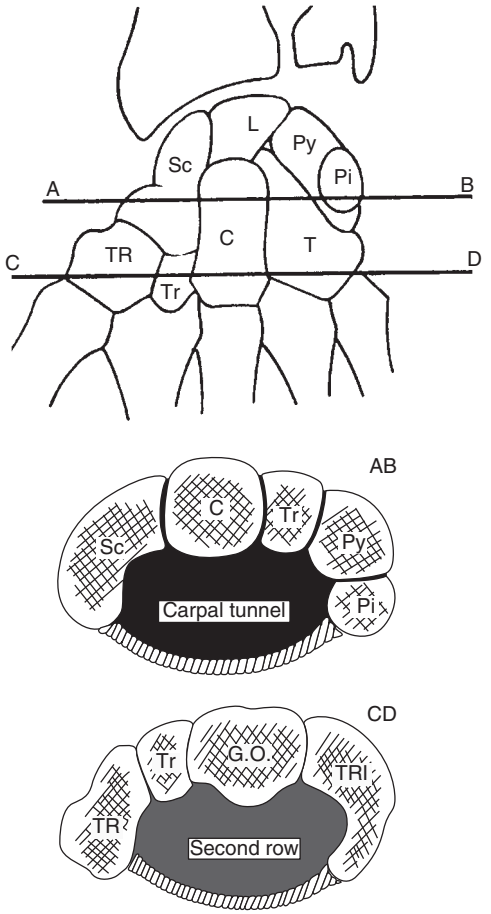

Figure 12.15

The concavity of the wrist at two levels: AB, cross-section of the first row, and CD, cross-section of the second row. Scaphoid bone (SC); lunate (L); pyramidal (Py); trapezoid (TR); capitate (C); urciform (T).

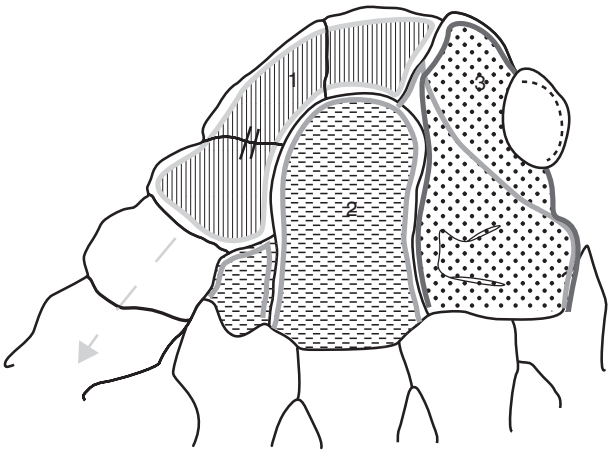

Figure 12.16

The three functional pivots: (1) scaphoid + trapezium; (2) capitate + trapezoid and hamate; (3) triquetrum and hamate.

on a bone, the pisiform, but, as has been pointed out, this is an independent bone in the first row. It is significant that all these muscles arise proximally on the inferior extremity of the humerus (epicondyle and epitrochlea), and they are therefore transarticular muscles with the usual biomechanic consequences (it is necessary to stabilize the joints which are crossed by muscles to avoid unwanted effects at this level; it is a well-known necessity in the kinetic chain).

2. This group includes the muscles which are 'in transit', crossing the two sides of the carpus. Some are firmly attached to the bony layer by fibrous sheets, which represent pulleys for the tendons going to the digits. There, again, they have a role in the mobility of the wrist.

Group 1

(a) Muscles which arise from the medial process (epicondylus medialis) of the humerus are *flexors* of the wrist (Figure 12.17). The tendon of the flexor carpi radialis passes through a specific canal at the lateral part of the carpal canal and inserts on the base of the second metacarpal and secondly on the trapezium and on the base of the third metacarpal. It is a flexor of the wrist, but acts mainly on the lateral side of the mediocarpal joint. Only when the extensor carpi radialis is active does it have a pronator action.

The palmaris longus is an inconstant muscle which is thinner. It tightens the annular ligament and the skin of the palm. It is a flexor of the wrist and also maintains the convexity of the carpal ligament in the forward direction – and this, according to Kaplan,[8] could reinforce the insertions of the intrinsic muscles on it.

These two muscles are innervated by the median nerve, and respond specifically to the C7 and C8 roots of the brachial plexus.

The flexor carpi ulnaris is an important muscle which ends on the pisiform and sends little expansions to the neighboring bones. The role of the pisiform is not fully appreciated. It spreads out the terminal insertion of the

flexor carpi ulnaris and so increases the moment of the flexor force of this muscle. Owing to the mobility of this little bone on the triquetrum, the tendon maintains a favorable leading angle whatever the movement of the wrist (flexion, extension, or inclination). The tendon of the flexor carpi ulnaris extends the ulnar axis of the forearm into the ulnar fingers. This muscle is a strong flexor of the wrist, but also generates a medial inclination (except in one important flexion – illustrated by Duchenne de Boulogne).[9] It is innervated by the ulnar nerve (C8 and D1).

(b) Muscles arising from the lateral epicondylus are *extensors* of the wrist (Figure 12.18). The extensor carpi radialis longus, which arises from the inferior part of the humerus, ends on the dorsal base of the second metacarpal. The extensor carpi radialis brevis is more precisely a muscle arising from the radial epicondyle and inserts on the base of the third metacarpal. Both are extensors of the carpus, but only the longus gives a lateral inclination (Figure 12.18, arrow). Some anomalies are possible for these two tendons, and this can be useful to know when tendon tranfer is chosen. They are innervated by the radial nerve and correspond to roots C5 to C8 of the brachial plexus.

Group 2

For the muscles in this group, the wrist is only a passage zone for their tendons before they reach the digits. These tendons are:

- *dorsal* – all the extensors communis and proprius, and laterally the extensor and abductor tendons for the

Figure 12.17

The flexor muscles of the wrist. From radial to ulnar: flexor carpi radialis, palmaris longus, and flexor carpi ulnaris. Adapted from Tubiana et al.[1]

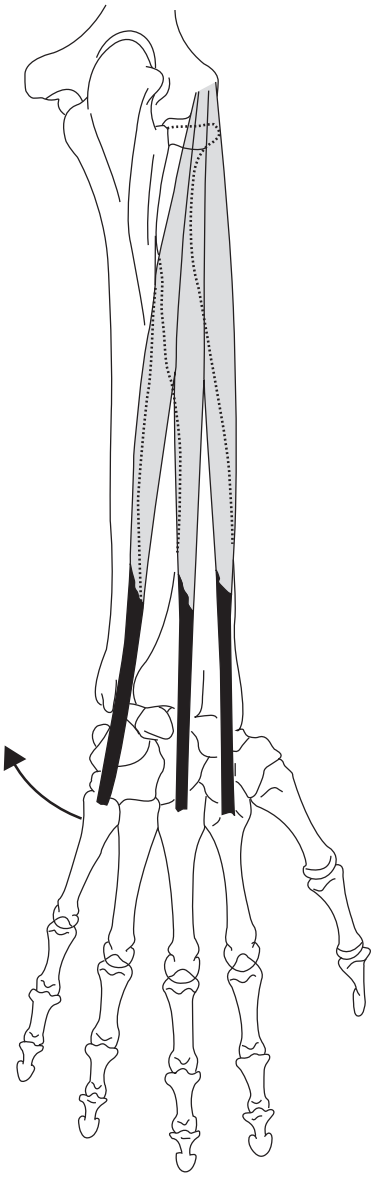

Figure 12.18

The extensor muscles of the wrist. From radial to ulnar: extensor carpi radialis longus, extensor carpi radialis brevis, and extensor carpi ulnaris. All these muscles are inserted distal to the carpus. Adapted from Tubiana et al.[1]

thumb (Figure 12.19a and b); they are firmly applied on the carpus by the annular dorsal ligament and also by particular fibrous sheets on the radial inferior extremity

- *palmar* – flexors: superficialis and profundus of the digits, and long flexor of the thumb; they are gathered in the carpal canal, under which they diverge towards the digital rays (Figure 12.20a and b).

The existence of the two joints, the radiocarpal and mediocarpal, can justify a teleological point of view: they avoid a sharp angular aspect of the wrist during motion (flexion and extension). Mechanically it is possible to consider the wrist as two different pulleys, which have the same transverse axis but two different diameters (Figure 12.21). More precisely, the pulleys are not the mobile element, but the tendons are sliding in their hollows (with the help of the synovial sheaths). Moreover, the situation is different in flexion and extension:

- *During flexion*, the pulley which has the greater radius is formed by the carpus, and the extensor tendons are the gliding elements; the pulley having the shorter radius comprises the annular carpal ligament, on which the palmar tendons are the gliding elements. Because of the difference between the two radii of the pulleys, the displacement of the extensors is much more important than for the flexors.
- During *extension* the two surfaces of the carpus are the pulleys, and the anterior palmar ligament loses this role. The difference in the dimensions of the pulleys is minimal and the course of the extensors and flexors tends to be similar.

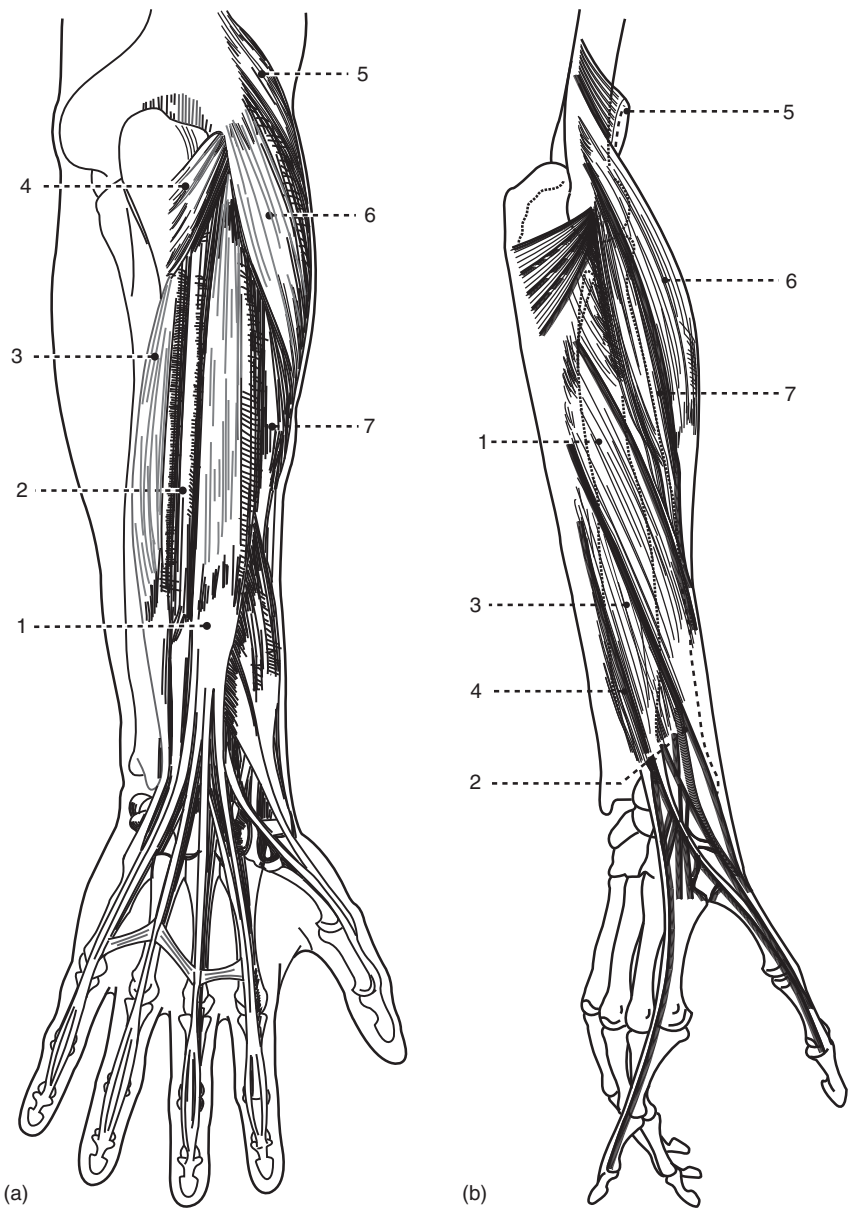

Figure 12.19

(a) The superficial muscles of the dorsal forearm. *Posterior*: (1) extensor digitorum communis; (2) extensor digiti minimi proprius; (3) extensor carpi ulnaris; (4) anconeus. *Lateral*: (5) brachioradialis; (6) extensor carpi radialis longus; (7) extensor carpi radialis brevis. (b) The deep and lateral muscles of the dorsal forearm. *Posterior*: (1) abductor pollicis longus; (2) extensor pollicis brevis; (3) extensor pollicis longus; (4) extensor indicis proprius. *Lateral*: (5) brachioradialis; (6) extensor carpi radialis longus; (7) extensor carpi radialis brevis. Adapted from Tubiana et al.[1]

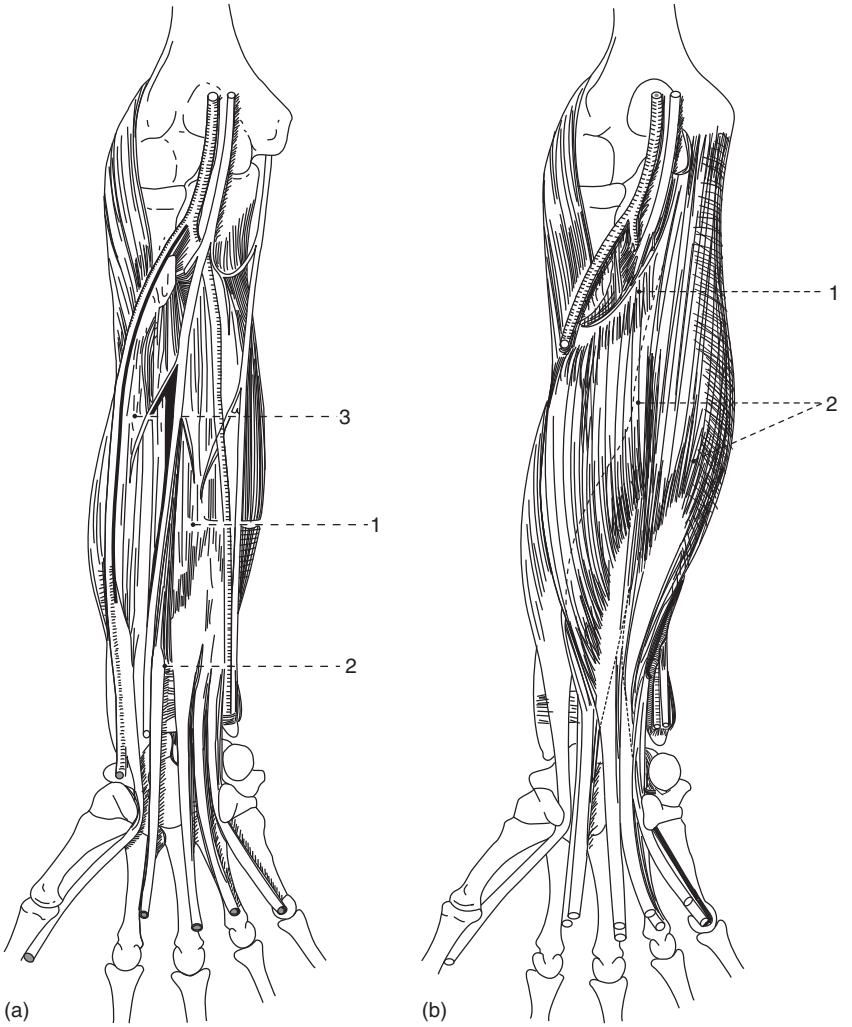

Figure 12.20

(a) The deep muscles of the anterior forearm. (1) Flexor digitorum profundus; (2) the tendon of the index finger is more individualized than that of the other fingers; (3) flexor pollicis longus. (b) The flexor superficialis. (1) The fibrous arch of the flexor digitorum superficialis; (2) the deep digastric fascia from which the tendon of the index and ring finger arise. Adapted from Tubiana et al.[1]

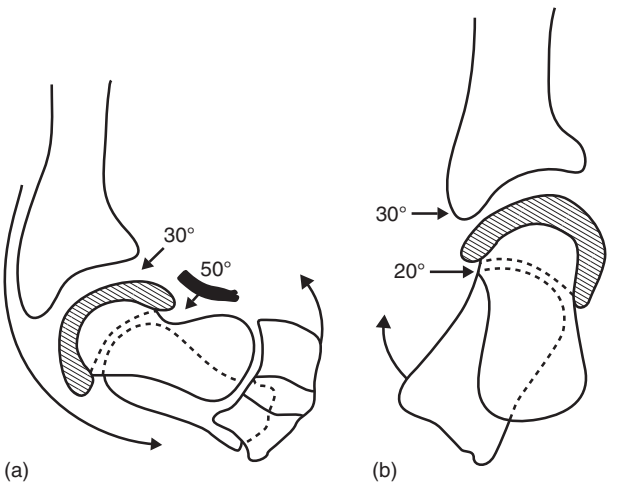

Figure 12.21

The two pulleys of the wrist. (a) During flexion the great pulley comprises the dorsal sides of the radiocarpal and mediocarpal joints. The anterior retinaculum is the little pulley for the flexor tendons. The extensors being stretched, flexion of the digits is somewhat difficult. (b) When the wrist is straight or extended the effect of the two pulleys is almost identical, so this is the functional position.

If one accepts this interpretation of the pulleys some physical derivatives should be considered. Normally, a pulley transmits to the two parts (upstream and downstream) of a tendon (which passes under this pulley) the same forced generated by the muscle. For example, if one considers an extensor tendon, it generates a force F which is proximal to the wrist. Below the wrist this force F is identical, but can be opposed totally or partially by forces which impede extension of the wrist: for example the weight of an object (W), or the weight of the limb at the level of contact (G) (Figure 12.22). Thus, at the level of the wrist, we will have exerted on the carpus and always in the direction of flexion, a resulting force equal to F+W+G. If one wants to keep the wrist in the same position this force exerted on the carpus must be counterbalanced by extension forces of the epicondylar muscles, and also by forces of the flexors of the elbow to balance the effect of gravity. It is then possible to imagine the complexity of the forces exerted by the muscles of the upper limb which are *all* affected, more or less, by a movement or a position of the hand. Moreover, unwanted effects can arise when the movement is thwarted. In such a condition, digit flexors can get an extensor effect simply by the prevalence of the force exerted on the annular ligament (force applied on the axis of the pulley).

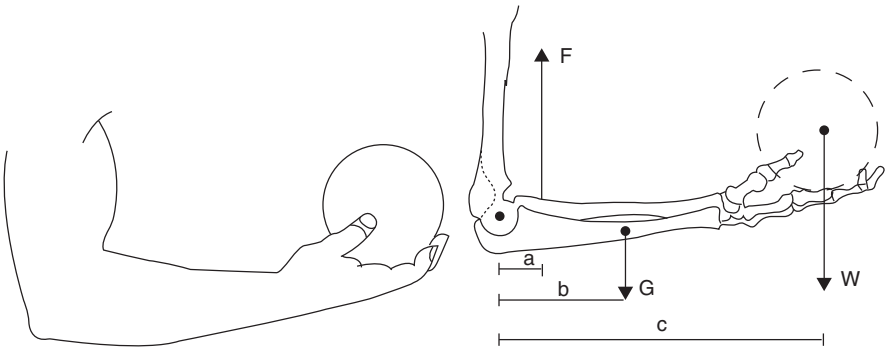

Figure 12.22

System of forces acting on the forearm, measured from the transverse axis of the elbow: F = force exerted by flexors of the elbow; G = force exerted by gravity; W = force exerted by the weight of the object handled; a, b, and c are the distances to the axis of the elbow. Adapted from Delamarche et al.[10]

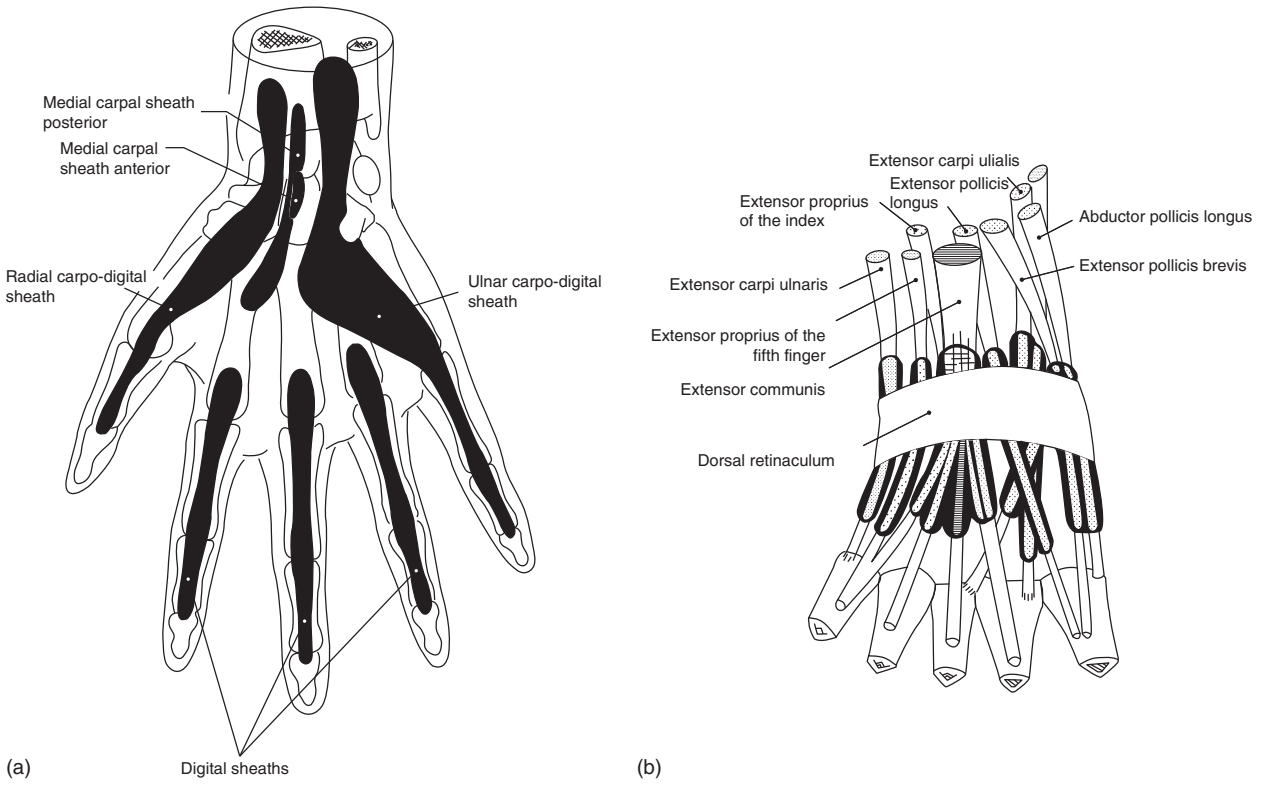

Figure 12.23

The synovial sheaths of the tendons: (a) around the flexors tendons; (b) around the dorsal tendons.

We have not considered the frictional forces, that exist betwen the tendons and the bones or ligaments which act as a pulley. For these, the serous bursae serve to decrease the friction, and almost all the tendons on the two faces of the wrist are surrounded by synovial sheaths (Figure 12.23a and b). The frictional forces of the bone at the level of the joints during movement can be predominant when the cartilage is destroyed by a pathologic phenomenon (e.g., rheumatism).

In conclusion, movements of the wrist, either of flexion or of extension, are all linked. It is thus difficult for synergistic muscles to oppose those which are not predominant (the activity of which does not generate the movement): some are necessary to stabilize a joint in the articular chain, some to slow down or enhance the fluidity of movement of a skeletal piece, and so on. All these activities being simultaneous, they are ipso facto synergistic, with both different and complementary effects. It is then reductive to say, as classically, that the flexors of the digits are synergistic with the wrist extensors.

Nevertheless, there are functional positions of the wrist. They are multiple because they depend on the desired gesture and the projected handling of the object. Some positions in extension of the wrist are necessary, for example, for writing. The neutral flexion position and ulnar inclination is usual in prono-supination movements or in the thumb–index pinch. It is well known, moreover, that the extreme positions of flexion or extension of the wrist disturb the muscle activity by the fact that the pulleys unbend or stretch flexors or extensors. It is difficult to

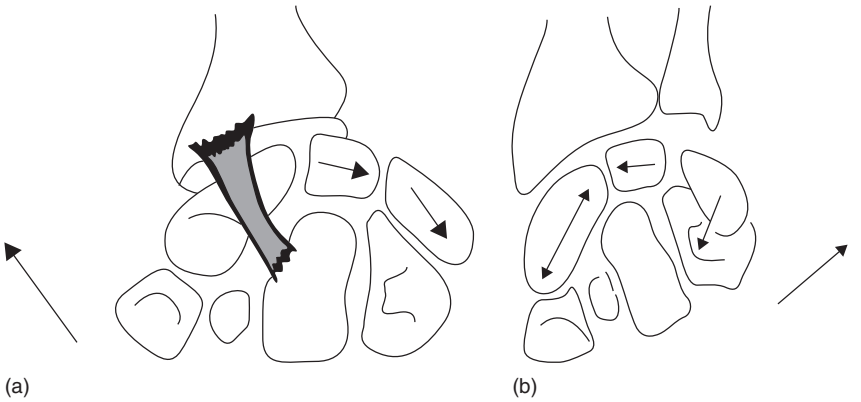

Figure 12.24

Some movements of the carpal bones studied with radio-cinema. (a) During radial deviation with some flexion of the wrist: the scaphoid seems to be shorter and the lunate is inclined on the ulnar side. The hamate is sliding on the triquetrum. The radio-scapho-capitate ligament maintains the scaphoid in its middle part and this allows the rocking motion. (b) During ulnar deviation the hamate slides on the triquetrum, the lunate is further under the radial extremity, and the scaphoid has its maximal height.

know the exact situation of each carpal bone in these complex movements.

It should be remembered that X-ray images are produced by rays tangential to the surfaces. Images are not easy to interpret and some errors can be made. For example, in a movement one bone can seem to have a different aspect and a different position in relation to the neighboring one, which looks stable only because it has a more regular form. Then to speak of mobility of the first is incorrect.

Nevertheless, with the use of a radio-cinema (X-ray movie) some motions of the carpal bones are true. For example, in a position of ulnar deviation the triquetrum slides on the superior surface of the hamate, this may be explained by the displacement helping to diminish the stretch of the ulnar ligamentous system. Also, in flexion and radial inclination the scaphoids tend to be more horizontal and the semilunar tends to subluxate anteriorly (Figure 12.24a and b). It does not seem essential to interpret these situations in normal physiology. However, in contrast, in traumatic lesions a description of the orientation of the bones is useful and can explain the mechanism of the trauma. Such descriptions are also essential for the diagnosis of the so-called 'wrist instabilities', which can have a number of different causes. In 1972, Linscheid et al[11] published a classification of wrist instability which is still commonly used (e.g., rocking chair of the semilunar, ulnar, palmar, or dorsal translation of the carpus). Taleisnik[12] added the distinction between static (permanent) and dynamic instabilities (only visible on radiocinematographies). The mediocarpal joint seems to be important in this respect. The role of the lunate is essential for many authors. It helps to distinguish between the DISI (dorsal intercalated segment instability) and the VISI (volar intercalated segment instability).

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13 Restoration of extension of the wrist and digits

Raoul Tubiana

Loss of active extension in the wrist and digits constitutes a considerable functional and cosmetic handicap. This severe disability can be caused by neurologic, muscular, or tendinous lesions (Figure 13.1).

Etiology

Traumas are the most common cause of loss of active extension of the wrist and digits due to a lesion in the nerve pathway from brain to arm, or to muscular or tendinous injuries. However, these lesions can also be the result of various non-traumatic peripheral neuropathies or non-traumatic muscular or tendinous lesions of metabolic, vascular, inflammatory, congenital, or degenerative origin. We shall only consider those lesions that can benefit from a reconstructive surgical treatment.

In this chapter, restoration of extension of the wrist and/or digits will be considered in very different conditions, such as radial nerve injuries, injuries of the extensor tendons of the digits, congenital pollex flexus adductus, and extensor tendon ruptures in rheumatoid arthritis.

Neurologic lesions

Traumatic paralyses can be caused by nerve division, avulsion, loss of nerve substance, or compression.

Paralyses of the extensors of the wrist and digits are usually classified according to the level of the neurologic lesion (see Chapter 2).

Cerebral lesions

Head injuries and stroke patients are dealt with in Chapter 30.

Cervical spinal cord lesions

The paralysis of extension of the wrist in high-level tetraplegia is a sign of poor prognosis. On the other hand, the existence of active extension in the wrist permits the use of tenodesis to re-establish a functional grip (see Chapter 29).

Brachial plexus lesions

Loss of active extension in the wrist and digits can result from brachial plexus lesions caused by trauma or by compression due to tumors or anatomic anomalies.

All cervical roots can be involved, as the posterior cord, from which the radial nerve originates, receives motor fibers

Figure 13.1
Loss of active extension in the wrist and digits.

from all the roots. In some cases, only the sixth and seventh cervical roots are involved, or even only C7, in which case the brachioradialis is active.

Lesions of the cervical spinal cord and of the brachial plexus are discussed in Chapter 20. We just want to emphasize here the importance of restoration of wrist extension, which is a fundamental step in the treatment of extensive paralyses of the hand.

Radial nerve lesions

The radial nerve arises from the posterior cord and is the nerve of extension for the upper limb. It furnishes all of the extensors of the elbow, the wrist, and the digits; however, the distal two phalanges of the fingers can be extended by the intrinsic muscles in the hand (Figure 13.2a).

Lying behind the axillary artery initially, the radial nerve runs distally in the arm by winding around the posterior aspect of the humerus from medial to lateral. The close anatomic relationship of the radial nerve with the humeral shaft accounts for the high incidence of radial nerve injuries in fractures of the humerus.

The spiral humeral groove is in fact an osteo-muscular groove, enclosed prosteriorly by the long head of the triceps, limited proximally by the insertions of the lateral head and distally by the insertions of the medial head of the triceps (Figure 13.2b).

Several anatomic studies of the humeral osteo-muscular groove have been performed. Wilhelm[1] observed that at the distal part of the groove, the nerve is crossed by the deep tendinous portion of the lateral head of the triceps which can be a source of compression.[2] Lotem[3] described a fibrous arch composed of fibers from the lateral head, which differs from the hiatus of the lateral intermuscular

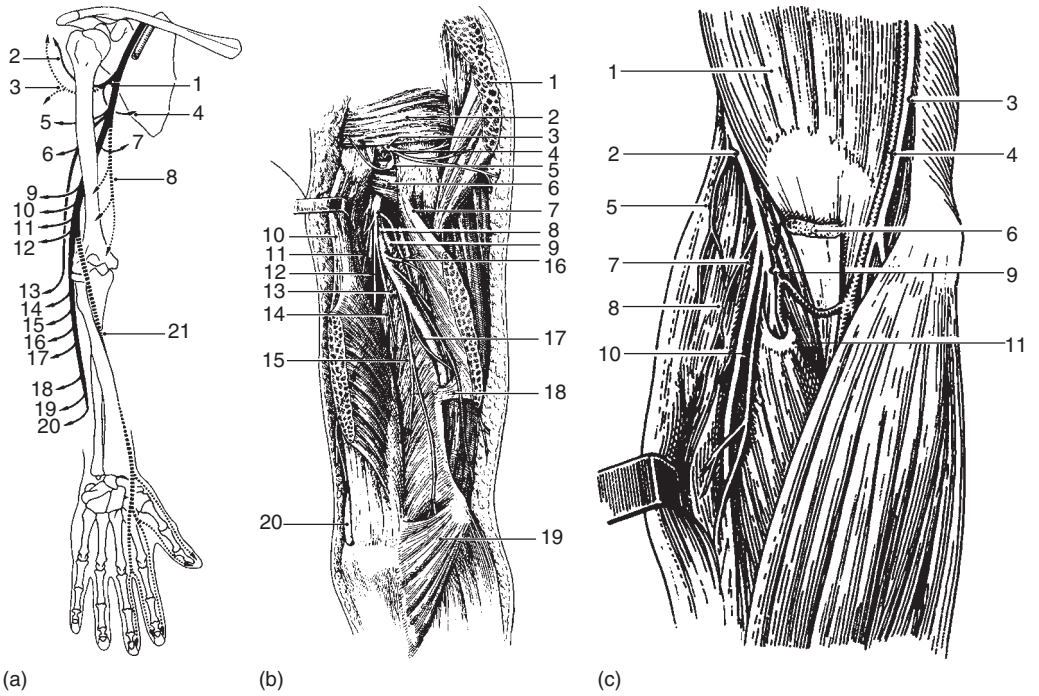


Figure 13.2

(a) The radial and axillary nerves; muscles supplied and cutaneous distribution (the forearm is pronated): (1) axillary nerve; (2) deltoid; (3) cutaneous branch to shoulder; (4) teres minor; (5) triceps (long); (6) triceps (lateral); (7) triceps (medial); (8) medial cutaneous branch; (9) brachioradialis; (10) extensor carpi radialis longus; (11) extensor carpi radialis brevis; (12) supinator; (13) anconeus; (14) extensor digitorum communis; (15) extensor digitorum to fifth digit; (16) extensor carpi ulnaris; (17) abductor pollicis longus; (18) extensor pollicis brevis; (19) extensor pollicis longus; (20) extensor indicis proprius; (21) anterior sensory branch. The sensory branches are shown as dotted lines. (b) Dissection of the posterior arm to show the axillary and radial nerves (right arm): (1) deltoid muscle (posterior part reflected anteriorly); (2) teres minor; (3) axillary nerve emerging with posterior humeral circumflex artery; (4) through the quadrilateral space; (5) branch to teres minor, showing a ganglion; (6) teres major; (7) lateral head of triceps; (8) radial nerve with the profunda brachil artery (9); (10) long head of triceps; (11) nerve to long head of triceps; (12) medial cutaneous nerve of arm; (13) short head of triceps; (14) superior nerve to short head of triceps; (15) inferior nerve to short head of triceps and anconeus; (16) superior nerve to lateral head of triceps; (17) inferior nerve to lateral head of triceps; (18) lateral intermuscular septum; (19) anconeus; (20) ulnar nerve. (c) The radial nerve at the level of the elbow: (1) brachialis; (2) radial nerve; (3) median nerve; (4) brachial artery; (5) branch to brachioradialis; (6) cut end of biceps tendon; (7) superficial branch of radial nerve; (8) branch to extensor carpi radialis longus; (9) posterior motor branch of radial nerve; (10) branch to extensor carpi radialis longus; (11) arcade of Frohse.

septum more distally. Lubahn and Lister,[4] Allieu and Lussiez,[5] and other authors found similar arches, which may account for some high radial nerve entrapment.

In the arm, the major branches extend successively: the triceps, the brachioradialis, and the extensor carpi radialis longus (ECRL).

The radial nerve divides at the level of the bicipital groove into its two terminal branches: the deep posterior motor branch and the superficial anterior sensory branch (Figure 13.2c).

High radial palsy refers to a lesion of the nerve above its division. Most often, it is in the context of a traumatic lesion, especially with a fracture of the humerus (see the discussion of radial nerve injuries and fractures of the humerus later in this chapter) or sometimes an external compression syndrome (lover's or Saturday night palsy). Paralyses of the brachioradialis and of the wrist extensors with wrist droop indicate a high radial palsy. The triceps is generally not involved, as its innervation is mainly from branches with origin in the axilla.

The nerve for the extensor carpi radialis brevis (ECRB), arises in more than half of the cases (58%) from the superficial terminal branch,[6] so that when there is a lesion in the terminal motor branch, extension of the wrist can be maintained in some cases.[7]

The posterior motor branch, the posterior interosseous nerve, penetrates the supinator muscle by passing under the arcade of Frohse. This arcade is fibrous in about one-third of cases and may compress the nerve. The nerve winds around the neck of the radius between the two heads of the supinator. In 25% of cases, it lies against the periosteum for about 3 cm (bare area) when the forearm is supinated; it is more vulnerable at this level.[8] The nerve then emerges from the supinator in the posterior compartment and runs along the posterior aspect of the interosseous membrane. It sends motor branches for all the muscles of the posterior aspect of the forearm and sensory branches to the wrist and carpometacarpal joints.

A *low radial palsy* differs from a high radial palsy in that it spares the radial wrist extensors and the supinator. An associated paralysis of the extensor carpi ulnaris (ECU) results in radial deviation of the wrist during extension.

Paralysis of the extensor digitorum results in a deficit of metacarpophalangeal joint extension, although extension of the interphalangeal joints, including that of the thumb, remains possible by action of the median and ulnar innervated lumbricals and interossei.

In order to detect deficit of extension of the proximal phalange, one must maintain the wrist in extension to eliminate the tenodesis effect of wrist flexion.[9]

Deficits of motor function are variable, and can at first be confused with tendon ruptures at the wrist. The most common cause of these ruptures is rheumatoid arthritis. The clinical history is different – rupture of the extensors of all the fingers does not usually occur at one time; therefore, the patient who has an increasing inability to extend the fingers and thumb, which is associated with radial deviation and weak extension of the wrist, is probably suffering from paralysis.[10] The diagnosis is confirmed by additional tests:

- When the wrist is passively flexed, if the fingers extend slightly by a tenodesis effect, the continuity of the tendons is retained.
- Direct electrical stimulation to the muscles produces extension of the fingers.

Low radial palsy can have a traumatic origin: laceration of the posterior interosseous nerve or compression by fracture–dislocation of the elbow. It can also in a number of cases have a non-traumatic origin: constriction of the nerve at the proximal edge of the supinator from the arcade of Frohse. The first case of this constriction was described in an orchestra conductor by Guillain and Courtellemont[11] in 1905. Comtet and Chambaud[9] discovered a double constriction of the nerve before it entered the supinator. Since then, several constrictions have been found, with or without an arcade of Frohse.[12,13] Any anatomic anomaly in the radial tunnel can cause single or double constriction.[14,15]

Extrinsic compression can be caused by a tumor. Synovial cysts causing compression of the radial nerve are relatively rare.[16,17]

Leprous and poliomyelitis palsies and some rare neuromuscular diseases can disturb extension of the wrist and digits.

Treatment of neurologic lesions

As already mentioned, cerebral, cord and brachial plexus lesions are dealt with elsewhere in this book. Here, we shall cover restoration of extension in the wrist and digits after radial nerve paralyses.

The first treatment after a radial nerve division is surgical repair by direct suture or nerve graft.

The earlier the repair, the better the results. The more distal the nerve repair, the better the chances of a rapid restoration. It has been clearly demonstrated that palliative surgery for radial nerve palsy is now less frequently performed, because of the better results after microsurgical nerve repair techniques.[18]

Fracture of the humerus is the most frequent cause of radial nerve lesions, and the treatment of radial nerve injuries associated with fractures of the humerus is still debated.

Radial nerve injuries and fractures of the humerus

As it crosses the spiral groove on the posterior aspect of the humerus (see Figure 13.2b), the nerve is not in contact with the bone. They are kept apart by thin sheets of muscle. They come into direct contact only at the lateral supracondylar

border of the bone. At this level, the nerve crosses the inextensible posterior intermuscular septum to enter the lateral bicipital groove; it is somewhat stretched at this point, and lack of mobility accounts for its vulnerability in humeral fractures.[19] Bezes et al[20] reported 26 radial palsies for 246 fractures of the humerus.

Injuries of the radial nerve can be seen immediately after fracture in about 10% of fractures of the distal half of the humerus. Seddon[21] demonstrated in 1943 that division of the nerve before treatment was rare; most of the nerve lesions were contusion (neurapraxis) or compression (axonotmesis) with the possibility of regeneration. Consequently, emergency treatment should be directed at proper closed management of the fracture, with evaluation of the status of the nerve injury before any operative intervention is undertaken.

This conservative approach is still followed by many surgeons.

However, it seems that the severity of radial nerve lesions has increased with the frequency of motor accidents. The advantage of early surgery in these cases is obvious. In other respects, osteosynthesis procedures, aimed at early mobilization of the fractured limb, are more and more frequently performed, and, because of the surgical exposure, iatrogenic nerve lesions are more frequent.

Over a period of 10 years, Alnot et al[22] treated 62 patients with humeral fractures associated with radial nerve palsy, most of whom were referred after prior care. Orthopedic treatment was used alone in 14 cases. There were 6 plate fixations and 42 intramedullary nailings. A continuous nerve was found in 38 cases, but 24 of the 62 cases of radial nerve palsy resulted from nerve tears. Recovery was achieved in 37 of the 38 lesions with continuous nerve. Nerve divisions were treated by nerve suture or nerve graft. The outcome of primary nerve suture appears to be better than that of secondary grafts with proximal resection to healthy nerve tissue.

Some elements are helpful for the choice of an early surgical approach of the lesions:

1. The time of appearance of the paralysis:
   - When the paralysis exists before any treatment of the fracture, the nerve lesion can be of any type: neurotmesis, axonotmesis, or neurapraxis.
   - In the case of iatrogenic palsy, when the radial nerve paralysis appears after the treatment of a humerus fracture, the nerve lesion is usually only a compression after a close reduction, but can be a division after a surgical exposure.
2. The severity of the trauma and type of the humerus fracture:
   - Spiral fractures of the distal third of the humerus with important lateral angulation and overriding of a sharp distal fragment, or with intermediary fragments, are particularly dangerous for the nerve.

Progress in imaging techniques, especially magnetic resonance imaging (MRI), can now visualize a nerve division or even the position of the nerve between the bone fragments, but cannot evaluate an intraneural lesion.

In conclusion, it seems that fractures of the humerus associated with radial nerve paralysis with important lateral angulation or subsequent to high-energy trauma have a high risk of nerve division and should benefit from systematic nerve exploration. The early surgical approach will allow open treatment of the fracture and nerve repair before the growth of scar tissue. Minimally displaced fractures, on the contrary, are often associated with simple contusion of the nerve with capability for spontaneous recovery. Indications in intermediate cases are difficult. If an osteosynthesis procedure is performed in order to benefit from early mobilization, a systematic nerve exploration is recommended by Alnot and several authors.

In the case of a conservative approach to the lesions, electromyographic (EMG) studies are necessary to show early signs of regeneration in the brachioradialis, several weeks after the accident and before any clinical sign of muscle recovery. If after about 3 months there is no electrical or clinical sign of regeneration, a surgical exploration of the nerve is necessary.

Palliative treatment after radial nerve lesions

When radial nerve repair is unsuccessful or has not been done, restoration of wrist and digits extension is possible with tendon transfers.

Radial nerve palsy results in paralyses of the triceps, the supinator, and the brachioradialis muscles, and also causes paralyses of the extensors of the wrist, of the thumb, and of the fingers.[23]

Paralysis of the triceps is uncommon, because the site of the trauma on the upper limb is usually distal to the branches that innervate this muscle.

In paralysis of the supinator muscle, the loss of supination is compensated by the biceps and by the movements of the shoulder. Likewise, paralysis of the brachioradialis, which is essentially an elbow flexor, is compensated by the biceps and brachialis.

By contrast, three movements that are essential to hand function are lost and cannot be compensated for: extension of the wrist (all three wrist extensors are supplied by the radial nerve), extension and retroposition of the thumb (brought about by the extensor pollicis longus, abductor pollicis longus and extensor pollicis brevis), and extension of the metacarpophalangeal joints of the fingers.

Historical perspective

The evolution of palliative techniques for radial palsy has been continuous since the first attempts to use tendon transfers to obtain finger and thumb extension at the end of the 19th century.[24,25]

Boyes[26] compiled a list of 58 transfers described up until 1960. This list has lengthened since then.

For a long time, patients with paralyzed wrist extensors were treated by arthrodesis[27] or by tenodesis of the extensors carpi radialis (ECR) to the radius.[28]

Jones[29] is credited with re-establishing extension of the wrist by the transfer of the pronator teres (PT) to the ECR. This classic transfer has proved its value over the years and continues to be used.

The choice of transfers that Jones advised were as follows:

- PT transfer to the extensor carpi radialis brevis and longus (ECRB and ECRL)
- transfer of the flexor carpi ulnaris (FCU) to the extensor digitorum communis (EDC) of the third, fourth, and fifth fingers
- flexor carpi radialis (FCR) transfer to the extensor pollicis longus (EPL), to the EDC of the index finger, and to the extensor indicis proprius (EIP).

Jones[30] extended the transfer of the FCR to include the abductor pollicis longus (APL) and the extensor pollicis brevis (EPB).

Starr[31] seems to have been the first to mention the danger of using all the wrist flexors as transfers. He retained the FCU and transferred the FCR and palmaris longus (PL).

Zachary[32] demonstrated that it was necessary to retain one powerful active wrist flexor in order to stabilize the joint. His study showed that conservation of the PL is not always sufficient to stabilize the wrist.

The use of the FCU as transfer, which is the strongest flexor of the wrist, remains debated. Boyes[26], Tsuge and Adachi,[33] and Brand[34] recommend other combinations of transfers that leave the FCU in place. Biomechanical studies[34] have further analyzed these different types of transfers.

Principles of reconstructive surgery for radial palsy

A single transfer cannot act effectively on the tendon of five different muscles, as used by Jones.[30] A transferred tendon can act on different tendons of the same muscle, such as the EDC, but its action is dispersed or even cancelled if it is fixed on the tendons of different muscles that each have a specific function.

A tendon transfer used to extend the fingers will also extend the wrist if the latter is not stabilized. If the amplitude of movement has already been used to extend the wrist, the transferred tendon will not have sufficient action left to extend the fingers.

Therefore, the main lines of treatment are established in the following manner:

- Re-establish, by separate transfers:
  1. Wrist extension
  2. Extension of the proximal phalanges of the fingers
  3. Extension, spreading, and retropositioning of the thumb
- Conserve a strong muscle that will ensure stability of the wrist.

The choice of transfers and the surgical technique will be discussed for each of these three steps. We shall first evaluate previous techniques before describing the author's usual techniques.

Previous techniques

Re-establishment of wrist extension

Arthrodesis

For a long time, orthopedic surgeons who treated these lesions often performed arthrodesis of the wrist, because this procedure allows the utilization of freed muscles of the wrist for transfer. Now, everyone agrees that it is better to re-establish extension of the wrist, rather than an arthrodesis, because arthrodesis destroys the active tenodesis effect of wrist extension, necessary for a complete movement on the extrinsic tendons of the hand.

Tendon transfers at the wrist

Re-establishment of wrist extension is probably the most important aspect of palliative surgery for radial nerve palsy. Although there is general agreement regarding the use of the PT as the motor muscle, various other muscles are possible, including a wrist flexor a finger flexor, or even the brachioradialis (BR) when its innervation is preserved. However, the PT presents the greatest number of advantages because its force (1.2 kg) is somewhat superior to that of the ECRB or the ECRL alone, and its excursion (5 cm) is slightly greater to that of the ECR. The pronator quadratus (PQ) innervated by the median nerve continues to ensure pronation of the forearm. The main problem is the fixation of the PT on the paralyzed extensors of the wrist.

The three extensors (Figure 13.3) have different moment arms for wrist extension: the ECRL has a smaller moment arm than the ECRB for wrist extension, and its moment arm for radial deviation is greater than its moment arm for wrist extension, so that if the transfer is fixed on both tendons, the patient's wrist is forced into radial deviation when extended. Thus, it seems contraindicated to fix the transfer to both the ECRL and ECRB, as this would accentuate radial deviation and diminish extension of the wrist. Brand[34] recommends fixation solely to the ECRB. However, there is still a loss of active ulnar deviation and some radial deviation of the wrist. This is the major drawback of this transfer. Although patients are satisfied with the esthetic correction, many complain of fatigue and reduction in grip strength. Some authors have tried to compensate for this radial deviation by combining transfer

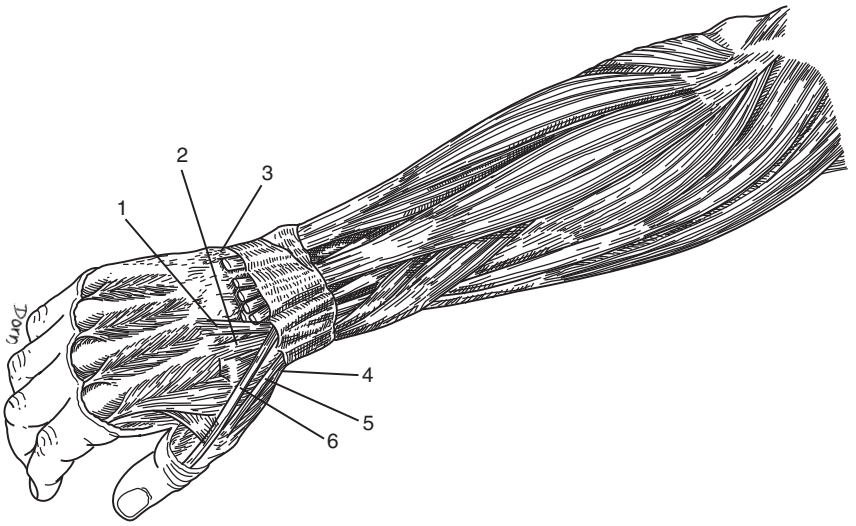


Figure 13.3
The three wrist extensors and the three thumb extensors: (1) ECU; (2) ECRL; (3) ECRB; (4) APL; (5) EPB; (6) EPL. The extensor digiti have been cut.

of the PT to the ECR and to the ECU at the same time.[35] This is not appropriate, because the extension moment arm of the ECU is even smaller than that of the ECRL and varies with rotation of the forearm; in pronation, the ECU exerts an action opposite to that of the radial wrist extensors.

Tendon transfers for the fingers

The FCU, the FCR or one or more flexor digitorum superficialis (FDS) may be chosen.

The FCU has a force of 2 kg, close to that of the extensors of the fingers; its excursion (3.3 cm) is less than that of the EDC (4.5 cm). The FCR is clearly weaker (0.8 kg) and its excursion (4 cm) is also less than that of the EDC. Thus, if a wrist flexor is transferred to the EDC, complete extension of the fingers can only be obtained by simultaneous flexion of the wrist. The use of the FCU, which is the strongest flexor of the wrist, is still debated: it reduces the force of flexion of the wrist and its ulnar deviation, and favors radial deviation. Tsuge and Adachi[33] and Brand[34] recommend combinations of transfers that leave the FCU in place. They proposed the following transfers:

- PT to ECRB
- FCR to EDC
- PL to EPL.

The transfer of the FCR while the FCU is conserved is certainly a technique that produces less radial deviation, but the strength of extension is less. However, one cannot hope to rely solely on the FCU left in place to maintain ulnar deviation while the wrist is extended, because the FCU cannot bring the wrist into ulnar deviation without flexing it at the same time.

Boyes[36] used two flexor digitalis superficialis (FDS) tendons. The transfer scheme is as follows:[37]

- PT → ECRL + ECRB
- FCR → APL + EPB
- FDS of IV → EPL + EIP
- FDS of III → EDC.

The balance of the forces between the paralyzed and transferred muscles is better, but the removal of the two FDS tendons reduces the strength and independence of these two fingers.

Tendon transfers for the thumb

The three long posterior muscles of the thumb column are the APL, the EPB and the EPL, all of which are relatively weak. These muscles can be divided into two groups, where axes of traction diverge and where courses differ (see Figure 13.3).

It is important to recall the following:

1. The excursion of the EPL is 5 cm and the excursions of the EPB and APL are only 2.5 cm. The same transfer should not be used on the three tendons whose excursions differ greatly.[38]
2. That the intrinsic muscles of the thumb contribute to the extension of the distal phalanx. In radial palsy, the distal phalanx of the thumb can be extended when the proximal phalanx remains flexed.

The usual motor muscles chosen are a wrist flexor or an FDS.

All of the possible combinations of transfers have been described.[39]

Different transfers do not require each muscle's individual action. Rather, separation of the thumb column in extension and retroposition is required, while avoiding, if possible, radial deviation of the hand. The simplest solution consists of making use of the same transfer for the EDC and the EPL, whose excursions are similar. However, the thumb loses all independence of movement, and although good retroposition of the thumb can be obtained, separation of the first commissure remains limited. To correct this defect,

one can use another transfer for the APL and EPB to obtain better separation. However, such a transfer might overload the radial component of the wrist.

Dunn[40] advocated transfer of the palmaris longus to the extensor pollicis longus for extension of the thumb. Scuderi[41] used the same transfer, but rerouted the EPL tendon lateral to Lister's tubercle, to minimize adduction when sutured with the PL. In this manner, abduction and extension of the thumb are obtained, and another transfer to the APL and EPB does not seem to be necessary.

However, the lateral location of the transfer prevents it from benefiting from the active tenodesis effect provided by wrist movements. Retroposition is not complete, and will only worsen with the tendon's tendency toward volar subluxation.

In some cases, an expansion of the EPB is inserted on the base of the distal phalanx of the thumb in addition to the normal insertion on the base of the proximal phalanx. Cuperman[42] advises systematic testing of the action of the EPB on the distal phalanx during surgery. When exerting traction on the EPB results in extension of the distal phalanx and abduction–extension of the thumb, it is preferable to fix the motor muscle directly to it rather than to the tendon of the EPL, because, as the EPB is in the same axis as the first metacarpal, it is not necessary to reroute it, as is done with the EPL.

The author's usual technique[43]

The choice of transfers is not original. The main changes made to the classic techniques consist of operative modifications. The following transfers are used:

- PT to the ECRB and to the ECRL rerouted
- FCU to the EDC and the EIP
- PL to the EPL rerouted.

The transfer for extension of the wrist is fixed only after the transfer on the digits, as the passive movements of the wrist are used to adjust the tension of the transfers.

Incisions

Rather than several small incisions, two long incisions are made on both sides of the forearm (Figure 13.4).

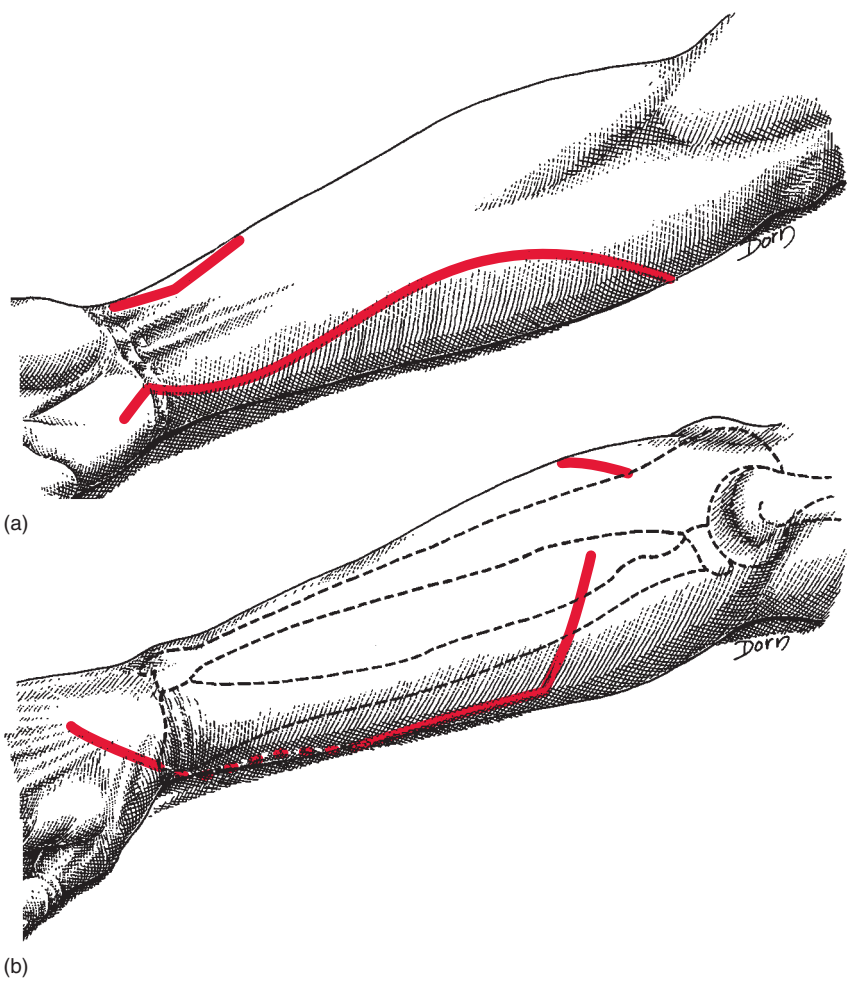


Figure 13.4
Exposure: (a) anterior approach; (b) dorsal approach.

Extensive exposure on both sides of the forearm has several advantages: It facilitates sufficient elevation and dissection of the motor muscles to provide a more direct pathway and avoid acute angulation of the tendon following transfer, and it allows lysis of the constantly present adhesions found around the length of tendons of the paralyzed muscles. These muscles may be partially excised if necessary.

Elevation of the transferred tendons

Elevation of the FCU and PL tendons

The FCU tendon is divided at the level of the distal flexion crease of the wrist. The muscle is freed, with a periosteal elevator throughout the length of the incision from its aponeurotic attachments. Occasionally, a small neurovascular pedicle joins the muscle distally, so it must be divided. The main pedicle is located proximally, joining the muscle at its deep surface about 6 cm from its origin from the epicondyle. The best method of protecting the pedicle is to expose it. The medial fascia is resected as necessary so that the FCU can pass from the volar to the posterior aspect of the forearm without angulation. A traction suture is passed through the end of the tendon to permit further manipulation. Because the muscle body attaches low on the tendon, it is preferable to strip the excess distal fleshy fibers, conserving only 5 cm of the exposed tendon (Figure 13.5). This reduces the cosmetically unattractive bulge of the transfer beneath the wrist and facilitates suturing.

The PL tendon is sectioned at its distal end. The sinuous anterior incision permits it to be freed throughout its length.

Freeing the extensor tendons

The large veins on the posterior aspect of the forearm are preserved. The dorsal fascia is thick distally; it is dissected so that the sutured tendons are able to glide readily in the subcutaneous tissues.

The EDC, EIP, EDM, and ECR are exposed. Traction is applied to these tendons in order to free them of adhesions within the osteofibrous tunnels (Figure 13.6).

Complete passive extension of the wrist and of each finger is essential.

Elevation of the PT

The antebrachial fascia is incised between the BR and ECRL. The tendon of the PT arises from the volar aspect of the forearm and curves around the lateral border of the radial diaphysis beneath the BR. The muscle is dissected and detached with a 2 cm strip of periosteum in order to make its insertion in the recipient tendons easier. A traction suture is placed in the end of the tendon, allowing traction on the PT.

At this point, the three motor muscles are ready for transfer.

Centralization of the ECRL tendon

To eliminate radial deviation of the wrist, we have tried several different procedures. First, the PT was transferred to the ECRB only. Then, to avoid the possibility of new adhesions between the two ECR tendons, which might create recurrent radial deviation, the ECRL tendon was divided distally and sutured to the ECRB. The correction of radial deviation still remained incomplete.

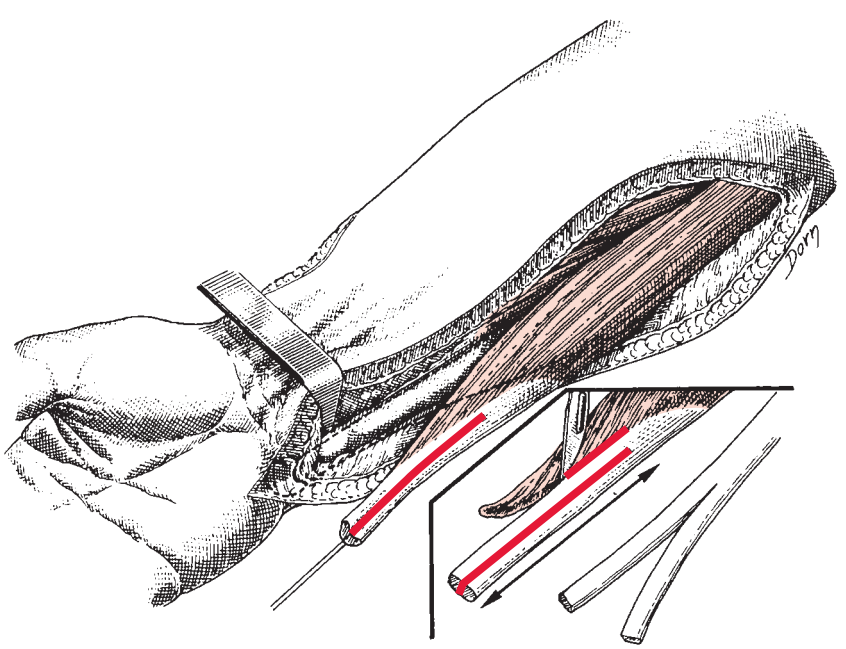


Figure 13.5

The FCU tendon is divided and the excess distal fleshy fibers removed. The tendon is split into two strips, each approximately 5 cm long.

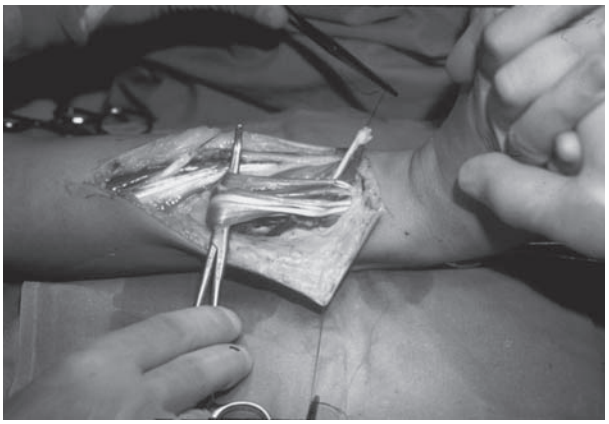


Figure 13.6
Freeing the extensor tendons.

We described in 1985 the centralization of the insertion of the ECRL tendon (Figure 13.7).[44] The tendon of the ECRL is divided at its insertion on the second metacarpal. It is pulled out of the dorsal retinaculum and freed up to the level of its musculotendinous junction. It is passed again under the retinaculum, but through the EDC tendon compartment. To make more room in this compartment, the EIP tendon is divided in the forearm at its musculotendinous junction, pulled out distally, and then passed over the retinaculum. The rerouted ECRL tendon is fixed to the ulnar side of the base of the third metacarpal with sutures and staples, medial to the insertion of the ECRB tendon, symmetrical to the ECRB with respect to the longitudinal axis of the wrist. This centralization is completed before any transfer to the thumb and fingers is performed.

Tourniquet release

It is important to release the tourniquet and secure hemostasis after the dissection, before fixation of the transfers, in order to set appropriate tension on the different transfers.

Fixation of the transfers

Fixation of the FCU on the EDC

The FCU is passed to the posterior aspect of the forearm.

As the tendon of the FCU is somewhat bulky, it is splinted into two strips, each approximately 5 cm long. One slip is passed through the tendons of the EDC, and the other, at the end of the procedure, will reinforce and adjust the sutures.

The suture is situated approximately 4 cm above the proximal edge of the dorsal retinaculum when the wrist is in a neutral position. When the wrist is completely flexed, the sutures must remain proximal to the retinaculum.

With an assistant holding the wrist in 45° of extension and the thumb and the fingers extended, the little, ring, long, and index finger tendons of the EDC are successively perforated with a sharp tendon clamp. These perforations are made in the oblique, distal, and radial directions. One strand of the split end of the FCU is pulled through the perforations. The tendons of the EDC are fixed to each other above the transfixation line with non-absorbable suture material, which prevents the hole from enlarging (Figure 13.8a).

If the EDC muscle is completely fibrous, 2 cm of the paralyzed muscle are resected proximal to this suture, in order to avoid an angulation at the level of fixation of the transfer.[45] Each perforated tendon is sutured independently to the motor slip. By varying the site of the suture of each tendon, it is possible to adjust the tension for each finger. Tension is more important for the radial fingers than for the ulnar ones. Motion in the wrist facilitates this adjustment. Secondary relaxation always takes place. Extension of the proximal phalanges should be possible when the wrist is in 25° of flexion.

Fixation of the PL to the EPL

The tendon of the EPL is sectioned at its musculotendinous junction in the lower third of the forearm and drawn distally out of its osteofibrous groove.

The technique of Scuderi has been modified. The EPL tendon is passed under the extensor retinaculum, but in the ECR compartment in place of the ECRL, which is previously rerouted. Thus, the EPL is in a more radial location and its new pulley prevents volar subluxation.

The PL, approached by the volar anterior incision, is also freed, so that it takes a straight course to meet the EPL. The two tendons are sutured on the radial side of the forearm, above the wrist, with the thumb in radial abduction, complete extension, and retroposition; the wrist in neutral position, and the PL under maximum tension. Reactivation of the EPL by the PL is sufficient to produce extension and abduction of the thumb column (Figure 13.9), making it unnecessary to perform another transfer to the APL, as its antagonist, the ECU, is paralyzed.

Fixation of the PT

The PT is passed subcutaneously for its new attachment into the ECR muscles. The periosteal strip the PT is fixed through the ECRB and the rerouted ECRL tendons just distal to their musculotendinous junction, with the transferred muscle being under tension. An assistant maintains the wrist in 45° extension, the metacarpophalangeal (MP) joints of the four fingers slightly flexed at 10°, and the interphalangeal joints in extension.

Fixation of the superficial slip of the FCU tendon

Adjustment of the tension of the FCU transfer on the digits is probably the most delicate part of the operation, which is why we prefer to fix last of all the second slip of the FCU tendon. Tension should be adjusted individually for each finger.

The EIP tendon, still unsutured, is now fixed to the FCU (see Figure 13.8b).

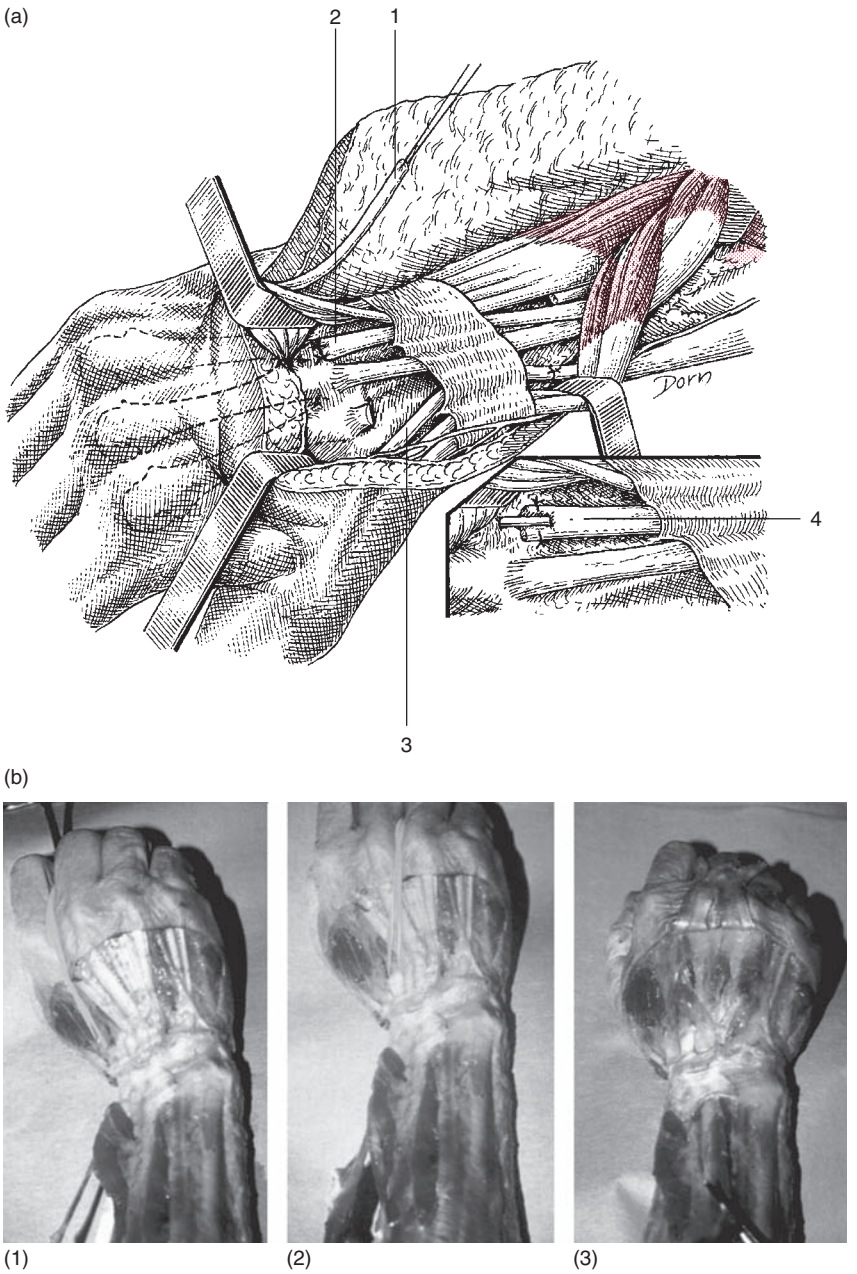


Figure 13.7

(a) Centralization of the ECRL tendon. The EPL tendon is rerouted. (1) EIP. (2) The ECRL tendon is passed through the EDC tendon compartment and fixed on the ulnar side of the base of the third MC. (3) The EPL is passed under the extensor retinaculum in the second compartment in place of the ECRL tendon to be sutured with the PL. (4) The ECRL tendon is fixed with sutures and staples. (b) Action of the centralization of the ECRL tendon. (1) In a normal wrist, traction of the ECRL causes marked radial deviation. (2) Traction on the ECRB also causes radial deviation of the wrist, although it is less pronounced. (3) Traction on the 'centralized' ECRL and on the ECRB provides extension of the wrist without deviation.

The extensor digiti minimi (EDM)

The EDM is left free unless complete extension by the EDC to the little finger alone is incomplete. This may be tested by pulling on the common extensor. If extension is incomplete, the EDM tendon should be tacked onto the transfer, but without excessive tension.

Technique maintaining the FCU in place and transferring one FDS

When the patient practices heavy manual labor without requiring great digital dexterity, one would tend to reinforce wrist extension by using a transfer of the FDS, especially in the dominant hand, and leaving intact the FCU. However, the technique utilizing the FDS is more delicate and the rehabilitation takes longer.

The following choices of transfers has been adopted:

- PT → ECRB and ECRL rerouted
- FDS of IV → EDC of II, III, IV and little fingers
- FCR → EPL + EIP
- PL → APL + EPB.

Incisions

The same approach is adopted on the posterior aspect of the forearm as in the previous procedure.

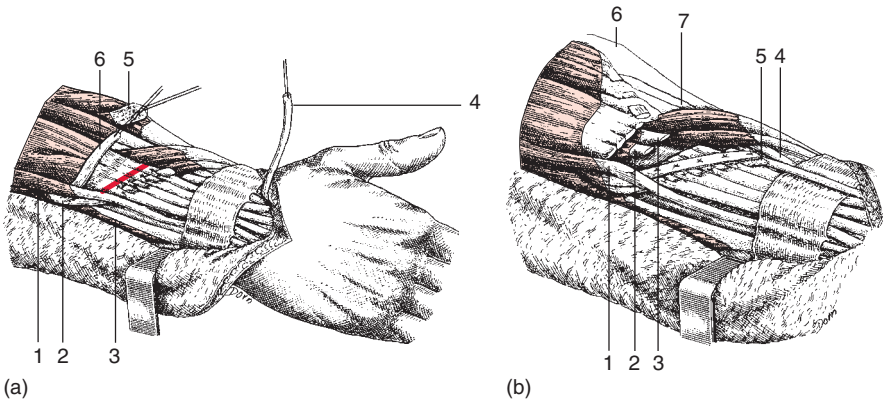


Figure 13.8

Transfer of the FCU to the EDC and EIP, of the PL to the EPL and of the PT to the ECR. (a) Fixation of the FCU to the EDC. One strip of the FCU is passed through each EDC tendon. Note the proximally placed suture on each EC tendon to prevent it spreading open at the fixation site. The wrist and fingers are in extension. (1) EDM; (2) FCU; (3) ECU; (4) EIP; (5) PT; (6) second strip of FCU tendon. (b) The EDC is divided proximal to the fixation when the muscle fibers are completely fibrous. (1) EDM fixed on the FCU; (2) proximal part of the EIP; (3) proximal part of EIP; (4) rerouted EPL tendon; (5) fixation of the EIP tendon on the FCU; (6) the PT tendon is fixed on the rerouted FCRL and on the ECRB; (7) rerouted EPL sutured to PL.

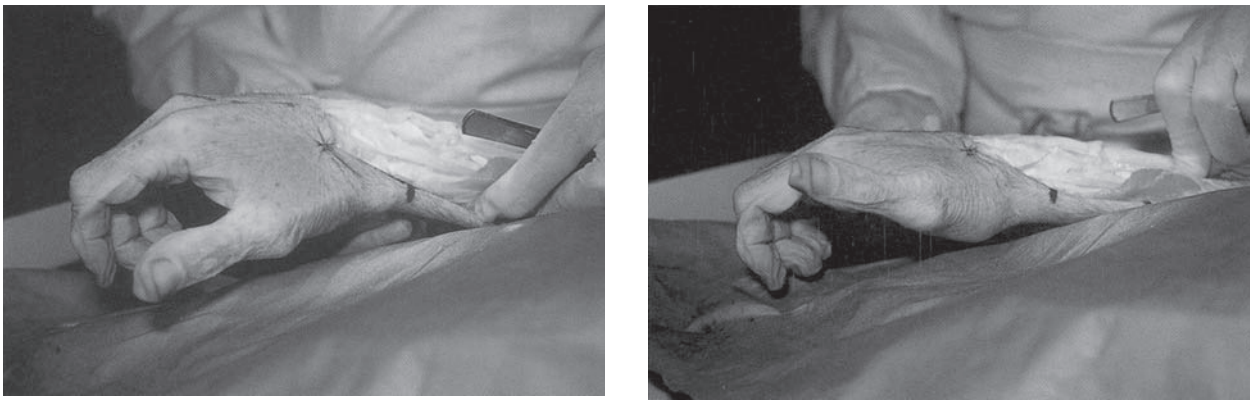


Figure 13.9

Transfer of the PL to the EPL tendon. (a) Scuderi's technique: the EPL tendon is rerouted lateral to Lister's tubercle. (b) Tubiana's modification: the EPL is rerouted through the ECRL compartment, showing better retroposition of the thumb.

On the volar aspect, a transverse incision is made at the base of the ring finger for the division of the FDS tendon proximal to the chiasma. The longitudinal sinuous incision in the forearm is more central than in the previous procedure.

On the dorsal aspect of the forearm

1. Elevation of the EIP, EPL, EPB and APL tendons (Figure 13.10a). All of these tendons are divided at the level of their musculotendinous junction and are extracted from their respective osteofibrous compartments.
2. Centralization of the ECRL tendon, as in the preceding technique.

On the volar aspect of the forearm (Figure 13.10b)

1. The muscular belly of the FS of the ring finger is freed and the muscle is passed medially to the FDP. The volar interosseous pedicle is located and retracted. A large window is made in the interosseous membrane at the proximal edge of the pronator quadratus, with fibrous membrane removal performed deep to the muscle. If the orifice is too proximal, the tendon will

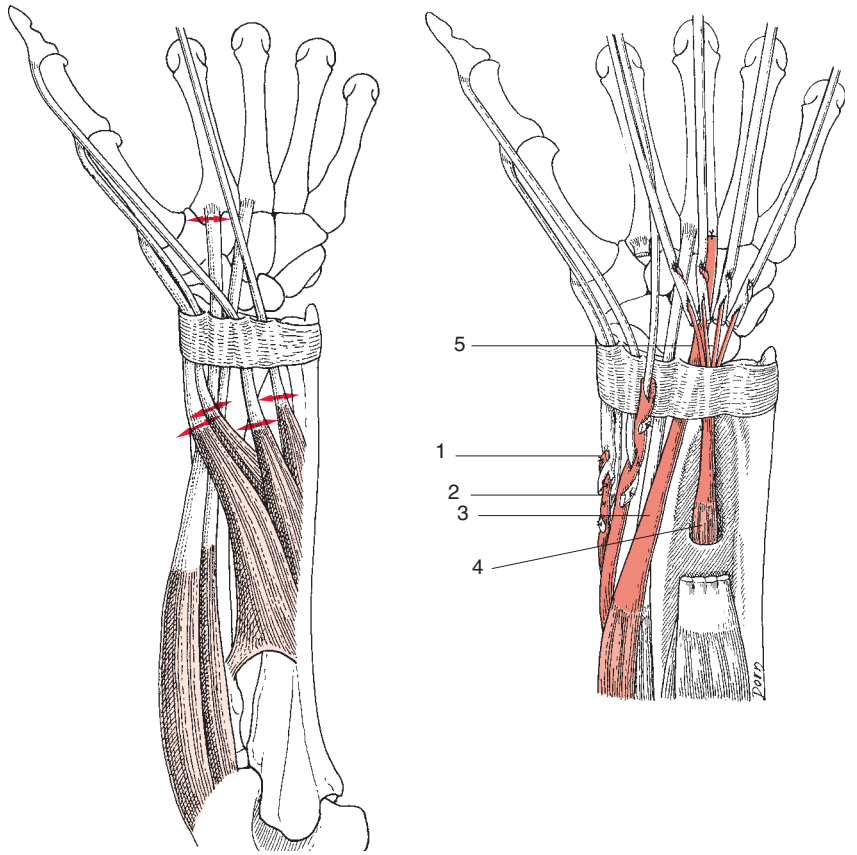


Figure 13.10

(a) Site of tendon divisions. (b) Transfer of the FCR and FDS tendons. (1) The PL is fixed on the EPB and APL tendons. (2) The EPL tendon is rerouted through the second compartment and the EIP tendon is passed over the retinaculum. These two tendons are fixed to the FCR. (3) The ECRL is rerouted. (4) The FDS is passed through the interosseous membrane, just proximal to the quadratus muscle. (5) FDS transfer to the EDC tendons distal to the retinaculum.

not have a direct route and may be caught on the distal edge of the window.

2. The FCR tendon is divided at the level of the wrist, and the FCR is freed up to the middle third of the forearm and passed posteriorly around the radius.
3. Elevation of the PL and PT.

Transfer of the FDS tendon to the EDC tendons

The FDS tendon is rerouted through the interosseous membrane in the posterior compartment and is then passed under the extensor retinaculum at the site of the extensor digitorum tendons, which have been resected. The FDS tendon is divided into four bands, which are sutured to the four EDC tendons on the dorsal aspect of the hand, distal to the extensor retinaculum, with the fingers held passively in extension and the wrist in 40° extension.

Transfer of the FCR to the EPL rerouted and EIP

The FCR tendon is fixed both to the EPL and to the EIP. The EPL tendon is rerouted through the second compartment as before and transfixed into the larger FCR tendon. This suture should be as proximal as possible, in order to prevent the limitation of gliding under the retinaculum.

The EIP fixation can be more distal, because its tendon is passed superficial to the retinaculum.

Transfer of the PL to the APL and EPB

The tendons are sutured on the radial side of the forearm, above the wrist, with the thumb in radial abduction, complete extension, and retroposition.

When the PL is missing, a tenodesis is possible. The APL and EPB tendons are divided at their musculotendinous junction about 6 cm above the radial styloid process. These tendons circle the styloid insertion of the BR and are then sutured back to themselves under strong tension (Figure 13.11).

Transfer of the PT to the ECRL and ECRB

The periosteal strip of the PT transfixes the ECRL and ECRB at the level of their musculotendinous junction as in the preceding technique.

Postoperative care

A volar splint, prepared before the operation, is applied to maintain the wrist in 50° of extension and 10° of ulnar deviation, with the interphalangeal joints in extension; the MP joints in 15° of flexion; the thumb in extension, abduction, and complete retroposition; and the forearm in full pronation. The splint should never place the wrist and

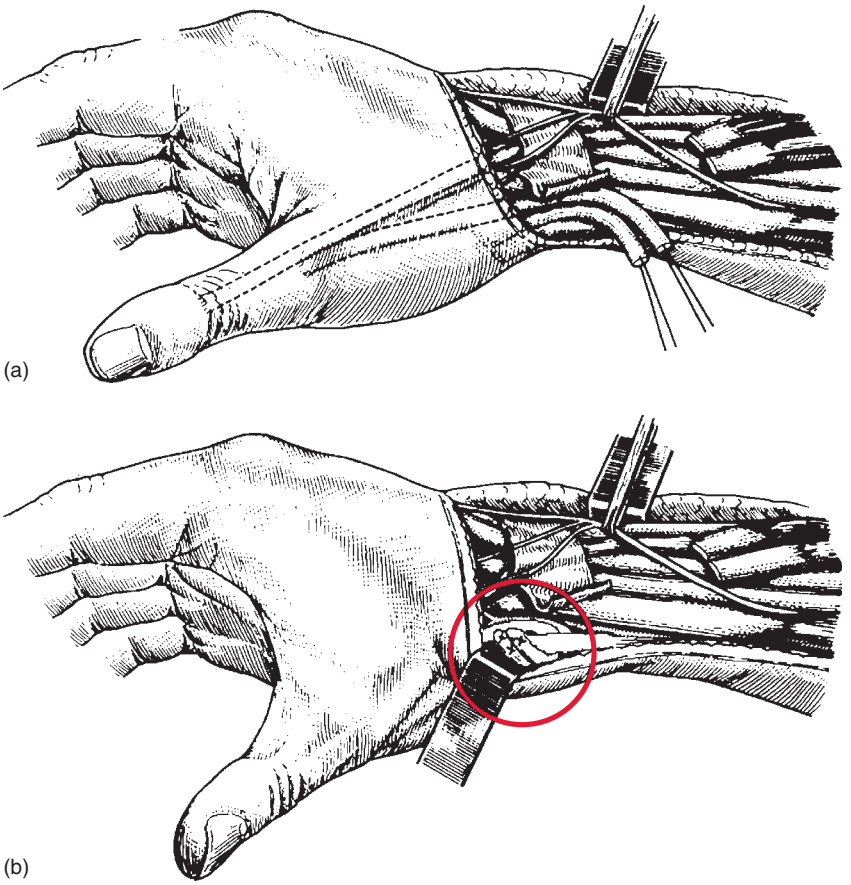


Figure 13.11

Tenodesis of the APL and EPB. Tendons around the insertion of the BR tendon. (a) Division of the APL and EPB tendons at their musculotendinous junction. (b) The two tendons circle the styloid insertion of BR and are sutured to themselves, with the thumb in complete extension and anteposition.

fingers in complete extension, and should extend above the elbow so that pronation and supination are avoided. This splint is worn continually for 2 weeks, after which the interphalangeal joints only are freed. The support is removed after 3 weeks, and is replaced by a dynamic splint composed of a dorsal antebrachial support on which is fixed a system of dynamic elastic appendages with harnesses placed on the volar aspect of the phalanges. Almost the entire palmar aspect of the hand is free.

Progressively, starting with the fifth postoperative week, active extension and complete flexion of the fingers are allowed. It is important to move the fingers and the wrist separately.

Indications for palliative surgery

Just as for all other palliative surgery for paralysis, the surgeon must, in each case, take into account the prognosis for reinnervation of the paralyzed muscles, the characteristics of each patient, and the extent of the paralyses.

Prognosis for nerve repair

With improvements in nerve repair techniques, it has become more common to treat patients with incompletely recovered radial nerve repairs rather than only those with a pure radial nerve palsy.

Although nerve repair has made undeniable progress, the time required for reinnervation of the paralyzed muscles may be so long after proximal lesions of the radial nerve that one may advise early tendon transfers.

The indications for early transfers are limited to instances in which, for any reason, nerve repair cannot be done, or the theoretical chance of nerve recovery is remote or projected to be very late (see Chapter 23 on timing of reconstructive surgery).

Particularities of each patient

There is now a greater tendency to conserve the FCU. However, in our standard technique, we transfer the FCU, because the technique is simple, the rehabilitation is easy and in most cases the results are satisfactory. We transfer the FDS only in young and cooperative patients, because the rehabilitation takes longer. Therefore, removal of FDS tendons is avoided if independence of individual fingers is desired, especially in those occupations utilizing keyboards.

In the absence of the PL, we prefer to use an FCR type of transfer. A tenodesis of the APL and EPB tendons wrapped around the insertion of the BR tendon is possible (Figure 13.11).

Extent of the paralysis

The extent of the paralysis depends on the nature and the level of nerve lesion and on the possible association with other peripheral nerve injuries.

The nature of the nerve lesions

A partial division of the radial nerve causes paralysis of only some extensor muscles and the palliative treatment must be adapted to the extent of the paralysis. Compression neuropathies of the radial nerve have been described at different levels.

Compression proximal to the elbow or at the level of the elbow results in muscle weakness or even in incomplete or complete paralysis. These paralyses usually recover after conservative treatment or surgical decompression; reconstructive surgery is rarely indicated.

Posterior interosseous nerve palsy

In distal palsy, affecting only the posterior interosseous nerve, innervation of the BR and ECRL is retained, and thus active wrist extension persists. The origin of the branch for the ECRB is variable, and it may be clinically difficult to confirm whether one or two radial wrist extensors are active.

Persistent innervation of the ECR muscles causes severe radial deviation. In this case, removal of the FCU results in an uncompensated disequilibrium.

In these distal paralyses, transfer of the FCR to the EDC seems particularly indicated. The ECRB and FCU must always be preserved.

When a severe radial deviation persists, one solution is to suture the FCU tendon to the ECU tendon; this technique makes the FCU into a pure ulnar deviator.[34]

Extensive paralyses

Whatever their etiology, the choice of transfers is limited in extensive paralyses caused by association of radial palsy with other paralyses.

It is first necessary to re-establish active wrist extension, as this movement provides flexion of the fingers when combined with tenodesis of the finger flexors and a thumb index lateral grip.

When the PT is paralyzed, a flexor of the fingers or a wrist flexor, or even the BR (if it still has its innervation) can be utilized. The BR is also innervated by the radial nerve, but its branches are more proximal than those of the ECR. Similarly, its nerves' origins in the cervical cord are higher, so this muscle is sometimes the only one that can be transferred in cervical cord paralyses. The BR originates on the distal third of the humerus and is essentially an elbow flexor. An active elbow extensor is required to allow the BR to be used as a wrist extensor. Although the BR is one of the most powerful muscles of the forearm, its excursion is very limited (1.3 cm). However, this can be considerably augmented by freeing all of its proximal forearm connections and disinserting its proximal portion on the humerus, with care being taken to avoid tension on a rather short neurovascular pedicle[46] (Figure 13.12).

When neither the BR nor a flexor of the fingers or of the wrist is usable, a transfer of the latissimus dorsi harvested with its fascia from the posterior crest of the ileum is able to reach the ECRB tendon.[47,48]

A free muscle transplant innervated by an intercostal nerve is also possible. Athrodesis of the wrist is avoided as much as possible.

Association with other peripheral nerves paralyses

High radial and high median nerve paralysis

In this severe type of paralysis, a wrist arthrodesis was used for a long time in order to use wrist flexors as transfers.[49-51] Now the transfer of the latissimus dorsi to the ECRB provides wrist extension and increases the strength of the grasp by a tenodesis effect. Flexion of the index and long

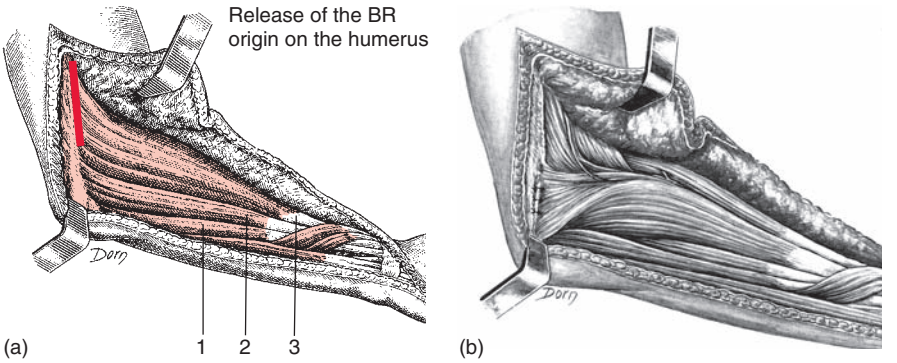


Figure 13.12

Transfer of the brachioradialis (BR). (a) The BR is essentially an elbow flexor. To increase its range of motion, it is necessary to free its muscular fibers and tendon adherences in the forearm. (1) ECRB; (2)ECRL; (3) BR. (b) To reduce its moment arm at the elbow, the proximal portion of the BR, inserted on the humerus, is elevated along with its accompanying periosteum and is folded upon itself. The neurovascular pedicle is quite short (4–5 cm), and must be located on the deep aspect of the muscle.

fingers can be restored by side-to-side suture of the flexor digitorum profundus (FDP) tendon of the ring finger to the two radial flexor profundus. The FCU is transferred to the EDC and the EPL. The choice of transfer to restore thumb opposition depends on the extent of ulnar nerve innervation in the flexor pollicis brevis (FPB). Frequently, both heads of the FPB are innervated with sufficient thumb anteposition, and arthrodesis of the thumb interphalangeal joint will provide enough stability for thumb pinch.

If the function of the FPB is inadequate, a transfer of the abductor digiti minimi (ADM) to the abductor pollicis brevis (APB), will restore thumb opposition. Forearm pronation is restored by rerouting the biceps brachi tendon around the proximal radius,[52] and a neurovascular skin island flap transferred from the ulnar aspect of the ring finger pulp will restore the thumb pulp sensibility.

The choice of transfers is broader in distal radial and median paralysis.

High radial and ulnar nerve paralysis

One should restore wrist and digits extension plus intrinsic function of the fingers and ring and little fingers flexion.

Wrist extension can be restored by a PT transfer.

Flexion of the ring and little fingers is obtained by side-to-side suture of FDP tendons to the index and long fingers corresponding tendons.

To prevent a claw deformity and allow active extension of the fingers' interphalangeal joints, different procedures can be used:

- The FDS of the long finger is transferred to the EDC and to the EPL tendons, associated with a capsulodesis of the MP volar capsule.
- Or the long finger FDS is used according to the Zancolli[53] lasso procedure for the MP joint stabilization associated with a tenodesis of the digits extensor tendons fixed in the radius and activated by wrist extension.

Radial stabilization of the index finger should be increased to improve the strength of the thumb lateral pinch. Transfer of the palmaris longus prolonged by the palmar fascia[54] is indicated in such cases (see Chapter 19).

High radial, median, and ulnar nerve paralysis

The functional loss is extremely severe and can be compared with a complete avulsion of the brachial plexus (see Chapter 20).

In these very extensive paralyses, no local transfer is available; only regional muscle transfers are possible using the biceps brachi[48] or the latissimus dorsi. If the latissimus dorsi is denervated, a free neurovascular muscle transplant may be the only possibility, and should be used for wrist extension, associated with several procedures of flexor and extensor tenodesis (see Chapters 20 and 29).

Muscular and tendinous lesions

Most of the muscular and tendinous lesions responsible for loss of active extension of the wrist and digits (Figure 13.13) also have a traumatic origin: dorsal wounds in the forearm (sometimes with loss of muscular substance), or division, rupture, or avulsion of extensor tendons of the wrist and digits.

Non-traumatic muscular or tendinous lesions can also cause a deficit or a lack of extension of the wrist and digits. They can be of vascular, metabolic, inflammatory, or degenerative origin. The most common of these diseases is rheumatoid arthritis.

We shall consider first injuries of the extensor tendons of the fingers and of the thumb, then extensor tendon ruptures in rheumatoid arthritis.

Injuries of the extensor tendons of the fingers

The repair of the extensor tendons of the digits is influenced by the location of the injury.

The International Federation of Societies for Surgery of the Hand has adopted the classification of the extensor tendons of the fingers into eight zones proposed by Verdan. The extensor tendons of the thumb are classified into four specific zones, and they share two zones with the extensors of the fingers at the wrist and at the forearm (Figure 13.14).

In the fingers, the extensor tendons are thin, superficial structures, lying immediately adjacent to the skeleton, and, when damaged, they tend to adhere to the skin and to the bone. Their range of motion is much less than that of the flexors, and therefore it is more difficult to compensate for any loss of length.[55] Generally, the approach to treatment varies according to whether the lesion is open or closed, recent or chronic. Because of the superficial position of the extensor tendons in the hand, their treatment following traumatic injuries depends largely on the condition of the skin. Crush injuries and avulsion wounds are frequent; their treatment should not impair flexion of the fingers, which represents the main function of the hand. Fracture reduction and fixation, tendon repair, and reconstruction of skin cover should be done simultaneously. However, if there is any risk of joint contamination, a delayed repair is preferable.

At the level of the fingers, the three phalanges constitute a multiarticular chain;[56] an injury of the extensor apparatus at one level may alter the balance of the whole finger.

Thus, division, rupture or avulsion of the central extensor tendon inserted on the base of the middle phalanx can lead to a boutonnière deformity. The two lateral extensor tendons sublux volarly and form a buttonhole through which the flexed PIP joint protrudes. The proximal retraction of the extensor apparatus increases the traction on the distal joint, which becomes hyperextended.

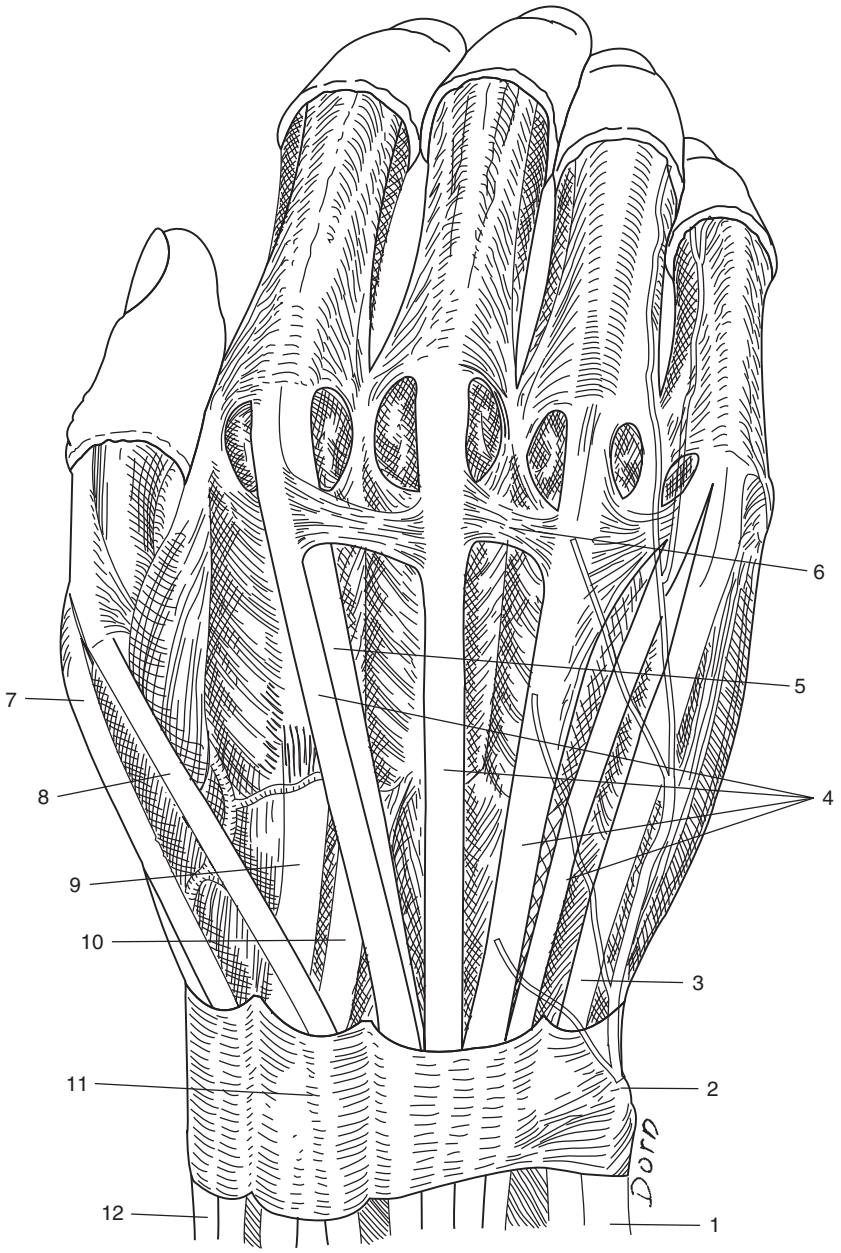


Figure 13.13

Extensor tendons of the wrist, thumb and fingers: (1) extensor carpi ulnaris; (2) sensory branch of the ulnar nerve; (3) extensor digiti minimi; (4) extensor digiti communis; (5) extensor indicis proprius; (6) junctura tendinae; (7) extensor pollicis brevis; (8) extensor pollicis longus; (9) extensor carpi radialis longus; (10) extensor carpi radialis brevis; (11) extensor retinaculum; (12) abductor pollicis longus.

Likewise, persistent flexion of the distal phalanx resulting from a lesion of the terminal extensor tendon can result in a swan-neck deformity caused by the proximal retraction of the extensor apparatus and excessive traction of the central extensor tendon.

It is important to treat these deformities at an early stage, when they are still correctable.

Clean lacerations at the MP level are sutured, followed by dynamic splinting.

Lacerations of the hood or sagittal bands are also repaired with interrupted 6-0 nylon sutures to prevent subluxation of the extensor tendon. If the joint is open, the capsule and the tendon are repaired separately.

Contaminated wounds are left opened, particularly with human tooth injuries. The wound is debrided and irrigated after a culture has been taken. A splint immobilizes the wrist in extension and the MP joint in 10° of flexion. The tendon may be repaired secondarily.

Extensor tendon injuries at the metacarpal level have a better prognosis than in the fingers, when tendons are thick enough to accept buried core type sutures.

Delayed suturing remains feasible for a long time in lesions distal to the juncturai tendinum, because these intertendinous connections prevent retraction of the proximal end. Delayed suturing, however, is not indicated in tendon lacerations proximal to the juncturai tendinum, which may be followed by important tendon retraction, because of the risk of severe extrinsic extensor tightness and the limitation of the flexion of the finger.

Associated lesions include extensive skin damage following crushing or avulsion, tendon tissue loss, and fractures of the underlying metacarpals. The treatment should stabilize

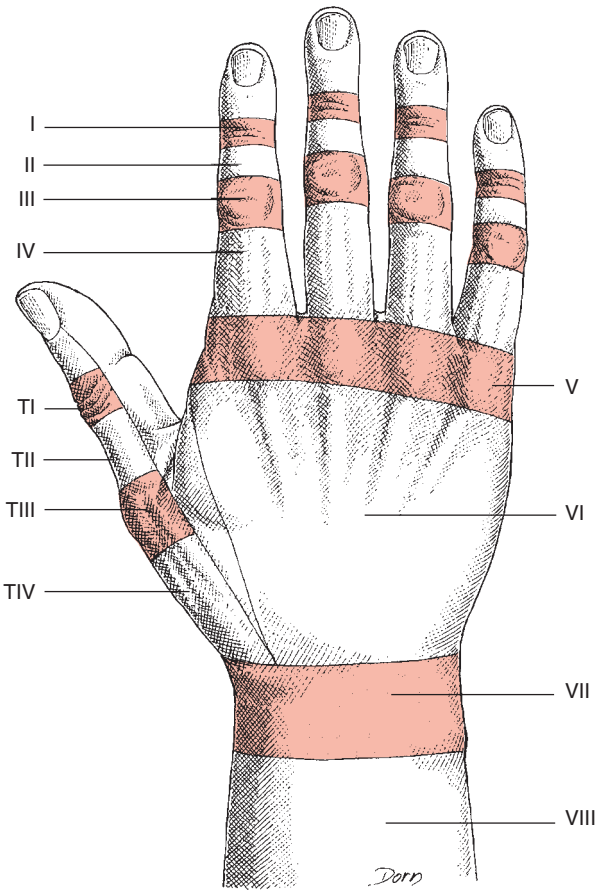


Figure 13.14

The extensor tendons of the fingers cross eight zones. The extensor tendons of the thumb cross six zones: four specific to the thumb, which are preceded by the letter T (thumb). Two zones are shared with the extensors of the fingers: zones VII or TV (wrist) and VIII (forearm).

the fractures, assure a good skin cover, repair the tendons, and prevent stiffness of the joints.

Late reconstruction of the extrinsic extensor tendons is in many ways similar to that of radial palsy or rheumatoid extensor tendon rupture. The main difference is the risk of scarring and adhesion after injuries. Suture to an adjacent tendon is the simplest method of repair. In more severe injuries involving several tendons, two alternatives are possible: tendon transfers or intercalated tendon grafts.

- Tendon transfer is our preferred method. When available, the extensor indicis proprius (EIP) provides an excellent transfer for the reactivation of one or two fingers extensor tendons. There is also the possibility of using the EPL or an FDS.
- Tendon grafts are only effective if the muscle contractility still has an adequate range of motion, and if there is not much adhesion and scarring.

Minigrafts have been used to bridge a short tendon loss.[57]

Injuries of the extensor tendons of the thumb

Extension of the thumb differs from that of the fingers in several respects (see Figure 13.13). The mechanism of phalangeal extension is simpler, because only two phalanges are involved. Each phalanx has its own extrinsic extensor: the EPB for the proximal and the EPL for the distal phalanx extension. As for the long fingers, intrinsic muscles participate to the extension of the distal phalange through the dorsal expansions of the abductor pollicis brevis (APB) and adductor pollicis (AP) to the EPL.

The movements of the first metacarpal control the motion of the thumb ray. Opening of the first metacarpal (anteposition–abduction) results from the action of both the intrinsic muscles (APB and opponens) and extrinsic muscles (APL and EPB). The EPL, whose course is quite different, because it is reflected around Lister's tubercle, adducts the first metacarpal while extending the thumb phalanges. Such complex action requires a much greater range of motion (5 cm instead of 2–5 cm) than that of the APL and EPB (see Chapter 16).

At the interphalangeal joint level, division, rupture, or avulsion of the EPL tendon creates a situation similar to that prevailing at the distal interphalangeal (DIP) joint of the fingers. The antagonistic action of the FPL produces a flexion deformity of the distal phalanx. In open injuries, the joint capsule is usually damaged by the injury, and the first aim of treatment consists of protecting the joint against infection. The tendon ends are brought together by positioning the joint in extension. The interphalangeal (IP) and MP joints and the wrist are immobilized in extension over a period of 4 weeks. Delayed tendon repair may be attempted if necessary.

Closed injuries are treated by 7 weeks of continuous splinting of the thumb, with the distal phalanx in extension.

At the level of the proximal phalanx and of the MP joint, the two extensor tendons (EPL and EPB) and the expansion of intrinsic muscles should be repaired. Care should be taken not to ignore an injury to the EPL when the distal phalanx retains some extension through the action of the intrinsic muscles, or an injury to the EPB when the MP joint can still be extended by the EPL.

At the level of the thumb metacarpal and of the wrist, the tendons form into two groups, which border the anatomic snuffbox. Each group – the APL and EPB radially and the EPL ulnarly – runs into a separate fibro-osseous compartment, clothed in its own synovial sheath at the lower end of the radius.

Ruptures of the EPL tendon are quite common, often preceded by fractures of the lower end of the radius. The origin of the rupture seems obvious, by attrition of the EPL tendon over a bony displacement or the extremity of a K-wire. However, these ruptures may occur after undisplaced radius fractures, at the level of Lister's tubercle; they probably result from ischemia.[58] There is a great variation in the time gap between injury and rupture. One should

not be misled by the presence of extension movements at the distal thumb phalanx. The impossibility for the patient of moving the thumb ray into retroposition with the thumb in abduction is a sign of EPL rupture. The lacerated tendon is explored through a curved longitudinal incision. The gap between the two tendon ends is usually large.

Although a tendon graft from the palmaris longus can give a good result, most authors use an extensor indicis proprius transfer (Figure 13.15a).

A curved longitudinal incision is made on the ulnar side of the head of the second metacarpal. The EIP and EDC are isolated. The ulnarly located EIP is freed and divided 1 cm proximal to its junction with the EDC. The distal stump of the EIP is sutured to the index EDC tendon. A second incision is made just proximal to the wrist dorsal retinaculum. The EIP is easily identified at the wrist because it has the most distal muscle belly. It is delivered with traction to the proximal wound. A third curved incision is made on the radial side of the MP joint of the thumb. The EIP tendon is then directed subcutaneously over the thumb MP joint. Nalebuff[59] advises not to connect the transfer with the distal stump of the ruptured tendon, but to pass the EIP tendon directly to the thumb MP joint level and weave it into the extensor mechanism (Figure 13.15b), with the thumb in complete abduction, extension, and retroposition, and the wrist in 15° extension.

The transfer should be sewed in tightly, because it will stretch when motion begins after 4 weeks of immobilization.

Congenital pollex flexus adductus

Congenital pollex flexus adductus is a fixed deformity of the MP joint of the thumb flexed into the palm. The deformity is due to the absence or hypoplasia of the EPB. The flexion–adduction of the MP joint is determined by the thenar muscle forces (which are not balanced, because of the absence of the EPB), alone, even with a normal EPL[60] (Figure 13.16a).

Pollex flexus adductus can be unilateral or bilateral: it is usually isolated, although it can be found in the context of an extended arthrogryposis.

The contracture of the palmar structures – skin, intrinsic muscles, and flexor pollicis longus – is not very severe at birth, and in some cases early treatment maintaining the thumb in abduction and extension with a splint for a few weeks may cure the anomaly. Even if it does not, this treatment prevents further contraction of the palmar structures and facilitates surgical correction.

Surgery must first release the first web space when necessary. The skin is incised along the opposition crease, and the different aponeurotic layers are divided until the thumb abduction is obtained. The EIP tendon is then transferred to the remnant of the EPB tendon (Figure 13.16). The ECRL tendon can be transferred instead, if the EIP is itself abnormal. The skin defect is filled by a full-thickness skin graft. Sometimes, in older children, it may be necessary to perform an arthrodesis of the MP joint.

Extensor tendon ruptures in rheumatoid arthritis

Extensor tendon ruptures associated with dorsal rheumatoid tenosynovitis are quite common. They are more frequent than flexor tendon ruptures. They are caused by attrition against a prominent bone spur or by direct invasion by hypertrophic synovitis associated with ischemic necrosis. They mostly occur in aggressive rheumatoid diseases.

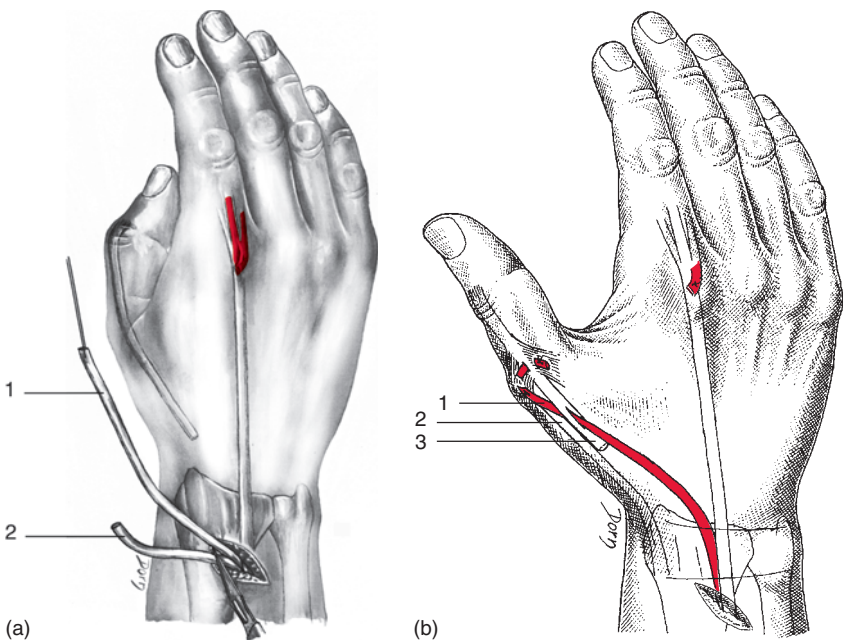


Figure 13.15

Rupture of the EPL. EIP tendon transfer. (a) Elevation of the EIP tendon, on the ulnar side of the index EDC tendon. The distal stump of the EIP is sutured to the EDC tendon. Through an incision proximal to the wrist dorsal retinaculum, the EIP tendon is pulled out and then transferred subcutaneously over the thumb's MP joint. (1) EIP; (2) proximal stump of EPL. (b) The EIP tendon is passed through the distal stump of the ruptured EPL tendon and then into the extensor mechanism at the MP joint level. (1) EIP; (2) EPB; (3) distal stump of EPL tendon.

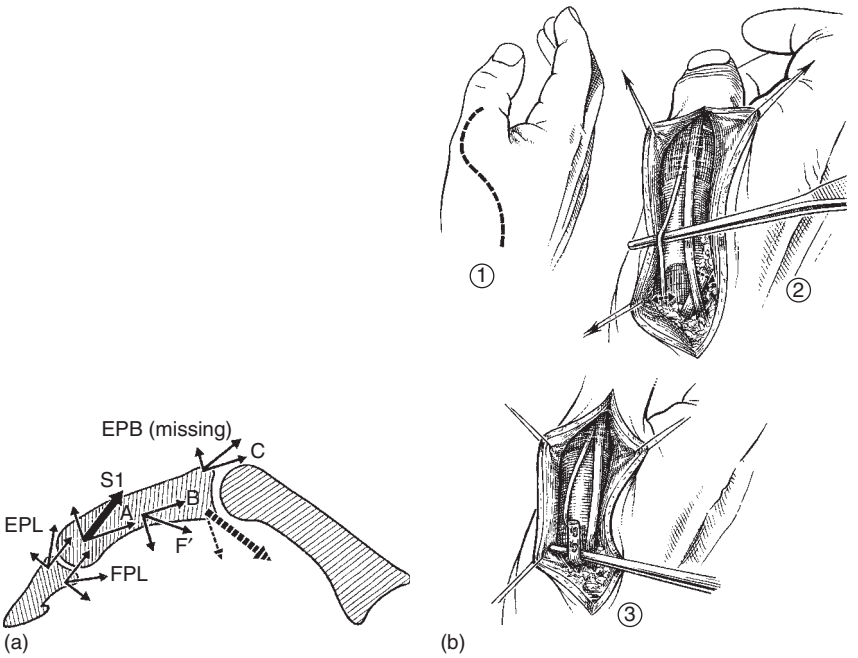


Figure 13.16

Pollex flexus adductus. (a) In EPB absence or hypoplasia, the EPL cannot extend the thumb's proximal phalanx, because its resultant force $\mathbf{S}_1$ rather induces an extension–retroposition of the first metacarpal, and the proximal phalanx is maintained in flexion by the predominant action of the thenar muscles. (b) (1) Skin incision. (2) Division of the remnant of the EPB tendon. (3) In this case, the ECRL is used as a transfer. In the case of total absence of the EPB, the extensor indicis proprius tendon is transferred.

They can sometimes be first confused with a posterior interosseous nerve palsy; the clinical tests in favor of a nerve compression are described at the beginning of this chapter.

Their treatment must include a tenosynovectomy with tendon repairs.

Extensor tendon ruptures are usually treated by adjacent sutures and tendon transfers, sometimes by joint fusion, and occasionally by tendon bridge grafts. Direct repair between the tendon ends is rarely possible, because of the rheumatoid lesions and of the proximal muscle contracture.

The tendons transfer technique in rheumatoid arthritis is more complex than in paralysis, and the results are less assured because deformity or stiffness of the wrist and digital joints have adverse effects. In particular, the tenodesis effect of wrist motion is often missing. Also, the choice of transferable tendons is restricted by synovitis invasion of the tendons, and the poor conditions of the path in which the transferred tendon has to glide. Tendon transfers cannot work properly across dislocated joints. If these joints need surgery, tendon transfers should be delayed.

The most common extensor tendon ruptures are those to the digital extensors of the little finger at the distal end of the ulna and of the EPL at Lister's tubercle.

Ruptures of the extensor pollicis longus (EPL)

These ruptures are usually located at Lister's tubercle. Interphalangeal motion is still possible through the intrinsic muscles.

We have already described the treatment of ruptures of the EPL by an EIP transfer, which usually gives satisfying results. The EPB[61] and a slip of the APL[62] have also been used as transfers.

Ruptures of the extensor tendons of the fingers

The site of ruptures is usually at the wrist level; however, it may be more distal when the rupture is caused by invasion of the tendon by synovitis.

At the wrist level

The extensor tendons, with their synovial sheaths, pass under the dorsal retinaculum on the dorsum of the wrist before diverging toward the fingers. Synovium is not confined under the extensor retinaculum and can extend to the dorsum of the hand. Tendon ruptures are frequent at this site, by attrition and direct invasion. Rupture of the extensor tendons of the little finger is most common, and is often the prelude to ruptures of the extensor tendons of the ring finger and then of the other finger extensor tendons. Tendon rupture of the little finger constitutes a indisputable indication for surgery, not only to repair the tendon but also to prevent other ruptures. A synovectomy should be performed as soon as possible, with careful removal of any bony projection that might have been responsible for tendon attrition.

Two extensor tendons are usually described at the level of the little finger: the extensor digitorum communis (EDC) and the proprius. In fact the proprius is often the only functional extensor of the little finger (in 32 cases of 57 for Schenck[63]).

The diagnosis of rupture of the finger extensor tendons relies on the defect of active extension of the proximal

phalanx. The middle and distal phalanges can be extended by the interosseous muscles.

The diagnosis of rupture of the extensor tendons in the little finger is not always made, for the defect in extension of the finger may be due to subluxation of the tendon. To make this diagnosis, the finger is placed in passive extension, and the patient is asked to maintain this position. If the patient is unable to do this, there is probably a rupture. When possible, one can also flex the wrist, which, as a result of a tenodesis effect, will extend the finger if there is a subluxation but not if there is a rupture.

Single rupture to the little or ring finger

Adjacent side-to-side suture is the best procedure when the distal stump is long enough.

After completion of the dorsal tenosynovectomy, the dorsal retinaculum is relocated under the extensor tendons. It will provide a gliding plane for the tendons, and reinforces the wrist capsule. No functional disturbance usually results from this procedure, because the limitation of wrist extension in most rheumatoid patients will avoid a bowstringing effect of the tendons (which occurs at more than 40° of wrist extension). Tendon repair is carried out by suturing the ruptured tendons to the adjacent digits at full extension.

If side-to-side suture cannot be done, the best solution is then transfer of the EIP to the ruptured tendon. The EIP is an excellent tendon to use, because it is an in-phase transfer; the excursion is the same as the ruptured tendon, and the disability to the index finger is minimal.

Double rupture to the ring and little fingers

This is the most common double rupture, usually caused by attrition. The EIP is transferred to the little finger extensor tendons and the ring EDC tendon is sutured side-to-side to the middle (Figure 13.17a).

Adjustment of the tension of the transfer is probably the most delicate part of the operation. Flatt[10] advises to pull the motor tendon unit distally about two-thirds of its total travel distance and hold the fingers fully flexed and the wrist in slight extension. In order to assure proper tension, Nalebuff[64] recommends that the surgery be carried out under intravenous lidocaine anesthesia. Before closure, the tourniquet is deflated and motor power returns within 2–3 minutes. The patient can then demonstrate range of motion. Local infiltration is used for skin closure.

Triple and quadruple ruptures

These are more difficult to treat, not only because use of the EIP transfer is no longer sufficient, or even possible, but also because all other tendon transfers present drawbacks. The two most convenient transfers are the *extensor pollicis longus* (EPL) and the *flexor digitorum superficialis* (FDS).

The EPL has a long tendon, and a good 5 cm range of motion. It is not strong, but is sufficient for the extension of two digits. It can only be used if the MC joint of the thumb needs an arthrodesis; this is quite common in rheumatoid hands. In cases of triple ruptures, the EPL may be transferred to the fourth and fifth extensor tendons, and the third extensor tendon is sutured side-to-side to the index communis extensor tendon (Figure 13.17b). The EPL tendon is divided at the level of the thumb MP joint. The thumb MP joint is fused. The IP joint of the thumb will be extended by the intrinsic muscles, and additional power can be obtained by suturing the extensor pollicis brevis to the distal stump of the EPL.

In multiple ruptures, when two motor transfers are available, one motor is used for the index and long finger,

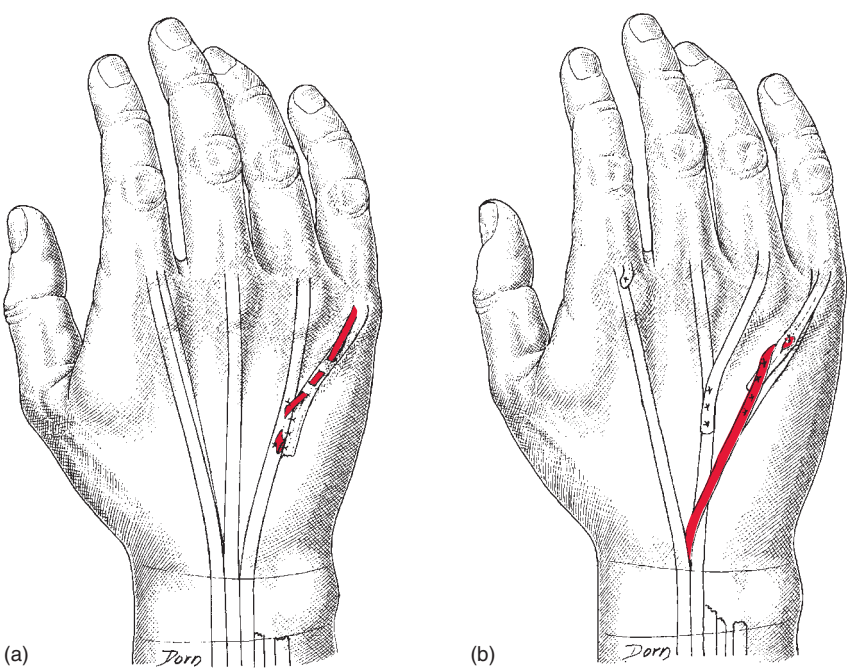


Figure 13.17

Extensor tendon ruptures. (a) Rupture of the little finger extensor tendons. Adjaced side-to-side sutured. (b) Rupture of the ring and little finger extensor tendons. The EIP tendons is transferred to the fifth extensor tendons. The fourth EDC tendon is sutured to the third.

sutured with relatively taut tension for precision grip, and the other sutured, with less tension on the ring and little fingers used for grasping.

One FDS can be used – the third or the fourth. It is a strong muscle with a long tendon and a long range of motion, and it can be transferred to three digits (Figure 13.18). However, it may have been weakened by flexor synovitis, and its removal reduces the strength, and independence of the finger from which it was taken. We prefer to use a direct passage through the interosseous membrane. The muscle is passed ulnarly to the FDP. The volar interosseous pedicle is retracted and a large window is made in the interosseous membrane at the proximal edge of the pronator quadratus. A subcutaneous passage around the radius is used when scar existed from previous dorsal wrist surgery.

Wrist extensors and wrist flexors have been advocated for transfer, but their tendons are often not long enough and they have only a short range of motion. These may work when there is a good wrist motion to add a tenodesis effect. In cases of wrist destruction or arthrodesis, multiple wrist tendons are available, but with limited range of motion. In such cases, it is more effective to use a transfer with a long range of motion. An intercalary 'bridge graft' using the PL tendon may be another possibility in cases of multiple extensor ruptures; however, the results are not as good as after tendon transfers.

Rheumatoid finger deformities

They usually result in lack of extension at one or several finger joints.

When the MP joints have been invaded by the synovial pannus, the distention of the fibrous elements at the joint leads to a flexion deformity of the MP joint. Hypertrophy of synovium at the MP joint level is often associated with an ulnar displacement of the extensor tendons. Ulnar subluxation of the extensor tendons is certainly not the only cause of ulnar deviation of the fingers. However, their displacement into the intermetacarpal space contributes to the fixation of this deformity.

Distention by rheumatoid pannus of the sagittal bands that constitute the proximal insertion of the extensor tendon on the MP joint volar plate will allow volar subluxation of the base of the proximal phalanx under the action of both the long flexor tendons and the intrinsic muscles. This subluxation increases the traction of the extensor apparatus on the base of the middle phalanx, being one of the most frequent factors in the development of a swan-neck deformity.

Rheumatoid boutonnière deformity is caused by synovitis of the proximal interphalangeal (PIP) joint – weakening of the dorsal capsule and of the central extensor tendon

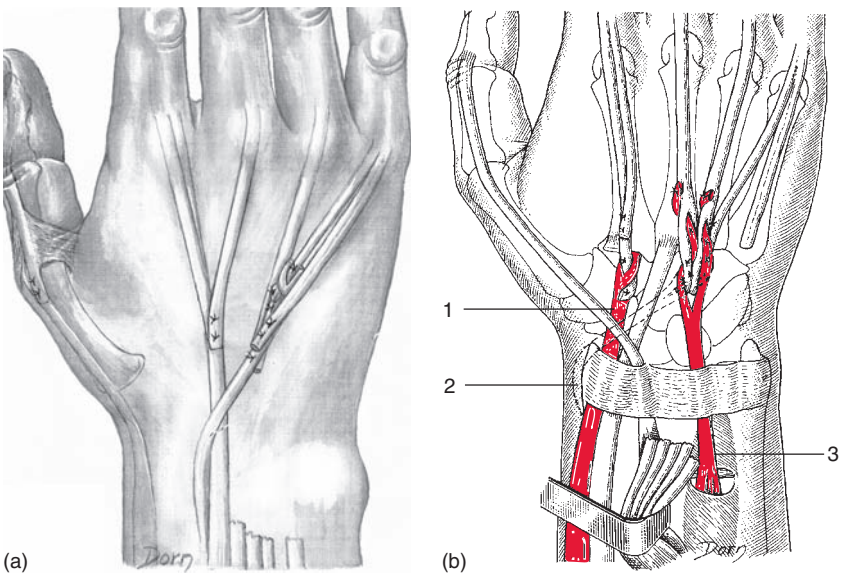


Figure 13.18

(a) ELP transfer. Triple extensor ruptures: EPL tendon transfer. The EPL tendon is divided proximally to the MP joint. The MP joint of the thumb is fused. The IP joint of the thumb will be extended by the intrinsic muscles and by the suture of the distal stump of the EPL tendon to the EPB tendon, which has been removed from its insertion at the base of the proximal phalanx. A temporary K-wire is placed obliquely through the thumb IP joint to maintain extension. The proximal end of the EPL tendon is transferred to the fourth and fifth extensor tendons and the third extensor is sutured to the second EDC tendon. (b) FDS transfer. Quadruple extensor ruptures. In this case, a flexor superficialis has been used, either the third or the fourth, to extend three fingers, and the ECRL has been transferred to the index extensors (1). The FDS tendon is transferred either directly through the interosseous membrane (3) or around the radial aspect of the forearm (2). This subcutaneous passage is chosen when there is a tendency for an ulnar deviation of the fingers or when previous dorsal wrist surgery with scarring has been performed.

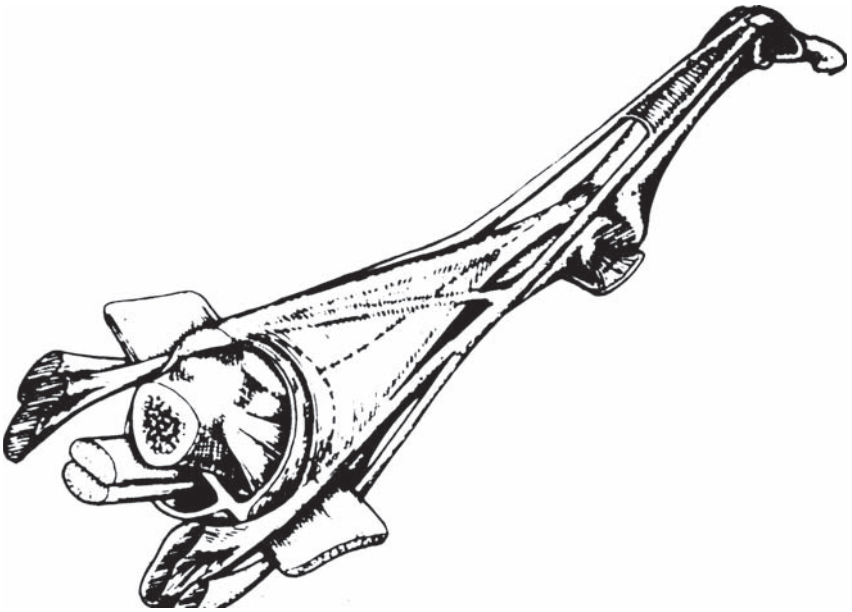


Figure 13.19

Ulnar subluxation of the extensor tendon at the MP joint, associated with ulnar deviation of the finger, will increase the extensor tendon traction on the base of the middle phalanx and produce a swan-neck deformity.

Figure 13.20

Rheumatoid hand: subluxation of the base of the proximal phalanges with ulnar deviation of the fingers and marked flexion deformity.

inserted at the base of the middle phalanx leads to elongation, rupture or detachment of this tendon, and loss of PIP extension.

Articular synovitis in the distal interphalangeal joint can cause rupture of the terminal extensor tendon, which has two important functional repercussions: (1) an inability to extend the distal phalanx (mallet finger) and (2) proximal retraction of the extensor apparatus, reinforcing its traction on the middle phalanx and leading to a swan-neck deformity if the PIP joint is lax, as is often the case in rheumatoid arthritis.

Classification

We have adopted a common four-stage classification of all chronic finger deformities with respect to musculotendinous imbalance.[43] This classification proceeds with some modifications, from Nalebuff's classification of rheumatoid swan-neck deformity:[65]

Stage 1: There is no limitation of passive motion in any finger joint

Stage 2: Tenodesis effect: limitation of motion in one joint is influenced by the position of other finger joints

Stage 3: Fixed deformities: X-rays do not show articular lesions; limitation of joint motion is not influenced by the position of another finger joint; the limitation may be due to soft tissue contracture or adhesions

Stage 4: Radiography shows significant articular lesions.

This classification is used as a guide for the therapeutic indications at each joint level.

We shall summarize these indications.

At the metacarpophalangeal joint

Chronic rheumatoid deformities at this level are essentially ulnar deviation and flexion deformity. We use the classification already mentioned:

Stage 1: no limitation of passive motion

The extensor tendon is usually subluxated on the ulnar side of the joint, it is mobilized by incising the extensor hood on the ulnar side. The joint capsule is opened and synovitis excised. The radial fibers of the extensor hood are then reefed to realign the tendon over the middle of the joint.

Stage 2: tenodesis effect

Ulnar subluxation of the extensor tendon associated with ulnar deviation of the finger may increase the extensor

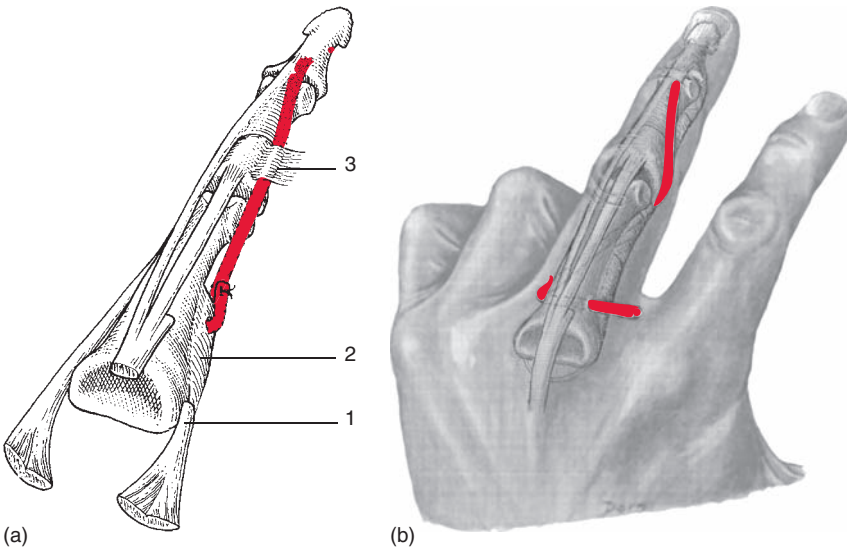
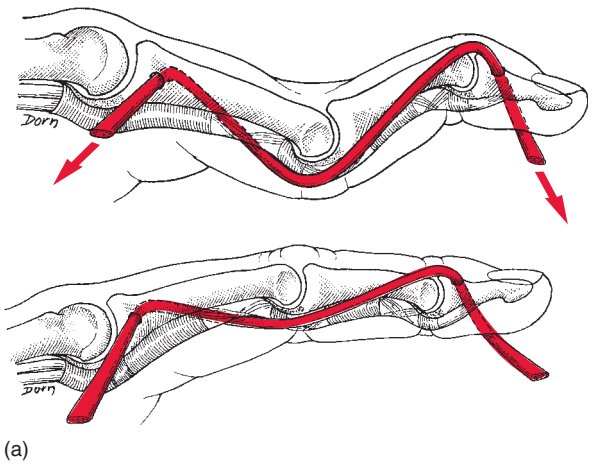


Figure 13.21

(a) Oblique retinacular ligament reconstruction: lateral band technique.[57] (1) interosseous muscle; (2) flexor tendon sheath; (3) Cleland's ligament. (b) The lateral band spiral technique.[66]



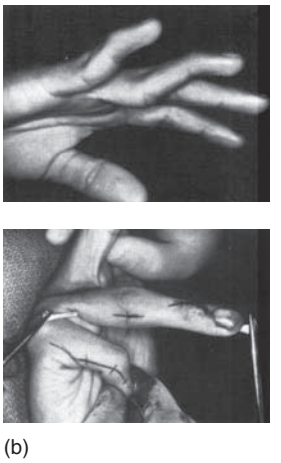


Figure 13.22

(a) Spiral oblique retinacular ligament reconstruction: Littler's[67] tendon graft technique.[67] Two through-and-through holes are made: the first in the distal phalanx in an anteroposterior direction proximal to the nail matrix and distal to the FDP insertion: the second transversely through the base of the proximal phalanx. The slender graft crosses the interphalangeal joints and makes them act together.
(b) Clinical example.

tendon traction on the base of the middle phalanx and produce a swan-neck deformity (Figure 13.19).

Surgical correction is indicated before the deformities become fixed. The extensor apparatus should be realigned over the middle of the MP joint. An ulnar intrinsic release is performed, and PIP joint hyperextension must be corrected to prevent recurrence of the deformity.

Stage 3: fixed flexion deformity

Articular surfaces are intact. A soft tissue release is performed usually in conjunction with a MP joint arthroplasty.

Stage 4: lesions of the articular surfaces (Figure 13.20)

If surgery seems necessary, especially when there is a marked flexion deformity of the MP joints, the only choice is an MP joint arthroplasty. The most commonly used is the Swanson silicone rubber implant arthroplasty.

PIP joint deformities

Two deformities are seen at this level in the course of rheumatoid arthritis: the boutonnière and the swan-neck.

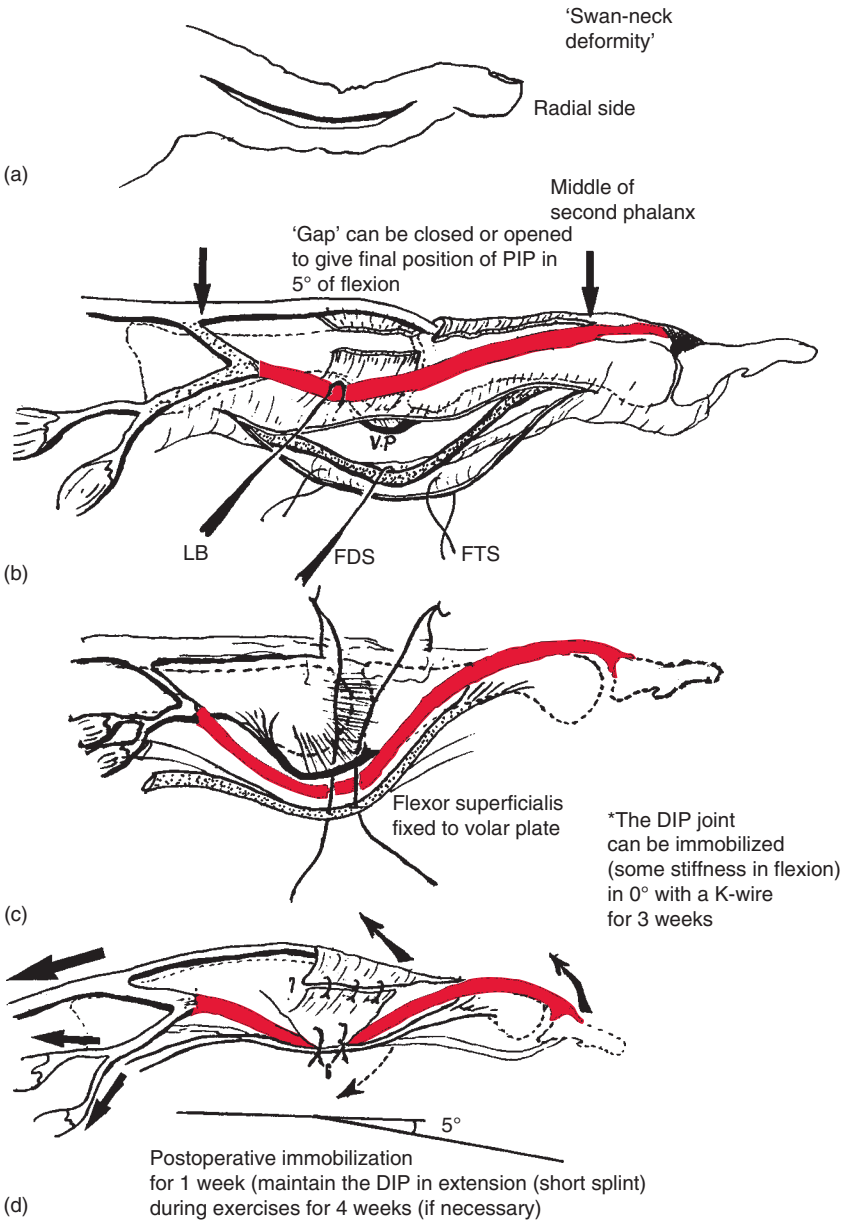


Figure 13.23

Transloation of the lateral extensor tendon by kind permission of Zancolli.[68]

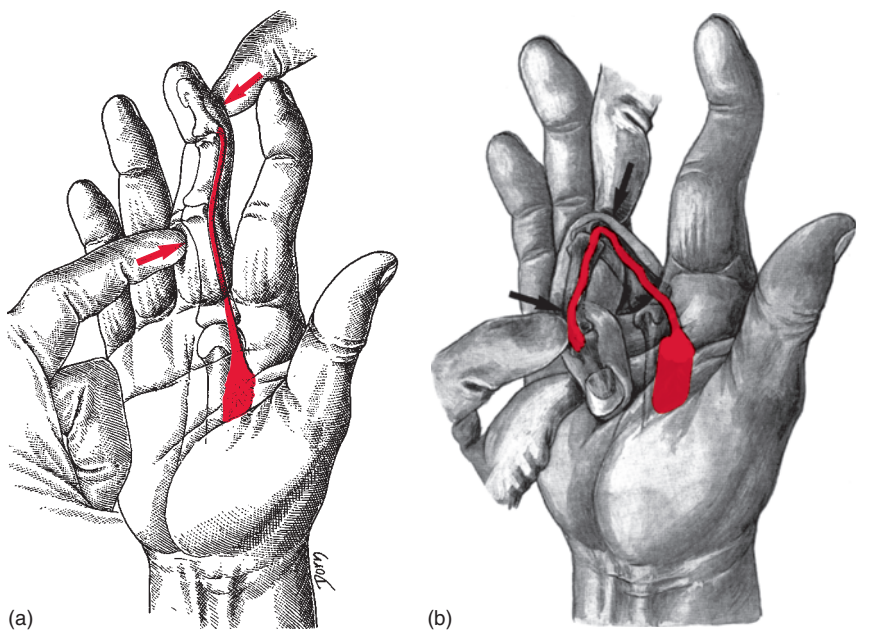


Figure 13.24

Intrinsic tightness. (a) The Bunnell–Finochietto test. When the proximal phalanx is maintained in extension, the distal phalanges cannot be flexed. (b) Flexion of the proximal phalanx allows flexion of the distal phalanges.

These are progressive conditions. Corrective splinting and exercises are used at all stages; alone or in conjunction with surgery, they constitute an essential step prior to any operation.

The rheumatoid swan-neck

It is important to treat swan-neck deformity at an early stage to prevent stiffness of the PIP joint in extension. It is also necessary to treat the cause of the deformity, which in rheumatoid arthritis has its origin at a different level from the wrist to the distal interphalangeal joint.

A common cause is excessive traction on the central extensor tendon inserted on the base of the middle phalanx.

Stage 1: no limitation of passive motion in any finger joint

Treatment is aimed at correcting the dynamic imbalance at the two interphalangeal joints. Many surgical procedures have been proposed; they should be selected taking into account the quality of the extensor and flexor tendons.

Correction of PIP joint hyperextension can be obtained by different types of tenodesis. Littler[57] suggested reconstruction of an oblique retinacular ligament, using the ulnar lateral extensor tendon, held volar to the PIP joint axis, thus serving as a check-rein to prevent PIP hyperextension (Figure 13.21a). This ligament also serves to tenodesis the DIP joint into extension as the PIP actively extends. The original proximal attachment was through a window in the flexor tendon sheath at the A2 pulley level. For a more secure repair, the lateral band may be rerouted volarly in a spiral fashion, across the digit and then through the bone at the base of the proximal phalanx[66] (Figure 13.21b). A modification of this technique uses a tendon graft if the quality of the extensor tendon is poor[67] (Figure 13.22).

In translocation of the lateral extensor tendon,[68] the lateral extensor tendon, usually on the radial side, is freed at the level of the PIP joint. It is subluxated volarly in front of the volar plate and maintained in this position by suture of the volar plate to the FDS tendon. An oblique retinacular ligament is created. This technique is simple and effective (Figure 13.23).

Flexor superficialis tenodesis can also be used when the quality of the extensor tendon is poor. One slip of the FDS, proximally divided, is fixed to the flexor tendon sheath, at the A2 pulley level.

Stage 2: tenodesis effect

The Bunnell–Finochietto test (Figure 13.24) indicates an intrinsic muscle contracture. Resection of the oblique fibers of the interosseous hood at the level of the proximal phalanx restores flexion of the distal phalanges when the proximal phalanx is held in extension[57] (Figure 13.25).

Stage 3: fixed deformity

Articular surfaces are intact. Contracture of the extensor apparatus and of the dorsal skin, and later of para-articular structures, fixes the deformity. Nalebuff[64] has shown that gentle manipulation into flexion, associated with splinting and an exercise program, may progressively correct the deformity. If not, one should proceed to a dorsal soft tissue release by freeing the lateral extensor tendons from the central followed by a PIP joint tenodesis to prevent recurrence of the deformity (Figure 13.26).

Stage 4: joint stiffness with PIP articular surfaces destroyed

The only two alternatives are arthroplasty or fusion of the PIP joint. The choice depends mostly on the condition of the two other joints of the finger.

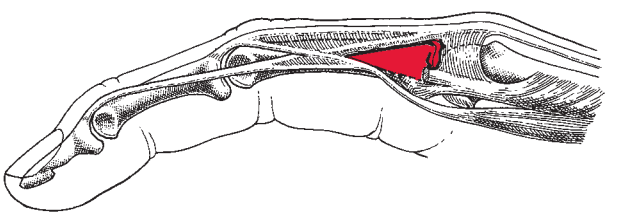
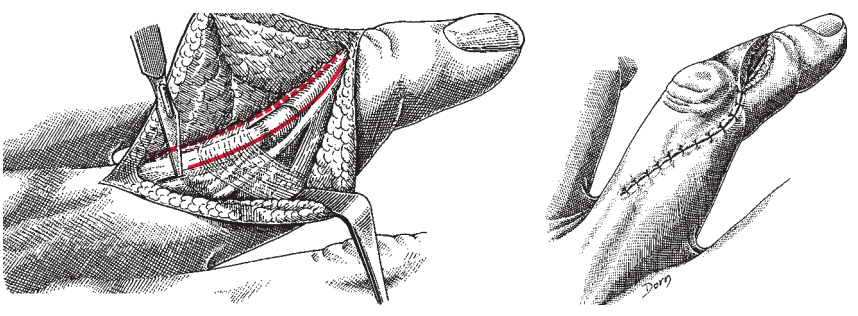


Figure 13.25

Triangular resection of the oblique fibers of the interosseous hood and of the central band of the interosseous tendon. Neither the central tendon, not the lateral bands of the extensor and of the interosseous tendons, nor the transverse fibers of the intrinsic aponeurosis should be resected.



(a) (b)

Figure 13.26

(a) Soft tissue release: longitudinal incisions along the central extensor tendon. The two extensor lateral tendons are tenolyzed and mobilized from the central tendon in order to recover their normal volar shift. (b) The incision is closed proximally so that the extensor mechanism is covered at the joint level. The distal third of the incision, over the middle phalanx, is left open.[64]

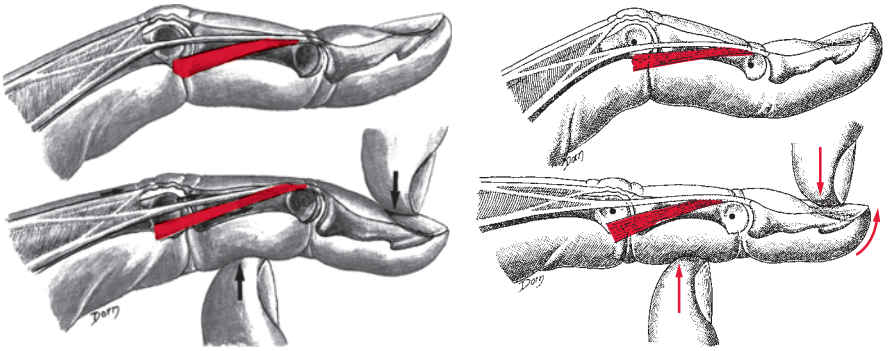


Figure 13.27

(a) Boutonnière deformity – stage 1. When the middle phalanx is maintained in extension, flexion of the distal phalanx is possible. Conservative treatment consists in splintage of the PIP joint in extension and active flexion exercises of the DIP joint. (b) Boutonnière deformity – Stage 2. When the middle phalanx is maintained in extension, flexion of the distal phalanx is not possible. The Haines–Zancolli test is positive. Terotomy of the lateral extensor tendons at the level of the middle phalanx will correct hyperextension of the DIP joint, and retraction of the proximal portion of the extensor apparatus will reduce PIP joint flexion if there is no bony adhesion.

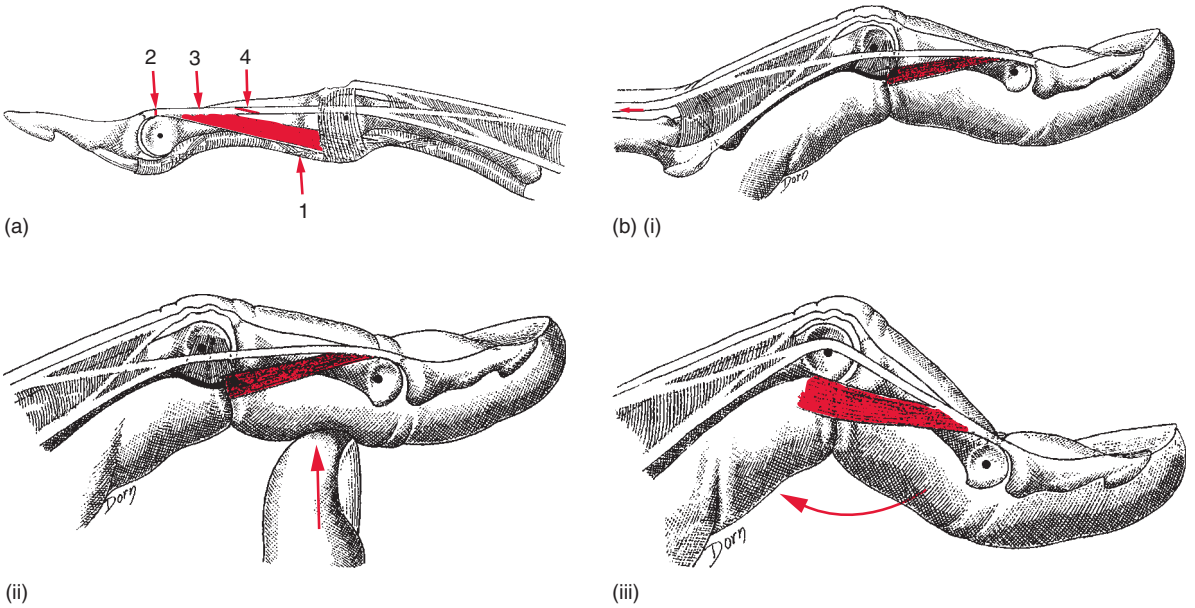


Figure 13.28

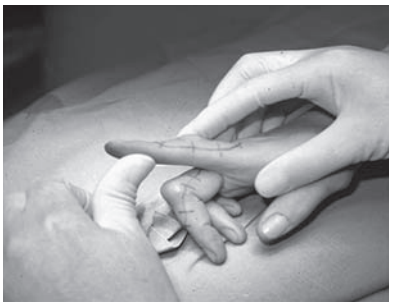
Different sites of tenotomy used for treatment of boutonnière deformity. (a) (1) Division of oblique retinacular ligament. (2) Tenotomy of terminal extensor tendon. (3) Tenotomy of terminal extensor tendon proximal to distal insertions of oblique retinacular ligament. (4) Tenotomy of lateral extensor tendons. (b) Boutonnière deformity – Stage 3. Both interphalangeal joints present limitation of motion caused by soft tissue contracture. (i) Proximal retraction of extensor apparatus causing hyperextension of MP joint. Contracture of oblique retinacular ligaments limiting flexion of distal phalanx. (ii) Contracture of PIP joint volar plate and accessory collateral ligaments blocking passive extension of joint. (iii) PIP joint flexion is still possible; the DIP joint remains in hyperextension, and the distal phalanx is excluded from pretension. In Stage 3, procedures aimed at rebalancing the extensor system should only be attempted if the interphalangeal joint contractures are passively corrected preoperatively. If, in spite of an adequate conservative program, complete passive correction is not obtained, or is only obtained by surgery, it is wise to renounce attempts to repair the extensor apparatus and to be content with a distal extensor tenotomy when useful PIP joint flexion is still possible. If the PIP joint is too stiff, and the flexion deformity important, the extensor tenotomy may be associated with PIP joint fusion in a function position.

The rheumatoid boutonnière deformity

Unlike the rheumatoid swan-neck, which can originate at different levels of the extensor mechanism, boutonnière deformity originates only at the PIP joint.

The functional loss caused by PIP joint flexion remains less important than the PIP joint stiffness in extension. For this reason, treatment should involve minimal risk. Paradoxically, it is the lack of DIP flexion that is, in most cases, more disturbing than the PIP flexion.

(a)



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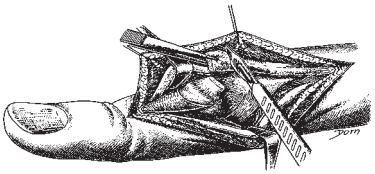

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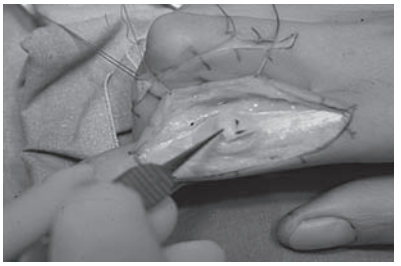

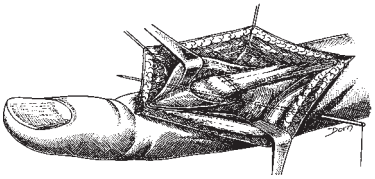

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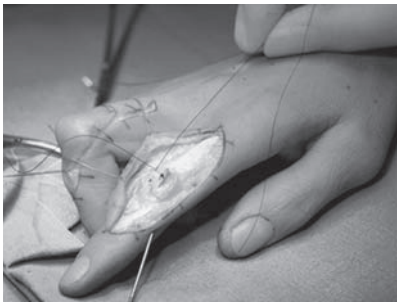

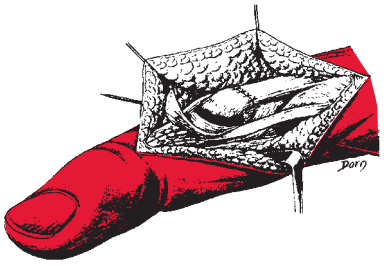

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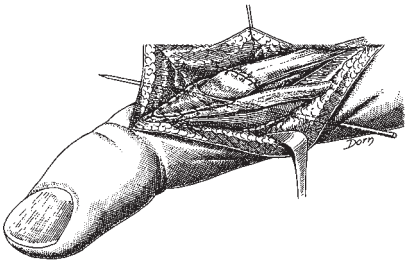

(f)

Figure 13.29

Boutonnière deformity: reconstruction of the extensor apparatus. (a, b) Complete extension of the PIP joint must be obtained before surgical reconstruction of the extensor apparatus. (c) Shortening of the central tendon. The central extensor tendon and the two lateral extensor tendons are located and freed, shortening of the central tendon for about 3 mm. The distal insertion of the tendon is preserved. (d) The PIP joint is temporaily held in extension by an oblique K-wire. The central tendon is sutured to itself and an oblique tenotomy of the lateral tendon is performed. (e) The distal phalanx is flexed. The lateral extensor tendons are sutured one to the other at their distal ends. The triangular lamina, when preserved is overlapped over them. (f) Results.

Boutonnière – Stage 1: no limitation of passive motion in any finger joint (Figure 13.27a)

Splinting in extension at the PIP level, combined with active flexion exercises of the DIP is indicated.

Boutonnière – Stage 2: complete passive mobility of the three finger joints

Although there is passive mobility, when the middle phalanx is maintained in extension, flexion of the distal phalanx is not possible, because of the contracture of the oblique retinacular ligament (tenodesis effect) (Figure 13.27b).

The surgical treatment theoretically consists of partial resection of the contracted oblique retinacular ligament to correct the distal joint hyperextension.[52]

A tenotomy of the lateral extensor tendons at the level of the middle phalanx seems more appropriate for restoration of the extensor mechanism balance (Figure 13.28a).

Boutonnière – Stage 3: Joint stiffness, with articular surfaces intact on X-rays

Soft tissue contracture limits PIP and DIP joint motion. Flexion of the PIP joint is still possible, but the lack of DIP flexion prevents complete rolling of the finger necessary for a normal grasp and hampers pinch grip (Figure 13.28b). A corrective splint on the PIP joint combined with active mobilization of the DIP joint is applied. As progress is made, the splint can be altered. In cases of persistent deformity, indications for surgery should be very cautious and candidates carefully selected. The flexion deficit of the distal phalanx can be more disabling to the grip than the extension deficit in the PIP, especially in the ulnar fingers. However, reconstruction of the extensor apparatus at the PIP joint is difficult, and surgery must not jeopardize flexor function in an attempt to gain extension. If complete passive extension of the PIP joint is obtained (Figure 13.29a,b), reconstruction of the extensor apparatus may be considered (Figure 13.29c–e). Both the extension of the PIP joint and the flexion defect of the DIP joint must be corrected by shortening the central extensor tendon and correction of the volar subluxation of the lateral extensor tendons.

If complete passive extension of the DIP joint is not obtained, tenotomy of the lateral extensor tendons is performed (Figure 13.30).

It is often wiser in cases of persistent deformity at the level of the two interphalangeal joints to just make a tenotomy of the lateral extensor tendons without correction of PIP flexion deformity.

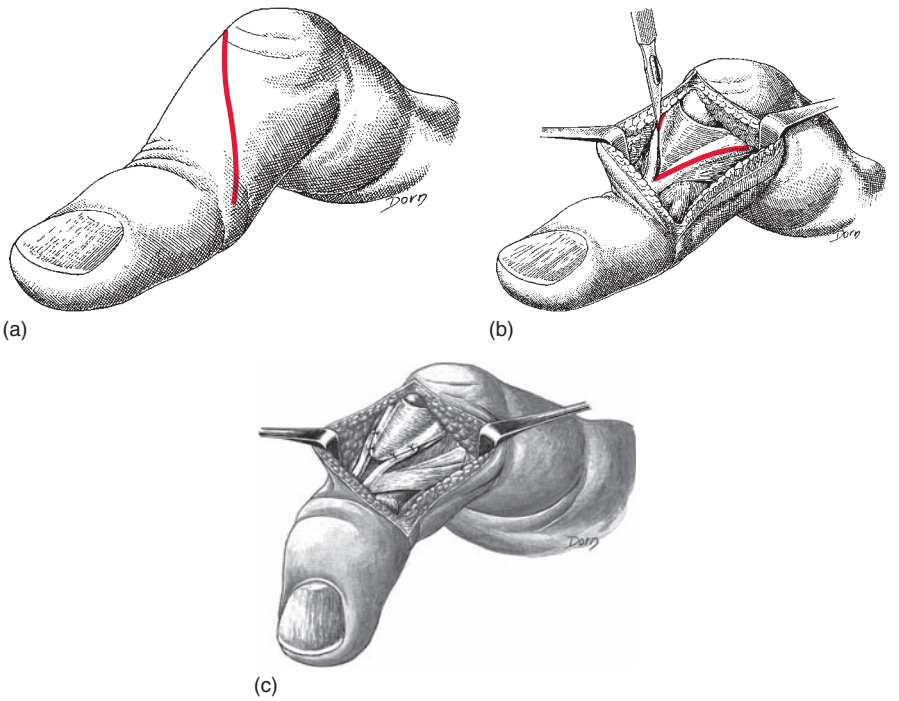

Figure 13.30

(a) Oblique tenotomy of the lateral extensor tendons. Incision on the dorsum of the middle phalanx. (b) The two lateral extensor tendons are divided obliquely. It is important to free the proximal portion of the extensor apparatus with a blunt spatula passed under the lateral tendons. It is often not necessary to divide the distal portion of the lateral border of the lateral extensor tendons into which the oblique retinacular ligament is inserted in order to obtain complete flexion of the distal phalanx. (c) The obliqueness of the division allows lengthening of the distal extensor apparatus without loss of contact between the ends of the divided tendon. A splint holds the PIP joint in as much extension as possible and the DIP joint in neutral position. Active mobilization of the distal phalanx is started after 1 week. A splint holds the IP joints in extension between exercises.

Boutonnière – Stage 4: Fixed deformities with lesions of the articular surfaces

Treatment in by PIP arthrodesis associated with extensor tenotomy. PIP arthroplasty is only possible when the extensor tendons are still usable.

At the DIP joint

Loss of distal phalanx extension constitutes a *mallet finger*. The rheumatoid mallet finger can also be classified in four stages:

- Stage 1: mallet deformity with no limitation of passive motion and absence of deformity at the PIP joint. Treatment is by synovectomy and splintage.
- Stage 2: Tenodesis effect. There is DIP joint flexion with PIP joint hyperextension. The best solution is a DIP joint arthrodesis beetwen 10° and 25° according to the finger involved. Littler's spiral oblique retinacular reconstruction can be another procedure for correction of the deformities of the two interphalangeal joints.
- Stages 3 and 4: Fixed deformity. Persistent symptomatic deformity may benefit from fusion of the distal joint.

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14 Biomechanics of the fingers

René Malek

The ulnar type fingers, of which there are four, share a similar constitution, although the fifth has a special hypothenar musculature. They have three phalanges and three joints (metacarpo-phalangeal (MP), proximal interphalangeal (PIP), distal interphalangeal (DIP)). They are called the 'long fingers'.

Anatomy

Bone and ligament structures

The *metacarpophalangeal joint* is a condylar-trochlear joint.

1. Joint extremities: the heads of the metacarpal bones are asymmetric and convex, flattened transversally, and with a bolster on the ulnar condyle, with the exception of the head of the fifth finger, which has a radial bolster. In the profile the condylar surfaces are lower than the inferior edge of the bone (Figure 14.1). Thus the radius of the movement of the first phalanx increases during flexion. However, the transverse axis of flexion remains the same, there is only a cam effect. The base of the first phalanx represents a glenoid excavation, whose surface is only a third of the articular surface of the metacarpal head. This would explain the existence of a fibrocartilage which extends the surface of the phalanx anteriorly. This fibrocartilage remains mobile to permit the joint extreme flexion, when it slides under the metacarpal head owing to a deep synovial cul-de-sac.
2. Capsule and ligaments: the capsule is somewhat loose. The lateral ligaments have three parts (Figure 14.2):
   - The metacarpophalangeal part, which extends from the metacarpal head and is inserted into the lateral tubercule of the base of the first phalanx. The radial ligament is the most resistant. These ligaments are oblique regarding the longitudinal metacarpal axis because their proximal insertion is very dorsal and their distal insertion is rather volar because the lateral tubercules in which they are inserted are in a more palmar position. Moreover, this obliquity is different for each one (the radial ligament is more oblique) and this causes an obligatory pronation of the first phalanx during flexion and a supination during extension.[1]
   - The metacarpoglenoidal part whose fibers are situated deeper and are more horizontal (vertical regarding the longitudinal axis of the metacarpal bone) and which has some insertions in the intermetacarpal (or interglenoidal) ligaments and the flexors' sheath.

These two parts of the ligament have a different tension, according to the flexion–extension of the joint (as noted), taking into account the shape of the metacarpal head and the resulting cam effect. This anatomic

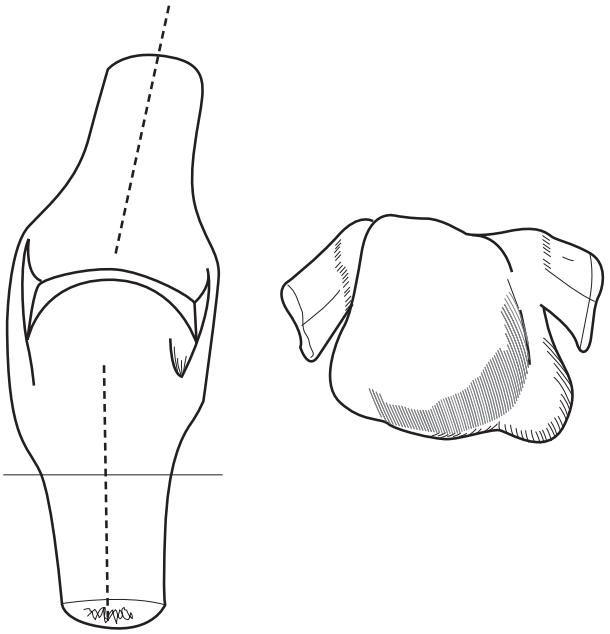

Figure 14.1

The heads of the metacarpal are asymmetric and convex with a bolster on the ulnar condyle.

*with V Gasiunas for the translation in English

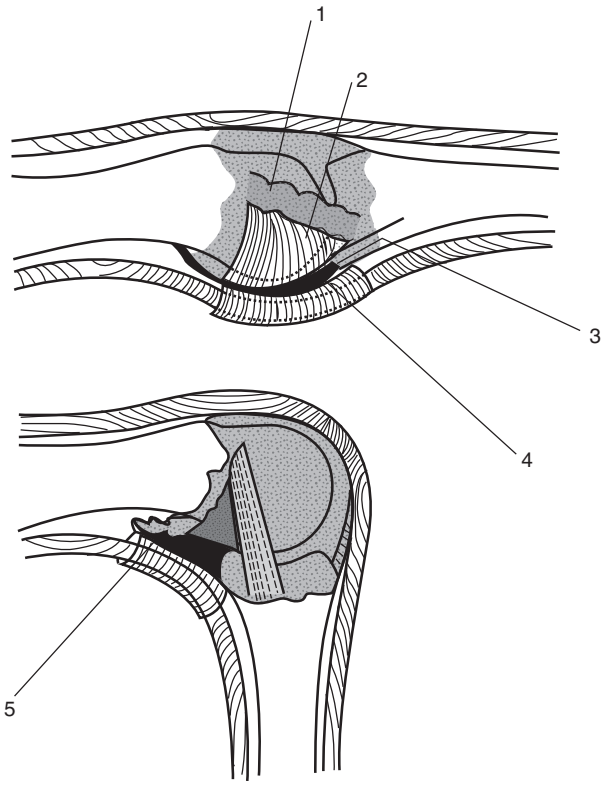

Figure 14.2

The collateral ligament of the metacarpo-phalangeal joint. 1) head of metacarpal bone; 2) base of first phalanx; 3) collateral ligament; 4) accessory part (glenoidal) of the ligament; 5) volar plate. The collateral ligament is tight in flexion and relaxed in extension. Adapted from Tubiana et al.[2]

fact is important and explains the variabiity of tension at the level of the lateral ligaments, which are tight in flexion and slack in extension, allowing the possibility of lateral movements of the metacarpophalangeal joint.

- The third part of the fibers reinforces phalangeal insertion of the fibrocartilage.

The *proximal interphalangeal joint* is also a condylotrochlear joint, which, in this case, does not allow movements other than flexion and extension.

1. Articular extremities: the distal part of the first phalanx presents two condyles of almost perfect spherical aspect (in contrast to the metacarpal head). Thus there is no cam effect in flexion. The trochlea at the base of the second phalanx has two glenoid cavities which are separated by a blunt crest, corresponding to the slight intercondylar groove. There is also a glenoid fibrocartilage which increases the articular surface.
2. Ligaments: There are two lateral ligaments, radial and ulnar, divided into two parts, which tie the phalangeal head to the base of the second phalanx and to a glenoid fibrocartilage. These lateral ligaments are stretched to

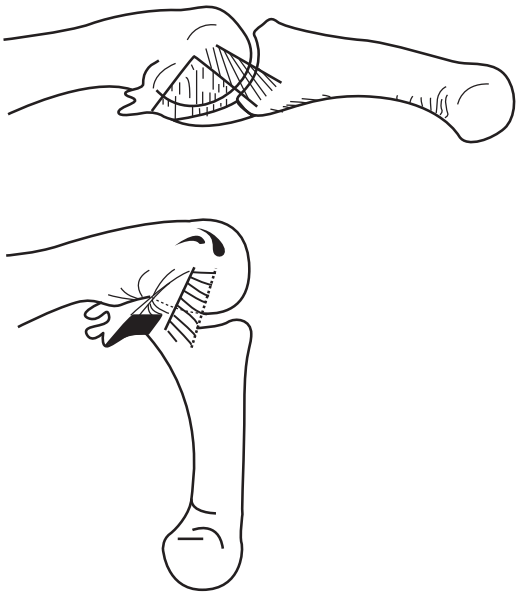

Figure 14.3

In the PIP joint tension of the collateral ligaments remains the same in flexion and extension.

the same degree during flexion and extension, and there is no possibility of lateral movement in any degree of flexion (Figure 14.3).

The *distal interphalangeal joint* is constructed according to the same model, described above. Only flexion and extension are possible.

The joints' ligamentous structures are tightly maintained by the *tendinous and fibrous structures*, which will be described later.

The joints of the fingers are mainly used in flexion, which is the necessary position to handle objects. It can be considered that two tendinous planes exist, that one palmar and one dorsal, that remain in contact with the skeleton and correspond to the flexor and extensor tendons. In addition, a number of tendinous and ligamentous structures located on the sides of the fingers give solidity to these two planes and make them work in a simultaneous and a synergistic manner. This complexity emphasizes that the biomechanics of the fingers must be studied as a whole, not divided into flexion and extension, because all those structures are connected together and depend on each other. Thus an anatomic description will be given before the global functional study.

Fibrous and tendinous structures

Flexor tendons and their sheaths: the palmar plane

Every finger has two tendons which correspond to the flexor superficialis and flexor profundus muscles. These are extrinsic muscles because their bellies are situated in the forearm, and this emphasizes the functional unity formed by the hand and the forearm.

The *profundus tendon*, which gives insertion to the lumbricales muscles (see later), ends at the base of the distal phalanx (Figure 14.4).

The *superficialis tendon* ends on the second phalanx. Hence the tendons must cross, and there is a perforating and a perforated tendon. The latter is the superficialis tendon, which is divided at the MP level into two strips which pass around the profundus tendon to insert in the lateral aspect of the middle phalanx, having been tightly interlaced (chiasma tendinosum camperi) (Figure 14.5).

Flexor muscles have a mixed innervation: the superficialis flexor of the fingers is innervated by the median nerve. The two ulnar bellies of the flexor profundus (for the fourth and fifth fingers) are innervated by the ulnar nerve, while the two radial bellies are innervated by the median nerve.

The tendons are kept in contact with bone by fibrous inextensible sheaths, situated at the level of the metacarpal head, at the proximal and at the middle phalanx. This layout is necessary to avoid a tendon detachment from the bone plane in flexion forming a bowstring. To allow flexion, the fibrous sheaths are replaced by less resistant oblique and crossed fibers, in front of the joint flexion areas (Figure 14.6).

These fibrous sheaths form reflection pulleys for the tendons and must be broad enough to avoid an angulation, which might have an undesirable effect on the tendon sliding. The pulleys bear different loads up and down, which depend upon the liberty of motion of the hand (the existence or not of inertial forces to be overcome at the extremity of the finger). These forces must be taken into account in the movements of the finger joints. When the pulley is close to the joint, one of these forces has a component that tends to dislocate the bone, in general in the palmar direction. Usually this force is counterbalanced exclusively by the resistance of the restraining joint structures (ligaments). Only in pathologic eventuality (e.g., rheumatoid arthritis) may this force lead to a joint dislocation.

Finally, at the MP level, the aperture of the fibrous sheath is formed by the less resistant arciform fibers. Thus the proximal part under the pressure of the flexor tendons can be somewhat distended and this, at the same time, not only permits the joint flexion but also, by displacing the entrance

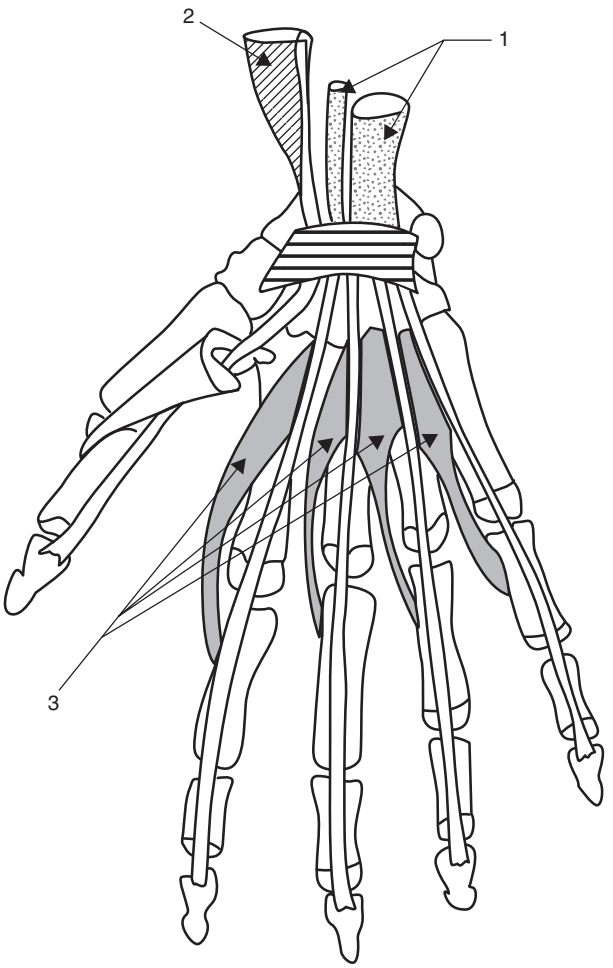

Figure 14.4

The flexor profundus muscle and the lumbricales.
1) flexor digitorum; 2) long flexor of the thumb; 3) lumbricales.

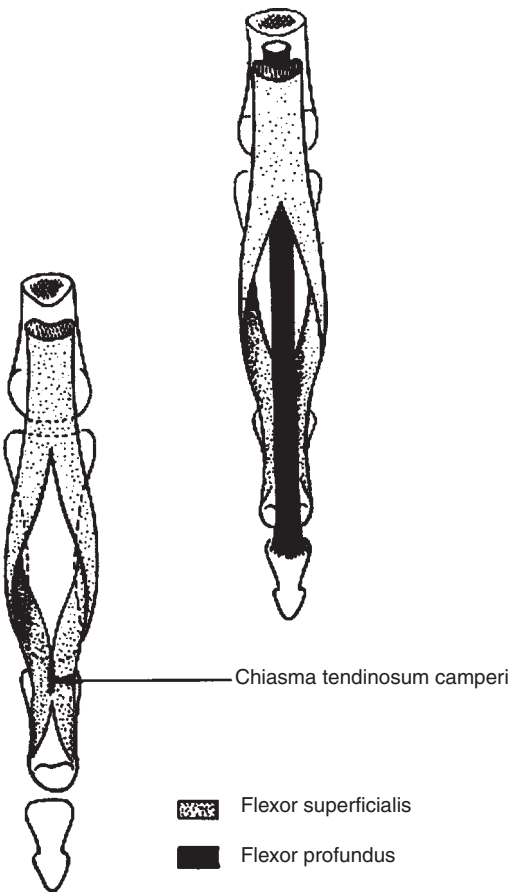

Figure 14.5

Description of the distal insertions of the flexor superficialis.

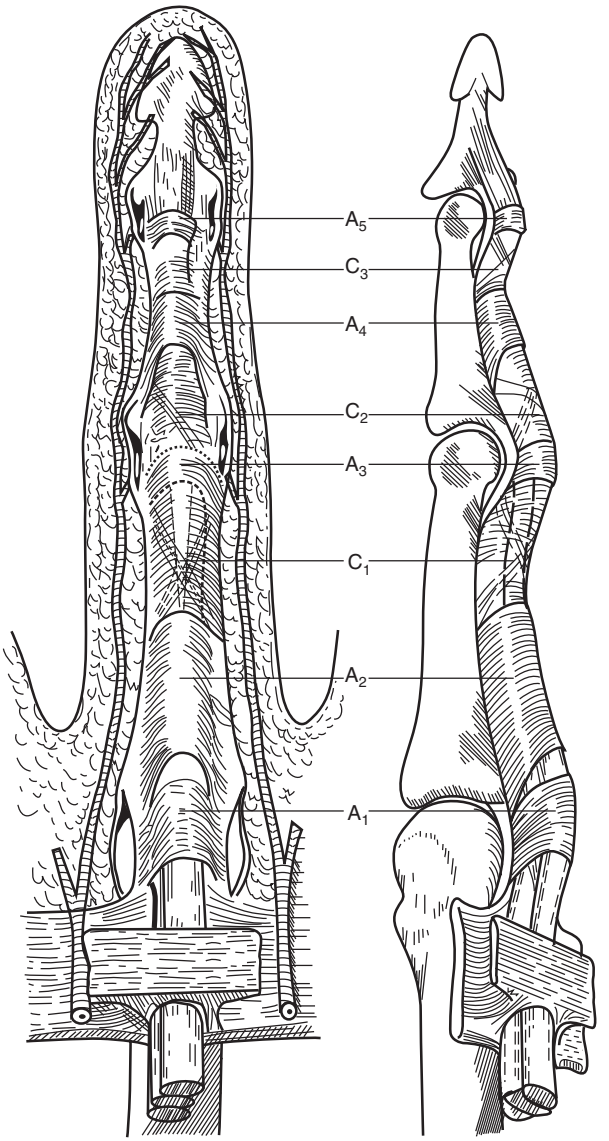

Figure 14.6

Disposition of the flexor tendons in the digits. The digital fibrous sheath of the flexor tendons, formed by five tight-annular pulleys (A1, A2, A3, A4 and A5) and three segments with loose cruciform fibers (C1, C2 and C3).

of the pulley downwards, accentuates the flexion effect of the tendon.

The flexor tendons are surrounded by a *synovial sheath* which facilitates their sliding in the osteofibrous canals built up between the phalanges and the fibrous sheaths. The synovial sheath of the fifth finger extends into the proximal part by the ulnocarpal sheath, which surrounds the flexor tendons in the palm and which continues in the carpal canal up to the wrist (Figure 14.7).

Every sheath is formed of two layers, one attached to a tendon and another attached to the deep surface of the canal. Between them there is a small quantity of a synovial type fluid.

The sliding is effected by 'rolling' of the two layers, forming a cul-de-sac at the extremities of the sheaths, passing beyond the border of the fibrous sheath proximally (Figure 14.8).

In the prearticular zones, between the thick fibrous sheaths, the tendons are nourished by deep vessels forming true vascular mesos inside the synovial sheaths. Persistent avascular zones remain in between, with which the surgeon should be familiar when he performs sutures and tendon grafting.

Extensor tendons: dorsal plane

These also come from extrinsic muscles. Their endings are quite complex: the extensor proprius of the index finger and the extensor proprius of the fifth finger have their distal insertions in common with the extensor digitorum communis tendon. The extensor tendon is broad and flattened. First it gives off a small tendon strip at the finger base, which first sticks to the inferior part of the MP joint's capsule and then inserts into the base of the first phalanx. This ending is not constant (half of the cases for Kaplan).[3]

At the dorsal aspect of the proximal phalanx the tendon divides into three strips:

- one median, the most solid of which inserts into the base of the middle phalanx
- and two lateral, which surround the first one, join each other at the distal half of the middle phalanx and insert into the base of the distal phalanx (Figure 14.9).

The two form a kind of skeleton of the dorsal apparatus of the finger reached by many ligamentous structures.

The extensor muscles, including the extensor proprius of the index finger and the extensor proprius of the fifth finger, are innervated by the radial nerve.

Lateral elements: intrinsic muscles and ligaments

These elements of the hand are purely intrinsic and have the particularity to cross the sides of the fingers, narrowly joining the palmar and dorsal planes.

The muscles These are represented by the palmar and dorsal interossei, and the lumbrical muscles, which have a biomechanics that is not easy to elucidate.

1. The *palmar interossei muscles* (there are three of them) (Figure 14.10) arise from the anterior half of the lateral aspect of the metacarpal bone, which is the most distant from the hand axis, in the intermetacarpal space. It ends on the finger which extends this metacarpal bone. The tendon of palmar interosseus is anterior or volar with respect to the transverse intermetacarpal ligament. It divides into a triangular sheet presenting two sorts of fibers:
   - the proximal fibers pass transversely to join their homologs from the opposite side and to form a

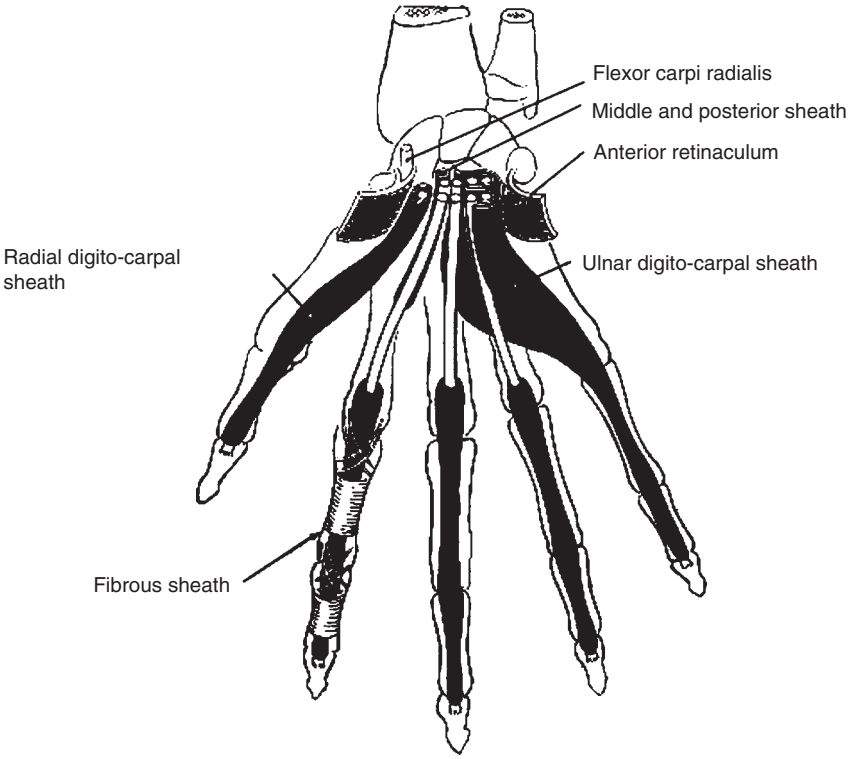

Figure 14.7

The synovial sheaths of the flexor tendons.

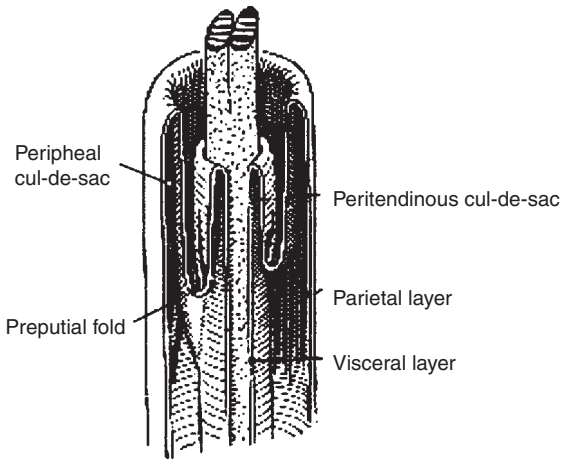

Figure 14.8

Superior extremity of the synovial sheath of the digit allowing the sliding of the tendons.

fibrous arcuated structure: the interosseous horn; this expansion is triangular, and

- its distal fibers, which are oblique, almost longitudinal, form a strip which joins the lateral strip of the extensor tendon (already described) and with it constitutes the lateral tendon which inserts into the base of the distal phalanx (see Figure 14.9). The majority of authors (excluding Kaplan[3]) consider that there is no insertion of the palmar interossei into the lateral tubercle of the base of the proximal phalanx. Some oblique fibers join each other from the opposite side at the level of the proximal interphalangeal (PIP) joint to form a triangular sheet. The existence of the first palmar interosseus in the first intermetacarpal space is controversial: its fibers would be part of the flexor pollicis brevis and it is why there are only three palmar interossei described. Furthermore, the middle ray does not have any of them.

2. The *dorsal interossei muscles* (there are four of them – one for every intermetacarpal space) (Figure 14.11) arise from the lateral aspect of the metacarpal bone, situated close to the axis of the hand, and from the dorsal half of the metacarpal, which, as has been seen, gives the origin of the palmar interossei. Then, the third metacarpal gives the origin of the dorsal interossei of the second and third intermetacarpal spaces. Every one of them ends by a double tendon (some authors think that the belly is also divided, as two distinct motor nerve branches exist). One of the tendons inserts into the lateral tubercle of the base of the first phalanx of the digit, which has given the largest insertion. The second continues within a triangular sheet and ends in the same manner as the tendon of the palmar interosseus: its dorsal fibers enter into the dorsal expansion of the interossei, and its palmar oblique fibers form a strip which joins the strip of the extensor tendon to form the lateral tendon (see Figure 14.9). These tendinous structures are applied against the lateral aspect of the finger and at the level of the MP joint are located behind the transverse intermetacarpal ligament. The palmar and dorsal interossei muscles are innervated by the deep branch of the ulnar nerve.

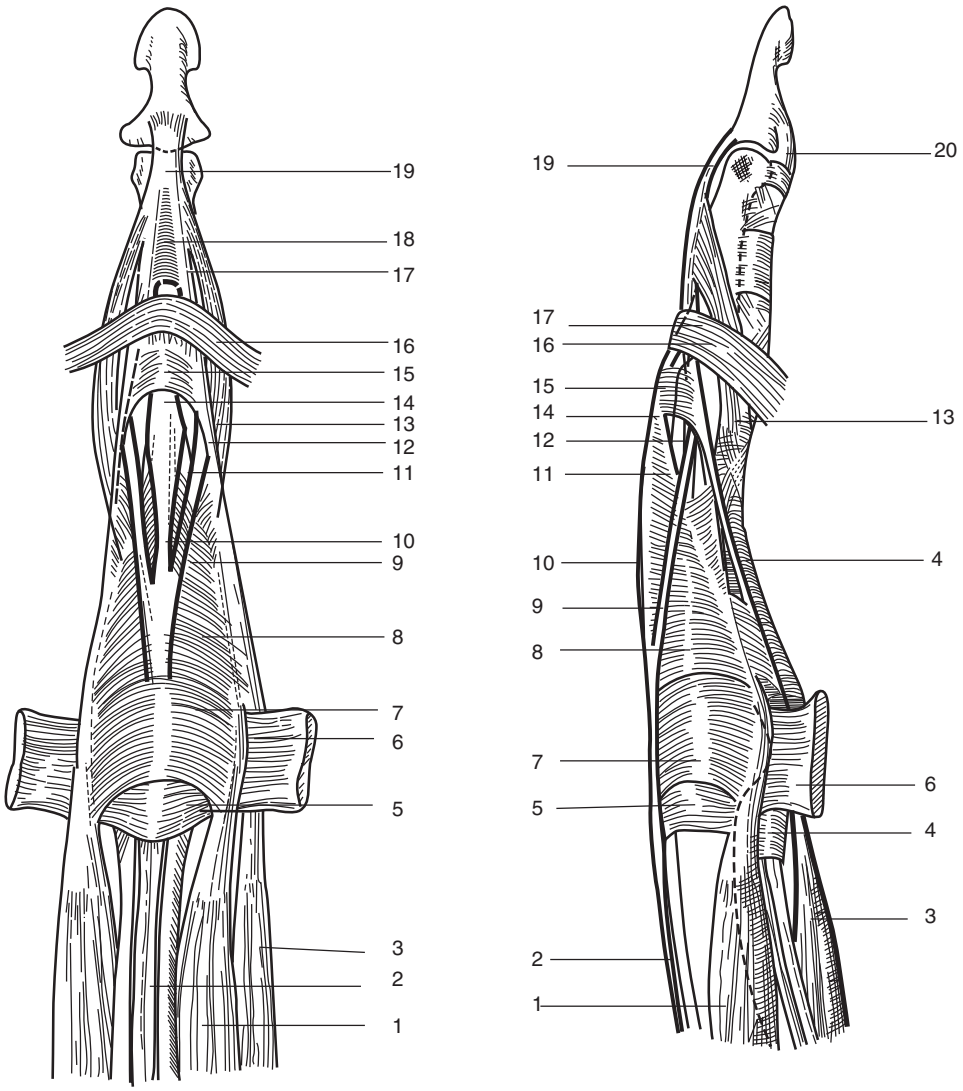

Figure 14.9

Anatomy of the upper extremity of the synovial sheath facilitating the sliding of the tendons. Extensor apparatus of the fingers: (1) dorsal interosseous; (2) extensor communis tendon; (3) lumbrical; (4) flexor tendon sheath; (5) sagittal band; (6) transverse metacarpal ligament; (7) extensor hood (transverse fibers); (8) extensor hood (oblique fibers); (9) lateral band; (10) central extensor tendon; (11) interosseous middle band; (12) lateral extensor tendon; (13) oblique retinacular ligament; (14) central extensor tendon; (15) spiral fibers; (16) transverse retinacular ligament; (17) lateral extensor tendon; (18) triangular ligament; (19) distal extensor tendon; (20) profundus tendon. From Tubiana et al.[2]

3. *The lumbrical muscles* (Figure 14.12) are small muscles extended between the tendons of the flexor profoundus and the dorsal extensor tendons, thus they have no direct osseous insertion.
   - In the first and the second intermetacarpal space they arise in the palm from the radial side of the tendons, 2 or 3 cm distally regarding the carpal canal (they come into the latter when the flexors contract).
   - In the third and the fourth intermetacarpal space they have a feather form, arising from the two neighboring flexor digitorum profundus tendons under the same conditions.

   The lumbrical muscles end with a flattened strip which joins the free border of the extensor tendon's radial side without taking part in the construction of the dorsal fibrous expansion of the interossei (Figure 14.13). These muscles are often the subject of individual variations. The first and second lumbrical muscles are innervated by the median nerve, the third and the fourth by the ulnar nerve.

Fibrous and aponeurotic elements In addition to the tendinous extremities, fibrous formations exist which have proved their physiologic importance.

1. The *sagittal fibers* that some authors describe, like one of the extensor tendon endings, arise from the tendon proximally with respect to the dorsal aspect of the MP joint and obliquely, inferiorly and anteriorly plunge into the palmar transverse intermetacarpal ligament,

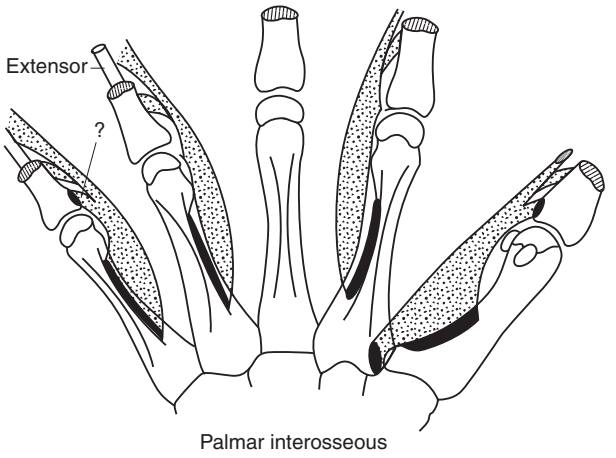

Figure 14.10

The division into three strips which end the extensor tendon.

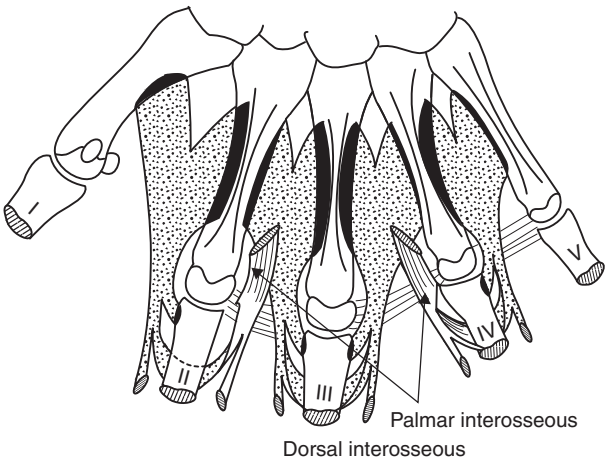

Figure 14.11

Palmar interossei.

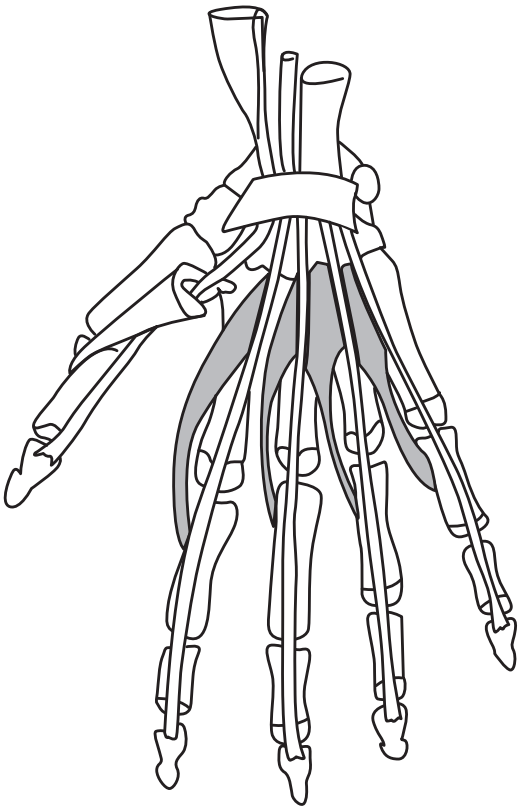

Figure 14.12

The lumbricales muscles.

and, more precisely, into the borders of the fibrocartilage. The sagittal fibers probably take part in extension of the first phalanx, as a relationship exists between them by means of the fibrocartilage – they form a kind of ring or a 'lasso', which elevates the base of the phalanx when this is in the initial position of extension. In other words, it gives a hyperextension of the MP joint. Because of this system, the absence of the extensor tendon's osseous insertion into the first phalanx is totally compensated for. Moreover, it permits a flexion mobility of the dorsal aponeurosis, the position of the MP joint not being important. In the case of MP hyperextension (as can be seen in ulnar palsy) all the force of the extensor tendon is transmitted anteriorly by the means of these sagittal fibers, whilst the extensor loses its capacity to act upon the interphalangeal joints (Figure 14.14).

The sagittal fibers also play a role in maintaining the tendons over the metacarpal heads, preventing them from luxation into the intermetacarpal grooves. In their distal part, the sagittal fibers press the tendons of the dorsal interossei against the capsule of the MP joint. Thus, at the base of the finger exists a cylindrical, fibrous, tendinous, and ligamentous tube, which is flexible, rather like a pipe maintaining the tissues surrounding the finger.

Finally, the fibers become continuous with those of the dorsal transverse ligament and the juncturae tendinum or intertendinous anastomoses. These anastomoses, situated more proximally than the MP joints, insure that the tendons are firmly united during extension, and play a role in keeping the metacarpal bones in a row (Figure 14.15).

2. The *triangular sheet* is formed by some oblique fibers from interossei which come together at the posterior aspect of the second phalanx. It keeps the lateral strips from moving apart during flexion (Figure 14.9).
3. *The two retinacular ligaments* (Figure 14.13):
   - The *transverse retinacular ligament*, which is thin but resistant, arises from the palmar aspect of the PIP capsule and inserts to a lateral border of the extensor tendon, passing superficially regarding the origins of the Cleland's fascia (see below).
   - The *oblique retinacular ligament* is better known and has an aspect of a tendon. It is stretched from the flexor tendon sheath at the distal quarter of

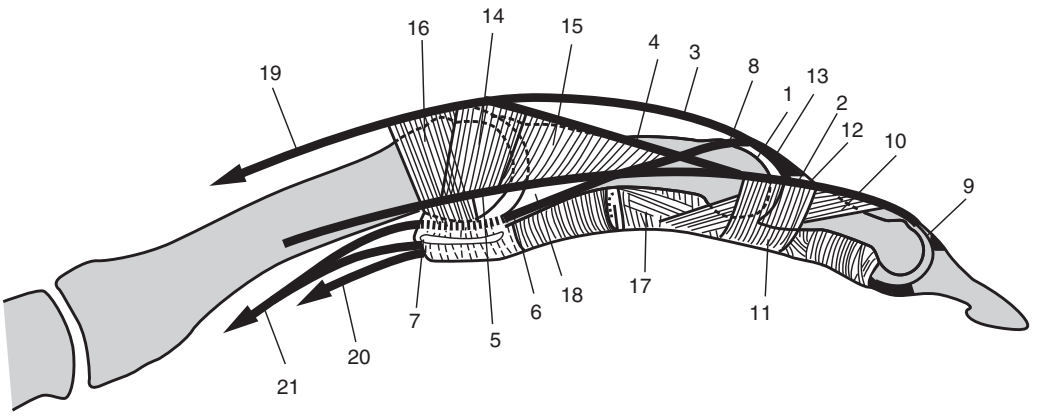

Figure 14.13

Diagrammatic view of the profile of a finger showing the insertions of the intrinsic and extrinsic muscles and the retinacular ligaments. There is a certain symmetry between the fibrous formations at the level of the metacarpophalangeal joint and the proximal interphalangeal joint. (1) Central or middle extensor tendon; (2) Lateral extensor tendon; (3) Central band of the long extensor; (4) Lateral band of the long extensor; (5) Interosseous tendon; (6) Lumbrical tendon; (7) Deep transverse intermetacarpal ligament (or interglenoid); (8) Central band of the interosseous; (9) Terminal extensor tendon; (10) Oblique retinacular ligament; (11) Transverse retinacular ligament; (12) Triangular ligament; (13) Insertion of the extensor digitorum into the second phalanx; (14) Transverse fibers of the interosseous hoods; (15) Oblique fibers of the interosseous hoods; (16) Sagittal bands; (17) Fibrous sheath of the flexor tendons; (18) Insertion of the interosseous on the base of P_1; (19) Tendon of the extensor digitorum; (20) Superficial flexor tendon; (21) Deep flexor tendon.

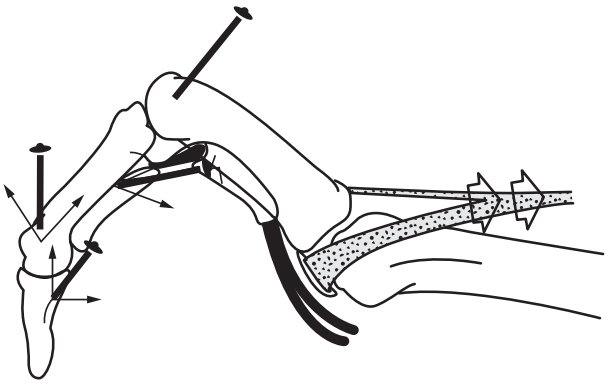

Figure 14.14

Predominant action of the sagittal fibers of the extensor when MP joint is hyperextended.

the first phalanx and joins the lateral strip of the extensor tendon, which inserts its fibers into the base of the third phalanx. It is deeper than the transverse retinacular ligament, so it is an element which makes a kind of tenodesis between P2 and P3, being anterior with respect to the axis of the PIP flexion, and posterior with respect to the axis of the DIP flexion. Because of this, one can speak of an active tenodesis when a joint's flexion leads ipso facto to a flexion of the other one, and on the contrary during extension.

4. *Cleland's ligament* (Figure 14.16): on each side of the PIP joint there are ligamentous fibers which bind the skin to the skeleton of the finger. These fibers are divided into two divergent cone-shaped fascicles, the anterior one going towards the extremity of the finger, the other one going upwards. In the angle of their intersection as they insert into the skeleton at the base of the second phalanx, the transverse retinacular ligament is tightly clasped.
5. *Grayson's ligament* (Figure 14.17), which is more palmar than Cleland's ligament, arises from the flexor tendons' sheath and extends transversely up to the skin of the finger. Whilst Cleland's ligament is dorsal with respect to the neurovascular bundle, Grayson's ligament is palmar; this arrangement has the ability to immobilize and protect the bundle during flexion and extension.

The cylindrical characteristics of the finger are practically permanent. To understand the biomechanics of the finger, consider that the finger, especially its lateral and dorsal parts, is not only where the 'tendons slide'. The tendons are maintained within the cylinder of the finger by a firm layer of skin, to form a uniform structure which undergoes the mechanical forces of variable intensity and direction. These forces direct the joints' movement.

Dorsal aponeurosis of the finger (Figure 14.18) plays a crucial role in all movements. At the level of the digit, the confluence between the common extensor tendons, the endings of the lumbrical and interosseous muscles, the transverse and oblique retinacular ligaments, and the triangular sheet is called the dorsal aponeurosis. This is a triangular formation, concave anteriorly, with an inferior summit inserted into the distal phalanx. This is its fixed point (shown by the star in Figure 14.18). Its superior border is represented by the arch-shaped edge of the fibrous expansion of the interossei. Its lateral borders are oblique, going behind distally. They are tied (in the mobile manner) at their proximal part to the intermetacarpal

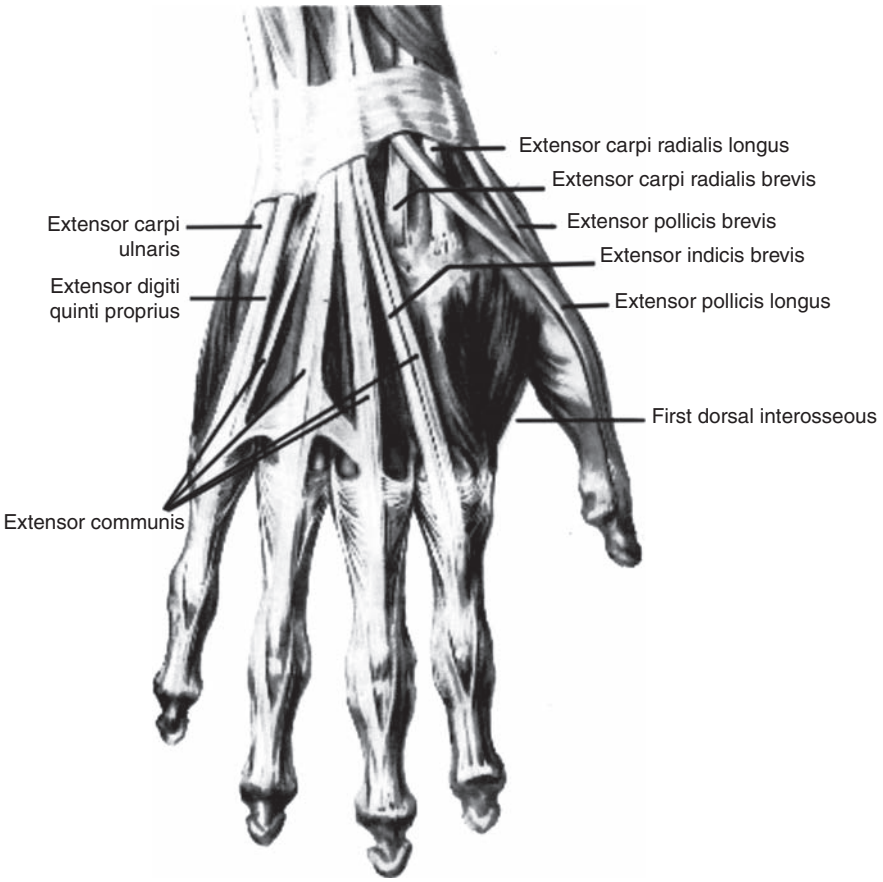

Figure 14.15

General disposition of the tendons at the dorsum of the hand.

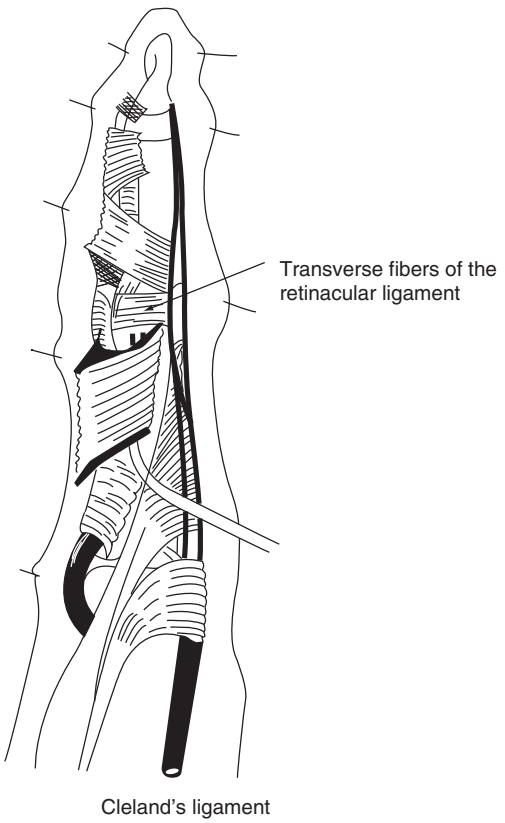

Figure 14.16

Cleland's ligament.

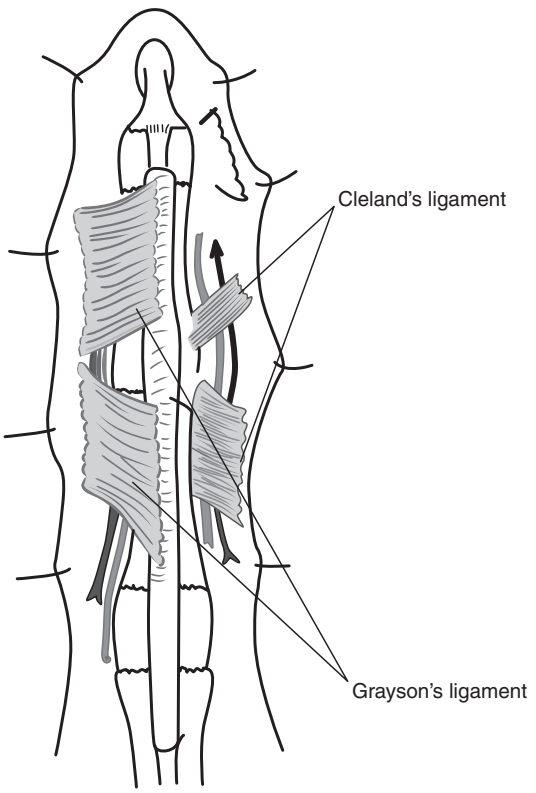

Figure 14.17

Grayson's ligament.

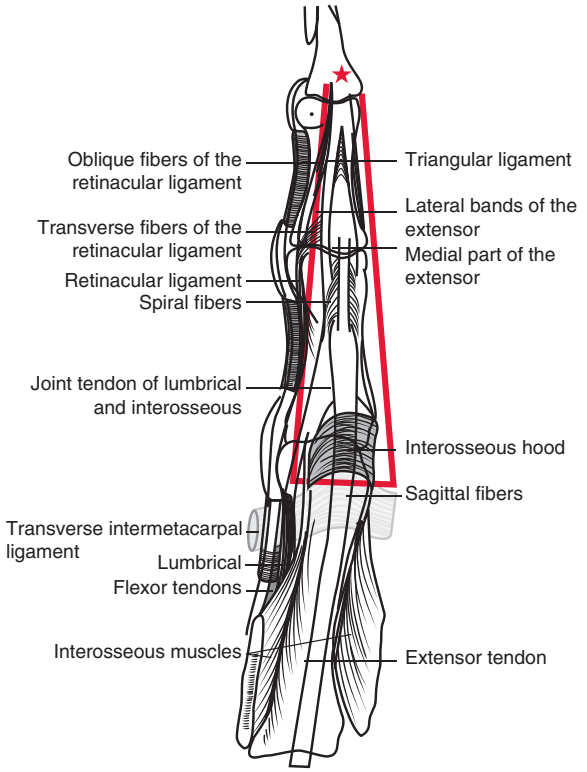

Figure 14.18

The dorsal aponeurosis of the digit, considered as an anatomic entity.

space by the tendons of the intrinsic muscles (so-called wing tendons), and more distally to the palmar aspect of the finger by the retinacular ligaments. It seems that this aponeurosis is inextensible in the two planes, vertical and transverse. However, its tendinous framework structure allows it to receive and transmit orientated mechanical forces generated by muscles, and this only by an adaptation of its resistance in a variable fashion. Above all, its sliding all in one piece regarding the skeleton intervene in the joint play. It is only tied to the underlying skeleton (not taking into account the distal insertion into the distal phalanx) by the insertion into the second phalanx of the median strip of the common extensor. Finally, it may be that the division into three tendons (which are reinforced zones of the dorsal aponeurosis) allows a greater possibility for (controlled) flexion of the PIP joint (like a reed being bent).

The review of this quite complex anatomy permits a more comprehensive study of the biomechanics of the fingers in the following section.

Biomechanics

Three basic notions enable us to understand the fingers' biomechanics. They are:

1. the *skirt effect*, which is explained by the sliding of the dorsal aponeurosis
2. then, the particular distribution of the forces generated by the muscles in the multiarticular system with an *intercalary bone* in between, taking into account proximal and distal muscular insertions
3. finally the *preferential transmission* of forces created by muscles regarding the relative orientation of their bellies, or, their fibers and the tendinous fibers which they extend (fan-shaped tendon).

1. The 'skirt effect' – everyone knows that a skirt which is below knee level in the posterior view goes up several centimeters when the hips are flexed. This is what happens at every joint of the finger (especially at the MP joint), because the dorsal aponeurosis is fixed at the extremity of the finger (Figure 14.19). Mathematically, the sliding observed at its superior border is equal to the sum of arcs run over on the surface of each phalangeal extremity as if these were a part of a sphere. The length of the stars depends on the angle of flexion. When this angle becomes close to 60° (more precisely, close to the radian value, which is 57.29°) the distance of the shift on the dorsal side is equal to the ray of the sphere.[4] Moreover, as already mentioned, at the level of the MP joint there is a cam effect. Then the value of the ray of the metacarpal's head is variable and increases anteriorly. So it is clear that the more the finger is flexed, the greater is the sliding of the aponeurosis (Figure 14.19).

   In addition to the value of the proximal arc corresponding to the sum of arcs of the different joints involved in the movement must be added the difference between the extreme radii of the metacarpal head. This difference might reach 7 mm.[5] For example, when the middle finger is flexed at all its joints at 90°, the sliding of the aponeurosis, measured at the upper border of the fibrous expansion of the interossei, is equal to about 18 mm. With such a value of sliding, one can understand that the insertion of the extensor in the base of the proximal phalanx (when it exists) is completely relaxed, whatever the position of the wrist may be.
2. The second important notion is that the osseous architecture of the finger is multiarticular. The flexor and extensor tendons act like the reins inserted at the corresponding surface of the phalanx, whether the middle or the distal phalanx is involved. The flexor and extensor muscles act simultaneously (one carries out the motion, the other one is used to make it fluid or to slow it down). Whatever the case, these forces are applied on the intercalary bone, situated proximally.

   A study of the vectors of these biomechanical forces (according to the classic law of the parallelogram) reveals evidence of two types of forces:
   - The *active vectors*, perpendicular to the bone, going in the direction of flexion for the flexor tendons, and in the direction of extension for the extensor tendons. The predominance of one of these vectors creates a movement of part of the skeleton in one direction or another.

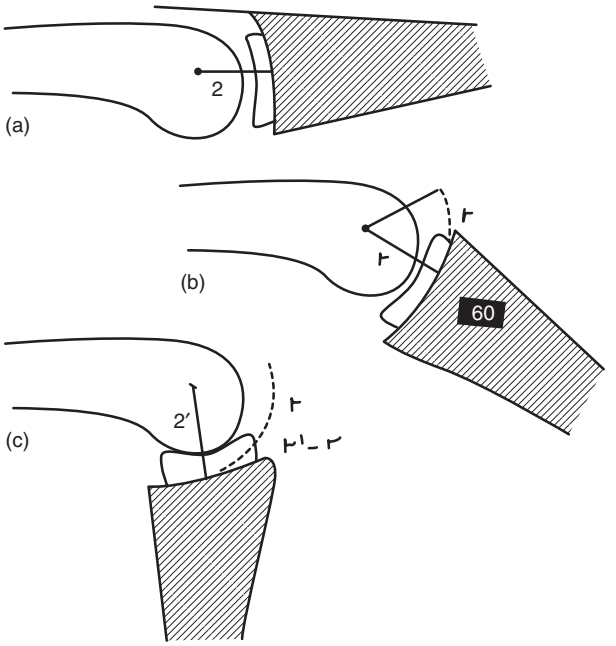

Figure 14.19

a) situation of the radius r measurement (between the center of the metacarpal head and the superior border of the dorsal aponeurosis).
b) when flexion is about 60° the radian of the arc is equal to the radius r.
c) The cam effect makes that the radius is greater for a flexion more than 60° and the sliding is greater.

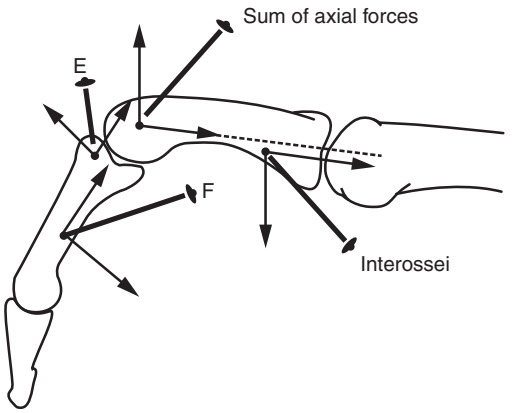

Figure 14.20

There are two mechanical forces applied on the second phalanx, generated by the extensor and the superficialis tendons. The parallelogram construction shows two axial forces which add up and two forces perpendicular to the bone which have an opposed direction. Their difference determines whether a flexion or extension movement appears. The resultant force of the axial forces is applied to the head of the first phalanx. The main role of the intrinsic muscles is to oppose it. E, extensor communis; F, flexor superficialis.

- On the other hand, this analysis shows the existence of other obligatory interfering forces – called *axial forces*, because they have a direction coinciding with the axis of the bone. One can conceive that the flexor and extensor axial forces are added to each other. They act on the head of the bone situated above and they are far from negligible, because the leading angle between the tendon and the bone is rather small. So, these axial forces are liable to extend the articulation situated more proximally. Thus, if we schematize these forces, starting at the extremity of the finger, i.e., distal phalanx, the resultant force (the sum of the two of them) will act on the head of the middle phalanx, which will be implicated in extension. If the latter is not desired, the axial force can be divided into a new axial force and a force of extension, which must be counterbalanced with an equal force. At this level (we are at the level of the middle phalanx) it is the flexor digitorum superficialis (FDS) which will exert the antagonistic force.

If we continue to analyze these forces by the parallelogram, towards the root of the finger, at the head of the proximal phalanx, which is an intermediary bone in the digital osteoarticular chain, the resultant sum of axial forces generated below induces a motion of the MP in extension. The flexors cannot oppose it, because they have no insertion into the proximal phalanx. Thus, to prevent extension intervention by another element of MP flexion is needed. This role is reserved for the intrinsic muscles, essentially the palmar and dorsal interossei, by means of their common fibrous expansion, and the dorsal interossei by the skeletal insertions on the phalanx (Figure 14.20). Paralysis of the intrinsic muscles causes the well-known claw deformity of the fingers with hyperextension of the MP, which will be described below.

3. The third important notion is the possibility of different actions of some of the hand muscles as they have several insertions distal to a joint. This is called the 'fan-shaped' tendon effect (Figure 14.21). The force, generated by a muscle whatever is its mode of action (isotonic or isometric contraction, or a slowing-down activity with elongation of the belly), and when there is no change of direction because of a pulley, is transmitted in a straight line and along the great axis of the muscle and its tendinous fibers. A simple example is given by the insertion of the dorsal interossei. When the MP joint is in complete extension the predominant force acts on the phalangeal tendon, so the muscle has the function of abductor of the finger into which it inserts (there is no action on the middle finger). When the MP joint is flexed, the force generated by interossei acts predominantly on the fibrous expansion of interossei, around the base of the proximal phalanx; this is possible because the sliding of the dorsal aponeurosis has begun. Thus, this force will contribute to the flexion of the MP joint, and this action will increase as flexion of the digit is wanted by the individual. Only in light flexion (or in the absence of full extension) can the force of the interossei be transmitted by means of

Figure 14.21

The fan-shaped tendon. (a) When the metacarpophalangeal joint is completely extended the main action of the interossei is abduction–adduction of the first phalanx owing to their osseous insertions. When flexion is beginning these muscles act as extensors of the two distal phalanxes. (b) When the metacarpophalangeal joint is flexed, the intrinsic muscles contribute to the flexion of the first phalanx and tend to fix it. F, flexion.

its lateral strip, that, together with the extensor's strip will form the lateral tendon, which on its own represents the border of the dorsal aponeurosis. This is the moment when the activity of the interossei will contribute either to the extension of the middle and distal phalanges, or to slowing down the flexion of these two phalanges.

When there is complete flexion of the MP joint and extension of the IP joints, the force of the interossei passing through only the fibrous expansion, in addition to the contribution of the MP flexion, immobilizes the dorsal aponeurosis. This explains the difficulty of flexing the IP joints (in conjunction whith the fact that the flexors are relaxed, a fact which may be compensated for by the wrist extension).

General considerations

In the light of these three essential notions it is possible to understand how the digital apparatus functions. It will then be possible to examine the physiologic consequences and therapeutic applications.

It has become clear in the sections above that it is not possible to study separately the fingers' flexion and extension, which are tightly related movements.

It must also be noted that the mechanical forces vary according to the movement – whether it is free (no contact with an object) or whether it is related to handling an object or manipulation.

There is some amplitude of hyperextension of the finger joints, variable by age and dependent on joint laxity, which is peculiar to any adult individual. It is in the range of 30° in the MP, 0° or 5–10° in the PIP joints, and 20° (passively) in the distal interphalangeal (DIP) joints. It should be remembered that the usual position of departure of the finger joints is in the direction of flexion.

The degree of flexion of the fingers is close to 90° in the MP, 100° in the PIP, and 80° in the DIP joints. In digito-palmar flexion, which brings the pulp into contact with the palm, flexion of 77% in the MP joint, 20% in the PIP joint, and 3% in the DIP joint takes place, according to Littler.[6]

The role of the wrist is fundamental in finger physiology. In fact, muscles which are called extrinsic have their bellies in the forearm and their tendons go along the dorsal and palmar sides of the wrist. The posterior aspect might be considered as a pulley which acts on the extensor tendons when the wrist is being flexed. The anterior aspect would also be considered as a pulley of the flexor tendons, but this time when the wrist is being extended and without the action of the palmar retinaculum. There is a 'skirt effect' in the extensors, as well as in the flexors, which will modify the tendons' length. When the wrist is flexed the extensors are stretched and this facilitates the opening of the hand. When the wrist is in extension the flexors are stretched, which facilitates the finger flexion.

The order or the rhythm of flexion and extension of the fingers (in 'free' motion) is very precise: during complete flexion it begins in the PIP joint, continues in the MP, and ends at the DIP joints (because of a slower rhythm of motion).

Regarding the question of what are the respective actions of the extrinsic and intrinsic muscles in the movements of the fingers, a dynamic electromyographic study by Long et al[7] has yielded some detailed biomechanical data. Nevertheless, the interpretation of the results must be made with some reservation. Long himself says that electromyography has no quantitative value – only a qualitative value which can determine whether the muscle which is being studied participates in the precise movement. Moreover, he reminds us that the three types of muscular activity – isotonic contraction with shortening of the muscular belly, isometric contraction without shortening, and, in contrast, contraction with lengthening – are related to the number of activated motor units in the muscular body. The activity leaves an electromyographic line only when the movement develops a certain force. Long notes the weakness of the electromyographic signals during muscular activity with lengthening. However, he did not say that the 'electric silence' he mentioned corresponds to a complete inactivity. Along the same lines, Long points out that the role of 'viscoelasticity' in the physiology of the fingers should be reconsidered. Even if there are no electric signs, this state might correspond to an activity with lengthening, which may be very often found in the hand to stabilize the intercalated bone or to increase the fluidity of a slowing down motion in another muscle.

To summarize, as far as we are concerned, during the movement of a finger all the muscles of the hand, intrinsic as well as extrinsic, interossei and lumbricals, are implicated to various degrees and with some modulation.

During flexion the flexor digitorum communis (FDC), FDS, and extensor communis contract together at the beginning, while the direction of motion is flexion because the flexors prevail over extensors. The flexor profundis predominates during the 'free' movement without a counterbalance. It generates flexion of the three articulations by the simple play of fibrous pulleys and retinacular ligaments. The superficial flexor does not act substantially until there is a necessity to counterbalance an excessive force at the PIP joint – a force acting by the resistance of the object in the direction of the PIP extension, for example in the firmly closed grip. But we have already seen the role of the axial forces, which, if they are not overcome, will lead to a claw finger (MP hyperextension and flexion of the two IPs). These are the actions of intrinsic muscles, which will play a role in equilibration. First the interossei and lumbrical muscles will slow down the descent of the dorsal aponeurosis fixed to the finger's extremity. Their distal tendons in the beginning are situated in the direction of their bellies, and this permits the action on their terminal strips. Afterwards, when the finger is being flexed, only the interossei will exert an action on the flexion of the proximal phalanx by means of their fibrous expansion, and they will also act as a counterbalancing force against the axial forces (sum of the forces of the flexors and extensors). By means of their action on the fibrous expansion, and consequently on the dorsal aponeurosis, which is prevented from sliding distally, the interossei will permit greater or lesser flexion of the IP joints. This flexion is also modulated by the common extensor, which will restrict the flexion of these joints by its insertion in the middle phalanx and the insertion of its lateral strips in the distal phalanx.

The DIP flexion relaxes the retinacular ligament, which facilitates the flexion of the PIP. The flexion of the PIP and MP joints relaxes the lateral strips of the interossei; this action is essentially transmitted by the means of the fibrous expansion.

In conclusion, all the muscles participate in flexion. The particular role of the lumbrical muscles will be emphasized later.

During extension the role of the central tendon of the common extensor is interpreted in different ways. Contraction of this muscle necessarily exerts tension on the dorsal aponeurosis and thus on its first bone insertion (if we discard the insertion to the P1, which is not clear), which is localized at the base of the second phalanx. In the palmar aspect the superficial flexor tendon is also put under tension, but this time it is the extensor which predominates. That is, by the play of the axial forces (exerted on the head of the P1 backwards) of these two muscles, the extension of the MP joint could take place if the departure of the motion is flexion. This extension of the proximal phalanx must be controlled by the intrinsic muscles in the same manner as described in the flexion study.

Extension of the distal phalanx is obtained by tension on the retinacular ligament at the PIP level, and by tension on the lateral tendons by stretching the lateral strips of the common extensor. The extension of the interphalangeal joints is also reinforced by the action of interossei and lumbrical muscles as their tendons extend their bellies.

So what is the exact role of the lumbrical muscles in this rather complex physiology? Their proximal insertions 'navigate' with the deep flexors' tendons. Thus, they are not fixed, contrary to their distal insertions, which share the dorsal aponeurosis (lateral extensor's tendon) insertion.

In the situation that we have just mentioned (flexion of the MP and the extension movement of the IP joints), the flexors are relaxed and the contraction of the lumbrical muscles draws those tendons towards the extremity, facilitating the extension. Whether the lumbrical muscles have any specific action on the extension of the IP joints is controversial, because their terminal fibers become bent as the MP joint flexes. The fibers of the extensor will extend the distal phalanx in relation to the middle phalanx as they pull the dorsal aponeurosis. The lumbrical muscles will act on the distal joints only when their fibers are less bent in relation to their global axis.

It has been said that, in the flexion position, the distal insertion of lumbrical muscles follows the dorsal aponeurosis. However, their length is modified or relaxed very little. On the contrary, their proximal insertion follows the movements of the flexor profundis tendons which have a tendency to elongate the muscular bellies, of the lumbricales. Long says that they do not enter into action, at least in the 'free' movement. In fact, as was shown by Rabischong,[8] these little muscles are very well supplied by proprioreceptive elements, i.e., the neuromuscular spindles and the Golgi complexes. They are probably the origin of myotactic reflexes and, thus, act as servomotors: the myotactic reflex, which has the neuromuscular spindle as a departure point, leads to the action of its own muscular fibers, which should react to passive stretching by the contraction of its motor units. Yet the lumbrical muscle may be stretched very little. In this state of affairs it cannot be a slowing-down element of flexion, because it is a small muscle without any power. Nevertheless, it may play an important role in somehow 'smoothing' the flexor tendon's displacement. This is a 'damper' in comparison with a servomechanism.

As far as Golgi complexes of the lumbrical muscles are concerned, they have another role because they measure the force exerted on the tendons by the motor units (of the lumbrical muscles). These two types of proprioreceptive elements may regulate the flexor's contraction by handling information and because they are the departure point of greater inhibition of the motor neurons.

The anomalies of the lumbrical muscles do not seem to influence this interpretation of their physiology.

During the adduction and abduction movements of the fingers, the role of the interossei muscles is well known. The insertion into the first phalanx far from the axis of the hand enables the dorsal interossei to abduct the fingers. There is a priori no action on the third finger. It probably depends on an interosseus muscle on one side which might be more activated.

The action of the palmar interossei muscles (which have no bone insertion, and which insert into the most distant

point of the dorsal aponeurosis of the finger in relation to the axis of their intermetacarpal space) is the adduction of the fingers. There are a certain number of anatomic variations concerning the bone insertion of the palmar interossei, but, as for the lumbrical muscles, there is no modification in this physiology. Abduction and adduction activity is easy to understand. It has the maximal force when the MP joint is in full extension. In this position the collateral ligaments are relaxed, which permits the movement.

The first dorsal interosseus muscle is very powerful because it has to oppose the pressure of the thumb in the thumb–index pinch. In the position of extension, the interossei muscles act in a rotational direction, which allows good adaptation of the fingers to a handled object. Abduction or adduction of the fingers (extended or not) is very difficult when the MP joints are flexed. There are some articular reasons (e.g., loss of laxity because of tension on the collateral ligaments) and muscular reasons (because the osseous insertions of the intrinsic muscles are brought closer, and because their principal force is mainly transmitted to the fibrous expansion).

The role of the extrinsic tendons in lateral movement has yet to be elucidated. The extensor tendons converge in the direction of the wrist and they are held rather firmly at the dorsal aspect of the MP joints, so they have an adductor action on the fingers at this level. This action is counterbalanced by the resistance of ligamentous structures and the interossei muscles. One can conceive the pathologic consequences.

The flexors have the same action, but this time it is the lumbrical muscles which counterbalance the effect of adduction.

Pathological deformities of the fingers

Mallet finger

This is the simplest lesion of the extensor apparatus and it consists of the loss of the insertion of the distal dorsal aponeurosis. It occurs in the case of traumatic rupture, or by pathologic distention connected with rheumatoid lesions of the dorsal synovial tissue of the DIP joint.

No force is applied in the direction of extension, so the distal phalanx is drawn by the profound flexor tendon into flexion. The dorsal aponeurosis slides in the proximal direction, leading to tension on the middle tendon. The PIP

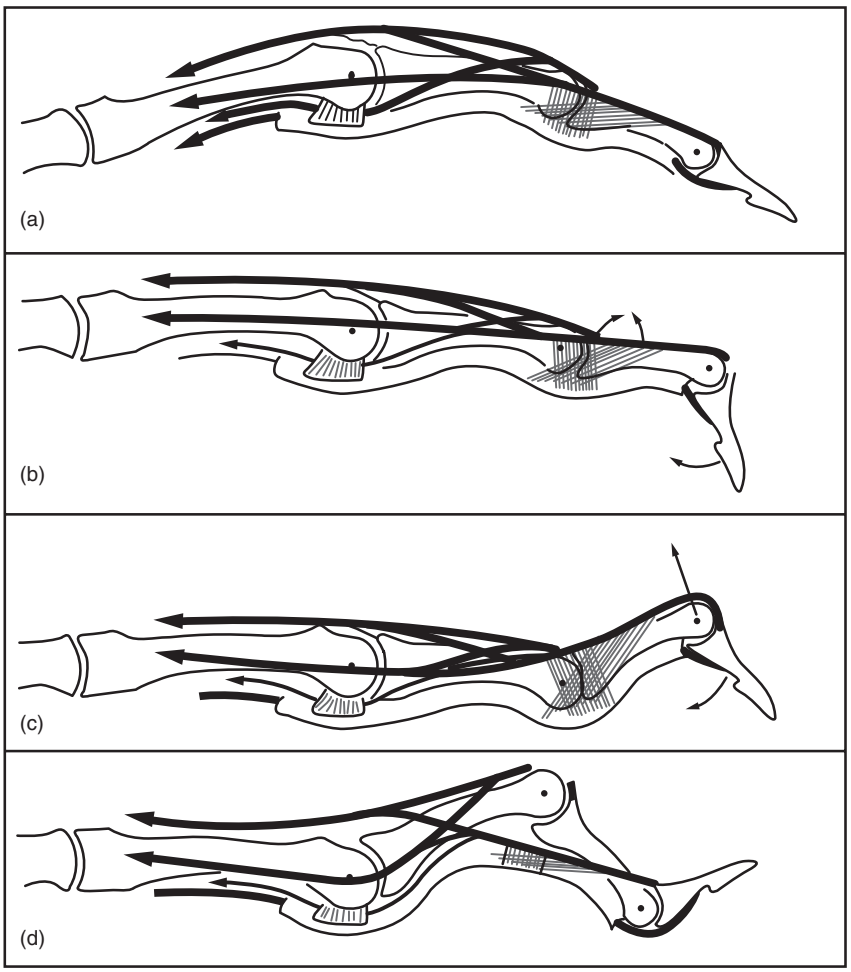

Figure 14.22
Digital deformities: (a) normal anatomy; (b) mallet finger; (c) swan-neck deformity; (d) boutonnière deformity.

joint is directed in extension, depending on the movement chosen and the level of activity of the flexors. In some cases of hyperlaxity, the articulation may result in hyperextension, which would oppose the direction of axial forces exerted by the flexor and extensor tendons. In addition to the mallet deformity, another deformity – swan-neck deformity – can occur. The retinacular ligament in its oblique part is not implicated in this deformity, but its retraction may affect the repair of the distal insertion of the dorsal aponeurosis into the base of the distal phalanx. Significant osseous avulsion may cause palmar subluxation of the base of the distal phalanx. On the other hand, it facilitates a good repair by the appearance of a callus which reinserts firmly into the dorsal aponeurosis.

Swan-neck deformity

Without a lesion of the extensor's terminal insertion, probably facilitated by laxity of the PIP joint, hyperextension of this joint may take place in the three following cases:

- paralysis or insufficiency of the flexor superficialis
- over-retraction of the intrinsic muscles
- excessive traction on the middle extensor tendon.

In the rheumatoid hand this may occur when the wrist is fixed in a flexion position, when there is ulnar luxation of the extensor tendon at the MP level, or when its proximal attachments to the MP joint are destroyed. This deformity is always associated with a phalangeal 'zigzag', as the DIP joint is in flexion and the PIP is in extension. This arises because of the predominant action of the axial forces which result from the activity of the extensor and the flexor profundus, and which are directed against the dorsal surface of the head of the middle phalanx.

The retinacular ligament, which could theoretically oppose this displacement, should be relaxed to render possible the deformity, and this persists as the tissues retract.

Boutonnière deformity

This deformity is rarer and less disabling. It may be post-traumatic, related to a rupture of the median extensor's tendon, associated with a lesion of the triangular sheet. This pathology can be observed in the rheumatoid hand as the synovitis distends the capsule and disrupts the median tendon. It is followed by the flexion of the PIP joint by the action of the superficialis tendon and also the axial forces. There is a tendency for the lateral extensor tendons to subluxate palmarly. These tendons, passing more anteriorly before the transverse axis of flexion, participate in the flexion of this joint. The PIP makes a kind of hernia because of the pathologic gap of the dorsal aponeurosis. The latter has a tendency to retract proximally and this leads to extension of the DIP, a position that will be fixed by the retraction of the retinacular ligaments.

The fifth finger and the hypothenar eminence

The fifth finger (or auricular) is a peculiar ulnar digit owing to its mobility (still inferior to that of the thumb) and also because of a special musculature that forms the hypothenar eminence.

Skeletal anatomy

The mobility of the fourth and fifth digits is noticeable, and this contrasts with the almost complete stiffness of the second and third digits. The ring finger has some mobility, but not comparable with the little finger, even if the two are articulated by a unique carpal bone – the hamartum – and therefore their mobility is somewhat linked.

The inferior surface of the hamatum is nearly quadrilateral, divided in two parts by a crest:

- the area in contact with the fourth metacarpal base is flat in a transverse axis, and more often concave in the anteroposterior direction
- The area for the fifth metacarpal is globally in an oblique plane, oriented toward the vertical axis of the hand. It is always concave from back to front, and convex in its transverse axis.

The basis of the fourth metacarpal presents a 'horseshoe-shaped' surface for the joint with the hamatum, and also another surface on its dorsal aspect, which is flat and is articulated with the capitatum. The fourth metacarpal has only gliding movements of short range in the two directions of flexion and extension.

The fifth metacarpal is also joined with the hamatum and the fourth metacarpal. The joint surface is concave in a transverse axis, and convex in the other axis, perpendicular to the first.

This joint is an arthrodia, but movements occur around the two axes, i.e., flexion–extension and abduction–adduction. As the flexion–extension axis is not perpendicular to the diaphysis of the metacarpal, there exists a rotation. This rotation is 'obligatory' (see the thumb rotation study in Chapter 16) and resembles an 'opposition' to the thumb, and so facilitates the grip. The obliquity of the joint has already been described, and the effect of the palmar deepening of the carpus anteriorly adds to this orientation of the fifth digit.

Muscles of the fifth digit – hypothenar eminence (Figure 14.23)

There are five muscles, if the palmaris brevis is included. They also include the palmar interosseous of the fourth intermetacarpal (the fourth dorsal interosseous muscle is not

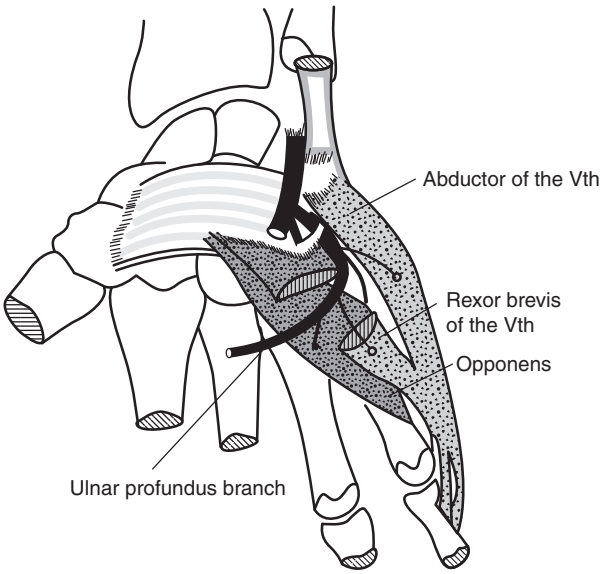

Figure 14.23

The hypothenar eminence, its muscles and the passage of the ulnar profundus branch. Adapted from Brizon and Castaing.[9]

a muscle of the fifth digit) and the three intrinsic muscles that form the hypothenar eminence:

- The palmaris brevis is a thin quadrilateral muscle, beneath the integument of the ulnar side of the hand, which arises, by tendinous fasciculi, from the transverse carpal ligament and the palmar aponeurosis. The fibers are transverse and insert into the skin of the ulnar border of the palm.
- The palmar interosseous muscle of the fourth space arises from the anterior half of the fifth metacarpal diaphysis and ends on the same side, on the basis of the first phalanx. It gives a dorsal expansion which inserts on the dorsal aponeurosis and on the extensor digiti quinti.
- The abductor digiti quinti is located on the ulnar border of the palm of the hand. It arises from the pisiform bone and from the tendon of the flexor carpi ulnaris. It ends in a flat tendon, which divides into two slips, one inserted into the ulnar side of the first phalanx of the little finger, the other into the ulnar border of the dorsal aponeurosis of the extensor digiti quinti proprius.
- The flexor digiti quinti brevis lies on the same plane as the preceding muscle, but on its radial side. It arises from the convex surface of the hamulus of the hamate bone, and the volar surface of the transverse carpal ligament. It inserts into the ulnar side of the base of the first phalanx of the little finger. It is separated from the abductor at its origin by the deep branches of the ulnar artery and nerve. It is sometimes missing and the abductor is then more developed.
- The opponens digiti quinti is triangular and is located beneath the two preceding muscles. It arises from the convexity of the hamulus and the contiguous portion of the transverse carpal ligament. It is inserted on the whole length of the fifth metacarpal along its ulnar margin.
- Some variations of these muscles are often observed during surgery.

It is important to consider the action of these muscles. The palmaris brevis wrinkles the skin of the hypothenar eminence, preventing it from gliding on the deep structures during a strong grasp. The main role of the fifth digit is apparent during flexion, extension, and abduction of this digit. Three muscles generate the flexion of the first phalange: the palmar interosseous, the flexor digiti quinti, and the abductor digiti quinti. Because they are inserted on the base of the first phalanx they give a special force of flexion to this digit. One of the effects is to insure the grip on a tool handle, between the fifth digit, the hypothenar eminence, and, in front of these, the cup of the hand. Its depth is accentuated by flexion of the fifth metacarpal at the level of its joint with the hamatum. Rotation of the digit has already been studied, and in extreme flexion the pulp of the little finger enters into contact with the base of the thenar eminence. Normally the heads of the metacarpal bones describe a little arch, and it is only when a special grasp force is needed that the fifth head 'descends' below the fourth and the third.

Paradoxically, it is said that when, in contrast, an individual does not have great strength in his digits (in his intrinsic muscles), the head of the metacarpal appears to be below the normal plane. It is a test of general strength in musicians.[10]

Extension of the fifth digit is produced by the extensor apparatus, i.e., the two extensors which draw the dorsal aponeurosis proximally. As in the normal physiology of the fingers, the intrinsic muscles (palmar interosseous and hypothenar muscles) facilitate extension of the two distal phalanges when the metacarpophalangeal joints are almost in extension. When they are in flexion, they can only fix the dorsal aponeurosis, and the maximum of their action is flexion of the first phalange.

Abduction of the digit, which corresponds to an ulnar deviation, is important when it is necessary to widen the palmar surface of the digits in some conditions: before grasping a voluminous object, or when supporting a flat one (e.g., a waiter and his platter). Pianists also use also this abduction in some tuning.

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15 Restoration of flexion in the digits

Raoul Tubiana

Loss of flexion of the digits can jeopardize one of the essential functions of the hand: the grip. This severe disability can be caused by neurologic, traumatic, pathologic, or congenital lesions.

Grasp, pinch, and prehension

Digitopalmar grasp as well as thumb–fingers pinch are only the mechanical components of prehension. Prehension can be defined as all of the functions put into play when an object is seized by the hand: intent, permanent sensory control, and a mechanism of grip. Ocular control, useful at the beginning of a normal prehension, becomes necessary during all stages of prehension when sensation is absent or insufficient.

Having at his disposal different types of grips, man can choose and adapt his pattern of prehension not only to the object seized but also to the purpose of the grip.

The choice of the type of grip is preselected; the hand then adapts to the form of the object.

Grasp consists of three stages: first, opening of the hand; next, closing of the digits in order to seize the object; and, finally, regulation of the force of grasp.

Opening of the hand requires the simultaneous action of the extrinsic extensors and intrinsic muscles. It is proportional to the volume of the object grasped. 'Before we can grasp, the extensors of the wrist must contract or else the flexors of the fingers will pull the wrist into flexion. Before we can pinch, the long abductor of the thumb has to spring into action or else the carpo-metacarpal joint will flex and the MP joint will drop'.[1]

A variety of combinations are possible for positioning the mobile elements of the hand in order to seize an object and adapt to its form.

Schematically, the functional attitudes of the grips can be dividing into power grips, where the flexed digits maintain the object against the palm (Figure 15.1), and precision grips (Figure 15.2) between the pulps of the thumb and of the flexed fingers.[2] These two types of grip depend less on the form of the object than on the reason for which the object is grasped.

The thumb is absolutely indispensable for precision grips. It gives both stability and control of direction. The thumb is also very useful for power grips, forming a buttress that resists the pressure of the object grasped with the other fingers.

The thumb is not indispensable for all types of power grip. Certain grips use only a simple hook, without precision, formed by the long fingers flexed, which is controlled by the powerful digital long flexor muscles (Figure 15.3).

The role of the intrinsic muscles assumes increased importance when agility and precision are necessary.

Regulation of the force of grip must be varied according to the weight, fragility, surface characteristics, and utilization of the object. Precise and continuous sensory information is indispensable for regulation of pressure and in preventing premature release.

The anatomy and physiology of flexion of the fingers has been described in the preceding chapter. You will recall that flexion of the fingers is brought about by the combined action of:

1. The flexor digitorum profundus (FDP), which flexes the distal phalanx and reinforces the flexion of the two proximal phalanges (Figure 15.4)

Figure 15.1
Power grip.

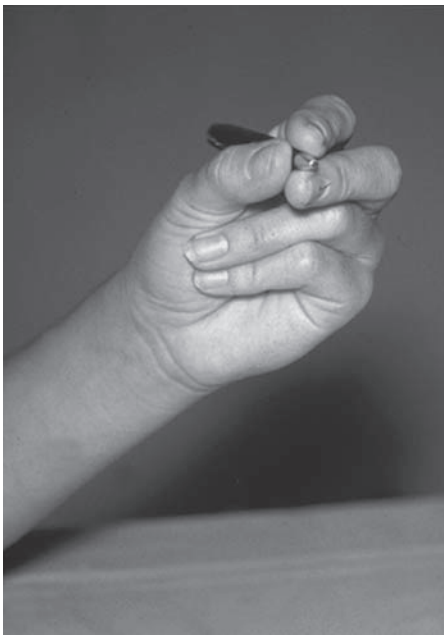


Figure 15.2
Precision grip.

Figure 15.3
Hook formed by the long fingers without participation of the thumb.

2. The flexor digitorum superficialis (FDS), which flexes the proximal interphalangeal joint
3. The interosseous muscles, which contribute to the flexion of the proximal phalanx, when the interosseous hood is displaced distally on the dorsal aspect of the proximal phalanx (Figure 15.5).

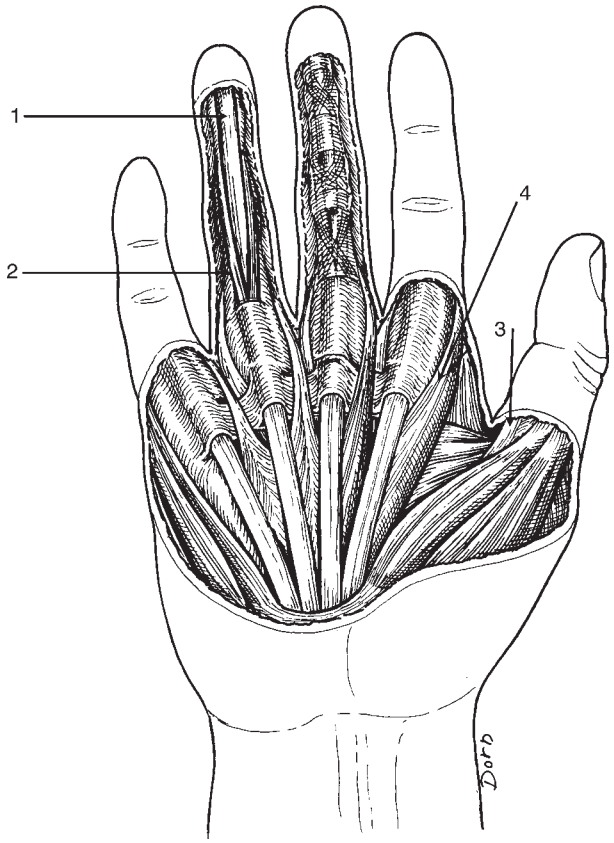


Figure 15.4
The flexor tendons with their fibrous sheath: (1) flexor digitorum profundis; (2) flexor digitorum superficialis; (3) flexor pollicis longus; (4) lumbrical muscle.

Nerve supply according to grip pattern

Sensation of the ulnar palm and fingers and innervation of the FDP muscle of the ulnar fingers, important in the digitopalmar grasp, depend on the ulnar nerve. Also, intrinsic muscles of the thumb required for power grip – the adductor pollicis and the deep head of the flexor pollicis brevis (FPB) – depend on the ulnar nerve.

Sensation of the radial part of the palm and radial digits, the FDP of the radial fingers, and the flexor pollicis longus (FPL), important in precision handling, are controlled by the median nerve. Also, the opponens pollicis (OP), the abductor pollicis brevis (APB) and the superficial head of the FPB, required for precise positioning with the object, are innervated by the median nerve.

The FDS for the four fingers is innervated by the median nerve: FDS action is discreet during unopposed flexion, but becomes predominant in flexion against resistance.

Opening of the hand depends on the radial nerve.

Peripheral nerve lesions

The median and ulnar nerves are involved in the flexion of the digits.

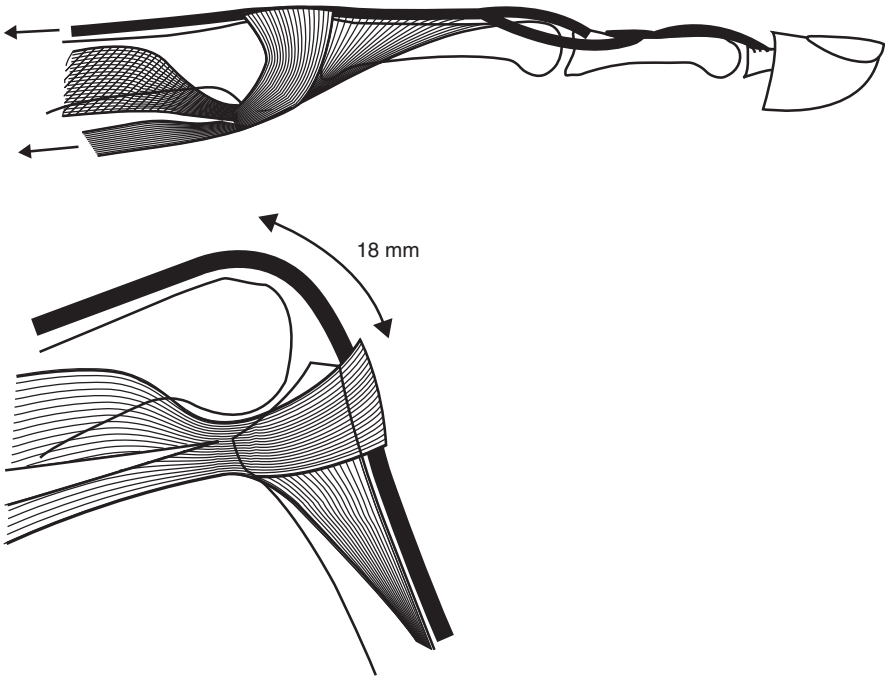


Figure 15.5
Distal displacement of the interosseous hood.

Median nerve injuries

The median nerve (Figure 15.6) arises in the brachial plexus by two roots: one from the lateral cord (C6–C7) and the other from the medial cord (C8–T1). It descends in the anteromedial compartment of the arm with the brachial vessels, and enters the anterior compartment of the forearm, covered by the bicipital aponeurosis. It crosses anterior to the ulnar artery and passes between the two heads of the pronator teres (PT), then under the fibrous arch joining the two heads of the FDS. It gives off the anterior interosseous nerve proximal to the FDS fibrous arch, which supplies the FPL, the flexor profundus to the index finger, and the pronator quadratus.

The median nerve then runs distally in the forearm closely bound to the deep surface of the FDS within the muscle sheath. As the muscle changes to tendon, in the lower half of the forearm, the median nerve is at first lateral to the index flexor tendon, then anterior to it. It passes under the flexor retinaculum, and, as it emerges from the carpal tunnel, it divides into its terminal motor and sensory branches. The median nerve is the most important sensory nerve.

In low median nerve palsy, the thenar intrinsic muscles innervated by the median nerve – the APB, the OP, the superficial head of the FPB and the radial lumbricals – are paralyzed.

In high median nerve palsy, the PT, the flexor carpi radialis, the FDS, the index and long finger flexor profundi, the FPL, and the pronator quadratus muscles are also paralyzed.

The median nerve can be compressed at several points along its course (Figure 15.7): under the bicipital aponeurosis (lacertus fibrosus), between the heads of the pronator teres, under the FDS fibrous arch, and within the carpal tunnel. Dissociated median nerve palsy resulting from compression (or division) of the anterior interosseous nerve produces a characteristic deformity: during pinch, the distal phalanges of the thumb and index finger cannot flex, and stay in extension. The main disability in median palsy is *the sensory loss in the radial aspect of the hand*, which reduces considerably the functional benefit of tendon transfers. However, in spite of this handicap, it seems reasonable to restore fingers' flexion and in some cases thumb opposition. It must be kept in mind that restoring sensation (or at least protective sensation) may be possible with a late nerve repair made years after division of the nerve; this does not apply to motor recovery.

Restoration of thumb index and long finger flexion can be obtained by brachioradialis, extensor carpi radialis (ECR) or extensor carpi ulnaris (ECU) transfers. These muscles have less potential excursion than the flexor profundi or FPL, and satisfactory flexion can only be obtained if the transfer's excursion is amplified by the tenodesis effect of wrist motion. The ECR brevis (ECRB) is preferably not used for digit transfer, because it is the main wrist extensor.

The ECR longus (ECRL) can be transferred to index and middle finger flexor profundi tendons; the tension is adjusted so that the finger is fully flexed when the wrist is extended to 45°. In a simpler way, the index profundus tendon can be sutured, side to side, to the conjoint middle, ring, and little finger profundus tendons. With flexor superficialis paralysis, a swan-neck deformity can develop if the proximal interphalangeal (PIP) joint is lax. Tendon transfer to the index and middle profundus tendons may aggravate the deformity.

The brachioradialis must be freed of fascial attachments throughout the forearm before its transfer to the FPL. Restoration of thumb opposition is the subject of Chapter 17.

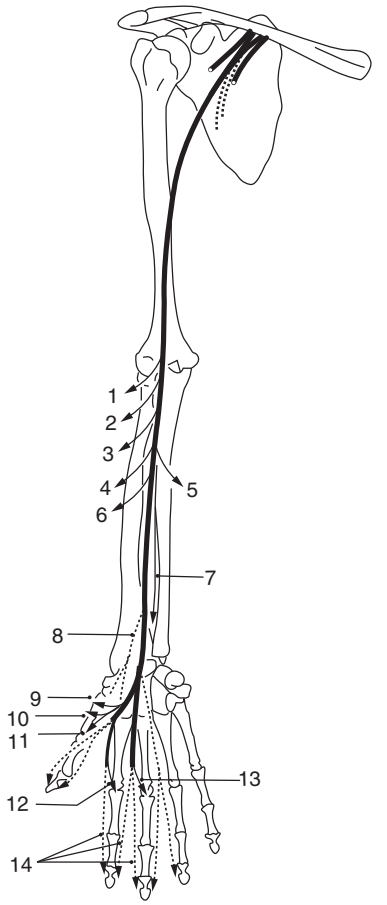


Figure 15.6

The median nerve; muscles supplied and cutaneous distribution: (1) pronator teres; (2) palmaris longus; (3) palmaris brevis; (4) flexor digitorum superficialis; (5) flexor digitorum profundus to second and third digits; (6) flexor pollicis longus; (7) pronator quadratus; (8) palmar cutaneous branch; (9) abductor pollicis brevis; (10) superficial branch to flexor pollicis brevis; (11) opponene pollicis; (12) first lumbrical; (13) second lumbrical; (14) digital nerves (sensory). The sensory branches are shown as dotted lines.

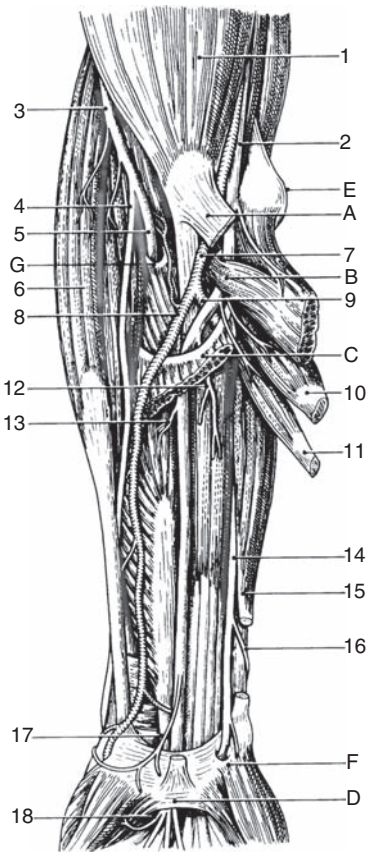


Figure 15.7

Dissection of the anterior aspect of the forearm to show the median nerve, radial nerve, and ulnar nerve; the common sites of compression of the three nerves are shown. (1) biceps brachi; (2) median nerve; (3) radial nerve; (4) sensory branch of radial nerve; (5) motor branch of radial nerve; (6) brachioradialis; (7) brachial artery; (8) radial artery; (9) ulnar artery; (10) flexor carpi radialis; (11) palmaris longus; (12) branch of anterior interosseous nerve to flexor digitorum profundus to index and middle fingers; (13) branch of anterior interosseous nerve to flexor pollicis longus; (14) ulnar nerve; (15) flexor carpi ulnaris; (16) dorsal cutaneous branch of ulnar nerve; (17) palmar cutaneous branch of the median nerve; (18) thenar branch of the median nerve. Common sites of nerve compression in the forearm. Median nerve: (A) expansion of biceps; (B) two heads of pronator teres; (C) flexor digitorum sublimis; (D) carpal tunnel. Ulnar nerve: (E) medial epicondylar groove; (F) Guyon's canal. Radial nerve: (G) arcade of Frohse.

Ulnar nerve injuries

The ulnar nerve arises from the medial cord of the brachial plexus (C7, C8 and T1 roots) (Figure 15.8). It runs medial to the brachial artery, goes through the medial intermuscular septum, and at the elbow level passes between the medial epicondyle of the humerus and the olecranon. It enters the forearm between the humeral and ulnar origins of the flexor carpi ulnaris (FCU) and descends within the anteromedial compartment of the forearm under cover of the FCU. At the wrist level, it runs with the ulnar vessels in the osteofibrous Guyon's canal. Just distal to the pisiform, it divides into its two terminal branches: the sensory superficial branch and the deep motor branch.

The ulnar nerve supplies the FCU and the two ulnar heads of the FDP. In the lower third of the forearm, it gives off the dorsal cutaneous branch, which supplies the skin of half the dorsum of the hand. Its terminal sensory branch supplies the ulnar part of the palm and the palmar skin of the little finger and of half of the ring finger. The deep terminal branch sends fibers to all the interosseous muscles, the ulnar lumbricals, the adductor pollicis, and the deep head of the FPB.

The ulnar nerve can be compressed at several points:

- at the arm, rarely by the inconstant expansion of the triceps, forming the 'Struthers' arcade'
- at the elbow level in the fibro-osseous tunnel between the medial epicondyle and the olecranon; next, the

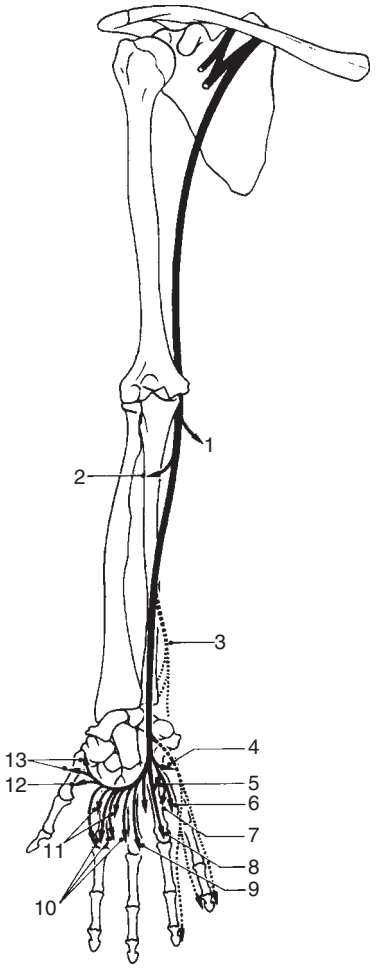


Figure 15.8

The ulnar nerve; muscles supplied and cutaneous distribution: (1) branch to flexor carpi ulnaris; (2) branch to flexor digitorum profundus supplying fourth and fifth digits; (3) dorsal cutaneous branch; (4) palmar cutaneous branch; (5) branch to abductor digiti minimi; (6) branch to opponens digiti minimi; (7) branch to flexor digiti minimi; (8) fourth lumbrical branch; (9) third lumbrical branch; (10) branch to palmar interosseous muscles; (11) branch to dorsal interosseous muscles; (12) deep branch to flexor pollicis brevis; (13) branch to adductor pollicis. The sensory branches are shown as dotted lines.

nerve runs between the humeral and ulnar heads of the FCU
- at the wrist level, as it courses through Guyon's canal
- in the palm, the deep terminal branch dives under a fibrous band between the pisiform and the hook of the hamate.

Restoration of the ring and little fingers flexor profundi in a nerve palsy has been debated. Bunnell[1] believed it inadvisable because the more long flexors that are working, the more obvious should be the claw hand deformity. Brand[3] recommended that tendon transfers not be considered unless there is also an associate high median palsy.

If there is marked weakness of the ulnar fingers, Omer[4] attaches the profundus tendons of the ring and little fingers to the profundus tendon of the middle finger, the index profundus being left free.

High median and high ulnar nerve paralyses

Functional loss is severe. The most important clinical problem is the total loss of volar sensibility; also, digitopalmar grasp as well as thumb–finger grip are lost.

The goals of salvage surgery are to restore a simple digitopalmar grasp and a stable type of key pinch, rather than an opposition thumb finger pinch used for precision activities, which are definitely lost in the absence of sensibility.

Many motor transfers are available: ECU, ECRL, extensor indicis proprius (EIP), thumb extensors, and brachioradialis. They all have been used in different patterns. Correction of claw-hand deformity is rarely necessary because clawing is minimal when long flexors are paralyzed.

The ECRL is a suitable transfer for finger flexion: it is transferred around the radial aspect of the forearm to the flexor digitorum profundus at about 4 cm proximal to the carpal ligament, the wrist being held in neutral position.

The brachioradialis freed in the forearm is a suitable transfer to provide key pinch; it is sutured to the flexor pollicis longus. Arthrodesis of the metacarpophalangeal (MP) joint of the thumb is indicated in case of instability.

Both the ECRL and brachioradialis have no adequate excursion for full flexion of the digits, and their transfer should be reinforced by active wrist extension.

Some protective sensibility can be restored in the radial side of the hand by a radial-innervated dorsal skin flap[4] (see Chapter 25).

Flexor tendon lesions

Flexor tendon lesions usually result from trauma; however, some diseases, particularly rheumatoid arthritis, can disturb or abolish digit flexion.

Flexor tendon injuries

The inclusion of flexor tendon injuries in a book on restoration of function after upper limb paralyses and muscular defects is certainly debatable. However, flexor tendon repair has been a major challenge for surgeons of my generation, and this book is probably my last opportunity to expose this fascinating problem.

Unlike their extensor counterparts, the flexor tendons of the fingers run an almost entirely intrasynovial course, which eases their gliding but renders surgical repair more difficult. These long tendons move several joints. They have a lengthy excursion, and transmit considerable force during a power grip.

Anatomy, physiology, surgical repair, and expected results vary with the location of flexor tendon injury.

The Committee on Tendon Injuries of the International Federation of Societies for Surgery of the Hand (IFSSH) has adopted a classification in five zones.[5] From distal to proximal, these are:

zone I: distal to the insertion of the flexor superficialis
zone II: common digital canal to the profundus and the superficialis
zone III: between the digital canal and the carpal tunnel
zone IV: carpal tunnel
zone V: the wrist.

The regions crossed by the long flexor of the thumb are preceded by the letter T (Figure 15.9).

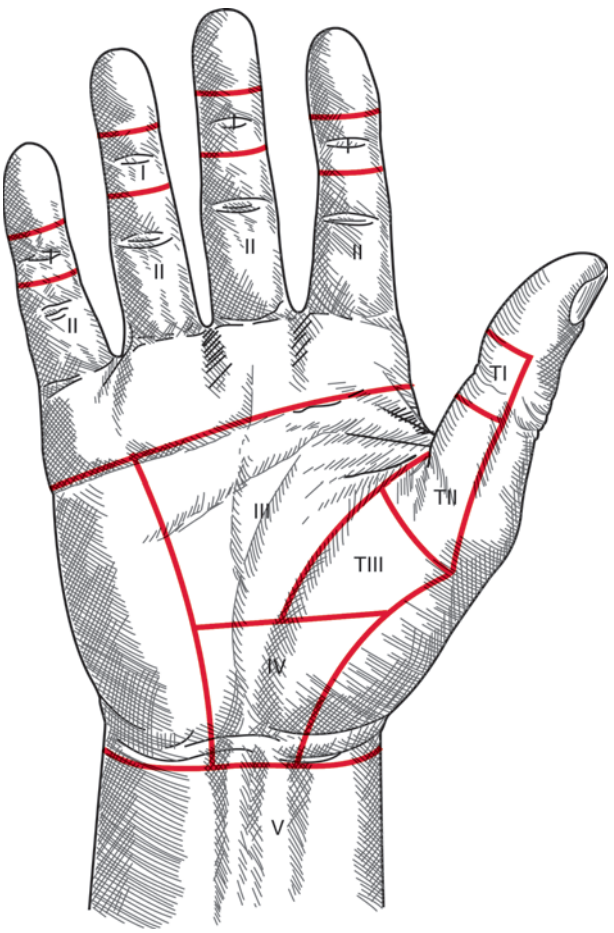


Figure 15.9
The five anatomic regions crossed by the flexor tendons of the fingers and thumb.

In fact, surgical indications are discussed with reference to only three main zones:[6]

- *The distal zone*: only the flexor profundus is present here, and the functional sequelae are limited.
- *The intermediate zone*: this is the former "no-man's land" of Bunnell, corresponding to the fibro-osseous digital canal. This zone poses the most difficult surgical problems, because of the intricate gliding relationship of the two flexor tendons, the proximity of fixed anatomic structures, and the poor blood supply.
- *The proximal zone*: this includes the palmar, carpal and wrist regions.

The tendons for the different digits are close together; this accounts for the frequency of multidigit injuries. However, the blood supply of the tendons is better and the prognosis for repair is also better.

Surgical repair of flexor tendon injuries

Repair of flexor tendons is difficult, because it requires both a strong tendon callus to resist traction of powerful muscles and, even more difficult to obtain, functional gliding planes, capable of ensuring the mobility of the tendons. In spite of considerable improvements in treatment methods, repair of the divided flexor tendon continues to be one of the more challenging problems in hand surgery.

The history of flexor tendon repair is very instructive in that it shows that concepts have changed with the progress of our scientific knowledge. For centuries, tendon surgery remained confusing, because nerves and tendons were supposed to be of similar origin, suggesting a strong risk of convulsions following surgery to repair these structures, Galen (AD 129–ca. 200) was the first to differentiate nerves from tendons.[7]

With the exception of Avicenna, the famous 10th century Arabian surgeon, tenorraphies were rarely performed until the 18th century. In 1767, Hunter performed a study investigating the tendon healing process on canine Achilles tendon.[8] In 1880, Nicoladoni of Vienna described techniques of tendon repair.[9] Biesalski[10] of Berlin used autogenous sheaths of paralyzed tendons to avoid adhesions (1910), and Lexer[11] of Jena reported his 1912 results with flexor tendon grafts. They were followed in 1916 by Leo Mayer,[12] who trained with Biesalski and described the blood supply of tendons, the role of mesotenon and paratenon, and many other points that will be mentioned below.

Sterling Bunnell between 1918 and 1928 elaborated the classic principles of flexor tendon repair.[1,13–15] He reviewed the anatomy and physiology of flexor tendons, outlined the necessity for an atraumatic technique, and established a protocol for 3 weeks' postoperative immobilization. Primary suturing was almost always doomed to failure in the digital canal. Although the tendon callus was usually satisfactory, adhesions with fixed surrounded structures were so extensive that tendon mobility was nil. In the face

of these poor results following primary suturing, in what he called 'no man's land', Bunnell in 1922 gave this advice: 'Close the skin, wait for the wound to heal, then perform a secondary repair as follows: excise the two flexors and graft the profundus tendon alone from the lumbrical to the digital extremity'.[14]

Tendon grafting is an ingenious concept that offers the advantage that the sutures are tension-free and can be placed in an optimal location away from the fibrous pulleys. In fact Bunnell's secondary one-stage tendon grafts have been an undeniable improvement compared with the poor results obtained after the usual criss-cross tendon suture in the digital canal, so that Bunnell's teaching was held as dogma by generations of surgeons for the treatment of flexor tendon lesions within the digital canal.

Some surgeons, including Boyes,[16,17] Littler,[18] and Pulvertaft,[19,20] published long series of single-stage tendon grafts on well-selected patients, in whom function has been restored in a high proportion of cases.

The secondary single-stage flexor tendon graft was still the main procedure used for flexor tendon repair in zone II when I received in 1951 a Fulbright grant. I spent 6 months with Bunnell in San Francisco; then I visited Boyes and Mason and Allen, Littler, and Pulvertaft. All of these famous hand surgeons used the same basic principles of tendon grafting, with some differences in the surgical technique. They had in common an enormous experience of this special procedure and an extremely meticulous surgical technique. They all admitted that the results of flexor tendon grafting were not constantly satisfactory and that a single-stage tendon graft gave good functional results when soft tissue is supple, passive mobility is complete, and at least two well-situated pulleys are present. Associated lesions such as contracted scars, joint stiffness, nerve and bone lesions, multiple tendon injuries, and unsuccessful attempts at primary suturing have an adverse effect on the results and seriously worsen the prognosis. This was clearly shown in an extensive study of 1000 cases of flexor tendon grafts by Boyes and Stark.[17]

Back in Paris, I tried to put into practice what I had learnt, and I published in 1960 the results of my first cases of flexor tendon grafts performed on well-selected patients.[21]

However, this long and delicate procedure, reserved for selected patients and performed by selected surgeons, could not fulfill the needs for the treatment of a very common injury. It seemed that other approaches to flexor tendon repair should be investigated.

What has changed in half a century

My long career has allowed me to follow the evolution of flexor tendon repair during half a century.

Evolution of the concepts

In the 1950s and 1960s, some surgeons recognized their own 'less-than-satisfactory' results with single-stage tendon grafts. This is not entirely surprising when one realizes that this surgical repair is far from physiologic. Flexion of the fingers is a complex mechanism involving the movement of two tendons with different excursions, gliding freely within a tight fibrous sheath lined with a synovial membrane. The trauma from the ablation of the severed tendons destroys the synovial sheath and a portion of the fibrous sheath. Additionally, only one tendon is reconstructed, when normal anatomy requires two.

Some courageous surgeons from the USA and Europe – Siler, Posch, Kleinert, Kessler, Verdan, Michon, and G Brunelli – tried to improve the technique of primary tendon repair in zone II, in order to avoid the systematic use of a secondary flexor graft. In fact, the father of the modern technique of primary flexor tendon repair was Harold Kleinert, who combined a new concept of tendon healing with preservation of the flexor tendon sheath, a new technique of tendon suture with a core grasping suture plus a peripheral running suture, and a new method of early semi-active mobilization.

When Kleinert et al[22] published their long-term clinical findings on primary flexor tendon repair in 1967, the results were so astonishingly good that the American Society for Surgery of the Hand (ASSH) sent to Louisville a special Committee for Control.

When primary tendon suture is contraindicated because of the poor condition of the finger, or late referral, a two-stage tendon graft is now usually considered, which involves a preoperative re-education, then an excision of the damaged flexor tendons and insertion of a silicone rod to induce pseudo-sheath formation and allow pulley reconstruction, and about 3 months later a secondary flexor graft.[23]

These new concepts were supported by considerable progress in the understanding of tendon physiology, nutrition of flexor tendons, and tendon healing, and by improved suture materials, suture techniques, and early postoperative rehabilitation, which have progressively changed the indications for and results of flexor tendon repairs.

Nutrition of flexor tendons

It was generally admitted that the tendon was an avascular structure, with low metabolic activity and minimal healing potential.[24,25] Direct continuation of vessels from muscles and bone was for a long time supposed to be minimal, with tendon nutrition depending primarily on the mesotendon and the vincula.[26] The flexor digitorum tendons have a precarious blood supply at the level of the digital sheaths. The vasculature is segmental, arranged in an arcading fashion, with each arcade supplying a specific portion of the tendon. The vinculum longum at the chiasma level is common to both tendons (Figure 15.10a). However, variations in the pattern of the vinculum longum are frequent.[27] The flexor pollicis longus tendon also has a segmental vascularization from the vincula in its digital notion. In its proximal portion, the tendon receives branches from the median nerve artery.

In spite of longitudinal intrinsic vessels, Lundborg et al[28] demonstrated the presence of several vascularly deprived

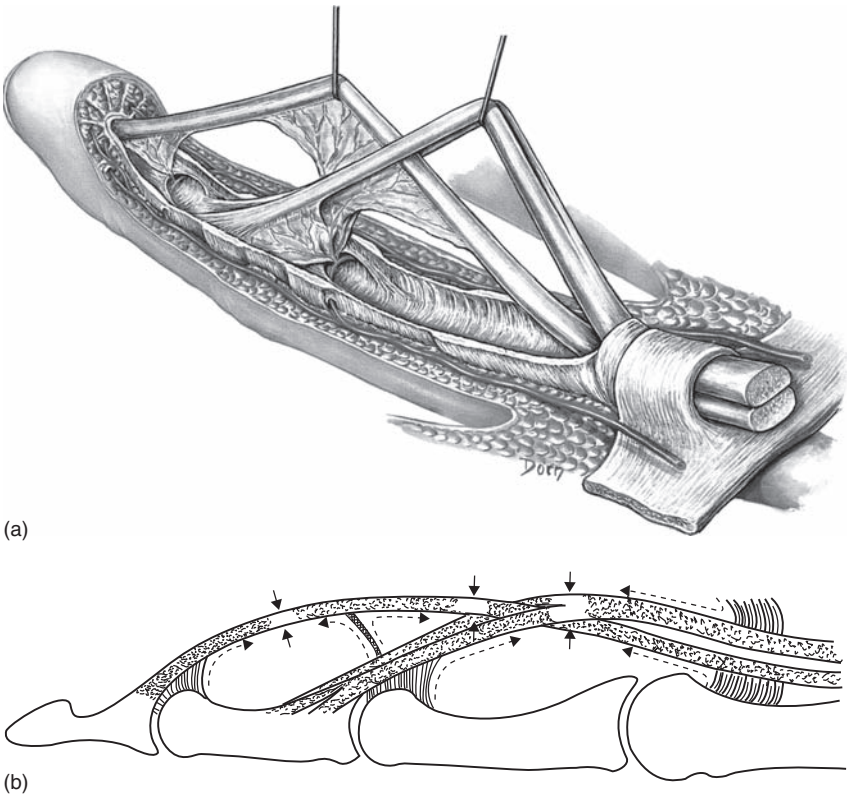


Figure 15.10

(a) The digital flexor tendons. The FDP passes through the FDS. The vinculum longum, at the chiasma level, is common to both tendons. Excision of the superficialis would destroy the profundus vascularization; (b) avascular segments on flexor tendons.

zones, 'avascular segments': one of the flexor superficialis, just proximal to the chiasma, and two of the flexor profundus, proximal and distal to the vinculum longum (Figure 15.10b). Also at the level of the proximal interphalangeal joint, the flexor profundus tendon is vascularized only along its dorsal aspect. The anterior 1 mm of the tendon, on which considerable pressure is exerted, is 'avascular' (Figure 15.11). In addition, the vascularity of the flexor tendons becomes poor with age[27] (Figure 15.12).

This segmental supply system, inadequate in some areas, emphasizes the importance of the nutritional role of the synovial fluid secreted by the cells of the synovial sheath.[29]

For Guimberteau,[30] flexor digitorum tendons are safely supplied in all parts, thanks to the continuous longitudinal system, and are supported by transverse relays such as branches from the ulnar pedicle, palmaris arcade, collateral arteries, and vincula. Like every organ, the tendon has a vascularization adapted to its function. Guimberteau described a 'sliding unit' that integrates the tendon, a peripheral 'multimicrovacuolar collagenous dynamic absorbing system' (MCDAS), situated between the tendon and its neighboring tissue that favors optimal sliding and the arteriovenous supply. The tendon may slide without any hindrance and without inducing any movement of other neighboring tissue; small vessels are part of the collagen framework and are submitted to the various deformations during movements.

Manske[31] used tracer materials to compare the role of vascular perfusion and synovial diffusion in supplying the nutritional needs of the flexor tendon within its sheath.

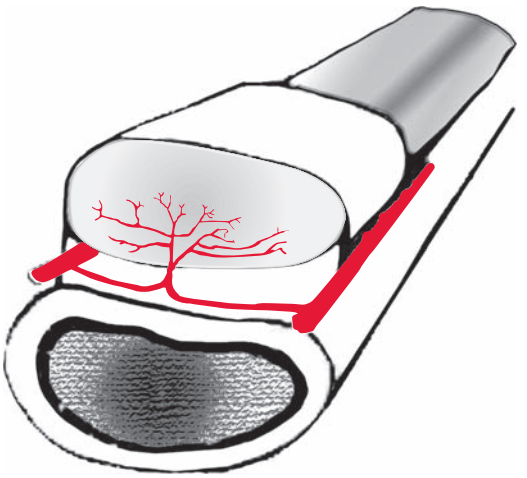


Figure 15.11

At the PIP level, the FDP tendon is only vascularized along its dorsal aspect.

He concluded that there is a dual source of nutrients, but that diffusion is a more effective pathway than perfusion.

It is now admitted that the nutritive mechanism of the flexor tendon in the digital canal depends on the blood supply from the vincular system, the longitudinal intrinsic vessels and the synovial fluid.[27-29,31-36] However, the relative roles of synovial fluid and epitendinous vascular ingrowth in healing flexor repairs within the digital canal is still debated. For Amadio et al,[37] synovial fluid is not sufficient

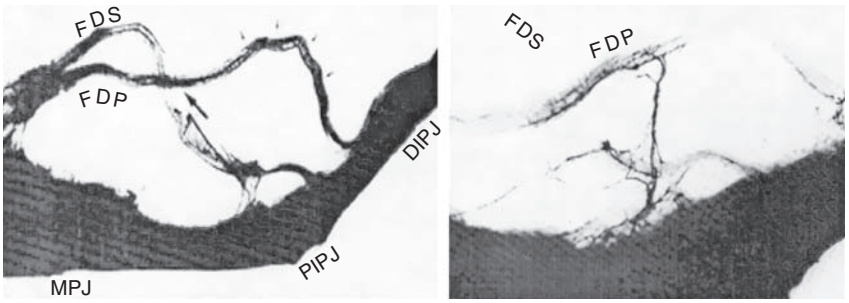


Figure 15.12

Changes in blood vessels distribution with age. (Reproduced with permission from Dr Matsui.)

to nourish the flexor tendons if the vincular system is avulsed. This is one reason to repair rather than resect the superficialis tendon, since resection destroys the vinculum longum to the profundus. It seems important to preserve the tendon vascularization as much as possible and to repair the digital collateral arteries when possible, because it has been proved that the results after flexor tendon repair are better when the tendon vascularization is good than when it has been damaged.

Management of the tendon sheath

It is now assumed that the flexor tendon sheath should be preserved as much as possible: the synovial sheath for the tendon's nutrition, and the fibrous sheath for its mechanical role. In 1975, Doyle proposed a new nomenclature of the flexor pulley system composed of five annular pulleys and three cruciate pulleys. Manske and Lesker added a palmar aponeurosis pulley, which in conjunction with the A1 pulley contributes to maintenance of flexor tendons at the MP joint (Figure 15.13).

Studies on the role of the pulley system in controlling the relationship between tendon excursion and joint mobility have shown that the A2 and A4 pulleys are functionally the most important. However, the essential function of these pulleys can be preserved even if only half of the length of the pulley is intact.

The theoretical advantage of sheath repair in a clean-cut tendon injury is that it serves as a barrier to the formation of extrinsic adhesions, and provides a smooth gliding floor for the repaired tendon and avoids entrapment of the sutured tendons by the cut edges of the sheath during postoperative tendon mobilization.[38]

After a period of 'almost obsessive closure of the tendon sheath',[39] closure of the synovial sheath is no longer considered a necessity by most hand surgeons. Partial release such as venting the pulley to allow free running of the repair, or simply laying the sheath back over the tendons without suturing, or not repairing a partial cut of any pulley, seems permissible when other pulleys and most of the sheath are not injured. One should remember that the friction of the repaired tendon within its sheath is increased by the bulk of the repair and the edema. Sheath management should avoid narrowing of the tendon gliding space.

During secondary tendon grafting surgery or tenolysis, at least the A2 and A4 pulleys should be preserved or reconstructed.

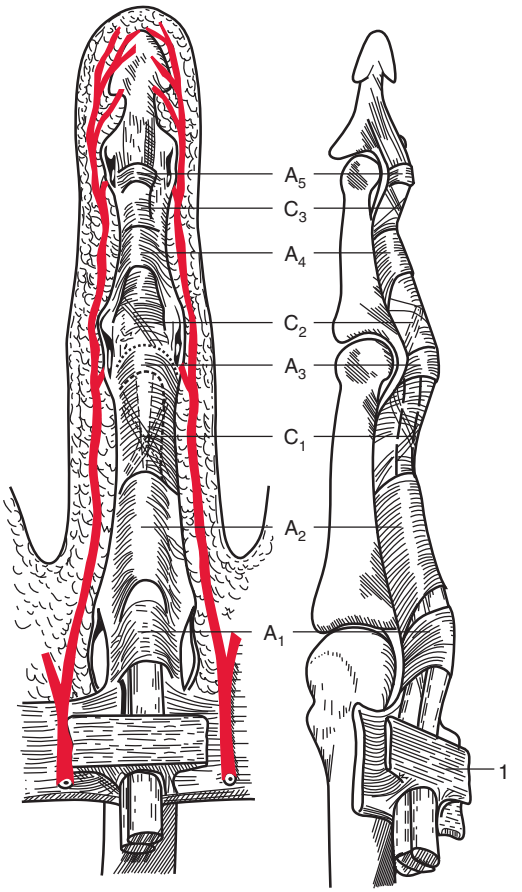


Figure 15.13

The flexor tendon sheath, with the five annular pulleys. Pulleys A2 and A4 have bony insertions, A1, A3 and A5 are inserted on volar plates. Between the annular pulleys are the three cruciform flexible portions. The natatory ligament of the palmar aponeurosis (1) constitutes another pulley.

Tendon healing

The scarring process in the tendon wound edges cannot be isolated from that which occurs in the adjacent tissues. This was the 'one-wound scar' concept described by Peacock.[25]

In 1962, Potenza,[40] by experimental studies of the mode of healing in canine flexor tendons, was able to prove that divided tendons repaired by criss-cross suturing and then splinted heal entirely by the ingrowth of fibroblastic tissue

originating outside the tendon. 'Tendons heal by the fibroplastic response of the surrounding tissues whose own integrity has been violated'. The greater the tissue trauma, the greater the fibroplastic response and hence, the greater the resultant adhesions.

The prevalent thinking (of Bunnell, Mason, Boyes, and others) was that one should excise the tendon sheath around the area of repair to allow neovascularization.

The 1941 experiments of Mason and Allen[41] showed that the resistance of a sutured tendon diminishes rapidly until the fifth day; this is the 'lag time', an inflammatory phase during which the strength of the repair is almost entirely imparted by the suture itself. After this time, there is a fibroblastic or collagen-producing phase 2, from the fifth day to 4 weeks. During phase 2, the resistance increases slowly, and on the 15th day returns to what it was on the day of the repair. The resistance of the healing scar continues to increase for 6 weeks. A remodeling phase (phase 3) continues until approximately 4 months.

These findings reinforced Bunnell's practice of 3 weeks' immobilization before allowing tendon gliding.

During the 1960s and 1970s, significant improvements in our understanding of the mechanisms of repair and in treatment methods occurred when some surgeons – Kleinert et al[22] and Kessler et al[42] – realized the influence of early tendon mobilization on the results of tendon repair. Laboratory experiments, in particular those by Gelberman, Manske and colleagues, have provided biologic verification of the effect of early digital mobilization on the rapidity and quality of flexor tendon healing. Movements of flexion and extension of the finger create a 'pump effect' that allows nutrition of the tendon, particularly on the avascular volar area of the tendon.[43,44]

Restoring gliding function in a divided flexor tendon is a complex biologic, biophysical, and biochemical problem. A large amount of research[32,33,35] has dispelled the previous concept that tendons lacked the intrinsic biologic capability to participate in their own healing. It is now admitted that tendon, which is a living vascularized tissue, possesses a definite intrinsic potential for repair. Lundborg[35] demonstrated that a flexor tendon divided and isolated in a synovial fluid environment, without vascular supply, is able to heal without any adhesion formation. Tendons have both an intrinsic and an extrinsic capability to heal.

For Matthews and Richards,[45] most adhesions are the result of three factors induced by the tendon repair, contributing in various degrees: suturing, synovial sheath excision, and immobilization.

The aim of the tendon repair is to favor intrinsic healing. The more intrinsic healing, the less adhesion formation. In a series of laboratory experiments, Gelberman et al[46–48] demonstrated that the application of early *passive* stress to repair canine tendons led to a more rapid recovery of tensile strength, fewer adhesions, improved excursion, and significantly shortened period of early repair site softening that was identified by Mason and Allen[41] after immobilization of the tendon repair. The important clinical significance of these studies was to show that early digital mobilization could alter the primary extrinsic mechanism of tendon repair in favor of intrinsic tendon repair, achieving a good example of mecano morphogenesis.

More recent studies[49,50] have reported that increasing the loads applied to the flexor tendon repair site by active early mobilization enhances the healing process.

From all these studies, it is now accepted that the most effective method of tendon repair involves the use of a strong suture technique, followed, when possible, by an early postoperative active controlled motion program.

Surgical technique

Surgical technique has evolved in many ways.

Surgical exposure

Interrupted incisions, which were once popular, necessitated difficult subcutaneous tunneling. Bruner's[51] anterior zig-zag incisions are now widely used (Figure 15.14). The neurovascular bundles in the digits are left in their beds. These incisions are not subjected to change in length during flexion and extension, and are free from significant scar contracture. When necessary, the digital incision is extended in a zig-zag fashion in the palm and wrist.

Every surgeon practising tendon surgery since Mayer and Bunnell has agreed that an 'atraumatic' surgical technique has an undoubted influence on results.

At present, the common use of magnification facilitates dissection and suturing. Great care should be taken to minimize any handling of the tendon surface, for this would predispose to adhesions; the tendon is grasped at the site of laceration. Often, additional openings in the tendon sheath are necessary, they should be made over the membranous area. Technique of tendon retrieval using a small flexible silastic tube is a great help. Hypodermic needles are passed transversally through sheath and tendons, to stabilize the tendon ends for repair (Figure 15.15).

Tendon suture

The method of flexor tendon repair has a great influence on the strength of the repair site during the 6 weeks after repair.

The suturing technique should avoid tissue compression while providing sufficient strength to allow early mobilization without gapping or rupture until such time as the healing tendon has sufficient strength to take over from the surgical repair. Gapping at the repair site becomes the weakest part of the tendon, unfavorably alters tendon mechanics, and attracts adhesions, with a resultant decreased excursion.[52]

There have been many improvements in tendon suture technique.

The classic criss-cross sutures used by Bunnell tend to strangulate the tendon's extremities, and have been abandoned (Figure 15.16a).

It is now admitted by most surgeons that flexor tendon repair should include a suture within the tendon – the core

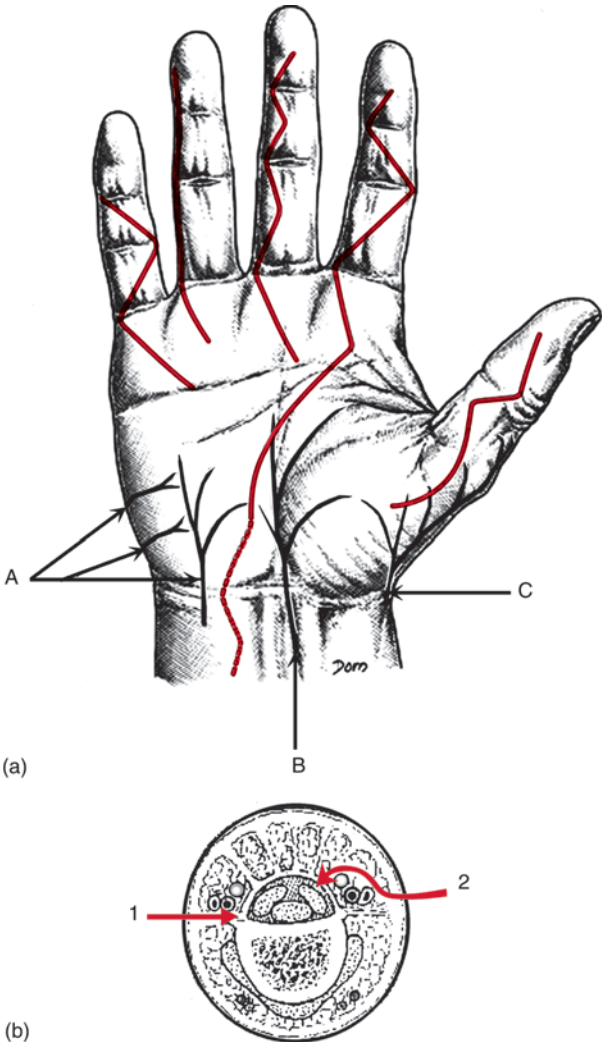


Figure 15.14

(a) Bruner's zig-zag and longitudinal midlateral incisions: zig-zag Bruner incision on the little finger; hemi Bruner volar incision on the long finger and on the thumb; longitudinal midlateral incision on the ring finger; digital incision extended in a zig-zag fashion in the palm and wrist on the index finger. (A) Sensory branches of the ulnar nerve. (B) Palmar cutaneous branch of the median nerve. (C) Sensory branches of the radial nerve. (b) Cross-section of a finger: (1) longitudinal midlateral incision; (2) Bruner's anterior zig-zag incision; the neurovascular bundle is kept in place.

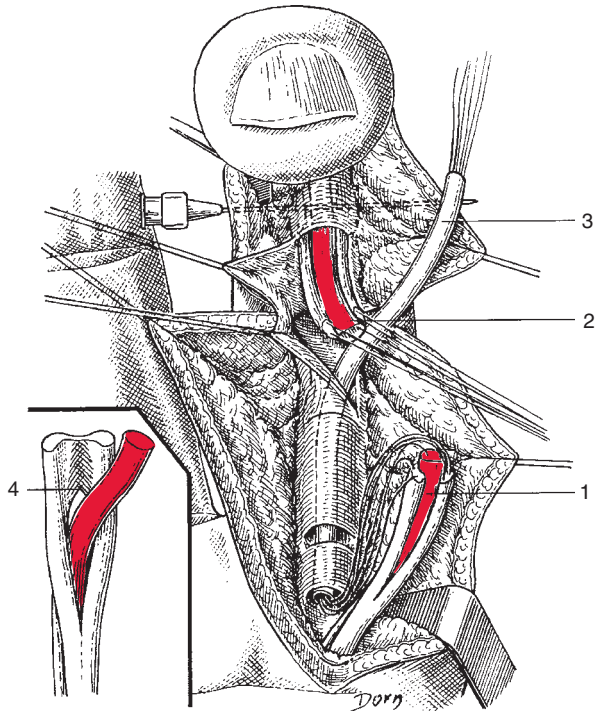


Figure 15.15

Exposure of the flexor tendon extremities. (1) Proximal tendon ends are exposed in the distal palm. (2) Distal ends are exposed by flexing the interphalangeal joints, and are maintained in a window cut in the C1 tendon sheath by a hypodermic needle. (3) A silicone rubber catheter is used as a guide through the pulleys. (4) A silicone rubber catheter is used as a guide through Camper's chiasma.

suture – that grips the tendon at a distance from the cut ends to relive tension, plus a circumferential suture around the edge of the repair to improve the gliding capacity of the repair.

Kirchmayr[53] published in 1917 a technique of suture that was reintroduced by Kessler[54] in 1973 under the term of grasping suture (Figure 15.16b), which presents in each tendon end two longitudinal and one transversal segments. Longitudinal segments follow the longitudinal orientation of the tendinous fibers, and the transverse segments provide anchoring in the tendon at about 1 cm from the end of each tendon. In order to avoid gliding of the transverse thread into the tendon, which may dissociate tendon fibers, a loop grasping a few fibers is necessary at each extremity of the transverse segments; the longitudinal segments should be dorsal to the horizontal one, to lock the suture. Urbaniak et al[55] published results of tensile strength analysis of tendon suturing methods, and found the Kirchmayr–Kessler-type grasping suture to be almost three times stronger than the Bunnell criss-cross stitch 5 days after the repair.

The looped grasping suture (Figure 15.17) introduced by Tsuge[56] offers an easy way of locking the suture.

Wade et al[57] and Diao et al[58] showed that the running circumferential suture not only allows a better gliding of the tendon repair, but also increases the tensile strength. Such a suture may provide a significant reduction of gapping between the tendon ends.

The position of the core suture would preferably be in the volar part of the tendon to minimize damage to the blood supply. However, there are significant strength and biomechanical advantages to dorsal rather than palmar placement of the core suture.[59,60]

For a time, a two-strand grasping suture with a running circumferential suture (Figure 15.12) received the approval of a majority of authors.[22,61] This kind of suture permits an early passive protected mobilization, but does not allow an immediately active rehabilitation.

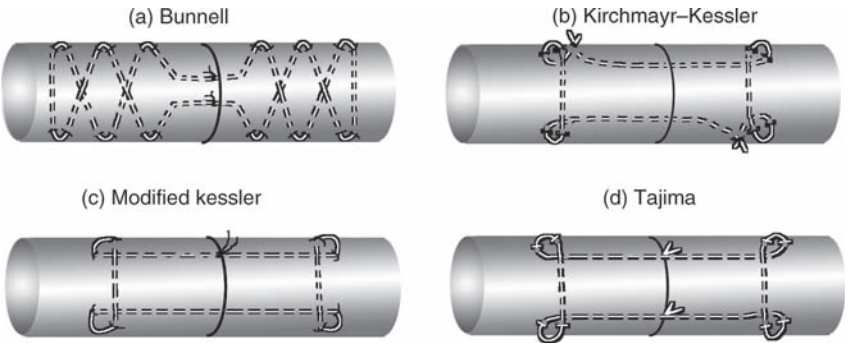


Figure 15.16

Evolution in tendon suture technique: (a) Bunnell's criss-cross suture; (b) Kirchmayr-Kessler grasping suture, with the longitudinal components lying dorsal to the transverse; (c) modified Kessler; (d) Tajima suture using double-armed needles.

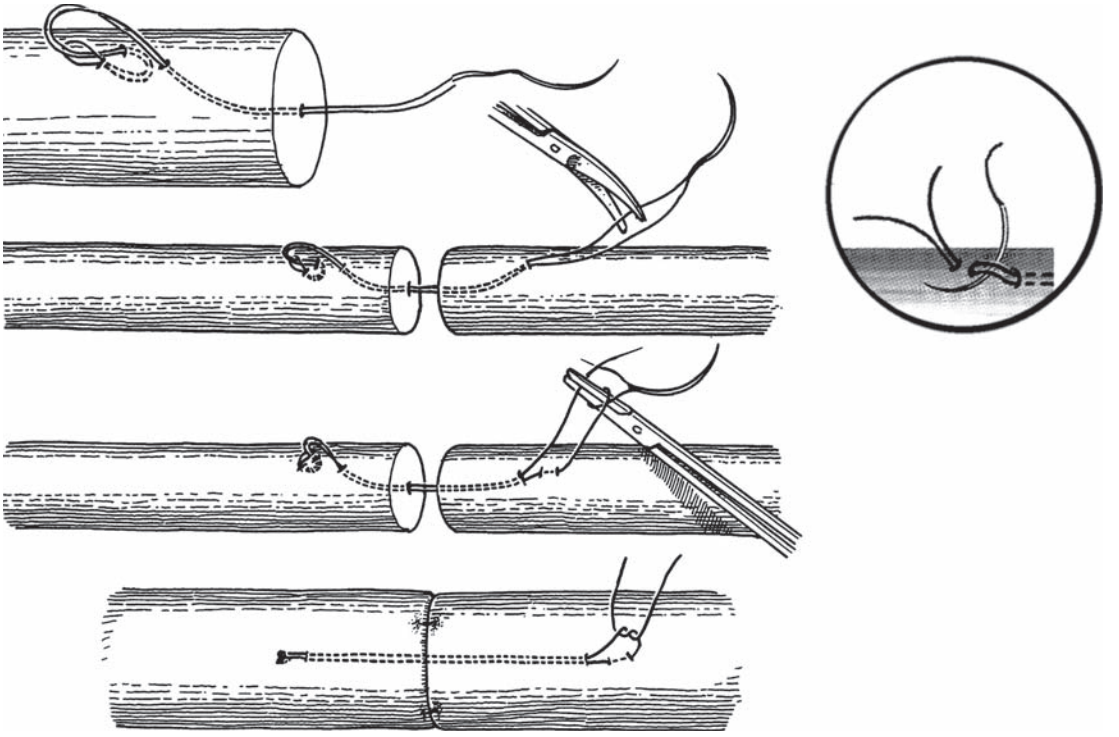


Figure 15.17

Tsuge looped grasping suture.

A tendon suture technique allowing an early active mobilization has been for a long time an inaccessible aim for flexor tendon repair. Early active motion generates a heavy stress on the repair, which has only been estimated recently (Table 15.1).

Surgeons should be using a repair technique that has a strength of about 4–5 kg force.

Numerous clinical and biomechanical researches have been undertaken in order to reinforce the strength of the repair with in vitro and in vivo evaluations (Le Nen et al. 1998).

Clinical investigators admit that the strength of a tendon repair is roughly proportional to the number of suture strands that cross the repair (Lee, 1990). It is now accepted that at least a four-strand core suture is necessary to allow early active mobilization. A six-strand core suture (three double cross-stitches) is used by Savage[62] (Figure 15.18) and even an eight-strand by Winters.[63] However, multistrand repairs are technically difficult, and they may even have harmful effects: the stiffness at the repair site increases with the number of strands, and the nutrition of the tendon may be compromised, causing edema and loss of gliding.

Table 15.1 *Estimated repair strength (core suture)*[52]

	0 week	*1 week (–50%)*	*3 weeks (–33%)*	*6 weeks (+20%)*
Two-strand	1800 g	900 g	1200 g	2200 g
Four-strand	3600 g	1800 g	2400 g	4200 g
Six-strand	5400 g	2700 g	3600 g	6500 g

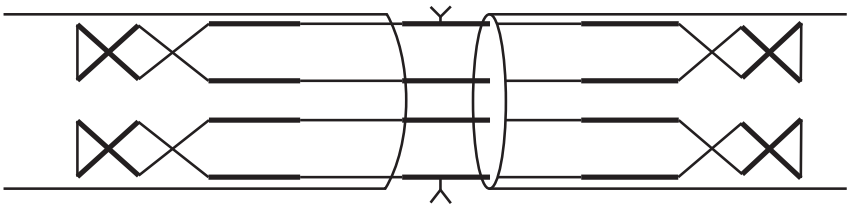
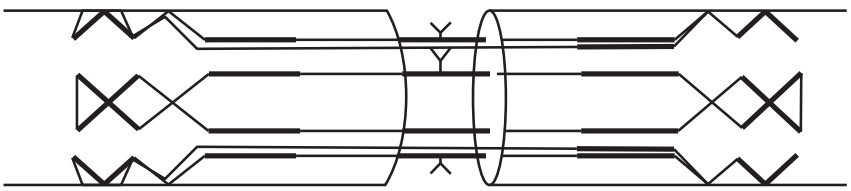


Figure 15.18

Savage's four- and six-strand sutures. The strength of a tendon repair is roughly proportional to the number of suture strands that cross the repair.



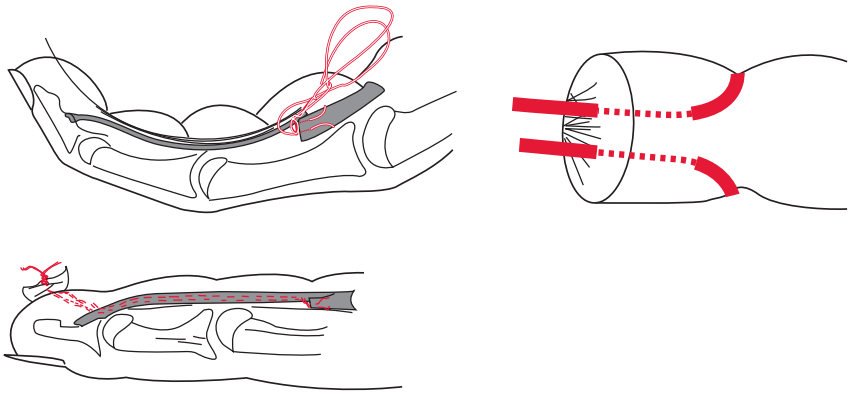


Figure 15.19

Another way of adding strength to a suture is to use a larger caliber of core suture. However, the knot is bulky at the flexor tendon junction. A distal technique of tendon suture fixation avoids the problem of suture knotting within the flexor tendon sheath.

The friction of the tendon within its sheath is increased by the bulk of the repair.

Another way of adding suture strength is to use a larger caliber of core suture, but the knot is bulky at the flexor tendon junction.

Repairs usually rupture at the level of knots.[64]

The fewer suture knots the better: a single-knot suture loop is stronger than a two-knot loop.[50] Pruitt[65] indicated that, whenever possible, it was best to locate knots outside the repair. However, bulky knots left over the tendon can create a conflict with the pulley system, especially if they are in an anterior position. It is preferable to place them laterally (Soejima et al. 1995).

An original technique of tendon suture fixation[66,67] avoids the problem of suture knotting within the flexor or tendon sheath (Figure 15.19). This technique is particularly used in distal flexor tendon repairs, and allows early active mobilization. Many other techniques of tendon suture have been described that allow active mobilization: the Becker bevel technique used when the flexor tendon cut is oblique[68] (Figure 15.20), pull-out wire suturing,[69] the Lee double-loop locking technique,[70] and the Messina double-armed suture.[71,72] Strickland[52] added a horizontal mattress core suture to the Tajima grasping suture with running peripheral epitendinous sutures (Figure 15.21).

In summary, the ultimate force in the suture technique increased significantly when the number of strands increased,

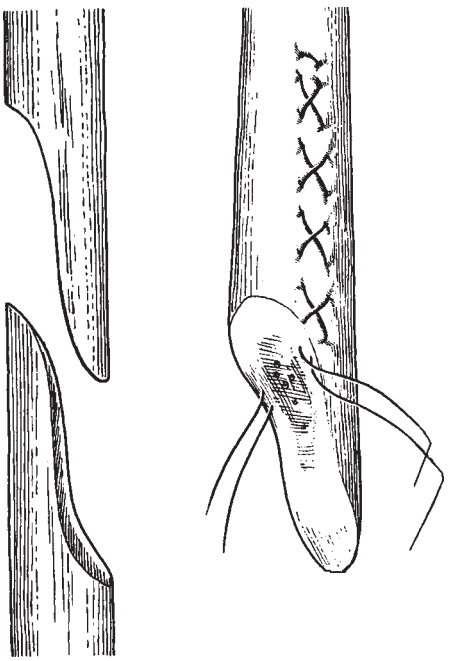


Figure 15.20

Becker's bevel technique.

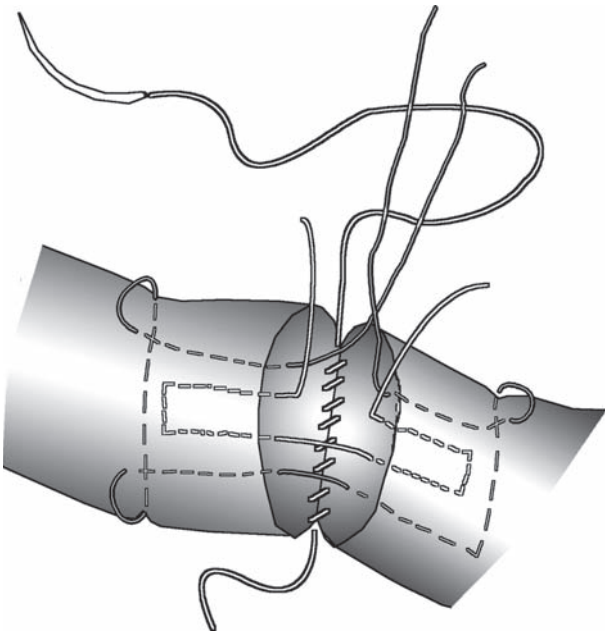


Figure 15.21

Strickland's flexor tendon repair (Indiana method): Tajima core suture with a dorsal running peripheral epitendinous stitch in progress. A mattress suture is added.

or the suture caliber increased. The suture configuration did not influence the ultimate force.[73]

The final sophisticated suture technique consists of two intratendinous stainless steel anchors, which are joined by a multifilament stainless steel suture. However, it seems wise to have more experience with voluminous metallic material before promoting these procedures.

The challenge of finding the ideal tendon suture technique allowing early active motion is not yet solved. We presently favor a four-strand core suture: a double Tsuge looped grasping suture or a double Kessler-type suture with only one knot[74–77] (Figure 15.22), associated with a Silfverskiold–Anderson[78,79] cross-stitch circumferential suture, well suited to keeping the free ends of the tendon from fraying and bulking. Silfverskiold stated that the suture bite should be placed 3–5 mm from the edge of the lesion (Figure 15.23).

The suture technique represents only the first part of the tendon repair; the second part, postoperative management, is essential for the quality of the results.

Suture material

Several new synthetic suture materials are readily available for use in tendon surgery, either non-absorbable (e.g., monofilament nylon) or absorbable sutures. The ideal material should be inert, of small caliber, but strong and pliable. Trail et al[80] have shown that monofilament polyglyconate (absorbable) and stainless steel wire each possess high tensile strength and provide good knot security, whereas braided polyester and polypropylene offer a reasonable compromise. Polypropylene is smooth; polyester in its usual braided form is rough, and so grips tissue well.

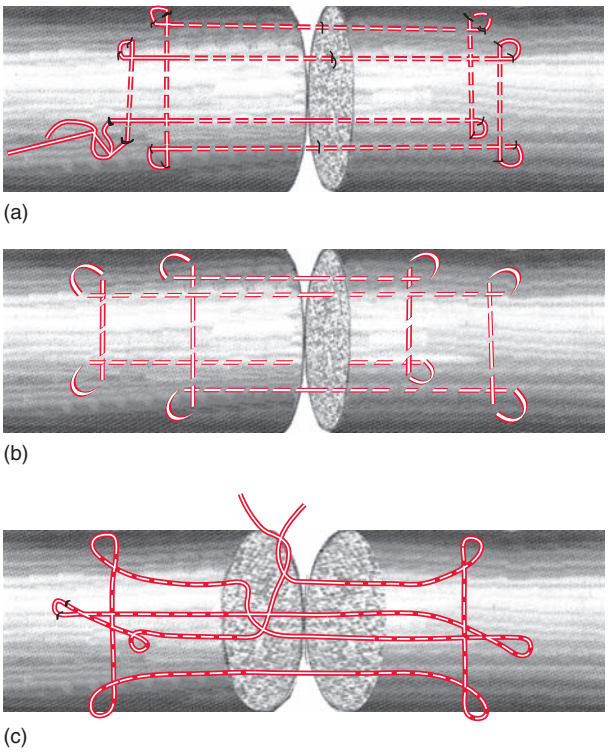


Figure 15.22

Double Kessler-type suture (four-strand) with only one knot. (a) Barrie: the knot is outside the repair. (b) Dubert: the knot is inside. (c) Lenen: a double modified Kessler in two planes with one knot has the best tensile strength because of a better distribution of the strength (in vitro evaluation).

4-0 braided polyester is usually used for the core suture and 6-0 polypropylene for the peripheral suture.

Postoperative management

The benefits of early mobilization have been indicated since Mayer[12] in 1916, but early mobilization was not originally used, because of the fear that tendon junctures did not unite; Bunnell's three-week postoperative immobilization was applied until progress in suture material and suture technique allowed techniques of early protected mobilization.

Everyone now agrees, after the work of Kleinert[22] and Gelberman,[43] that stressed tendons will heal faster and have less adhesions and better excursion than unstressed repairs. Postoperative immobilization is now used only on children and uncooperative patients.

Numerous methods of postoperative mobilization have been described. These methods can be schematically classified into three groups: controlled passive motion, semiactive mobilization, and early active mobilization.

Controlled passive motion

Duran and Houser[81] in 1975 proposed a controlled passive motion for the flexor tendon repair in zone II. A dorsal

splint maintains the wrist in 20° of flexion, the metacarpophalangeal (MP) joints in 60° flexion, and the interphalangeal joints in a neutral position. The proximal and distal interphalangeal (PIP and DIP) joints are mobilized separately (Figure 15.24).

It has been observed that passive MP joint movement produces no relative motion of the flexor tendons. DIP joint motion produces excursion of the FDP of 1 or 2 mm per 10° of joint flexion. PIP joint flexion results in excursion of both FDP and FDS of 1.5 mm per 10° of joint flexion.

Differential excursion between the two digital flexors is increased by positioning the wrist in flexion or extension, using after the third postoperative week a dorsal splint with a hinge allowing flexion or extension of the splint: the Mayo 'synergistic splint'[82] or the Strickland 'tenodesis splint'[83] (Figure 15.25). The wrist should be positioned in about 35° extension, with MP joints flexed to 90° to minimize the force required to achieve or hold full active composite digital flexion. The fingers extend within the restriction of the splint, with the wrist in 20° flexion.

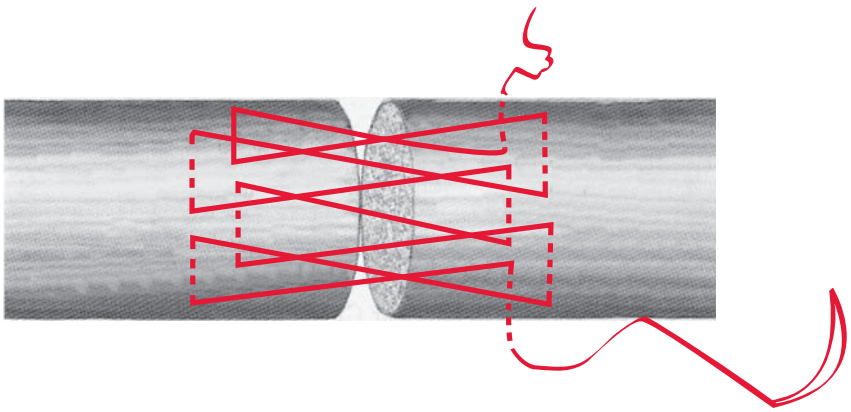


Figure 15.23

Silfverskiold cross-stitch circumferential suture.

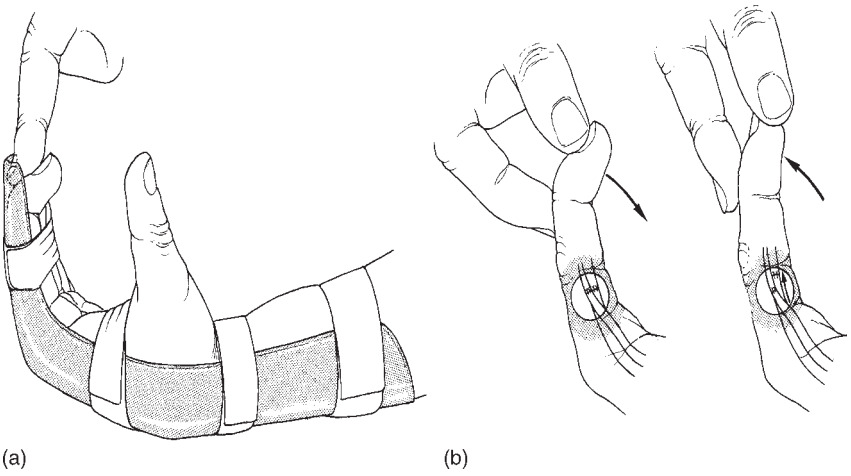


Figure 15.24

Controlled passive motion – Duran and Houser. (a) DIP joint motion produces excursion of the FDP of 1–2 mm per 10° of joint flexion. (b) PIP joint flexion results in excursion of both FDP and FDS of 1 mm per 10° of joint flexion.

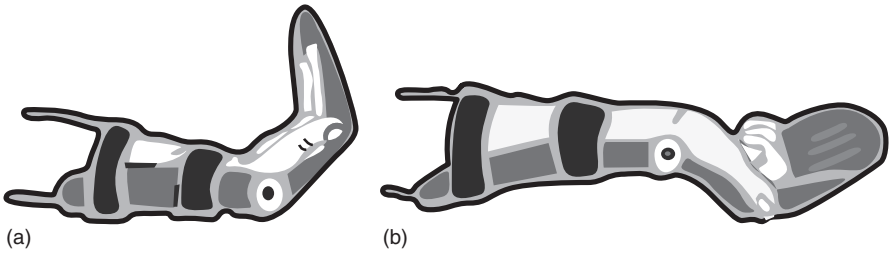


Figure 15.25

The Strickland hinged wrist splint or tenodesis splint. (a) Within the restraints of the splint, the patient passively flexes the digits while simultaneously extending the wrist. (b) As the patient allows the wrist to drop in flexion, the digits extend within the restriction of the splint (tenodesis effect).

Semi-active mobilization

Kleinert[22] developed a technique of protected mobilization consisting of active extension and passive flexion of the fingers. Rubber bands were fixed to the nails and coupled to the use of active extension against a limiting dorsal block splint (Figure 15.26a). Since then, rehabilitation techniques have evolved. The original Kleinert rubber bands splinting, although it has been a great improvement over Bunnell's 3 weeks of immobilization, may cause some problems in terms of achieving poor DIP joint flexion and extensor tendon tethering.

Chow[84] used rubber band traction incorporating a palmar pulley at the distal palmar crease level, which increases the tendon excursion in the sheath and the differential gliding between superficialis and profundus tendons (Figure 15.26b).

One problem that is often overlooked is that of dorsal joint tightness. These dorsal problems are the most common cause of long-term loss of full flexion with loss of grip strength.[85] In any splinting programs, the interphalangeal joints should be kept in extension with straps between the exercise sessions and at night, to minimize the likelihood of flexion contracture developing.

The protecting dorsal splint is retained during 6 weeks; return to usual activities is possible after 8 weeks, but heavy manual work only after 12 weeks.

Postoperative early active mobilization (POEAM)

As it is now established that early motion stress has the potential benefit to facilitate healing and to increase tendon excursion by limiting the amount of peritendinous formation, active flexion and extension exercises seem to be the best postoperative method of rehabilitation[68,86] when there are no contraindications.

A dorsal splint on the wrist and fingers, similar to the one used for controlled passive motion, is placed to protect the repair. About eight sessions of finger joint mobilization (both passive and active) are programmed every day. POEAM may start using the technique of 'place and hold'. Savage et al[87] have described a method of reducing flexor tendon force during active movement by holding the proximal phalanges of the uninjured fingers more extended than the operated finger, whose MP joint is kept in variable degrees of flexion. As it is not possible for the physiotherapist to be present at each session, the patient is taught, using the other hand, to perform the exercises himself. Frequent control by the therapist is essential. Results are more rapid when the collaboration of the patient is a motivating factor.

The tendon rupture rate in some centers is no higher for controlled active motion than in series using passive or semi-active methods of rehabilitation: 3.8% for Bellemere et al[88] and 5% for Elliot et al[89] However, the rupture rate is much higher for Peck et al.[90]

It is important to realize that early active mobilization cannot be prescribed after all flexor tendon repairs.

Some authors prefer that POEAM should be preceded by a period of passive mobilization, started 1 day after surgery in order to reduce edema and internal resistance.[91] The length of this period of passive rehabilitation can be extremely short (1 or 2 days when all conditions are favorable) or much longer, and is influenced by many factors:

- The necessity for a specialized therapist.
- An intelligent and motivated patient – POEAM requires the greatest level of patient comprehension.
- Tendon lesions: clean-cut division, and early repair are favorable factors. When the vascularization of the tendon ends is jeopardized, it is safer to use an early passive rather than an early active mobilization. For instance, cases with important retraction of the proximal tendon stump and devascularization should be mentioned to the therapist, because tendon healing takes longer.
- Quality of the repair: the suture technique must have the tensile capability of withstanding the forces involved with active muscle contraction during the early weeks of healing. Unknown quality of surgery or late referral contraindicate early active mobilization.
- And, of course, the site of the repair.

Site of repair

When the flexor tendon repair is in zone I, III, IV, or V, POEAM can be started very early, with extension of the finger being limited by a dorsal splint. In zone II (the digital canal), early active mobilization is also desirable, but the indications are limited by the factors already mentioned.

Although early active mobilization is now commonly used in many European and North American specialized hand centers, protected passive mobilization of the flexor tendon is still widely used, because in most countries, most

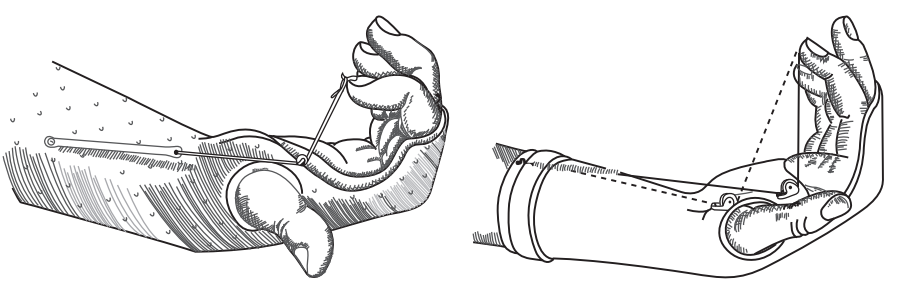


Figure 15.26

Semi-active mobilization. (a) Kleinert's 1967 postoperative dynamic splint.[22] (b) In 1988, Chow added a palmar pulley at the distal palmar crease level.[84]

acute flexor repairs are still done by trainees or surgeons not specialized in the hand.

What is essential for flexor tendon management is not only to have specialized hand surgeons and specialized therapists who understand tendon wound healing, but also they must be educated to collaborate on planning therapy.

Surgical indication in flexor tendon injuries

We shall now try to precisely define respective indications for tendon sutures, tendon grafts, and some other procedures, taking into account the following factors: the timing of the tendon repair, the existence of associated lesions, the characteristics of the patient, the surgical conditions, and the location of the lesion.

Timing of the repair

Indications for surgery are influenced by the time interval between the injury and the repair. One can distinguish early primary, delayed primary, and late repairs, but we must define what these terms mean, as they vary from one author to another. The Committee on Tendon Injuries of the International Federation of Societies for Surgery of the Hand (IFSSH) has adopted the following definitions:[5]

- primary repair: repair done within the first 24 hours following injury
- delayed primary repair: repair between 1 and 14 days following injury
- secondary repair: repair done after the second week following injury; early secondary repair is undertaken between the second and fifth weeks, and late secondary repair is done after the fifth week.

It seems to us that the distinction made between delayed primary repair and early secondary repair is quite arbitrary, because apposition of the tendon ends without undue tension is sometimes possible several weeks after tendon division.

Primary repairs

Contrary to Bunnell's advice to perform a secondary repair in the digital canal, flexor tendon injury is now considered a surgical emergency at all levels. The decision as how to manage the tendon will depend primarily on the state of the wound. It is important to stress the limits of primary suturing. The quality of the skin cover plays a large part in this decision. One will attempt a tendon repair only if the wound is likely to heal primarily. This precludes primary flexor tendon repairs in contused wounds. Contaminated wounds, especially bites, run a high risk of becoming infected, and are a definite contraindication to primary repairs. Lesions of both neurovascular bundles of a finger, edema that restricts passive flexion, unstable fractures, articular damage, and inadequate surgical facilities are contraindications to primary suturing.

In this early phase, the tendon retraction is not fixed, and suturing of the tendon ends is possible if there is no loss of tendon substance. Primary tendon suturing is therefore often feasible.

For a long time, great importance was attached to the time elapsed between injury and repair. Six hours was the limit, beyond which the wound was regarded as inevitably infected and no tendon repair would be attempted. Marc Iselin[92] showed that delay in repair could be safely prolonged, provided that the wound was clean, the hand immobilized, and the patient placed on antibiotics. This concept of 'delayed emergency' has been put into practice when surgical conditions were not initially favorable.

Surgical facilities and the experience of the surgeon have a marked influence on the results of tendon surgery. They are often inadequate at the time of primary repair. The requirements are a fully equipped aseptic operating room (like that used for bone surgery), an adequate instrumentation, and a surgeon experienced in tendon surgery.

When surgical conditions are favorable, primary suture should be done as soon as possible.

Secondary repairs

Secondary repair is carried out when the injury is seen late or when primary repair was not possible. Secondary suture is possible when the wound is clean as a result of previous debridment, apposition of the tendon ends is possible without tension, and there is adequate soft tissue coverage.

Secondary suture can be done within several weeks following the injury. This is facilitated if, immediately following the injury, the wrist and MP joint have been maintained in flexion, which limits the proximal retraction of the tendon and the destruction of the vincular network.

In practice, secondary suture of flexor tendons distal to the wrist can be carried out within 3–5 weeks in adults, and even later in children.

When the proximal tendon end is retracted in the forearm, shortening and scarring will make tendon suturing difficult. Some authors have found the results of secondary repair to be as good as those of primary tendon repair.[56,93] However, for most authors, the results following secondary suture are less favorable,[94] and POEAM is contraindicated.

Late repairs

After 5 weeks, retraction has become fixed, and a tendon graft is usually indicated.

Location of the lesion

Division in the distal zone

The flexor digitorum profundus alone is divided

Numerous types of repair can be used. Some aim at restoring tendon function, while others just attempt to stabilize the distal interphalangeal joint.

Restoration of tendon function can be obtained by reinsertion, suture, or grafting. Reinsertion is possible when the division of the FDP lies within 1 cm of the insertion. One should guard against excessive traction, which may result in residual fixed flexion, and can even restrict flexion of the profundus tendons of the other fingers, according to Verdan's 'syndrome of quadriga'.[95,96] It is preferable to suture the FDP tendon than to reinsert it with excessive traction.

Avulsed tendons should also be reattached whenever possible.

Tendon-to-bone repair can be performed using different techniques:

- Bunnell's pull-out wire[97] through the distal phalanx and the nail has been the standard technique for a long time. Marin Braun and Foucher[98] used barbed wire for such repairs.
- An anchor suture (Mitek) reduces the risk of infection from a transcutaneous device and avoids the risk of nail dystrophy.[99,100]

When the treatment has been delayed, the edematous tendon can be too swollen to pass freely through the A4 pulley. One-half of the tendon is excised longitudinally in the midline to allow tendon repair or reattachment. The 'double-barreled' configuration of the distal part of the FDP tendon facilitates the procedure.[101]

Grafting of the flexor profundus in the presence of an intact flexor superficialis can be considered in some favorable cases. The graft should be thin (plantaris) and should preferably be placed alongside the superficialis than through it within the fibrous sheath,[19,20] although good results can be obtained by a graft through the intact superficialis tendon.[102,103] A staged superficialis tendon reconstruction with silicone implant and secondary tendon grafting is possible.

Division of the flexor pollicis longus (FPL) tendon in the distal zone

The FPL can be advanced further than the flexor digitorum profundus because of the independence of the tendon and the absence of lumbrical muscle. The tendon can be surgically divided obliquely at the level of the musculotendinous junction, allowing lengthening of the tendon up to 2 cm[104] (Figure 15.27).

Stabilization of the distal joint, without tendon repair, on the long fingers or on the thumb can be obtained by tenodesis or arthrodesis.

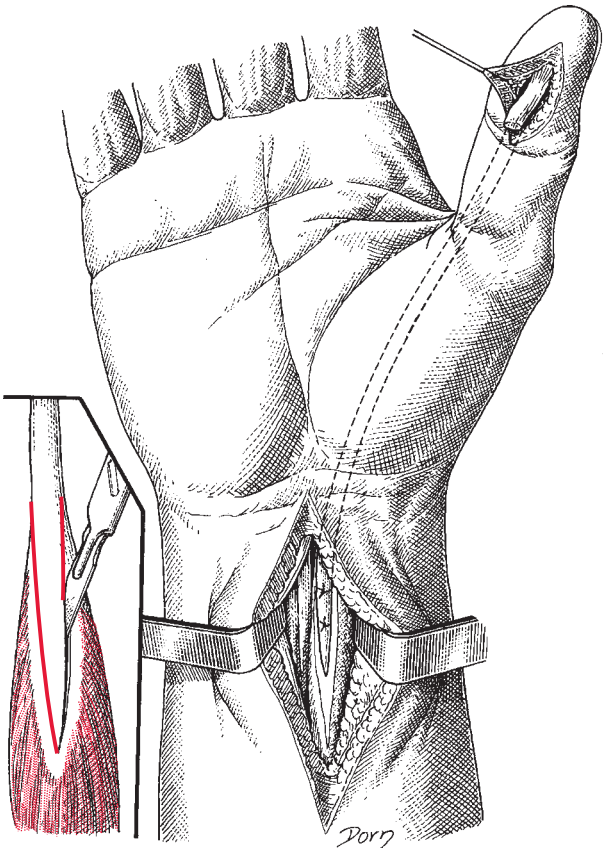


Figure 15.27
Lengthening of the FPL tendon (Rouhier's technique).

Division in the intermediate zone (digital canal)

In Bunnell's 'no-man's land', a single-stage tendon graft was the rule for a long time. Secondary single-stage tendon grafts are now rarely performed, because the operation is quite complex and single-stage grafting gives its best results when the digit is in good condition, with an adequate passive range of motion, a preserved pulley system, and at least one digital neurovascular bundle intact. In these ideal conditions, primary suture also offers the possibility of a good result in a shorter time. Repairing both tendons (FDP and FDS) better maintains the vascularity provided by the vincula, and assures independent flexion of each phalanx and greater flexion strength (Figure 15.28). The FDS slips should be repaired first. Careful attention must be made to their orientation. These thin slips require mattress-type sutures. More proximally, as the caliber of the superficialis tendon increases, a standard core suture can be used. Resection of the flexor superficialis tendon in order to have more room inside the digital canal would destroy the vinculum longum, thus increasing problems of tendon healing. However, resection of a slip of superficialis may facilitate gliding of the profundus.

When primary tendon suture is not possible, a secondary suture can sometimes be done if the local conditions have

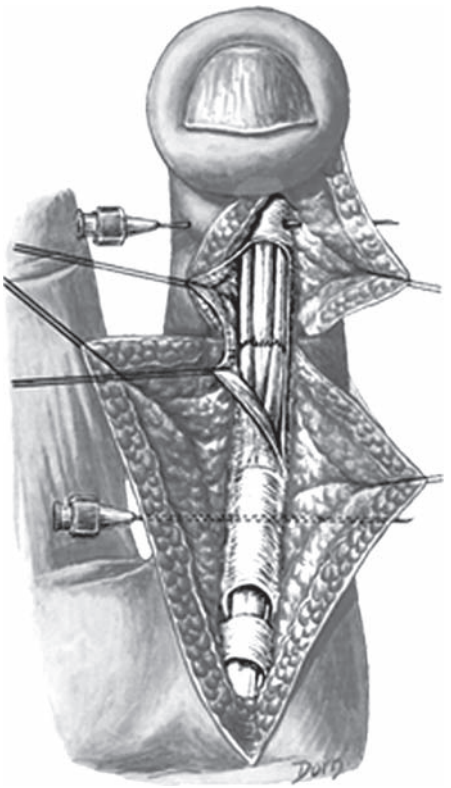


Figure 15.28
Flexor tendon repair in zone II.

turned favorably. However, late flexor tendon suturing with the proximal tendon stump retracted in the forearm often gives only poor functional results. A flexor tendon graft seems to be indicated in most cases.

Is there still a place for the single-stage tendon graft?

Staged reconstruction as developed by Hunter[105] currently enjoys the greatest popularity, but takes a very long time. It seems to me (maybe it is just a nostalgic reminiscence?) that the single-stage Bunnell flexor tendon graft still has a place when the tissues are supple and the flexor tendon sheath is functional with at least A2 and A4 pulleys (Figure 15.29). Recent improvements in tendon suturing and postoperative early passive mobilization should be used.

Two-stage tendon graft

The concept of using foreign material implants to induce the surrounding scar to form a pseudosynovial sheath was already investigated experimentally by Mayer[106] in 1936 with celloidin tubes, but he was unable to find a flexible inert material. Carroll and Basset[107] used silicone rods to induce pseudosheath formation. Silicone is chemically inert and flexible; however, it lacks strength and tear resistance. Since 1960, Hunter has pursued research on damaged flexor tendon mechanism replacement using passive or active

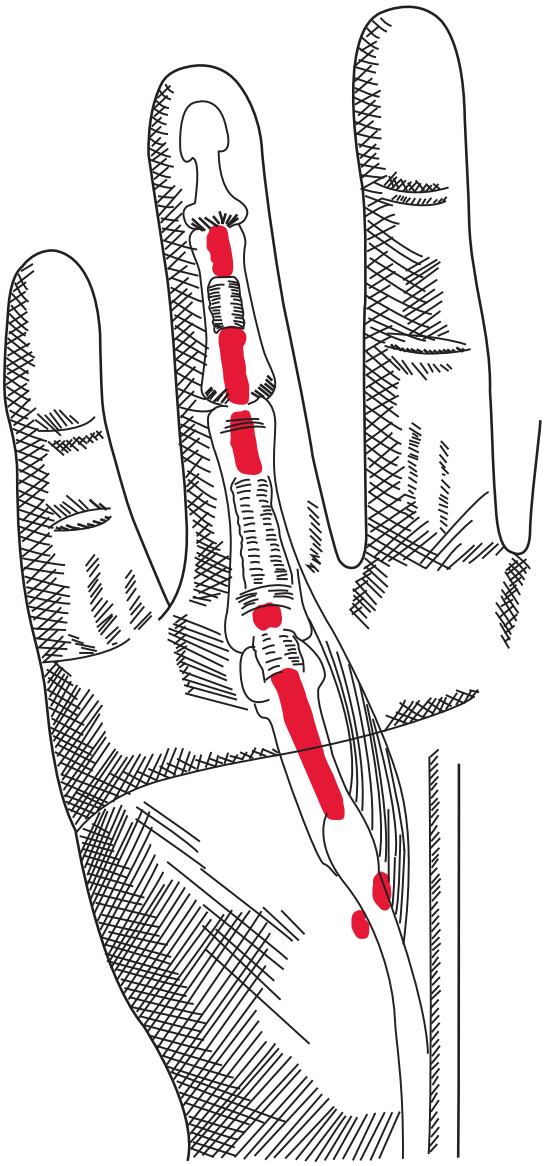


Figure 15.29
Single-stage flexor tendon graft (Bunnell): there is a single graft from the lumbrical to the digital extremity, a Pulvertaft 'fish-mouth' suture between the flexor digitorum tendon and the graft, and a distal pull-out fixation of the graft.

tendon implants, followed secondarily by a tendon graft. He developed the two-stage tendon graft procedure, which involves:

1. Preoperative re-education to achieve maximal passive mobility of the joints and suppleness of soft tissue.
2. Excision of the damaged flexor tendons and of fibrous tissue, and insertion of a silicone rod (a woven Dacron core molded into silicone rubber) (Figure 15.30).

   Pulley reconstruction is done at this stage, and constitutes a major step in flexor tendon repair. Various techniques can be used. The insertion band of the FDS at the level of the middle phalanx can reconstruct the A4 pulley (Figure 15.31a). A palmaris longus or a plantaris

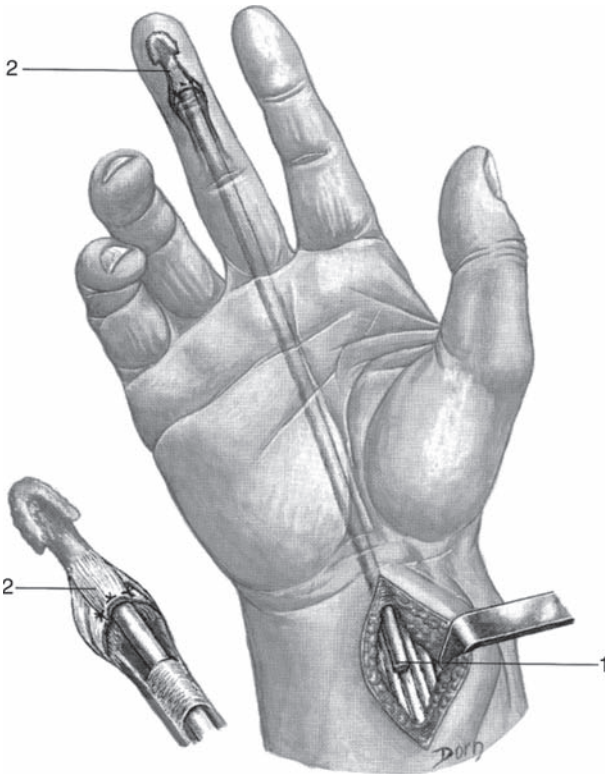


Figure 15.30

Flexor tendon reconstruction (Hunter), stage 1. Complete passive mobility must be recovered with excision of the damaged flexor tendons, a 1 cm stump of distal profundus tendon is preserved. The graft bed is prepared, and an appropriately sized silicone implant is sutured to the superficialis in the palm and brought proximally to the forearm through the carpal tunnel. (1) Proximal free extremity of the silicone implant. (2) The distal extremity is strongly sutured to the distal FDP stump.

tendon graft can be wrapped several times around the implant. For Kleinert,[108] weaving a tendon through 'the always present fibrous rim of the previous pulley' affords a good control of the tension. Lister[109] advises the use of retinacular tissue, which is similar to flexor tendon sheath and seems preferable to a tendon graft for pulley reconstruction. A strip 8 cm long and 1 cm broad may be taken from the extensor retinaculum (Figure 15.31b). Retinacular tissue may also be taken on the dorsal aspect of the foot. Nishida et al[110] reported that of all the sheath restoration methods, the use of the extensor retinaculum creates the least resistance to tendon gliding. The strip of retinacular tissue is passed around the phalanx and the tendon, deep to the extensor tendon over the proximal phalanx. It is passed superfical to it over the middle phalanx (Figure 15.31b). At least A2 and A4 are necessary, but better mechanics will be restored if more pulley support is supplied.[111]

3. After about 3 months, a long graft (plantaris or long toe extensor) is placed in the fibrous sheath formed around the implant (Figure 15.32). The graft is sutured to the proximal end of the implant in the distal forearm and pulled through the carpal tunnel, the palm, and the digital canal. Usually, the FDP is used as the motor. The distal extremity of the graft is sutured to the distal FDP stump.

Clinical outcomes associated with flexor tendon repair

Repair rupture, adhesions, and joint stiffness may require secondary operation. Extensive soft tissue injuries and long

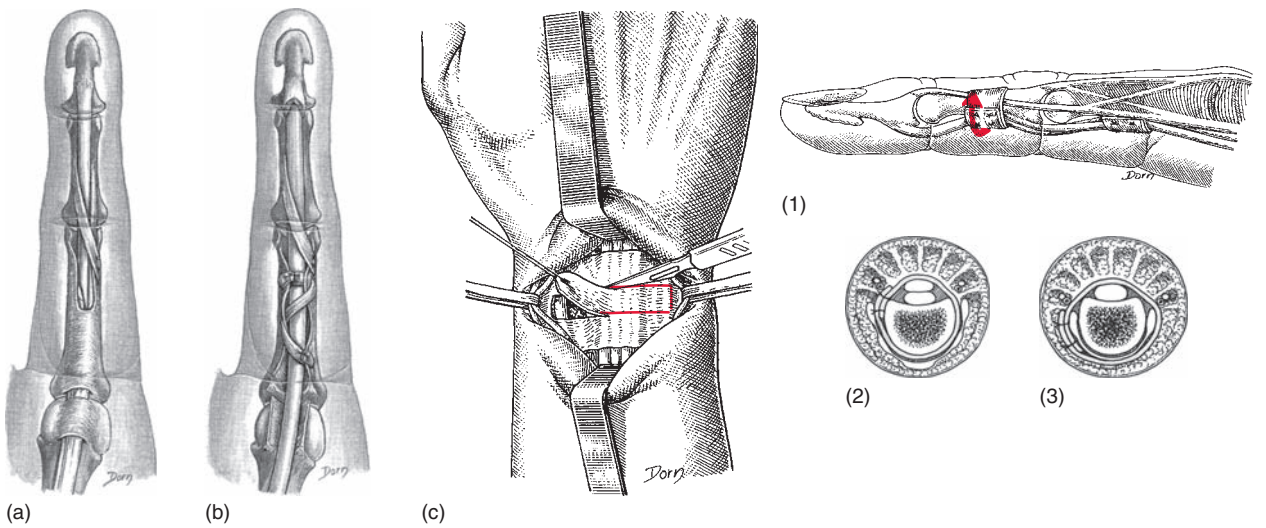


Figure 15.31

Pulley reconstruction – various techniques. (a) Insertion band of the FDS. (b) Tendon graft. (c) Lister's technique: (1) A strip is taken from the extensor retinaculum. (2) The strip is passed around the proximal phalanx, deep to the extensor tendon, for reconstruction of the A2 pulley. (3) The strip is passed around the middle phalanx, superficial to the extensor tendon for the reconstruction of the A4 pulley.

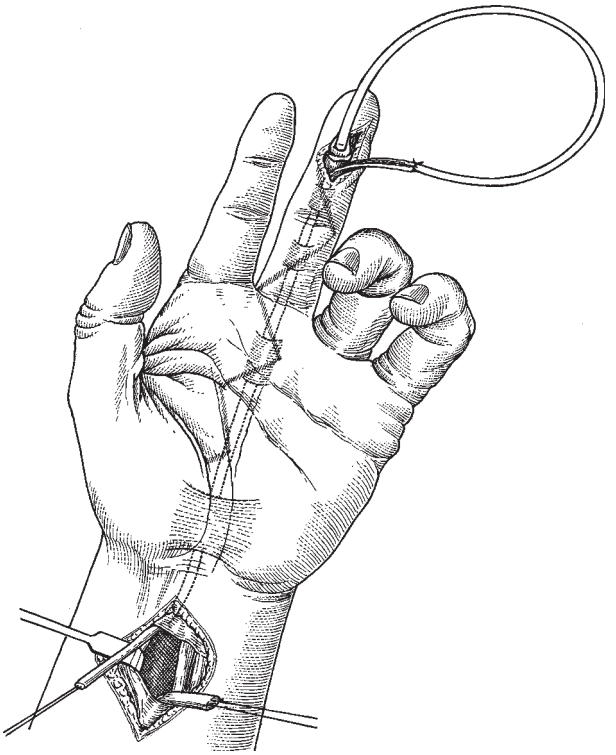


Figure 15.32

Hunter staged reconstruction – stage 2. A long tendon graft (the plantaris or the long extensor of one of the three central toes) is sutured to the proximal end of the implant and pulled through the carpal tunnel, the palm and the digital canal.

duration of surgery contribute to postsurgical edema. A bulky tendon increases its friction against the pulleys. A forceful pull to overcome the resistance may lead to rupture of the repair.

Misuse of the repaired fingers is another cause of rupture. Postoperative active motion must be properly applied and used with protection. If rupture occurs soon after the repair, a direct resuture may be attempted.

Dense adhesions may arise from the synovial sheath, from the pulleys or from the bony floor. Also, stiffness of the DIP and PIP joints is frequently observed. An appropriate rehabilitation program may prevent these complications, and must always be carried out before any secondary operation.

Salvage procedures

Several salvage procedures for secondary reconstruction have been described.

Pedicle tendon graft

Paneva-Holevich's pedicle tendon graft,[112] involves the use of the superficial flexor tendon of the respective finger as a pedicled graft. In the first stage, the FDP tendon is divided in the palm at the lumbrical muscle level. The FDS is cut 3 cm more distally, and the cut ends of the proximal stumps of the two flexor tendons are sutured end to end. Paneva-Holevich[113] has since added the excision of the digital portion of the two flexor tendons and the introduction of a silicone rod, according to Hunter's technique (Figure 15.33). Three months later, the FDS tendon is identified in the forearm and divided at its musculotendinous junction. The FDS pedicle graft is rotated distally, and is temporarily sutured to the proximal end of the implant and then pulled through the digital canal. Its distal extremity is sutured to the distal FDP stump.

The superficialis finger

The concept of the superficialis finger, initially described by Osborne,[114] has been suggested as a means of salvage for the finger affected by severe adhesions or failed tendon graft procedures, often associated with bone and neurovascular injury. In these situations, a more limited goal of a one-tendon, two-joint flexor system is justified to restore motion to the MP and PIP joints, with fusion of the DIP joint. The indications for the superficialis finger operation are an inadequate DIP joint caused by trauma, arthrosis, or extensor insufficiency, and multiple prior failed procedures on the flexor tendon system. A staged superficialis tendon reconstruction with silicone implant and secondary tendon grafting is performed, associated with arthrodesis of the DIP joint.

Composite grafts of tendons and sheath

Peacock and Madden[115] conceived the procedure of transferring both flexor digitorum tendons with their intact synovial sheath in order to eliminate adhesions. This includes not only the entire fibrous flexor sheath anteriorly, but also all the phalangeal periosteum and the volar plates of the interphalangeal joints posteriorly. This scientifically interesting procedure has been successfully tried by Hueston,[116] but seems of little practical use.

Transplantation of a vascularized tendon with microsurgical reattachment

The tendon graft can be taken from the back of the foot.[117] Guimberteau[118] described a salvage procedure in secondary flexor tendon repair, using a vascularized flexor tendon with its surrounding sheaths to form a sliding unit. He usually uses the flexor superficialis of a ring finger, with its surrounding mesotendon and its vascular branches from the ulnar artery. The flexor tendon graft is divided at the musculotendinous junction and at the decussation level, and is translated, after section of the ulnar vessels pedicle in the forearm, as an island retrograde tendon transfer. The transplant is inserted in zones I and II of the damaged flexor tendons in another finger (Figure 15.34).

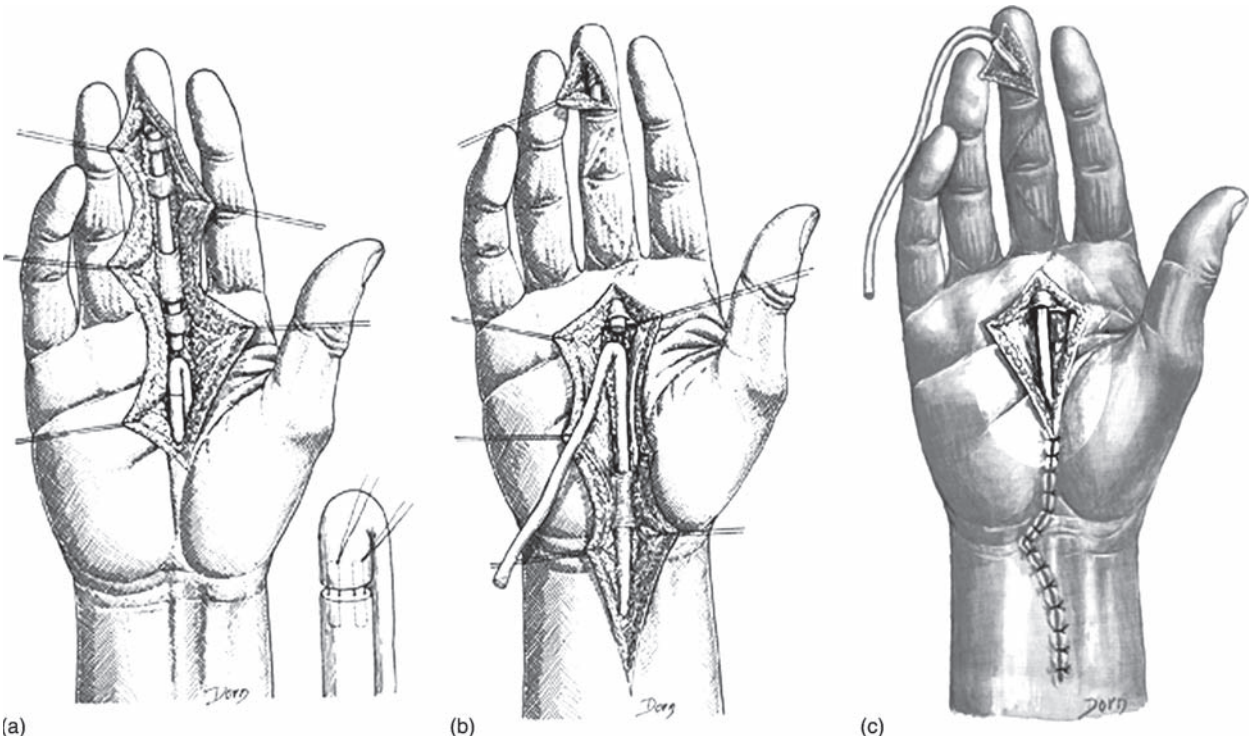


Figure 15.33

Pedicle tendon graft (Paneva-Holevich[113]). (a) Suturing of the proximal stumps of the FDS and FDP; implantation of a silicone rod. (b) The pedicle graft (from the FDS) is divided at its musculotendinous junction and rotated distally. (c) The graft is sutured as the proximal end of the implant and pulled through the distal canal.

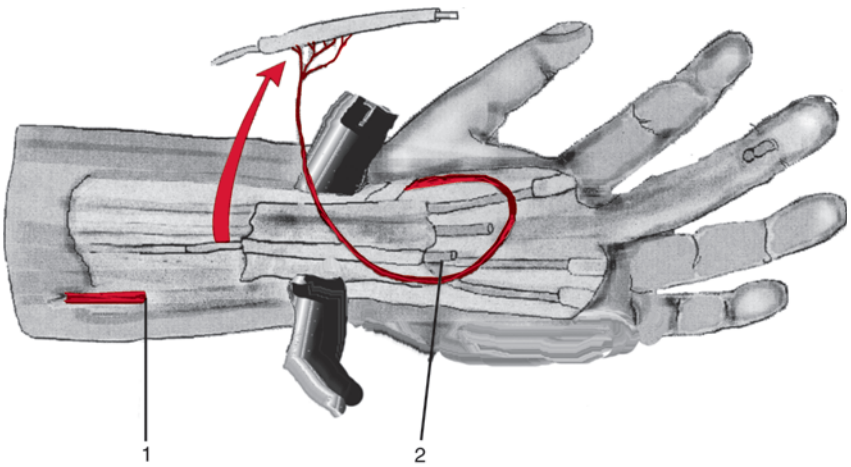


Figure 15.34

Free transplantation of flexor superficialis – Guimberteau procedure. (1) Section of the flexor superficialis of the ring finger at the level of the decussation and at the tendinomuscular junction. (2) Proximal section of the ulnar vascular pedicle; raising of the island tendon transfer. The transplant will be inserted on the long finger.

Division in the proximal zone

Although the flexor tendons cross three different topographical regions (zones III, IV, and V), the anatomic conditions are usually suitable for a primary repair. Suturing of both the profundus and the superficialis tendons is often possible.

On the anterior aspect of the wrist and distal forearm tendons, vessels and nerves are superficial and unprotected by the skeleton; this explains the high incidence of associated lesions. When local conditions are favorable, vessels, tendons, and nerves should be repaired.

An early secondary repair of the tendons and nerves is indicated whenever doubt persists as to the degree of asepsis of the initial wound.

Tenolysis

When the repaired tendon is capable of active mobilization, but active flexion is much more limited than passive flexion, adhesions or inadequate pulley function may be the cause. Tenolysis of adherent flexor tendon repairs or grafts is an excellent procedure when the indications and

the local conditions are adequate. However, it is a demanding procedure: the operative procedure must be very meticulous, and close cooperation is necessary between the surgeon, the therapist, and the patient for the postoperative mobilization program. Tendons of poor integrity have an increased likelihood of rupture, and require protective splinting and a controlled range of motion program.

The regional anesthesia technique at the wrist level[119] permits the patient to actively cooperate during the operation.

It is safer to wait 8 months after the repair before re-operating, on a well-motivated patient who presents with soft and supple tissues.

Total anterior tenoarthrolysis (TAT)

This procedure has been described by Saffar and Rengeval[120,121] for the treatment of severe contracture of the finger after flexor tendon surgery. It consists of detaching 'en bloc' the flexor apparatus and the volar plates of the interphalangeal joints from the underlying skeleton. The sliding of the soft tissue allows straightening of the finger. This procedure can only be used if there is good vascularization and innervation of the finger, with articular cartilages in good condition and tendons that are still working; it is a change of the active range of motion into a more functional arc.

A midlateral approach on one side of the finger is performed, extending from the digitopalmar skin crease to the end of the distal phalanx. At the base of the finger, the incision extends obliquely in the palm or follows the digitopalmar skin crease (Figure 15.35a). The neurovascular bundles are protected and the subcutaneous fibrous tissue is cut transversally. The periosteum of the two phalanges is incised on their lateral sides, and with an elevator the periosteum on the anterior surface of the two phalanges is detached. The PIP volar plate is in continuity with the raised periosteum, and the volar fibers of the accessory lateral ligaments are cut. In this way, the volar plate is freed from its bony attachments (Figure 15.35b). If necessary, the lateral ligaments are progressively sectioned under direct vision while trying to preserve their proximal part. The same step is carried out at the DIP joint, allowing straightening of the finger (Figure 15.35c). It is sometimes necessary to continue the incision around the pulp.

The sliding is in proportion to the severity of the 'hook finger'.

Palliative procedures

Arthrodesis of the distal phalanx can be used in some cases to stabilize the pinch when only the flexor superficialis is active.

Tendon transfers using the FDS from an intact finger are rarely indicated, except for damage beyond repair of the FPL. However, if multiple digits are involved, one can consider transferring the proximal end of a divided tendon for another digit, if the level of the injury is appropriate.

Tendon prosthesis: Hunter has fixed the two ends of his Dacron silicone implant, the distal end in the distal phalanx and the proximal end in the motor tendon. This prosthesis is still temporary. It permits not only the new sheath for the secondary graft but also active flexion of the finger and continued function of the motor muscle.

Conclusion

Surgical indications for flexor tendon injuries are complex, and surgical repair is often technically difficult. Flexor tendon repair should be done in hand centers by expert surgeons and therapists.

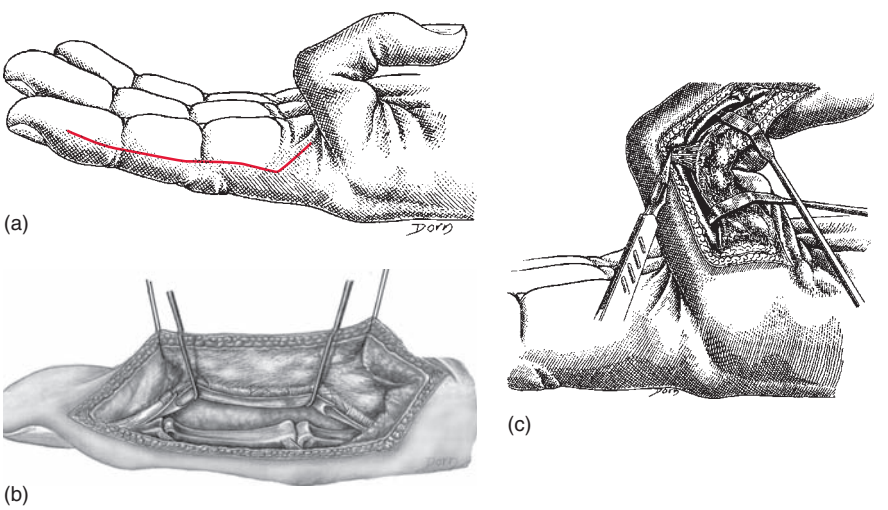


Figure 15.35
Total anterior tenoarthrolysis. (a) Total anterior tenoarthrolysis: incision. (b) The volar tissues slide along the skeleton; it is not always necessary to free the DIP joint. (c) Periosteal incision on the lateral sides of the proximal and middle phalanx; the ligaments of the PIP joint are cut under vision.

Loss of flexion of the digits in rheumatoid arthritis

In rheumatoid arthritis (RA), digital flexion can be impaired or lost due to flexor tenosynovitis, digital joint lesions, or tendon ruptures.

Flexor tenosynovitis

Flexor tenosynovitis results from a proliferation of tenosynovium around the flexor tendons at the wrist, palm or digit (Figure 15.36).

At the wrist level, flexor tenosynovitis results in compression of the median nerve, and, in addition, in adhesions of the flexor tendons, which often decrease active flexion of the digits.

At the hand level, flexor tenosynovitis impedes normal tendon excursion within the flexor tendon sheath. These tendon synovial sheaths are frequently involved in the inflammatory process, producing distention of the surrounding pulleys, which normally maintain the flexor tendons applied on the skeleton. Tendon invasion and adhesions often restrict tendon excursion and result in inability to actively flex the digit. There is an important difference in the active and passive range of motion, which is an important diagnostic point. Sometimes, a flexor nodule snaps against the edge of the annular pulleys and the digit may be locked in flexion.

Digital joint lesions

As a result of RA, digital joints can develop laxity and/or, at a later stage, stiffness.

At the MP joint

The capsule becomes distended by synovial proliferation. The distention of the sagittal bands and of the accessory collateral ligaments allows a volar displacement of the volar plate, which is fixed to the base of the proximal phalanx. The collateral ligaments are also stretched by subchondral erosions on the metacarpal head. Loss of capsular ligamentous support is the primary phenomenon that allows the development of two deformities: subluxation of the MP joint and ulnar deviation of the fingers. At the point of entry in the fibrous flexor sheath, the flexor tendons exert a traction force in two directions: one in a volar direction and the other in an ulnar direction for the index and middle fingers (Figure 15.37).

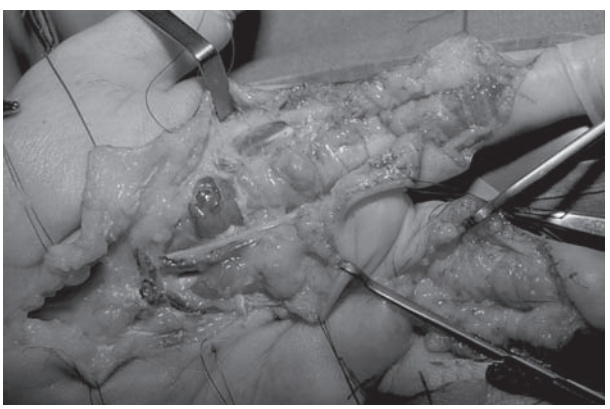


Figure 15.36
Flexor tenosynovitis.

(a) With distention of capsuloligamentous support, the base of the proximal phalanx subluxes volarly under the double action of the long flexor tendons and the intrinsic muscles. This subluxation increases the traction of the extensor tendons and limits the flexion of the finger.
(b) The index and middle finger flexor tendons exert an ulnar traction at the point of entry into the fibrous flexor sheath, which has no effect on a healthy MP joint, as it is applied to the metacarpal head. When the proximal part of the flexor tendon sheath attached to the volar plate is displaced volarly, the point of angulation of the flexor tendons is also displaced volarly and distally on a mobile element – the base of the proximal phalanx instead of the stable metacarpal head – and causes ulnar deviation of the finger.

At the PIP joint

Synovial proliferation here also results in joint laxity.

Any excessive traction of the extensor apparatus inserted on the base of the middle phalanx causes hyperextension of the PIP joint, especially if the joint is lax. Many factors in a rheumatoid hand can increase traction of the central extensor tendon and favor the development of a swan-neck deformity. These include MP joint subluxation, ulnar displacement of the extensor tendons, and rupture of the extensor tendon at the distal joint (mallet finger) with proximal retraction of the extensor apparatus. Loss of PIP joint flexion is very disabling, and swan-neck deformity needs early treatment before the deformity becomes fixed.

In all the digital joints, destruction of the articular surfaces will result in joint stiffness. This can be an indication for MP or PIP joint arthroplasty, for restoration of a useful grasp.

Flexor tendon ruptures

Flexor tendon ruptures in RA are less frequent than extensor ruptures. They are caused by both infiltration and attrition. The surgical repair depends greatly upon the type of mechanism that leads to tendon rupture.

At the wrist level, the most frequent cause of rupture is attrition. Direct invasion of the tendon by proliferative synovitis involves all locations where the synovial sheath is present: within the carpal tunnel, palm, or digital sheath.

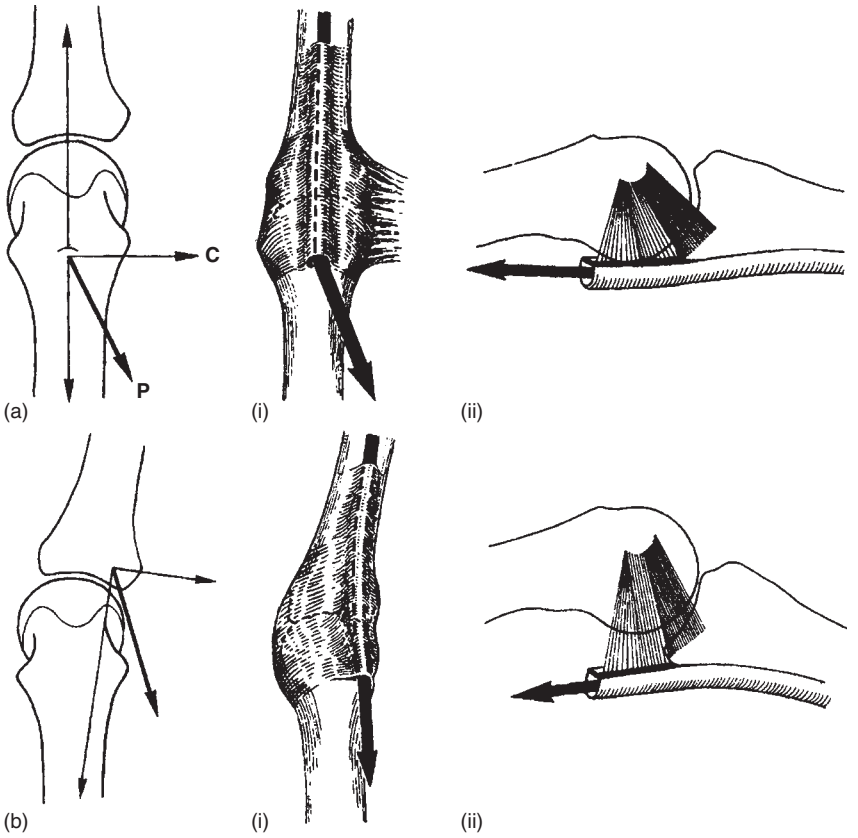


Figure 15.37

Forces created by the flexor digitorum tendons at the metacarpophalangeal (MP) joint level. (a) Normal index and long finger. (i) Anterior view showing the angulation of the flexor tendons as they enter into the sheath, and the two resultant forces, ulnar **C** and palmar **P**. (ii) Lateral view. The edge of the sheath acts as a pulley. The resultant ulnar force has no effect on the healthy joint, as it is applied to the metacarpal head, which is a fixed structure. The resistance of the healthy capsuloligamentous structures ensures no displacement of the orifice of the sheath. (b) In rheumatoid arthritis, when the MP joints have been invaded by the synovial pannus, the distention of the fibrous support leads to an anterior and distal displacement to the point of angulation of the flexor tendons. (i) The resultant ulnar force tends to press on the base of the proximal phalanx – a mobile element – and causes its ulnar displacement. (ii) For the same reasons, volar subluxation of the base of the proximal phalanx occurs.

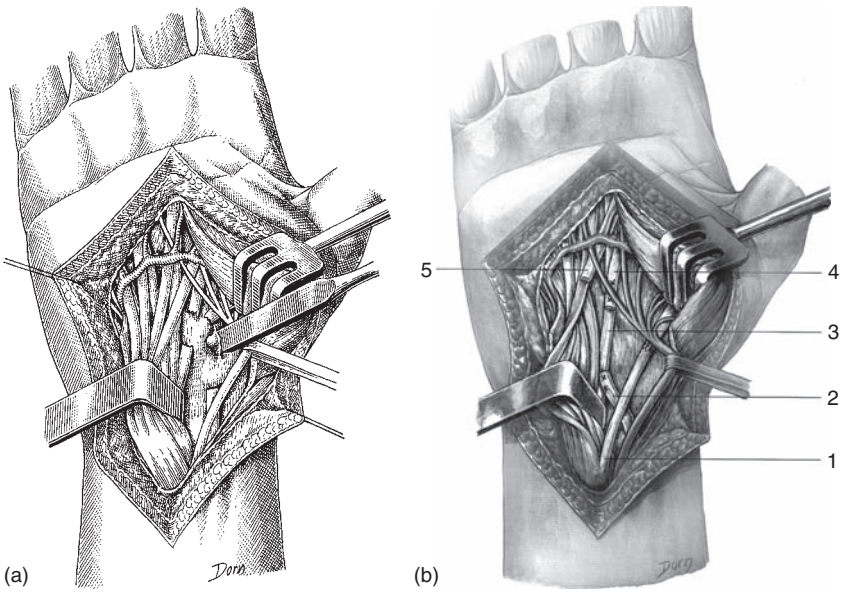


Figure 15.38

Flexor tendon ruptures at the wrist level: rupture of the FPL tendon and of the FDP of the index finger caused by attrition on a scaphoid bone spicule; the bone spicule is removed.

Attrition ruptures at the wrist have a better prognosis for the restoration of active flexion than do ruptures by invasion. The prognosis is also determined by the severity of rheumatoid disease and the effect of medical treatment, the degree of articular involvement, and the number of ruptured tendons. The goal of surgery is both to restore digit flexion and to prevent further ruptures of adjacent tendons.

Attrition ruptures

The most frequent cause of flexor tendon rupture is located over the scaphotrapezial region, the 'critical corner' of Mannerfelt.[122]

Due to RA, the scaphoid progressively assumes a horizontal position, and a bony spur can develop at the distal

part of the tubercle, which ruptures the FPL tendon. Once the FPL tendon has ruptured, the FDP of the index finger comes into contact with the spur, followed by the FDP of the long finger and the FDS of the index finger (Figure 15.38). Other sites of attrition have been described: the trapezium and the volar border of the distal radius.

Less frequently, the unciform process of the hamate and the distal edge of the ulna can be responsible for the rupture of the FDP of the little finger. These ruptures require urgent treatment in order to prevent further ruptures. One should start by treating the problem on the floor of the carpal tunnel. The bony spur is removed and the exposed bone is covered by a flap of adjacent soft tissue.

Treatment of FPL tendon rupture depends on many factors: the passive mobility of the thumb interphalangeal joint, the length of the gap between the two tendon ends, the quality of the adjacent flexor tendons, and the activity of the patient. An interphalangeal joint fusion is indicated if the joint is stiff or unstable. In other cases, it is better to restore FPL function by an intercalated graft from the palmaris longus or by transfer of a FDS tendon.

In isolated rupture of the FPL, an intercalated graft is preferred in recent ruptures, when the muscle has a satisfactory excursion. The tendon graft sutures must be performed away from the carpal tunnel.[123] Transfer of the ring finger FDS is preferred in older ruptures with a contracted muscle–tendon unit. Rupture of the index finger FDP tendon is treated by suture of its distal end to the adjacent intact tendon.

Ruptures by synovitis invasion

The diffuse tenosynovitis makes the diagnosis of rupture difficult.

Flexor profundus tendon ruptures at the palm level are treated by suture of the distal tendon end to the adjacent intact tendon. This is not possible at the finger level.

In isolated rupture of one flexor finger tendon in the digital canal, the primary goal of surgery is to protect the other flexor tendon by performing a meticulous synovectomy. If the superficialis is ruptured, it is not repaired. If the profundus is ruptured, the treatment should not jeopardize the remaining flexor superficialis function. Tenodesis or fusion of the DIP joint is adequate.

If both tendons are ruptured in the digital canal, poor results should be anticipated. A staged flexion tendon reconstruction using a silicone rod can be attempted in young motivated patients. In most RA patients, an arthrodesis of both the PIP and DIP joints in a functional position is the wisest choice, provided the MP joint is mobile.

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16 Biomechanics of the thumb

René Malek

General aspects

It is scarcely necessary to emphasize the importance of the thumb in hand function. The ability to oppose it to other fingers facilitates many ways to handle and manipulate objects. Duchenne de Boulogne[1] believed this facility led to humans dominating the animal kingdom. The expanses of the cerebral area devoted to its function support this conviction.

The late appearance of the opposition function (6th or 7th month of life) reveals the necessity of neurologic maturity for this very sophisticated function. Embryology already shows the singularity of the thumb: the embryonic hand is first tridactyl, and becomes pentadactyl towards the 60th day (Figure 16.1). The thumb takes its spatial position because, at the beginning of this period, the wrist becomes hollow. On the other hand, during development it is possible to distinguish two longitudinal rays in the forearm and the hand. The thumb takes part in the constitution of the lateral or radial ray, which is composed of the radius, scaphoid, trapezium, first metacarpal bone, and the two phalanges of the thumb (Figure 16.2). This division explains some sites of associated anomalies.

There are a number of imprecise notions about function which persist. Moreover, there is some confusion in the definition of the thumb movements because the planes of reference are not very well defined. On the other hand, the denomination of muscles (classically in connection with some finality) sometimes gives incomplete and wrong indices.

Opposition requires anatomic conditions, which will be discussed first.

Anatomic data

General aspects of the thumb's skeleton

The thumb is a short finger (Kaplan[2] considers it to be between 67% and 79% of the length of the index finger). Its extremity reaches the middle of the first phalanx of the index. This facilitates opposition to the pulps of the other fingers as they are flexed. The thumb has only three pieces of skeleton: a metacarpal, which is shorter and stockier than the ones of the neighboring fingers, and the diaphysis is somewhat more rounded and without well defined borders; and the first and the second phalanges, which are broader and more solid (Figure 16.2).

The question of which bone is absent has raised some controversies:

- In the theory of the triphalangeal thumb (which has nothing in common with a congenital pathology called 'thumb of three phalanges', which fits in the range of duplication pathology), it would be an absent metacarpal, or a metacarpal replaced by the trapezium. One of the main arguments in its favor is the existence of the proximal growth plate in the skeleton piece which would be called the first metacarpal, following the example of the phalanges and contrary to the metacarpal bones of the neighboring fingers, where the growth

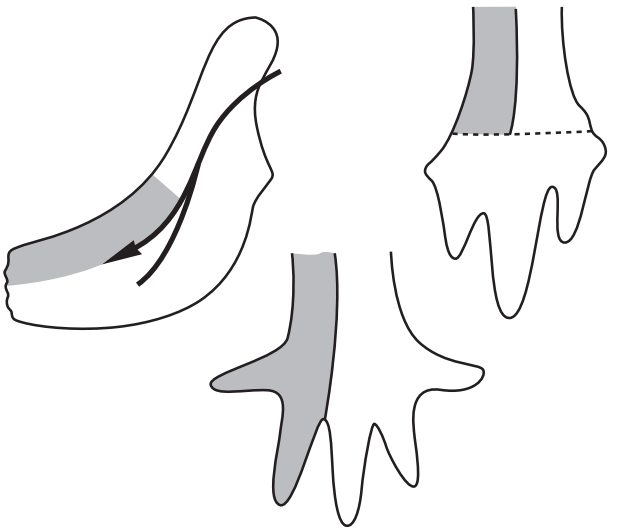

Figure 16.1

Embryology. The radial ray defect at three stages. The role of the interosseous artery has been pointed out. Anomalies of radius and thumb are very frequently associated.

*with V Gasiunas for the translation in English

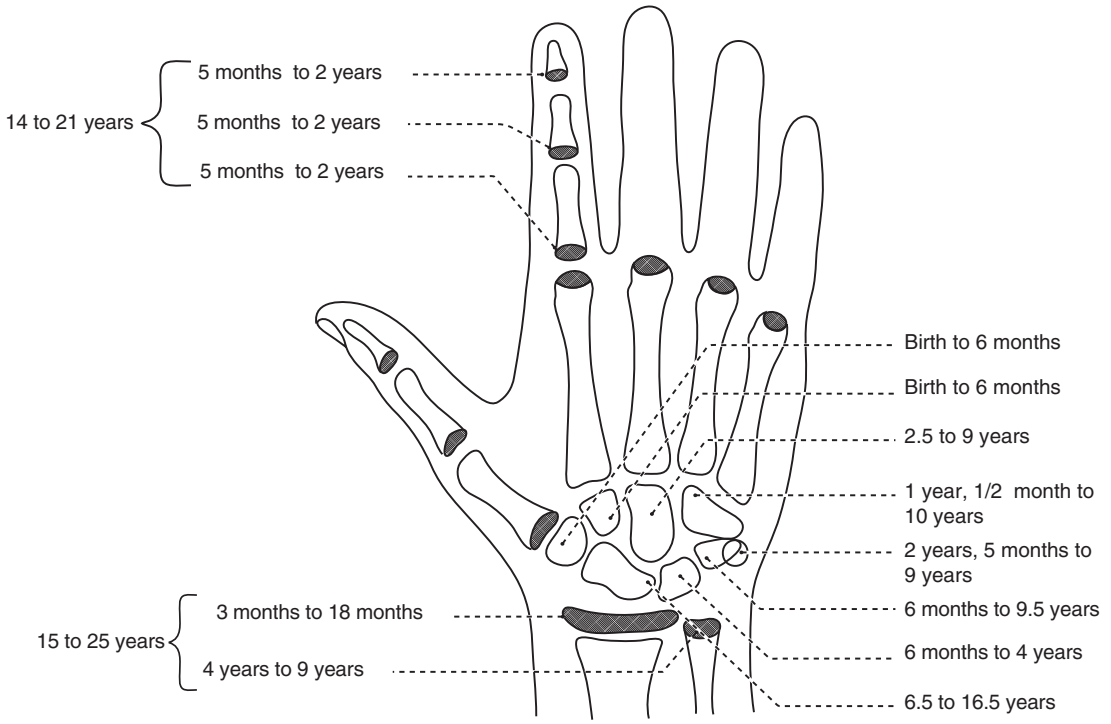

Figure 16.2

The ossification centers of the hand (date of appearance and date of fusion).

plate is situated distally. Nevertheless, this theory remains controversial.

- According to another theory it is the second phalanx which is absent or fused with the third phalanx. This would explain why the insertions of the intrinsic muscles are situated at the metacarpophalangeal level, as they are in the ulnar fingers.

It must be recognized that there is no practical impact of this problem, except that there are only three joints to provide the mobility of this digit.

Joints of the thumb

We will not cover the scaphotrapezial joint, although it is part of the thumb column, because it is in fact not very mobile. There have been many detailed discriptions of the direction and amplitude of the thumb's movements, covering the joints and their surfaces, with particular focus on the trapeziometacarpal joint, which is in the center of the specific opposition movements.

Although the anatomic detail is very interesting, it has helped to make the study of the biomechanics of the thumb very complex. Moreover, there is some confusion about the nomenclature of movements in their clinical assessment, and in the action of the muscles involved during these movements.

The trapeziometacarpal joint

This is also known as a 'saddle joint'.

The trapezium The joint surfaces and their orientation would be difficult to describe without taking into account the entire morphology of this small bone, which is part of the second row of the carpal bones.

Description

Why is it called the trapezium? Because there are at least two surfaces without contiguity, that correspond to a form of trapezium and are parallel to each other. To simplify, one could assimilate the bone to a form of a cube and then refer six planes to it. This facilitates the description, but does not match the exact situation of this bone in space. It is possible to describe the cube (its posterior surface being in the anatomic frontal plane), which presents (Figure 16.3) as follows:

- An anterior surface containing a rather voluminous structure, the tubercle of the trapezium (also called palmo-radial). It presents an internal crest, forming a limit to a groove that carries the tendon of the flexor carpi radialis muscle, which inserts into the second metacarpal bone.
- A posterior surface containing two tuberosities, a posteroradial (dorsoradial) tubercle and a posteroulnar (dorsoulnar) tubercle, separated by an excavation containing several small foramina for the vessels coming from the radial artery.
- A medial surface, which is a joint surface divided by a crest (perpendicular to the reference plane) into a concave superior surface articulated with the trapezoid bone, and an inferior area corresponding to the second metacarpal joint surface.
- A lateral rough surface situated subcutaneously.
- A triangular and concave superior surface, articulated with the distal pole of the scaphoid.

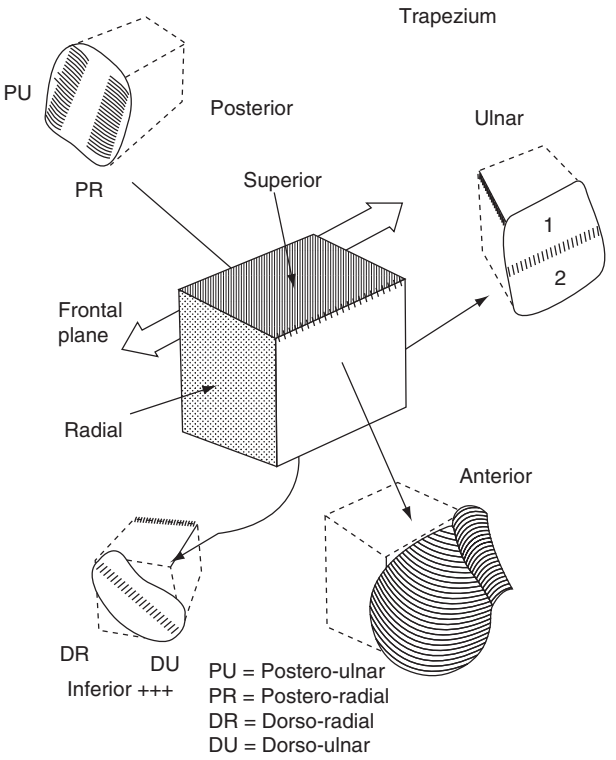

Figure 16.3

The trapezium can be likened to a cube presenting three faces: anterior with the tubercle of the trapezium and the furrow of the flexor carpi radialis; ulnar with two joint surfaces: 1 for the trapezoid, 2 for the 2nd metacarpal; superior joined with the scaphoid; posterior with two tubercles dorsoradial and dorsoulnar; radial subcutaneous; inferior joined with the first metacarpal.

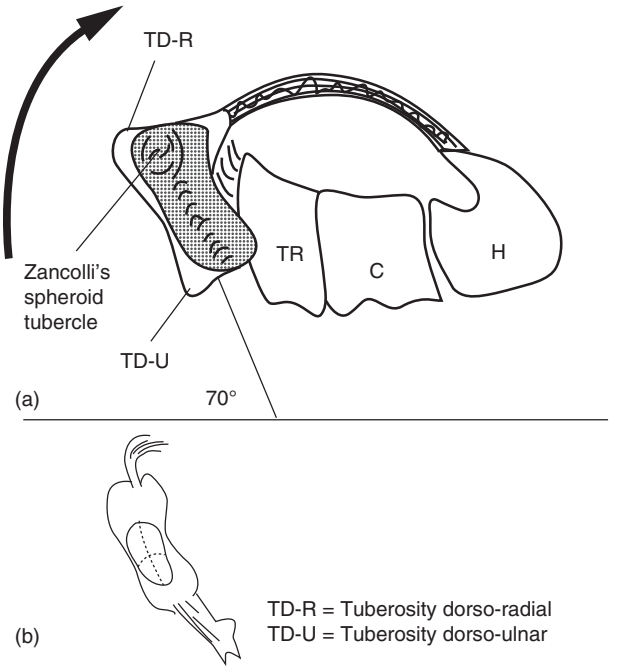

Figure 16.4

The rotated position of the trapezium and of the tubercles. The saddle of the horse whose head is in the posterior position.

- The largest inferior surface, representing the surface joint with the first metacarpal bone. It has been described many times, because it was (and it continues to be) thought that its configuration explains the complexity of the thumb movements.

The largest inferior surface has a form of a horse saddle, oblique anteriorly and laterally, and presents a double curve. It is concave in the anteroposterior aspect and convex transversally. This arrangement results in the formation of a crest in the great axis. Moreover, this surface represents a curvature with a medial concavity. Retaining the equestrian example, its form recalls a saddle for a horse with scoliosis – should it ever exist.[3]

In addition to this saddle-shaped joint surface, Zancolli[4] described a 'spheroid' joint surface, a zone that is convex in all directions, corresponding to a widening of the anterior part of the saddle surface's longitudinal crest; the zone covers the contour of the dorsolateral tubercle. This surface is much smaller than the saddle, but its role is significant in the rotational movements of the thumb (Figure 16.4a).

Orientation

The volume of the trapezium bone is situated in a particular position which modifies the orientation of its different surfaces as described by the anatomists according to the usual anatomic planes.

The wrist, for instance, represents a marked anterior concavity which is even more accentuated on each side as there are the tubercules of the scaphoid and trapezium (radiopalmar tubercle) laterally, and the pisiform bone and the apophysis of the unciform bone medially. In these prominences the anterior annular carpal ligament inserts and transforms the groove into the carpal canal.

Regarding the theory, the cube turns clockwise medially. When the anterior surface turns anteromedially, the groove of the flexor carpi radialis muscle's tendon goes directly to its insertion in the base of the second metacarpal bone. The medial part of the trapezium, having a double joint with the trapezoid and the second metacarpal bone, becomes nearly posterior. The posterior surface turns laterally and the dorsoradial tubercle becomes anterior in relationship with the dorsoulnar tubercle. The 'saddle' surface is oblique from the medial towards the lateral, and from the posterior towards the anterior aspect, and Zancolli's spheroid surface is practically anterior. The concavity of the saddle is turned towards the palm. Globally, this surface makes an angle with the frontal plane of 70° (20° with the sagittal plane) (Figure 16.4a).

Moreover, the axis of the trapezium (perpendicular to the two surfaces of the scaphoid and metacarpal) is oriented laterally in the same direction as the great axis of the scaphoid, but its saddle surface is tilted anteriorly and forms an angle of 35° with the horizontal plane (Figure 16.4b).

Altogether, the joint surface with the metacarpal bone is oriented medially, anteriorly, and downwards.

The joint surface of the first metacarpal bone This complies classically with the trapezium bone's surface in the inverse manner. It is also divided into three parts. The first one is in the form of a saddle, perpendicular to the one of the trapezium, and with inverse curvature, i.e., concave latero-medially and convex anteroposteriorly, creating a 'metacarpal crest'. So this extremity fits together with the trapezium's saddle (the surfaces having the form of two letter Ys, perpendicular to each other, which leads to the concept of such a system as a type of universal joint.

The contact with the trapezium saddle is relatively firm, and adapted according to the degree of tension in the structures of the ligaments and capsules. Many authors deny the possibility of rotational movements, by not considering the 'obligatory' rotation, which will be discussed later.

Zancolli[4] paid particular attention to the possible contact between the metacarpal joint surface and the 'spheroid zone', which he had described in the 'saddle' of trapezium (contact between two roughly convex zones). This is the zone which allows the rotational movements by dint of an existing metacarpal subluxation.

The two other parts of the joint surface correspond to two apophyses, which are the extensions of the metacarpal crest. Each apophysis has a joint surface concave in all directions. In the opposition and retroposition movements, these two surfaces come into contact with the spheroid zone and, during transition from one position to another, the metacarpal crest spans the summit of the spheroid zone with some incongruence.[4]

Of the two basal apophyses, one is dorsal and the other one is palmar, according to the axis, which is perpendicular to the great axis of the scaphoid bone. This gives us an idea of the orientation of the first metacarpal bone, which has the anterior surface facing anteriorly and medially. The spike of the palmar apophysis is intracapsular.

The base of the metacarpal bone has also a prominence which corresponds to the insertion of the tendon of the long abductor muscle, opposite to the ulnar tubercle on the other side, into which the ulnopalmar ligamentory complex is inserted.

It must be noted that in the trapeziometacarpal joint the only mobile surface is the metacarpal, whereas the surface of the trapezium takes part in the movements of the wrist[9] (as a whole or between each of the bones). This is why the comparison of the movements of the joint to the movements of a universal joint does not seem appropriate.

To keep the horse analogy, the following must apply:

- The horse is oriented posteriorly, with its cephalic extremity directed towards the second metacarpal (Figure 16.4b).
- The horse rider, who would correspond to the first metacarpal bone overlapping the saddle, is in a precise position: he would have the two legs (almost amputated where the stumps correspond to the apophyses of the metacarpal bone) on each side of the saddle, with his pelvis perpendicular to its great axis. But the horse rider looks in front of him and medially, and has a non-physiologic body torsion, which would explain how the thumb pulp can be oriented towards the pulps of the other fingers.

Thus, the first metacarpal bone may move on the saddle in two main axes (Figure 16.5a):

- an axis of the saddle which makes it lean forwards and backwards

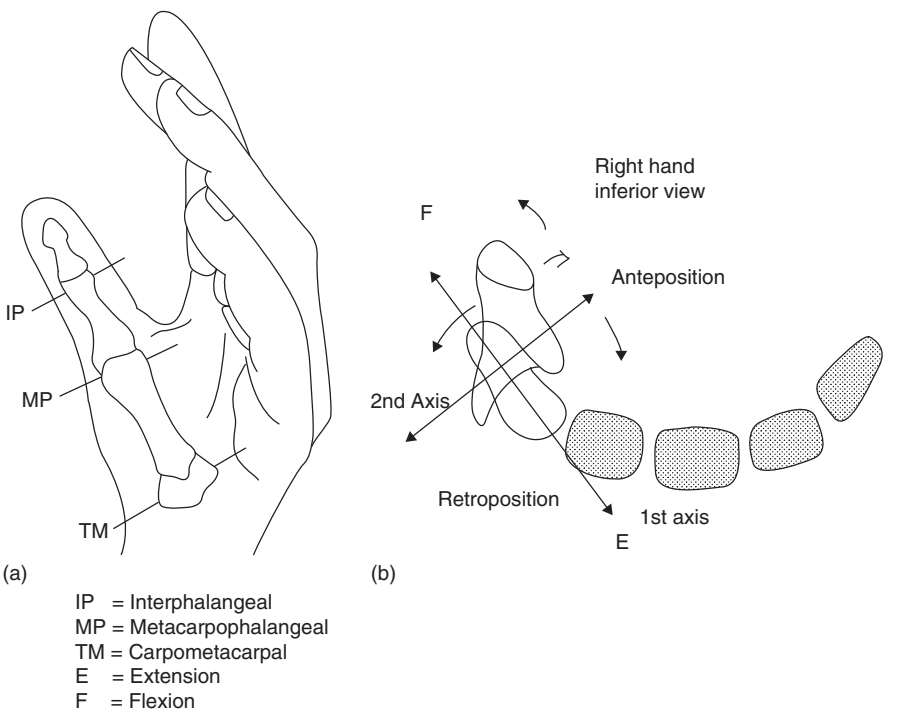

Figure 16.5

(a) The axes of the three joints of the thumb are almost parallel. (b) The mobility of the first metacarpal and the definition of the movements: anteposition is flexion and adduction; retroposition is extension and abduction. There is always a combination of the two possible movements around the two axes.

- an axis which is perpendicular to the first and makes it lean to one side (radial) or the other (ulnar).

However, the first axis is roughly parallel to the axis of flexion and extension of the metacarpophalangeal as well as the interphalangeal joint (Figure 16.5b).

That is why, in order to simplify along the lines of Zancolli[4] we propose naming the movements of the first metacarpal bone in the same manner: flexion and extension. The flexion of the first metacarpal bone involves an anteposition, so one could talk about the flexion-anteposition. The extension involves a retroposition so the terms could be associated, such as extension-retroposition[5] (Figure 16.5a). The second movement in the axis, perpendicular to the first, would be abduction, which takes the metacarpal bone away from the axis of the hand, and adduction, which brings the metacarpal bone closer to the ulnar side.

The concept of 'drawing aside' the thumb from the second metacarpal bone is interesting.[6,7] It is measured in terms of an angle, which varies according to the position of the thumb at the moment of measurement, and it is not easy to measure clinically. The first metacarpal makes a maximum angle of 45° with the second bone; the minimum value also depends on the volume of the musculature of the first web space, and is never less than 15° (Figure 16.6).

The metacarpal joint surface may slide over the surface of the saddle, which will then modify the axis of movement, added to the possibility of displacement[4] (Figure 16.7).

Naturally, these two types of mobility may combine in opposition, leading to an excursion over the approximately conical joint surface (circumduction).

This situation is also complicated by the possibility of thumb rotation, which can be seen at the level of every segment of the finger. This is covered in a later section.

The problem of naming the movements is arduous, as there is no consensus, and therefore some difficulties may be experienced in the clinical descriptions.

Restraining structures The *capsule* is generally rather lax, and is reinforced by the four *ligaments* which limit the amplitude of joint movements (however, these are not the only limiting factors) (Figures 16.8–16.10). The four ligaments consist of the following:

- The intermetacarpal ligament, which is situated between the bases of the first and the second metacarpal bones, at the bottom of the first web space, and which limits the opening of the latter.
- The oblique posteromedial ligament, which emerges at the posterior aspect of the trapezium and is often associated with the preceding one and forms a ligamentory

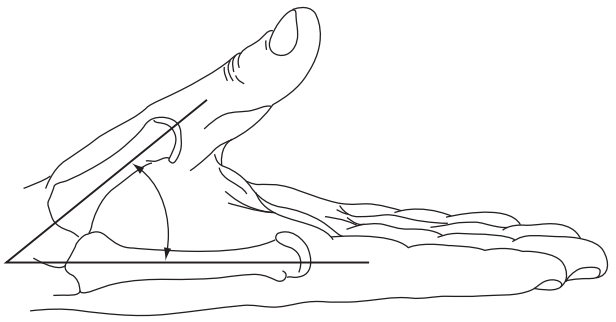

Figure 16.6

'The angle of separation' formed by the intersection of the axes of the first and second metacarpals in a sagittal plane. Reproduced with permission.

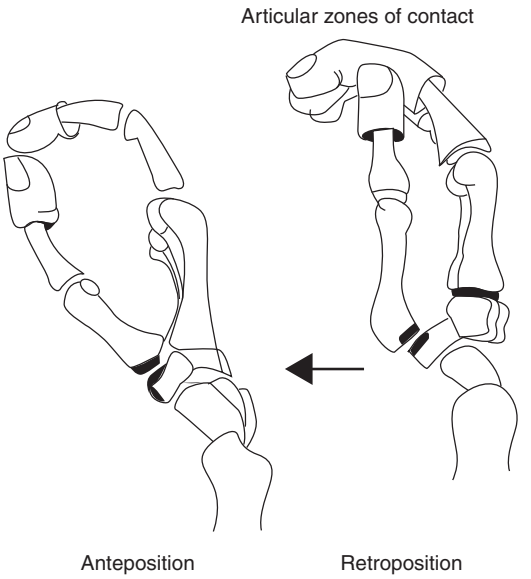

Figure 16.7

The usual subluxation of the base of the metacarpal and the change in the zone of contact. Adapted from Zancolli.[4]

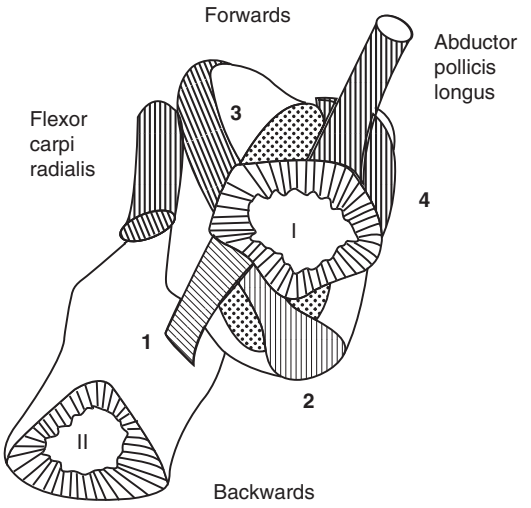

Figure 16.8

Ligaments of the trapeziometacarpal joint, inferior view. 1, Intermetacarpal; 2, posteromedial; 3, oblique anteromedial; 4, straight anterolateral. Adapted from Zancolli.[4]

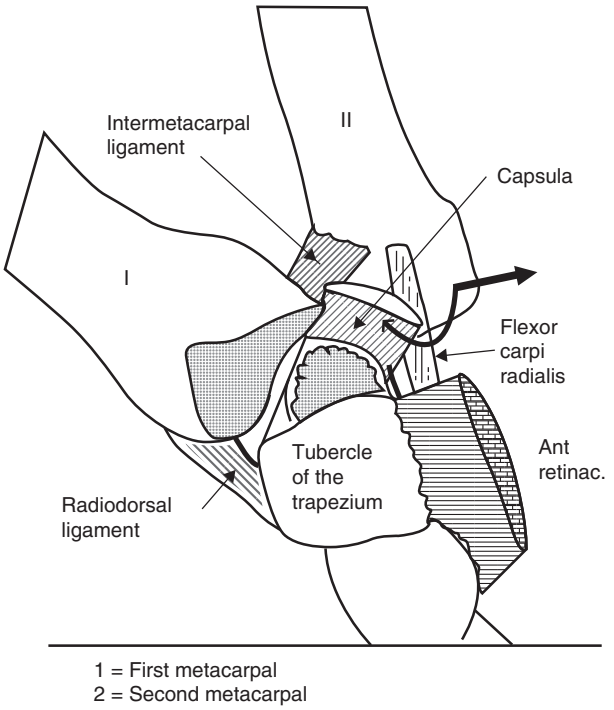

Figure 16.9

The anterior aspect of the joint, artificially dislocated, showing the recessus of capsula.

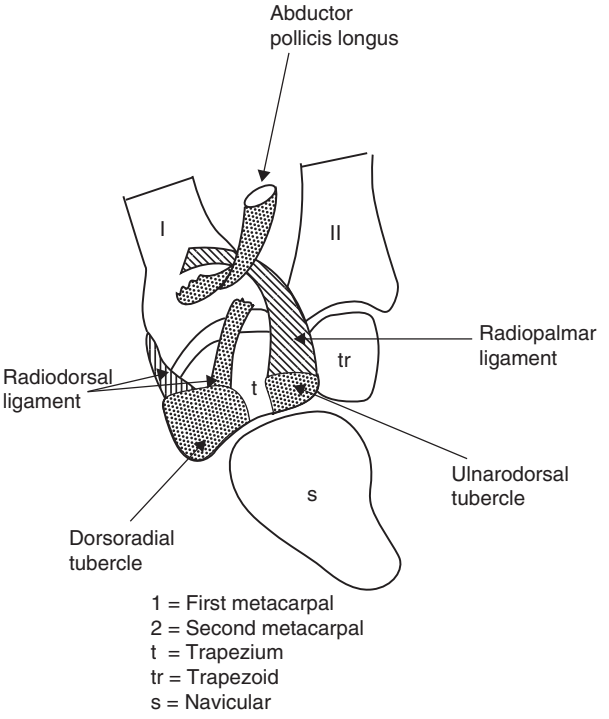

Figure 16.10

The posterior aspect of the joint.

'ulnopalmar' complex. Emerging from the dorsoulnar tubercle of the trapezium, it travels in an oblique fashion until it joins the insertion of the intermetacarpal ligament at the base of the first metacarpal.

Those two ligaments are considered to favor the trapeziometacarpal joint's stability, but also have an action of pronation, as they act together with muscles of opposition (the pronation may reach 90°).

- The oblique anteromedial ligament, which arises from the anterior aspect of the border of the flexor carpi radialis groove and inserts into the metacarpal base. This ligamentory bundle prevents hyperextension of the joint and would be a factor of supination in the movement of retroposition.
- Finally, the straight anterolateral ligament, which is situated between the radiodorsal tubercle of the trapezium and the base of the metacarpal bone, passing around the tendon insertion of the long abductor. It is stretched in flexion and abduction.

The intrinsic and extrinsic *tendons of the periarticular muscles* are also a means of restraint, limiting the amplitudes of the movements that they determine.

So what are the possible movements in this very exceptional joint? In fact the movements are of rather limited amplitude, contrary to what one could expect in view of the number of mechanical and mathematical studies this joint has inspired. For many reasons, it is now possible to conclude that the mobility of the thumb is determined only partially by the morphology of the trapeziometacarpal joint surfaces.

The metacarpophalangeal joint

This is also a very important joint of the thumb. Its construction follows the lines of the other fingers' metacarpophalangeal joint. It is a condylar, two-degree of freedom joint, which functions in flexion-extension and, very importantly, in abduction-adduction.

Joint surfaces The metacarpal head is convex in two directions – the width is greater than the height – and presents two supports, with the medial one more prominent anteriorly. The base of the first phalanx is concave in two directions, and at its anterior border presents the insertion of the glenoid fibrocartilage, which is mobile in relationship to the phalanx as it undergoes flexion. Close to its insertion, the fibrocartilage contains two flat sezamoid bones, one surface of which is covered by cartilage in continuity with the joint cartilage, whereas the other gives the insertions for the thenar muscles: the lateral sezamoid bone receives the tendons of the abductor pollicis brevis, and the flexor pollicis brevis, and the medial one the adductor pollicis and the tendon of the first palmar interosseus muscle.

Restraining structures These comprise the joint capsule, which is rather lax and has a large palmar recessus, and collateral ligaments, which have a metacarpophalangeal part, and a metacarpoglenoid part which is attached to the fibrocartilage. In the metacarpophalangeal part, the medial ligament is shorter and is stretched more rapidly than the

lateral ligament, during flexion. The latter is also held by the phalangoglenoid ligaments which are inserted into the sezamoid bones, attached to each other by intersezamoid ligaments. The insertions of the intrinsic muscles press the fibrocartilage against the metacarpal head in extension (which assumes stretching of the intrinsic muscles); in flexion, with an active shortening of muscles, the fibrocartilage, in its sezamoid portion, has a tangential position in relationship to the body of the metacarpal bone, because of the action of its ligamentory attachments.

The mobility of the metacarpophalangeal joint is not important. The ligamentory arrangement and the muscle insertions limit the flexion, which is in the range of about 50° to 70°. The unequal length of the collateral ligaments leads to pronation of the first phalanx while flexed. Asymmetric condyles also contribute to this structural rotation, which is added to the rotation of the metacarpal bone at the level of the trapeziometacarpal joint, as we have seen above. Active and passive extension are very limited, except very particular cases. Radial inclination caused by the sezamoid bones is significant. It may reach 30° in extension. Ulnar inclination is less marked. It should be noted that, if we refer to passive motion, it is because the handling of objects implies external forces of resistance which act on the position of the skeletal pieces. This is also true for muscular action.

The interphalangeal joint

This is a trochlear type joint and has only one axis of mobility, which is roughly transverse at the level of the head of the first phalanx. This axis is tilted in relationship to the longitudinal axis of the diaphysis. There is also a condylar asymmetry of the same type as described for the metacarpal. So, the rotational movement of the second phalanx of 5° to 10° will be added to the same kind of movements at the other levels of the thumb. The active flexion of the interphalangeal joint reaches 80° to 90°. The active extension is only of a few degrees, in contrast to the passive extension, which may be much more.

Muscles of the thumb

The muscles of the thumb consist of two types:

- extrinsic muscles, which have their bellies located in the forearm, and
- intrinsic muscles, which form the thenar eminence muscles and the interossei muscles of the first web space.

Thenar eminence

The majority of authors consider that there are four thenar muscles, which, from the superficial to the deeper layers are:

- abductor pollicis brevis
- opponens pollicis
- flexor pollicis brevis and its two parts
- adductor pollicis and its two bundles.

It must be emphasized again that their nomenclature does not necessarily correspond with their real physiology, and this may lead to a misunderstanding of their action.

Abductor pollicis brevis This is a small and relatively weak muscle, which is situated above the much more voluminous opponens pollicis muscle. It arises at the anterior aspect of the anterior annular ligament of the wrist in its lateral half and the neighboring scaphoid tubercle. It inserts into the lateral tubercle of the base of the first phalanx, and into the radial sesamoid. It gives the fibers which go round the metacarpophalangeal joint and insert into the dorsal aponeurosis of the thumb and the tendon of the extensor pollicis longus, forming half of the fibrous expansion (Figure 16.11).

Opponens pollicis This is located under the abductor pollicis brevis, and also arises from the anterior annular ligament of the wrist. It has a quadrangular shape and its fibers insert into the lateral half of the anterior surface of the first metacarpal bone. It is a rather voluminous muscle which has a fundamental role in stabilization of the articular chain of the thumb (Figure 16.12).

Flexor pollicis brevis This is formed by two bellies: a superficial one which emerges from the anterior annular ligament of the wrist under the other intrinsic muscles described above, and another in the bottom of the carpal groove on its radial side. These two bellies join each other and insert into the lateral sesamoid and neighboring tubercle of the

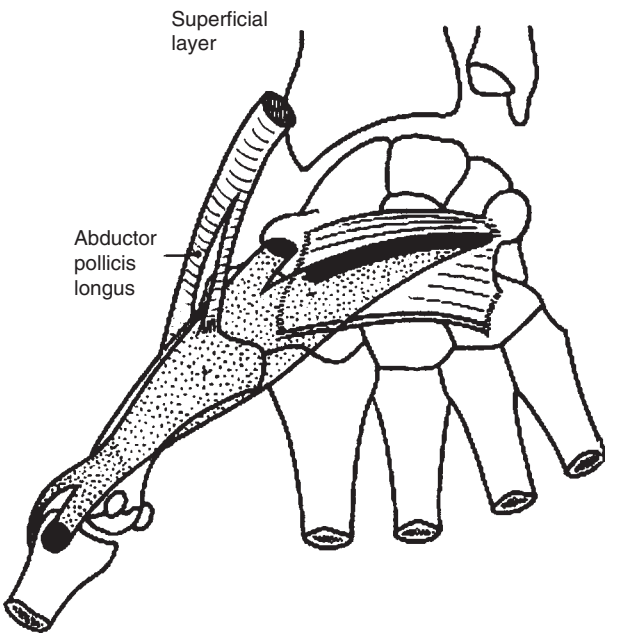

Figure 16.11

The abductor pollicis brevis (insertions in black). Adapted from Brizon and Castaing.[8]

first phalangeal base. Between the two bellies passes the tendon of the flexor pollicis longus beyond the carpal canal. This tendon will cross the deep belly of the flexor pollicis brevis superficially to go under the first fibrous intersesamoid pulley (Figure 16.13).

Adductor pollicis This is a large, fan-shaped muscle which arises by muscular and aponeurotic fibers from the bases and anterior aspects of the second and the third metacarpal bones, as well as from the neighboring carpal bones. Two bellies can be described: one transverse, proximal, and palmar, and another deep, with the more oblique fibers which form an arch at the base of the third metacarpal bone under which pass the deep branch of the ulnar nerve and the deep arterial arcade. The two bellies insert into the medial sesamoid and the neighboring tubercle of the base of the first phalanx. Together with an inconstant palmar interosseous muscle, adductor pollicis sends dorsal expansion fibers to the extensor tendon, which, together with the homolog fibers coming from the abductor pollicis brevis, form a dorsal fibrous expansion, which is the same structure as in the ulnar fingers. This expansion covers the lateral collateral ligament of the metacarpophalangeal joint. In the case of a severe luxation of the joint, this expansion (of which the superior border is free and quite solid) may interpose between the joint and the proximal part of the ruptured ligament and thus impede any cicatrization with the distal part of the ligament. Finally, some of the terminal fibers of the adductor reinforce the flexor tendon's sheath (Figure 16.14).

As well as the opponens pollicis muscle, one can distinguish thenar-lateral sesamoid muscles, which are the abductor pollicis brevis and the flexor pollicis brevis, and the thenar-medial sesamoid muscles, which are the adductor pollicis and the first palmar interosseus. The muscles of the two groups are the flexors of the first phalanx, but they are also extensors of the metacarpal bone. The lateral muscles are pronators, whereas the medial ones are supinators of the thumb.

The eccentric situation of the thenar muscles should be noted – these are situated anteriorly with regard to the first

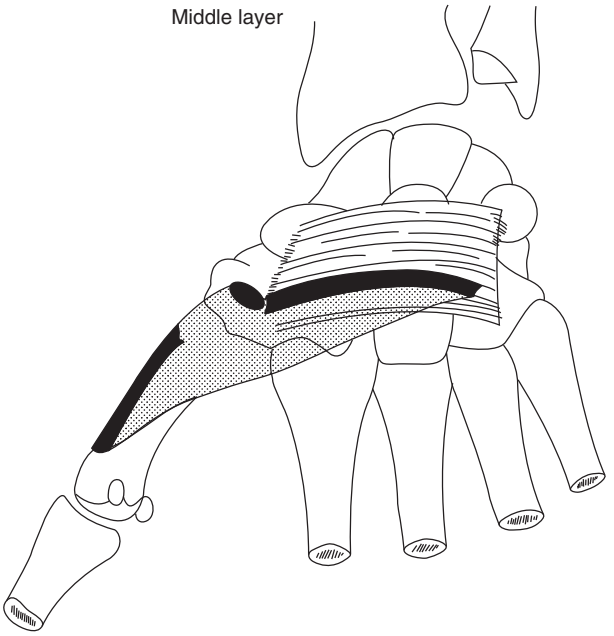

Figure 16.12
The opponens. Adapted from Brizon and Castaing.[8]

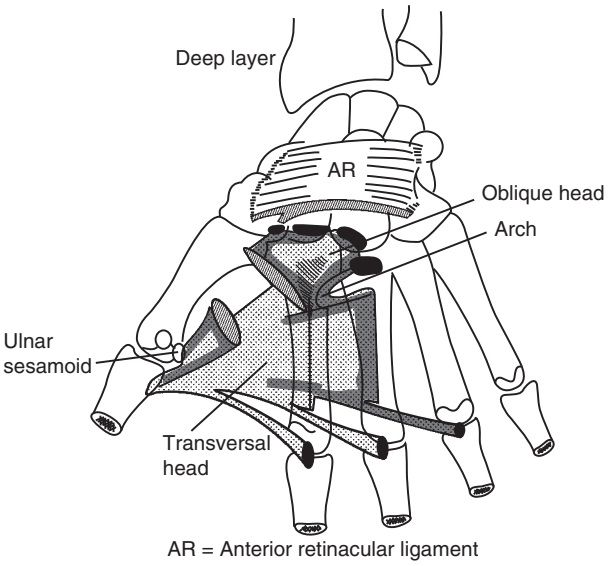

Figure 16.13
The two heads of the adductor. Adapted from Brizon and Castaing.[8]

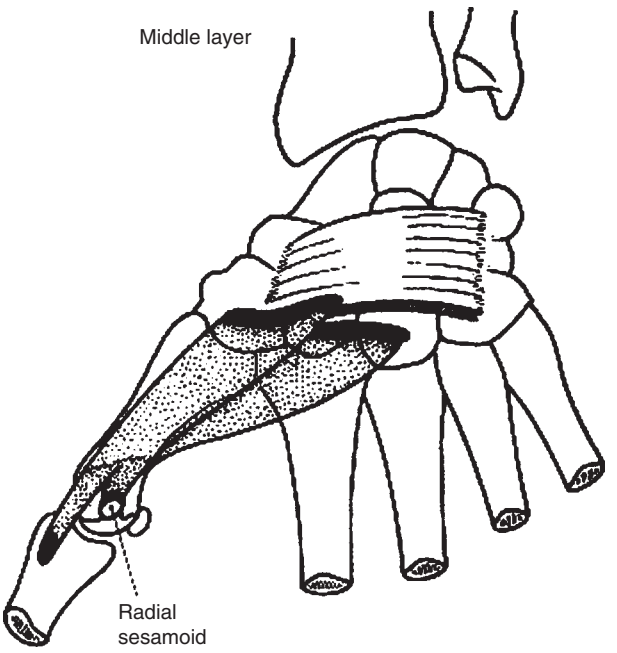

Figure 16.14
The flexor pollicis brevis and its two heads. Adapted from Brizon and Castaing.[8]

metacarpal bone, and also anteriorly in relationship to the oblique plane containing the transverse axes of the three joints of the thumb. These features give considerable torque and force in comparison to the volume of the muscles. The intrinsic muscles of the thumb are stronger than the extrinsic ones. Compression and shearing forces at the level of the trapeziometacarpal joint are significant.

Some intrinsic muscles are distinct from the others because they belong to the first web space. These are:

- The frequently non-existent first palmar interosseus muscle, which arises from the first metacarpal bone and inserts into the medial sesamoid bone. It is difficult to isolate it from the free border of the adductor pollicis, which shares the same action. A number of authors prefer to reserve the name of 'first palmar interosseus' to a muscle situated in the second interosseus space.
- The first dorsal interosseus muscle which is a very important muscle from the anatomic and functional point of view, because it takes part in the pinch. Thus this muscle is studied together with the intrinsic thumb muscles. It arises from the two borders of the first web space, i.e., from the dorsal side of the first and second metacarpal diaphyses. It forms a belly, which is often very voluminous (the prominence of which is seen at the posterior aspect of the first web space), and it ends at the radial side of the base of the first phalange of the index finger. It is inconstant but, when it exists, it gives fibers for the dorsal expansion and constitutes part of it. It flexes the first phalanx of the index finger, extends the two distal phalanges by means of the dorsal aponeurosis, and has an abductor (radial displacement) action on the metacarpophalangeal joint. It resists the forces applied on the lateral side of the index finger, and is also a stabilizer of the trapeziometacarpal joint because it acts against the dislocating effect of the adductor and flexor muscles.

Extrinsic muscles

There are three extrinsic muscles on the dorsal side, and only one on the palmar side. Their bellies are situated in the forearm and are tendinous at the thumb level. These muscles are active not only on the thumb, but also on the wrist, which they cross. Equally, the position of the wrist will modify the action and force of the extrinsic muscles of the thumb.

Dorsal muscles (Figure 16.15) These are a part of the deep muscle layer in the posterior forearm region. In order of appearance, from high to low, they are:

- the abductor pollicis longus
- the extensor pollicis brevis
- the extensor pollicis longus, which is above the extensor indicis proprius muscle.

It must be emphasized that the muscles which emerge higher in the forearm do not come down as low at the level

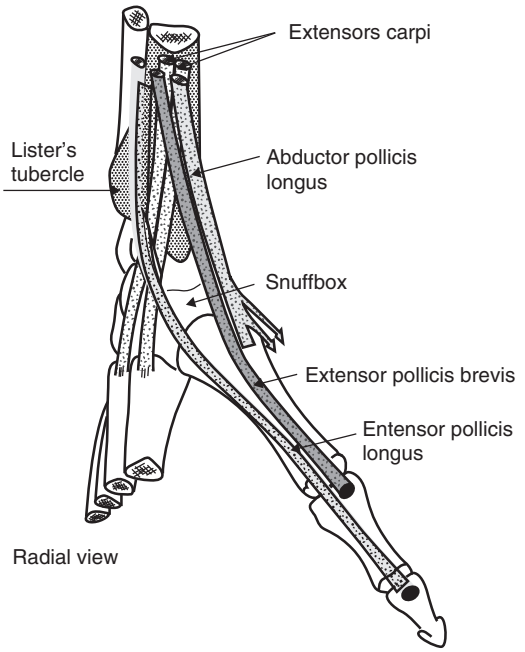

Figure 16.15

The dorsal and lateral tendons of the thumb. The 'snuffbox' is delimited by the two extensors, and the extensor carpi radialis, brevis, and longus are located at its superior part.

of the thumb column. All are oblique as they come downwards and laterally around the wrist and at the dorsal aspect of the first web space. When the thumb is at rest, the first web space is in a different plane in relationship to the surface of the dorsal hand, and forms an angle of 40° to 80° with it. The tendons are attached to the radius by means of fibrous sheath's (fixed points), and during the movements of the thumb they are mobile under the dorsal skin of the first web space.

The abductor pollicis longus

This has a strong tendon which arises rather high above the wrist, crosses two extensor carpi radialis brevis and longus dorsally in an oblique fashion, above the anatomic 'snuff box', and then enters an osteofibrous canal at the lateral aspect of the radial epiphysis and inserts at the posterior surface of the base of the first metacarpal. It often sends the expansion towards the abductor pollicis brevis. The abductor longus is not, in fact, an abductor of the thumb. It acts as an extensor, but also as an adductor which pulls the metacarpal bone backwards (the horse rider falls backwards and laterally) when moving it apart from the axis of the hand (that is when it is an abductor). It is also a wrist flexor (the only flexor innervated by the radial nerve), and it also leads to a radial tilt of the wrist.

The extensor pollicis brevis

Often its thin tendon comes into the same groove as the tendon of the long abductor, and then inserts into the

base of the first phalanx. It has the same action on the wrist and is also an extensor of the trapeziometacarpal joint. It is classically an extensor of the metacarpophalangeal joint. According to experience, its absence (congenital malformation) or its section leads to a 'pollex flexus adductus', i.e., a thumb flexed into the palm with a flexion of 90° at the metacarpophalangeal joint, despite the existence of a normal extensor pollicis longus. (There is also an extension at the trapeziometacarpal joint.) The explanation resides in the fact that the flexors of the first phalanx (intrinsic and extrinsic) generate a force on flexion on the first phalanx, which is no longer counterbalanced when the extensor pollicis brevis is absent. Moreover, the axial force of the extensor pollicis longus, associated with the force of the flexor tendons, worsens the flexion of the metacarpophalangeal joint rather than counterbalancing it.

The extensor pollicis longus This is a tendon which is vertical in the beginning and then changes its direction as it passes around Lister's tubercle of the radial epiphysis (at this point it may be damaged in the rheumatoid hand), crosses the lower part of the radial extensors of the wrist, and passes to the dorsal surface of the thumb. It receives the dorsal expansions of the intrinsic muscles before it inserts into the base of the second phalanx. This muscle acts on the wrist by extending it and leads to a radial inclination. It is somewhat lateral at the metacarpophalangeal joint and therefore contributes to the radial inclination of the first phalanx jointly with the lateral sesamoid muscles (Figure 16.16). Finally it is an extensor of the trapeziometacarpal joint (the horse rider leans backwards) (retroposition), and because of that it takes part in the latero-lateral pinch with the second metacarpal bone – still possible in certain extended paralyses.

In general, radial inclination of the wrist relaxes the dorsal extrinsic muscles of the thumb and vice versa – the latter ones need the ulnar inclination to develop all their force. Finally, one must know that there are frequent anomalies regarding these three extrinsic muscles.

Palmar muscles The flexor pollicis longus, in contrast to the preceding muscles, is rather voluminous. It arises from the anterior surface of the radius in the area free from insertions that exists between the bicipital tuberosity and the pronator quadratus muscle. It passes through the carpal canal laterally to the flexor tendons (it is the most lateral element), bends below the trapezium's tuberosity, and afterwards, obliquely, passes laterally and anteriorly in relationship to the axis of the carpal canal, and between the two bellies of the flexor pollicis brevis. Then it crosses the deep belly and proceeds between the two sesamoid bones to insert at the base of the second phalanx (Figure 16.17).

From the metacarpophalangeal joint the flexor pollicis longus passes into two fibrous sheaths which press it against the skeleton: one at the level of the sesamoid bones and the fibrocartilage, and the other at the level of the diaphysis and the head of the first phalanx. When it enters into the carpal canal, the flexor pollicis longus

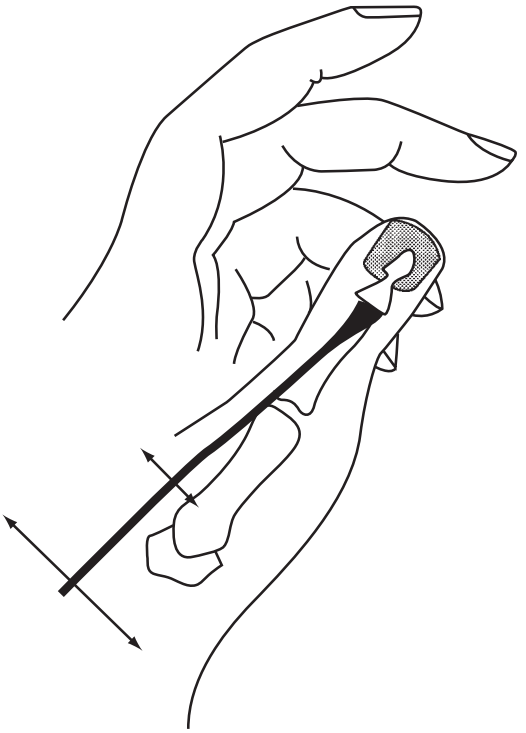

Figure 16.16

The extensor pollicis longus has a transverse mobility related to the degree of ante- or retroposition of the thumb.

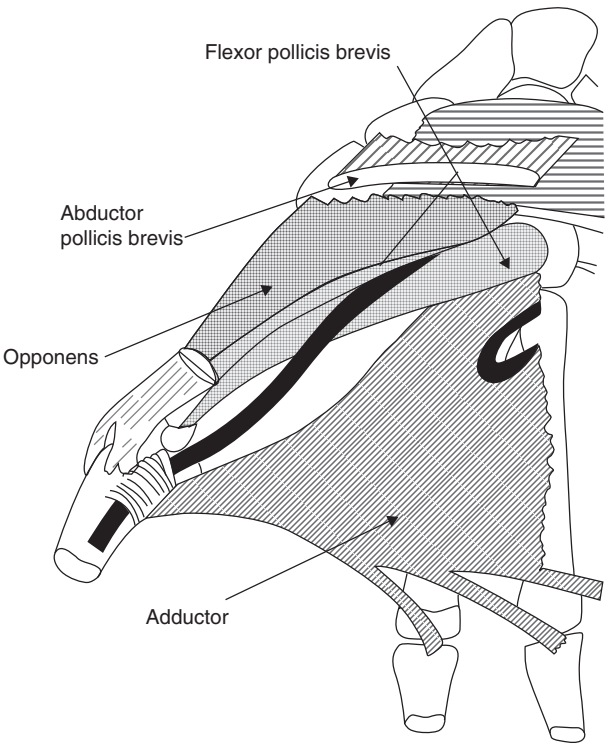

Figure 16.17

The thenar eminence muscles: the route of the tendon of the flexor pollicis longus between the two heads of the flexor pollicis brevis. Adapted from Brizon and Castaing.[8]

is surrounded by a synovial sheath, distinct from the synovial sheath of the flexor communis tendons.

The flexor pollicis longus is sometimes comparable to the flexor communis, as it splits into two strips near the first phalanx. The tendon is rather mobile as it performs a run of some 12 mm. Its action is mainly the flexion of the second phalanx of the thumb. As the proximal pulley exists, in the case of external resistance which occurs because of contact with an object, the flexor pollicis longus becomes a flexor of the first phalanx. In the two cases, by the means of the mechanism of axial forces, it is an extensor of the trapeziometacarpal joint. Its oblique direction under the carpal canal does not, however, allow it to exert all its force on an object held between the thumb and the fingers. This is a notion advocated by Brand,[9] which is contradictory to the classic notion according to which the thumb's force while holding an object is referred to the flexor pollicis longus (for this author the adductor pollicis and flexor pollicis brevis have a 30% bigger force because of a better leverage). Nevertheless, when the object held is resistant, the force of the flexor pollicis longus acts on the pulley at the entry of the sheath at the level of the metacarpophalangeal joint, and then the classic notion holds true.

Finally, the wrist movements are more significant here than for the dorsal muscles (which allow the use of this tendon in tetraplegia by an active tenodesis effect).

Motor innervation of the thumb

The dorsal extrinsic muscles are all innervated by the radial nerve. The flexor pollicis longus is innervated by the median nerve. In the case of the intrinsic muscles, the lateral sesamoid muscles are innervated by the median nerve the medial sesamoid muscles by the ulnar nerve. The interossei muscles in the first web space are innervated by the ulnar nerve. There are some anatomic variations for the interossei and thenar muscles.

Topographic anatomy

As the muscles of the thumb have already been described, a topographic study will be used to summarize. *Superficially*:

- The first web space is wide, covered by soft and flexible skin. Its border presents two principal folds which medially prolong the two flexion folds of the metacarpophalangeal joint, and which appear alternatively in the thumb's movements.
- Palmarly, one can clearly identify the relief of the thenar eminence (related to the volume of the opponens pollicis, which is covered by the abductor pollicis brevis), which is limited medially by the thumb's opposition fold or the 'life line', distally by the free border of the skin of the first web space, and superiorly by the inferior transverse wrist fold.
- Dorsally, there is a palpable dorsal aspect of the metacarpal bone under the skin, which is raised by the three extrinsic tendons, mainly by the tendon of the extensor pollicis longus, whose transverse mobility is visible. There is often a considerable relief of the dorsal interosseus muscle of the first web space. The anatomic 'snuffbox' is situated at the base of the thumb and between the tendons of the extensor pollicis longus and abductor pollicis longus.

The surface of the first web space – the dorsal – represents a third anatomic zone of the hand, in addition the palm and the dorsum. This is a triangular zone with a superior summit, situated at a different angle in relationship to the plane of the metacarpal bones, which depends upon the opposition of the thumb, the zone where the extrinsic tendons pass under the skin (Figure 16.18).

At a greater depth, the aponeuroses define the thenar or palmar-lateral compartment, which is situated between the

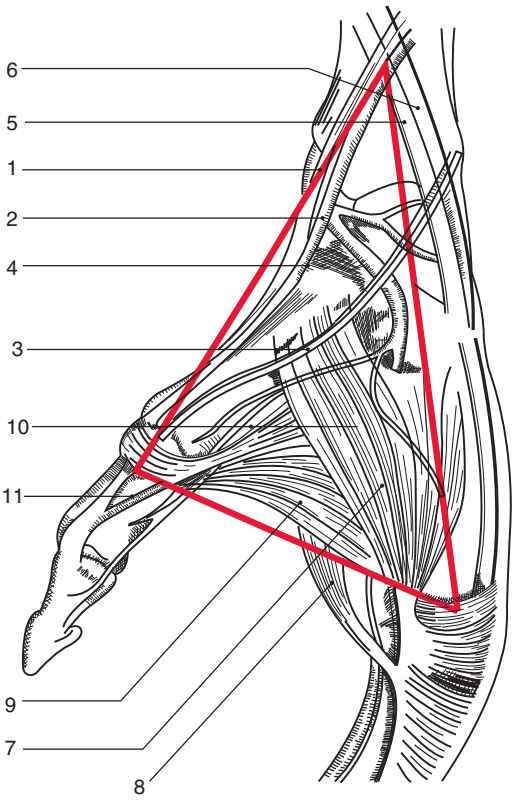

Figure 16.18

The third topographic area of the thumb corresponds to the triangular zone of the dorsal aspect of the first commissure: (1) abductor pollicis longus; (2) extensor pollicis brevis; (3) extensor pollicis longus – the last two tendons define the snuffbox where the following pass; (4) the radial artery and the tendons of; (5) extensor carpi radialis longus and; (6) extensor carpi radialis brevis; (7) the first dorsal interosseous; (8) the first lumbrical; (9) adductor pollicis (oblique head); (10) adductor pollicis (transverse head); (11) the dorsal slip of the adductor for the extensor apparatus.

deep palmar aponeurosis of the two first web spaces posteriorly and the thenar aponeurosis anteriorly, which is attached to the anterior border of the third metacarpal bone. This insertion makes it clear that there is a true septum formed by the adductor pollicis (called the mesothenar by Winslow[10]). This muscle lines the posterior surface of the compartment, leaving the first intermetacarpal space posteriorly together with the first dorsal interosseus muscle. This septum is mobile because of the thumb's movements. Thus, in the position of extreme opposition it moves around the tendons of the index finger.[11]

Biomechanics

Basic physiologic data

Every muscle is formed by motor units (a nerve fiber and some muscular fibers), activated in greater or lesser numbers depending upon the necessary force required to produce the needed effect. These units are assembled together into the fascicles of the nuscle fibers, which are clearly seen at the surface of every muscle. Their activation is not only quantitative (number of motor fibers), but it is also influenced, by the insertions of the muscle – the skeleton. Their 'predictive' effect is therefore variable and depends on the initial position, but it changes during movement. As emphasized by Berthoz, there is a kind of motor wave which passes over the entire muscle, and even diffuses to the adjacent muscles. The question asked by Brand, as well as by other authors previously, was whether the intrinsic thenar muscles could be considered as a unique mass. That supports the idea that the motor wave is at the origin of the opposition movement, as it involves the muscles in a successive manner.

Mechanical forces generated by muscles can always be broken down by the classic parallelogram method into:

- vectors perpendicular to the skeletal axis, and therefore the only forces generating the movement (efficient forces), and
- parasite vectors, called axial forces, which are parallel to the skeletal piece and which are exerted on the head of the bone situated above, mediated by the base of the skeletal piece situated below. At this new level the very same axial force may be broken down into an axial force and an efficient force capable of moving the bone above.

This would explain that, in order to move a segment without moving the segments above it, a stabilization must be applied to this segment by the muscles which will counterbalance the parasite forces. Thus, the entire muscular system of the extremity, and even of the whole organism, is gradually activated.

In other words, muscles around the joint – above as well as below it – are activated, even if the movement is simple. In the human organism there is no sector of muscular inactivity.

When a muscle is bi- or multiarticular, the position of the joint above has an impact on the muscular action below (in our case, it is, as we have seen, the position of the wrist for the extrinsic muscles of the thumb).

It must be remembered that in a movement of taking an object, the first phase is the phase of approach (of the whole body and the extremity in particular). At the end stage of the phase the hand and the fingers are put in a suitable position for the rest of the movement, the position of which will vary depending on the manner in which the object is taken and the handling chosen. This is due to the brain, which facilitates anticipation of the movement, and its result when it is possible. When the hand comes into contact with the object, the visual information (the only initial feedback) is extended by the sensory information (superficial and deep sensitivity). So the stage of taking the object begins, which is quickly transformed into handling and manipulating. At first the hand will be opened because the extensors of the fingers and the thumb are moved apart. Afterwards the thumb is opposed, and it will be adapted to the second stage of grasping.

The movement of opposition

As we have seen, in the beginning the movement of opposition of the thumb is favored by the anatomic orientation of the trapeziometacarpal joint. There is a real difficulty, if not to say real confusion, in the naming of the thumb movements, as the planes of reference are not related to the spatial description of the thumb position. The terms flexion, extension, abduction, and adduction are commonly used, but they may induce some confusion if it is not taken into consideration that they are described according to different axes and planes.

When the thumb is considered, one should not refer to classic anatomic planes (transversal or sagittal) that are used, for example, to describe the movements of the ulnar fingers. We prefer to refer to:

- a plane which is approximately perpendicular to the longest length of the trapezium joint surface, and which is parallel to the axes of flexion and extension of the metacarpophalangeal and interphalangeal joints
- a second plane, perpendicular to the first, passing through the base of the first metacarpal bone.

Then one could consider that the thumb has movements of flexion, extension, and ulnar or radial inclination.

We have already seen what the terms of anteposition and retroposition correspond to. At each joint of the thumb there is a rotational movement added, that we shall describe by only considering the longitudinal axis of the skeletal piece, as well as the generating factors, which will be described further.

The only difficulty in describing the movements of the thumb in this manner, in order to have an eventual

means of evaluation, is to know which position is 0. This is a position where there is least tension of ligaments and the muscular action is minimal. It would be useful to define it. (It must be noted that the same difficulties are faced in all types of joint evaluation.)

Evidently, in the clinical examination there is a neutral position of the thumb, and this is the only suitable position to define in terms of the classic anatomic planes. It is assumed that the frontal plane contains the second and third metacarpal bones. The plane of the first web space contains the first and the second metacarpal bones, and forms an angle of about 70° with the previous plane. There is an angle of about 40° between the two metacarpal bones, as the thumb is considered to be in a neutral position. In this neutral position, the metacarpophalangeal and interphalangeal joints are flexed, at about 30° each. So the neutral position of the thumb is the position where the thumb is parallel to the index finger, and the first metacarpal bone is in the intermediate position between flexion and extension. This is the reference point for measurements in clinical assessment.

In addition, in contrast to the numerous studies mentioning volumes of circumduction during opposition, we have seen that the movements of each joint (i.e., flexion-extension, radial or ulnar deviation, rotation) do not have a large amplitude, at least as far as active mobility is concerned. There are several reasons for this: osteoligamentory and muscular – because the thenar muscle mass represents a real obstacle (medially to the first metacarpal bone).

Returning to the rotation of the thumb at the metacarpophalangeal joint the rotation implies two components:

- An automatic one, related to the sliding of the metacarpal joint surface over the 'saddle' of trapezium, for which, as we saw, it has the form of an arch. The 'scoliosis' of the saddle, together with the sliding, leads to the obligatory rotation round the vertical axis, theoretically situated in the center of the curvature. The rotation is confirmed by possible subluxation of the trapeziometacarpal joint, which may be seen and palpated in maximal adduction of the joint[4] (see Figure 16.7).
- The other component is 'intra-articular' and corresponds to a variable adaptation of the trapeziometacarpal joint. This is an adaptation of the two torus-shaped surfaces, where, more schematically, the base of the metacarpal bone is Y-shaped; it is bigger than the 'saddle' and thus may turn around itself. This rotation becomes possible as there is some laxity in the restraining structures such as capsule and ligaments. It is a consequence of the predominant muscular contraction of the medial or lateral sesamoid muscles.

This rotation has been a real point of discussion as it was thought that it could take place because of a lack of contact between articular surfaces – which is not correct because the surfaces are toroidal.

In the retroposition of the thumb, the contact is established by the joint surfaces of the basal apophyses of the metacarpal bone and the 'spheroid' zone of the trapezium.[4] This contact allows a rotation, induced by the muscles.

The notion of the obligatory rotation (sometimes called conjoint), which was often mentioned referring to the works of MacConnail,[12] must be mentioned. This is a notion, according to which a movement like flexion or abduction leads to rotation ipso facto. This may happen only when the movements are assessed according to a reference axis, which is not a real axis of motion in the strict sense of the word. As a simple example, consider flexion of the thigh of x° in relationship to the body in the sagittal axis. If the movement takes place in the axis of the femur neck it necessarily leads to an external rotation of the extremity.

For the thumb, as we stated, the rotation takes place around the axis of the metacarpal diaphysis and there is no possibility of obligatory rotation. However, the situation is different for the joints discussed below.

There are also rotational factors at the metacarpophalangeal joint, which appear when it is flexion and lateral inclination. The first one is an obligatory rotation linked to the fact that the axis of flexion is not perpendicular to the diaphysis because of the asymmetry of the metacarpal head, due to a more prominent medial condyle, and also a collateral medial ligament which is shorter than the lateral one. The second, more important factor of rotation is linked to the insertions and different traction forces of the intrinsic muscles, as some of them pull in the direction of supination, while the others pull in the direction of pronation (external sesamoid muscles). These are articular factors linked to a possible laxity in certain positions which relax the ligaments.

This is the explanation for the flexion-radial inclination and pronation of the metacarpophalangeal joint which is the most frequently used, and the most useful for dealing with the handling of objects.

Finally, in the interphalangeal joint, the obliquity of the transverse axis of flexion in relation to the diaphysis of the first phalanx leads to pronation, which is a true obligatory or conjoint rotation.

One can see that all these rotatory components of the thumb have a different nature at different levels, and are additive, giving an impression that there is a very high degree of rotation in opposition pulp to pulp with the other fingers (in the so-called circumduction movement).

It is important to consider the exact situation and action of every muscle. If the opponens pollicis muscle is taken apart, the intrinsic muscles emerge at the zone which circumscribes the lateral part of the carpal canal. This field of insertion is situated medially and slightly superiorly, regarding the trapeziometacarpal joint. All the bellies are situated medially to the metacarpal bone. Distally, they are all inserted into the base of the first phalanx, directly or via the sesamoid bones and glenoid fibrocartilage. Thus they all take part in flexion of the metacarpophalangeal joint.

Either medial or lateral sesamoid muscles exert a pressure on the head of the first metacarpal bone. Breakdown of the

forces provides the evidence that there is a force, which represents the sum of all the axial forces, which is applied on the anteroinferior surface of the metacarpal head and has a tendency to accentuate the flexion of the metacarpophalangeal joint and also leads to a radial inclination at the metacarpophalangeal level. In addition to these axial forces other forces are added, such as those exerted by the flexor proprius and by the extensors – all have a tendency to drive the first metacarpal bone apart from the second one and, therefore, to increase the measured angle between them.

The intrinsic muscles are like shrouds of a mast, which are fixed to a socket joint. This link should be stabilized by other shrouds with a counterbalancing effect, but the tendons of extrinsic muscles add to the same action of flexion of the metacarpophalangeal joint and extension of the trapeziometacarpal joint (because of their axial forces).

The opponens pollicis is the only muscle capable of counteracting the axial forces and stabilizing the first metacarpal bone, which explains why its volume is important and why its functional value is essential.

Having clarified this, how does the opposition take place? First of all, it must be emphasized, the thumb is normally in a position of opposition. Furthermore, in the thumb-fingers grasp, the movements of the ulnar fingers do a large part of the work. Thus, the large movement of circumduction, commonly described for opposition, is not really a rule.

Clinical observation of the thumb reveals that there are three preferential movements. The first one drives the thumb into contact with the lateral surface of the index finger. The excursion of the thumb goes from the base of the first phalanx to the last one, and the movement ranges from extension of the first metacarpal bone (retroposition) to an almost maximal flexion (anteposition). Beginning with the middle of the index finger, another, very visible movement is present under a form of a true abduction of the first phalanx (radial inclination) – this time at the metacarpophalangeal joint. Throughout this movement, pronation of the entire thumb column takes place. It is the sum of two types of rotation: rotation of the metacarpal and rotation of the first phalanx. It is possible to observe this movement when palpating the extensor pollicis longus and abductor pollicis longus. Those two tendons will lead to an inclination in opposite directions of extension-retroposition (extensor pollicis longus) and flexion-anteposition (abductor pollicis longus). It is also possible to observe, under the skin, a palpable simultaneous contraction of the intrinsic muscles of the first web space and the thenar muscles. This first type of opposition resembles the movement of the windscreen wiper of a car.

The second type is a similar movement of the thumb, but this time in order to oppose the pulp of the thumb to the pulps of the other fingers. The immobility of the thumb is quite marked; it is in the position of extreme flexion of the trapeziometacarpal joint (anteposition), maintaining a large space in front of the second metacarpal bone, from which it stays separated by the volume of the contracted thenar muscles. The rotation in pronation is very significant (because of the action of the lateral or radial thenar muscles). Thus, these are the lateral movements of the metacarpophalangeal joint (radial or ulnar inclination) which make the pulp of the thumb pass successively in contact with the pulps of all the other fingers. It is more the action of extrinsic muscles than the action of the sesamoid ones, which act in the opposite direction – one against the others. As a rule, the interphalangeal joint of the thumb stays in the position of extension and the fingers are demiflexed (the fourth and fifth fingers are oriented in such a manner that they promote the thumb's opposition).

The third position of the thumb is frequent: it is a position, where the thumb's column is in almost neutral position, with the interphalangeal joint in extension and where the other fingers are positioned in front of the relief of the thenar eminence, with all the joints flexed (but in such a manner that they are not flexed to the same degree, as they put the pulps in a position almost parallel to the thumb's column). This is the position that is usually seen when an instrument or a sports racquet is handled. The thumb serves as a buttress to oppose the pulps of the other fingers. The bottom of the palm receives and stabilizes the instrument, improving handling.

Finally, a less frequent position of the thumb must be considered, where it tends to disappear and to take a position in the plane of the palm (maximal extension or retroposition). It is not really capable of doing this because of the anterior situation of the trapeziometacarpal joint in relation to the frontal plane passing through the metacarpal bones. The first metacarpal, therefore, is oblique downwards and backwards, with respect to the second metacarpal bone. However, the hand seems 'flat' because of the relative disappearance of the thenar eminence.

Pathologic postures of the thumb

In the light of these functional and in particular mechanical considerations which have been described, certain deformities or angular postures may be explained:

- Froment's sign in ulnar palsy
- congenital or acquired pollex flexus adductus
- Z-shaped thumb, often observed in rheumatoid arthritis.

Froment's sign, described in ulnar palsy, Froment's sign manifests itself by a marked flexion of the interphalangeal joint when the patient is asked to take and to hold a thin object between the thumb and the index finger. The most probable mechanical explanation is a deficit of the metacarpophalangeal flexors, related first to the thenar muscles, innervated by the ulnar nerve palsy, and, secondly, to the automatic pursuit of a supplementary force by the flexor pollicis longus. This force is localized in the metacarpophalangeal joint because its full effect cannot take place unless the interphalangeal joint is flexed completely to

make it possible to act on the proximal pulley of the first phalanx (P1). By the axial force developed at the interphalangeal joint, flexor pollicis longus will lead to a hyperextension of the first phalanx, which will be aggravated by the axial force of the extensor pollicis longus and the direct force of the extensor pollicis brevis – all those forces being in imbalance with the metacarpophalangeal joint's flexors. The m. opponens pollicis cannot make anything in this case and zig-zag deformity most often accompanies the Froment's sign.

According to Brand,[9] some patients prevent these mechanical effects induced by the palsy, and to control their finger joints, they put their interphalangeal joint in hyperextension and attempt to block it in this position when the object is taken, in order to allow the flexor pollicis longus to act immediately on the metacarpophalangeal joint and prevent hyperextension at this level. But, then, extension of the trapeziometacarpal joint occurs, manifesting as an inverse Z of the thumb.

Pollex flexus adductus has multiple causes; it is also called thumb-in-palm or clasped thumb. The thumb is flexed at the metacarpophalangeal joint at 80° or more. The deformity is permanent and cannot be corrected by voluntary action by the patient: by staying in front of the palm the thumb cannot be opposed to other fingers; its hyperflexion at the metacarpophalangeal joint is a mechanical barrier to undergoing circumduction.

In the congenital pollex flexus adductus, the problem is due to the anatomic or functional absence of the extensor pollicis brevis. Extension of the metacarpophalangeal joint is impossible in spite of the action of a normal extensor pollicis longus. First the forces of the thenar muscles are no longer directly counterbalanced. Second, the extensor pollicis longus develops an axial force which is applied on the palmar aspect of the head of the first metacarpal bone. To that force, the axial force of the flexor pollicis longus is added, aggravating the flexion of the metacarpophalangeal joint and, at the same time, leading to extension of the trapeziometacarpal joint extension (Figure 16.19).

This neonatal anomaly has two clinical manifestations. One is probably a consequence of the fetal position as the thumb is flexed and covered by other fingers. By the lack of movement, this prevents the development of the extensor pollicis brevis. In this case the deformity is reversible if the metacarpophalangeal joint is maintained in extension by a cast or by a striping for several weeks. The other manifestation is related to a congenital absence of the extensor pollicis brevis and is always surgical.

These two forms would very quickly be complicated by 'retraction' of the palmar soft tissues (skin and aponeurosis), which would need a surgical intervention (first space dissection and palmar cutaneous graft to the opposition fold). Only the second case needs a tendon transfer to the extensor pollicis brevis (extensor indicis proprius or extensor carpi radialis longus). Pollex flexus adductus is frequently observed in arthrogryposis.

In the isolated *radial palsy*, the abductor pollicis longus, extensor pollicis longus, and extensor pollicis brevis muscles are paralyzed, as is the extension of the thumb. Only the interphalangeal joint may be extended, because of the dorsal expansions of the abductor pollicis brevis and adductor pollicis, but the force is reduced. Nevertheless, it must be noted that the axial forces coming from palmar muscles (flexor pollicis longus and thenar muscles) may lead to extension of the trapeziometacarpal (but not the metacarpophalangeal joint), which is favorable for handling an object.

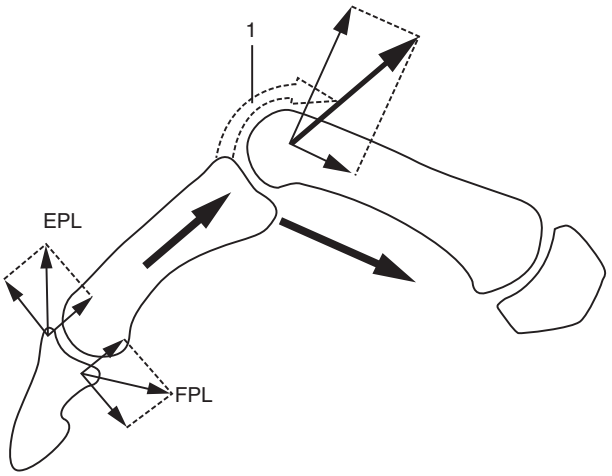

Figure 16.19

Mechanical explanation of the thumb-in-palm formity when extensor pollicis brevis (1) action is missing when extensor pollicis longus (EPL) exists.

In *other palsies* there is no particular vicious posture. They may manifest only by the emaciated thenar eminence or the extreme position of the thumb.

The *Z-shaped thumb* is specific, and is observed in rheumatoid arthritis. The deformities and their causes were studied by Tubiana. They take place in almost 60% of cases of rheumatoid arthritis cases. The Z-shaped thumb is caused by synovitis-induced destruction, which affects the metacarpophalangeal joint first. The increase in dorsal lesions destroys the insertion of the extensor pollicis brevis, and the extensor pollicis longus is moved medially. The flexion of the P1 quickly becomes irreducible. Extensor pollicis longus leads to hyperextension of the P2. Consideration of the axial forces can help to explain the trapeziometacarpal joint's hyperextension and the maximal flexion of the metacarpophalangeal joint.

When the lesions of rheumatoid arthritis occur in the trapeziometacarpal joint, the base of the metacarpal bone becomes dislocated radially and is in a position of flexion and ulnar drift, with retraction of the first web space as a consequence. The rest of the digital column may either take a reverse Z position (hyperextension of the metacarpophalangeal joint, flexion of the interphalangeal joint), or a Z-shaped position, comparable to the position first described with flexion of the metacarpophalangeal and hyperextension of the interphalangeal joint.

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17 Restoration of thumb opposition

Raoul Tubiana

The thumb ray is shorter, more proximal, more mobile than the others (Figure 17.1a). It can project in front of the palm to oppose itself to the other four rays (Figure 17.1b). Opposition of the thumb is a complex movement that follows an arc of circumduction in a palmar and ulnar direction, allowing not only precision and power grip but also extensive contact between the pulp of the thumb and the pulps of the fingers, which are the sensory organs of touch.

Although opposition is not specific to humans, as many other primates have an opposable thumb, the erect posture on lower limbs permitted the opposition of the thumb ray in the human hand to acquire its specificity.

Opposition is the result of:

1. The forward tilt of the radial carpo-metacarpal column (or thumb column).
2. The mechanics of the three joints of the thumb column.
3. Muscular tractions.

Thumb opposition includes successive stages that necessitate the conjoint action of extrinsic and intrinsic muscles of the thumb, innervated by the three principal nerves of the upper extremity:

1. Opening of the first web space is due to extrinsic muscles: the extensor pollicis longus (EPL), the extensor pollicis brevis (EPB), and the abductor pollicis longus (APL). They are innervated by the posterior motor branch of the radial nerve.
2. Anteposition of the first web space is due to the lateral thenar muscles: the abductor pollicis brevis (APB), the opponens (OP), and the superficial head of the flexor pollicis brevis (FPB). They are innervated by the median nerve thenar motor branch.
3. Adduction is due to the adductor pollicis (AP), and the deep head of the FPB. They are innervated by the ulnar nerve motor branch.
4. Retroposition of the thumb column is ensured by the extensor pollicis longus. It is innervated by the radial nerve.

The territory and action of the radial nerve are well defined: it innervates the posterior extrinsic muscles used to extend the thumb column and to open the hand. The median and ulnar nerves together ensure the movement of opposition, and the interdependence of their actions is mirrored in the frequent overlapping of their respective territories. The innervation pattern of the hand's intrinsic muscles is the subject of frequent variations that make surgical indications somewhat imprecise when they are based on involvement of one of these nerves. Several links between the median and ulnar nerves have been described in the forearm[1,2] and in the palm of the hand.[3–6]

For these reasons, it appears preferable to classify thenar muscle paralyses according to the muscles involved and to the function lost, and not according to the nerve lesion.[7]

Classification of paralyses of the thumb

Depending on the extent of paralyses, thumb paralyses are classified as follows:[8]

- Group I: paralyses of intrinsic muscles of the thumb only. They can be subdivided into three groups:
  1. Paralyses of the lateral thenar muscles causing loss of opposition of the thumb.
  2. Paralyses of the adductor associated with partial or predominant ulnar nerve innervation of the FPB.
  3. Paralyses of all the intrinsic muscles of the thumb.
- Group II: paralyses of intrinsic muscles of the thumb associated with paralyses of the extrinsic muscles of the thumb and conservation of active wrist extension.
- Group III: paralyses of the intrinsic and extrinsic muscles of the thumb, without active wrist extension. This group can be subdivided according to whether or not wrist extension can be recovered with a transfer.

Furthermore, one must take into consideration the associated lesions in each group involving, for example, sensory disorders, contractures, stiffness, or instability.

We shall see that restoration of thumb opposition is not indicated in many categories of these paralyses (see below: 'Indications for surgical restoration of thumb opposition').

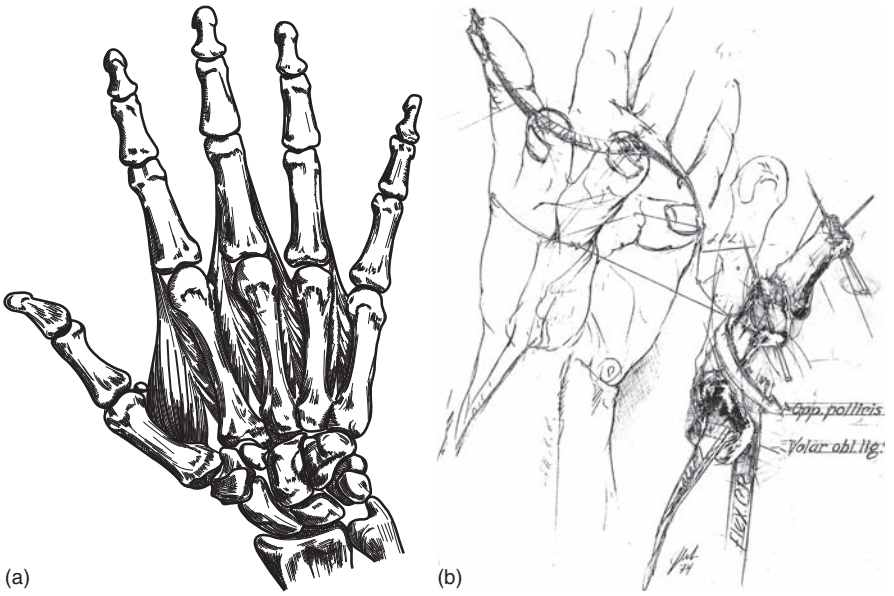


Figure 17.1

(a) The thumb ray is a polyarticular chain made up by the trapezium, the first metacarpal, and two phalanges. The trapeziometacarpal (TM) joint is functionally the most important joint. It was called by Cruveihier a 'reciprocal fitting joint' and a 'saddle joint' by Fick. The articular facet at the base of the metacarpal is concave dorsovolarly and convex radio-ulnarly. It fits together with the saddle-shaped part of the trapezium. (b) Opposition is a complex movement of the three joints of the thumb column allowing its anteposition and pronation and an extensive contact between the pulp of the thumb and the pulps of the fingers, as shown in this beautiful drawing by J.W. Littler (by kind permission of the late Dr Littler).

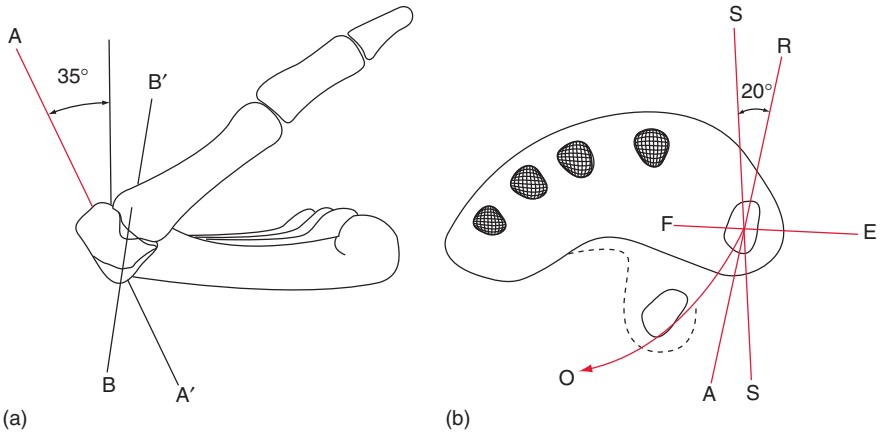


Figure 17.2

Terminology used for the movements of the thumb metacarpal. The trapezial distal articular surface is inclined: (a) 35° palmarly (with respect to the transverse plane); (b) 20° radially (with respect to the sagittal plane). A, anteposition; R, retroposition; F, flexion; E, extension; A–R, anteposition–retroposition axis; F–E, flexion–extension axis; S–S, sagittal plane; O, opposition with pronation.

Terminology of thumb movements

As already mentioned in the preceding chapter, there is some confusion over the terminology of thumb movement. This is caused by the complexity of the movements at the level of the trapeziometacarpal (TM) joint and the necessity to differentiate between the movements of the first metacarpal and those at the level of the two other joints.

The trapezial distal articular surface is inclined:

- 35° palmarly (with respect to the transverse plane)
- 20° radially (with respect to the sagittal plane).

Because of the obliquity of the axes at the TM joint (Figure 17.2), the trapezial system of reference will differ from the three classic planes of movement (transverse, sagittal, and frontal).[9]

Based on the trapezial system, the first metacarpal is able to perform two angular movements in relation with two axis of mobility: anteposition–retroposition and flexion–extension. The term 'abduction' is confusing, as it can be used for any movement of separation between the first and second metacarpals in any direction. In addition to the angular movements, an axial rotation of the thumb column is combined with anteposition. The mechanics of this longitudinal rotation are analyzed in the preceding chapter. It is due to the connected action of the three joints of

the thumb column, to the passive tension of the dorsal trapeziometacarpal ligaments, and to the active pull of opposition muscles.

Longitudinal rotation is only possible if the first metacarpal follows the course of the circumduction arc, with anteposition, in the ulnar direction. Pushing the first metacarpal in a plane parallel and near to the palm *does not* produce longitudinal rotation, as the fascicles of the dorsal trapeziometacarpal ligament remain lax.[10]

Longitudinal rotation is basically produced at the TM joint and not at the metacarpophalangeal (MP) joint as it was supposed to be by Winslow[11] or Bunnell;[12] however, the MP joint and the interphalangeal (IP) joint also contribute to the longitudinal rotation. In fact, the whole osteo-articular chain contributes to the longitudinal rotation in pronation.

The muscles

Movements of the thumb are produced by two groups of muscles: the extrinsics located in the forearm, prolonged by tendons in the hand that are the long muscles of the thumb, and the intrinsics located in the hand (see the preceding chapter).

The extrinsic muscles

There consist of the three long posterior muscles (C7 root) (Figure 17.3) and the flexor pollicis longus (FPL).

The *extensor pollicis longus (EPL) tendon* ends at the dorsal base of the distal phalanx. The EPL helps to extend the two phalanges of the thumb and exerts a lateral deviation at the MP joint level. It also exerts significant action on the first metacarpal, which it brings into retroposition, extension, and supination. Fick[13] named it the 'extensor pollicis adducens'. Without the opposing action of the lateral thenar muscles, extension of the distal phalanx under the effect of the EPL would produce retroposition of the metacarpal and would prevent opposition. When the lateral thenar muscles are paralyzed, this antagonistic action of the EPL impedes the re-establishment of anteposition by a transfer, and sometimes in longstanding lateral thenar paralysis, supination contracture necessitates the rerouting of its tendon outside Lister's tubercule (see Figure 17.17).

The *extensor pollicis brevis (EPB)* is a weak muscle that participates in the extension of the MP joint when the IP joint is flexed, and thus plays a part in the precision grip. It is also an extensor (or radial abductor) of the first metacarpal.

The *abductor pollicis longus (APL)*, whose tendon is slightly more palmar, has little effect of anteposition. The APL plays an important role in stabilizing the TM joint and acts as an antagonist of the adductor pollicis. Furthermore, it is a radial stabilizer of the wrist and an antagonist of the extensor carpi ulnaris (ECU).

These three posterior muscles of the thumb can be divided into two groups whose axes of traction diverge and whose courses differ considerably (Table 17.1). The same transfer should not be used on the three tendons.[14]

The *flexor pollicis longus (FPL)* is the only extrinsic muscle of the thumb that is innervated by the median nerve (Figure 17.4). It flexes the distal phalanx and contributes to flexion of the proximal phalanx. It plays an important role both in precision pinch, which requires movement of the distal phalanx, and in power grip. Its action is not that of a pure adductor, as Duchenne[15] showed, and it has only a minor direct effect on the TM joint. Indirectly, however, it reinforces the adductor power of the EPL by placing it under tension.

The four extrinsic muscle tendons cross the wrist joint. The position of the wrist will influence the action and strength of these muscles. Wrist radial deviation releases these muscles, while ulnar deviation increases their action.

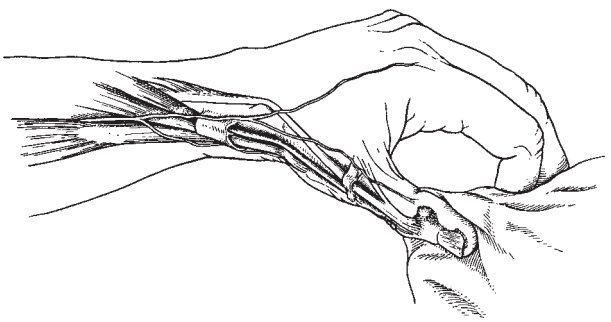


Figure 17.3

The three extrinsic posterior muscles of the thumb from radial to ulnar: APL, EPB and EPL. The EPL tendon passes on the back of the distal end of the radius, in a narrow oblique groove limited radially by the Lister tubercule. It is separated from the EPB by a triangular depression (when the thumb is fully extended) jocularly termed the anatomic snuff-box, crossed deeply by the radial artery and superficially by sensory branches of the radial nerve.

The intrinsic muscles

In the thumb, the intrinsic muscles are stronger than the extrinsics – it is the opposite in the long fingers.

Table 17.1 *Maximum excursion of the extrinsic muscles of the thumb*

Flexor pollicis longus (FPL)	5 cm
Extensor pollicis longus (EPL)	5 cm
Extensor pollicis brevis (EPB)	2.8 cm
Abductor pollicis longus (APL)	2.8 cm

Figure 17.4

The flexor pollicis longus tendon passes between the two heads of the FPB, then enters its osseo-aponeurotic canal.

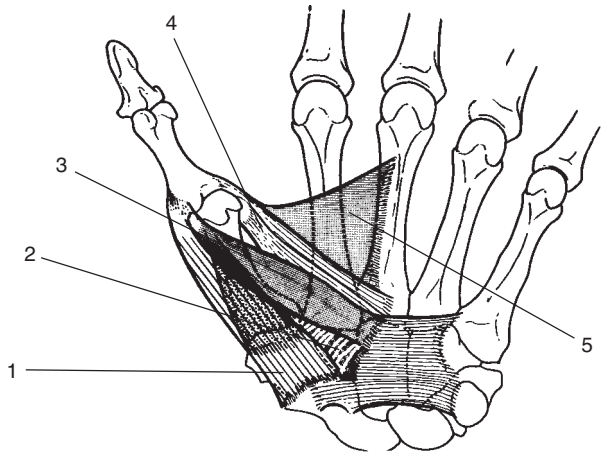


Figure 17.5

Intrinsic muscles of the thumb: (1) APB; (2) OP; (3) FPB; (4) AP oblique head; (5) AP transverse head (slide).

Table 17.2 *Values of the thumb muscles*

Muscles	*Muscular work* (kg m)
Extrinsic	
Flexor pollicis longus (FPL)	1.2
Extensor pollicis longus (EPL)	0.3
Abductor pollicis longus (APL)	0.5
Extensor pollicis brevis (EPB)	0.1
Intrinsic	
Abductor pollicis brevis (APB)	0.5
Opponens pollicis (OP)	0.4
Flexor pollicis brevis (FPB)	0.5
Adductor pollicis (AP)	1.5

The four intrinsic muscles of the thumb can be divided into two groups:[16] lateral and medial (Figure 17.5). However, all the thenar muscles are medial to the first metacarpal.

The three lateral or 'thenar proper' muscles form a cone whose apex is centered at the MP joint. They are arranged in two planes. The *abductor pollicis brevis (APB)* the most superficial, has its fibers nearly parallel to the first metacarpal. It is the muscle of anteposition. It also pronates and causes lateral deviation of the proximal phalanx and contributes to extension of the distal phalanx. Duchenne[15] called it the opposing phalangeal muscle of the thumb, because, by its action, the tip of the thumb can reach the distal phalanges of the fingers.

The deeper *opponens (OP)* acts only on the first metacarpal, because it does not have a phalangeal insertion. Thus, it is the main 'opposing muscle' of the first metacarpal. It also plays a role in stabilizing the first metacarpal.

The *flexor pollicis brevis (FPB)*, situated on the same plane as the opponens and medial to it, has a more oblique course. It is therefore a weaker muscle for anteposition than the APB, and a stronger adductor than the opponens. It also contributes to the flexion of the proximal phalanx. It permits the thumb to contact the middle phalanx of the fingers. It present two heads – a superficial and a deep head – which form a muscular groove for the tendon of the FPL.

The *adductor pollicis (AP)* – the strongest muscle of the thumb (Table 17.2) – presents an oblique and a transverse head, inserting on the medial sesamoid and ulnar aspect of the base of the proximal phalanx of the thumb. It approximates the first metacarpal to the second and third, and also produces inward rotation into supination, which is the opposite action to that of the lateral thenar muscles. In addition, the oblique head of the adductor sends a dorsal expansion to the EPL (Figure 17.6), thus contributing to extension of the distal phalanx of the thumb. This dorsal expansion is symmetric with that of the APB on the radial side.

During opposition, a wave of contraction sweeps through the thenar muscles, from lateral to medial (Figure 17.7). The lateral thenar muscles essentially cause pronation and anteposition, placing the thumb in the position for grasp; the medial thenar muscles are supinator and adductor, placing the thumb on the object and grasping it.

It is essential to remember that in the thumb, the intrinsic muscles are stronger than the extrinsics – which is the opposite of the situation in the fingers.

The adductor has a force greater than that of all the three lateral thenar muscles together. If palsy of the adductor is combined with palsy of the FPB, the imbalance becomes considerable.

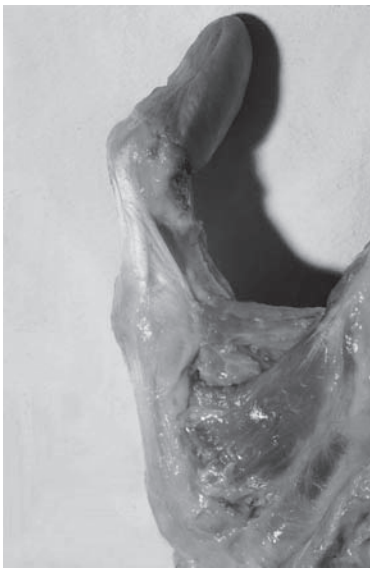


Figure 17.6

The oblique head of the adductor sends a dorsal expansion to the EPL, symmetric with that of the APB on the radial side.

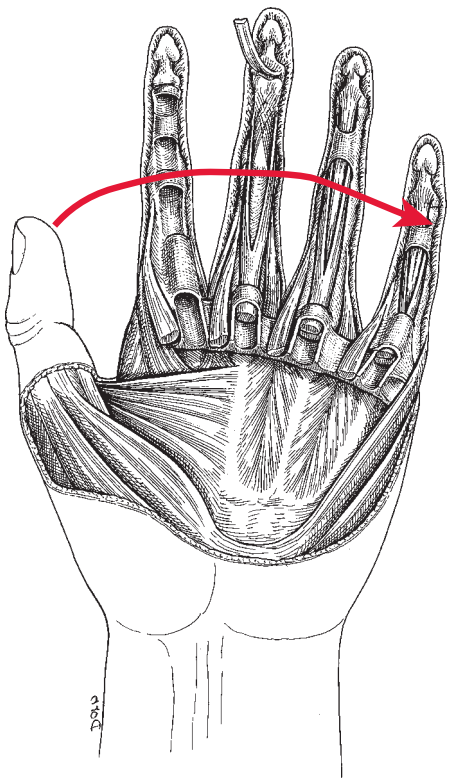


Figure 17.7

During opposition (follow the arrow), a wave of contraction sweeps through the thenar muscles from lateral to medial. (1) APB; (2) FPB; (3) FPL; (4) AP.

An isolated median nerve lesion at the wrist will theoretically jeopardize anteposition only. In fact there are many anatomic variations in the nerve distribution. Between the lateral thenar muscles innervated by the median nerve and the medial thenar muscles innervated by the ulnar nerve, the deep head of the FPB forms a true transitional zone. It frequently receives a double innervation. In most cases, it is the ulnar territory that spills over. In a patient with a median nerve palsy, if the ulnar nerve supplies the two heads of the FPB, the movement of opposition is still possible. Although movement of opposition is reduced by half in both anteposition and pronation, this may be misleading. The existence of such a movement risks misdiagnosis of a median nerve palsy if the absence of contraction in the APB and the OP is not sought systematically. Likewise, in isolated paralyses of the ulnar nerve, the presence of opposition due to the lateral thenars may cause a misdiagnosis if one does not look for atrophy of the first web and other signs of ulnar palsy.

Clinical diagnosis may be even more difficult when there are anastomoses between the median and ulnar nerves. In the forearm, the Martin-Grüber anastomosis is not unusual, for it has been suspected in 6 out of 40 cases after selective blocking of the ulnar nerve[17] with maintenance of innervation of the first dorsal interosseous and/or the adductor. The Riche-Cannieu anastomosis in the palm between the terminal branches of the median and ulnar nerves can provide mixed innervation of the adductor.

Clinical evaluation of intrinsic muscle paralyses

The thenar muscles are the flexors and stabilizers of the MP joint. Their paralysis can cause an instability that seriously compromises pinch, when the thumb proximal phalanx is put into hyperextension[18] and tends to push the metacarpal head medially. Before any reconstructive surgery, it is essential to test the stability of this articulation, which is indispensable for a strong thumb–finger pinch. This stability reveals a great deal of individual variation. Patients with a lax MP joint are at the greatest risk for instability after paralysis.

At the level of the IP joint, extension depends partly on the thenar muscles, which, as shown by Duchenne,[15] have attachments through their dorsal expansions into the extensor apparatus like those of the intrinsic muscles at the level of the fingers. Although these expansions to the EPL arise from both the APB and the adductor, an extension lag of the distal phalanx is not found after an isolated paralysis of the median nerve. On the contrary, after an isolated ulnar nerve paralysis, a flexion deformity of the distal phalanx occurs when an object is grasped. This sign has been described by Froment[19] (Figure 17.8).

The interpretation of the Froment sign has been the subject of numerous commentaries.[20] Thus, for Bunnell,[12] flexion of the distal phalanx is due to the collapse of the thumb column and is secondary to the hyperextension of the MP joint that causes tension on the FPL. It is, in effect, sometimes possible to diminish the flexion of the distal joint by stabilizing the MP joint in slight flexion. One can,

however, observe effort-induced flexion of the distal phalanx of the thumb unaccompanied by hyperextension of the proximal phalanx, thus justifying Froment's interpretation of the sign that he had described (lack of extension of the distal phalanx by the paralyzed adductor).

It is clinically difficult to evaluate the state of an FPB hidden under an intact APB. Electromyography is of little help. One can estimate in an empiric fashion the predominance of ulnar innervation of the FPB if, when Froment's sign is elicited, contraction of the FPB does not prevent hyperextension of the MP joint. In a case of ulnar paralysis in which the thumb MP joint is hyperextended (Jeanne's sign: Figure 17.9) when the patient is asked to form a pinch between the thumb and fingers, one may assume that, in addition to adductor paralysis, the FPB is in large part paralyzed.

The flexion deformity of the thumb's distal phalanx is functionally disabling, because it hinders the most useful type of pinch. Furthermore, it indirectly contributes to increasing supination of the thumb when the pronator thenar muscles are also paralyzed. Indeed, pressure from the index is exerted during finger–thumb pinch on the dorso ulnar aspect of the distal phalanx of the thumb, forcing the thumb into supination, a movement contrary to opposition (the 'crank-handle' effect of Brand:[21] Figure 17.10). It is therefore essential to correct this deformity and ree-stablish pronation of the thumb.

Stabilization of the MP joint and correction of supination of the thumb are absolutely necessary for the re-establishment of a functional opposition.

In *associated paralyses of the median and ulnar nerves*, sequelae are compounded. The thumb column is in the plane of the palm, drawn backward and in supination by the EPL; the MP joint is hyperextended, while the IP joint remains flexed. 'Not only is the first metacarpal positioned in the plane of the second, but also the thumb is rotated on its longitudinal axis. As a result, the anterior aspect of the thumb faces forward as the fingers do.'[15]

Anesthesia of the palmar aspect of the hand is variable. When it is total, the only useful sensitive pinch that persists is between the thumb and the posterolateral aspect of the proximal phalanx of the index finger, which is innervated by the radial nerve. This further accentuates retroposition of the thumb, which progressively becomes fixed in a position contrary to opposition.

Gradually, these paralyses become complicated by deformities, trick movements, soft tissue contractures, and joint stiffness. These all make the restoration of opposition more difficult, and thus must be corrected as soon as possible.

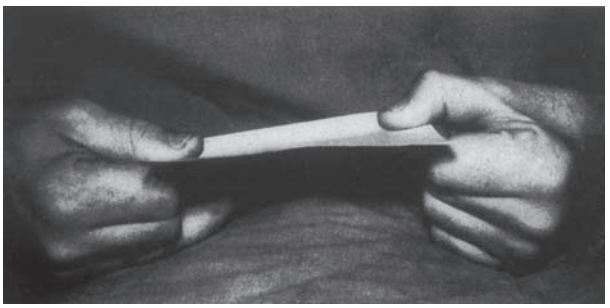


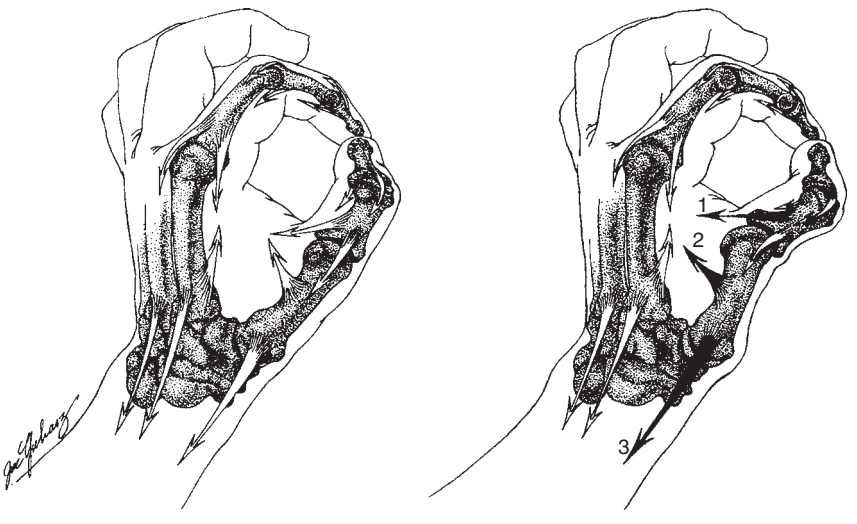
Figure 17.8
Froment's sign.

Etiology

Paralyses of thumb opposition have several causes: traumatic, infectious, vascular, etc., acting at different levels.

The etiology varies widely from country to country.

In traumatic cases, the first decision is to attempt direct repair of the nerve lesion. In spite of advances in nerve repair, the percentage of motor recovery in the intrinsic muscles after proximal nerve lesions remains problematic in adults, and may need 2 years, thus justifying for some



(a) (b)

Figure 17.9
Terminal thumb–index pinch in ulnar palsy. (a) Flexion of the distal phalanx (Froment's sign). The thumb MP joint is stable. One can estimate that the FPB has still some activity. (b) The MP joint is hyperextended (Jeanne's sign). One may assume that, in addition to adductor paralysis, the FPB is in large part paralyzed. (Courtesy of Dr L Mannefelt.)

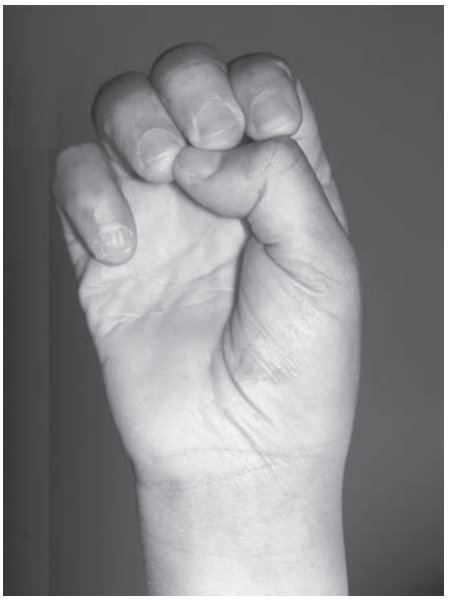


Figure 17.10
The crank-handle effect.

surgeons an early reconstructive procedure (the timing of palliative surgery is dealt with in detail in Chapter 23).

Leprosy is probably the most common cause of thumb paralysis in the world. A general medical treatment is essential before any surgical repair is undertaken.

In poliomyelitis in which considerable improvement follows the acute phase, it is safer to wait in order to evaluate the prognosis before performing reconstructive surgery. However, in children especially,[22] fixed deformities may affect the skeleton and render the restoration of opposition impossible. Thus, it is important to prevent fixed deformities from the onset of disease.

Certain neurologic conditions such as Charcot–Marie–Tooth disease may benefit from surgery if the progression is very slow.

Restoration of thumb opposition

History of opponenplasty

For a long time, surgeons showed little interest in restoring opposition of the thumb. Spitzy[23] suggested that the loss of opposition should be treated by an arthrodesis of the first carpo-metacarpal joint.

Steindler[24] was the first to perform an opponenplasty by means of transfer of the FPL tendon split longitudinally: the radial half of this tendon was pulled around the lateral side of the metacarpal and fixed at the dorsal aspect of the base of the proximal phalanx.

Ney[25] was the first to use transfer of EPB.

Huber[26] and Nicolysen[27] independently advocated the transfer of the abductor digiti minimi muscle to the thumb. This procedure was revived by Littler and Cooley[28] in 1963.

Bunnell[29] described his first attempts at restoring opposition of the thumb. He began by transferring the flexor superficialis of the ring finger subcutaneously and transversely across the palm, and inserting it on the lateral aspect of the first metacarpal. This produced adduction and pronation, but not anteposition. Dissatisfied with the results, he decided to construct a pulley near the pisiform, using as a motor unit the FDS, the flexor carpi ulnaris (FCU), or another muscle prolonged by a graft. Finally, he altered the distal insertion, moving it from the first metacarpal to the posteromedial aspect of the base of the proximal phalanx. Thus, Bunnell's pulley operation developed gradually until he laid out its principles in his classic article in the *Journal of Bone and Joint Surgery*.[12]

Some worthwhile technical improvements were made later, particularly those of Royle,[30] Thompson,[31] Littler,[32] Le Coeur,[33] Merle d'Aubigné et al,[14] Riordan,[34] White,[35] Zancolli,[10,36,37] Brand,[21,38] Ramselaar,[39] Tsuge and Hashizume,[40] and Burkhalter.[41]

However, in spite of all these efforts, tendon transfers still often fail to restore good function to a paralyzed thumb. This is frequently due to the choice of a procedure inadequate to meet the specific needs of the patient. The surgeon who practices a routine operation for re-establishing opposition will not satisfy the needs of the different clinical varieties of thumb paralyses. We have tried since 1968 to define the surgical indications according to the paralyzed muscles, remaining motor muscles and other potential restoration needed.[7,8,42,43]

Indications for surgical restoration of thumb opposition

As already mentioned, the major problem is to decide which different forms of pinch should be re-established. It is evident that an operation directed at making possible strong and precise opposition of the thumb and a pulp-to-pulp grip can be undertaken with a reasonable chance of success only if full opposition movement can be accomplished passively. Numerous obstacles may hinder this movement, such as contractures of skin and muscles of the first commissure (see Figure 17.17), joint stiffness, or instability. When these obstacles cannot be overcome, it comes necessary to limit surgical ambitions and simply try to achieve a useful lateral thumb–index pinch.

The necessary conditions for a useful reconstruction of an opposition grip are:

1. A mobile and stable thumb column.
2. A free thumb web allowing a passive complete movement of opposition away from the palm.
3. A transfer with sufficient excursion, reinforced by active wrist extension (tenodesis effect).
4. A mobile and stable opposable element.
5. As much as possible, a useful pulp sensibility.

These conditions are rarely obtained in complicated and extensive paralyses.

For this reason, it may be preferable in these cases to forego restoring opposition. A lateral thumb–index pinch is more easily achieved, as it can be activated by the extrinsic muscles alone (see Chapter 19 on restoration of thumb lateral pinch). Treatment must be adapted to each case, taking into account the condition of the intrinsic and extrinsic thumb muscles, the presence and distribution of sensory loss, the sites of scarring, the mobility of the joints, and the occupation of the patient. One should not consider isolated treatment of thumb paralyses: the condition of the other components in the hand, with which the thumb must come into contact, in order to accomplish pinch, must always be taken into account, as well as the extent of the palsy in the upper limb. Only in favorable cases, as defined above, will suitable musculotendinous transfers allow restoration of thumb opposition grip.

Different techniques are used to restore thumb opposition – essentially tendon transfers; however, muscle transfers, tenodesis, and joint arthrodesis have some useful indications.

Tendon transfers

Restoration of thumb opposition by means of tendon transfers differs, depending on the extent of the palsy and the condition of the joints of the thumb column.

Several variations can be described in terms of the three essential elements of the procedure: the motor muscle, the course of the transfer (which may involve a pulley), and the distal fixation.

Motor muscle

It is preferable to use a motor whose removal causes the least functional loss, with a tendon long enough to be fixed directly to the thumb, and whose power and excursion are adapted to the paralyzed muscles. The power needed for re-establishing thumb anteposition is less than that required for a thumb flexion–adduction grip.

Several transfers may be used from the palmar or the dorsal aspect of the forearm, each with its own advantages and disavantages.

Palmar transfers

Flexor digitorum tendons

The *flexor digitorum superficialis (FDS)* to the ring finger is usually chosen,[29–31] as its loss is less noticeable than in the other fingers – except, of course, in high ulnar palsy, when its removal would abolish flexion of the digit altogether because of the flexor profundus paralysis. One must beware of using a paralyzed and reinnervated superficial flexor, which is rarely strong enough to function as a good opposition transfer. Also, before the superficial flexor is removed, the two flexors must be carefully tested separately. The FDS to the little finger is usually quite weak.

One should remember that the removal of the FDS causes not only a loss of power of the finger, but also a loss of functional independence, as flexion of the finger now depends solely on the flexor profundus.

An incision at the base of the ring finger allows removal of the tendon just proximal to Camper's chiasma, in the gap between the two proximal pulleys A1 and A2.

The *flexor digitorum profundus (FDP)* tendon is only used when the donor digit is to be amputated. It must never be taken from a finger whose superficial flexor action is to be preserved.

The flexor pollicis longus

This was the first tendon transfer used to restore opposition by Steindler.[24] It since has been used by several authors.[44–46]

The palmaris longus

This is the muscle whose transfer causes the least functional loss. However, its tendon is too short to reach the thumb. It may be prolonged by pretendinous bands of the superficial palmar fascia[47] (Figure 17.11).

It can be used in spite of its weakness to restore anteposition.

All these palmar transfers innervated by the median or ulnar nerves only provide a re-arrangement of the forces of flexion. They do not bring into play any supplementary forces, as is the case with dorsal transfers.

Dorsal transfers

The dorsal transfers, because of their location, are often spared in wrist wounds involving the median nerve.

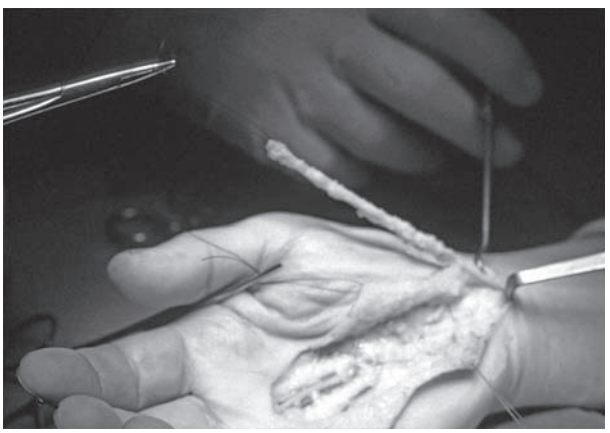


Figure 17.11

Camitz procedure. The PL tendon is prolonged by the pretendinous bands of the palmar fascia.

The *extensor indicis proprius (EIP)* is one of the best choices for opponenplasty.[10,41,48,49] The tendon removed from the ulnar aspect of the corresponding common extensor has an adequate length and strength, and the extension deficit on the index does not lead to any damaging consequences (when the technique is correct) (see Figure 17.18).

The *extensor digiti-quinti (EDQ)*, although weaker, has been used.[50] However, the EDQ can be the only fifth finger extensor tendon, and is often not long enough.

The *extensor pollicis brevis (EPB)* has frequently been proposed as a transfer for opponenplasties[10,38,42,51] (Figure 17.12). It is important to realize that its removal is not without harmful effects. Indeed this muscle is indispensable for maintaining extension of the MP joint while the distal phalanx is flexed during precision pinch; this action should be retained whenever possible. Besides, this tendon is somewhat variable:[52] it is often too slender, and may have atypical insertions into the first metacarpal, which would make its transfer difficult. Its main disadvantage is suggested by its name (brevis); however, in a few cases, the tendon extends to the distal phalanx. Its tendon's usual distal insertion on the mid-dorsal aspect of the base of the proximal phalanx is an ideal site for attaching an opposition transfer when the MP joint is stable. Thus, since Ney,[29] several authors have made use of the distal end of the EPB to extend a transfer.[12,53] However, this tendon can slip laterally over the side of the MP joint, and thus becomes a flexor. To avoid this complication, Kaplan et al[54] recommended wrapping the tendon of the EPB around that of the EPL before suturing it to the motor tendon (Figure 17.13).

The *extensor pollicis longus (EPL)* has a longer tendon than the EPB and a better excursion. However, after its transfer, retroposition of the thumb column is always diminished, and the technique of its distal fixation is quite complex to avoid the risk of a distal thumb joint flexion deformity. EPL transfer is preferably used for restoring opposition in high median and ulnar nerve palsy (see the Bureau and Decaillet procedure – Figure 17.23).

The *abductor pollicis longus (APL)* is the essential stabilizer for the TM joint; also, its tendon is short with limited excursion, so that it has seldom been used for an opponen transfer. Edgerton and Brand[55] advocated the advancement of the APL distal insertion after rerouting the tendon around the tendon of the palmaris longus, which acts like a pulley.

The frequent existence of several APL distal tendons may allow the elevation of one of them, prolonged by a graft.[56]

The other muscles of the forearm, the brachioradialis, and the wrist flexors and extensors may be used to restore thumb movement, but their short tendons need to be lengthened.

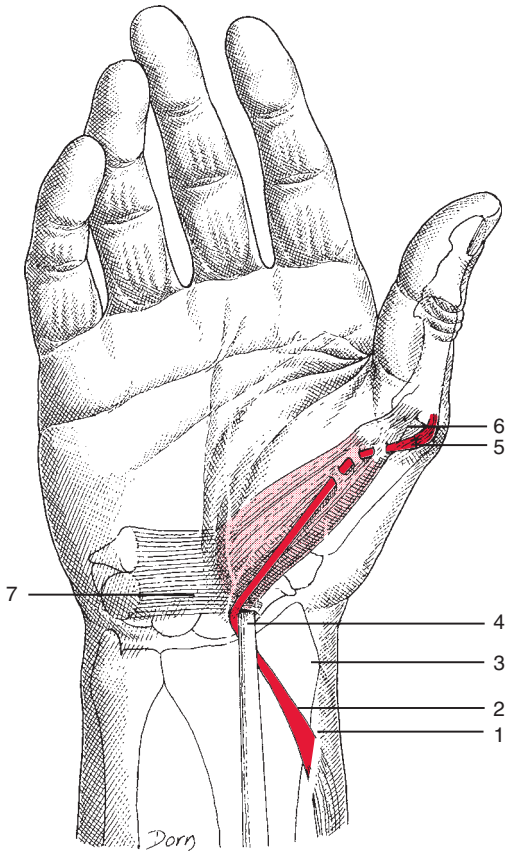


Figure 17.12

EPB transfer. The EPB tendon is rerouted and must be fixed back to its own distal end. (1) Sensory branch of radial nerve. (2) Rerouted EPB tendon. (3) Forearm incision. (4) FCR tendon. (5) The EPB is fixed back to its own distal end. (6) Thumb incision. (7) Palmar cutaneous branch of median nerve.

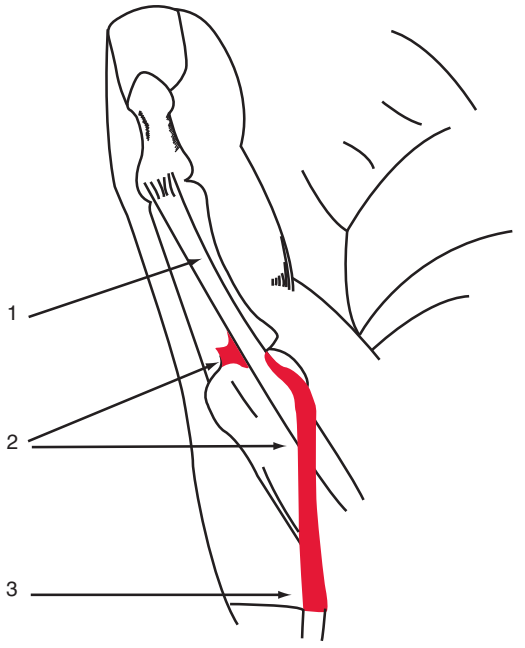


Figure 17.13

The EPB tendon is wrapped around the EPL in order to prevent its sliding over the side of the MP joint.[54] (1) EPL; (2) EPB; (3) suture of the EPB with a motor tendon.

Course of the transfer

The course of the transfer is of considerable importance, for it determines the movement of the thumb created by the transfer.

The direction of the transfer must be as direct as possible, avoiding marked angulation. Nonetheless, changes in direction are often necessary to obtain the action desired from the transfer; they are determined by the pulleys.

For the restoration of thumb opposition after a lateral thenar muscle palsy, the aim of the surgical treatment is to restore anteposition with rotation into pronation of the thumb column. The direction of the transfer is that of the APB parallel to the first metacarpal. The combined action of the transfer with that of the conserved adductor pollicis allows opposition to be re-established. A pulley is only needed if the transfer does not pull in the axis of the first metacarpal. Duparc et al[57] emphasized the value of a direct trajectory through the interosseous membrane (IOM), when using a posterior forearm muscle. With this straighter trajectory, the transfer gains in length and strength. The direct path of the transfer allows maximal advantage to be taken of the active tenodesis effect of wrist flexion and extension. No pulley is necessary (Figure 17.14). When crossing the IOM, the orifice must be at the proximal border of the pronator quadratus to avoid marked angulation against the fibrous membrane. The window must be large enough to allow easy gliding of the motor muscle belly and its tendon. Injury to the interosseous pedicles should be avoided.

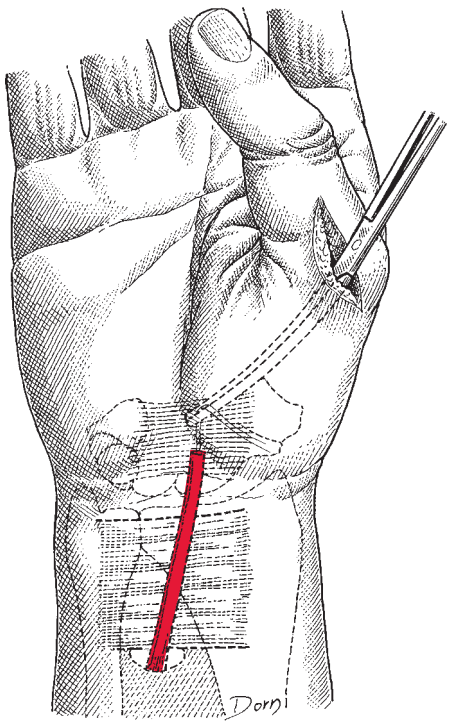


Figure 17.14

A dorsal transfer can be transferred volarly through the interosseous membrane (10M). The 10M opening is usually made proximal to the upper border of the pronator quadratus.

The pulley

A pulley is necessary when the transferred muscle does not pull in the axis of the movement to be restored. Altering its position will influence the relative degree of anteposition or adduction of the thumb. The more proximal, superficial, and near to the flexion–extension axis of the TM joint the pulley, the more marked is the anteposition movement, with a concomitant decrease in the force of adduction (Figure 17.15). The more distal, deep, and medial the pulley, the more pronounced is adduction. These observations have been confirmed by cadaver experiments.[58]

One should also take into account the friction of the pulley on the transferred tendon, which is proportional to the angulation. This friction reduces the power exerted by the motor muscle. The pulley should be in a fixed

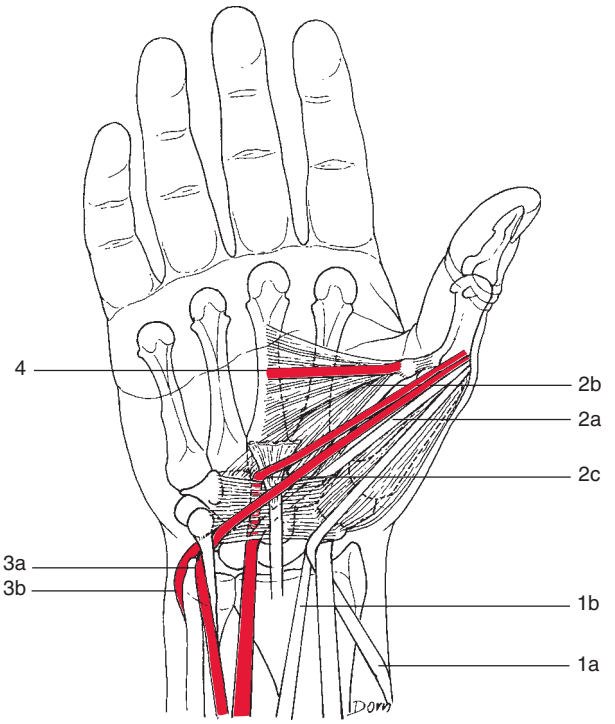


Figure 17.15

Schema showing path of the transfers in each variety of intrinsic paralysis of thumb musculature. (1) Lateral thenar paralysis: (1a) A superficial and proximal pulley around the FCR tendon is needed if the motor is in a lateral location. (1b) Direct path without pulley. (2) Adductor pollicis paralysis: (2a) With complete deficit of the FPB. (2b) The FPB has a strong enough contraction to prevent hyperextension of the MP joint. (2c) Transfer around the ulnar border of the palmar aponeurosis. (3) Paralysis of all intrinsic muscles of the thumb: (3a) Transfer around the FCU tendon. (3b) Transfer around the ulnar head. (4) Restoration of lateral pinch.

position, and large enough, and the angulation should not be excessive.[39]

In *median nerve palsy* restoration of thumb opposition depends on the re-establishment of anteposition by the transfer. A pulley is only useful if the transfer is not in the axis of the TM joint. For example, the dorsal tendon of the EIP can be rerouted palmarly around the FCR tendon or around the ulna or through the IOM, in order to take a path parallel to the APB (Figure 17.16).

In *ulnar nerve palsy* anteposition is conserved by the lateral thenar muscles, and adduction is possible because of the EPL; however, the strength of the thumb–finger grip is reduced considerably because the paralyzed adductor is the most powerful muscle in the thumb. If, as is frequently the case, the two heads of the FPB are predominantly innervated by the ulnar nerve, the FPB palsy is added to that of the adductor.

The aim of the transfer is not to re-establish movement of the adductor, which consists of bringing together the first and second metacarpals while placing the thumb in supination; rather, it is to improve the function of the thumb by increasing the strength of the grip without compromising its pronation and anteposition. The path of the transfer will be transverse or oblique in the palm, according to the state of the FPB.

In *combined median and nerve palsy*, the path of the transfer is oblique in the palm. The best site for the pulley according to Bunnell is at the proximal pole of the pisiform.

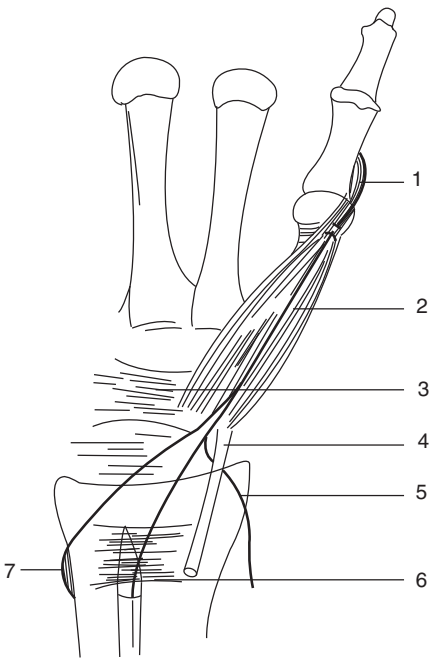


Figure 17.16

The various paths for EIP transfer: (1) distal insertion of the EIP on the EPL; (2) APB; (3) palmar retinaculum; (4) FCR tendon; (5) path of the EIP around the radius and the FCR tendon; (6) through the IOM; (7) around the ulnar. When the FCR is paralyzed, its tendon cannot be used as a pulley.

In the original procedure, the transferred tendon was passed around the distal part of the FCU tendon.

The pulley can be constructed with a tendon graft or a strip removed from a nearby tendon. One often has the advantage of using the existing anatomic structures as pulley, such as the tendons of the FCU or the FCR when they are not paralyzed (otherwise the tendon has a tendency to become overstitched), the medial border of the wrist, the superficial palmar fascia, a deep vertical septum of this fascia,[55] Guyon's canal, or the pisiform bone. Snow and Fink[59] cut a hole in the flexor retinaculum to serve as a pulley.

Some complications at the pulley site must be borne in mind: stretching of the pulley, displacement from the point of reflection when the pulley is not fixed (thus altering the course of the transfer), and finally the formation of adhesions between pulley and transfer. This last complication is most common when the pulley is contructed from a split tendon or lies in a scar area.

Fixation of the transfer

Several important mechanical factors should be considered when attaching the transfer, including the site of insertion of the muscle transfer, the angle of insertion of the transfer in relation to the bone, and the condition of the joints. Two factors must be considered: the level of insertion, and the position of fixation in relation to the axis of rotation of the thumb column.

Site of insertion

Level

The metacarpal is moved like a simple lever by an applied force. The effect of this force is roughly proportional to the length of the lever arm as represented by the distance between the point of insertion of the tendon and the TM joint. The more distal the insertion, the greater is the resultant force for a given muscular pull, and vice versa.

Position of fixation in relation to axis of rotation

To obtain internal rotation with pronation of the thumb, which is essential for opposition, a proximal transfer must be attached to the thumb column on its radial aspect. When the transfer is attached to the thumb column on its ulnar side, it produces an external rotation with supination of the thumb contrary to opposition.

Angle of insertion of the transferred tendon

By widening this angle, the active component of the force that is perpendicular to the skeletal element, is also increased. This was used by Le Coeur[33] when he placed a bony wedge between the transfer and the metacarpal.

Condition of the thumb column joints

In order for the transfer to act on the thumb, it is essential not only that the TM joint retain its mobility, but also that all thumb joints be stable. In particular, the TM joint is stabilized by the APL and the OP, and the MP joint is stabilized by the adductor and FPB. If these muscles are paralyzed, their stabilizing action must be replaced. Stability of the TM joint is rarely affected by palsy of the intrinsic muscles of the thumb, because an active APL persists. At this level, it is adduction contracture and stiffness that are feared.

On the other hand, instability of the MP joint is frequent and differs from one patient to the next; it also depends on the type and extent of the paralyses of the intrinsic muscles.

If the MP joint anterior capsule is lax, the proximal phalanx is placed in hyperextension, forming a swan-neck deformity of the thumb, accentuated during pinch by pressure of the index on the dorsal end of the flexed distal phalanx of the thumb. This digital pressure also produces supination of the thumb. It is therefore necessary for the MP joint to be stabilized when in use, either by the transfer's insertion acting as a ligamentoplasty or by tenodesis,[60–62] by capsulodesis,[10] or by an arthrodesis in which the first metacarpal and proximal phalanx form a functional unit, on which the transfer can act.

In conclusion, restoration of opposition in a paralyzed thumb must take into account: the type of palsy, the muscles available for transfer, the path of the transfer, the mode of fixation, and the condition of the joints and of the soft tissues. Each hand is a specific problem, and cannot be treated by a routine procedure.

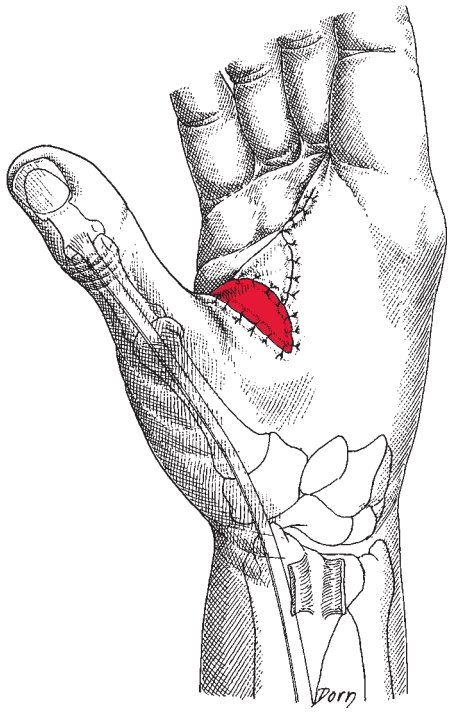


Figure 17.17

Liberation of the first web space with Z-plasty and skin graft and rerouting of the EPL tendon on the radial side of Lister's tubercle.

Restoration of opposition according to the varieties of thumb paralyses

Paralyses of lateral thenar muscles

The aim of the palliative treatment is primarily to restore anteposition.

The *motor muscle* can be relatively weak, since it acts only in positioning and does not have to supply the strength of the pinch, which is furnished by the intact adductor. Thus, the EIP, EPB, or palmaris longus may each suffice.

In absence of physical therapy and splinting in opposition, the action of the adductor and of the EPL, no longer antagonized by the paralyzed muscles, encourages supination contracture of the first commissure. Such a contracture must be corrected before any tendon transfer, using skin grafts. A rerouting of the EPL tendon, on the radial side of Lister's tubercle, is sometimes necessary (Figure 17.17).

The *direction* of the transfer is that of the APB toward the radial aspect of the thumb column.

A *pulley is useful only* if the transfer is not in the axis of the first metacarpal. It must be proximal to the wrist and superficial.

A *pulley is no longer necessary* when a dorsal tendon is passed through the interosseous membrane.

The *distal insertion* should be made on the radial side of the thumb column. The modalities of fixation depend on the muscle used as a transfer. When the EPB is used, it should be fixed back to its own insertion in order to re-establish an extension of the MP joint independent of that of the IP joint (see Figure 17.12).

Technique preferred by the author

In palsy of the lateral thenar muscles, I prefer to transfer the EIP, which is more constant than the EPB or the PL, and has a longer tendon. The tendon is transferred volarly. I prefer to use a direct trajectory through the interosseous membrane (Figure 17.18). However, for cosmetic reasons, I have often avoided two forearm incisions by passing the transfer around the radial aspect of the wrist and around the FCR tendon.

The transfer is passed through the tendon of the APB, dorsal to the flexion–extension axis of the MP joint, and is fixed to the dorsal expansion of the APB to the EPL (Figure 17.19).

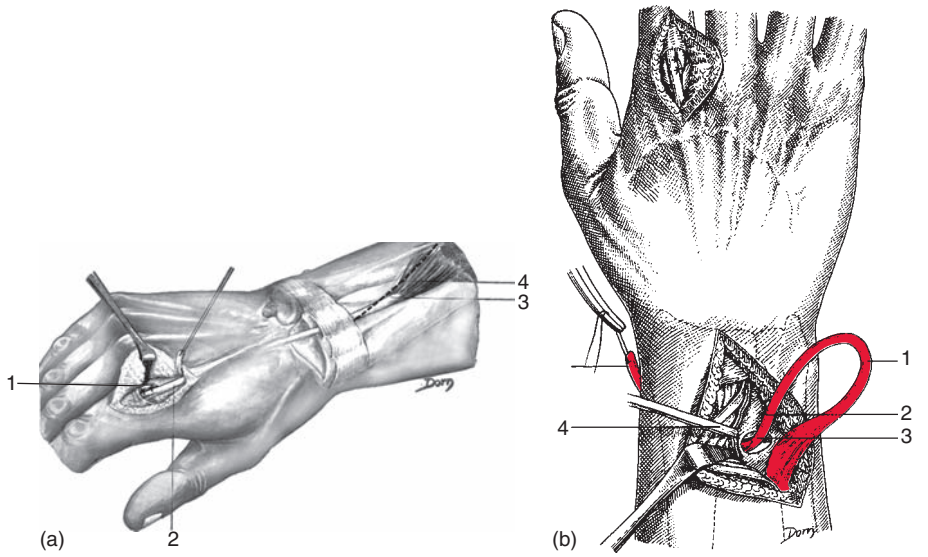


Figure 17.18

EIP transfer. (a) Elevation of the EIP tendon, which is ulnar to the EDC tendon. (1) The distal cut end of the EIP is fixed to the tendon of the EDC of the index. (2) EDC of the index. (3) EIP. (4) Forearm dorsal incision. (b) (1) The EIP tendon is extracted proximally into the forearm. It is easy to recognize, because it has the most distal muscle belly of all the digital extensors. It is then transferred volarly, preferably through the interosseous membrane. (2) The opening in the interosseous membrane to usually made proximal to the upper border of the pronator quantities; (3) it must be large enough to allow easy passage of the muscular body of the EIP, about 3 cm long and 1.5 cm wide. Care is taken to protect the interosseous neurovascular pedicles (4).

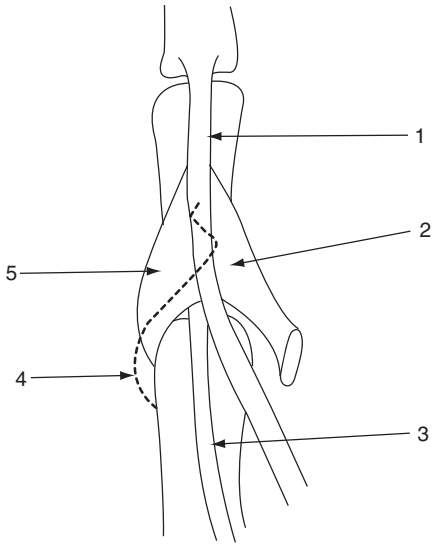


Figure 17.19

Fixation of the transferred EIP tendon to the APB and then on the dorsal expansion of the APB to the EPL. (1) EPL tendon. (2) Expansion of the AP to the EPL. (3) EPB. (4) The EIP tendon is passed through the dorsal APB expansion and is fixed on the EPL.

The tension of the transfer is adjusted so as to provide maximal anteposition of the thumb opposite the ring finger, with the wrist in slight flexion (5°), and the MP and IP joints in extension. A splint maintains the thumb in anteposition and the wrist in neutral position for 4 weeks. Rehabilitation is then started. The wrist must remain in rectitude during the exercises so as to avoid a tenodesis effect. It seems useful to encourage slight motion of the thumb during the splint period in order to activate the cortical reintegration of the transferred muscle.[63,64]

Paralyses of the adductor associated with partial or predominant ulnar innervation of the FPB

In ulnar nerve palsy, the adductor is paralyzed but anteposition is conserved.

Opposition is possible, but the strength of the thumb–finger grip is considerably reduced.

The choice of the motor muscle, the path, and the fixation of the tendon transfer for an adductorplasty must take into account the condition of the FPB muscle (see Chapter 19).

Distal median–ulnar palsy

All intrinsic muscles of the thumb are paralyzed. The thumb column is activated only by the extrinsic muscles. The thumb column is in retroposition rotated in supination, the phalanges flexed, and the atrophy of the thenar eminence is extreme.

'The tonic prevalence of the EPL, gives the human hand the appearance of a monkey's hand.'[15]

Anesthesia of the palmar aspect of the hand is total. The function of the thumb is limited to a narrow lateral pinch with the posterolateral aspect of the index finger, innervated by the radial nerve.

These lesions are usually caused by large wounds on the anterior aspect of the wrist or distal forearm in industrialized countries. The main problems are the lack of sensibility and the extent of the scar tissue, which will influence the choice of the motor muscle and the path of the transfer. Before considering reconstructive surgery, the surgeon must prevent contractures and fixed deformities and try to restore some sensibility. This may be possible years after the injury with a late nerve graft repair, even if this does not apply to the motor power. Other procedures are possible for the restoration of some sensibility on the thumb pulp (see Chapters 25–26). In particular, the sensate area on the dorsal aspect of the proximal phalanx of the index finger can be transferred as an island flap with the first dorsal intermetacarpal artery, to the thumb (Foucher and Braun's kite flap[65]) (Figure 17.20).

Restoration of thumb opposition is only possible if a complete passive movement of opposition is re-established, and seems to be only useful if there is some pulpar sensibility. If not, it is preferable to restore a strong thumb–index lateral pinch.

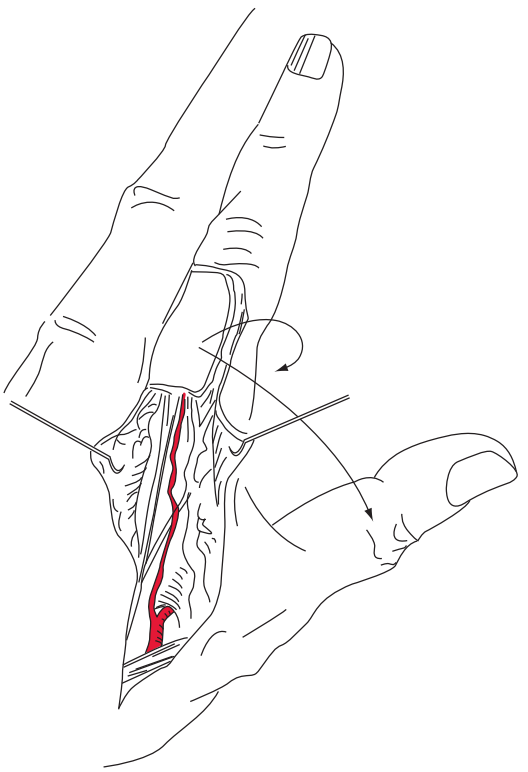


Figure 17.20
The kite flap.

If restoration of thumb opposition is decided, one will have to re-establish (1) anteposition and pronation of the thumb column, (2) some power of the thumb grip in flexion–adduction, (3) extension of the thumb distal phalanx for a greater pulpar contact, and (4) stabilization of the MP joint, whose stabilizing muscles (AP and FPB) are paralyzed.

The *motor muscle* should be powerful and have a good excursion. The FDS of the ring finger, when available, is the first choice, but if for any reason this transfer is not chosen, the EIP, although less powerful, can be used. The pulley is placed in an intermediate position, slightly proximal and superficial relative to the pisiform, to produce a greater anteposition effect.[29]

The *path of the transfer* should be superficial, oblique parallel with the direction of the FPB, in the direction of the radial aspect of the thumb MP joint.

The *fixation* of the transfer should be used, when possible, to stabilize the MP joint and to extend the distal phalanx.

Techniques preferred by the author for distal median–ulnar palsy

Transfer of the FDS of the ring finger

Three incisions are necessary:

1. An oblique or zig-zag incision over the proximal phalanx of the ring finger in order to remove the flexor superficialis.
   The tendon transferred has to be long enough to be able to stabilize, with its two bands of insertion, the often lax MP joint of the thumb. We use the technique described by Zancolli[10] for its lasso procedure: the tendon is harvested through a hole made in one side of the flexor tendon sheath, 0.5 cm proximal to the PIP joint flexor crease, in order to be proximal to the Camper's chiasma. One band of insertion of the FDS is cut. Tension is applied on the tendon, delivering the second band into the hole in the tendon sheath, thus obviating the need to section the tendon sheath all the way across. The second band of the FDS is cut in turn.
2. An angled incision on the anteromedial aspect of the wrist, elevating a medially based flap opposite the pisiform in order to extract the distal end of the tendon to be transferred and to make a pulley. The line of the incision must not correspond to the course of the transfer.
3. An S-shaped longitudinal incision centered over the dorsal aspect of the MP joint of the thumb for fixation of the transfer.

The *desired path* is that following the superficial head of the FPB. For the tendon to lie in this position, the pulley should be near the pisiform or in a more proximal location (see 3a and 3b in Figure 17.15).

A simple procedure consists of passing the transfer around the tendon of the FCU. The transfer is pulled subcutaneously under the skin of the thenar eminence in the direction of the radial aspect of the MP joint of the thumb. The two insertion slips of the FDS, after being woven through the tendon of the APB, are fixed on the ulnar side of the joint and act as a tenoplasty. Both of these slips are passed behind the MP joint, one proximal and the other distal to the joint space. Each slip transfixes the tendon of the EPL while the distal joint is maintained in extension. Then the bands are passed through the capsuloligamentous tissue from posterior to anterior in contact with bone. The two tendon ends are then fastened to one another under tension with the proximal phalanx held in slight flexion (10°) (Figure 17.21).

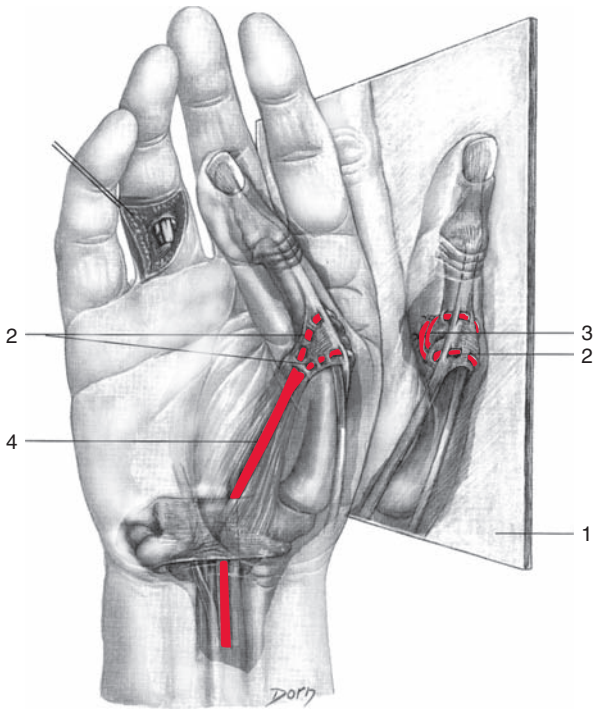


Figure 17.21

Paralysis of all intrinsic muscles of the thumb; FDS transfer to the thumb. The best site for the pulley is at the proximal pole of the pisiform. The simplest procedure consists of passing the transfer around the tendon of FCU. However, if this muscle is paralyzed, its tendon stretches and the direction of the transfer will not be maintained. In this event, it is advisable to perform a tenodesis of the paralyzed FCU tendon to the ulnar proximal to the pulley. Fixation of the FDS transfer: (1) Mirror. (2) The two terminal slips of the FS are passed on either side of the dorsal aspect of the MP joint. Each slip transfixes the EPL tendon, the distal phalanx maintained in complete extension. (3) Each slip passes through the capsuloligamentous tissue from posterior to anterior in contact with the bone. The two tendon ends are then fastened to one another under tension on the ulnar volar aspect of the joint to prevent MP joint hyperextension with the proximal phalanx held in slight flexion (10°) and the distal phalanx in complete extension. (4) FDS transfer.

Evaluation of tension: before fixing the slips, it is essential to place them under suitable tension. The proximal phalanx must be maintained in slight flexion and the distal phalanx in extension. The thumb, in anteposition, must be opposite the ring finger while the wrist is in neutral position. When the wrist is completely flexed, the thumb should go into retroposition. When the wrist is in complete extension, the thumb is brought into opposition.

Using two transfers

When, for any reason, an FDS is not available, I prefer to use two transfers, rather than the EIP or EPL alone. Other transfers are also available in distal median–ulnar palsy, and even if they are weak, their action is reinforced by the tenodesis effect of the wrist movements.

A double transfer allows the anteposition and flexion–adduction to be adjusted more effectively, provides better stabilization of the thumb column, and more effectively prevents flexion of the distal phalanx. Depending on the circumstances, either the EIP, EPB, or PL is used, each with a different path adapted to the special case of each patient. It seems preferable to keep the EPL in place, because it will reinforce adduction.

It is essential to avoid contradictory action of the two transfers – their actions should be complementary. To achieve this, the 'adductor' tendon is given not a transverse course but an oblique one similar to that of the FPB muscle. Both transfers should also be inserted on the radial side of the thumb column so as to counterbalance supination of the thumb. The imbalance in supination is less when there is palsy of all the intrinsic muscles of the thumb than when there is isolated palsy of the lateral thenars, for the adductor is also paralyzed. The anteposition transfer should follow the path of the APB and insert on the EPL. Essentially, it will serve to increase separation of the thumb from the palm (Figure 17.22).

The two transfers coordinate their action to stabilize the MP joint by preventing its hyperextension, and will prevent flexion of the distal phalanx.

Several forms of transfer are available, and it is necessary to choose the best suited in each case, taking into consideration such factors as the respective power of the two transfers, the articular stability, and the existence of scars.

Restoration of opposition in proximal palsies

These conditions concern the lesions proximal to the proximal third of the forearm. The overall upper limb restoration problem has to be evaluated according to the remaining possibilities.

The general principles of tendon transfers remain applicable; however, the restricted number of motors available and joint instabilities make the restoration more complicated.

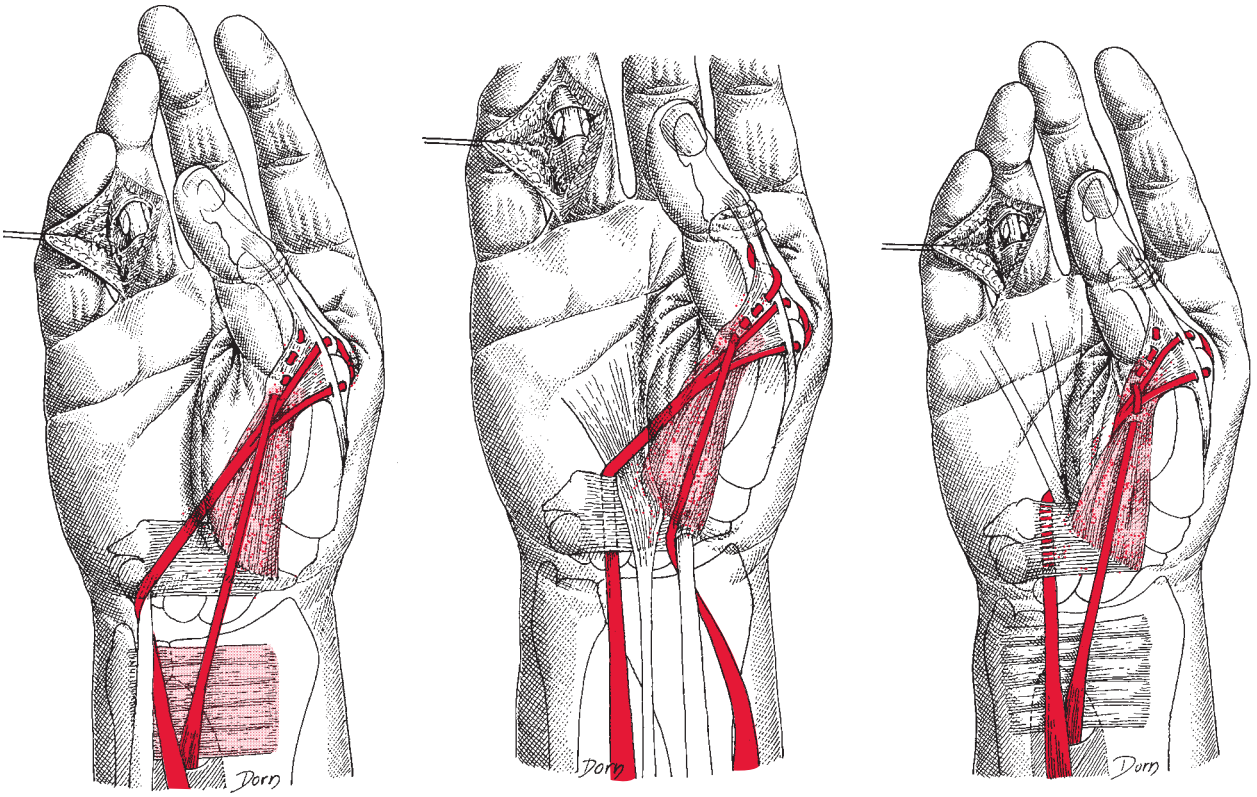


Figure 17.22

Using two transfers for median and ulnar palsy. Possible combinations in the path of the two transfers: one for anteposition and one for flexion–adduction; both transfers should be pronators of the thumb. There is a progressive increase in the force of the pinch when the pulley for the flexor–adductor transfer is more distal and deeper.

Isolated proximal palsy of the median nerve

Pronation of the forearm, wrist, thumb, index, and middle finger is paralyzed. Nerve repair usually allows restoration of extrinsic muscles; the problem is restoration of the lateral thenar muscle. If the intrinsic palsy does not recover, an opponenplasty is required. Transfer of the EIP through the IOM is our best choice (see Figures 17.16 and 17.19).

Several transfers of the intrinsic muscles innervated by the ulnar nerve can in certain conditions replace the paralyzed thenar muscles when the tendons of the forearm muscles are not usable (see below).

Proximal median–ulnar palsy

Disability is maximum, and the few remaining muscles innervated by the radial nerve must preserve wrist extension (which has a crucial role of tenodesis effect on the transfers) and restore thumb and finger flexion. EIP transfer is a good choice for opponenplasty, but it is often already used for another transfer. An MP joint fusion is indicated in case of instability, which is common in extensive palsies.

EPL transfer

The EPL has a good excursion and a long tendon, and its transfer, according to the Bureau and Decaillet[66] procedure is our preferred technique in proximal median–ulnar palsy (Figure 17.23).

A curvilinear incision that passes radial to the MP joint of the thumb is used to approach the EPL. The tendon is divided proximal to the MP joint.

A second incision on the dorsal aspect of the wrist and lower third of the forearm enables one to extract the tendon and pass it through the interosseous membrane, just proximal to the pronator quadratus. This central path makes it possible to benefit from the active tenodesis effect of the wrist, and gives supplementary length to the tendon. Through an anterior incision, the tendon is recovered in the forearm and passed around the tendon of the FCU, which plays the role of a pulley. Next, it follows a subcutaneous path toward the MP joint of the thumb and is woven through the tendon of the APB. Finally, it is solidly attached by a transfixation suture through its distal end, proximal to the MP joint, so as to contribute to the distal joint

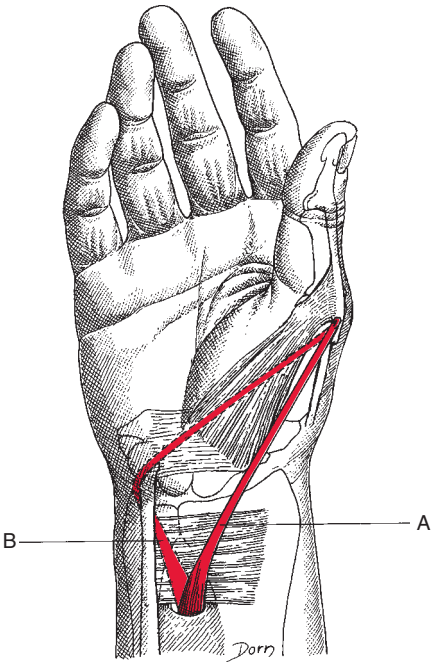


Figure 17.23

Transfer of the EPL in proximal median–ulnar palsy. The tendon is divided proximal to the MP joint and passed volarly through the interosseous membrane. It can have a direct subcutaneous path toward the MP joint of the thumb and acts as a transfer for anteposition (A) or is passed around the tendon of the FCU, which plays the role of a pulley and acts as a transfer for opposition (B). In both cases, it is attached to its distal end and remains an extensor of the thumb joints.

extension, while the MP and IP joints are maintained in complete extension. The reconstructed EPL tendon risks subluxating laterally at the MP joint if the suture is distal to this joint. A K-wire is placed obliquely through the distal joint to maintain it in extension. A splint maintains the wrist in neutral position and the thumb in opposition for 1 month.

Proximal median–radial palsy

The lack of the tenodesis effect produced by wrist movements worsens the problem. Groves and Goldner[67] have described a transfer of the FCU on the FDS of the ring finger. Then the FDS, motorized by the FCU may be transferred to restore opposition.

Intrinsic muscle transfers

Transfers of intrinsic muscles innervated by the ulnar nerve can also be used.

The abductor digiti minimi (ADM). Transfer of the ADM was independently proposed by Huber[26] and by Nicolaysen.[27] It was popularized by Littler and Cooley.[28]

Flexor digiti minimi (FDM). Pallazi[68] proposed the use of the FDM, using the same principles as with the ADM. When severing the FDM, great care must be taken in freeing the FDM from the ADM, which lies over it, and in freeing it up to the hamatum carefully, in order not to damage its neurovascular bundle. The transfer is routed in the same way transversally subcutaneously through the palm and fixed to the APB.

Other intrinsic muscle transfers. Orticochea[69] transferred the deep portion of the FPB, innervated by the ulnar nerve, to the dorsal expansion of the abductor in order to increase pronation of the thumb.

For poliomyelitis patients deprived of available extrinsic muscles, De Vecchi[70] proposed the transfer of the distal insertion of the adductor pollicis from the medial sesamoid to the lateral sesamoid.

Technique preferred by the author: transfer of the ADM (Figure 17.24a)

An incision is made extending from the wrist flexion crease to the posterior aspect of the proximal phalanx of the little finger by passing along the lateral aspect of the hypothenar eminence. A thenar incision is made over the posterolateral aspect of the MP joint of the thumb to expose the expansion of the APB and to expose the tendon of the EPB. The ADM is detached in the following manner:

1. The distal insertion on the proximal phalanx must be dissected very carefully in order to conserve the maximal length of the tendinous segment; it can be extended by the retrovascular fibrous band of the little finger.
2. The proximal insertion to the pisiform is also detached, conserving only its insertion to the flexor retinaculum and the FCU. The neurovascular pedicle, which enters the deep proximal portion of the muscle, is carefully isolated in order to avoid excessive tension. The freed muscle is rotated 180° and follows the path of the APB. The distal fixation uses the two heads of the transferred muscle, on the APB tendon and on its dorsal extension (Figure 17.24b). The transfer of this relatively powerful muscle limits the abduction of the little finger and weakens its strength. The cylindrical grip strength of the ulnar border of the hand may be jeopardized. Nevertheless, this muscle transfer can be very useful when the tendons of the forearm muscles are not usable, or the muscles are destroyed in the case of Volkmann's contracture. This transfer can also be used for congenital hand disabilities; it has interesting implications for reinforcing opposition in a pollicized thumb.[71] Finally, restoration of volume to the thenar eminence improves the appearance of the emaciated paralyzed hand; these esthetic considerations may be the determining factor in choosing the procedure for re-establishing opposition of the thumb in some patients.[72]

The wrist does not need to be immobilized after an intrinsic muscle transfer, since it plays no role in the movement of this transfer.

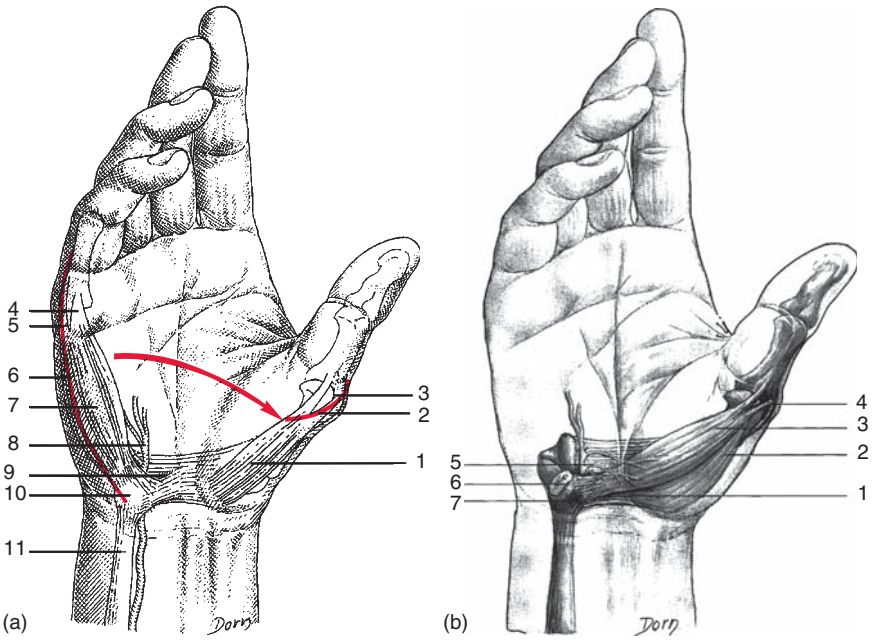


Figure 17.24

Transfer of ADM. (a) Incisions: (1) APB; (2) EPB; (3) thumb incision; (4) tendon of the ADM inserted on the base of the proximal phalanx; (5) retrovascular fibrous band; (6) hypothenar incision; (7) ADM; (8) ulnar pedicle; (9) Guyon's canal; (10) pisiform; (11) FCU. (b) Fixation of the muscle transfer: (1) flexor retinaculum; (2) APB; (3) ADM transferred; (4) EPB tendon; (5) neurovascular pedicle for the ADM; (6) Pisiform; (7) insertion of the ADM to the FCU tendon.

Associated median-ulnar and radial nerve palsy

This is almost the same condition as a distal brachial plexus palsy; the surgical possibilities are the same, and will use the few remaining motors. Reconstruction of a lateral grip is more appropriate than restoration of thumb opposition.

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18 Restoration of the intrinsic muscle function of the long fingers

Raoul Tubiana

The intrinsic muscles of the long fingers consist of the interosseous and lumbrical muscles (Figure 18.1). Their complex action combines with the extrinsic flexor and extensor muscles of the fingers to allow the independence of movement of each phalanx.

Each phalanx has a wide range of movements in flexion and extension. Flexion is ensured by the exceptionally large excursion of the long flexors; however, that of the long extensors is less important, and their action has to be relayed by the intrinsic muscles.

History

As far back as the second century AD, Galen[1] discovered the anatomy of the interosseous muscles, but the famous anatomist from Pergamum thought that their only action was flexion of the proximal phalanges of the digits. It was only in 1543 that two pupils of Vesalius from Padova, Columbus and Fallope, gave a better anatomic description of these muscles. Columbus described the distal insertion of the lumbricals on the extensor digitorum communis, and Fallope established the action of the interosseous muscles as extensors of the distal phalanges. Winslow[2] in 1752 was more precise, for he found that the interosseous and lumbrical muscles act as flexors of the proximal phalanges and extensors of the two distal ones. However, all these investigators considered these muscles to be weak auxiliaries of the long flexors and extensors of the digits.

Duchenne[3] in 1867 studied the mechanism of the movements of the digits and demonstrated that the intrinsic muscles were indispensable to ensure the freedom of movement of each phalanx. It is interesting to read the description provided by Duchenne, as translated by Kaplan:[4]

> 'It would be impossible to imagine a more ingenious mechanism favorable to simultaneous flexion of the proximal phalanx and extension of the two distal phalanges than presented in the anatomic arrangement of the terminal tendons of the (interosseous muscles) and lumbricals.
>
> 'In the first part of their course from the head and anterolateral aspect of the metacarpal to the dorsal aspect of the head of the proximal phalanx, the tendons of the (interosseous muscles) and lumbricals have an oblique dorsal and distal direction so that during contraction of these muscles, motion occurs in the metacarpophalangeal joint, while the fixed point is at the distal end of the proximal phalanx. This arrangement produces flexion of the proximal phalanx in proportion to the force acting on the distal end of the lever represented by this phalanx.
>
> 'In the second part of their course from the distal end of the proximal phalanx to the base of the distal phalanx, the same tendons pass over the dorsal aspect of the two distal phalanges, paralleled to their longitudinal axis. As a result of this, the contraction of the (interosseous muscles) produces only extension of the distal and then the middle phalanges. But the contraction of the (interosseous muscles) and lumbricals acts equally throughout the whole course of these tendons and thus produces simultaneously the two opposite movements of flexion of the proximal phalanx and extension of the two distal phalanges.
>
> 'To understand well the marvelous ingenuity of the means used by nature to produce the movements and their

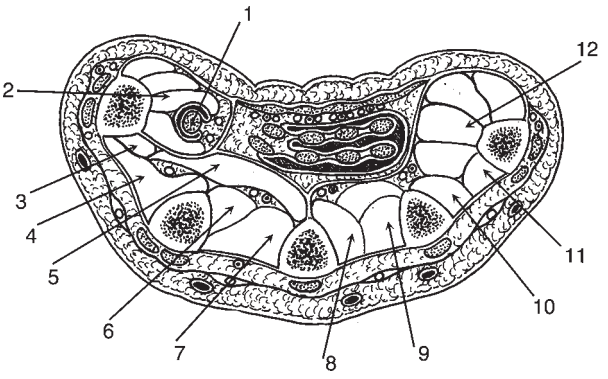


Figure 18.1

The intrinsic muscles: (1) flexor pollicis longus (FPL); (2) lateral thenar muscles; (3) first palmar interosseous (1st PIo); (4) first dorsal interosseous (1st DIo); (5) adductor pollicis (AP); (6) 2nd PIo; (7) 2nd DIo; (8) 3rd DIo; (9) 3rd PIo; (10) 4th DIo; (11) 4th PIo; (12) hypothenar muscles.

mechanisms which I explained, it is necessary to obtain the same results by other mechanical combinations. This is exactly what I attempted.'

Anatomy

The interosseous muscles

The structure of the interosseous muscles is particularly complex. Each interosseous muscle is composed of a number of muscular bundles of different lengths, taking their origins on the lateral or palmar surfaces of the metacarpal shafts. The muscular bundles have a relatively independent nerve supply, arising from the deep branch of the ulnar nerve. They continue into tendinous slips, entering the finger posterior to the interglenoid (deep transverse metacarpal) ligament. The tendinous slips insert distally at different levels in the fingers. These anatomic peculiarities allow for several classifications. Albinus[5] in 1724 classified the interosseous muscles according to their origins into three palmar adductors and four dorsal abductors of the fingers; this classification was accepted for almost 200 years. They can also be classified according to their insertions into deep and superficial, proximal or distal.[6,7]

The deep insertions are into the lateral tubercles on the base of the proximal phalanx and into the capsule of the metacarpophalangeal (MP) joint. They allow lateral movements of the fingers and prevent posterior dislocation of the muscle.

The superficial insertions occur at three separate levels in the finger (Figure 18.2):[8]

1. One group of muscle bundles runs into the transverse fibers surrounding the posterior aspect of the MP joint and into the base of the proximal phalanx, and forms the interosseous hood. The angle of approach of the palmar interosseous at the MP level is greater than the angle of the dorsal interosseous. The interosseous muscles flex the MP joint by means of the interosseous hood.
2. A second group runs more distally over the dorsal aspect of the proximal phalanx. The oblique fibers blend with the central band of the extensor tendon, forming the central extensor tendon, and insert into the base of the middle phalanx, which they extend.
3. A third group of distal fibers blends with the fibers coming laterally from the lumbrical and runs into the lateral band of the extensor tendon, forming the lateral extensor tendon.
4. The two lateral extensor tendons join together on the dorsum of the distal interphalangeal joint (DIP), forming the distal extensor tendon, which inserts into the base of the distal phalanx.

The lumbrical muscles

The lumbrical muscles are four small fasciculi that arise from the tendons of the flexor profundus in the palm.

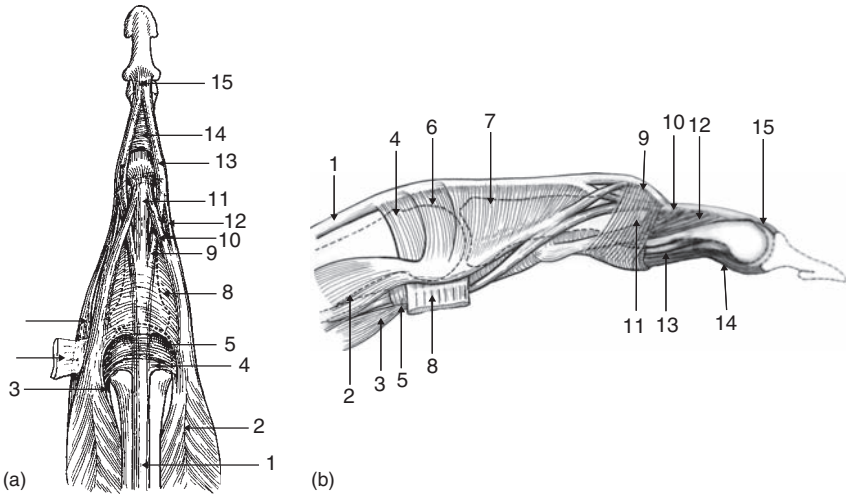


Figure 18.2

The intrinsic muscles of the long fingers and the extensor apparatus. (a) Dorsal view: (1) extensor digitorum comminis (EDC) tendon; (2) dorsal interosseous; (3) insertion of the interosseous on the metacarpophalangeal (MP) capsule and on the base of the proximal phalanx; (4) sagittal band; (5) transverse fibers of the extensor hood; (6) transverse intermetacarpal ligament; (7) lumbrical muscle; (8) oblique fibers of the extensor hood; (9) lateral band of the extensor tendon; (10) interosseous central band; (11) central conjoint extensor tendon; (12) interosseous central band; (13) lateral extensor tendon; (14) triangular lamina; (15) terminal extensor tendon. (b) Lateral view of the radial side of a long finger: (1) EDC tendon; (2) dorsal interosseous muscle; (3) lumbrical; (4) sagittal band; (5) flexor pulley A1; (6) transverse fibers of the extensor hood; (7) oblique fibers of the hood; (8) transverse intermetacarpal ligament; (9) central extensor tendon; (10) lateral extensor tendon; (11) transverse retinacular ligament; (12) oblique retinacular ligament; (13) flexor digitorum superficialis (FDS); (14) flexor digitorum profundus (FDP); (15) terminal extensor tendon.

Each passes to the radial side of the corresponding finger, superficial to the transverse intermetacarpal ligament, and terminates on the lateral extensor tendons (Figure 18.3). The angle of approach to the dorsal aponeurosis is greater than that of the interossei.

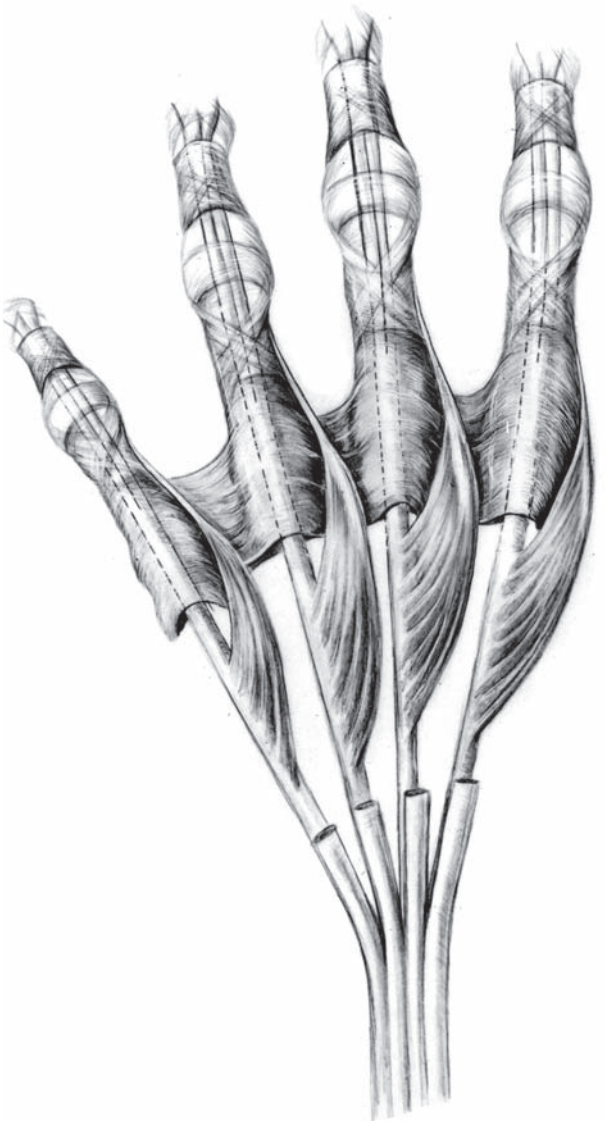


Figure 18.3
The lumbrical muscles, palmar view.

This is the general arrangement. There are different patterns in the insertions of the interossei and lumbrical muscles for each finger, and even for each side of each finger, allowing greater functional individuality. The index and little fingers have quite specialized intrinsic muscles because of their peripheral location. The index finger has the first dorsal interosseus, whose digital insertion is entirely osseous. The radial aponeurotic wing is formed almost entirely by the first lumbrical. On the little finger, the tendinous wing on the ulnar side is formed by the abductor digiti minimi.

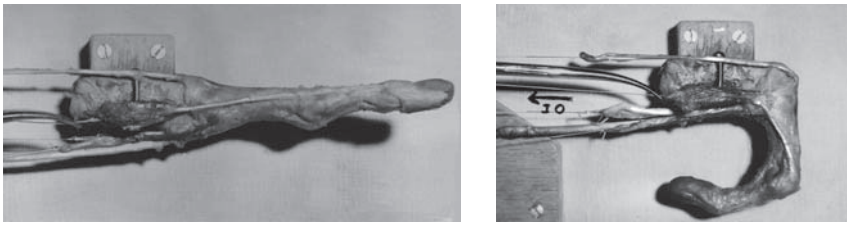
Action of the intrinsic muscles

The *interosseous muscles* produce lateral movements of fingers through their insertions on the lateral aspect of the base of the proximal phalanges. If they are paralyzed, these movements are considerably diminished. Slight lateral movement persists, owing to the proximal convergence of the long extensor tendons.

The interosseous muscles also play an essential role in flexion–extension movements of the phalanges. Schematically, they flex the proximal phalanx and extend the two distal phalanges. These movements depend on the MP joint position. When this joint is in extension (Figure 18.4a), the interosseous hood covers the MP articular space. The oblique fibers of the interossei are put into tension and extend the distal phalanges. In MP joint flexion, the hood is pulled distal to the articular space and the force of the interossei is applied to the back of the proximal phalanx, thus reinforcing its flexion, but they lose their extensor action on the distal phalanges (Figure 18.4b). Although the actions of the interossei are dependent on the position of the MP joint, they are less dependent on wrist movement.

The *lumbrical muscles* are longer than the interossei, giving them a greater contractile potential, though their volume is much less than that of the palmar interossei. Unlike the interosseous muscles, the lumbrical muscles are able to extend the two distal phalanges whether the MP joint is in extension or flexion.

The integrity of the lumbricals at the level of the index and long fingers, in cases of isolated paralysis of the ulnar



(a) (b)

Figure 18.4
(a) When the MP joint is extended, all the force of the interossei is applied in extension of the two distal phalanges. (b) When the MP joint is flexed, the force of the interossei muscles is applied on the sling on the dorsum of the proximal phalanx. However, they can no longer extend the distal phalanges.

nerve, is sufficient to prevent development of the claw deformity. However, this deformity is latently present even though it is not exhibited. During a strong grip between the thumb, index, and long fingers, the MP joint has a tendency to hyperextend while the interphalangeal (IP) joints are hyperflexed (hyperflexion sign of Mannerfelt). This latent deformation is evident when the patient is asked to flex the index and long finger MP joints while maintaining the IP joints in extension ('lumbrical plus' position). Slight pressure on the volar aspect of the proximal phalanx will cause immediate flexion of the proximal interphalangeal (PIP) joint. These patients are hindered in their ability to grasp small objects with precision and strength, although movements of the index and long fingers made without effort appear normal.

However, the lumbricals appear primarily to be IP extensors. The action of these small slender muscles between the flexor profundus tendons and the extensor apparatus is subtle: they participate in extension of the distal phalanges by pulling distally on the flexor profundus tendon when this muscle is at rest. As has been shown by Long,[9] this permits a reduction of the resistance of the flexor profundus and indirectly facilitates the action of the common extensor on the distal phalanges. Contraction of the lumbrical muscles, whose relatively free play is little impaired by attachments to the dorsal hood, also contributes directly to the extension of the distal phalanges, regardless of the position of the MP joint, whether the proximal phalanx is extended or flexed (Figure 18.5).

By contrast, the interosseous muscles have a decreasing extensor effect at the IP joints as flexion of the MP joint increases (see Figure 18.6). This explains why, despite their feebleness, lumbrical muscles can form an 'active diagonal system' between the flexors and extensors at the proximal part of the finger.

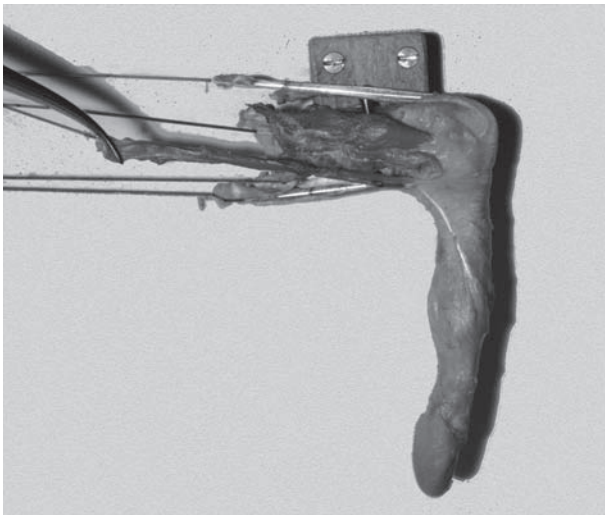


Figure 18.5

With the MP joint in flexion, the lumbrical is able to extend the distal joints.

Thus, these muscles, which have the same innervation as the corresponding flexor profundus, play a coordinating role between the extensor and flexor systems. Rabischong[10] demonstrated the richness of the sensory receptors at their level; thus, these small, weak muscles are true proprioceptive organs.

Innervation

All interosseous muscles are innervated by the ulnar nerve.

The lumbrical muscles have the same innervation as the corresponding flexor profundus; the first and second lumbricals are innervated by the median nerve; the third and fourth are innervated by the ulnar nerve.

Etiology

Intrinsic muscle deficits in Western countries most often result from direct trauma to the ulnar nerve or from long-standing nerve compression, at the level of the nerve trunk or more proximally[11] – in particular, impingement of the lower cervical nerve roots or lesions of the lower elements of the brachial plexus. However, ulnar nerve paralyses secondary to leprosy and poliomyelitis are still frequent in developing countries in the tropics and subtropics.

Clinical features

Dysfunction of intrinsic muscles of the fingers results in various clinical aspects. The most typical is the *claw-hand deformity*.

It is important to be aware that the classic claw deformity is only one aspect of intrinsic muscle paralyses, and that every ulnar paralysis does not result in a claw-hand. Also, there are many other dynamic problems disturbing prehension that are caused by paralysis of intrinsic muscles of the fingers.[12]

The claw deformity

The claw consists of hyperextension of the proximal phalanx while the two distal phalanges are in flexion. This deformity is caused by the paralysis of the intrinsic muscles, when the extrinsic muscles (i.e., the long flexors and the long extensors of the fingers) are active.

Mechanical analyses (Figure 18.6) show that the forces of the extensor digitorum communis (EDC) and of the flexor digitorum superficialis (FDS) on the middle phalanx have a component that produces extension of the proximal phalanx. It is this force that the intrinsic muscles (lumbrical and interossei) normally oppose. When these are paralyzed, no force exists to prevent the proximal phalanx from swinging into hyperextension. Inversely, this hyperextension is

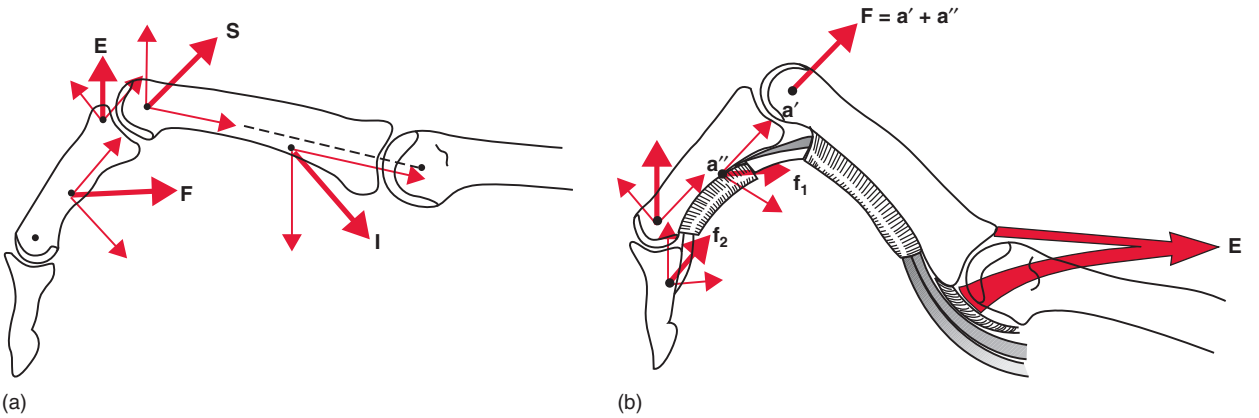


Figure 18.6

(a) Mechanical explanation of the claw-hand deformity. Force **E** of the EDC contributes to carrying the proximal phalanx into hyperextension. The force of the flexors also produces extension in the proximal phalanx Force **F** of the FDS has an axial component **a′**. Force $\mathbf{f_2}$ of the flexor profundus has an axial component that, as it is transmitted to the head of the middle phalanx, can be broken down into an axial force **a′** and an extension force on the middle phalanx counteracted by the flexion component of $\mathbf{f_1}$. The sum of **a′** and **a″** represents the total force of extension acting on the middle phalanx **F:** Force **E** of the EDC and force **F** of the FDS each has an axial component. The sum **S** of these is applied to the head of the proximal phalanx over which it extended. Force **I** of the intrinsic muscles counteracts this extension force at the proximal phalanx. (b) When the intrinsic muscles are paralyzed, no force exists to prevent the proximal phalanx from swinging into hyperextension. The EDC, which exhausts its action at the level of its proximal insertion **E**, has no effect on the distal phalanges. Note that hyperextension of the MP joint is only seen if the EDC and FDS are active.

seen only if the EDC and FDS are active. The long extensor, which exhausts its action at the level of its proximal insertions, has no effect on the distal phalanges. MP joint hyperextension is prevented initially by the volar plate of the joints, but capsular resistance progressively yields, and the deformity will worsen.

This deformity is present in varying degrees, depending on the extent of the paralysis and the suppleness of the digits. It involves only the ring and small fingers in cases of isolated ulnar paralysis, because the two radial lumbricals, innervated by the median nerve, prevent deformity of the index and long fingers when the hand is at rest (Figure 18.7). The third lumbrical may be innervated by both the median and ulnar nerves, and clawing is more severe in the little finger than in the ring finger.[13] Deformity is more marked when the nerve lesion is distal and the long flexors are intact.

It involves the four fingers (Figure 18.8) in cases of associated paralysis of the ulnar and median nerves.

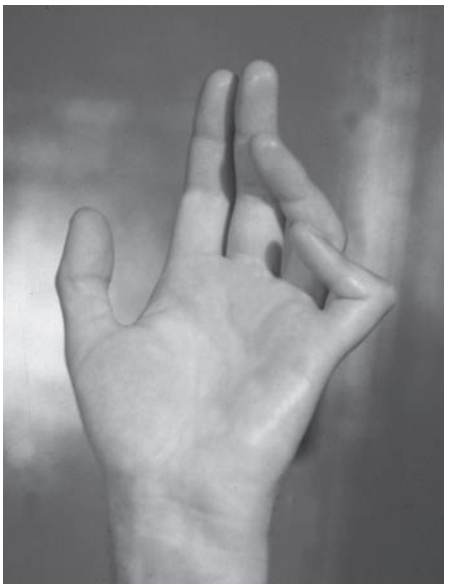


Figure 18.7

Isolated distal ulnar paralysis.

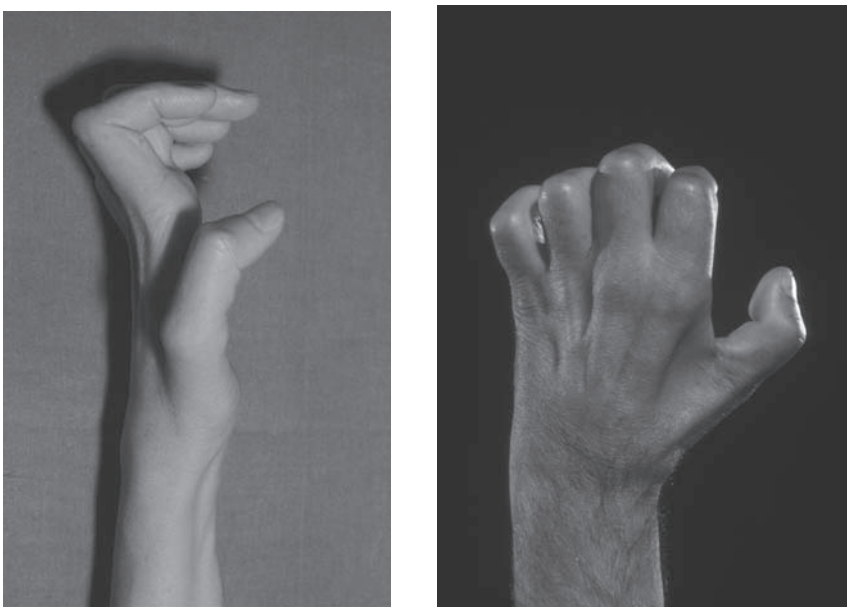
Bouvier's test

Bouvier,[14] while examining a hand with an ulnar paralysis, noted 'The patient could flex all the fingers equally, but he was unable to bring then into complete extension. However, and remarkably, the patient was able to achieve complete extension when he was pressing with his other hand on the proximal phalanx, thereby suppressing the action of the extensors at that level' (Figure 18.9).

The claw is called reducible when prevention of hyperextension of the MP joints allows extension of the distal phalanges.

Lack of strength of prehension

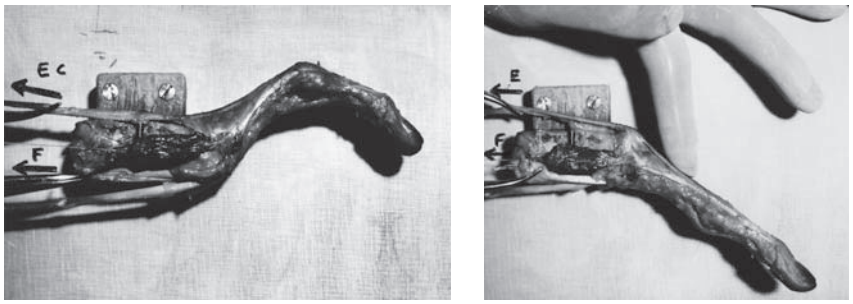
For a long time, intrinsic muscle strength has been underestimated. This strength is far from negligible, especially in the peripheral digits. Lateral pinch between thumb and index is possible because the extensor pollicis longus (EPL) tendon acquires an adduction action. The index finger has



(a) (b)

Figure 18.8

Associated paralysis of the ulnar and median nerves: (a) lateral view; (b) dorsal view.



(a) (b)

Figure 18.9

Bouvier's test. (a) Claw deformity. (b) Correction of the deformity: prevention of hyperextension of the MP joint allows extension of the distal phalanges by tension of the EDC.

no active abduction, and the strength is very diminished because of the first dorsal interosseous paralysis to which is added paralysis of the adductor pollicis and often of the flexor pollicis brevis (FPB) (see Chapter 19 on restoration of thumb–index lateral pinch).

The interdigital grip is also lost, because the interossei normally control movements of the fingers. In particular, paralysis of the third dorsal interosseous will prevent active adduction of the little finger toward the ring finger and the little finger is maintained in abduction because of the unopposed action of the extensor digiti minimi (Wartenberg's sign[15]).

The more proximal the ulnar paralysis, the more marked is the deficit of the strength of finger flexion, because the flexor profundus paralysis of the ring and little finger is added to the intrinsic paralysis. When there is concomitant proximal median nerve paralysis, all the digital flexors – intrinsics and extrinsics – are paralyzed.

A considerable lack of thumb strength is always added to the lack of finger strength, because the adductor pollicis (AP) and often the FPB are paralyzed. Measurements performed by Mannerfelt[16] showed that residual strength of thumb adduction in ulnar palsy is only 20% of normal.

Disturbance in finger flexion

Normally, finger flexion begins at the level of the PIP and MP joints and is followed by DIP joint flexion. In cases of interosseous paralysis, the sequence of flexion is altered (Figure 18.10a). Finger flexion is accomplished by the long flexors alone, which act first on the distal phalanges. The MP joint flexes last. With flexion, the pulps of the fingers shave the palm, and the hand cannot grasp large objects. This 'dyskinetic finger flexion' is one of the most awkward functional consequences of these paralyses.

This inversion of the sequence of phalangeal flexion causes a modification of the location of pressure zones during grasp. Normally during grasp, the pressure over the zone of contact is widely distributed on the palmar aspect of the fingers, on the metacarpal heads, and on the thenar and hypothenar eminences. When an ulnar-paralyzed hand grasps an object, it is the extremities of the flexed fingers that do the grasping, and the pressure on these limited zones of contact is considerable (Figure 18.10b). This can cause ulceration of the fingertips, especially when problems of pressure distribution are associated with a sensory disturbance.

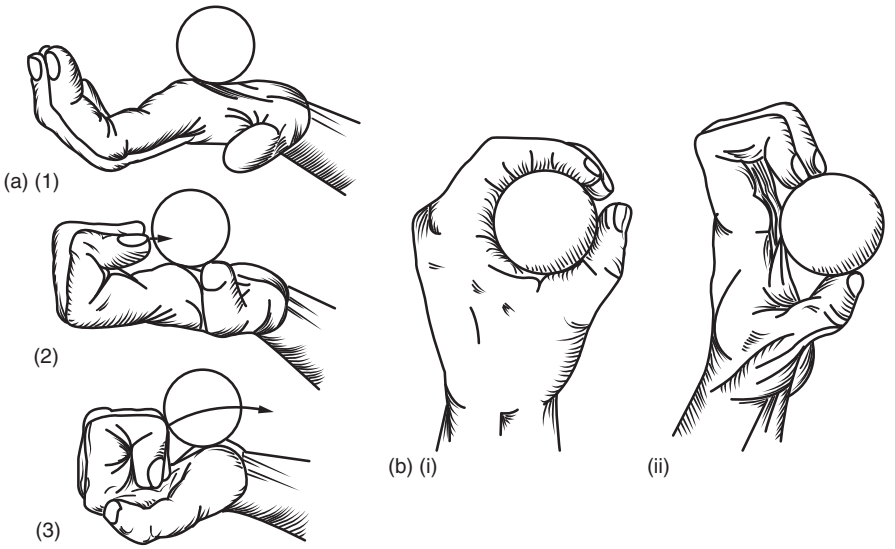


Figure 18.10

Disturbance in finger flexion. (a) With intrinsic paralysis, there is an inversion of the sequence of flexion. Flexion begins with the distal phalanges. (b) Excess of pressure on the fingertips. Comparison of pressure zones in normal grasp (i) and grasp in a hand with intrinsic paralysis (ii).

Treatment: physiotherapy

Early physiotherapy attempts to maintain mobility of the finger joints and to prevent deformity.

A *lumbrical bar splint* fitted over the dorsum of the metacarpal heads and proximal phalanges of the ring and little fingers is particularly useful. This splint prevents the MP joints from hyperextension without impending finger flexion. It will not improve grip strength, but will allow the extrinsic extensor tendons to extend the interphalangeal joints and diminish the likelihood of fixed contractures.

Surgical restoration of the function of intrinsic muscles of the finger

Timing of reconstructive surgery

Early reconstructive surgery is indicated when nerve lesion is proximal and recovery of distal motor function will be very delayed or problematical. Early surgery attempts to prevent deformity by stabilization of the MC joints in slight flexion (see Chapter 23 on timing of reconstructive surgery).

Late palliative treatment is indicated when nerve repair is unable to recover motor function.

Palliative treatment has two aims:[17]

- correct the claw deformity and improve finger motion
- increase grip strength

Correction of the claw-hand deformity can be achieved by any procedure preventing hyperextension of the MP joints using tenodesis, capsulodesis, or finger tendon transfers. However, only the addition of a tendon transfer from the wrist motor muscles or from a more proximal muscle can increase grip strength.

We will describe in turn tendon transfers, tenodesis, and capsulodeses.

Tendon transfers

Several techniques of tendon transfers have been used for correction of the claw deformity and reinforcement of grip strength.

Factors to be considered for the transfer include the choice of the motor, the course of the transfer, and the site of insertion.

Choice of motor muscle

The strength of the motor should be adapted to the deficit.

The more commonly performed procedures include transfer of a wrist motor or transfer of an extrinsic muscle of the fingers (flexor or extensor).

Only wrist motors will increase grip strength, but they must be lengthened by tendon grafts.

Path of the transfer

According to Bunnell,[18] the transfer (or graft) should run along the lumbrical canal and pass volar to the deep transverse intermetacarpal ligament. One should decide whether the transfer should pass volar or dorsal to the wrist. While a dorsal transfer becomes lax with extension of the wrist, a volar transfer may be expected to restore a more physiologic grip with the wrist extended (Figure 18.11). When the deformity is longstanding, the patient will have developed the habit of flexing the wrist to extend the fingers: this habit is very hard to discard. In these cases, a dorsal approach is preferable (Figure 18.12).

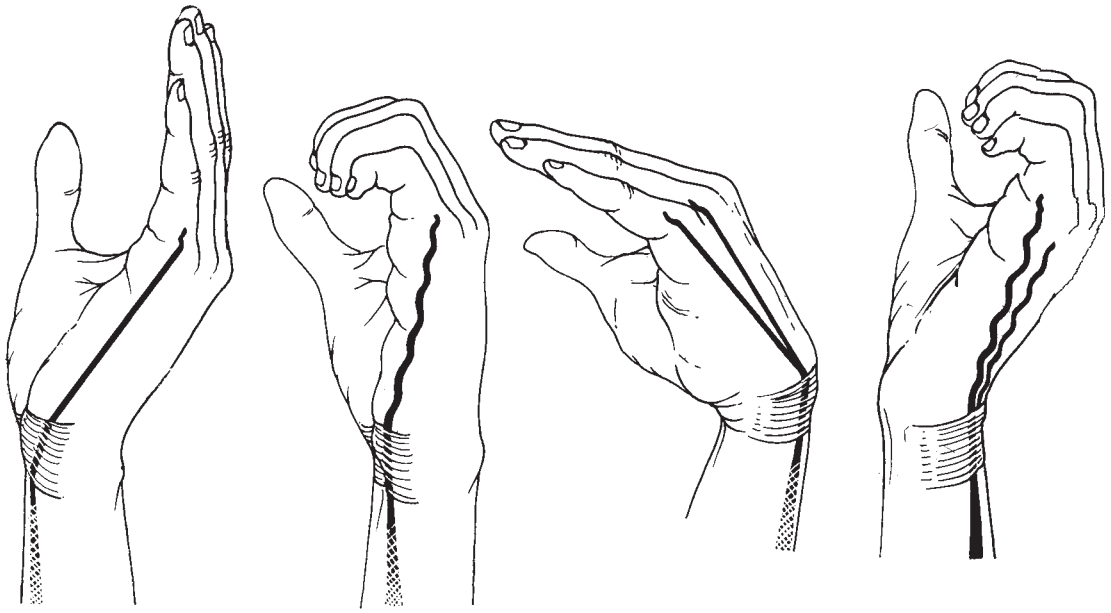


Figure 18.11

A transfer dorsal to the wrist is tightened with wrist flexion, while a transfer volar to the wrist is tightened with wrist extension.

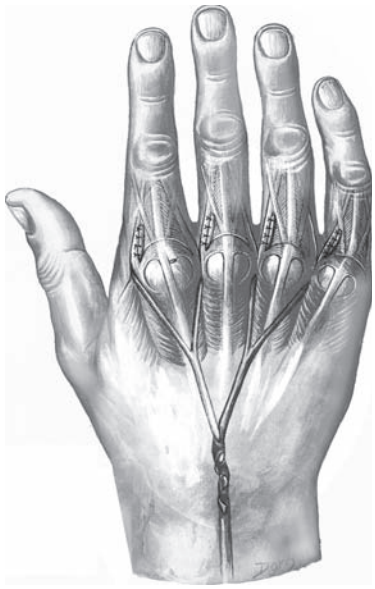


Figure 18.12

Dorsal approach. Transfer of the EIP. The EIP tendon is transferred to the two ulnar fingers passing dorsal to the wrist and palmar to the deep transverse metacarpal ligament.[19] A tendon graft is fixed to the transfer for the two radial fingers.[20]

Point of insertion

Side of insertion on the finger Bunnell inserted the transfers to reproduce intrinsic-like action on both sides of each finger. Later authors advocated unilateral insertions usually on the radial side of the fingers.

How far palmar to place the insertion (Figure 18.13a) The more palmar the insertion of the transfer with regard to the axis of rotation of the MP joint, the more marked will be the flexion of the joint. A transfer passing dorsal to the deep transverse intermetacarpal ligament and fixed to the tendon of the interosseous muscle has a moment of flexion less than that of a transfer passed volar to the ligament (Figure 18.13a).

How far distal to place the insertion (Figure 18.13b) In Bunnell's technique, the transfer was inserted into the oblique fibers to the interosseous hood, the MP joint being kept in flexion and the IP joints in extension. The dangers of transforming the claw deformity into a swan-neck by an overpowerful transfer are well known. These dangers are magnified if the flexor superficialis is sacrificed and the IP joints are lax. A distal insertion of the transfer into the interosseous hood is only useful if it is difficult to achieve active extension of the distal phalanges with the MP joints stabilized in slight flexion.

When stabilization of the MP joint in flexion allows the IP joints to extend, it is functionally more useful to reinforce proximal phalangeal flexion than distal phalangeal extension. The transfer will then be fixed more proximally, to the interosseous tendon, into the proximal phalanx[21,22] or in the fibrous flexor sheath.[24]

How many fingers should be rebalanced?

Although clawing of the index and long fingers is usually absent in low ulnar nerve palsy, inclusion of all four fingers in the transfer is recommended for improved asynchronous finger motion and dexterity.

It is not possible to mention the many tendon transfer procedures devised for the treatment of intrinsic muscle

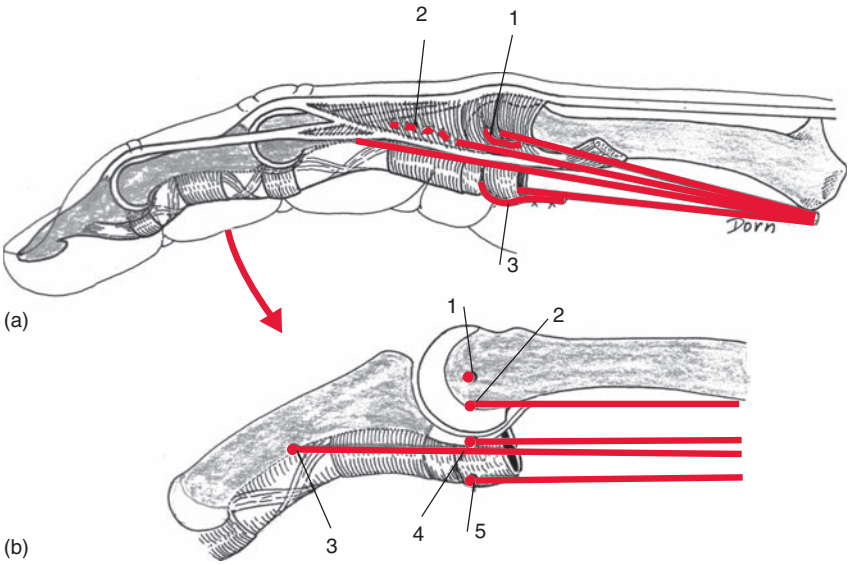


Figure 18.13

How far palmar and how far distal to place the insertion. (a) (1) Insertion on the transverse fibers of the extensor hood. (2) On the oblique fibers of the hood.[18] (3) On the pulley A, of the flexor tendon sheath.[23] (b) (1) Axis of rotation of the metacarpal head. (2) Site of the interosseous tendon. (3) Insertion into the proximal phalanx.[21,22] (4) MP joint volar plate. (5) Pulley A1.

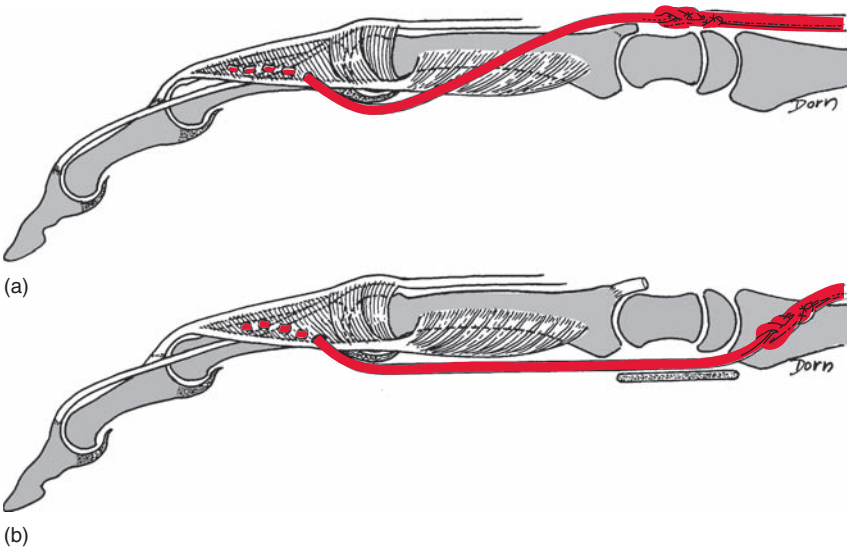


Figure 18.14

Brand's techniques. (a) The transfer is dorsal to the wrist. (b) The transfer is passed through the carpal tunnel. With a volar path of the transfer, a more physiologic grip may be expected with the wrist extended. However, a transfer dorsal to the wrist may be preferable in longstanding cases in which flexion of the wrist associated with extension of the fingers cannot be corrected by occupational therapy.

paralysis. Three procedures using different motor transfers will be described:

- transfer of a wrist motor
- transfer of an FDS
- transfer of the extensor indicis proprius (EIP).

Transfer of the extensor carpi radialis longus (ECRL)

Brand[25] started by transferring the ECRL, prolonged by four dorsal tendon grafts fixed on the interosseous hood (Figure 18.14a) Later, the wrist extensor and the grafts were passed through the carpal tunnel[10,26] (Figure 18.14b).

Four slips of tendon graft are required to prolong the ECRL for insertion into the proximal phalanges (Figure 18.15). The plantaris tendons from both lower extremities are readily accessible, and each will typically supply two tendon graft lengths. These slips are harvested through limited incisions using a tendon-stripping instrument.

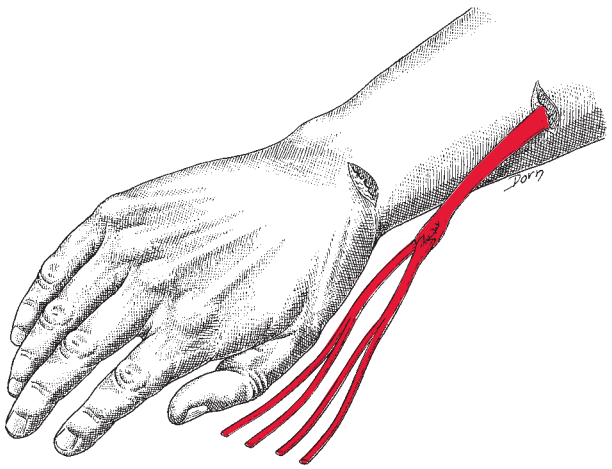


Figure 18.15

The ECRL tendon is prolonged by four slips of tendon graft for insertion into the proximal phalanges.

The long toe extensors may be used if the plantaris tendons are absent or of insufficient size.

The division of terminal slips must be in the proximal palm, so that their course is not interrupted by the vertical septa of the palmar aponeurosis. A palmar incision is used to provide good exposure.

The superficial palmar aponeurosis is divided. Short lateral incisions are made on the base of the fingers. A curved tendon passer is placed through the lumbrical canal from distal to proximal to help deliver the transfer to the proximal phalanx of each finger. The slips are pulled together so that their progression is symmetric and none regresses as its neighbor is pulled distally. Such a regression indicates that an obstacle is impeding the tendon's progression or that the division of the tendon into slips is not proximal enough.

Brand prefers an ulnar side insertion in the extensor aponeurosis for the index finger to avoid separation of index and middle fingers, which allows small objects to slip through. He also believes that an ulnar insertion for the index facilitates its rotation in supination toward the thumb. Most authors still advocate a radial insertion for the index finger to restore the action of the first dorsal interosseous and to reinforce the resistance of the index finger against the pressure of the thumb.

Transfer of the flexor digitorum superficialis

In Bunnell's procedure all four FDS tendons were transferred. Borrowing all the superficialis not only is wasteful but inevitably influences the movements and grip strength of the fingers. As a result, several authors, including Littler[27] and Goldner,[28] suggested alternative methods whereby only one or two superficialis flexor tendons were taken, divided into bands, and inserted on one side only (usually the radial) of each finger (Figure 18.16).

Zancolli[24] described an ingenious technique called the 'lasso'. A superficial flexor tendon is used. The tendon is harvested through a hole made in the tendon sheath at a level proximal to the PIP joint, care being taken to be proximal to the chiasma. One band of insertion of the FDS is cut, and tension is applied, delivering the second band into the hole in the tendon sheath, which is cut in turn. The two bands are pulled into the palm. Each is divided into two slips, each sutured to itself after a loop (lasso) has been made around the proximal pulley (Figure 18.17a). Thus, it becomes a flexor of the proximal phalanx and stabilizer of the MP joint during extension of the finger.

The tension placed on the transfer depends on the laxity of the finger joints, because even a lasso procedure can cause a swan-neck deformity if there is too much tension or if the PIP joints are too lax, although it has no direct insertion on the extension aponeurosis. Zancolli recommended fixation of the lasso with the MP joints extended and maximum tension on the transfer.

The loop is sutured back to itself by three sutures, the most distal suture on the proximal border of the A1 pulley.

Transfer of the EIP

The EIP tendon is slightly short. To avoid a suture under tension, Zancolli[23] passed the tendon through the interosseous membrane of the forearm distal to the pronator quadratus, and then through the carpal tunnel. It is split and the two slips are fixed with the lasso technique (Figure 18.17b).

Simultaneous correction of claw deformity and abduction deformity of the little finger

Belmahi et al[29] have described a technique that allows the simultaneous active correction of both Wartenberg's sign and claw deformity of the ring and little fingers with ulnar nerve palsy. The FDS of the ring fingers is divided into two strips up to the proximal part of the palm. The radial strip is used in a Zancolli's lasso procedure to treat the claw deformity of the ring finger. The ulnar strip is wrapped around the base of the little finger, using palmar and dorsal approaches at the level of the proximal phalanx, like a necktie, being medial to its radial pedicle, dorsal and superficial to its extensor apparatus, and then lateral to its ulnar pedicle (Figure 18.18). It is then recovered in the palm and sutured to itself, with the wrist in neutral position, the little finger in complete adduction, and 30° MP flexion.

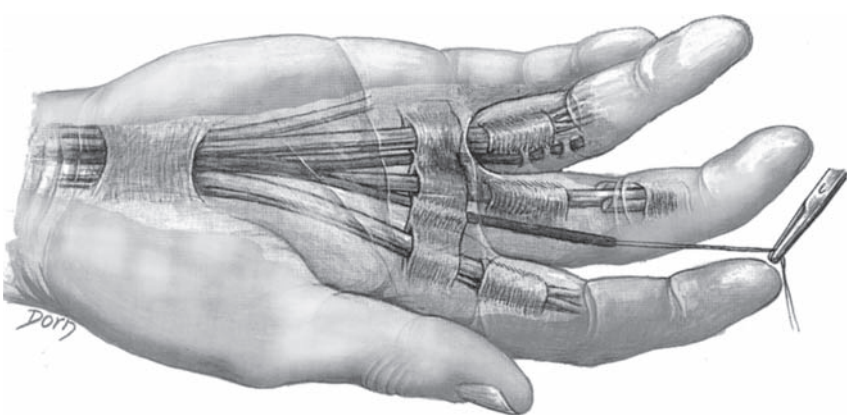


Figure 18.16

Alternative Bunnell's technique. One or two FDS tendons are divided into bands passed through the lumbrical canal under the intermetacarpal ligament and are fixed dorsally on the oblique fibers of the interosseous hood on the radial side of each finger.

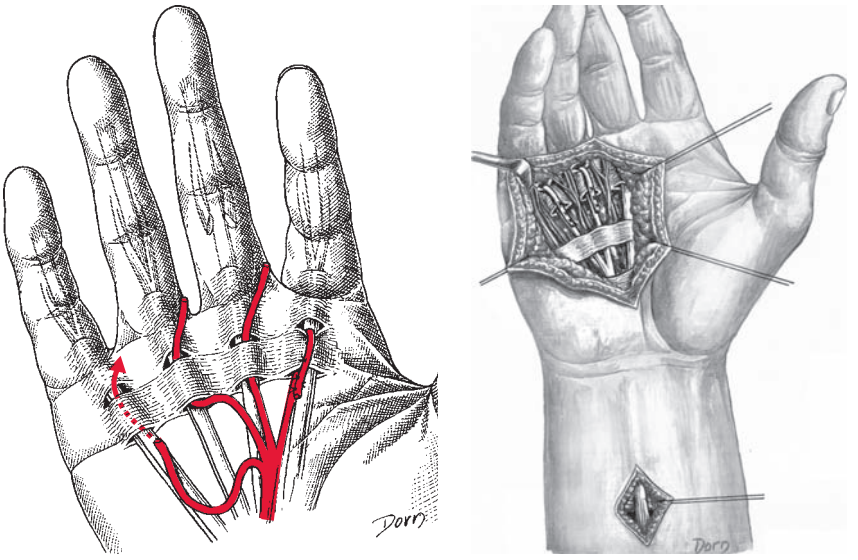


Figure 18.17

(a) Zancolli's lasso technique. The FDS tendon of the long finger is harvested proximal to its chiasma. It is divided into four bands; each band is inserted at the base of each respective finger using the lasso procedure around the A1 pulley of the flexor tendon sheath. Note that the most distal suture on the loop is fixed on the proximal border of the A1 pulley. (b) Zancolli's transfer of the EIP, through the interosseous membrane, for ulnar palsy.

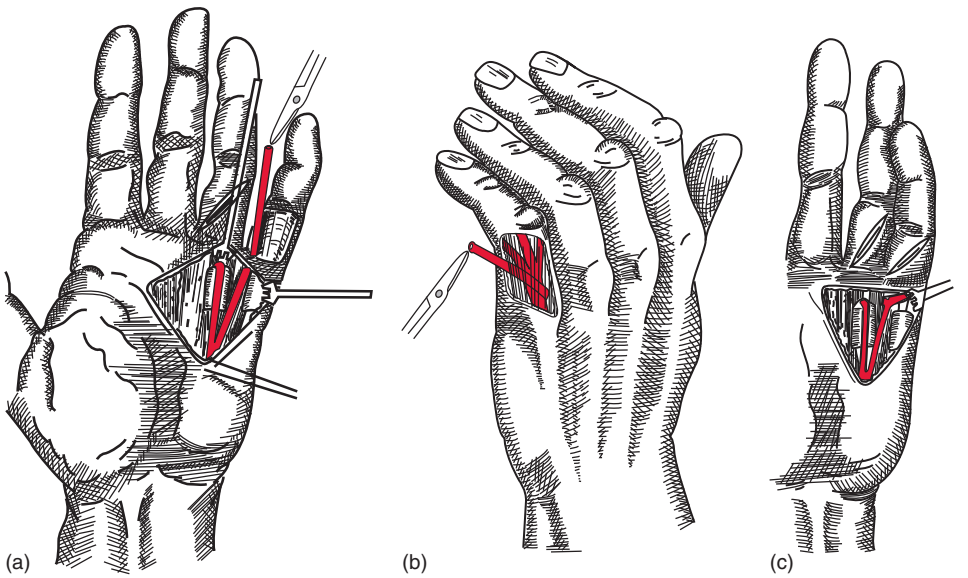


Figure 18.18

Belmahi's technique for simultaneous correction of claw deformity and abduction deformity of the little finger. (a) The FDS of the ring finger is divided into two bands. The radial strip is used as a Zancolli's lasso procedure. (b) The ulnar strip is wrapped aroud the base of the little finger, using palmar and dorsal approaches, and will be sutured to the interosseous hood after adjustment of the tension. (c) The ulnar strip is sutured to itself or to the flexor tendon sheath if it is not long enough, with the wrist in neutral position, the little finger in adduction, and 30° of MP flexion.

Postoperative care

When the transfer is fixed on the extensor aponeurosis, the IP joints are directly extended by the transfer; the immobilization includes the wrist in slight extension, the MP joints in 60° flexion, and the IP joints in extension. After 3 weeks, the IP joints are mobilized, where the MP joints remain in flexion for 2 weeks more.

The category of transfers that aim only at actively flexing the MP joints without directly extending the IP joints allows early mobilization of the IP joints. It is sufficient to maintain immobilization of only the MP joints in 60° of flexion for 1 month.

Tenodeses

These procedures aim only at preventing hyperextension of the MP joints, thus allowing the long extensors to extend the distal phalanges. Many techniques of tenodesis have been described for the treatment of paralysis of the intrinsic

muscles of the fingers. When a tendon fixed at its two extremities crosses one joint, it simply limits that joint's range of motion. When it crosses two joints, it can transmit the movement of one joint to that of the other joint; it becomes a 'dynamic tenodesis'.

We cannot describe all the tenodesis procedures used for correction of the claw-hand. The simplest involves a tendon graft fixed to the metacarpal, passed through the lumbrical canal in front of the deep transverse metacarpal ligament, and then fixed to the interosseous hood.[23]

A modification of this procedure used on leprous patients fixes the graft proximally to the extensor tendon itself.[30]

In Parkes' tenodesis,[31] a plantaris tendon graft is passed through the lumbrical canal. It is fixed distally to the interosseous hood and proximally in the flexor retinaculum (Figure 18.19). Its tension maintains graduated MP flexion between 45° in the little finger and 30° in the index finger, with the IP joints in extension. These grafts are activated by the extrinsic muscles of the fingers.

If the proximal end of the tenodesis is attached above the wrist, movements of the wrist may result in activation of the tenodesis. For example, in Fowler's technique,[19] a graft is fixed to the extensor retinaculum, whereas in Tsuge's technique,[32] the transfer is the tendon of the brachioradialis divided at its musculotendinous junction, then reflected and split into four strips, each of which is fixed in the interosseous hood.

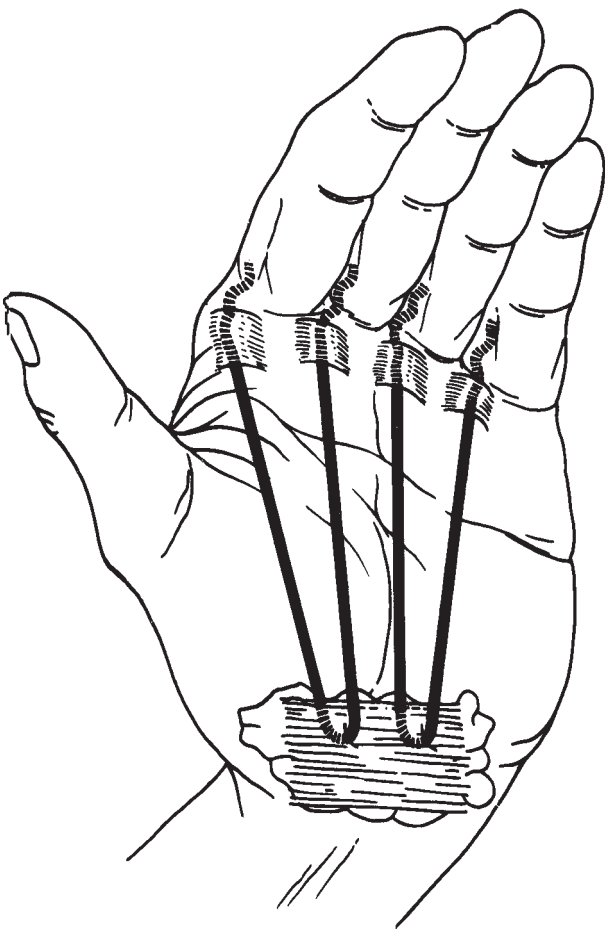


Figure 18.19
Parke's tenodesis.

Capsulodesis

Zancolli[23] advocated the use of capsulodesis to prevent MP joint hyperextension. A transverse incision in the distal palmar crease provides access to the MP joints. The radial side of the A1 pulley is opened and the flexor tendons are retracted laterally (Figure 18.20).

Zancolli makes a longitudinal division in the volar plate and advances the two sides to be sutured through drill holes in the metacarpal neck, creating slight MP flexion (between 15° and 30°). The MP joints are immobilized in flexion for a month, while the IP joints are left free.

Fixation to bone introduces a degree of technical difficulty but appears to strengthen the capsulodesis.

To simplify the procedure for 'surgery en masse' in leprosy, Bourrel[33] devised an H-shaped capsular incision. This defines two capsular flaps: one based proximally and the other distally. A transverse resection of 6 mm from one or the other of the H-flaps is made as required, and the edges are approximated.

One of the advantages of capsulodeses is that they do not necessitate the plundering of an already handicapped hand for tendon grafts. Also, they can correct the claw deformity whatever the position of the wrist, and they can be redone in cases of failure.

In fact, to approach the volar aspect of the MP joints in capsulodesis procedures, one must retract the flexor tendons and divide the A1 pulley. This in itself constitutes a 'pulley advancement' as advocated by Bunnell[34] (Figure 18.21). The effect of this advancement for Bunnell is to increase the flexion of the MP joint. By thus altering the points of application of the flexion forces, some improvement in the sequence of phalangeal flexion is obtained. The disadvantage of pulley advancement is that it favors ulnar drift of the fingers.

Indications of surgery for the treatment of intrinsic muscle paralysis of the long fingers

Surgical indications are governed by the claw deformity and the severity of the functional problems.

It must be determined whether the claw can be actively corrected by Bouvier's test (see Figure 18.9), and, if not, whether passive correction is possible.

Functional problems alone may justify surgery. An abnormal flexion sequence, beginning distally, is often a greater problem than the claw. The weakness of grip is determined by the level and extent of the paralysis. It is important to increase the strength of finger flexion when

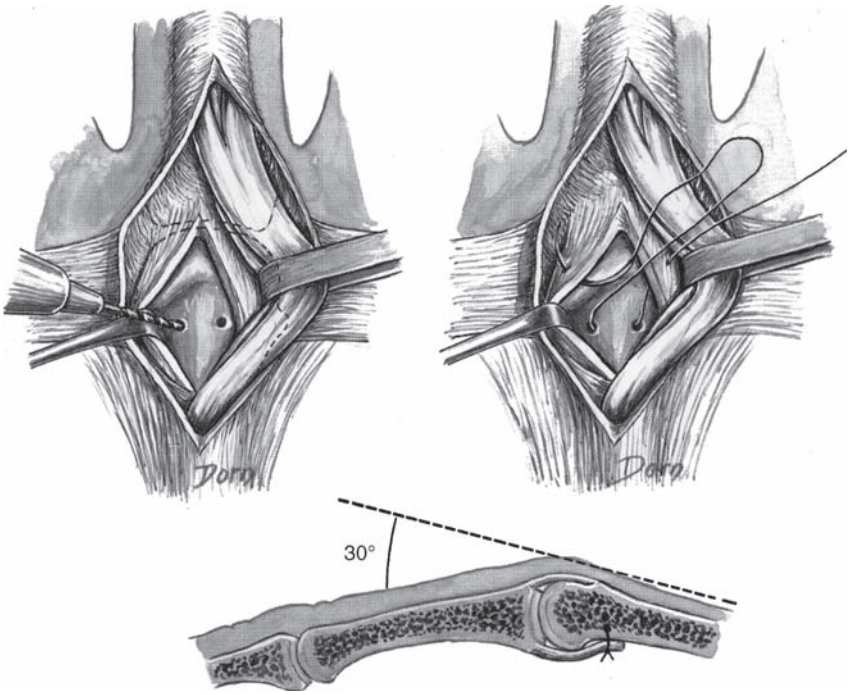


Figure 18.20

Capsulodesis. Zancolli's technique of transosseous fixation. To simplify the procedure, Bourrel[33] has devised an H-shaped capsular incision: this defines two capsular flaps: a transverse resection of 6 mm is made; and the capsular edges are approximated. The use of 'bone anchors' has facilitated bone fixation.

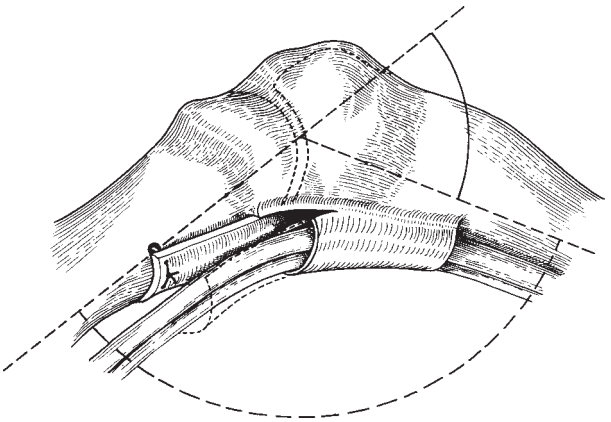


Figure 18.21

Pulley advancement.

paralysis of the long flexors is added to paralysis of the intrinsics.

Reinforcement of thumb–index lateral pinch is another important problem (see Chapter 19 on restoration of thumb–index lateral pinch).

When the claw is actively correctable

In these cases, MP hyperextension can be prevented by tendon transfer, tenodesis or capsulodesis. All these methods can correct the deformity.

Passive techniques have essentially only one effect, namely, correction of the deformity. Their use should be limited to the following conditions:

- when transfers are not available, because of extensive paralysis
- when there is a possibility of intrinsic muscle recovery after long delays
- when conditions for surgery are unfavorable.

These easy passive procedures allow surgery 'en masse' in developing countries.

Tendon transfers are indicated in all other conditions

The choice is influenced by the age and expectations of the patient, the level and extent of paralysis, and also eventually on associated lesions, such as injury to the volar wrist structure, or the laxity of finger joints, and on the particular neads of each patient. One must distinguish between distal paralysis in which flexor digitorum profundus (FDP) strength is maintained, and proximal paralysis, in which the FDP is lost. One must not forget that superficialis tendons from the ring and little fingers cannot be used in high ulnar palsy, because this would deprive these digits of an active flexor.

Use of a finger flexor for transfer simply redistributes balance within the hand, but diminishes grip strength if it is inserted into the interosseous hood. Transfer of a wrist flexor or extensor muscle–tendon unit will increase grip strength, and is preferable in proximal paralysis and even in distal paralysis for young patients with high functional demands. For other cases, a weak transfer such as that of the EIP will sufficiently stabilize the MP joint, correct the claw, and restore physiologic flexion of the finger.

It must be remembered that restoration of thumb strength and reinforcement of the thumb–index lateral pinch will also require another transfer (see Chapter 19).

Figure 18.22

Prevention of hyperextension of the MP joints does not allow extension of the distal phalanges: the claw is not actively correctable.

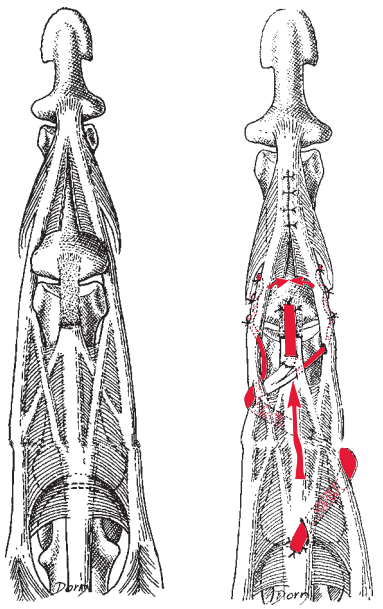


Figure 18.23

The trident graft. The graft woven through the extensor apparatus is divided distally into three bands: a central band fixed to the base of the middle phalanx, and two small bands detached from the graft proximal to the PIP joint and fixed to the lateral extensor tendons, which are brought out on the dorsal aspect of the joint. The graft can be activated by a tendon transfer.

When the claw is not actively correctable (Figure 18.22)

In this case, passive procedures that aim to prevent hyperextension of the MP joint are not adequate. One should look for the cause of this problem and try to correct it.

When passive motion of the three finger joints is conserved, a deficit of the extension of the distal phalanges, despite the maintenance of the MP joints in flexion, in most cases indicates a lesion of the extensor apparatus. An extension deficit greater than 30° is an indication for a transfer fixed on the interosseous to provide distal extension.

In longstanding flexion deformity of the PIP joint, the extensor apparatus distends and the lateral extensor tendons sublux to either side of the joint, resulting in a boutonnière deformity. The boutonnière may be corrected by a trident graft[35] woven through the extensor apparatus, which reinforces the central extensor tendon and corrects the palmar subluxation of the lateral extensor tendons (Figure 18.23).

A number of other factors can prevent active correction of the claw deformity: skin contracture, flexor or extensor tendon adhesions, and articular stiffness. These should be analyzed and treated before any transfer.

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19 Restoration of thumb–index lateral pinch

Raoul Tubiana

Thumb–index lateral pinch involves the palmar surface of the terminal phalanx of the thumb and the lateral aspect of the index finger (Figure 19.1). Its restoration is particularly indicated in extensive and complicated upper limb paralyses.

Anatomy–physiology

Several muscles are involved in the thumb–index lateral pinch, mainly the following:

- The *adductor pollicis (AP)*, the strongest muscle of the thumb ray, not only adducts the first metacarpal but also extends the distal phalanx and flexes the proximal phalanx.
- The *extensor pollicis longus (EPL)* not only contributes to the extension of the two phalanges of the thumb, but also brings the first metacarpal into retroposition and extension, and the proximal phalanx into ulnar deviation.

The AP and EPL are not only adductors of the thumb column, they also have a rotation action in supination.

- The *first dorsal interosseous* (Figure 19.2), a complex functional unit, adducts the first metacarpal and flexes the metacarpophalangeal (MP) joint of the index finger. It has two different fiber lengths: 3–6 cm for the bundles originating from the first metacarpal and 1–6 cm for the fibers originating from the second metacarpal.[1] Its muscle mass fraction of 1.4% is more than that of the extensor indicis proprius (EIP) (1.1%).
- The *flexor pollicis longus (FPL)* and *brevis (FPB)* also play a role in thumb adduction (see Chapter 16 on biomechanics of the thumb).

The EPL is innervated by the radial nerve, and the AP and first dorsal interosseous by the ulnar nerve. If, as is frequently the case, the two heads of the FPB are predominantly innervated by the ulnar nerve, the motor deficit in the case of ulnar palsy is considerable and the stability of the thumb column is jeopardized. In the absence of the stabilizing action of the AP and of the FPB, the MP joint of the thumb becomes lax and is displaced in hyperextension during the pinch (Jeanne's sign), while the distal phalanx flexes (Froment's sign) (Figure 19.3).

The index ray is still balanced in isolated ulnar palsy, due to the action of the first lumbrical muscle innervated by the median nerve, which prevents a claw deformity.

When the thumb is apposed to the side of the index finger, if index resistance cannot be maintained by the first dorsal interosseous, then either the index will collapse ulnarwards, or the middle finger will be brought in to support the index.

If, in median–ulnar paralysis, the lumbrical muscles of the index and middle fingers are also paralyzed, the middle finger will not be able to support the index, and the articular chains of both the thumb and index rays will be destabilized.

Clinical aspect

Thumb–index lateral pinch has different clinical aspects according to the extent of the paralyses and according to the level of the pinch on the lateral side of the index.

The pinch between the pulp of the thumb and the lateral side of the proximal phalanx of the index finger, or *key pinch*, is probably the most useful to be restored. It persists even in median–ulnar paralysis, because of the action of the EPL, but is very weak and needs to be reinforced. The thumb in supination tries to contact a sensible skin area on the dorsolateral aspect of the index finger, innervated by the radial nerve.

The more distal the pinch on the lateral aspect of the index, the more longitudinal rotation of the thumb column in pronation is found (Figure 19.4).

However, restoration of lateral pinch necessitates the possibility of an active separation between the thumb and index. This means abduction of the first metacarpal and extension of the thumb MP joint, and absence of soft tissue contraction in the thumb web.

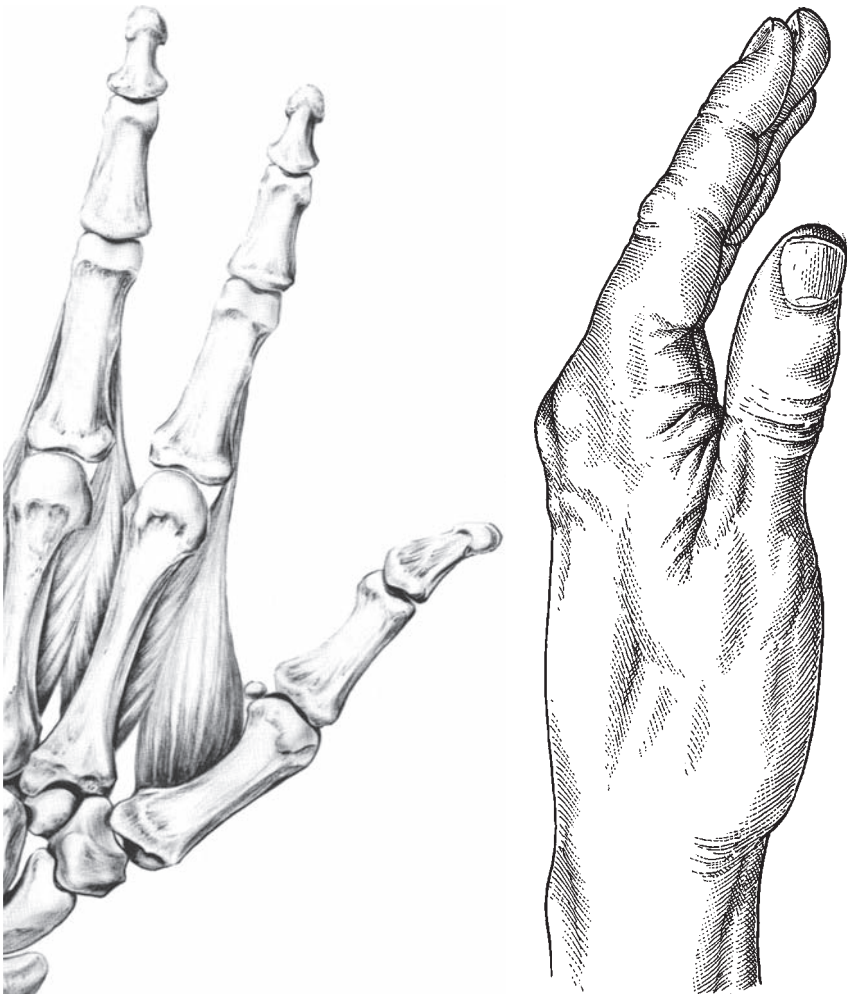

Figure 19.1
Thumb index view: (a) osteomuscular anatomy; (b) lateral view.

Treatment

Restoration of thumb–index lateral pinch necessitates adduction of the thumb and an active resistance of the index finger. The problem is quite different in case of ulnar palsy and in associated median–ulnar palsy. Thumb adduction can be restored by a direct tendon transfer in the case of ulnar palsy. The choice of a transfer is more limited in combined median–ulnar palsy, especially if nerve lesions are proximal. In extensive proximal paralyses, tenodeses utilizing wrist movement may be the only possibility for restoration of a thumb–finger pinch. We shall describe:

- direct tendon transfer for thumb adductorplasty
- active thumb tenodeses
- stabilization of the index finger.

Thumb adductorplasty

Many procedures have been described to restore thumb adduction. They must take into account the etiology and the extent of the paralyses. The AP muscle, innervated by the ulnar nerve, can be paralyzed by an ulnar nerve lesion, by combined median–ulnar paralysis, or more proximally by a brachial plexus or cervical spine lesion.

In ulnar nerve palsy

The AP is paralyzed, but anteposition is conserved. Thumb opposition is possible, but the strength of the thumb–finger grip is considerably reduced – which is the main complaint of the patients.

The therapeutic goals are to (1) increase adduction power for the grip, (2) stabilize the MP joint, (3) correct the extension defect in the distal phalanx of the thumb, and (4) often reestablish abduction of the index to provide support and resist pressure from the thumb; (5) anteposition and pronation of the thumb column should not be jeopardized.

The motor muscle can be a flexor digitorum superficialis (FDS) – preferably that of the ring finger when the palsy is distal. When the palsy is proximal, the FDS of the middle finger may be used, but this may reduce even more the power of the digitopalmar grip, and it is better to use a muscle innervated by the radial nerve, such as the EIP or

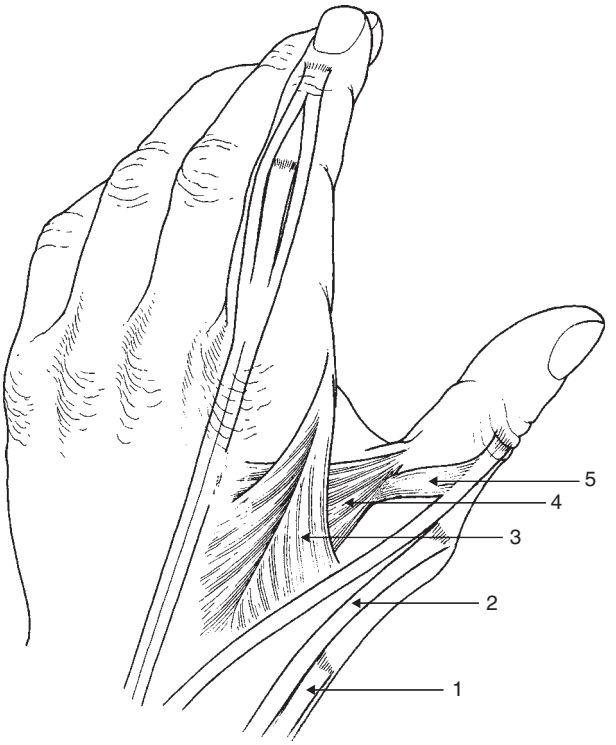

Figure 19.2

Dorsal view of the thumb web: (1) abductor pollicis longus (APL); extensor pollicis brevis (EPB); (3) first dorsal interosseous; (4) adductor pollicis (AP); (5) expansion of the AP to the extensor pollicis longus (EPL).

the extensor carpi radialis longus (ECRL). Use of the brachioradialis (BR) has been advocated by Omer.[2] The FDS and the EIP tendons are long enough; however, the ECRL or the BR must be lengthened by a tendon graft.

Path of transfer. It is necessary to take into account the condition of the lateral thenar muscles.[3] If the MP joint is put in hyperextension when the patient is asked to form a pinch between the thumb and fingers (Jeanne's sign) one may assume that the FPB is in large part paralyzed. If, on the contrary, when the patient is requested to produce such a pinch, the MP joint is put into slight flexion (reverse Jeanne's sign), one may assume that the FPB muscle is producing an effective contraction.

In cases of extensive paralysis of the FPB, the persistent lateral thenar muscles abductor pollicis brevis (ABP) and opponens pollicis (OP) would be unable to counterbalance the effect of a powerful deep transfer re-established in the path of the transverse fibers of the adductor inserted on the ulnar aspect of the thumb column; rather, it is the action of the FPB that must be restored and reinforced. The transfer should be deep, and should approach the thumb from its radial side like the FPB in order to be fixed to the tendon of the APB as well as to the EPL.

When the FPB is still active, the path of the transfer may be more transversal, and some authors fix the transfer on the adductor tendon on the ulnar side of the thumb column.

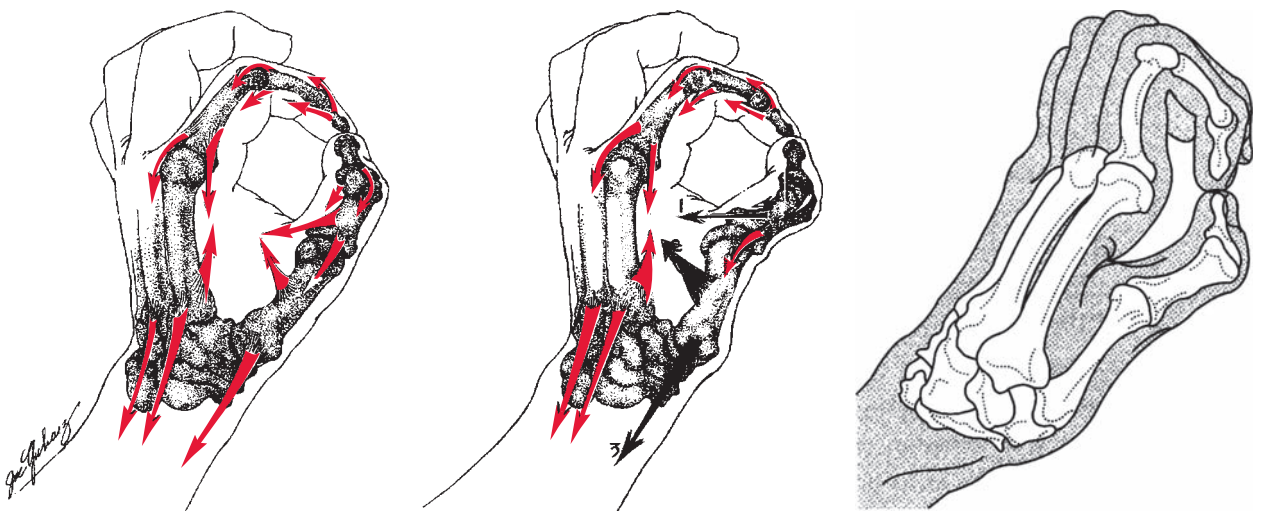

Figure 19.3

Thumb–index terminal pinch in ulnar nerve palsy is less efficient than a lateral pinch. (a) When the thumb MP joint is still stabilized by the FPB, the patient can make an 'O' between the thumb and index in spite of the flexion of the interphalangeal joint (Froment's sign). (b) In the absence of the stabilizing action of both the AP and FPB, the patient cannot make an 'O' (Jeanne's sign). (c) In median–ulnar palsies, the first and a second lumbrical are also paralyzed, in addition to the interosseous. The index MP joint has the tendency to hyperextend (Mannerfelt). (Parts (a) and (b) by courtesy of Dr L Mannerfelt.)

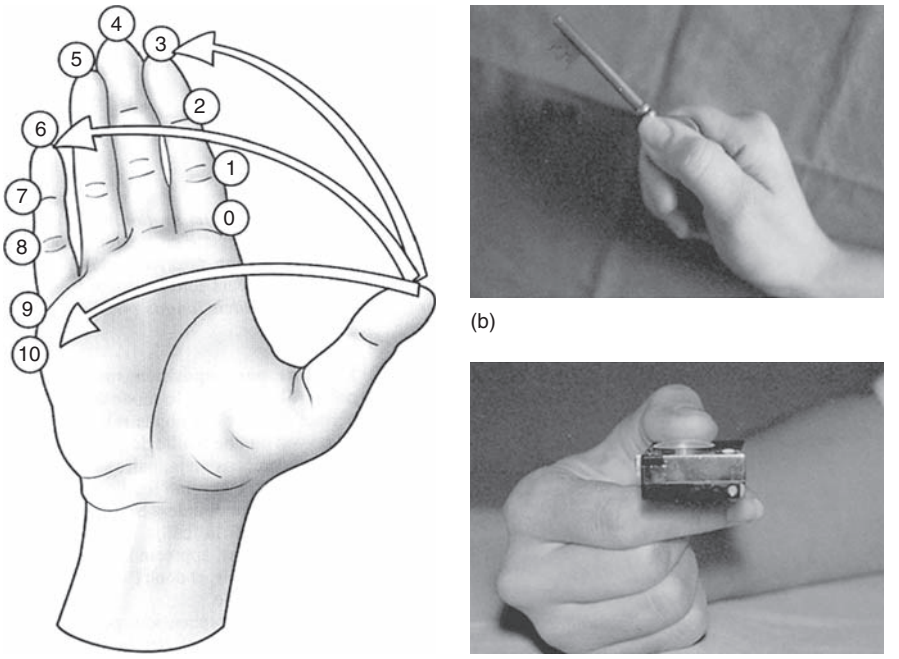

Figure 19.4

(a) Kapandji's method for clinical evaluation of thumb–index pinch. The method is based on the successive thumb–fingers pinch, on the lateral aspect of the index, and then during the wide course of opposition and of the ulnar aspect of the little finger. Eleven stages are defined. The first three stages show the relation of the thumb's pulp with the lateral aspect of the index: in stage 0, the pulp of the thumb is located on the lateral aspect of the proximal phalanx of the index finger; in stage 1, the tip of the thumb is in contact with the lateral aspect of the index middle phalanx; in stage 2a, the tip of the thumb is in contact with the lateral aspect of the index distal phalanx. The more distal the pinch, the more longitudinal rotation of the thumb column is found. (b, c) Two examples of thumb–index lateral grip. Lateral pinch is not only one of the most useful varieties of grips, but also the easiest to reconstruct surgically.

Techniques preferred by the author

1. In the case of predominant ulnar nerve innervation of the FPB, when there is Jeanne's sign, the motor muscle is preferably strong, and I use the FDS of the ring finger in distal ulnar nerve palsies. Four incisions are necessary (Figure 19.5a): one at the base of the ring finger to remove the tendon, a second in the palm distal to the flexor retinaculum, a third above the wrist, and a fourth opposite the MP joint of the thumb. The tendon is divided proximal to the Camper's chiasma, and is pulled out proximal to the wrist. It will be rerouted to follow a new path.

I prefer to avoid an overly acute angle in the transfer in order to reproduce the transverse direction of the adductor. I reserve this path for the transfer in cases of severe paralyses when it is sufficient to restore a lateral grip and not opposition.

A long curved forceps (Figure 19.5b) is introduced through the palmar incision, in a proximal direction, and continued into the carpal, deep in relation to the deep flexor tendons of the fingers. The tip of the forceps, exposed proximal to the wrist, is used to reroute the transfer laterally toward the thenar eminence. It is finally brought in the direction of the FPB toward the radial side of the MP joint (Figure 19.5c). The transfer is woven into the tendinous insertion of the dorsal expansion of the APB, and finally fixed to the EPL, with the distal phalanx kept in extension. When there is an instability of the MP joint, the fixation of the transfer is used to stabilize the joint (see Figure 21 in Chapter 17).

2. When the FPB remains active preventing hyperextension of the MP joint when it contracts, I prefer to use a less powerful muscle for the transfer. Transfer of the EIP is preferably used. The EIP tendon is drawn out above the wrist and passed volarly through the interosseous membrane (Figure 19.6), and then into the carpal tunnel under the flexor tendons. It is fixed into the tendinous insertion of the APB and its dorsal expansion is fixed to the EPL.

An alternative path for the transfer is possible, avoiding forearm incisions. The EIP tendon can be rerouted around the ulnar border of the third metacarpal bone (Figure 19.7), and then across the palm volar to the adductor pollicis muscle and dorsal to the finger flexor tendons and neurovascular structures.

In median–ulnar palsy

All intrinsic muscles of the thumb are paralyzed; the thumb column is activated only by the extrinsic muscles.

The thumb ray is in retroposition rotated in supination, with the phalanges flexed. The main problems are the lack

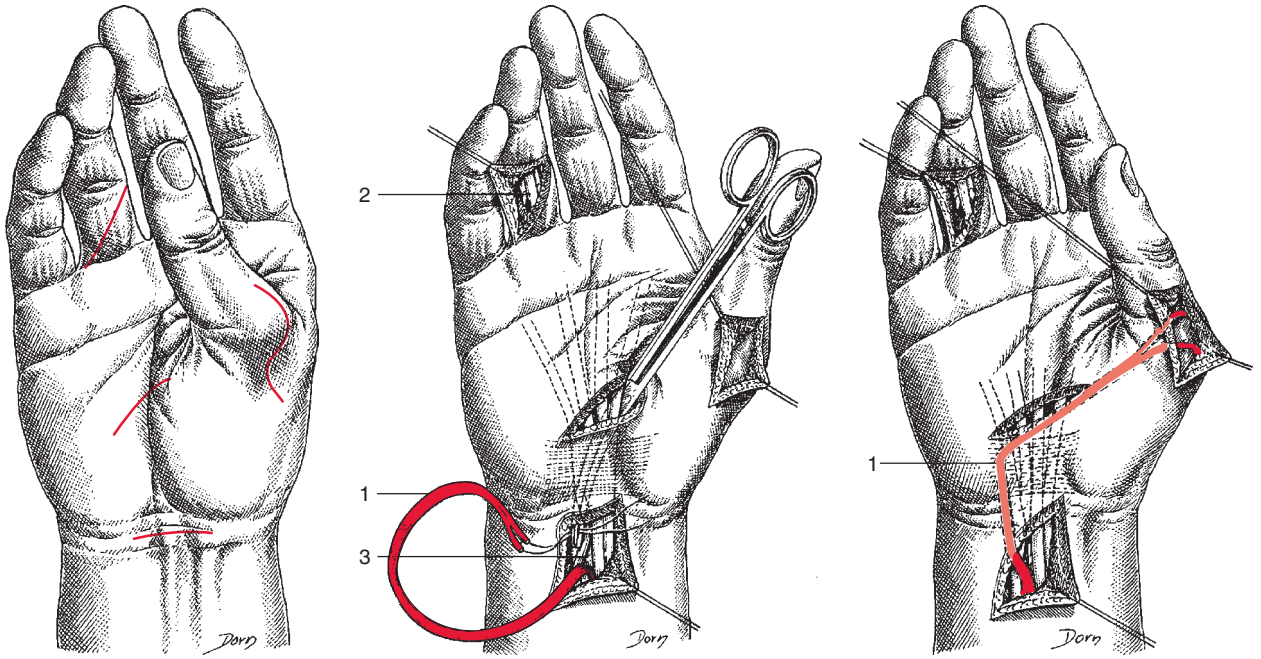

Figure 19.5

(a) Transfer of the FDS of the ring finger for paralysis of the AP and an important deficit of the FPB: the four incisions. (b) The FDS of the ring finger is passed around the ulnar border of the flexor tendons and deep to them in the carpal tunnel. (1) FDS of ring finger; (2) FDP of ring finger; (3) FDS of the little finger. (c) The rerouted FDS tendon (1) follows the direction of the FPB toward the radial side of the MP joint to the thumb.

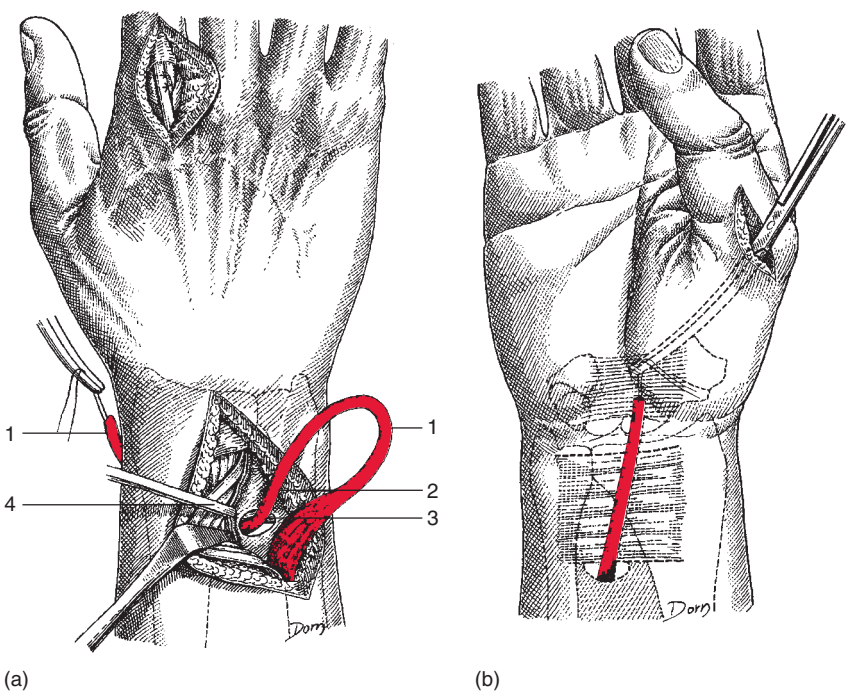

Figure 19.6

EIP transfer. The direct path of the transfer allows maximal advantage to be taken of the active tenodesis effect of wrist flexion and extension. No pulley is necessary. (a) Dorsal view. (b) Palmar view. (1) The EIP tendon is extracted proximally into the forearm. It is easy to recognize, because it has the most distal muscle belly of all the digital extensors. It is then transferred volarly, preferably through the interosseous membrane. (2) The opening in the interosseous membrane is usually made proximal to the upper border of the pronator quadratus (3); it must be large enough to allow easy passage of the muscular body of the EIP, about 3 cm long and 1.5 cm wide. Care is taken to protect the interosseous neurovascular pedicles (4).

of sensibility in the palmar aspect of the fingers and early contracture of the first commissure preventing passive movement of opposition. In most of these cases, it is preferable to restore a strong thumb–index lateral pinch rather than thumb opposition (see in Chapter 17).

The *motor muscle* can be the FDS of the ring finger when the palsy is distal. When the associated median–ulnar palsy is proximal, the EIP or the ECRL lengthened by a tendon graft (palmaris longus or a long toe extensor) are utilized. We are reluctant to use the extensor carpi radialis brevis

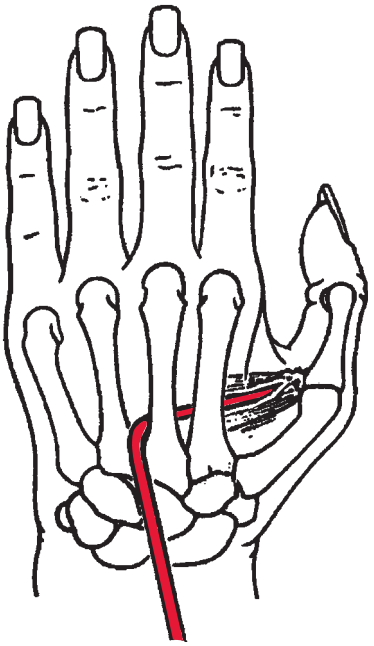

Figure 19.7

The EIP tendon is rerouted around the ulnar border of the third metacarpal bone.

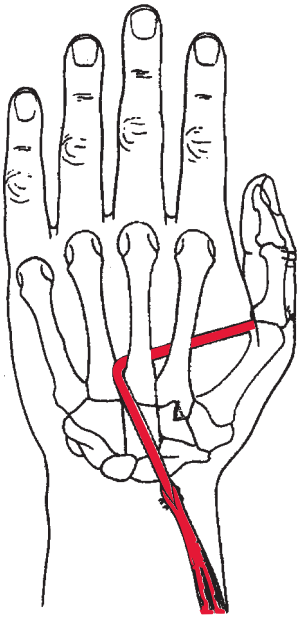

Figure 19.8

ECRL transfer prolonged by a tendon graft.

(ECRB), the main extensor of the wrist, although it has been used by several authors, with good results on thumb pinch strength.[4–6]

The BR can also be used, but only if the elbow is stabilized by the triceps – if not, it will exhaust its action in flexing the elbow. Of course, the choice of the motor for thumb adductorplasty is influenced by the procedure used for the correction of the intrinsic muscles paralysis of the long fingers.

Path of the transfer

The path depends on the motor chosen. A *flexor superficialis* tendon is rerouted after its passage through the carpal tunnel.

In the case of an *EIP* transfer, the tendon is withdrawn under the extensor retinaculum into the forearm. It is then rerouted subcutaneously in a straight line with the fourth extensor compartment and passed through the 3–4 intermetacarpal space to the palmar aspect of the hand. The EIP tendon end then follows a subcutaneous tunnel created with a curved clamp toward the MP joint of the thumb.

In *ECRL transfer*, the ECRL tendon is divided at its insertion to the base of the index metacarpal and withdrawn under the extensor retinaculum into the forearm. A tendon graft is necessary. The graft is passed bluntly along the palmar aspect of the transverse head of the adductor and between the third and fourth metacarpals to the dorsum of the hand. It will be sutured to the stump of the ECRL tendon in the dorsal forearm. As the two tendons are of different caliber, a Pulvertaft 'fish-mouth' transfixation procedure is used (Figure 19.8). Careful positioning of this junction is necessary, away from the intermetacarpal space and the extensor retinaculum.

Site of insertion of the transfer

The transferred tendon or the graft is woven into the tendon of the AP and of its dorsal expansion to the EPL, with the wrist in neutral position and the thumb ray fully extended.

Postoperatively, the hand is placed in a splint with the wrist in neutral position and the thumb ray extended. After 4 weeks, passive mobilization is begun; active mobilization is started at 6 weeks.

Stability of the thumb MP joint is essential to ensure a strong grip. In cases of joint laxity; different techniques for stabilizing the joint should be used: tenoplasty, capsulodesis, or arthrodesis.

Active thumb tenodeses

When paralyses of the intrinsic muscles of the thumb are added to extensive paralyses of extrinsic muscles, it is essential to take into account whether active extension of the wrist is possible.

In order to obtain a useful thumb grip in cases of extensive paralyses, several conditions must be fulfilled:

1. Stabilization of one or two joints of the thumb ray in order that one motor tendon will be able to control the three joints.
2. Active wrist extension will reinforce the action of the transfers. It is necessary for the wrist to have active extension of at least 30°.
3. Obviously, it is preferable to have two transferable motor units to the thumb: one to ensure its opening and the

other its closure. This latter motor unit should be more powerful because it ensures the pinch strength.

4. If there is only one active muscle transferable to the thumb, it is preferable to use this to ensure closure of the thumb. The BR is often transferable, because its motor branch from the radial nerve is more proximal than those of the extensor carpi radialis, and similarly its nerve's origin in the cervical cord is higher. If no local transfer is available, regional muscle transfers are possible using the biceps brachi or the latissimus dorsi.
5. When there is no active transferable muscle to the thumb, closing movement can be obtained by tenodesis, provided that the wrist has active extension. Active extension of the wrist restored by a transfer produces closure of the pinch; flexion due to gravity results in its opening *when the forearm is pronated*. BR transfer to the ECRB is often used on tetraplegic patients for restoration or reinforcement of wrist extension.
6. On which hinge should one base re-establishment of the lateral pinch? This can be either the trapeziometacarpal (TM) joint or the MP joint.[7] The basal joint appears anatomically more favorable. It was the first choice for Moberg's key grip operation[8] using the FPL tenodesed to the volar surface of the radius. Brand[9] modified Moberg's original procedure: the FPL is rerouted under the finger flexors in the palm, and then into Guyon's canal (Figure 19.9). Later, Allieu[10] modified the key grip procedure again by fixing the FPL to the EPL through the radius distal extremity to adjust the key pinch precisely (Figure 19.10).

Conservation of MP joint motion is used in Zancolli's EPL tenodesis.[7]

All of these procedures are described in Chapter 29 on Tetraplegia.

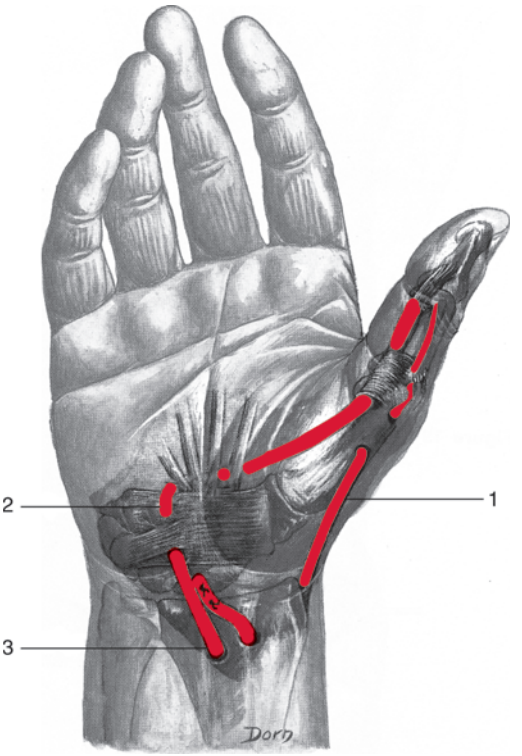

Figure 19.9

Extensive paralysis: re-establishment of lateral pinch in high-level tetraplegia. Brand's modification of Moberg's original procedure: the FPL tendon is rerouted under the finger flexors and then into Guyon's canal. (1) Tenodesis of thumb extensors. (2) The FPL tendon has a transverse trajectory and is then passed into Guyon's canal. (3) Tenodesis of the FPL, fixed on the radius. A wrist extensor is necessary for activation of these tenodeses.

Stabilization of the index finger

Transfer of the EIP to the tendon of the first dorsal interosseous was proposed by Bunnell.[11] In this procedure, the course of the EIP is changed to the radial aspect of the index. Because of the angle of approach, stabilization of the MP joint, but not abduction, is provided. Solonen and Bakalim[12] improved this technique by passing the EIP around the EPL, which acts like a dynamic pulley (Figure 19.11).

A *transfer of the EPB*[13] has a better angle of approach, but its transfer leaves a deficit of extension of the thumb MP joint. However, this transfer is recommended when the thumb MP joint is fused.

Use of the FDS was proposed by Graham and Riordan;[14] the tendon is divided proximal to the Camper's chiasma and is pulled out proximal to the carpal tunnel, it is passed radially and subcutaneously in the 'snuffbox' area, and then sutured to the first dorsal interosseous. It seems inappropriate

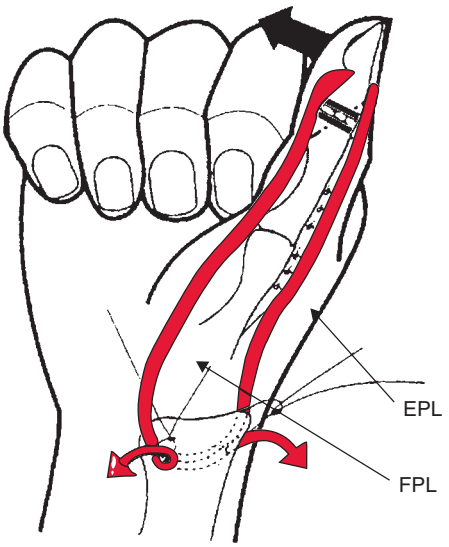

Figure 19.10

Allieu's modification of the key grip operation.

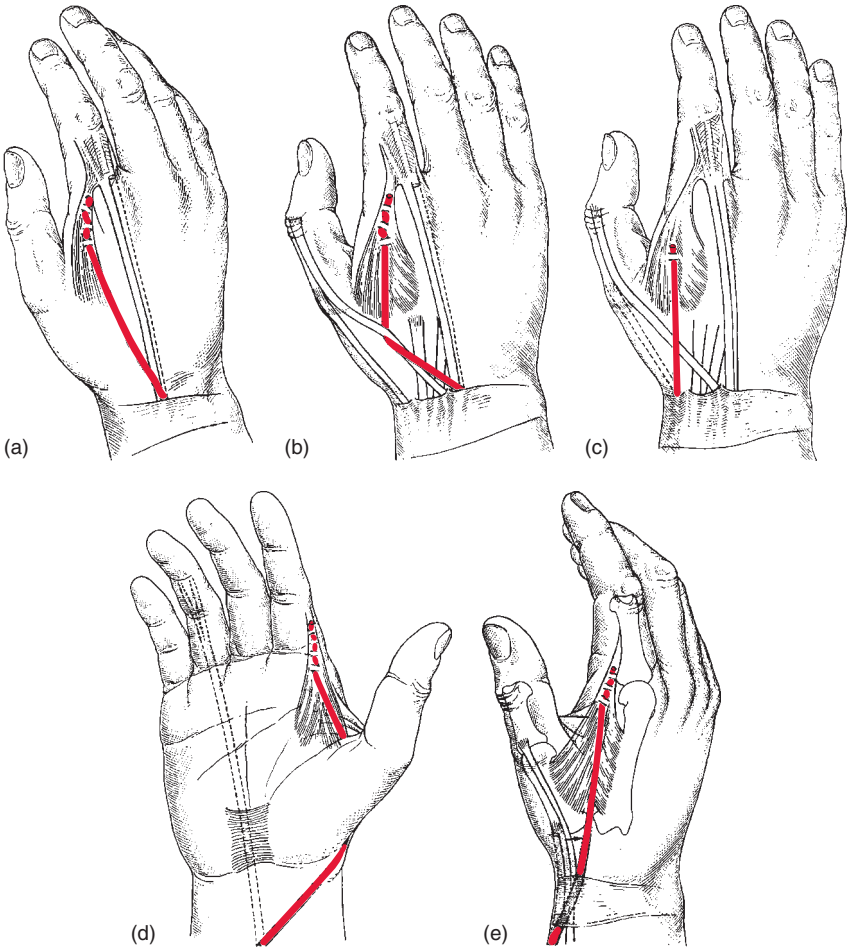

Figure 19.11

The different procedures used to re-establish abduction of the index finger: (a) Bunnell;[11] (b) Solonen and Bakalim;[12] (c) Bruner;[13] (d) Graham and Riordan;[14] (e) Neviaser et al.[15] (Reproduced with permission from Tubiana R. Paralyses of the intrinsic muscles of the hand. In: Tubiana R, ed. The Hand, Vol 4. Philadelphia: WB Saunders, 1993: 256–98.[3] Copyright Elsevier.)

Figure 19.12

The PL tendon prolonged by the palmar fascia and sutured to the first dorsal interosseous (Camitz).[18]

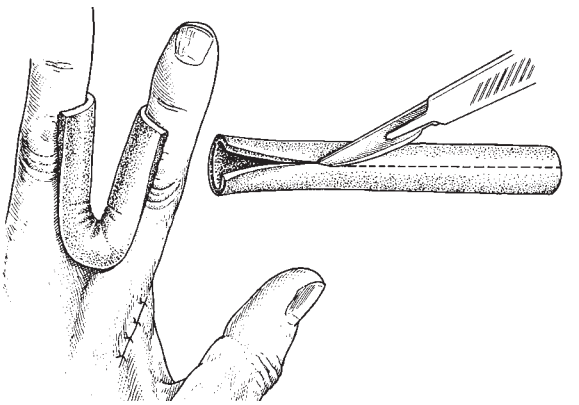

Figure 19.13

Maintenance of abduction of the index finger by a drain split longitudinally and placed in the second web space.

to use such a powerful and precious tendon for this function. In a more economical fashion, a strip of the ring finger FDS can be transferred across the palm and sutured to the first dorsal interosseous tendon, with the other strips being used for correction of the claw on other fingers.

Neviaser et al[15] described *abductor pollicis longus (APL)* transfer. Several tendons from the APL occur in over 96% of patients. The direction is favorable, but the excursion of the APL is short, and its tendon must be extended with a graft. The range of index finger abduction in nine consecutive patients was 58% (range 21–104%) of the unaffected side.[16]

Even an extensor digiti minimi transfer has been used.[17] We have used with satisfactory results the Hirayama

procedure.[18] The palmaris longus (PL) prolonged by the palmar fascia (as in Camitz's procedure),[19] is transferred around the radial aspect of the wrist and sutured to the first dorsal interosseous (Figure 19.12). Fixation is performed with the wrist in 10° of extension, and the index MP joint at 30° of flexion and 20° of radial deviation. The direction of the resultant transfer is parallel to the first dorsal interosseous, and its course palmar to the MP joint stabilizes this joint in flexion. A drain split longitudinally in the second web space provides a convenient splint during the postoperative period (Figure 19.13).

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20 Update and strategy in the treatment of adult brachial plexus injuries

Santos Palazzi, Juan P Cáceres, and José L Palazzi with the collaboration of José L Borrero

Introduction

For decision-making regarding treatment, the following should be noted:

1. There is fast and complete recovery with neuropraxia or axonotmesis with no sequela. No surgery is necessary: physical therapy and psychological support are needed.
2. When there is no recovery, surgery based on the following is necessary:
   - *Age*. There is no lower age limit for brachial plexus surgery. In general, surgery is not indicated in patients over 60 years in age, except those with partial paralysis.
   - *Time since injury*. In view of frequently found associated injuries and the possibility of spontaneous recovery, we prefer to perform surgery between the second and fourth months. By this time, improvement in the patient's general condition allowing serial examinations is noted. In view of muscle atrophy and irreversible nerve damage, results are quite poor when surgery is performed a year or longer after injury. Easier dissection in view of minimal fibrosis is the main reason for supporting early surgery (1 week). In our opinion, urgent nerve repair is only indicated when it is associated with a vascular insult that requires immediate surgery.
   - *Psychologic status*. The patient should be properly informed of the nature of his or her injury and complications, and must accept long-term therapy and treatment, which may last over 3 years.
3. Delay surgery when the proximal muscles show improvement and Tinel's sign progresses distally. Perform serial clinical and electromyographic examinations to follow nerve regeneration (usually 1 mm/day).
4. When there is out-of-sequence recovery, as seen when muscle reinnervation does not follow an expected proximal-to-distal chronology, we delay treatment for 8–10 weeks. After this time, if no clinical evidence of reinnervation is noted, the situation is ignored (if the muscle group is not functionally important) or the specific nerves that have not recovered are surgically explored. Careful, conservative exploration is mandatory in order not to injure nerves that have recovered. In some instances, we have found a dual lesion such as musculocutaneous nerve avulsion as it enters the muscle.
5. Recovery can also be dissociated – that is, motor and sensory recovery do not match. This may occur when the traction lesions involve an unknown number of axons or fascicles in different areas. In general, in these situations, it is best to accept spontaneous recovery and not to perform surgery, which could otherwise disturb the recovery obtained. Early secondary procedures may be indicated in these situations.

In our series (Table 20.1) supraclavicular injuries correspond to 65% of all adult traumatic injuries, which are mostly (85%) the result of traffic accidents. Surgical strategy and results are largely dependent on the type of injury suffered: preganglionic (avulsion), postganglionic (rupture), or dual lesions.[1,2] As an outline:

- Nerve grafts are used to repair postganglonic injuries.
- Neurotizations are used to repair preganglionic injuries.
- In order to further improve function, secondary surgeries will be needed over 60% of the time.

Up until the 1980s, brachial plexus surgery was mostly a technical problem. Today, it is a decision-making situation, in which the final outcome must be considered. Both the initial nerve surgery and the secondary palliative procedures must be considered equally important.

Table 20.1 *Epidemiology of traumatic adult brachial plexus lesions (n = 524) (follow-up from 1972 to 2001)*

Sex:			
Male	400 (84%)	Double level	36 (7%)
Female	84 (16%)	Traffic accident	448 (85%)
		• Motorcycle	304 (68%)
		• Car	72
		• Run over	38
		• Other	12
Age:			
Men	22 (range 5–56)	Work accident	46
Women	25 (range 8–43)	Sports	18
		Assault	8
		Others	4
Supraclavicular	341 (65%)		
• Partial	136 (40%)		
• Total	205 (60%)		
Infraclavicular	147 (28%)		
• Partial	103 (70%)		
• Total	44 (30%)		

Treatment: functional objectives

Restoration of elbow flexion becomes the primary motor functional objective in total brachial plexus paralysis. As a minimum, neurotizations aim to restore voluntary elbow flexion, which may indirectly benefit shoulder stability.

The second objective is to correct glenohumeral subluxation through repair or neurotization of at least the suprascapular nerve or though static orthopedic procedures such as tenodesis or arthrodesis.

In addition, functional objectives include wrist extension, finger flexion, and opposition as a minimum.

Also, a primary objective should be to obtain useful sensory return to the hand in at least the primary median nerve distribution.

Surgical exploration

Exploration of the adult plexus injuries calls for exposure of the entire plexus so as to determine injury level (which can be dual), as well as extent (Figure 20.1).

Surgical exploration always begins at the supraclavicular triangle with a zig-zag incision along the posterior (lateral) border of the sternocleidomastoid muscle. It then continues along the superior border of the clavicle and along the deltopectoral groove. In some situations, we perform a transverse cervical incision about 3 cm along the clavicle, which provides better cosmetic results.

In either case, after incising the deep cervical fascia, the omohyoid muscle is either retracted or divided, and the transverse cervical vessels are ligated and divided. The phrenic nerve is identified as it travels along the anterior scalene muscle. The C5 root corresponds to the level at which this nerve crosses the lateral border of the muscle.

Exploration of roots C8 and T1 may require partial removal of the scalene muscle or clavicular osteotomy.

The infraclavicular plexus is approached through the deltopectoral area, leaving the cephalic vein attached to the deltoid muscle. The pectoralis minor muscle is divided at its insertion and later reattached. If necessary, the pectoralis major muscle is reflected with a Z-plasty tenorraphy close to its insertion.

Repair according to findings (Figure 20.2)

Open postganglionic injuries

In general, these are nerve injuries caused by knife or iatrogenic incidents. If treated early, these are the only injuries that allow direct nerve repair. Nerve grafts will be used for late repairs. Restrepo[3] reported 217 cases in which 116 suffered a major vascular injury (carotid or subclavian vessels) and in which 28 suffered injury to the pleural apex. Vascular and nerve repair in the same setting, is always recommended.

In-continuity, closed, postganglionic injuries

If there is total loss of function, the intraoperative finding of an in-continuity lesion makes intraoperative decision-making quite difficult. Electrophysiologic techniques help in deciding whether or not to proceed with neuroma resection. Some authors consider these studies important. In view of technical difficulties, after a few trials, we have discontinued using intraoperative motor evoked potential recordings.

Functional recovery following neurolysis with or without epineurectomy may be obtained in axonotmetic lesions (Sunderland grades II–III).

There is always doubt, however, as to whether surgery was necessary, and spontaneous recovery might have ended up with the same result. Internal neurolysis is recommended for situations of non-traumatic pathology (tumors, plexitis, etc.).

Non-continuity, closed, postganglionic lesions

Neuroma resection and tension-free nerve grafting is mandatory in neurotmetic lesion (Sunderland grades IV–V).

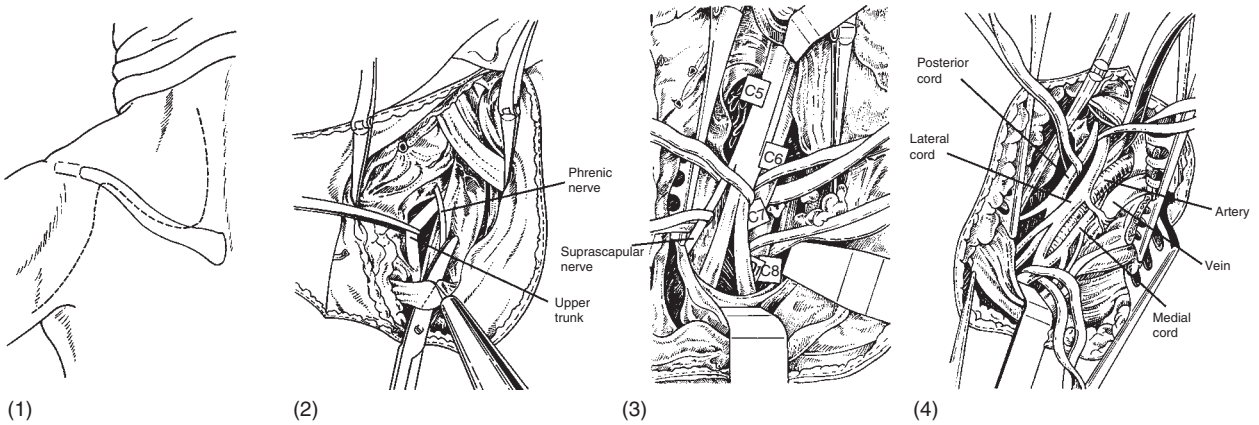

Figure 20.1

Surgical scheme. (1) Surgical exposure: incision. (2) Supraclavicular exposure: the phrenic nerve is brought to the 5th cervical nerve. (3) Supraclavicular exposure: upper, medial, and lower trunks. (4) Exposure of the infraclavicular brachial plexus: access to cords and axillary artery and vein.

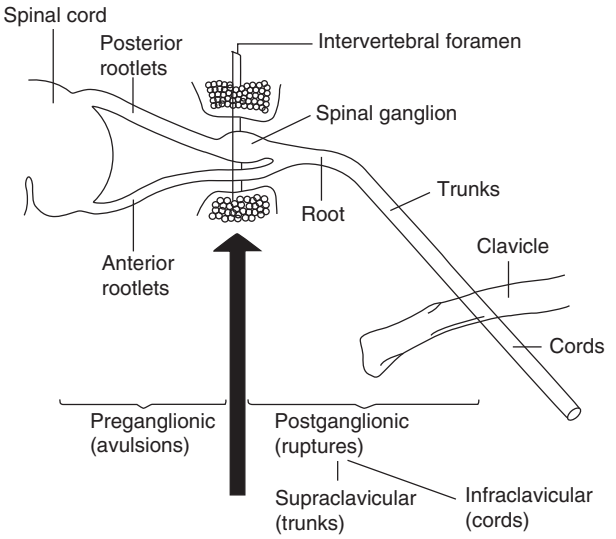

Figure 20.2

Diagram of pre- and postganglionic lesions.

Since there is tissue loss in all traction lesions, a nerve graft to bridge the gap between nerve ends is required.[4] The use of fibrin glue allows for easy, fast, and precise nerve repair.[5,6] After measuring the nerve gap to be bridged, nerve grafts are glued together at their respective proximal and distal ends. The grafts are cut to size, obtaining a fresh, sharp, transverse cut. Graft ends are then glued[7] to nerve ends. The coaptation is further secured with several 9-0 nylon sutures.

Nerve grafts are oriented in their respective quadrants.

Following the fascicular pattern described by Narakas[8] and Alnot's teachings, fascicles in the anterior portion of a root correspond to flexors and abductor muscles; the posterior portion corresponds to extensors and abductor muscles (Figure 20.3). Since revascularization is critical for graft survival, graft bed quality is more important than graft length. Grafts are driven following a quadrantic orientation.

Supraclavicular postganglionic injuries should be considered as peripheral nerve repairs at a more proximal level. In these cases, a proximal stump is always present, and only the graft beds and their length cause concern. Direct nerve reconstruction is mandatory.

Two major problems must be considered for supraclavicular root injuries:

1. *Proximal root stump quality*. Ruptures that display a well-defined fascicular pattern must be distinguished from those which display a thinned-out proximal stump indicative of a diffuse stretch injury, which carry a much worse prognosis.[9]
2. *The distal nerve elements needing repair*. Longer nerve grafts as close as possible to their motor target are preferred. Axonal dispersion may occur when grafts are placed more proximally.[10,11] Reinnervation, as complete as possible, of a single important muscle group is preferred over scanty reinnervation of several muscle groups.

Avulsions (preganglionic injuries) (Table 20.2)

At present, neurotization offers the only possibility of repair for root avulsion injuries. Neurotization is a nerve transfer in which a healthy nerve is, either totally or partially, detached from its intended target and directed to a receptor nerve controlling an affected area. This becomes a nerve-to-nerve transfer where the expected recovery has greater functional importance than the loss incurred by the donor nerve (Figure 20.4). In 1903, Harris and Low[12] implanted 'healthy neighbor nerves' into distal stumps of C5 and C6 in three patients.

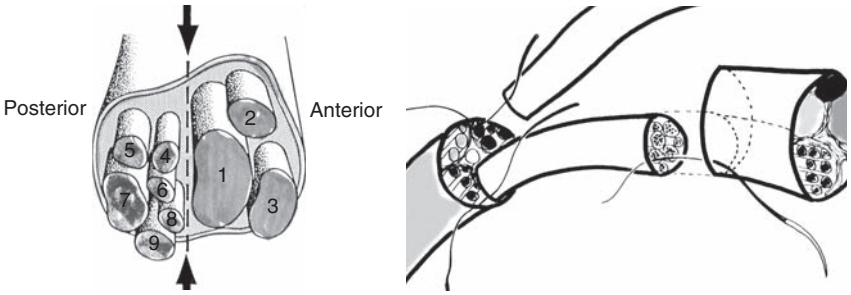

Figure 20.3

(a) Posterior fascicles of roots innervate abductor and extensor muscles. Anterior fascicles of roots innervate adductor and flexor muscles. (b) Quadrantic drive of the nerve grafts.

Table 20.2 *Topography of avulsions*

Individual	
C5: 1	
C6: 24	
C7: 34	
C8: 12	
D1: 4	
Combinations	
C5, C6: 30	C5, C6, C7: 34
C6, C7: 4	C7, C8, D1: 78
C7, C8: –	C6, C7, C8, D1: 78
D1: 38	
All	
27	

It was probably Seddon and Yeoman who, in 1963, provided the first description of technique and results after nerve transfers.[13] On four occasions, they coapted intercostal nerves to the musculocutaneous nerve.

Neurotization can be either intraplexural or extraplexural. The latter can be ipsilateral or contralateral, depending on the nerve being transferred.

(a) Intercostal nerves III, IV, V, and VI, which result in minor motor deficits (unless there is an associated phrenic nerve paralysis), are more commonly used. If there is phrenic nerve injury, particularly in babies, we do not use intercostal nerves for neurotization in view of concerns with possible respiratory insufficiency. The sensory distribution for intercostal nerve VI (breast area) must be taken into consideration, particularly in women.

At least three intercostals are needed to reinnervate the musculocutaneous nerve. Nerve connections can be performed either via grafts or directly without using grafts to the musculocutaneous nerve proper or to the biceps muscle branch to avoid axonal dispersion.

End-results at 2 years after musculocutaneous nerve neurotization with intercostals range between 70% and 50% of useful function (M3).

One must consider intercostal muscle paralysis in cases of Brown-Sequard syndrome.

Millesi's[1] idea of reinnervating the musculocutaneous nerve with intercostals III, IV, and V, and the motor branch to the triceps with intercostals VI and VII, should be considered. As a second stage, the triceps is transferred over the biceps to increase elbow flexion power.

Birch[13] suggests that one should always perform neurotization to the long thoracic nerve (Bell) to reinnervate the serratus muscle.

Muset[14] proposes neurotization of the thoracodorsal nerve to reinnervate the latissimus dorsi.

(b) Dividing the spinal accessory nerve distal to the motor branches for the upper trapezius is the most commonly performed transfer (and with the best results). Neurotization of the suprascapular nerve provides active shoulder stability.[8,15]

One must rule out supraspinatus tendon tear using sonography or magnetic resonance imaging (MRI). C5, C6, or C7 avulsion will have better results than complete lesions. Narakas[8] prefers this direct neurotization without grafts, even in cases where C5 or C6 are not avulsed. We follow this theory, believing that it prevents co-contractions. Direct suture is possible in 90% of cases.

Kotani and Allieu[16] utilize the spinal accessory for musculocutaneous neurotization. Results are good, in spite of inequality in myelin fiber count for the spinal accessory (±1800) and for the musculocutaneous (±6000) and the use of long grafts.

(c) In 1998, Narakas[17] suggested transferring the hypoglossus nerve (cranial nerve XII) to the plexus and to the musculocutaneous nerve in particular. Besides Sloof's series,[18] in 1993, for obstetric paralysis, the largest published series is that of Malessy,[19] with 12 patients. Of these, only 2 obtained active flexion ±M3. We have never utilized this method.

(d) Gu Yu Dong[20] utilizes the phrenic nerve to the musculocutaneous nerve.

(e) Gu Yu Dong[21] describes transferring the contralateral healthy side C7 root extending it with a trans-thoracic graft. Receptor nerves include the musculocutaneous, the median, or both. On three occasions, we have used this transfer with the following variations:

- We use the anterior fascicle of C7 after electrical stimulation and verifying motor response.

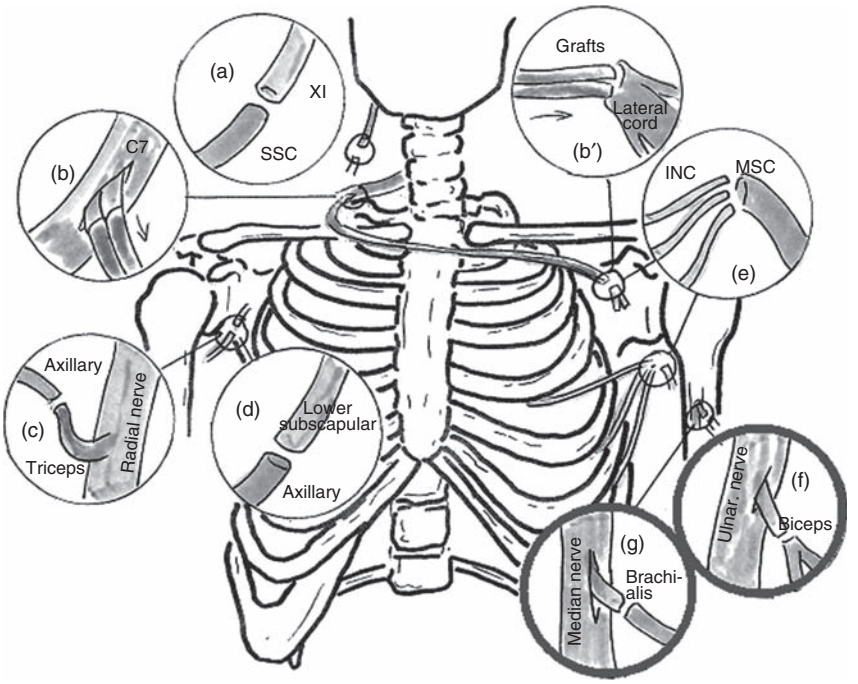

Figure 20.4

Scheme of proximal neurotizations: (a) XI → suprascapular (SSC);[8] (b, b′) contralateral C7 → lateral cord;[21] (c) nerve to long head of triceps → axillary;[24] (d) lower subscapular → axillary;[22] (e) intercostals (INC) → musculocutaneous (MSC);[13] (f) ulnar → biceps motor nerve;[27] (g) median → brachialis motor nerve.[27]

- We use two sural nerve trans-thoracic grafts.
- Later, as a second stage (8–10 months), neurotization of the musculocutaneous nerve and the branch to the median nerve is carried out. In two cases, elbow flexion (M3) and median nerve sensation have been obtained.

(f) Other transfers or neurotizations that have been described include:
- ipsilateral phrenic nerve
- medial or lateral contralateral nerve to the pectoralis major[11]
- motor nerves to pectoralis minor (considered excellent as a transfer to the axillary nerve, which can be done end-to-end or end-to-side)
- lower subscapular nerve (motor nerve from the teres major) to the axillary nerve[22]
- ipsilateral cervical plexus.[23]

(g) Nerve transfer using the nerve to the long head of the triceps to the anterior branches of the axillary nerve through the posterior approach is a reliable and effective procedure for deltoid reconstruction.[9,24]

(h) The best neurotization for elbow flexion, however, is that of transferring fascicles from the ulnar nerve to the biceps motor branch.[4,25] This is our preferred choice in cases of C5, C6, or C7 avulsion and functioning C8 and T1.

The ulnar nerve is dissected at the level of the middle third of the arm. An epineural incision is made, and fascicles corresponding to extrinsic muscles, usually in the anterior, lateral aspect are identified using a low-voltage nerve stimulator. Prior to this, the biceps motor branch is identified. This branch is found 8–14 cm distal to the inferior border of the pectoralis major. In 90% of cases, there is only one branch. A direct nerve-to-nerve coaptation is then performed, either over or under the short head of the biceps (see Figure 20.4).

In those rare cases in which, in addition to upper root avulsion, there is an ulnar nerve injury, we have used median nerve motor fascicles instead.

In a series of 41 patients followed over 7 years, we have used the ulnar nerve 34 times and the median nerve 4 times.[26] The results are summarized as:

	Good	*Useful*	*Poor*	*Nil*
C5, C6 avulsion	5	4	1	1
C5, C6, C7 avulsion	9	15	3	3

In search for better outcomes, Oberlin[27] proposed a double nerve transfer:
- fascicles for the ulnar nerve (FCU) ±3000 motor axons to the biceps motor nerve (3300±415)
- fascicles for the median nerve (PT) ±1800 motor axons to the brachialis motor nerve (2900±462).

The number of myelinated nerve fibers matches between donors and recipients. In our opinion, this double neurotization is indicated in cases of C7 avulsion, long preoperative delay, and poor strength of ulnar fascicles on intraoperative stimulation.

Disadvantages are a longer operation than single neurotization, a risk of complete anesthesia of thumb and index fingers, and a risk of interosseous anterior nerve lesions.

Dual lesions

We have seen distal avulsion (intramuscular) of the musculocutaneous muscle (a dual lesion), 11 times.

In all cases, fascicles from a sural nerve graft were implanted directly into the muscle and sealed with fibrin glue (direct muscular neurotization[28]).

We have been unsuccessful in four cases of axillary nerve avulsion. Useful shoulder stabilization after supraspinatus muscle reinnervation was obtained in five cases of distal suprascapular nerve avulsion.

Frequent lesions and repair outline

Partial injuries

Postganglionic C5, C6 with intact C7, C8, T1

For the shoulder, we need at least two motor muscles:

- Spinal accessory to suprascapular nerve.
- Nerve graft from C5 to superior trunk.

For the elbow:

- Direct repair with grafts C5, C6 to anterior and posterior divisions of the upper trunk.

Preganglionic C5, C6 intact C7, C8, T1

For the shoulder:

- Spinal accessory to suprascapular.
- Lower subscapular to axillary.
- Motor nerve of long head of triceps to anterior branch of axillary nerve (preferred).

For elbow flexion:

- Ulnar nerve fascicles to nerve to biceps.

Preganglionic C5, C6, C7; intact C8, T1

For the shoulder:

- Spinal accessory to suprascapular nerve.
- If triceps muscle has good strenghth, transfer of triceps long head motor nerve to anterior part of axillary nerve.
- If triceps muscle is weak, pectoralis minor motor nerve to axillary nerve.

For elbow flexion:

- Double Oberlin procedure.

For wrist and finger extension:

- Tendon transfer for extension of the wrist, metacarpophalangeal joints and thumb extension.

Table 20.3 *Results for C5, C6 and C5, C6, C7 supraclavicular lesions (n=64)*

	Useful	*Not useful*
Partial supraclavicular C5, C6 (*n*=30)		
C5, C6 post (12)	9	3
C5 post, C6 pre (10)	7	3
C5, C6 pre (8)	3	5
Shoulder	66%	34%
Elbow flexion	78%	22%
Partial supraclavicular C5, C6, C7 (*n*=34)		
C5, C6, C7 post (8)	6	2
C5, C6 post, C7 pre (8)	5	3
C5 post, C6, C7 pre (12)	6	6
All pre (6)	2	4
Shoulder	45%	55%
Elbow flexion	66%	34%
Wrist - hand	26%	74%

post, postganglionic; pre, preganglionic.

- Intercostal nerves III and IV to triceps nerve, to thoracodorsal nerve in order to reinnervate the latissimus dorsi.

A summary of results on repair after upper plexus injuries is given in Table 20.3.

Klumpke's paralysis (C8, T1) with C5, C6, ±C7 functioning

These injuries are infrequent and represent 5-8% of plexus injuries. They result in motor and sensory loss equivalent to a high median and ulnar nerve injury. Complete loss of hand function occurs if there is associated C7 avulsion. In the adult, repair does not produce functional recovery of intrinsic muscles, even postganglionic injuries. Still, we believe that repair should be carried out for it may provide protective sensation to the ulnar side of the hand and reinnervation of the proximal ulnar nerve.

Secondary surgery should be carried out early. Trapeziometacarpal arthrodesis, as well as tenodesis and transfers allowing partial automatic pinching similar to cases of tetraplegia, should be performed.

Different neurotizations with intercostal nerves, or from nerves from the contralateral plexus, yield poor or minimal end-results when attempting functional motor reinnervation of the hand. This is because:

- There is a large distance between the donor nerve and the sensory or motor target organs.
- The intercostals are semi-autonomic and lack the needed sophistication to produce complex motor function in the hand.

Neurotization of the anterior interosseous nerve (epitrochlear branch) of the median nerve with the brachialis muscle branch was proposed by Accioli[29] in 1999.

The brachialis muscle branch is served by roots C5, C6, C7. It is usually a single nerve, measuring 1.8–2.4 mm in diameter and ending up as two or three intramuscular branches. It contains 2090 ± 462 myelinated fibers (Figure 20.5).

In cases of C8 and T1 avulsion injury, sparing injury to C5, C6, Accioli's technique is direct suture between the muscle branch to the brachialis muscle and the epitrochlear branch of the median nerve (*Technique A*).

We have found, on occasion, a responsive epitrochlear branch, not needing a transfer. In these instances we proceed differently:[30,31]

- *Technique B*. We employ end-to-side nerve connection, using the brachialis muscle branch end and the lateral brachial cutaneous nerve end (the sensory component of the musculocutaneous nerve) into the side of the median nerve in an area where intraoperative nerve stimulation produces no response. The objective is to perform a more distal neurotization, expecting some reinnervation of wrist and finger flexors and at least protective sensation in the median nerve distribution of the hand.
- *Technique C*. Wrist and finger extension are the primary goals after C7, C8, T1 avulsions. For this, we perform neurotization of the posterior interosseous nerve, the branch of the radial nerve with the brachialis muscle branch. In these cases, a nerve graft between the brachialis muscle branch and the posterior interosseus nerve is needed.

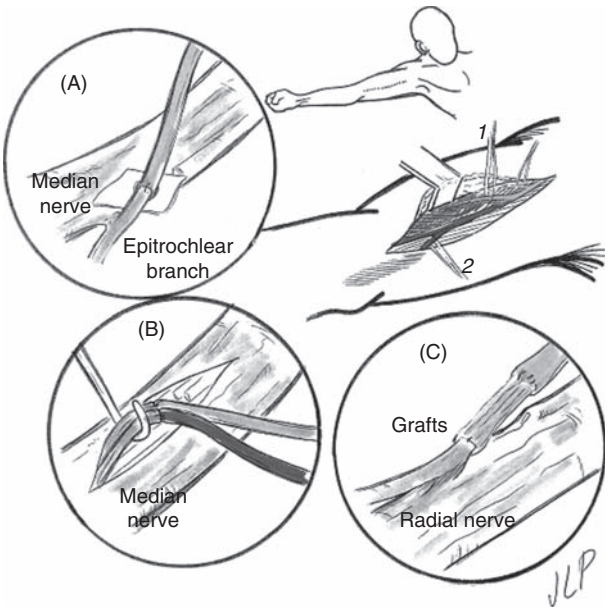

Figure 20.5

Scheme of surgical techniques of neurotization by the brachialis muscle motor nerve: (A) to the epitrochlear branch of the median nerve; (B) brachialis motor nerve and lateral cutaneous nerve neurorraphy to non-active fascicles of the median nerve (below the exit of the active epitrochlear branch; (C) nerve grafts from the brachialis motor nerve to the deep motor branch of the radial nerve (posterior interosseous nerve).

In conclusion, for avulsions of C8 and T1, ±C7, neurotization of the median or radial nerves with the muscle branch to the brachialis provides a distal connection aimed to reinnervate distal muscles, which in addition may serve for future tendon transfers. Adding sensory fibers from the lateral antebrachial cutaneous nerve may provide sensation in the median nerve distribution of the hand.

Total supraclavicular injuries

These are severe injuries, involving the entire upper extremity. They are usually associated with neurogenic pain, which becomes more difficult to treat when its onset is early.

An outline of treatment follows:

C5, C6, C7 postganglionic; C8, T1 preganglionic

- Spinal accessory to the suprascapular nerve for the shoulder; grafts from the three postganglionic roots to the lateral cord, posterior cord, and lower trunk.

C5, C6 postganglionic; C7, C8, T1 preganglionic

- Spinal accessory to the suprascapular nerve; grafts from the two postganglionic roots to the lateral cord and posterior cord.

C5 postganglionic; avulsion of C6, C7, C8, T1

Two situations may arise:

(a) If C5 has a good fascicular pattern and size:
- Spinal accessory to the suprascapular nerve.
- Grafts from C5 to the anterior fascicle of the lateral cord proximal to the pectoral branches.
- The intercostal will be saved for a future free vascularized muscle flap.

(b) If root C5 is doubtful:
- Spinal accessory to suprascapular grafts from C5 to the posterior cord, hoping to partially reinnervate the deltoid and triceps.
- Intercostals III, IV, V to the musculocutaneous, frequently adding intercostals II to a muscle branch of the pectoralis major.

If the spinal accessory or the intercostals are not available:

- Suspension tenoplasty of the shoulder, usually using the coracoacromial ligament.
- Neurotization using contralateral C7 posterior division to the anterior fascicles of the lateral cord for elbow flexion, wrist flexion, and some sensation of thumb, index, and medius.

Results are summarized in Table 20.4.

Table 20.4 *Results for total supraclavicular lesions*

C5 postganglionic. Rest preganglionic (*n*=31)	
• Shoulder subluxation:	
Improvement	20
Nil	11
• Elbow flexion:	
M3 or +	16
Less	15
• Sensibility of radial part of hand	5
• Pain:	
Improvement	24
No change	7
Avulsion of five roots (*n*=28)	
• Shoulder subluxation:	
Improvement	12
No change	16
• Elbow flexion:	
M3 or +	13
Less	15
• Pain:	
Improvement	19
No change	9

Discussion

There is no doubt that advances in anesthesia, microsurgical technique, and knowledge of pathophysiology have produced improvement in the treatment of these patients.

Whenever proximal stumps are suitable, direct repair is always preferred. Neurotization in cases of avulsion is part of the armamentation that permits offering some functional end-results.

Today, secondary operations cannot be separated from primary nerve surgery.

For total lesions, particularly in avulsions, the greatest problem is reinnervation and restoration of function to the wrist and hand. We should mention that, for this problem, Asian authors advocate, for finger flexion and extension, primary use of free muscle transfers (gracilis) connected to intercostals or the contralateral C7.[32]

For total avulsion cases, Brunelli, at the Narakas Club Meeting in 1999, proposed transfer of the entire healthy contralateral ulnar nerve to motorize the paralyzed arm.

Reimplantation of avulsed spinal nerve roots into the spinal cord is perhaps the most ambitious therapeutic modality.

Reimplantation of avulsed roots was first published by Bonney[33] in 1979. Two types of injury were pointed out. One was the true avulsion injury, in which spinal nerve rootless or even anterior horn cells are torn off the spinal cord. These are severe injuries, which are more frequent. The second type involves a somewhat more distal rootlet disruption, which allows the possibility of surgical repair.

In 1977, Jamieson and Birch reimplanted C7, C8 anterior rootlets using nerve grafts.[13] Anterior rootlet stumps in the spinal cord were noted on microscopic evaluation. After anterior root reimplantation, Carlstedt et al[34] experimentally demonstrated functional motor recovery in non-human primates. In 1995, they published their results of five cases after nine reimplantations in humans through a posterior approach, in which only the anterior roots were reimplanted using fibrin glue to secure one to three nerve grafts per root.[35]

In our opinion, these technological advances are positive steps toward improvement after brachial plexus injuries, which usually have a grave functional progress. For this condition, Bunnell's words apply well: 'when you have nothing a little bit is a lot'.

Neurologic pain

Neurogenic pain is described by the patient as burning, lancinating, flashing, or numbing. Its treatment is complicated and multidisciplinary, since it may include medical, psychological, or even neurosurgical involvement.

As an outline:

1. In view of the danger of addiction, the use of opiates in patients with chronic pain is not recommended.
2. In general, nerve medication is a combination of:
   - *Anti-epileptics*: clonazepam 0.5–12 mg/day or carbamazepine 100–800 mg/day. Lately, results seem better using gabapentin 300 mg three times per day or pregabaline.
   - *Antidepressants*: amitriptyline is initiated as 25–100 mg/day. Pain relief is believed to occur via serotonin and norepinephrine receptor blockade.
   - Central nervous system analgesics: tramadol 50–100 mg/day.
   - *Transcutaneous nerve stimulation* (TNS): this is performed by placing an electrode at the neck (C3, C4 dermatome) and another at the axilla (T2, T3), for 20 minutes, three or four times per day. The mode of action is believed to be via local inhibitory systems acting in the dorsal horn (α and β fibers). Acupuncture may yield a similar effect.
   - *Mental relaxation and pain control*: social and psychosomatic balance improves with this method.
   - In patients what are not candidates for surgery or in patients who have had unsuccessful surgery, a *cannabis-based* medicine might be of use.[36]
   - *Neurosurgical treatment for intractable neurogenic pain*: nerve stimulation through epidural electrodes in the cervical spine may help, particularly in postganglionic injuries. For preganglionic injuries, the technique of dorsal root entry zone ablation (DREZ), consisting of electrocoagulation of the anterolateral entrance of the posterior root in the posterior horn, described by Nashhold is used.[37]

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21 Primary repair of the obstetric plexus

Alain Gilbert

Introduction

The name obstetric palsy (la paralysie obstetricale) was given in 1872 by Duchenne de Boulogne in his book *De l'electrisation localisée*.[1] He recognized the traumatic origin of the lesion, although some controversy has existed until recently. Modern series in which operative exploration has been carried out have confirmed the pathology.

The etiology is a tearing force due to traction on the head or arm.

There are two basic types of lesion:

1. Overweight babies (>4 kg) with vertex presentation and shoulder dystocia who require excess force by traction, often with forceps or ventouse extraction for delivery. This results in upper plexus injury, most commonly to the C5 and C6 and occasionally the C7 roots, but never the lower roots (Figure 21.1).
2. Breech presentation, usually of small (<3 kg) babies, requiring excessive extension of the head and often manipulation of the hand and arm in a fashion that exerts traction on the upper roots as well as on the lower roots. This may cause rupture or avulsion of any or occasionally all of the roots (Figure 21.2).

Clinical presentation

The initial diagnosis is obvious at birth. After a difficult delivery of an obese baby by the vertex presentation or a small baby by the breech, the upper extremity is flail and dangling. A more detailed analysis of the paralysis pattern of the various muscles of the upper extremity is not necessary, as the picture will change rapidly. Examination of the other extremities is important to exclude neonatal quadriplegia or diplegia. Occasionally, birth palsy may be bilateral. Forty-eight hours later, a more accurate examination and muscle testing can be performed.

At this stage, it is usually possible to differentiate the two types of paresis:

1. *Erb–Duchenne-type paralysis of the upper roots*. The arm is held in internal rotation and pronation. There is no active abduction or elbow flexion. The elbow may be slightly flexed (lesion of C5–C7) or in complete extension (lesion of C5–C6). The thumb is in flexion, and sometimes the fingers will not extend. As a rule, the thumb flexor and the flexors of the fingers are functioning. The pectoralis major is usually active, giving an appearance of forward flexion of the shoulder. There are no vasomotor changes or gross impairment of distal sensation.
2. *Complete paralysis*. The entire arm is flail and the hand is clutched. Sensation is diminished and there is vasomotor impairment giving a pale or even 'marbled' appearance to the extremity. Often, Horner's sign is present on the affected side (Figure 21.3).

A shoulder X-ray should be taken to eliminate fracture of the clavicle or the upper humerus, which can occur in

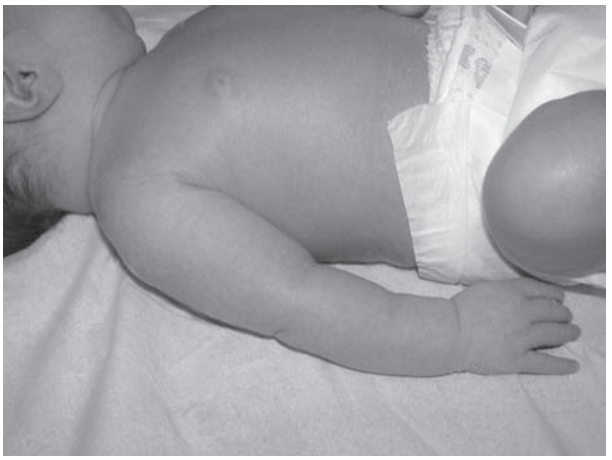


Figure 21.1
Typical C5 C6 paralysis.

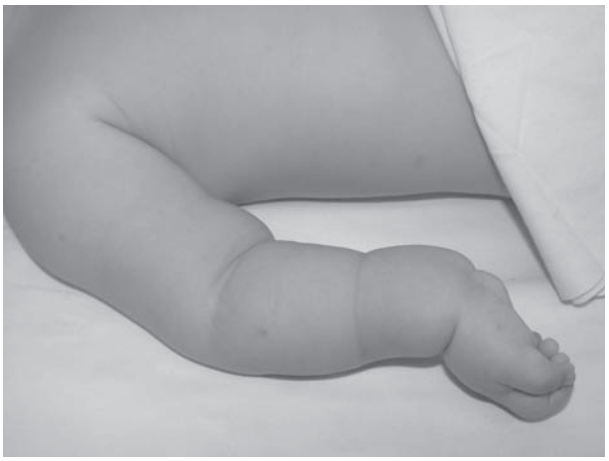


Figure 21.2
Complete paralysis.

Figure 21.3
Horner's sign.

association with the paresis. Occasionally, a phrenic nerve palsy can be detected by fluoroscopy.

The clinical development during the first month is variable, and many pareses will recover during this stage.[2] However, Wickstrom et al[3] reported that only 10% of total palsies recover to any useful extent. These patients should be carefully evaluated at the age of 3 months clinically and by electromyography (EMG) and cervical myelography. Gentle physiotherapy should be used during this recovery period to minimize the development of contractures while awaiting spontaneous recovery. At this stage, complete paralysis with Horner's sign will remain unchanged, and early operation should be considered in these babies at 3 months.

Paralysis of the upper roots may show spontaneous recovery during the first 3 months. These babies should be treated with physiotherapy and assessed clinically and by EMG by the age of 3 months.

Spontaneous recovery

The literature reports varying rates of spontaneous recovery from 7% to 80%. Useful guidelines are given in the thesis of Tassin,[4] who came to the following conclusions:

1. Complete recovery is seen in those infants showing some contraction of the biceps and the deltoid by the end of the first month and a normal contraction by the second month.
2. No infant in whom neither the deltoid nor the biceps contract by the third month can be expected to obtain a good result. Testing the deltoid can be difficult. As a result, assessment of the biceps is the most reliable indicator for operative intervention. If there is no evidence of any recovery in the biceps by the end of the third month, operation is indicated. Clinical assessment is more reliable than electrical testing. If surgery is not undertaken, some recovery will continue to take place spontaneously, but it is likely to be less satisfactory than that following surgery.

Indications for operation

If recovery of the biceps has not begun by 3 months, the prognosis is poor and surgical repair of the plexus is indicated. The following clinical situations pose particular problems:

1. Complete palsy with a flail arm after 1 month, particularly with Horner's syndrome, will not recover spontaneously and is a prime candidate for surgery. These babies are best treated by early operation at the age of 12 weeks. Absence of recovery of the hand is the most important factor, even with recovery of the shoulder or elbow.
2. Cases of complete palsy of C5 and C6 occurring after breech delivery with no sign of recovery by the third month have to be explored. In these cases, avulsion injuries of the upper roots are common.
3. The commonest C5, C6, and sometimes C7 palsies almost always show some sign of recovery, which can be misleading, and which has in the past encouraged a conservative approach. If, however, after careful examination, the biceps is completely absent at 3 months, surgery should be considered. It is important to look for biceps recovery but not elbow flexion, which can be given by the brachioradialis muscle. Great difficulty arises when infants are seen late, i.e. towards the sixth to eighth months, and show minimal recovery of biceps function. The parents are often encouraged by the beginning of recovery, and will not accept the idea of an unsatisfactory final result. Under these conditions, it is difficult for the surgeon to advise surgery that cannot promise a definitive result. In order to avoid this situation, it is important to try to make decisions by the third month.

When the operation has been decided, the patient should be explored in depth in order to minimize any operative or anesthetic risks and to predict the lesions as precisely as possible.

Preoperative exploration

EMG is done at this stage. Although it rarely gives precise indications on the extent of the lesion and the quality of recovery, it may be very useful in predicting avulsion injuries. The association with evoked potentials will give even more precision. The avulsion of an isolated root in the central or lower part of the plexus is not clearly shown by EMG. Association with Horner's syndrome will be almost pathognomonic[5] of T1 avulsion.

However, in upper root avulsions, especially after breech delivery, the EMG is very clear, showing a total absence of muscle reinnervation. This absence, in conjunction with the obstetric record, will almost certainly confirm the avulsion at exploration.

Some authors[6] have developed methods of exploration that could give a better precision in the prognosis of obstetric lesions.

Conventional radiology is necessary in order to assess the possibility of diaphragmatic paralysis, which can sometimes occur at birth concurrently with the plexus lesion. It is important to know the status both medically and legally before the operation.

Myelogram and CT myelography

For many years, we used to take myelograms of our patients.[7] The results were not entirely satisfactory, with a large number of false positives and false negatives. The advent of computed tomographic (CT) myelography has changed the situation and gives much precise information. However, the indications for CT myelography are rare, as there is little information that we cannot obtain by direct observation during operation. Furthermore, this examination needs general anesthesia in a neonate.

It can be useful mostly in upper root lesions suspected of avulsion. In these cases, the roots are often in place and the operative diagnosis is difficult. The myelogram may be very useful in these cases (Figure 21.4).

Magnetic resonance imaging

MRI has been used extensively for brachial plexus injuries. Although it may give excellent results in adults, we feel that it is unreliable in children (Figure 21.5). With improvements in surface antennas, this will probably change, but at the moment the necessity for general anesthesia and the lack of information lead us to prefer, when necessary, the CT myelogram.

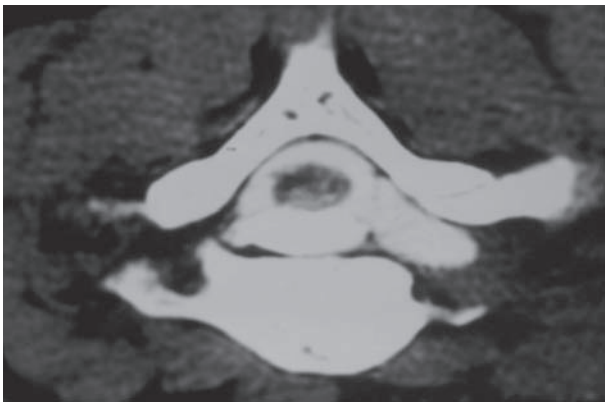


Figure 21.4
Myelogram showing a large meningocele.

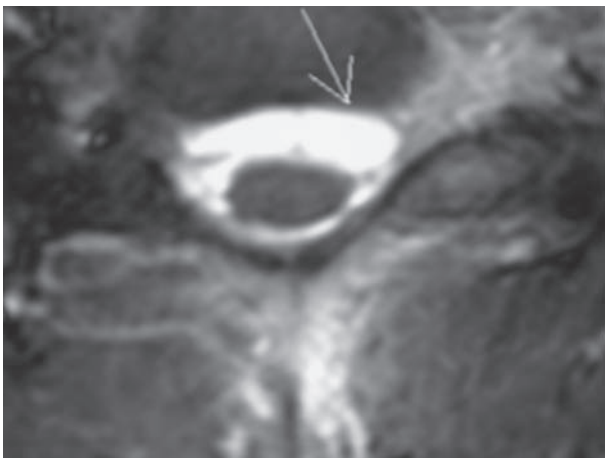


Figure 21.5
The MRI is more difficult to interpret.

Operative procedure

The child is in a supine position, with a small towel rolled and placed under the superior spine and scapula, in order to allow a large opening of the neck and thoracobrachial area. The head is turned completely to the opposite side, with the neck in slight hyperextension. The neck and upper thorax are prepped, as well as the entire upper extremity and both legs and knees. No tourniquet is used.

For several years, we used preoperative evoked potentials, but we have now stopped doing this, as the results were quite unpredictable and the variations due to anesthesia or temperature too large.

The incision will vary according to the type of injury. For C5 C6 or C5 C6 C7 lesions, we use only a supraclavicular triangular flap based on the posterior border of the sternocleidomastoid (SCM) muscle and the superior aspect of the clavicle.

In complete palsy, the incision extends distally over the deltopectoral groove.

The skin is infiltrated with a 1/1000 solution of epinephrine. This usually raises the heart rate by 15 or 20 beats/min. In order to obtain a good efficiency and a bloodless field, it is best to wait for the rhythm to return to its pre-injection level.

Approach

After supraclavicular incision, the skin and subcutaneous fat are lifted and held with a stay suture. If the SCM muscle insertion on the clavicle extends laterally, it may be necessary to desinsert its lateral part.

The plexus is covered with a thick layer of fat and multiple ganglia. This layer is lifted from its medial position, over the jugular vein, and reflected laterally. The field is then stabilized with a self-retaining retractor.

To approach the plexus, it is necessary to cut the omohyoid muscle and divide the transverse cervical vessels.

The plexus is then visible, and the first move is to separate and protect the phrenic nerve. In following it upwards, C5 is found automatically, as the nerve has a connection with the root in almost every case.

The anterior scalene is retracted gently, and the roots can be seen and dissected free. As they are followed distally, the neuroma is obvious, hard and surrounded with scar, often reaching the superior part of the clavicle.

The suprascapular nerve is systematically dissected free, along with the long thoracic nerve and its branches, when the neuroma is lifted.

Assessment of the extent of the lesion, the quality of the roots, and the length of the defect can then be done.

When the infraclavicular approach is needed, the second flap is lifted and sutured to the skin of the thorax. The pectoralis major is desinserted from the inferior part of the clavicle and retracted. The retractor will also take the pectoralis minor, demonstrating the upper, middle, and lower trunks and their branches.

A large periosteal flap is designed on the clavicle with a lateral pedicle. It is elevated and the posterior periosteum is elevated. The clavicle is then cut obliquely, using an electric saw. Using a 12G-K wire, holes are drilled into the two opposite pieces of bone. Once they are elevated, the posterior periosteum is sectioned laterally, making another flap. It is necessary at this stage to also cut the subclavian muscle. The two parts of the clavicle are held with a self-retaining retractor, and the whole plexus appears.

Every trunk is dissected and isolated. Care is exercised not to injure the subclavian artery and vein. The lower trunk has to be followed up to its division from C8 and T1, and the two nerves are dissected up to the foramen. This may be dangerous due to the proximity of the vessels. It is, however, absolutely necessary, and no decision can be taken without appraisal of the roots at the level of the foramen.

Intraoperative stimulation may be of help, especially when there is a suspicion of avulsion (Horner's syndrome, EMG) and the root is found in the foramen. In these cases, if there is no response to direct stimulation, the root will be considered as avulsed, cut at the foraminal level, and grafted.

At the beginning of our experience, we left several of these roots in place, with the hope of some recovery if there was no visible proof of avulsion. We always regretted it, and these patients went on to poor results in the hand.

The situation is different in the upper roots, after breech delivery, as we shall see later.

Repair of the lesions

Neuromas are excised largely and systematically. There is no point in doing neurolysis if the clinical situation has led to a surgical exploration. Neurolysis does not provide improvement,[8] and there are very few indications left.

When the roots are avulsed, the ganglion is often found with its small motor branch. This part should not be sacrificed, as sometimes only one or two roots are available for grafting. Directing the grafts on the small motor root will allow grafting of the whole plexus, which otherwise could not be repaired due to the discrepancy between the donor roots and the large volume of the trunks to graft.

These repairs done directly on the motor component have given some very satisfactory results.

Harvesting the nerve grafts

In babies, we use only the sural nerve and on rare occasions the superficial cervical plexus.

We feel that the use of trunk grafts such as the vascularized ulnar nerve should never be done, as this could sacrifice the chances of recovery in the hand.

The sural nerve is harvested by posterior zigzag incisions, after epinephrine injection. The nerve is too fine to be taken by separate incisions, and its anatomy[9] precludes the use of a stripper.

Some authors have proposed that the sural nerve be harvested endoscopically. Capek et al[10] applied the technique to pediatric cases. This is appealing, as the scars of this area are usually visible. The drawbacks are the necessity to start the operation in a prone position and then turn the patient over, and mostly the length of the procedure, which in some cases took several hours.

Preparing the nerve grafts

The sural nerve is, in most cases, Y-shaped,[9] and it is important to keep as much length as possible.

The defect between the two ends is measured at the plexus level, and the grafts (usually both legs) are divided on the table, performing a cable. We feel that discussion

about the direction of the grafts is not important: if one chooses to use the grafts in an anterograde direction, there will be a loss of axons through the multiple branching; if they are used in a retrograde manner, the distal part will have many empty conduits. There has never been any proof that one way or the other is better. The grafts are cut and placed on the table for their final organization. The end of each cable bundle is then glued to make a trunk, and this trunk is sharply cut transversally. Then, in a bloodless field, the graft is placed at once between the plexus extremities and simply glued in place.

We started gluing the nerves almost 20 years ago, immediately after Narakas' work. We have found the use of fibrin glue to be safe, and it has given better results than sutures. We use only glue without any sutures. It is also used with end-to-end and end-to-side anastomoses.

End-to-side anastomoses

Several authors have shown the feasibility and effectiveness of end-to-side anastomoses in the experimental arena. Very few reports have yet shown results in the clinical arena.

There are cases where there is no simple solution to repair the plexus:

- Isolated avulsion of one or two roots may occur, especially with breech delivery. The alternative may be a complicated extraplexual neurotization.
- In repair of extensive lesions, there may be a lack of donor area (rupture of a small C5 avulsion of C6 C7 with intact C8 T1). A relatively less important root (C7) may be sutured to C8.
- In complete upper roots avulsion, neurotization of the musculocutaneous nerve by the ulnar trunk[11] may be done in an end-to-side manner instead of sacrificing a bundle from the ulnar nerve.

The recipient root is dissected and the perineurium is split longitudinally. No dissection is done inside the nerve. The end of the avulsed nerve is placed into the opening and simply glued. It should hold without tension, and we do not use sutures even in this situation.

If it is proven that the results of end-to-side anastomosis equal those of neurotizations, its simplicity will make it a valuable tool.

Neurotizations

A large number of extraplexual neurotizations have been described in the literature – mainly for adults. We have used many of them in obstetric palsy, but the development of multiple intraplexual repairs and end-to-side anastomoses has reduced their indications. We are reluctant to use the intercostal nerves, especially in complete paralysis, since we studied ventilatory capacity in these children and showed that the extensive use of intercostals would decrease this capacity severely.[12] This has been confirmed by Gu.[13]

The occipital nerve is very small and too far from the plexus itself.

We still use the terminal part of the spinal accessory nerve. It can be found in the wound, and its size allows repair or grafting of a suprascapular nerve or a musculocutaneous nerve. It should be taken after its division, in order not to affect the trapezius muscle.

We have often used the medial nerve of the pectoralis major. This is easy to find, is of good size, and can be sutured end-to-end with the musculocutaneous nerve. It is very useful in cases of upper root avulsion to use a branch of the ulnar nerve to reinnervate the musculocutaneous nerve. However, our results show that it is better to use it in pure C5 C6 lesions, but that it is often too weak when C7 is injured. The results were not so good in these cases.

The contralateral C7 root has been used by some surgeons[13–17] and applied to obstetric paralysis by others.[18,19] The medicolegal situation, as well as the length of grafts necessary, has prevented us from using it. The possible advent of end-to-side anastomoses will reduce the number of indications for this procedure.

The lesions

Many types of lesions are encountered in obstetric plexus paralysis (Figure 21.6).

The most common is the neuroma in continuity. This is mostly found in the upper plexus; the level is usually at the junction of C5 and C6, but it can sometimes extend proximally, often distally to the clavicular area.

Faced with this lesion, there has been a tendency by several authors[8] to preserve continuity and perform only a neurolysis. Some have been able to assess their patients and demonstrate the inefficiency of the procedure.[8] We have always considered that when the clinical decision to repair the plexus has been taken, the neuroma must be removed and grafted. At the beginning of our experience,[12] a small series of 15 of these neuromas were excised and sent to pathology for serial studies. They showed an almost complete absence of fibers at the distal end of the neuroma. There are almost no indications for isolated neurolysis in this surgery. The intraoperative discovery that a neurolysis may be the appropriate treatment generally comes from excess in the indication.

Rarely, there is a complete rupture of the roots or trunks. The extremities may be difficult to find among a scarred area.

Avulsion injury is the most severe lesion. It may be suspected during the approach if there is an uncommon amount of scar, with difficult dissection of the roots. The spinal ganglion and the motor branch are usually found. It is important to preserve them, as it may be useful to repair the motor root directly.

No diagnosis of avulsion should be accepted until the root has been dissected up to the foramen. A number of

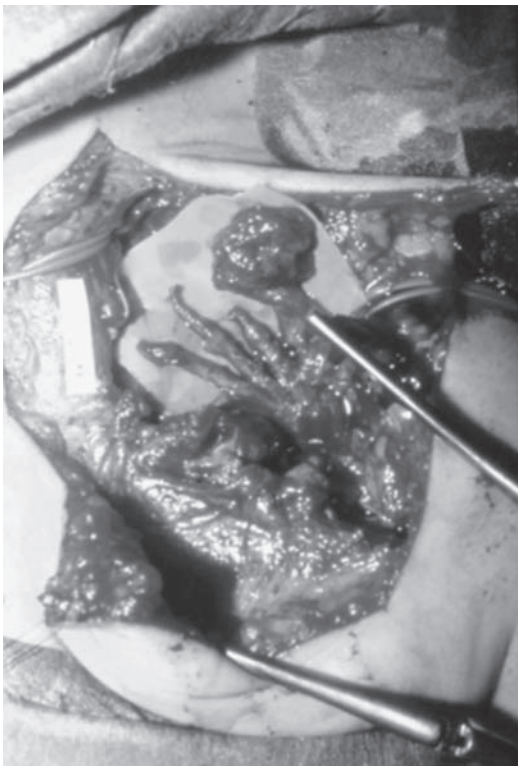


Figure 21.6

A complete lesion with rupture of two upper roots and avulsion of the others.

authors have determined the diagnosis of avulsion on clinical grounds, associated with EMG and myelography. These examinations are very helpful, but only dissection can confirm the diagnosis.

This exploration of the lower roots C8 T1 can only be done safely with osteotomy of the clavicle.

There are difficult cases where the root is inside the foramen, with all signs of avulsion. This situation is found, in particular, in upper root lesions after breech delivery. It is then necessary to make a decision. In upper root lesions, we know from experience[20,21] that approximately 50% of these roots will recover – at least partially. We prefer to close the wound and wait 6 months. After this delay, those patients who have not recovered will be reoperated and these roots, considered as avulsed, will be neurotized. In lower roots, decision has to be taken immediately, as reoperation may become dangerous in this area. If there is an associated Horner's syndrome and no response to electrical stimulation, the root is considered as avulsed, and cut at the foramen, to be neurotized.

Strategy of repair

When there are a sufficient number of donor roots, repair by grafts does not pose a problem.

Problems arise when there are no donor roots (isolated avulsion) or there are only one or two roots with three or four avulsed roots. We then have to mix grafts, neurotizations, and end-to-side sutures.

The technical aspects may vary with the lesion, its extent, and the length of grafts, among other things. The philosophy stays the same: we have priorities:

- The first priority is hand function, and especially finger flexion and thumb movements. These movements are almost impossible to recover through secondary transfers.
- Wrist and finger extension are also essential if the hand is to be usable.
- The next most important movement is elbow flexion, which can be used only with shoulder external rotation
- Elbow extension and shoulder abduction will come after.

The choice of distal grafting will be determined by these priorities. According to the number of usable roots, the repair can be more or less ambitious. In using this strategy, one of the main questions is the function after use of a single root to try to repair several movements, sometimes antagonists.

Our experience in very severe lesions has shown that in many cases, the child is able to independently control the movements he or she will recover, even from the same root.

In some instances, we have found co-contractions between biceps and triceps, which are difficult to treat (see below).

In terms of reliability, at this stage, we will use the grafts for the key movements: some neurotizations (spinal accessory, ulnar nerve, and pectoralis major nerve) for the biceps, and end-to-side for the secondary aims or if nothing else is possible. The results in 436 cases operated on up until 1996 showed the following distribution of root lesions:

1. C5 and C6: 48%
2. C5 to C7: 29%
3. Complete involvement: 23%, almost all of which were avulsions.

Postoperative care

Stretching the reconstructed area must be avoided in the first 3 weeks postoperatively. This can be achieved by a plaster cast. Physical therapy is then resumed by gentle passive exercises and encouraging voluntary movement. Every effort should be made to counteract retraction and internal rotation of the shoulder and flexion of the elbow. Physiotherapy should be continued throughout the recovery period (usually for 2 years), but regular physiotherapy should then be discontinued. The recovery is slow. It can be seen 4–6 months after direct suture and at 6–10 months following graft reconstruction. It can continue in upper plexus lesions for more than 2 years and in complete lesions for more than 3 years.

Results

Since 1977, more than 3600 patients have been seen and 842 operated for plexus repair.

Between 1976 and 1995, 436 patients were operated on and have been reviewed, with more than 4 years' follow-up.[22–24] The results in the shoulder using the Mallet scale[25] showed the following:

C5 C6 (Figure 21.7)

- At 2 years:
  > grade IV (good–excellent): 52%
  grade III: 40%
  grade II: 8%
- After 2 years, one-third of patients had secondary surgery:
  13 subscapularis releases
  33 latissimus dorsi transfers
  6 trapezius transfers
- A new evaluation was done at 4 years (after tendon transfers); with the following results:
  grade IV: 80%
  grade III: 20%
  grade II: 0%

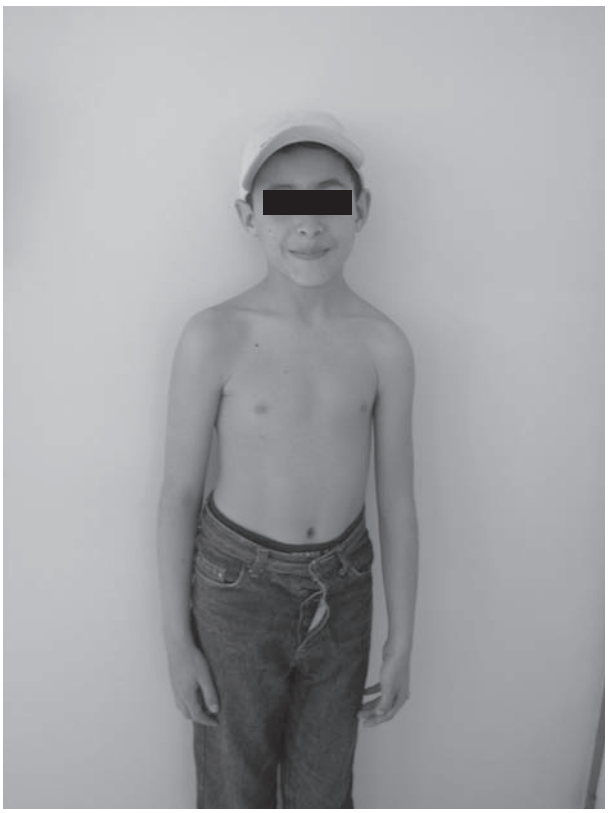
C5 C6 C7

- At 2 years:
  grade IV: 36%
  grade III: 46%
  grade II: 18%
- After 2 years, one-fourth of the patients had secondary surgery:
  7 subscapularis releases
  24 latissimus dorsi transfers
  1 trapezius transfer
- Results were evaluated again at 4 years:
  grade IV: 61%
  grade III: 29%
  grade II: 10%

Complete paralysis

The shoulder results in complete paralysis are less satisfactory, because parts of the upper roots destined for the shoulder and elbow have to be sacrificed in order to obtain function in the hand. The shoulder results at 4 years are as follows:

grade IV: 22.5%
grade III: 42%
grade II: 35.5%.



(a)

(b)

Figure 21.7(a, b)
Results 10 years after C5 C6 repair.

Alternatively, the prognosis for the hand, which is very poor following spontaneous recovery alone, provides 83% of hands with some function and useful function in 75% of patients 8 years after a neurotization.

Complications

There have been no operative deaths. The overall complication rate in the present series was 1%, including phrenic nerve lesions, lesions of the thoracic duct, wound infections, and vascular lesions, all of which have been managed satisfactorily without late sequelae.

Conclusions

Based on the results of the author's series, the following recommendations can be made:

1. Babies who do not recover biceps function by the age of 3 months should be considered for immediate operation.
2. Primary suture without tension is rarely possible. Nerve grafting is usually necessary for root or trunk ruptures.
3. In the presence of root avulsions, an internal neurotization should be attempted between different roots, particularly as children seem to have a far greater capacity to accommodate to differential neurotizations.
4. When it is not possible to perform an internal neurotization, an external neurotization can be performed using one or more of the following donor nerves (in order of preference): the pectoral nerves, the intercostal nerves, or the accessory nerve.
5. The reconstruction should be protected from excessive motion for the first 3 weeks.
6. Physiotherapy should be continued up to 2 years of age, but then continued by the parents in the form of play and activities of daily living.
7. Secondary surgery can be considered when it is clear that recovery following reconstruction is no longer progressing.

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22 Sequelae of the shoulder in obstetric palsy

Alain Gilbert

Summary

After spontaneous recovery or surgical repair of the obstetric brachial plexus, the sequelae at the shoulder level may be disabling, but can be treated surgically. Lack of passive medial rotation should be rapidly treated by subcapularis release in order to avoid secondary joint incongruence. The paralytic shoulder can be improved by latissimus dorsi transfer or trapezius transfer. These operations give constant improvement, but the results are better in young patients. Lack of appropriate rehabilitation and disuse will lead to decreased mobility, a recurrence of the medial rotation contracture, and joint anomalies, with retroversion of the glenoid and posterior subluxation of the humeral head.

Early sequelae

After an obstetric lesion of the brachial plexus, spontaneous recovery or repair of the plexus will allow generally good function of the shoulder. However, in both cases, there are patients in whom the muscle recovery will be late or incomplete. Usually the lateral rotators come back very late and, in contrast, the medial rotators are not paralyzed and recover quickly. There is an imbalance between the muscular groups. The internal rotators tend to be hyperactive and can become contracted. The medial rotation contracture is a common feature. It should be limited by physiotherapy, but sometimes the imbalance is so strong that the joint becomes fixed in internal rotation.

This internal rotation contracture may occur very early at age 6–8 months, but more often at 16–20 months. This position can become deleterious after some time and provoke some anomalies of the joint. The first anomaly will be the flattening of the humeral head and posterior subluxation of this head. After several years, the head is deformed, posteriorly dislocated, and the glenoid is small, flattened and retroverted. At this stage very little can be done. This will happen in 8–10 years. It seems that up to approximately 3 years, the deformity of the joint is limited, allowing a useful release. It is mandatory to treat this contracture rapidly when it occurs. After no more than 3 months of unsatisfactory physical treatment, surgery is the only solution.

In the young child, the main cause of internal rotation is contracture of the subscapularis. Joint contracture is not a primary cause, and the bone anomalies (coracoid) occur later.

In the past, several techniques have been described:

- Sever proposed section of the subscapularis tendon, associated with capsular resection.[1]
- Saloff-Coste[2] preferred to lengthen the tendon.

These techniques are very effective but their main drawback is the loss of internal rotation. Carlioz and Brahimi[3] proposed the release of the subscapularis muscle from the scapula. This operation preserves the function of the muscle.

Technique of subscapularis release

This operation is only possible if the joint remains congruent and the humeral head is round. It is necessary to verify this anatomy preoperatively; the arthroscan and magnetic resonance imaging are the most precise methods.

The infant is positioned on its back with the shoulder elevated by means of a large cushion; the shoulder should be free. The incision is made along the axillary border of the scapula with the arm in abduction (Figure 22.1). The anterior border of latissimus dorsi is identified and retracted posteriorly (Figure 22.2). The angle of the scapula then sticks out and is positioned by a strong suture, which is used for traction (Figure 22.3). The deep surfaces of the blade of the scapula and the subscapularis are seen. The angular part of the muscle attachment is divided (Figure 22.4). The teres major, which is quite close, should be respected.

The subscapularis is then released progressively, but keeping it extraperiosteal. The fibrous bands are sectioned

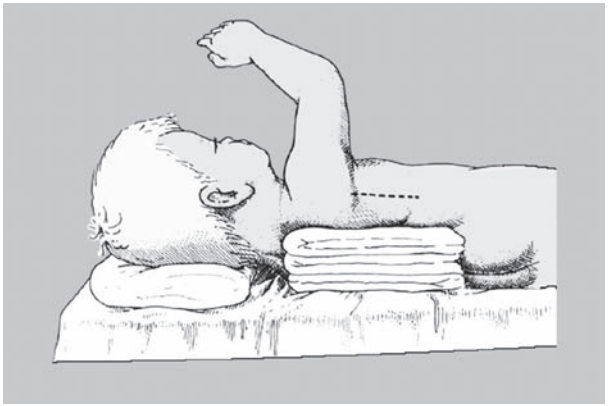

Figure 22.1
Positioning for subscapularis release.

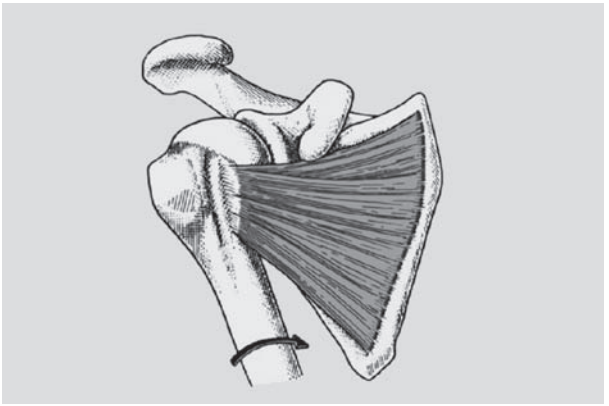

Figure 22.4
Desinsertion of the muscle at its border.

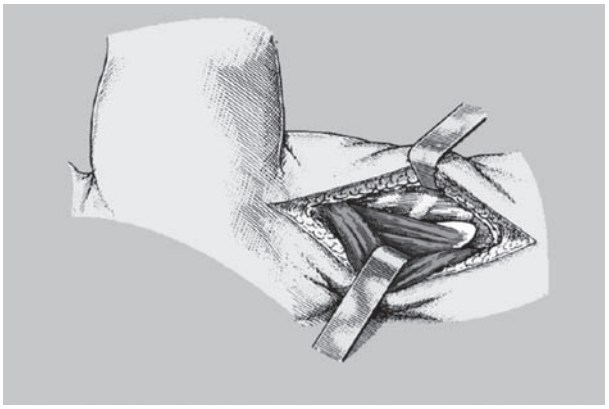

Figure 22.2
The latissimus dorsi is retracted.

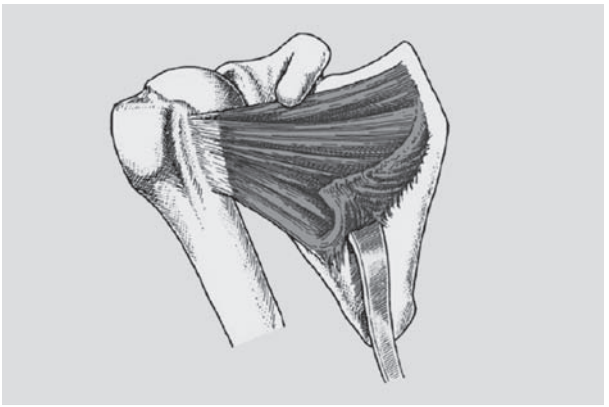

Figure 22.5
The extraperiosteal release is complete.

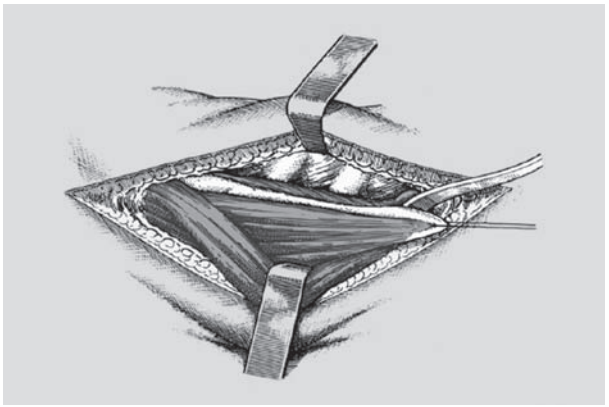

Figure 22.3
Placement of a traction suture in the scapula.

one by one (Figure 22.5). The release should be complete, particularly at the superomedial angle of the scapula. (Desinsertion that progresses too far medially and may injure the serratus anterior or the superior border of the pedicle of the subscapularis should be avoided.) The result is an immediate gain of motion at the end of the disinsertion, often surpassing 60 to 70°. It is never necessary to open the joint capsule (Figure 22.6).

A drain is left in place for 48 hours. The infant is placed in a cast, with the elbow at the side, in a position of maximal external rotation for 3 weeks (Figure 22.7). After removal of the plaster, intensive physiotherapy is started. It is crucial to maintain the good external rotation obtained by surgery. Only if the external rotator muscles become active will the passive rotation be maintained. If, after 5 or 6 months, there is still no active lateral rotation and progressive loss of passive rotation, a tendon transfer should be effected. If it is not done in time, the contracture will recur.

Usually about 50% of patients recover active rotation after simple release.[4] Some of the remainder should have operations for muscle transfer, but only 20% accept it or are followed closely enough. When the patient lives far away, or if the family does not seem to be compliant, it is better to effect a latissimus dorsii transfer at the same time as the release.

For the others, the transfer will be done after a minimum of 6 months of physiotherapy, on a supple joint (Figures 22.8–22.11).

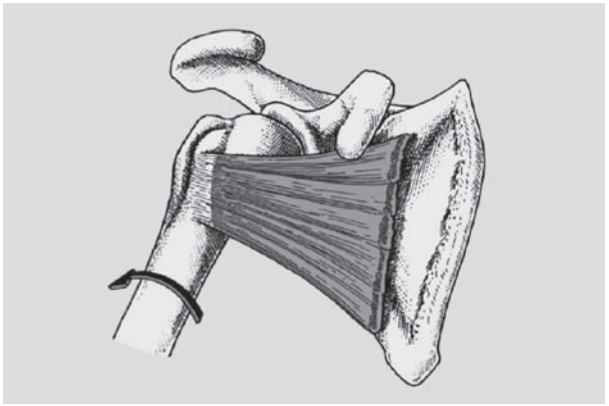

Figure 22.6
At the end of the desinsertion, external rotation is free.

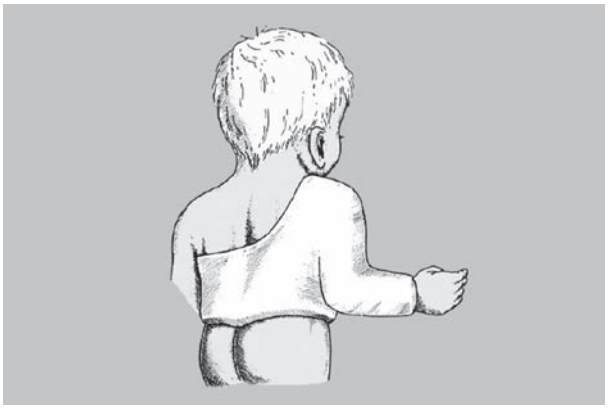

Figure 22.7
Cast immobilization for 3 weeks.

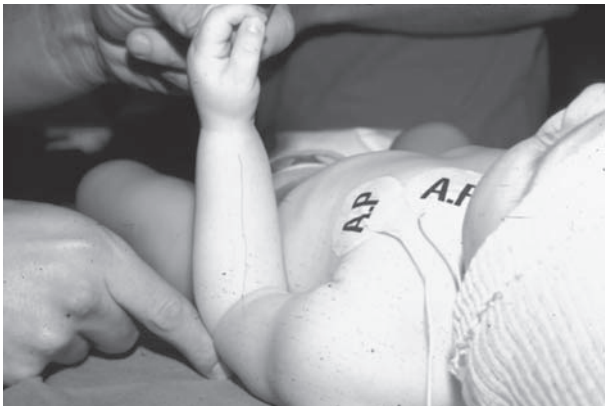

Figure 22.8
Preoperative position.

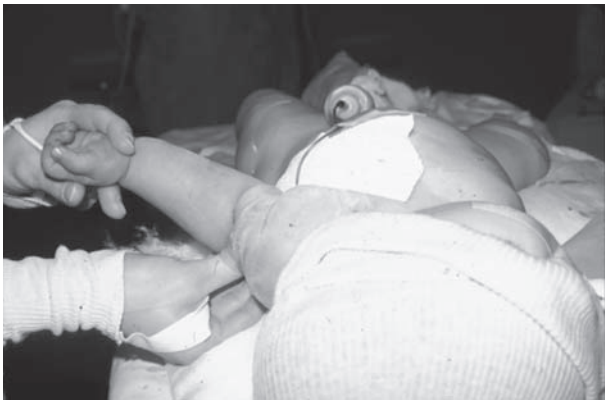

Figure 22.9
Immediately postrelease.

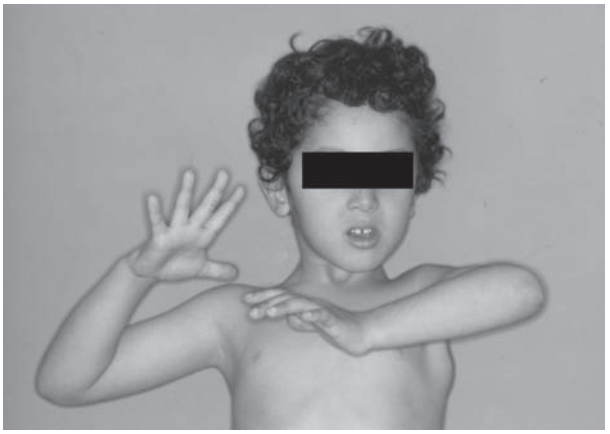

Figure 22.10
A preoperative 'trumpet sign'.

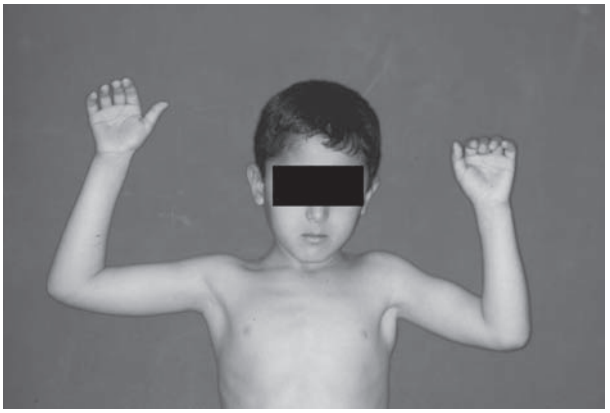

Figure 22.11
Postoperative result.

Technique of latissimus dorsi transfer (Figures 22.12–22.16)

The patient is positioned on the opposite side, with the affected arm and the shoulder prepped and draped free. An incision is made vertical and posterior to the shoulder, and following the lateral border of the scapula. The latissimus dorsi muscle is freed from the teres major and the scapula, and its tendon of insertion on the humerus is isolated. The tendon is divided at its insertion to the humerus, and the muscle is freed, taking care to preserve its neurovascular

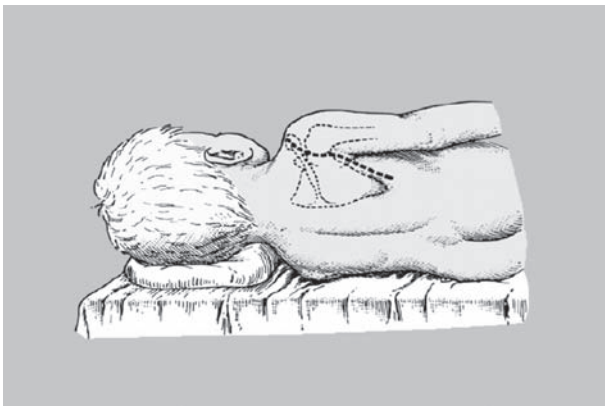

Figure 22.12
Positioning and incision for latissimus dorsi transfer.

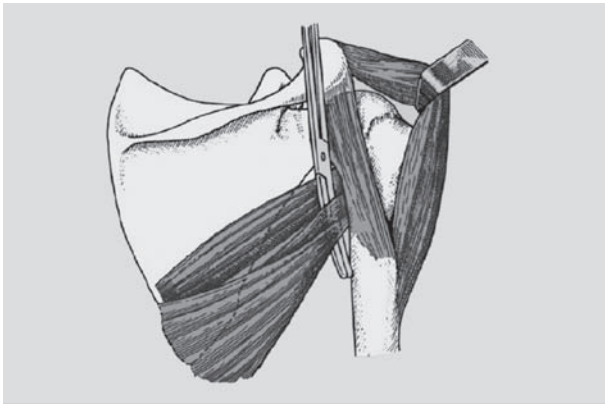

Figure 22.13
Isolation of the latissimus dorsi tendon.

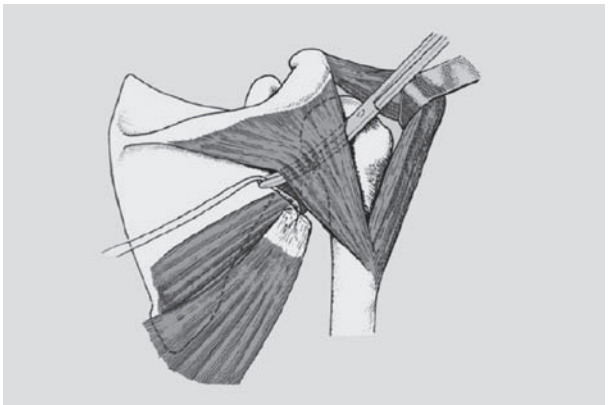

Figure 22.14
Passing under the posterior deltoid.

pedicle. A transdeltoid incision between the middle and posterior parts of the deltoid provides access to the humeral head and the insertion of the external rotators. The latissimus dorsi tendon is then passed under the posterior deltoid and fixed either to the rotator cuff or directly to the

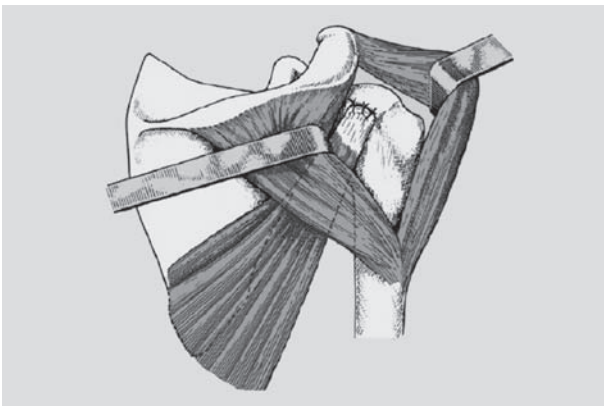

Figure 22.15
Fixation to the humeral head.

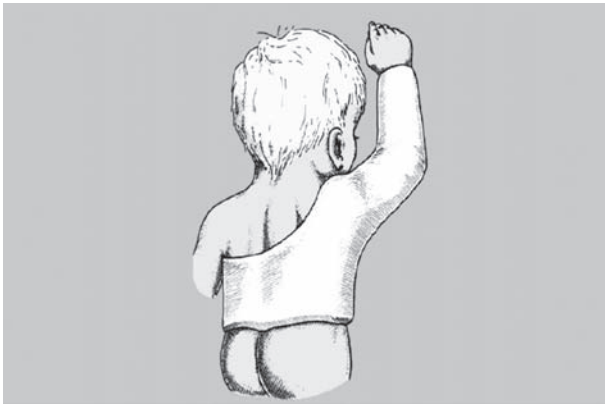

Figure 22.16
Abduction casting for 6 weeks.

humeral head in the young child. A stout, non-resorbable suture is used to fix the transfer while holding the shoulder in 120° of abduction and external rotation. The infant is immobilized in this position in a cast for 6 weeks.

Between 1982 and 1999, 203 children (110 boys and 93 girls) with birth-related brachial plexus palsies had latissimus dorsi transfer by the same surgeon (AG). Indications were age older than 2 years, less than 90° abduction and/or lack of active external rotation, a radiographically limited dysplasic glenoid (normal or flattened, without shoulder dislocation), and supple passive shoulder range of motion (ROM). Of the children, 89 had C5-C6 palsy, 72 had C5-C6-C7 palsy, and 42 had complete brachial plexus palsy. Microsurgical repair of the brachial plexus had been done in 57% of patients with C5-C6 palsy (n = 51; mean age, 4.4 ± 1.8 months), in 86.3% of patients with C5-C6-C7 palsy (n = 63; mean age, 4.6 ± 2.5 months), and in 68.3% of patients with complete palsy (n = 28; mean age, 5.5 ± 5.4 months). Subscapularis release was done at a mean age of 2.5 ± 2.1 years in 20.2% of children (n = 41).

At the time of latissimus dorsi transfer, the mean age of patients with C5-C6 palsy was 4.1 ± 2.7 years, and the mean age was 3.7 ± 1.8 years for patients with C5-C6-C7 palsy

and 3.9 ± 1.7 years for patients with complete palsy. The mean follow-ups were 5.5 ± 3.8 years for patients with C5–C6 palsy, 5.5 ± 3.5 years for patients with C5–C6–C7 palsy, and 6.2 ± 3.6 years for patients with complete palsy (range 1–15 years). Clinical evaluations were done preoperatively at 1, 3, 6, 10, and 15 years. Shoulder abduction (0–180°), passive external rotation with the elbow close to the body (0–90°), and active external rotation (0 = no active external rotation, 1 = active external rotation <30°, 2 = active external rotation >30°) were assessed.

Patients were divided preoperatively into three groups based on Gilbert's classification:[4] group 0–1 (26 children), group 2–3 (121 children), and group 4–5 (56 children). Finally, patients were assigned to three groups based on age at surgery: younger than 3 years (n = 80), 3–7 years (n = 109), and older than 8 years (n = 14). Data were analyzed using one-way ANOVA, significance was set at $p < 0.05$, and data were expressed as mean ± standard deviation (Figures 22.17–22.19).

C5–C6 palsy

Children with C5–C6 palsy achieved the best results in terms of abduction and active external rotation, in particular those in group 2–3. When mean preoperative abduction (105.34° ± 40.6°) was compared with mean postoperative values at 1 (131.14° ± 37.6°), 3 (136.16° ± 33.2°), and 6 years (129.19° ± 40.2°), the increase was significant ($p < 0.001$). However, values decreased (to 119.17° ± 39.5° and 105° ± 46.5°) at 10 and 15 years, respectively, with a loss of statistical difference from the preoperative values ($p > 0.05$). The difference between preoperative (56.96° ± 21.8°) and postoperative (65.36° ± 17°) passive external rotation was significant only at 1 year ($p < 0.01$). Differences at 3, 6, 10, and 15 years were not significant ($p > 0.05$). The improvement in active external rotation was significant at all times

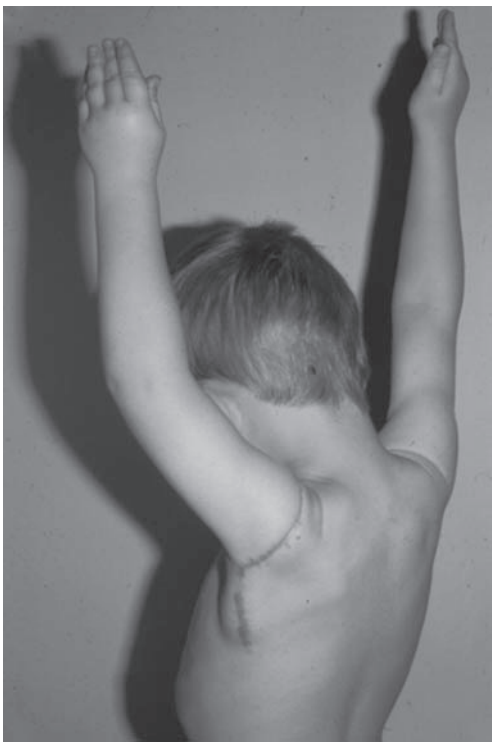

Figure 22.17
Result after 3 months.

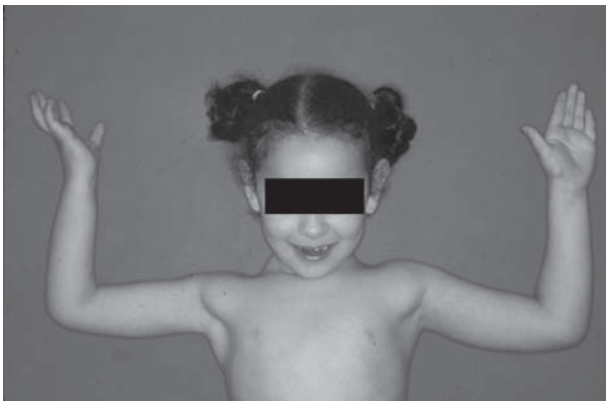

Figure 22.18
Six months postoperatively: external rotation.

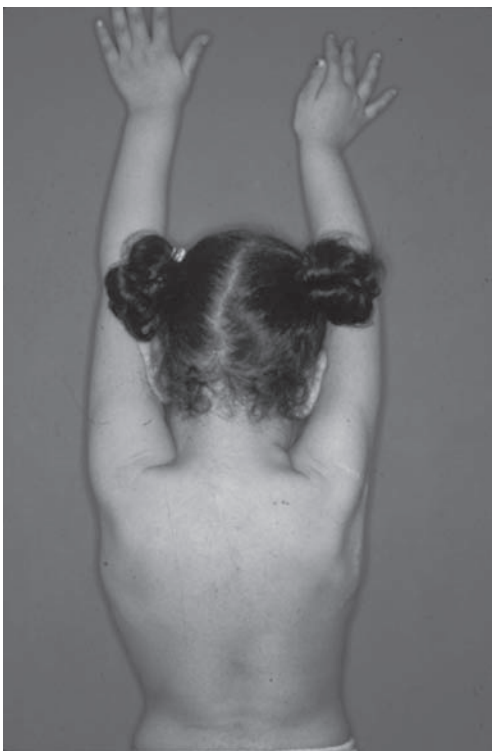

Figure 22.19
Six months postoperatively: abduction.

($p < 0.001$), increasing from 0.4 ± 0.3 preoperatively to 1.5 ± 0.5 at 1 year, and slightly decreasing at 15 years (1.4 ± 0.6).

The three preoperative groups based on Gilbert's classification were composed of 8, 45 and 36 children, respectively. Patients in group 0–1 had poor preoperative shoulder function. They experienced significant improvement in shoulder abduction until 10 years, and deterioration thereafter (Figure 22.2). The increase in passive external rotation was never significant ($p > 0.05$). By contrast, active external rotation improved significantly ($p < 0.001$) and remained significantly greater at all follow-up visits (Figure 22.3).

Children in group 2–3 had mild preoperative shoulder dysfunction and showed continuous and significant improvement ($p < 0.001$) in abduction at 1, 3, 6, and 10 years, with a peak at 15 years (Figure 22.2). The increase in passive external rotation was never significant ($p > 0.05$). By contrast, active external rotation improved significantly ($p < 0.001$) until 15 years (Figure 22.3). Patients in group 4–5, who had good preoperative shoulder function, experienced a small improvement in abduction (Figure 22.2) and a similarly small improvement in passive external rotation. In contrast, active external rotation lost significance at 10 and 15 years (Figure 22.3).

C5–C6–C7 palsy

Children with C5–C6–C7 palsy exhibited modest gains in abduction and significant gains in active external rotation in the long term. The mean preoperative abduction was lower (82.5° ± 31.9°) in patients with C5–C6–C7 palsy than in patients with C5–C6 palsy (105.34° ± 40.6°). The postoperative increase was significant ($p < 0.001$) at 1 (104.63° ± 37.3°), 3 (103.94° ± 37.1°), 6 (100° ± 34.2°), and 10 years (98.4° ± 34.1°), but not ($p > 0.05$) at 15 years (75° ± 21.2°). The improvement in passive external rotation was significant only at 1, 3, and 6 years ($p < 0.05$). The improvement in active external rotation was significant at all times ($p < 0.001$), increasing from 0.5 ± 0.3 preoperatively to 1.5 ± 0.6 at 1 year, and remaining high at 6 (1.6 ± 0.5), 10 (1.6 ± 0.6), and 15 years (1.4 ± 0.6).

According to Gilbert's classification, there were 10 children in group 0–1, 46 in group 2–3, and 16 in group 4–5.

No significant improvement was experienced by patients in group 0–1 in terms of abduction (Figure 22.6) or passive external rotation (both $p < 0.05$). By contrast, active external rotation improved significantly ($p < 0.05$) (Figure 22.7). In children in group 2–3, who had mild preoperative impairment, abduction improved significantly until 10 years, but deteriorated at 15 years (Figure 22.6). A significant increase ($p < 0.05$) in passive external rotation was recorded at 1, 3, and 6 years, whereas active external rotation improved significantly until 10 years, but declined at 15 years (Figure 22.7). Children in group 4–5, who had good preoperative shoulder function, experienced little improvement in abduction (Figure 22.6) and in passive external rotation. In contrast, active external rotation rose significantly at all follow-ups (Figure 22.7).

Complete brachial plexus palsy

Children with complete palsy experienced long-term improvement only in active external rotation. The preoperative data of children with complete palsy were consistently worse than those of the other two groups. The improvement in abduction was significant only at 1 and 3 years (99.26° ± 33.1° and 98.52° ± 30°, respectively; both $p < 0.005$), whereas a progressive decline was observed at 6 (82.86° ± 18°; $p > 0.05$), 10 (68.46° ± 28.1°; $p > 0.05$), and 15 years (10° ± 14.1°). The improvement in passive external rotation was significant ($p < 0.05$) only at 1 and 3 years. The increase in active external rotation was significant at 1, 3, 6, and 10 years ($p < 0.001$ or $p < 0.005$), increasing from mean preoperative values of 0.2 ± 0.3 to 1.2 ± 0.5 at 1 and 3 years, to 1.1 ± 0.8 at 6 years, and to 0.9 ± 0.6 at 10 years. The difference was no longer significant at 15 years (1 ± 1.4).

There were 8, 30, and 4 children in Gilbert's three classes of shoulder function, respectively. Patients in group 0–1 had a non-significant improvement in abduction (Figure 22.8) and in passive external rotation at all times (both $p > 0.05$). Active external rotation deteriorated at 3, 6, and 10 years. Patients in group 2–3 experienced an abduction improvement at 1, 3, and 6 years, and a consistent decline of values at 10 and 15 years (Figure 22.8). The increase in passive external rotation was never significant ($p > 0.05$), whereas active external rotation improved at 1, 3, 6, and 10 years, but declined and lost significance at 15 years (Figure 22.9). Patients in group 4–5 had a poor and non-significant improvement in abduction at 1, 3, and 6 years before being lost to follow-up (Figure 22.8). There was no improvement in passive external rotation, whereas active external rotation increased significantly ($p < 0.05$) until 6 years (Figure 22.9).

Age at surgery

There were no differences in the results of children of various ages. When analyzing the results based on the age at surgery, children with sequelae of C5–C6 palsy had improvements in abduction (Figure 22.10) and external rotation at 6, 10, and 15 years that seemed to be greater in younger patients. No significant differences ($p > 0.05$) were observed among age groups. In C5–C6–C7 palsies, the three groups based on age at surgery were composed of 31 children younger than 3 years, 38 children 3 to 7 years, and three children older than 8 years. There were no significant differences in abduction and external rotation improvements among these age groups. In cases of complete paralysis, there were no significant differences in abduction and external rotation among the patients younger than 3 years ($n = 8$), patients 3 to 7 years ($n = 31$), and patients older than 8 years ($n = 3$).

In severe cases, where the latissimus dorsi is very weak, the trapezius may be used. Its action will be more of abduction and it will provide less external rotation, but, in most cases, by carefully choosing its insertion it will be possible to correct some of the internal rotation deformity.

Technique of trapezius transfer (Figures 22.20–22.22)

The patient is installed in a lateral position so that the arm is free. A V–Y incision is drawn on the shoulder, the anterior incision is made on the clavicle, the posterior on the spine of the scapula. The skin is incised but not lifted from the muscle. The trapezius is desinserted from its bony insertions on the clavicle and the scapula. The musculocutaneous flap is lifted as a whole. The flap is limited by the deep vascular pedicle, where dissection should stop.

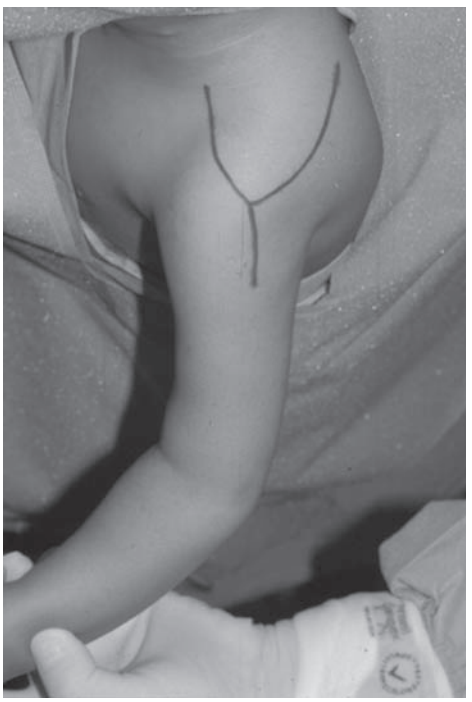

Figure 22.20
The incision for trapezius transfer.

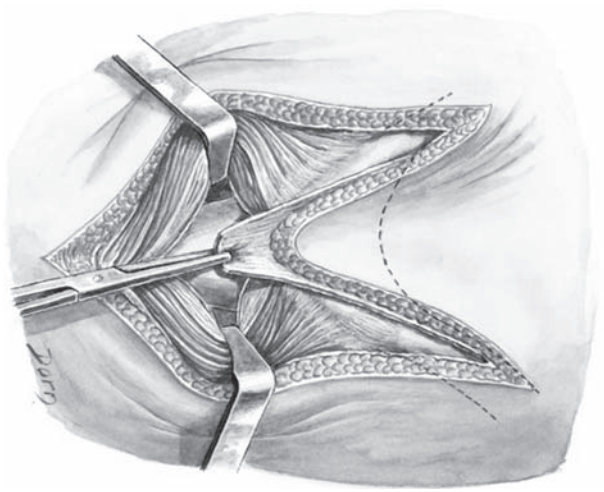

Figure 22.21
The muscle is lifted with the skin.

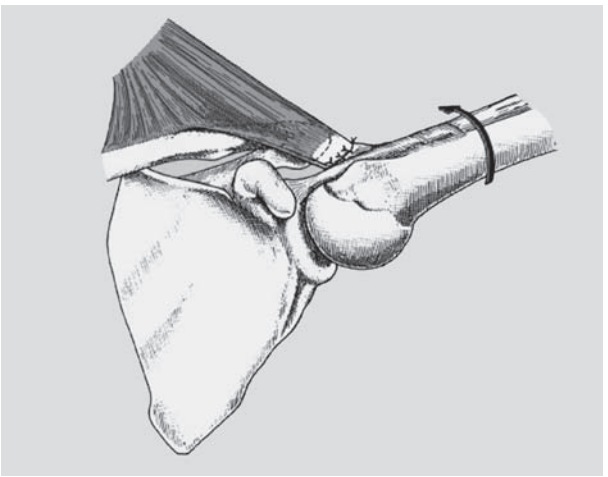

Figure 22.22
The tendon fixed on the anterior lip of the bicipital groove.

Distally, the tendon is lifted with the periosteum of the acromion. A T-shaped incision is made on the deltoid. The muscle is usually denervated. The two flaps are desinserted from the acromion. The humeral head is held in rotation and the bicipital groove becomes lateral. The trapezius tendon will be fixed on the anterior lip of the bicipital groove. The arm is held in abduction and external rotation. Fixation is made through the bone, at best with a bone anchor. The two deltoid flaps are closed over the trapezius tendon. The skin can be closed in a V fashion. The arm is immobilized for 6 weeks in a cast in abduction and external rotation.

This transfer has been described numerous times with different techniques of insertion. In the young infant it is possible to fix the transfer directly to the humerus without elongating it. The following important points should be respected:

- The splint is left for 6 weeks and is then opened, allowing physiotherapy in the splint for another 2 weeks.
- Re-education of the muscle progresses slowly and is begun in the supine position, eliminating gravity. Muscle strengthening may take 6 to 10 months.
- The muscle has to be worked intensively as it is usually too weak to give good abduction. After several months, the average abduction is around 40°.

In weak shoulders with a limited abduction, it may be necessary to effect two transfers at the same time. The most often used muscles are the latissimus dorsi and the trapezius (Figure 22.17). Other authors[5,6] have used the levator scapulae muscle with the latissimus dorsi.

After 6 weeks, the plaster is partially moved, leaving the inferior half and allowing for another 2 weeks rehabilitation in abduction but without adduction. Physiotherapy

will be long and difficult. It is best done lying, and in a pool, as the trapezius is rarely strong enough to rapidly obtain a direct abduction.

The results of our trapezius transfers have been reviewed[4]; in 14 cases we found an average improvement of 43° in abduction after the transfer. In most cases, due to the insertion of the tendon, there was some weak external rotation. These results are in accordance with the literature[7-10] (Figures 22.23-25).

Technique of levator scapulae transfer

It is possible to use the levator scapulae transfer. The use of this transfer is indicated in severely paralyzed shoulders (grade 0) in which neither abduction nor external rotation are present. The rationale is to actively center the humeral head into the glenoid fossa in order to improve the action of the transfer to the deltoid. As the levator scapulae muscle is innervated by the cervical plexus it is always active, even in severe obstetric paralysis, and can be usefully transferred.

The skin incision is the same as used for the trapezius transfer, prolonged posteriorly on the spine of the scapula and vertically following the inner border of the scapula until the inferior angle. Detaching the trapezius to perform the transfer to deltoid at the same time facilitates exposure of the upper part of the vertebral border of the scapula and consequently the scapular insertion of levator scapulae muscle. The levator scapulae is detached from the scapula with a long strip of eriostemon as far as the inferior angle, in order to obtain a long tendon for its distal reattachment to the greater tuberosity. It is important to take care of nerves and vessels for the rhomboid muscles lying in a deeper and more medial plane compared to the levator

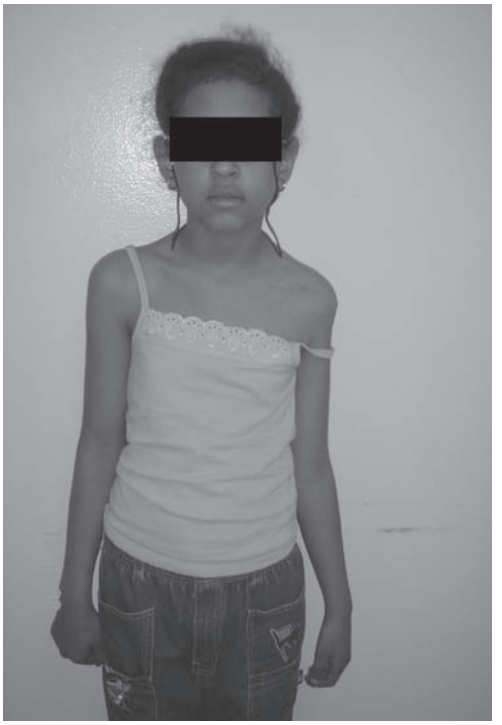

Figure 22.24
One year after trapezius transfer.

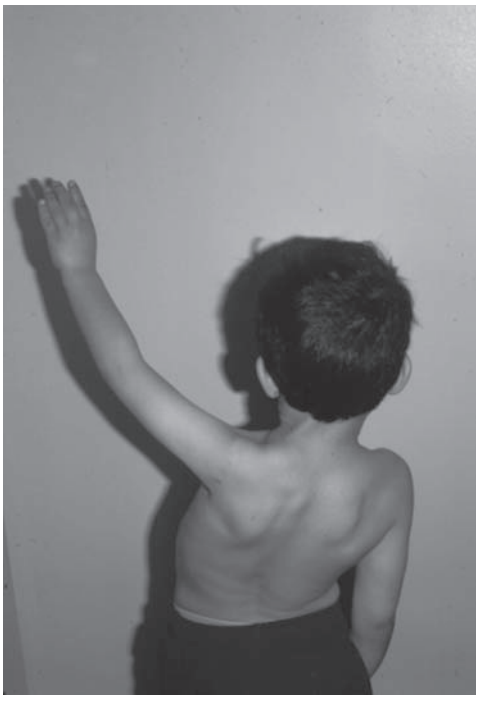

Figure 22.23
Result after 6 months.

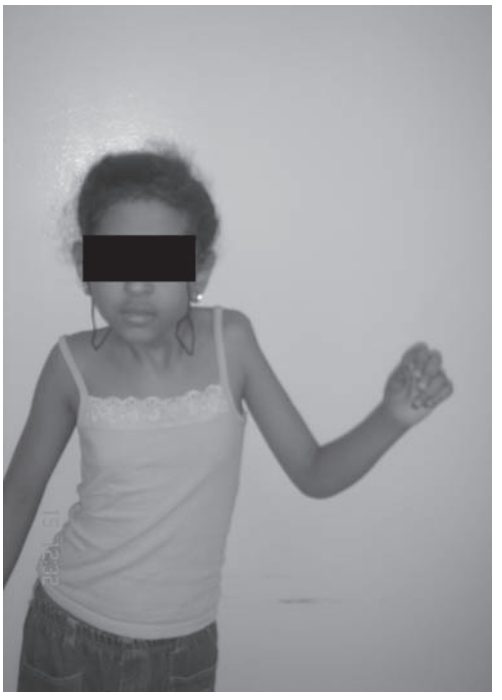

Figure 22.25
One year after trapezius transfer.

scapulae. Special care must be taken when detaching the muscle at its distal insertion to the upper medial angle of the scapula; it is important to maintain a sufficiently strong continuity between the muscle and its prolongation through the periosteal strip. After detaching the levator scapulae from the scapular angle with its periosteal prolongation, it is sufficient to free it proximally no more than 2–3 cm until it can be seen that the direction of the transplant is completely straight towards the great tuberosity.

The distal insertion to the humerus is performed in young children with a 3–4–0 thread, while in adults a bone to bone fixation is used for a stronger insertion, by means of harvesting a piece of bone from the scapula at the end of the periosteal strip so that it can be fixed with a screw to the great tuberosity.

The donor area at the inner scapular border has to be repaired by reinserting the rhomboid muscles in order to preserve its function (remember that the rhomboid and serratus together are responsible for bascule movements of the scapulothoracic joint).

After the insertion of the levator scapulae transfer to the humerus, the trapezius is transferred to substitute for the paralyzed deltoid.

Results

In shoulder 0 the results obtained by the double transfer of levator scapulae and trapezius depend on the scapulothoracic joint active function. A series of 6 cases with no active scapulothoracic joint motion were all upgraded from shoulder 0 to shoulder II, which means an average improvement of approximately 15° on our results of the trapezius transfer alone.

Although in groups III or IV there is no need to do extensive surgery and a single latissimus dorsi transfer is sufficient, the lower groups 0, I, and II are different as the latissimus may be absent or weak and there is a need to support the transfer by another muscle. Narakas[5] and Raimondi[6] like to associate latissimus and teres major, as did Sever[1] and Merle d'Aubigné before them. Gilbert[4] adds the teres major only in cases where the latissimus is weak or pale.

We have already discussed the joint transfer of latissimus and teres major. We use this transfer in cases where the latissimus is pale, thin, and weak, while the teres has a favorable appearance. The two tendons are detached and separated: the latissimus tendon is moved to the rotator cuff, and the teres major tendon is sutured on the latissimus at the musculotendinous junction or on the infraspinatus tendon. In a severely paralyzed shoulder with limited and poor function (shoulder I) we like to associate the trapezius with the levator scapulae and latissimus dorsi or the latissimus dorsi and the teres major. In 20 patients, this procedure has allowed us to obtain a shoulder III in 17 cases, with good scapulothoracic function, and 3 shoulder II when the scapulothoracic joint was unstable (Figures 22.26 and 22.27).

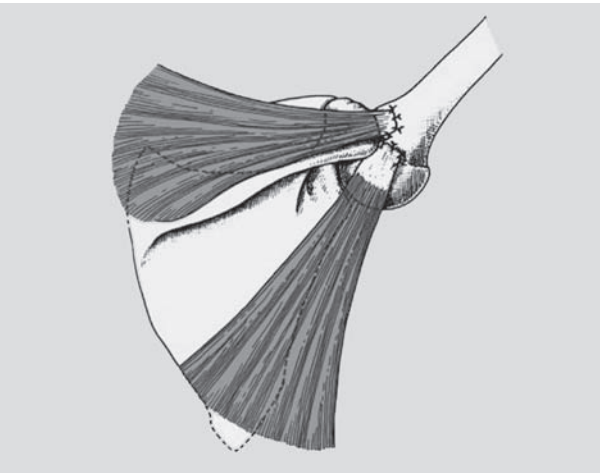

Figure 22.26

Double simultaneous transfer of trapezius and latissimus dorsi.

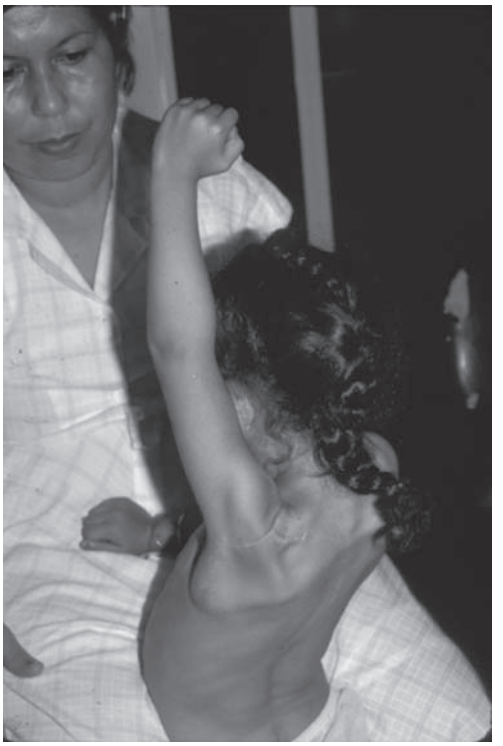

Figure 22.27

Result after double transfer.

Our indications are the following:

- Shoulder III–IV: isolated latissimus dorsi transfer
- Shoulder II: trapezius transfer + latissimus dorsi or levator scapulae
- Shoulder 0–I: trapezius (+ levator scapulae)

Medial rotation defect

Lack of active internal rotation is not rare, but does not commonly need treatment. In the past 25 years, we have

had to treat this defect in 7 cases only. In these cases, the pectoralis major was active, showing that it has no important role in medial rotation. The pectoralis can be used for reconstruction of internal rotation.

Transfer of pectoralis major for medial rotation

The patient is placed supine with the shoulder elevated. The arm is abducted and the incision is made in the deltopectoral groove. The tendon of the pectoralis major is followed to its insertion on the humerus and sectioned. The arm is then rotated 90° inward and the tendon fixed to the bone anteriorly in maximum internal rotation. Fixation is done with a bone anchor (Figure 22.28). After fixation, the incision is closed and the arm immobilized in a soft dressing with the hand on the opposite shoulder for 4 weeks. Rehabilitation will be started after this month. This procedure gives some internal rotation, but never a complete result.

Late shoulder deformity

In patients where the imbalance has not been treated, or where treatment has failed, the longlasting malposition will induce joint deformities, more severe as time passes:

- Flattening and posterior subluxation of the humeral head with retroversion of the humeral neck. The glenoid does not develop normally; it is flat and retroverted.
- Later, the joint becomes non-congruent, with posterior dislocation and severe medial rotation. The coracoid is elongated and has a downward position. The acromion may be larger anteriorly and laterally.[11] The glenoid is retroverted.[12–14]

In the child or adolescent with a deformed shoulder, the head can be seen posteriorly. Abduction is limited and there is usually no external rotation. The situation has to be studied radiologically: either by ultrasonography, computed tomography (CT) scan,[15] or magnetic resonance imaging (MRI).[16] We believe that the best and more accurate results can be obtained with CT scan 3D reconstruction, which shows the extent of the anomalies.

There is sometimes slight pain, however very often the situation is painless but the patient seeks functional and cosmetic improvement. The difficult questions for the surgeon are:

- Can we improve the situation? At what cost?
- Even if the functional improvement is limited, should we try to reintegrate the humeral head in order to prevent later arthritis and pain?

The answers to these questions are not easy and certainly differ. Several authors,[17–19] even in cases of posterior dislocation, advocate a surgical reduction, using soft-tissue releases, often associated with tendon transfers and sometimes with rotation osteotomies. Birch[20] advocates, at least in not too severe cases, reintegrating the head and reconstructing the joint with a complex operation combining:

- osteotomy of the coracoid
- lengthening of the subscapularis tendon
- if necessary, medial rotation osteotomy in order to correct the retroversion, once the normal external rotation is obtained.

This is certainly a very interesting suggestion, but we still need to see the long-term functional results of these operations.

At this time, we have tried in several cases to treat the medial rotation contracture in isolation by resection of the coracoid. This can be done either in older children or even after recurrence of subscapularis release and any case where the release is contraindicated because of some anomaly in the joint.

Technique of coracoidectomy and ligament release The patient is installed supine with the shoulder elevated and the arm draped free.

A short S-shaped incision is made from the clavicle to the tip of the coracoid, felt by palpation. The deltoid and pectoralis muscles are retracted and the long coracoid approached. With a scalpel, the periosteum is incised longitudinally and elevated from the bone. When the periosteum is completely elevated, the coracoid is resected as close as possible to its base with a Liston bone cutter. It is then very important to cut the coraco-acromial and coraco-humeral ligaments; once this is done, it is usually not necessary to add any procedure and a reasonable external rotation is obtained (30° to 40° after an initial −15°, −20°). The muscles are approximated over a drain and the skin closed. There is no immobilization and the physiotherapy is started immediately. The patient is instructed to keep his hand at the back of his head as much as possible, especially when sleeping.

The results of this simple operation are excellent and give a constant improvement, limited but enough to make

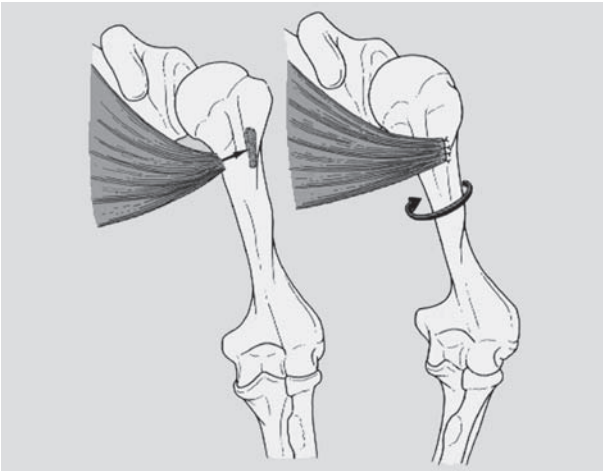

Figure 22.28
Transfer of pectoralis major.

the patient happy. However, this does not solve the problem of the patient with posterior dislocation. In some cases, after coracoidectomy, the head reintegrates the glenoid in external rotation, but when the patient does medial rotation, it comes out again. The retoversion could be treated by a medial osteotomy such as Birch, but there is also a severe anomaly of the glenoid. We have tried in two cases to add a posterior approach with stabilization by a bone block, as the glenoid is too small for an osteotomy. The head was stabilized, but at the cost of more stiffness to the shoulder.

The treatment of the late shoulder deformities is so unsatisfactory that anything should be done to avoid these anomalies. It is paramount to follow and treat immediately any type of shoulder deformity in the young child. Alternatively, if we consider that this shoulder dislocation cannot be treated and does not represent a particular risk for the future, osteotomy of the humerus can produce excellent cosmetic and functional improvements.[21–24]

Late sequelae

The late sequelae are a function of the severity of the joint anomalies. These anomalies are common and often very important, although there is usually no correspondence between the degree of anomaly and its clinical demonstration: limitation of movement is common but pain is rare.

The already noted deformities become more obvious with time and clinically noticeable:

- Posterior subluxation is not unusual, sometimes with complete posterior dislocation. A CT scan shows a flattened humeral head, looking to a new posterior glenoid. The normal glenoid is flat and hypoplastic, demonstrating the vanity of attempts to relocate the head.
- The head is posteriorly rotated, aggravating the situation. Clinically, the shoulder has little or no external rotation.
- The radiologic exam usually shows a large coracoid process and a large acromion, covering the head. This acromion is visible but it is not clear whether there is a clinical consequence. It seems that this hypertrophied acromion is responsible for a limited passive abduction of the shoulder. However, several attempts at resection of this acromion have not brought a significant improvement.

It is important to remember that these anomalies develop over many years and have nothing in common with traumatic dislocation. It may be possible to prevent aggravation and, in some cases, to relocate the head in a young child; it is difficult to imagine that a completely disorganized joint would be accessible to reconstruction. Some rare surgeons (Birch et al[20]) have tried a combination of anterior relocation with a posterior block, often associated with a rotational osteotomy. They claim improvement in anatomy and movement, but these results need to be duplicated.

In most cases, nothing will be done for the patient with considerable deformities and limitations but no pain except to observe him. The patient aged 15–16 will then disappear for many years. We now know that he may reappear 10, 15, or 20 years later with pain. The problem will then be early arthritis and the answer, arthroplasty, is controversial as the anatomy is abnormal, the joint stiff, and the muscles hypoplastic.

The evolution of the shoulder in obstetric palsy is an unsolved problem, and efforts should be made towards early diagnosis of the anomaly and prevention of its development.

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23 Timing of reconstructive surgery after peripheral nerve lesions

Raoul Tubiana

Reconstructive surgery is indicated after nerve palsy in the upper extremity once it is certain that nerve recuperation is impossible. In fact, the problem is complex because it is often difficult to determine precisely the likehood and quality of potential nerve regeneration. The situation was less trying some time ago, when the prognosis for recuperation after proximal nerve lesions was then so bad that one could advise tendon transfers at the time of nerve repair, almost without risk.[1] With progress in nerve repair, early tendon transfers are now seldom required.

Considerations affecting recovery of nerve function after nerve repair

The prognosis after a traumatic lesion of a peripheral nerve depends on many factors. Countless neurophysiological studies have been devoted to axonal regeneration and to the reinnervation of muscle fibers and sensory receptors.

Some factors depend on the quality of the repair. Other factors that affect the result are beyond the control of the surgeon.

Unfavorable factors

Functional recovery is affected by a variety of factors acting at different levels of the nervous pathway, from the neuronal cell body to the peripheral tissues innervated by the injured axon.

Factors intervening in the proximal segment

The axon is under the trophic control of the cell body, which may itself have been damaged by the lesion.

Site and nature of lesion

The site of the lesion can influence prognosis in a number of ways. The more proximal the lesion, the farther the axons have to travel to reinnervate the extremity of the limb. In addition to the distance, there are dangers inherent in the delay in reinnervation. The obstacles include exhaustion of the nerve cell, interstitial fibrosis, progressive atrophy, and degeneration of the muscle fibers.

Another factor that worsens prognosis in proximal lesions is the extent of retrograde axonal degeneration, which is always greater in lesions close to the cell body. This phenomenon can lead to the death of nerve cells, or at best may reduce the regenerating capacity of the axon, which may be unable to reach the extremity of the limb. Retrograde degeneration is also influenced by the severity of the trauma, the site of nerve division, and the nature of the injury. It is more marked after avulsions and after injuries by high-velocity missiles.[2]

From the prognostic point of view, open injuries, are more disturbing than closed injuries, because of the risk of infection and cicatricial tissue formation. Associated traumatic lesions of muscle, bone, and blood vessels increase the scarring around and ischemia of the damaged nerve. The state of the 'nerve bed' also influences the quality of the repair. It is probably fair to say that the greater the degree of trauma, the less likelihood of achieving good functional regeneration.

Factors intervening in the distal segment

The cicatricial tissue that joins the ends of a severed nerve or develops at the site of a crush injury has a marked effect on regeneration to an extent that varies with its volume, density, nature, and architecture. It can have a constrictive effect as it interferes both with the growth in length and later with the maturation of the regenerating axons. In addition, the large increase in collagen deposited around the Schwann tube within the endoneural spaces of the distal segment results in a reduction of their diameter, and this in turn is bound to affect the completeness of regeneration.[2]

Factors intervening at the periphery

A regenerating axon can mature fully only if it can establish appropriate connections with a functionally related end-organ.

The quality of anatomic and functional recovery depends on the number of regenerating axons that succeed in reaching the periphery and on the extent to which the original innervation has been restored.

The nerve involved

From a prognostic standpoint, the risks of directional errors concerning the axons seem greater when the nerve is 'mixed'. All motor nerves contain sensory fibers. Repair of predominantly motor nerves (e.g., the radial nerve) carries a better prognosis than that of 'mixed' nerves containing a high percentage of sensory fibers (e.g., the ulnar and, above all, the median nerve).

General factors

The age of the patient is of such importance that any analysis of results must be made by age groups if it is to be valid. Every series published so far confirms that the best results are obtained in young subjects. There is a rough correlation between the age of the patient and the level of functional recovery achieved. Only patients under the age of 13 years can be reasonably certain of achieving some degree of fine sensibility after suture.[3]

It has been suggested that in children the axons have a shorter distance to travel because the limbs are shorter, and so the capacity for regeneration is greater. The better return of sensibility in younger patients seems unlikely to be on the basis of an improved ability to remyelinate their sensory axons. It seems likely that the better sensibility regained in children relates to their intrinsic central nervous system adaptability rather than to more accurate or more efficient regeneration of individual axons.[4]

The effect of age on conduction velocity has been studied by several investigators. Thomas and Lambert[5] reported that in children 5 years old or less, conduction velocity is slower than that in the adult. This is readily explained by the fact that the rate of conduction is directly proportional to the diameter of the fibers, and this changes little after the age of 5. At the other end of the scale, conduction velocity decreases with age (by about 10% at the age of 60 years) owing to local ischemia and reduced permeability of the cell membrane.[6]

Effect of delay on prognosis

It has been established that the regeneration capacity of the nerve cell is less important than the degeneration of the denervated muscles.[7] Some nerve cells probably retain for years the capacity to grow new axons. By contrast, the period during which a denervated muscle can recover useful function is shorter. This delay in muscle recovery depends on the number of useful axons reaching the muscle and the number of surviving muscle fibers capable of reinnervation. Each of these must be considered.

The number of functional axons reaching the denervated muscles is necessarily smaller than normal by an amount that varies with the number of neurons surviving retrograde degeneration, the number of axons whose growth is stopped by fibrosis at the level of the injury, the number of axons misdirected outside the fascicles, and the number of axons misdirected within the fascicles.

The number of muscle fibers capable of reinnervation depends on the degree of destruction at the time of injury, the secondary degenerative changes in the muscles leading to atrophy and sclerosis, the capacity for connection between the extremities of the nerve fibers reaching the motor endplates, and the restoration of sufficient neuromuscular units to achieve useful motor function.

Gutmann and Young[8] believed that the longer the delay in reinnervation, the less the chances of return of motor function, because the proportion of reinnervated motor endplates is rapidly reduced when atrophy sets in. Also, the formation of new endplates slows because they are probably less efficient than the old ones. The number of fibers destined not to be reinnervated increases with time. It follows, therefore, that the earlier reinnervation occurs, the better are the chances for functional recovery.

It has been shown that the delay prior to a nerve repair affects the quality of the results. Figures published by the US Army Medical Services concerning nerve suturing performed during World War II showed that every delay of 6 days before the repair reduced the quality of the result by 1%.[9] Analyzing their results in microsurgical repair of nerve injuries, Zilch and Buck-Gramcko[10] found that 85% of their failures occurred when the repair was performed more than 4 months after the injury.

Delay between injury and nerve repair has much stronger effects on functional scores than on electrophysiologic measures.[11]

From a practical viewpoint, when assessing the theoretic chances for recovery, the surgeon should add the preoperative delay to the time needed for the growing axon to reach the paralyzed muscle (on the basis of an average of 1 mm/day). If the total is greater than 18 months, it is often wiser (especially if the patient is not young) to combine a nerve repair with palliative surgery to correct the motor deficit resulting from lack of function of the paralyzed muscles, provided this does not produce a further deficit.

By contrast, sensation can return after much longer delays – 7 years or more in some of our cases. Protective sensation, however, should not be confused with the return of 'stereognosis', which is difficult to obtain even with early repair.

Factors pertaining to treatment

The prognostic factors pertaining to treatment are extremely important.

The controversy over primary or late secondary repair of divided peripheral nerves has been a point of considerable argument. At present, the guideline is to repair the nerve at

the earliest convenient time. It is crucial that the nerve repair performed be as good as possible. With appropriate wound care, it is preferable to postpone a nerve repair until a qualified, experienced surgeon is available to perform the surgery under ideal conditions. He will choose the appropriate type of repair by suturing or grafting.

Surgical procedure has important relations with the quality of the nerve repair. The technique should be atraumatic, and excessive dissection and suturing under tension should be avoided.

The use of operating microscopes, microinstruments, microsutures, and fibrin glue has now become commonplace.

The nerve bed, at the site of the repair, should be cleared of scar tissue. Repair of the vessels, as is increasingly widely practiced, has a beneficial effect on the nerve repair, and probably also on the state and the number of surviving muscle fibers capable of reinnervation.

Indications for early reconstructive surgery

Advantages and risks of early reconstructive surgery

Advantages

Early reconstructive surgery in appropriate cases permits functional recovery and prevents the formation of deformities and trick movements that are difficult to eliminate. It also removes the need for cumbersome and constraining external splints.[14] It is for this reason that these operations have been referred to as 'internal splints'. Also, because all the tissues are still supple at this early stage, one can use less powerful transfers than are necessary in late reconstructive surgery. In addition, as Omer[12] mentioned, recuperation of a voluntary active motor function is an essential factor in sensory re-education.

What are the risks of this surgery?

There are two kinds of risk. First, harvesting of active muscles for use as transfers will increase the motor deficit unnecessarily if nerve recuperation eventually occurs. Second, deformities may develop because of excessive force after nerve recuperation.

The indications for these procedures are thus limited to instances in which (1) for any reason, nerve repair cannot be done; and (2) the theoretic chance of recuperation is remote or projected to be very late.

When there is a chance of nerve recuperation, the indications for early reconstructive surgery are rare. They are designed only to attempt a transitory means of aid. The techniques used must be chosen with great care. The possible transfers must not decrease the strength of the residual function and must not create new impairment in case of recuperation. It is essential not to use procedures that leave irreversible deficits.

The timing of reconstructive surgery is often guided by the level of the lesion.

Brachial plexus lesions

The extent of the paralysis and the difficulty in establishing the prognosis early considerably limit the use of early transfers after plexus injuries. However, in certain selected cases, it is possible to perform transfers or tenodeses at the wrist and the distal joints without incurring a long delay for an uncertain recovery. In particular, in the rare isolated distal lesions of C8 and T1, the nerve repair does not result in intrinsic muscle recovery in adults. Thus, one can perform reconstructive surgery early.

Peripheral nerve lesions

The decision for early reconstruction in peripheral lesions is guided by the time required for reinnervation of the paralyzed muscles. An estimate is made by adding the time that has elapsed before nerve repair to the time necessary for axonal regeneration to the paralyzed muscles, approximately 1 mm/day. If the total is more than 1 year, it may sometimes be wise to combine nerve repair with reconstructive surgery that will not compromise function in the case of reinnervation. Poor technical repair, delayed repair, proximal lesion site, unrepaired associated vascular lesions, and elderly patients are all factors that militate against successful recovery after nerve repair.

Proximal peripheral nerve paralysis

Early reconstructive operations are not usually performed after repair of the axillary, suprascapular, or musculocutaneous nerves, because it is preferable to await reinnervation of the muscles. It is possible to determine the extent of recovery after a few months.

Procedures for stabilization of the shoulder or flexion of the elbow are carried out only in cases of confirmed failure of nerve recovery.

Median nerve paralysis

The sensory component of the median nerve is so important that direct nerve repair is always recommended. It is possible to recuperate useful protective sensation even after very late repair (>5 years). Motor recovery after repair varies according to the site of the lesion. In distal lesions, one can expect useful recovery in a good percentage. The more proximal the lesion, however, the less chance there is for intrinsic recovery of the thenar muscles (Figure 23.1).

Thus, it is possible to discuss the possibility of an early opponensplasty in adults for lesions of the median nerve

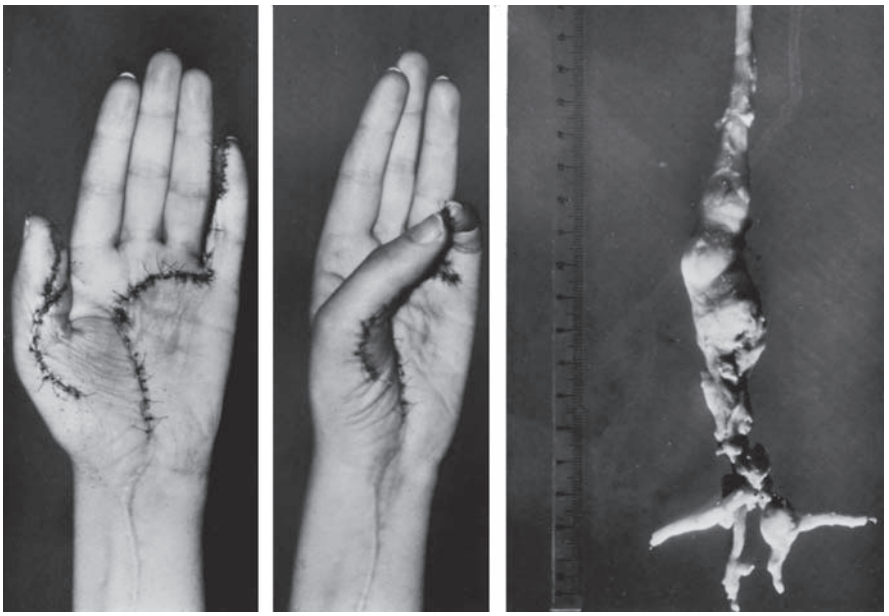

Figure 23.1

Removal of a tumor of the median nerve: immediate restoration of thumb opposition, with a transfer of the flexor superficialis of the ring finger to the thumb, associated with a neurovascular island flap from the little finger to the thumb pulp.

proximal to the elbow, Indeed, thumb opposition is endangered by contracture of the first web space and supination of the thumb, control of which by external splints is difficult.

Anteposition and pronation of the thumb is normally ensured by the abductor pollicis brevis. The power of the thumb grip in cases of isolated paralysis of the median nerve is ensured by the adductor pollicis. A relatively weak transfer is sufficient, such as the palmaris longus in cases of distal paralysis,[13] or an extensor muscle innervated by the radial nerve, such as the extensor indicis proprius, extensor digiti minimi, or extensor pollicis brevis. The use of these dorsal transfers for the patient does not require re-education, because the opening of the thumb coincides with the opening of the fingers. The path of the transfer must be palmar and parallel to the first metacarpal. The techniques for these transfers are described in detail in Chapter 18.

In case of motor recovery, these transfers will not leave any deficits if the proper technique has been respected. The sensory recovery will derive considerable benefit from use of a pulpar grip re-establishing a sensorimotor circuit, facilitating reintegration of the affected limb.

Ulnar nerve paralysis

After repair of the ulnar nerve above the proximal third of the forearm, an early reconstructive procedure may be considered, for the following reasons:

1. It is difficult to obtain useful functional recovery of the intrinsic muscles, especially in patients over the age of 40, because of the distal location of these muscles.
2. The finger metacarpophalangeal joints rapidly become stiff if they are not used, especially if they are in hyperextension. One must therefore strive to correct the ulnar claw before it becomes fixed. Simple stabilization of the metacarpophalangeal joints in slight flexion is sufficient to ensure extension of the distal phalanges and prevent joint stiffness. This stabilization can be achieved by tenodeses, capsulodeses, or tendon transfers. In this situation, procedures that do not require borrowing a tendon from the hand should be employed, such as tenodesis, using a tendon graft (Parkes tenodesis), or a capsuloraphy.

The strength of the thumb is much diminished because of the adductor paralysis, to which is often added paralysis of the flexor pollicis brevis. The patient compensates for the loss of the adductor by using the adducting action of the extensor pollicis longus, which brings the thumb column into retroposition and supination. If this deforming posture is not corrected early, the patient will have difficulty restoring opposition of the thumb if the nerve recovers or after a late transfer. Thus, when the chances for recuperation are small, one can propose an early transfer for restoration of flexion–adduction of the thumb, without reinforcing the tendency for supination. It is preferable not to use the far too important flexor digitorum superficialis at this stage, but instead to use an extensor tendon as motor (e.g., the extensor indicis proprius).

Associated paralyses of median and ulnar nerves

The arguments already discussed are equally applicable to these paralyses.

Transfer of the extensor indicis proprius can be used early to prevent retroposition and supination of the thumb induced by the extensor pollicis longus. Its tendon should follow a path subcutaneously from ulnar to radial sides and is inserted on the tendon of the abductor pollicis brevis and to its dorsal expansion to the extensor pollicis longus.

At the level of the fingers, the goal is to prevent hyperextension of the proximal phalanges. The early reconstructive procedures described for isolated ulnar paralysis can also be used here.

Radial nerve paralysis

One should be cautious in recommending early palliative operation after radial nerve repair, because recovery after repair is frequent. The most favorable prognostic factor is the great proportion of motor fibers and the proximal location of the muscles innervated by the radial nerve.

During the waiting period, it is extremely important to avoid flexion contracture of the wrist and extension contracture of the metacarpophalangeal joints.

Problems relating to radial nerve injuries and fractures of the humerus are examined in Chapter 13.

The indications for an early tendon transfer are rare. However, one should distinguish between transfers aimed at restoring wrist extension and those that extend the finger and the thumb.

Transfer of the pronator teres to the extensor carpi radialis brevis can be performed early. A longitudinal incision is made on the radial side of the forearm. Then, after the forearm is placed in pronation, the pronator teres is passed subcutaneously over the brachioradialis and the extensor carpi radialis longus brevis at the level of the musculotendinous junction. This transfer re-establishes wrist extension, and the pronator teres conserves its pronating action on the forearm.

If, by any chance, reinnervation occurs, regeneration of the wrist extensors reinforces the often insufficient action of the pronator teres transfer and does not hinder hand function if the wrist flexors are conserved. On the contrary, transfers to the finger and thumb extensors become too powerful after nerve regeneration, causing a constraint to finger flexion and to opposition that is harmful for function.

I had to reoperate on a pianist who suffered from an open fracture of the humerus; the radial nerve was crushed and divided. However, the nerve had been sutured, and tendon transfers were performed at the same time for wrist and finger extension. The radial nerve recovered more than had been hoped for, and the patient had his fingers fixed in hyperextension, so that I was obliged to take down the transfer on the fingers.

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24 Nerve entrapment syndromes provoking motor deficits in the upper extremity

Ioannis Tsionos and Dominique Le Viet

Nerve entrapment syndromes implicate compression of nerves by adjacent anatomic elements. This compression takes place particularly as the nerve traverses some 'naturally tight' passages, and it can be aggravated by the presence of some aberration in local anatomy. Such phenomena frequently have some dynamic composite; that is, they are manifested or intensified after some repetitive movements or in some specific position of nearby articulations.

Entrapment syndromes often produce transient sensory deficits and less frequently motor deficits. Recognition of impending motor deficits is of paramount importance, as the time limits then imposed are tight if someone is to expect motor recovery.

The true neurogenic thoracic outlet syndrome

The thoracic outlet syndrome (TOS) results from compression of the brachial plexus and/or the subclavian vessels at their passage through the thoracic outlet. Entrapment more frequently is encountered in the interscalene triangle, but it can also be produced at the opening between the first rib and clavicle or more distally as the neurovascular bundle passes under the coracoid apophysis and behind the pectoralis minor tendon.

Two main types of this syndrome have been recognized, according to a popular classification: (A) vascular type, subdivided into arterial and venous; and (B) neurogenic type, subdivided into true and disputed. Neurogenic TOS is the more common, but unfortunately the majority of cases belong to the disputed type, where neurologic symptoms are transient, atypical, and impossible to register objectively as precise neurologic deficits.

True neurogenic TOS is extremely rare in the general population, accounting for only about 1 per million subjects. The nerve territory implicated is commonly the one furnished by the anterior rami of the C8 and T1 spinal nerves or the lower trunk of the brachial plexus (lower neurogenic TOS). Exceptionally, upper plexus compression (implicating the C5, C6, and C7 roots, or the upper and middle trunk) may be accompanied by motor deficits (mainly in wrist and finger extension).[1] True neurogenic TOS seems to always have a limited spectrum of causes: either an abnormally large transverse C7 process or a cervical rib is present; that is, a bony aberration is always found (Figure 24.1). Compression ensues commonly from taut fibrous bands bridging these osseous anomalies with the first rib and limiting the space available for the plexus in the interscalene triangle.[2–4]

Symptoms and signs of lower neurogenic TOS

Patient age ranges from early adulthood to elderly, with a peak at 40 years. These cases may be referred to with incorrect diagnoses, such as cubital tunnel syndrome, carpal tunnel syndrome, or C8 or T1 radiculopathy. Symptom appearance is insidious. The patient (typically a woman) may complain of pain that is variable in intensity, intermittent, usually localized in the supraclavicular area, and reflecting in the medial arm and forearm. Paresthesias are reportedly intermittent and usually slight, which contrasts with established motor deficits, visible muscle wasting, and weakness in the hand. Symptoms are unilateral in the majority of cases.

Objective hypesthesia can be detected in C8 and T1 territory, that is, in the ulnar fingers, the ulnar border of the hand and the medial aspect of forearm and arm. This hypesthesia is usually rather slight. Muscle wasting and weakness concern preferentially the thenar muscles. The rest of the intrinsic hand muscles are usually less implicated, and this is even more so for forearm muscles. Ulnar finger clawing may be present.[3] Finger flexor reflexes may be decreased or abolished in some cases.[2,5] Digital compression of the

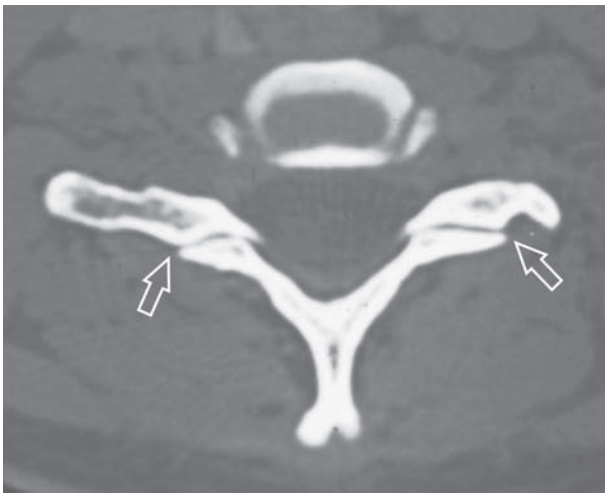


Figure 24.1
Bilateral cervical rib articulating with the transverse processes of the C6 vertebra.

interscalenic space and other provocation tests described for TOS are helpful in making the diagnosis.

Electrophysiologic studies

Electromyographic (EMG) needle studies show a chronic and slowly progressive neurogenic type of involvement with a C8–T1 nerve root distribution. Thenar muscles, hypothenar muscles, and the extensor pollicis brevis may be found to be influenced, confirming a C8–T1 pattern rather than a peripheral nerve one. Classically, findings in thenar muscles are more prominent.

Nerve conduction studies (NCS) are very helpful in making the diagnosis. Sensory NCS recordings from the index finger are normal, and those from the fifth finger show moderately low amplitudes. F-wave latency measurements on the ulnar nerve may show slowing due to proximal compression. No F-wave slowing is registered on the median nerve, proving absence of proximal compression on the C5, C6, and C7 roots or the upper and middle trunk.[2,4,6] Motor NCS shows low or abolished compound muscle action potential (CMAP) amplitudes in median-powered thenar muscles and to a lesser degree in ulnar-innervated intrinsic muscles.

Electrophysiologic study of the medial antebrachial cutaneous (MAC) nerve makes an important contribution to diagnosis. With stimulation at arm level and recording below the elbow, sensory nerve action potential (SNAP) is found to be severely diminished or non-obtainable – evidence against compression of the ulnar nerve at the elbow or wrist as well as against preganglionic C8–T1 root compression.

Differential diagnosis of neurogenic TOS

Aspects of this setting may simulate carpal tunnel syndrome, compression of the ulnar nerve at Guyon's canal, or cubital tunnel syndrome. Other neurologic conditions, such as syringomyelia, focal motor neuropathies, radiculopathies, spinal cord neoplasms, and lower trunk compression by neoplasms, may be confused with neurogenic TOS. A combination of clinical characteristics, imaging study findings, and results from electrophysiologic investigation will permit a correct diagnosis. For example, a preganglionic C8–T1 root compression may present the electrophysiologic profile of neurogenic TOS except for the SNAP in ulnar and MAC nerve territory, which is normal.[2]

Treatment of neurogenic TOS

Neurogenic TOS, once recognized, is treated surgically – a waiting attitude is likely to lead to more muscle wasting. Expectations from surgery should be modest:[4,5] although some amelioration in degree and ever some reversal of hand atrophy may occasionally be achieved after the operation, in most cases a successful result consists of stopping the progress of muscle wasting and alleviating the patient from pain and paresthesias. This impossibility of motor deficit reversal is most likely the result of the distance between lesion level and target muscles, the severity of compression, and the duration of compression before surgery.[4]

Especially for neurogenic TOS, a supraclavicular approach must be chosen, as bony abnormalities accompanied by taut fibrous bands are always found superior to the first rib and are thus unreachable by a transaxillary approach. The goal of the operation is to perform excision of abnormal bony and soft tissue elements and external neurolysis of the brachial plexus, when needed. Scalenectomy plus or minus first-rib resection are also performed.

Adopting the above moderate expectations, surgery seems to fulfill its goal, according to some small series.[2,6]

Neurogenic TOS after clavicular fracture

After a fracture of the clavicle, typically in its middle third, neurologic symptoms may ensue. Plexus compression can be present from the beginning, but it may also arise during or after fracture healing, the symptoms appearing from days to weeks to years after the trauma.[7]

There may be pain and paresthesias, but also motor deficits, sometimes occult, manifesting as difficulty with use of the limb and fatigability.[7] Symptoms due to vascular compression may also be present. Although clavicular fractures are common, these incidents are rare: in a series of 690 clavicular fractures, only two late brachial plexus palsies were noted.[8]

Compression is due to exuberant callus formation, healing in an extremely displaced position, non-union,[9,10] a pseudo-aneurysm of the subclavian artery,[11] or congenital pseudarthrosis.[12] Frequently, the medial cord is more severely involved.[7]

Delayed compression of the brachial plexus can be dealt with conservatively when it presents mainly with sensory symptoms: postural changes, shoulder girdle exercises, various bandages and analgesics may help reduce symptoms and widen the costoclavicular passage. However, patients with vascular occlusions, pseudo-aneurysms, and overt motor deficits or progressive neural deficits are candidates for prompt operative treatment.

Surgery on a healed clavicular fracture aims at simple removal of exuberant callus, or, in the case of malunion, at osteotomy, reduction of the malunion, and fixation.[7] Vascular problems should be dealt with simultaneously. A pseudarthrosis can be treated with fixation[7,9] or excision.[9,10,12] In the case where fixation is favored, stability is mandatory to avoid non-union or union with exuberant callus formation and recurrence of the compression.[7] Sometimes, a first-rib resection is added to widen the costoclavicular passage.[7] External neurolysis is performed as needed. Partial or complete alleviation from sensory symptoms is to be expected, and motor recovery is sometimes possible, even for intrinsic hand muscle involvement, probably due to a short duration of compression before surgery.

Long thoracic nerve entrapment

The long thoracic nerve (LTN) is formed mainly by branches from ventral rami of the C5, C6, and C7 roots, promptly after exiting the intervertebral foramen; it may also receive contributions from the C4 and C8–T1 roots. The C5 and C6 branches traverse the scalenus medius muscle, to be joined later by the C7 branch.[13–16] The LTN passes behind the plexus, axillary vessels, and the clavicle, and over the first rib, to then lie on the lateral aspect of the thoracic cage and supply the serratus anterior muscle.

Many causes of LTN palsy have been reported or presumed. In this long list, palsy after strenuous manual work or sport activities, or after a single violent incident (e.g., a sudden arm jerk or a blow to the shoulder) account for some 60% of cases[17] and are nearer to the notion of 'entrapment'. Palsies induced by body position during anesthesia (5%) and iatrogenic palsies (22%) also have a mechanical nature. This leaves only a minority of cases with infectious, miscellaneous (neuralgic amyotrophy, postinfection palsies, or direct trauma) or unknown causes.

Through its long course, the LTN is relatively fixed at its passage through the scalenus medius and on the lateral thoracic wall, over the proximal part of the serratus anterior. The terminal portion of the nerve is situated anterior to the inferior angle of the scapula.[15] This part of the nerve can be vulnerable to compression or stretching during movements of the shoulder joint.[15] Strangulation of the nerve's vascular supply may also play a role.[15] Besides, a fascial sling exists at the first intercostal space level,[18] extending between the first digitations of the serratus anterior and the brachial plexus and engaging the LTN. Putting the head and neck in contralateral rotation and tilt, and the upper extremity in abduction and external rotation, produces a bowing of the nerve[18] and puts extreme tension on its relatively fixed proximal part, by virtually doubling its length.[14] Thus, one of the above mechanisms, or a combination, might explain serratus anterior palsy of mechanical nature. When nerve stretching occurs, a vicious circle of palsy–winging–stretch–palsy deterioration may be established.

Symptoms and signs of LTN entrapment

The patient (typically a young male, affected on his dominant side[14,17]) becomes aware of the palsy because of shoulder dysfunction or a visible winging. In arm abduction and forward elevation, the scapula fails to be driven away from the midline, thus assuming a position of medial translation and downward rotation, and its medial border is no longer anchored against the thoracic cage (Figure 24.2). Thus, arm abduction and forward flexion are limited to some 90°–110°. The scapulohumeral angle (SHA) is increased in the resting position and increases even more with abduction.[19]

Scapular winging is well demonstrated when the patient is asked to push against a wall. Stabilizing the scapula with the examiner's hand (the scapular stabilization test[20]) or with the patient's own body weight ameliorates abduction.

A dull pain may appear during physical activity, radiating to the neck or upper arm.[13] Clicking or popping may be noted.[16] Even rotator cuff pathology may ensue.[13]

Electrophysiologic studies

Needle studies are the principal tool, NCS being more prone to variations.[13] Needle examination of other muscles of the same territory (C5–C6–C7), including paraspinal muscles, as well as sensory NCS are mandatory to exclude a nerve root lesion. Finally, other nerve root territories should be explored, to exclude a more generalized deficit (as in neuralgic amyotrophy).

Differential diagnosis

This includes other causes of serratus anterior dysfunction, as well as scapular winging from palsy of other muscles. Neuralgic amyotrophy is typically preceded by severe pain and it usually implicates more muscles. Trapezius or rhomboid palsy may produce winging, but the scapula is pulled laterally by the healthy serratus anterior.[20] Muscular dystrophies are usually characterized by low progression, bilateral deficits, and a specific pattern of involvement, as well as a particular electrophysiologic profile.

Because of a feeling of 'giving way' experienced by the patient in arm elevation, some patients have been

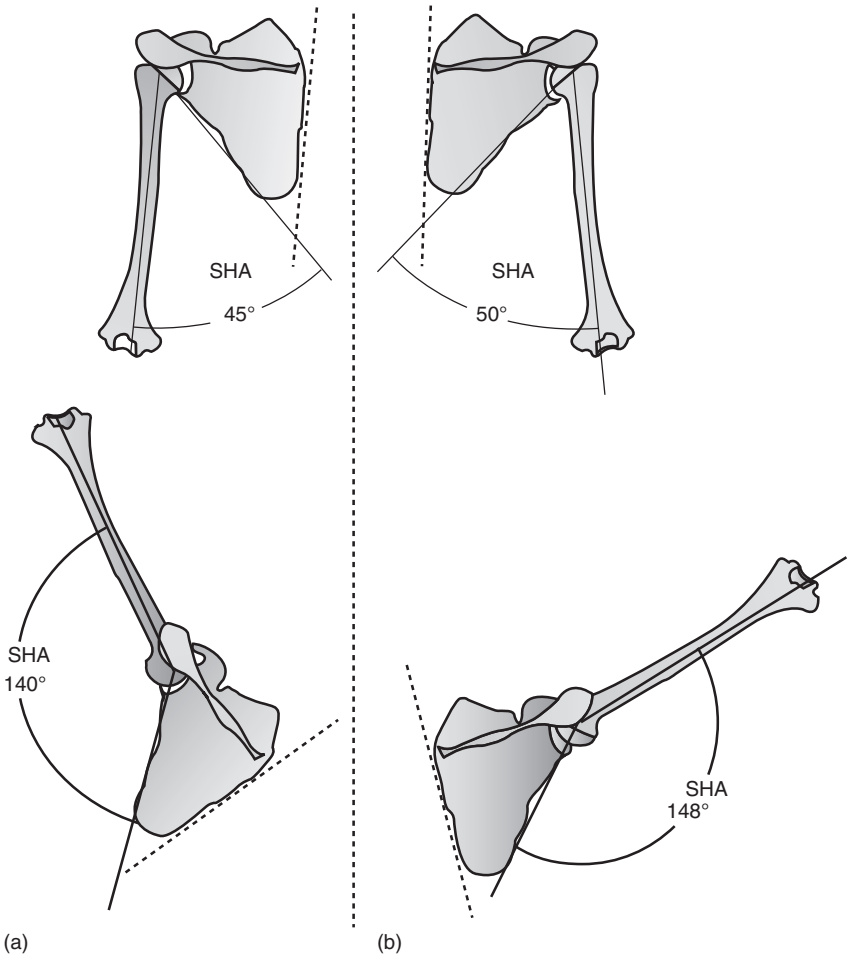


Figure 24.2

Scapulohumeral motion in patients suffering from long thoracic nerve palsy. The normal side (a) is used for comparison. In the resting position, the scapulohumeral angle (SHA) is greater on the side of the palsy (b) and the scapula is positioned more medially. During abduction, the scapula on the side of the palsy fails to move away from the axis of the body and to rotate upwards. Thus, abduction is limited and the extremity appears shorter.

diagnosed as suffering from shoulder instability.[20] Others experience symptoms and present signs of subacromial impingement.[20] LTN palsy must also be differentiated from deltoid or supraspinatus palsy, where the SHA is decreased.[19]

X-ray examination of the thorax, the cervical spine, and the shoulder is prescribed, sometimes supplemented by computed tomography (CT) or magnetic resonance imaging (MRI),[16] to differentiate this pathology from cervical spondylosis, disk herniation, tumors, and rotator cuff pathology.

Treatment of LTN entrapment

As patients with LTN entrapment are mostly young and physically active, they are unlikely to accept functional impairment. Happily, LTN entrapment has good chances of spontaneous recovery. Function returns after an average of 9 months, but may take up to 2 years.[14] The LTN is particularly long, and this may partly explain the length of the convalescence period.[15] No surgical treatment is generally recommended before 2 years have elapsed without recovery.[14] However, as this leaves the patient handicapped for a long period, some authors propose surgery somewhat earlier in motivated patients,[16,21] reserving a longer observation time for those suffering from neuralgic amyotrophy.[21]

Conservative treatment includes avoidance of active forward flexion or abduction of the arm beyond 90°, in the upright position.[20] Active and passive mobilization are performed without provoking winging, with the patient's own body weight fixing the scapula. Bracing devices are poorly tolerated.[22] Restoration of function is not always complete, and a minority of patients may continue to experience winging and disability.[16]

Beyond the above time limits, surgery may be proposed in a patient with disabling symptoms. Among all of the proposed surgical options (muscle transfers, various types of passive scapular stabilization using fascial bands or synthetic materials, and scapulothoracic fusion), transfer of the sternal head of the pectoralis major seems to be the first choice for these patients,[16,20,21,23] being both favorable from a biomechanical point of view and cosmetically acceptable. The insertion of the transfer is prolonged or reinforced with fascia lata[16,21,22] or a semitendinosus–gracilis autograft.[20] Postoperatively, strict physiotherapy protocols are followed.[20,22] Lifting more than 10 kg or participating in contact sports is prohibited for 1 year post operation.

Another method for treating non-recovering palsies is to transfer a functioning nerve onto the distal part of the non-functioning one, near the paralyzed muscle, thus prompting rapid re-innervation. Indications are proximal lesions on very long nerves, irreparable and neglected lesions in which time limits still permit a distal reinnervation, and lesions in continuity of undeterminable level or multiple-level lesions (as in traction injuries). This technique has been used with success in LTN palsies,[24,25] and a branch of the thoracodorsal nerve seems the most appropriate.[24]

Suprascapular nerve entrapment

The suprascapular nerve (SSCN) arises from the upper trunk of the brachial plexus, taking fibers from the C5 and C6 roots. It crosses the superior border of the scapula medial to the coracoid process, passing deep to the (superior) transverse scapular ligament, through the suprascapular notch, and supplying a branch to the supraspinatus muscle. In the supraspinatus fossa, it gives off some sensory branches to the glenohumeral and acromioclavicular joints. It then passes through the spinoglenoid notch, medial to the spinoglenoid ligament (inferior transverse scapular ligament). This ligament is found in more than half of the subjects.[26,27] In the infraspinatus fossa, it gives its terminal branches (three or more[28]) to the infraspinatus muscle.

Entrapment of the SSCN may be produced at its passage through the suprascapular or the spinoglenoid notch. Stretching and angulation may take place dynamically during scapulothoracic and shoulder motion, as the nerve is short and relatively fixed by the above passages and by the bellies of the muscles that it supplies.[29,30] Some anatomic variations may play a role – for example, the thickness of the transverse scapular ligament or an ossification of it,[31] the shape and depth of the suprascapular notch, the depth of the spinoglenoid notch, or the number of branches to the infraspinatus,[28] and the range of motion of the scapulothoracic and shoulder joints.[32] Ischemic lesions of the SSCN have even been proposed.[33] Prolonged protraction of the scapula, as during lengthy surgical procedures, may also cause SSCN palsy.[34]

Entrapment in the suprascapular notch (high entrapment) seems most common. Repetitive activity of the upper extremity,[35] including cross-body adduction movement, has been blamed. High entrapment may thus be produced by certain sports activities, such as swimming, tennis, and weightlifting, but tumors (e.g., osteochondroma[35]) and fibrosis of the notch due to healed scapular fractures are also causes of compression.

Proximal entrapment of the SSCN by scar tissue after an overzealous distal clavicular excision has been reported.[36]

Entrapment in the spinoglenoid notch (low entrapment) is less frequent. It can be produced especially in overhead activities, as is the case in volleyball players (where an incidence of 10–25% has been reported[28,32,37]), baseball players, and weightlifters. Nerve stretching around the base of the scapular spine during excessive shoulder motion may be the cause. Alternatively, compression in the spinoglenoid notch by the medial edge of the supraspinatus–infraspinatus tendon may happen in abduction and external rotation of the shoulder.[38] Another cause of low entrapment is the existence of ganglia,[39,40] sometimes formed after unhealed labral tears.

Symptoms and signs of SSCN entrapment

Sensory deficits are typically absent. The patient complains of dull aching or crushing pain at the top of the shoulder, which may be referred to the back, the arm, or the upper anterior chest wall,[35] worsening during repetitive use of the upper extremity. In advanced compression, there is weakness in shoulder abduction and external rotation, and muscle atrophy. In low entrapment, only the infraspinatus is weak; even among elite players, this isolated compression can be asymptomatic and may be revealed by a weakness in external rotation of the arm ('the infraspinatus test'[37]).

Cross-chest adduction of the arm, contralateral rotation of the head, and direct exertion of pressure over the suprascapular notch may all trigger pain. Operative treatment (first-rib resection, acromioplasty, etc.) has been tried in many of these patients, without relief from pain.[35]

Electrophysiologic studies

The gold standard is the needle EMG, recording from the supraspinatus and infraspinatus. Findings are limited to these two muscles or to the latter alone – otherwise a radiculopathy or a more diffuse brachial plexus lesion exists.[13] Thus, recording must always extend to other C5- and C6-innervated muscles.[13]

Differential diagnosis

Any pathology that produces pain over the shoulder area may be confused with SSCN entrapment: cervical spine disease, tumors of the region, pathology of the shoulder or acromioclavicular joint, coronary artery disease, etc. Some of them may show mild muscular atrophy of the supraspinatus and infraspinatus, either as a result of nerve compression (e.g., cervical root compression) or secondarily. Neuralgic amyotrophy may sometimes show isolated deficit in the SSCN territory.

Diagnosis is mainly based on clinical and electrophysiologic evaluation. X-rays, ultrasound examination,[39] and MRI[39–41] may give additional information as to notch morphology, presence of ganglia or other tumors, intra-articular pathology, and muscle atrophy.[41]

Treatment of SSCN entrapment

Conservative treatment should be tried when the lesion is mild and no anatomic cause of compression is identified.[13] It consists of limiting overhead and other repetitive activities that may be harmful to the nerve. Strengthening of the rotator cuff muscles and the scapular stabilizers is an important part of this treatment.

For some authors,[13] operative treatment is reserved for cases with pain refractory to conservative management, when muscular atrophy appears, or when an anatomic cause of compression (e.g., a ganglion) is recognized. In the experience of others,[35] early operative treatment avoids the risk of irreversible muscle atrophy and should be preferred in safely diagnosed cases.

For high SSCN entrapment, release of the transverse scapular ligament is more safely done through a posterior approach, after elevating the trapezius from the scapular spine.[35] Reshaping of the suprascapular notch is performed when there is callus formation in it.[35] In SSCN entrapment after lateral clavicular excision, the nerve is approached and liberated from its surrounding fibrotic tissue in the lateral posterior cervical triangle.[36]

In low SSCN entrapment by a ganglion, the nerve can be approached through the infraspinatus fossa, by partially detaching the posterior fibers of the deltoid.[40] Then, the ganglion and the spinoglenoid ligament are excised. Other forms of treatment include arthroscopic debridement of the ganglion cyst, and repair[42] or debridement[43] of the capsulolabral lesions, as well as minimally invasive CT-guided needle aspiration.

In low SSCN entrapment resulting from stretching of the nerve around the scapular spine, the spinoglenoid notch is better approached by the supraspinatus fossa, after detachment of the trapezius (thus also permitting release of the transverse scapular ligament[38]). The spinoglenoid notch is deepened by reshaping of the base of the scapular spine,[38,44] and the spinoglenoid ligament is excised. Splitting the supraspinatus–infraspinatus tendon longitudinally could be proposed on a theoretic basis, but this may put at risk the integrity of the cuff.[38] Adding a partial detachment of the posterior deltoid gives access to the nerve from both sides of the spinoglenoid notch, if needed.

Results depend on the severity and duration of compression. Not every palsy of the spinati is expected to recover, but pain relief is achieved.[35] Less than perfect results are paradoxically produced sometimes in cases with supposedly mild compression and minimal electrophysiologic changes. This is presumably explained by some other painful pathology in the nearby anatomic structures, not addressed by SSCN decompression.[45]

Axillary nerve entrapment

The axillary nerve (AXN) contains fibers from the C5 and C6 roots, running through the upper trunk and posterior cord of the brachial plexus. At the lower border of the subscapularis muscle, it passes dorsally through Velpeau's quadrilateral space. It then gives off motor branches to the teres minor and deltoid muscles, and cutaneous branches to the overlying skin. Being of particularly short length and fixed at the above passage and by its terminal motor branches, it is prone to injuries.

Among diverse causes of AXN neuropathy (shoulder dislocation, direct blow to the shoulder, direct pressure by crutches, etc.), entrapment of the AXN in the quadrilateral space ('quadrilateral space syndrome') has been recognized and reported sporadically in the literature.

Symptoms and signs of AXN entrapment

This is a rare syndrome, typically found in the dominant extremity of young active individuals.[46] Repetitive movements during sports and other activities may compress the axillary nerve or, in some, both the axillary nerve and artery[46] at their passage through the quadrilateral space.

The patient complains of pain that is vaguely localized in the anterior shoulder region and aggravated by arm abduction and forward flexion. Paresthesias in the lateral aspect of the shoulder and the upper arm and weakness in shoulder abduction may also be present. Abduction and external rotation of the humerus for 1 minute will frequently reproduce the symptoms.[46] A point of local tenderness may be found, corresponding to the quadrilateral space, near the teres minor insertion.[46]

As both the radial nerve and AXN derive from the posterior cord, fibrosis and compression in the quadrilateral space may also entrap the radial nerve, producing paresthesias with a superficial radial nerve distribution.[47]

Electrophysiologic studies

Slowing of conduction along the AXN and low CMAP amplitudes may be observed in the deltoid, as well as spontaneous activity and pathologic muscle fiber recruitment during voluntary contraction. In patients with mild intermittent symptoms, these studies may be normal.[46,48]

Differential diagnosis

Diagnosis requires a high index of suspicion. Routinely, symptoms are erroneously attributed to TOS or to subacromial impingement. No single test or examination can give a definite diagnosis. A subclavian arteriogram can show axillary artery occlusion in arm abduction and external rotation,[46,49] but this is not indispensable for the diagnosis,[48] as the cause of the syndrome does not seem to be ischemia,[48,49] but rather direct nerve compression. Besides, the incidence of false-positive arteriograms in the normal population is unknown. Compression of the AXN may alternatively be due to osseous[50] or soft-tissue tumors; thus,

X-rays as well as CT or MRI scans may be of help if such pathology is suspected. MRI may also show teres minor atrophy.[51]

Treatment of AXN entrapment

Conservative treatment should be tried,[46,49] insisting on avoidance of activities in abduction and external rotation. Should this be insufficient, exploration of the quadrilateral space and release of the adhesions around the neurovascular pedicle are performed through a posterior approach. Detachment of the posterior deltoid from the scapular spine is a part of the approach for some[46] and an avoidable option for others.[48] Fibrous bands encircling the pedicle are divided.[47–49] Partial or total sectioning of the teres minor tendon may be needed to adequately liberate the pedicle, but some feel[48] that this is not indispensable. At the end of the operation, axillary fat is encountered, and the surgeon must be able to easily pass a finger through the quadrilateral space into the axilla.[48] Postoperatively, pain is alleviated, and frank deltoid weakness, if present preoperatively, is promptly reversed, as the distance between compression site and muscle fibers is short.

Musculocutaneous nerve entrapment

The musculocutaneous nerve (MCN) contains fibers from the C5 and C6 roots, running through the upper trunk and lateral cord. Arising from the latter at the level of the inferior border of the pectoralis major, it pierces the coracobrachialis muscle after supplying a branch to it. It then comes to lie between the biceps and brachialis muscles, supplying both. At the elbow, the nerve contains only sensory fibers, and arises between the brachialis muscle and the biceps tendon or fascia. Becoming the lateral antebrachial cutaneous nerve, it pierces the antebrachial fascia and supplies two branches, which innervate the anterolateral and posterolateral forearm skin.

In this course, the MCN is fixed as it passes through the coracobrachialis. Another vulnerable site is the exit point between the brachialis muscle and the biceps tendon, where entrapment produces purely sensory symptoms and will not be addressed here.

Communication between the MCN and the median nerve may exist and convey fibers from the former to the latter.[52]

MCN lesions are mainly met in the context of a brachial plexus lesion. In some isolated lesions (e.g., after shoulder dislocations or prolonged arm positioning in abduction, extension and external rotation during surgery[53]) nerve stretching is the most plausible etiologic factor. In some others (e.g., after violent elbow hyperextension against resistance[53] or after strenuous muscular activity[54,55]), the presumed mechanism is compression of the MCN by coracobrachialis contraction,[13] but nerve stretching distal to this fixation point may also play a role under these circumstances.[13] Lesions produced during loss of consciousness[53] or after carrying a heavy object[56] result from external compression. Finally, iatrogenic MCN lesions may be provoked by arthroscopy or open surgery of the shoulder. From all these types, MCN palsy after physical activity presents a profile closer to nerve entrapment.

Symptoms and signs of MCN entrapment

As the presumed site of entrapment is the coracobrachialis muscle, this results in both sensory and motor signs. Elbow flexion and supination are weak; however, they are never completely abolished, even in total paralysis of the MCN, as the brachioradialis and the flexor–pronator group give some elbow flexion, and the supinator and brachioradialis muscles help in supination. The biceps reflex is weak or absent on the involved side. Hypesthesias and paresthesias appear in the lateral forearm skin. These deficits may be hard to detect,[57] as sensory overlapping exists.

Theoretically, in the case of communication between the MCN and median nerve distal to the coracobrachialis, a proximal MCN lesion may give signs in the territory of the median nerve.[52]

Electrophysiologic studies

Sensory NCS are performed on the lateral antebrachial cutaneous nerve. Motor NCS values are obtained by stimulation at the axilla or Erb's point and detection over the biceps muscle. Both are pathologic in MCN entrapment at the coracobrachialis level. EMG study of the biceps and brachialis muscles is also abnormal. Of interest (but technically difficult) might be an EMG study of the coracobrachialis, as this muscle is not expected to be involved in MCN entrapment[13,53] (its motor supply arises proximal to the muscle belly). In contrast, MCN stretching proximal to its passage through the coracobrachialis is expected to provoke detectable changes in needle examination of the coracobrachialis.

Differential diagnosis

A patient with MCN palsy occurring after strenuous physical activity has a characteristic history and findings on evaluation, and thus diagnosis can hardly be missed. In practice, only a biceps tendon rupture may present with a similar clinical setting: a complete one is easily diagnosed by the deformation provoked in the anterior arm musculature and a partial one by the pain triggered during palpation of the biceps tendon and the detection of a palpable

voluntary muscle contraction, despite pain. Neuralgic amyotrophy may show MCN palsy, but is rarely limited to this territory.[58]

Physical and electrophysiologic examination differentiate isolated MCN palsy from more generalized proximal lesions, such as stretching of the brachial plexus or cervical root pathology. Attention should be paid to territories corresponding to the C5–C6 roots, the upper trunk, and the lateral cord of the plexus, as lesions at these levels may be manifested by weakness of elbow flexion, besides other deficits.

Treatment of MCN entrapment

MCN proximal entrapment after physical activity seems to be a benign lesion, expected to recover when treated conservatively.[54,55] Some weeks to several months may be needed, depending on the degree of nerve damage.

Other MCN palsies produced by a mechanism of stretching or compression (extreme positioning of the arm, violent hyperextension of the arm and elbow, or shoulder dislocation) are also treated conservatively. Prognosis depends on the severity of initial nerve lesion, which in turn depends on the violence of the trauma: partial MCN palsies after minor traumatic incidents (e.g., elbow hyperextension), extreme upper extremity positioning, or prolonged external compression have a better chance of recovery[53,54,56,57] than complete paralysis after a major traumatic event. In the latter setting, when no recovery is detected by EMG studies within 6 months from injury, nerve grafting or neurotization techniques may restore some of the lost elbow flexion.

Radial nerve entrapment

The radial nerve (RDN) carries fibers mainly from the roots C6–C7–C8, running through all trunks, then through their posterior divisions and finally through the posterior cord. It passes anterior to the long head of the triceps and then runs obliquely slightly distal to the spiral groove, where a thin muscle layer from the medial head of triceps separates it from the humerus.

From the lower border of the axilla to the spiral groove, the RDN gives off branches to the triceps and anconeous muscles and the posterior cutaneous nerve of the arm (before entering the spiral groove), the posterior cutaneous nerve of the forearm, and the lower lateral cutaneous nerve of the arm. The latter two accompany the RDN in the spiral groove.

Emerging under the lateral head of the triceps, the RDN pierces the lateral intermuscular septum and enters the space between the brachialis and radial extensor muscles. This part of the RDN path, up to the passage through the supinator muscle, is known as the radial tunnel. Here, the RDN provides motor branches to part of the brachialis, the brachioradialis (BR), and the extensor carpi radialis longus (ECRL). At about the elbow level, it divides into a superficial sensory branch and the posterior interosseous nerve (PION). A considerable number of variations refer to the branching pattern of the RDN inside the radial tunnel.[59,60] At 2–10 mm proximal to its entry into the supinator muscle, the PION may be crossed by the medial edge of the extensor carpi radialis brevis (ECRB) origin.[59–62] It may also be crossed by a vascular leash from recurrent radial vessels.

The superficial sensory branch of the RDN innervates the dorsoradial half of the hand skin and the dorsal skin of the thumb and the proximal phalanges of the index and middle fingers.

The PION innervates the ECRB[59,60] and then swings around the radius, entering the supinator muscle, which it also innervates. The proximal border of the superficial layer of supinator muscle may have a tendinous or musculotendinous consistency[60] and is known as the arcade of Frohse. It has been classically considered as the distal boundary of the radial tunnel.[62] The PION finally exits under the distal border of the superficial layer of the supinator, which can also be tendinous or musculotendinous.[60,61]

At the level of emergence from the supinator, the PION gives off its terminal branches, sprouting from a common tassel.[63,64] The deep posterior forearm muscles – abductor pollicis longus (APL), extensor pollicis brevis (EPB), extensor pollicis longus (EPL), and extensor indicis proprius (EIP) – are mainly innervated by the descending branch of the PION.[63,64] Arising from the ulnar side of the tassel[63,64] are (a) recurrent branches innervating the proximal third of the extensor digitorum communis (EDC), mainly the extensors of the middle and ring fingers,[63] and (b) other branches innervating the EDC, extensor digiti minimi (EDM), and extensor carpi ulnaris (ECU). Finally, the PION gives some afferent innervation to the wrist joint.

Symptoms and signs of RDN entrapment

Motor deficits result from entrapment of either the RDN, before its division, or the PION:

Entrapment in the spiral groove under the lateral head of the triceps may cause acute (after intense muscular training)[65,66] or chronic compression.[67] The presence of a fibrous band originating from the lateral head of the triceps may contribute to the compression.[65,67] The triceps muscle is unaffected, but the radial wrist extensors and all posterior forearm muscles are deficient (Figure 24.3a). A slight sensory deficit over the dorsal skin of the first web space may be noted.[67] The triceps reflex is normal and the BR reflex is decreased or abolished. Entrapment here may also result from callus formation after a humeral fracture.[68]

Entrapment proximal to the above site can occur at the distal border of the teres major muscle[69] or from a fibrous band of the medial head of the triceps,[70] in combination with muscular overtraining. In compression reported during the windmill pitching motion in softball,[71] traction of the

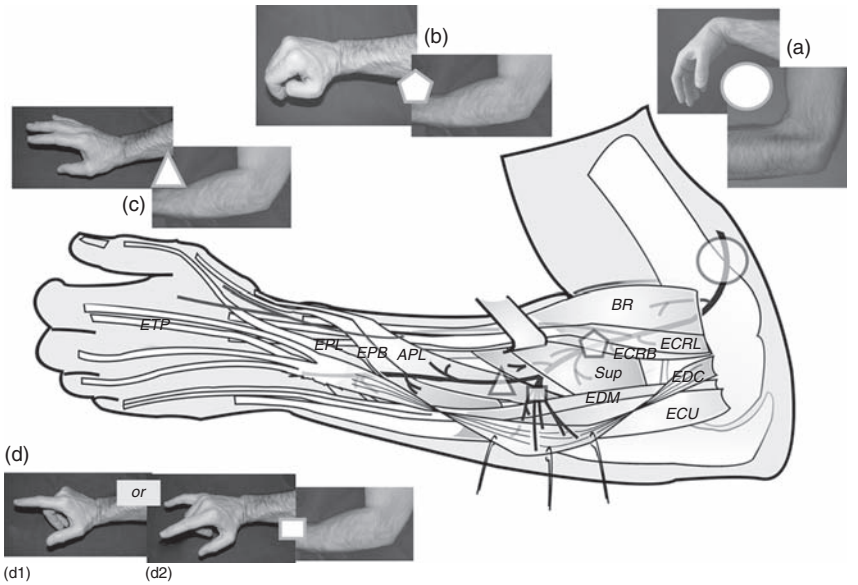


Figure 24.3

Entrapment of the radial nerve in the lower arm and forearm. The extensor digitorum communis (EDC) muscle has been raised, to uncover the deep dorsal forearm muscles. (a) (circle) Proximal to the radial tunnel. (b) (pentagon) At the entrance to the arcade of Frohse. (c) (triangle) At the exit from the arcade of Frohse, compression of the descending branch. (d) (rectangle) At the exit from the arcade of Frohse, compression of the transverse branches.

RDN plays a role. The triceps may be partly affected[69] and a sensory loss may be observed over the posterior arm and forearm.[72] The deltoid muscle is normal.

Entrapment inside the radial tunnel may be produced by compression from vascular leashes, the fibrous medial border of the ECRB, the arcade of Frohse, and tumors[73,74] and other space-occupying lesions.[75–77] Two types of clinical involvement may ensue from compression at this level:

1. Motor deficits are evident. Compression results from space-occupying lesions[64,73,75–77] or from a fascial thickening of the arcade of Frohse.[64] When compression is distal to the RDN division into superficial and deep branches, the BR, the ECRL, and probably the ECRB are spared, sensory involvement is absent, and the triceps and BR reflexes are normal ('PION palsy'). Finger extension and thumb extension–abduction are affected, but extension of the wrist is possible, with concomitant radial deviation, under the influence of the active ECRL, as the ECU is paralyzed (Figures 24.3(b) and 24.4). Once full-blown, this is a painless situation. Onset of palsy may be preceded by a period of intense use of the forearm.[78]
2. Pain in the lateral elbow area is the principal symptom,[79] with exacerbation during repetitive forearm rotation or elbow extension, sometimes with nocturnal flare ('radial tunnel syndrome' or 'resistant tennis elbow'). Tenderness is found during palpation of the radial tunnel. Pain is elicited or aggravated by resisted supination with the elbow extended (compression at the arcade of Frohse), by passive pronation with the wrist flexed (compression at the arcade of Frohse and/or by the ECRB medial edge), or by resisted extension of the middle finger (compression by the ECRB, contracting to stabilize the wrist).[62]

Entrapment at the exit from the supinator, where the PION starts to divide, may result in some pattern of partial PION palsy.[80] Lesions of all branches to the EDC or just the recurrent ones (Figure 24.3d) produces the 'sign of the horns' (drop middle and ring fingers, extension of little and index possible, extension and abduction of the thumb possible).[63] In lesions of all branches to the EDC and the EDM, a drop little finger is added[64] (Figure 24.3d1). Lesions of the main descending branch produce a drop thumb pattern,[64,81] probably with some weakness in index extension (Figure 24.3c). Tenderness to palpation at the exit of the PION from the supinator may be found.

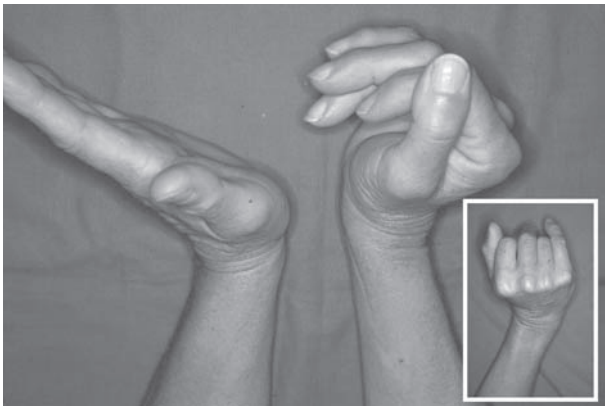


Figure 24.4

Posterior interosseous nerve palsy on the right: drop thumb, index, and finger. Radial wrist extensors are spared and deviate the wrist radially on extension (inset).

Electrophysiologic studies

They help detect the level of lesion and the presence of conduction block or axonal loss. They are valuable in the differential diagnosis and prognosis of the lesion. In acute

palsies, no definite information can be gathered as to the type of nerve lesion (neurapraxia, axonotmesis) before 3 weeks have passed from the onset. When performed with a delay of 2–3 months, electrophysiologic studies offer more solid evidence as to the chances of spontaneous resolution.[82] Besides, they can be extended into other nerve territories (including those with the same root supply) should the patient's history and clinical setting be not compatible with RDN entrapment.

Differential diagnosis

X-rays, ultrasound, and CT and MRI scans may help in detecting nearby tumors or non-tumorous conditions[73,77] and spinal column or central nervous system pathology.

Lesions of the C7 root or the posterior cord should be differentiated from RDN palsy by thoroughly evaluating other nerve territories. Partial PION palsies must be differentiated from tendon ruptures, for example in a rheumatoid arthritis context (where a PION palsy may also occur): the tenodesis effect will help in this. A drop ring and little finger may simulate ulnar claw, but there is no active extension of the metacarpophalangeal joints of these fingers.

Radial tunnel syndrome must be differentiated from lateral epicondylitis of the elbow. Apart from the slight difference in the localization of pain, a lidocaine injection into the radial tunnel produces finger drop and pain relief in patients with radial tunnel syndrome.

Lead intoxication, neuralgic amyotrophy,[58] hereditary neuropathy with liability to pressure palsies (HNLPP) and 'hourglass fascicular nerve compression'[83–85] may all mimic RDN palsy. HNLPP is a deficit in the myelin sheath with an autosomal dominant type of transmission, rendering peripheral nerves prone to demyelinization at usual compression sites. In 'hourglass fascicular nerve compression', an intraneural fascicular torsion of unknown etiology takes place. The radial and median nerves are the usual sites of involvement. The lesion is visible by peroperative inspection.

Treatment of RDN entrapment

Is it an acute or a chronic palsy? What is the time passed between palsy onset and presentation? What is the level of nerve compression? Is there any evidence for space-occupying lesions compressing the nerve? Answering these questions helps substantially in defining a therapeutic attitude towards RDN palsy.[82]

Acute palsies at the spiral groove level following muscular overuse are treated conservatively for about 3 months. Passive motion is preserved by splinting the wrist passively and the metacarpophalangeal joints dynamically in extension; thus, extension of the proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints is performed by the intrinsic hand muscles. Exploration is indicated if there is no evidence of recovery. The nerve is first approached into the interval between the brachialis and the BR and then followed proximally. The lateral intermuscular septum is incised, the lateral head of the triceps is released, and any accessory head or fibrous band is excised. If needed, the approach is extensible proximally, after release of the long head of the triceps.

Treatment of the acute PION palsy begins also with a 2- to 3-month period of observation, splinting and physiotherapy.[62,82] If there is no evidence of recovery at the end of that period, exploration should follow, performed by an anterior approach (between the BR and the brachialis: Figure 24.5), a posterior approach (between the radial wrist extensors and the finger extensors), or an approach through the radial extensors (e.g., between the BR and the ECRL). The approach through the BR–ECRL interval offers a good exposure of the proximal radial tunnel;[62,82] combined with the posterior approach,[62] it gives sufficient access to the exit from the supinator. All potential causes of compression should be dealt with.

For RDN compressions provoked by a mass, operative treatment should be the first choice.[82] An RDN palsy from exacerbation of elbow rheumatoid arthritis has been treated medically.[86] However, surgical treatment is advanced by some authors,[87,88] as this may avoid loss of time.

In radial tunnel syndrome, non-operative treatment is offered first. Apart from forearm splinting in supination, with the wrist in extension, and avoidance of physical activity that triggers the symptoms, non-steroidal anti-inflammatory medication may be administered. When surgical treatment is selected, epicondylitis should be dealt with, to maximize the chances of good outcome.[89] Disinsertion ECRL and ECRB act by denervating the lateral epicondylar area.[90] Disinsertion of the anterior and lateral parts of the supinator muscle origin contributes to denervation of the

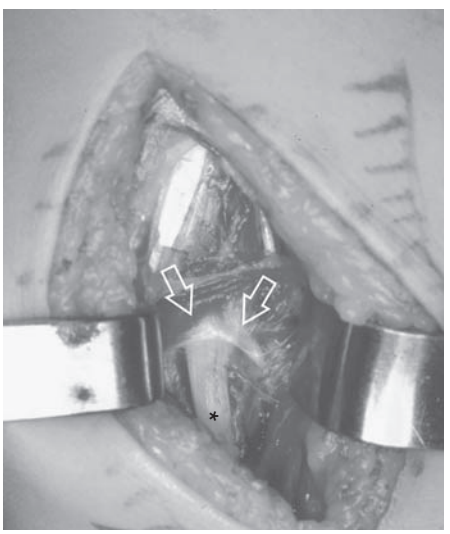


Figure 24.5

The entrance to the arcade of Frohse. The asterisk indicates the radial nerve and the arrows indicate the proximal tendinous edge of the supinator muscle.

lateral epicondyle and concomitant relaxation of the arcade of Frohse.[90]

Tendon transfers are seldom used in RDN palsy caused by entrapment, as at least incomplete and usually complete recovery will follow after observation or exploration. The problem presents sometimes for longstanding compressions with insidious progress to paralysis and more rarely in high RDN palsy after muscular effort[67] or in PION palsy. The reader is referred to other chapters of this book for a detailed discussion of reconstruction options after irreversible RDN palsy.

Another option is nerve transfer, according to which redundant median nerve motor branches (e.g., to the palmaris longus or to the flexor digitorum superficialis) are used to re-innervate the distal part of the paralyzed RDN.[91] Candidates are patients with non-recovering high RDN palsy, in whom permanent loss of motor endplate function has not yet occurred but for whom nerve grafting is not a choice, either because of a prolonged delay or because of the extent of the RDN lesion.[91]

Ulnar nerve entrapment

The ulnar nerve (ULN) contains nerve fibers from the C8–T1 roots, traveling successively through the lower trunk, its anterior division, and the medial cord. About the middle of the arm, it pierces the medial intermuscular septum and passes posterior to it, where it lies anterior to the medial head of the triceps muscle. Here, a fascia may joint the medial head of triceps and the intermuscular septum,[62] forming the arcade of Struthers.

At elbow level, the nerve runs inside the retroepicondylar groove, to then enter the cubital tunnel, the roof of which is formed by the ligament of Osborne proximally and the fascia covering the flexor carpi ulnaris (FCU) muscle. At the entrance to the cubital tunnel are given off branches to the FCU. There follow branches to the ulnar half of the flexor digitorum profundus (FDP), given off inside the cubital tunnel. The ULN then pierces the flexor/pronator aponeurosis to continue traveling in the forearm down to the wrist.

At several centimeters proximal to the wrist, the ULN gives off a dorsal cutaneous branch, which supplies the skin of the dorso-ulnar surface of the hand and the dorsal skin of the proximal phalanges of digits 4 and 5. A palmar cutaneous branch may also be given off. The ULN then enters Guyon's canal, passing deep to the volar carpal ligament and between the pisiform and the hook of the hamate. Here, it bifurcates into a superficial and a deep branch.

The superficial branch innervates the palmaris brevis muscle and supplies collateral nerves to digit 5 and the ulnar half of digit 4. The deep branch pierces the pisohamate ligament, and, on exiting from the canal, it plunges into the hypothenar musculature, turning around the hook of the hamate. It passes between the origins of the abductor digiti minimi (AbDM) and the flexor digiti minimi muscles, to then penetrate the opponens digiti minimi[62,92] and be found deep to the flexor tendons. It gives off branches to the hypothenar muscles and also to the two medial lumbricals, all interossei (including first dorsal interosseous, 1st DI), the adductor pollicis (AdP) and the deep head of the flexor pollicis brevis. Variations on this innervation (e.g., all-ulnar innervated thenar muscles) exist.

ULN compression at elbow level is provoked dynamically in elbow flexion, when tension is imposed on the elements forming the roof of the cubital tunnel. Besides, the ULN becomes taut over the medial epicondyle. Shoulder and wrist position may augment stretching.[93] The exact roles of nerve excursion, extrinsic compression, and stretching are not defined, but they may act in synergy. Compression of the ULN by flexor/pronator aponeurosis might occur,[94,95] as well as compression by the arcade of Struthers.[96] Modifications in local anatomy may predispose to this compression:[62,97] cubitus valgus, callus formation, heterotopic ossification, vestigial muscles (anconeus epitrochlearis: Figure 24.6),[98] a prominent medial head of the triceps,[98,99] subluxation of the ULN[99,100] (Figure 24.7),

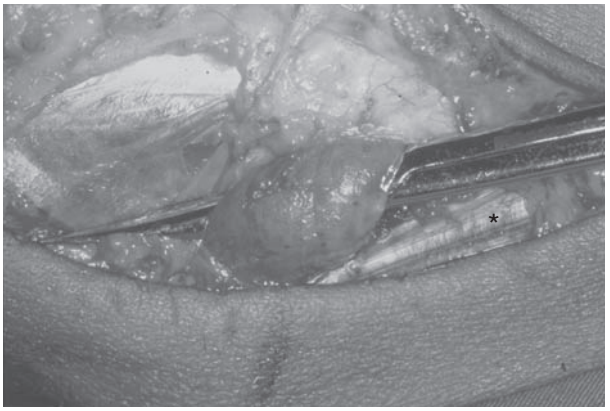


Figure 24.6

The anconeus epitrochlearis muscle. The asterisk is on the triceps tendon.

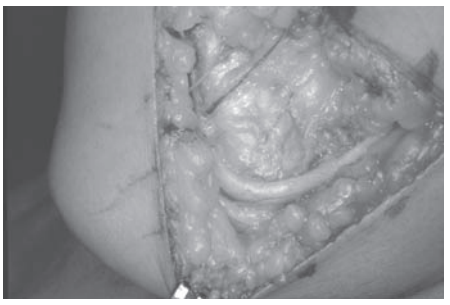


Figure 24.7

Snapping and anterior subluxation of the ulnar nerve around the medial epicondyle, on flexion of the elbow. The humerus is up; the forearm is on the left.

ganglia,[101] synovial osteochondromatosis,[102] tumors, metabolic diseases, rheumatoid arthritis of the elbow, etc.

At wrist level[92,103] or in the palm, ULN entrapment results from aberrations in local anatomy (anomalous muscles,[104,105] fibrous bands,[106] and piso-hamate coalitions[107]), fractures,[108–110] and tumors and other space-occupying lesions,[111–118] as well as from external pressure (e.g., in bicycle riders[119]). The following zones of compression can be defined here[62,103] (Figure 24.8): In *zone 1*, extending up to the bifurcation of the ULN, both deep and superficial branches are affected. In *zone 2* (extending up to the emergence of the hypothenar branches from the deep branch), compressions affect globally the deep motor branch of the ULN, and in *zone 3*, only the superficial branch is influenced. Zones 2 and 3 are superposed in the distal half of Guyon's canal; thus, compression at this level (e.g., by a fracture of the hook of the hamate) may produce mixed deficits. Two further zones have been recognized[92,119] to describe compressions in the palm: *zone 4* extends up to the emergence of the branches to interossei and lumbricals; *zone 5* consists of the last part of the motor branch, from where branches to the 1st DI and AdP emerge. Thus, compression in zone 4 spares the hypothenar muscles, but provokes clawing; compression in zone 5 influences only the ulnar-innervated thenar muscles, and clawing is absent. Aberrations from the above scheme do exist.[106,120]

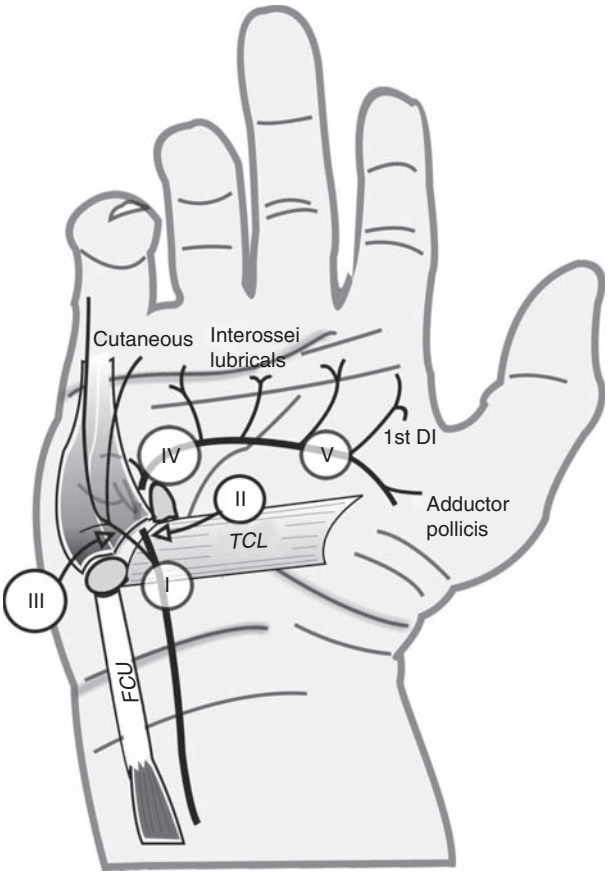


Figure 24.8

Zones of compression of the ulnar nerve at the wrist and in the palm. The palmar carpal ligament is not shown.

Symptoms and signs of ULN entrapment

Compression at elbow level

This produces numbness and paresthesias in the hand, with ulnar 1½ finger distribution, and also in the ulnar dorsal and palmar hand skin. These symptoms may be intermittent, triggered by elbow flexion and appearing only at night. In severe or prolonged compressions, permanent sensory loss ensues. Pain localized medially in the elbow or radiating down the hand may concurrently appear. Tinel's sign may be vividly elicited at the elbow. Sensory symptoms are reproduced with sustained elbow flexion, especially when combined with application of local pressure.[121]

Motor involvement, when present, concerns the intrinsic hand muscles and, less severely,[122] the FCU or FDP to the ring and little fingers. The FCU is spared more frequently than the FDP,[97] as branches to the former are given off just at the nerve's entrance to the cubital tunnel. The internal topography of the ULN at the level of compression may also play a role.[123] Clawing of digits 4 and 5 (Figure 24.9), and weakness in index and little finger abduction should be sought. A positive Froment's sign (switching from lateral to terminal pinch when trying to keep a sheet of paper between thumb and index against resistance) is more frequent[124] than a positive Wartenberg's sign (permanent abduction of the little finger under the influence of the EDC and EDM, unopposed by the paralyzed third palmar interosseous muscle). A positive crossing finger test[125] (inability to actively put the middle over the index finger) may also detect intrinsic palsy. Motor involvement typically affects the 1st DI more than the AbDM,[122] due to the spatial arrangement of fascicles within the ULN at elbow level. In advanced motor involvement, muscle atrophy may be visible in the intermetacarpal spaces. Motor involvement

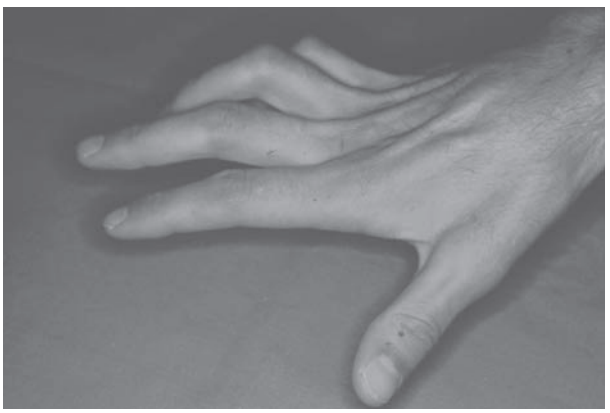


Figure 24.9

Ulnar clawing. Note the atrophy of the interosseous muscles (deepened intermetacarpal web spaces).

provokes a feeling of clumsiness in use of the hand and loss of force, especially in thumb–index pinch.

Among the classifications used in evaluating the extent of ulnar neuropathy, the one proposed by McGowan[126] has been widely accepted (Table 24.1). Dividing grade II into IIA and IIB is important in anticipating recovery.[127]

Compression at the level of Guyon's canal or more distally

1. Forearm muscles and dorsal and palmar cutaneous branches are spared.
2. The elbow flexion test is negative.
3. A positive Tinel's sign may be produced at wrist level.

Local signs may point to the presence of a mass at the wrist or distally. Hand involvement depends on the exact site of compression, as described above (zones). Clawing, when present, may be more evident, as the FDP to the ring and little fingers is unaffected.

Electrophysiologic studies

In motor conduction studies, recording is performed from the ADM or from the 1st DI. In sensory conduction studies, recording is performed antidromically from the little finger. Stimulation is performed at different levels (above and below the elbow). ULN conduction across the elbow is better measured with the elbow flexed, as in extension the nerve is slack and thus errors interfere in the estimation of the length across which latency and velocity are measured.[97] Conduction of the dorsal cutaneous branch of the ULN is also evaluated. Short-segment incremental measurements may sometimes disclose the precise level of compression in a wider anatomic region.[128,129] EMG studies are combined with the above to define the level of compression, its severity, and its duration.[97] A Murtin–Gruber anastomosis may be present, changing the interpretation of the findings.[97]

Table 24.1 *Grading of ulnar neuropathy according to McGowan,[126] as modified by Goldberg et al[127]*

Grade I:	Symptomatology positive, no objective findings
Grade II:	Objective decrease in sensory and motor function
IIA:	Intrinsic strength graded 4, no intrinsic atrophy
IIB:	Intrinsic strength graded 3, slight intrinsic atrophy
Grade III:	Paralysis of intrinsics, profound sensory deficit, advanced intrinsic atrophy

Differential diagnosis

X-rays and CT and MRI scans may show local bone and soft tissue pathology. MRI may even show signal abnormalities inside the ULN at the elbow, confirming compression at that level.

Clinical evaluation and electrophysiology help differentiate ULN compression from compression of the C8–T1 roots, the lower trunk, or the medial cord. For example, in preganglionic C8–T1 root compression, sensory conduction studies on the ULN are normal and sampling of C8- or T1-innervated muscles outside the ULN territory (abductor pollicis brevis, FPL, EDC, EIP, and EPB) shows denervation.

Central nervous system disorders such as cerebral stroke and amyotrophic lateral sclerosis may mimic palsy from ULN compression at the elbow, but the clinical setting, signs, findings, electrophysiology, and evolution are different, and differential diagnosis should be straightforward.

Treatment of ULN entrapment

ULN entrapment at the elbow

Any compression by abnormal anatomy or mass should be treated surgically. Otherwise, treatment depends on the extent of the neuropathy (using McGowan's grading or similar) and the response to conservative treatment.

For patients with intermittent sensory symptoms and no objective sensory or motor deficits, avoidance of keeping the elbow flexed for lengthy intervals and night splinting in extension may provide steady and durable relief from symptoms.[130,131]

Candidates for surgery are patients with objective sensory or motor deficits, those whose EMG studies show significant denervation, and those with grade I ULN compression whose symptoms are recalcitrant to conservative treatment.[132] Techniques of ULN decompression are simple in situ decompression, decompression with anterior transposition and decompression with some form of medial epicondylectomy.

In simple in situ decompression, the ligament of Osborne and the deep and superficial aponeurosis of the FCU are sectioned. Endoscopic methods[133] and mini-open approaches[134] have also been described. Proponents argue in support of this method as being minimally invasive and non-devascularizing. To some,[135] when compression is limited to the cubital tunnel, a portion of the fascia overlying the ULN and situated proximal to the ligament of Osborne may be left intact, assuring stability of the nerve in the retroepicondylar groove. Simple decompression treats that part of ULN entrapment caused by compression in the cubital tunnel and not so much the one due to strain-elongation of the nerve in the position of elbow flexion.

Decompression combined with anterior transposition additionally relieves the nerve from strain and/or subluxation over the medial epicondyle in elbow flexion; it also treats any

compression by mass or abnormal bone anatomy (e.g., cubitus valgus). When transposition is chosen, attention should be paid to some details: (a) branches of the ULN to forearm muscles should be saved; (b) the medial intermuscular septum should be excised, as it can otherwise provoke kinking of the ULN; (c) distal release of the ULN into the cubital tunnel should be performed by opening the flexor–pronator aponeurosis. Three variants of anterior transposition exist: *subcutaneous*, *intramuscular*, and *submuscular*. In subcutaneous transposition, posterior re-displacement of the ULN in elbow extension is prevented with the help of a fascial or fasciodermal sling.[136,137] In intramuscular and submuscular techniques, the ULN is placed into a well-vascularized bed.

In *decompression with medial epicondylectomy,* the cubital tunnel is opened, and strain to the nerve during elbow flexion is relieved by osteotomy of the medial epicondyle.[138] This technique was initially proposed in ULN entrapment secondary to post-traumatic osseous abnormalities at the elbow.[139] Better results may be achieved with this technique, in terms of patient satisfaction,[140] compared with anterior transposition. However, when an overzealous osteotomy is performed, the following risks exist:[141,142] (a) loss of the medial osseous contact point of the elbow may provoke irritation and traumatism of the nerve; (b) patients may be aware of anterior subluxation of the ULN in elbow flexion; (c) weakness of the flexor/pronator group may result; (d) valgus elbow instability may ensue. Thus, some form of partial osteotomy was proposed instead.[141–145] With a frontal osteotomy[142] (Figure 24.10), a clearance of some 8 mm is achieved for the ULN, which assures less tension in elbow flexion.

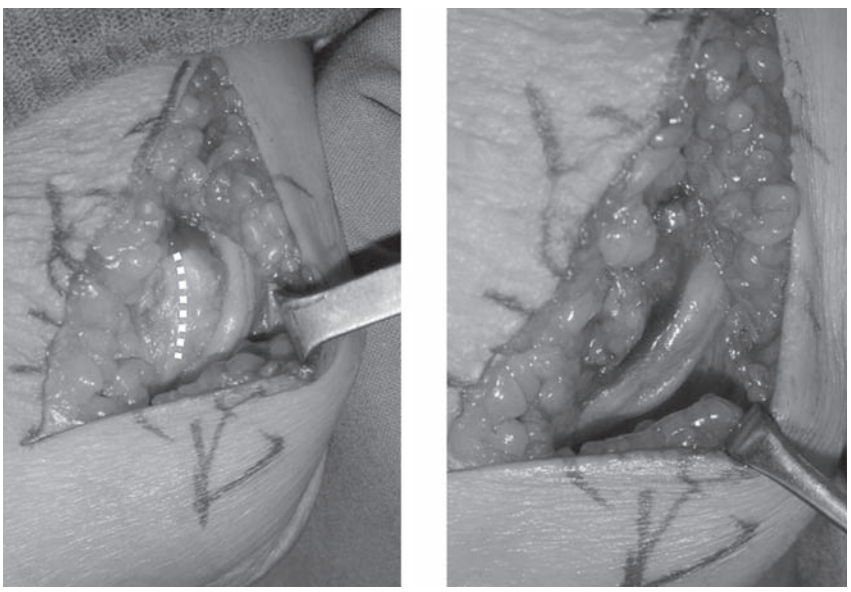
Actually, the choice of technique depends largely on the surgeon's preference, as prospective randomized clinical trials are lacking.[146] Nonetheless, the chance for complete recovery is always inversely related to the preoperative neuropathy grade.[145,147] Some conclusions might be drawn from meta-analysis:[146,148]

1. For minimal compression, non-operative treatment is successful in more than 50% of patients. Surgery, according to Dellon,[146] yields excellent results in more than 90% of patients, independently of technique. Mowlavi et al[148] found slightly better outcomes with medial epicondylectomy.
2. For moderate degrees of compression, submuscular transposition offers better results and has the least recurrence rate. Non-operative treatment has no place here.
3. For severe compression, according to Dellon,[146] all surgical techniques yielded excellent results in only up to 50% of patients for sensory recovery, and up to 25% of patients for motor recovery, and were followed by a recurrence rate of 30%. Subcutaneous transposition performed better. For the treatment of recurrences, submuscular transposition with neurolysis gave better results, while producing fewer recurrences. According to Mowlavi et al,[148] in situ decompression performed better.

Main complications after surgical treatment of ULN entrapment at the elbow are hematomas, neuroma formation from branches of the medial cutaneous nerve of the forearm, ULN instability, and recurrence of compression. The latter may be the result of inadequate decompression, scarring in the bed of the ULN, or creation of an iatrogenic compression.[62]

ULN entrapment at the wrist or more distal

When external compression is the cause, change of the occupational or recreational habits may bring improvement.



(a) (b)

Figure 24.10

Technique of partial epicondylectomy in the frontal plane.[142] (a) The dotted line represents the plane of the osteotomy. (b) After osteotomy, there is space for the ulnar nerve in elbow flexion. The humerus is up; the forearm is on the left.

When a mass is at the origin of the compression, surgical treatment is indicated. The problem of low ULN palsy after a displaced fracture of the distal radial metaphysis is a special case: when the palsy is concomitant with the fracture, accurate reduction may promptly reverse the deficit.[149] Otherwise, as well as in case of a tardy ULN palsy, exploration of Guyon's canal is indicated.[108,149]

ULN exploration at the wrist is performed through a longitudinal forearm incision crossing the wrist crease in a zig-zag fashion and continuing in the palm between the pisiform and the hook of the hamate. The volar carpal ligament is opened and the canal is inspected for any masses. Decompression of the deeper and more radially situated carpal tunnel can be performed, if needed. The motor branch of the ULN is followed as it plunges deep into the palm, by incising the pisohamate ligament and opening the fibrous arch of the hypothenar muscles. A different approach may be needed when entrapment is situated more distally, in zone 4 or 5.

Recovery depends on the severity and duration of compression, but motor deficits are more easily reversible than in ULN compression at the elbow, as the lesion is closer to the end-organs.

Median nerve entrapment

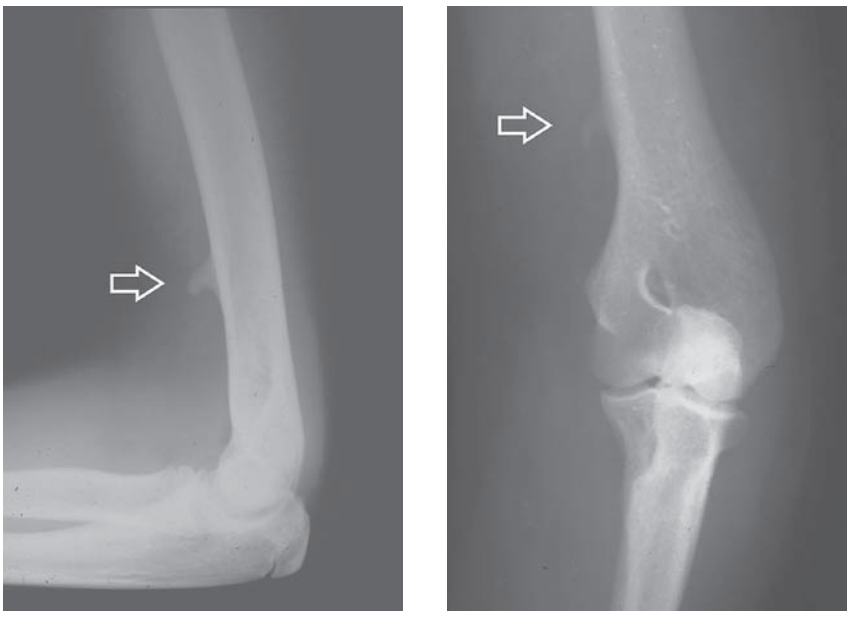
The median nerve (MDN) contains fibers mainly from the C6–T1 roots. The C6–C7 fibers are driven through the upper and middle trunk and then through the lateral cord; the C8–T1 fibers run through the lower trunk and the medial cord.

The MDN in the arm stays ventral to the medial intermuscular septum, and when a supracondylar process (Figure 24.11) or a ligament of Struthers is present, it may pass between these and the humerus. At elbow level, it runs deep to the lacertus fibrosus (LF) and gives off its first branches, destined to the pronator teres (PT) muscle, and then those to the flexor carpi radialis (FCR), the palmaris longus (PL), and the flexor digitorum superficialis (FDS). Usually, these latter branches arise under the humeral head of the PT, along with a second (distal) branch to the PT.[150]

The MDN usually runs between the humeral and ulnar head of the PT and then under the fibrous arch of the FDS, where it is closely related anatomically to its heads for the middle and ring fingers.[151] Approximately at this level,[152–154] it gives off the anterior interosseous nerve (AION).

The AION runs deep to the fibrous arch of the FDS, radial to the MDN, through the same orifice as the MDN or through a separate one. It supplies the flexor pollicis longus (FPL) muscle, the two most radial heads of the flexor digitorum profundus ($FDP_{II/III}$), and the pronator quadratus (PQ). It finishes in the wrist by supplying branches to the radiocarpal, distal radioulnar, midcarpal, and carpometacarpal joints.

The MDN follows a course under the radial border of the FDS muscle belly, supplying the latter with smaller motor branches. In the distal third of the forearm, it gives off a palmar cutaneous branch (PCB-MDN), which supplies the skin of the thenar eminence. At wrist level, the MDN enters the carpal tunnel, at the distal end of which it is divided into its terminal sensitive branches for the palmar surface and the dorsal aspect of the middle and terminal phalanges of the $3\frac{1}{2}$ radial digits. Motor branches supply the two radial lubricals. A recurrent motor branch is also given off, usually at the distal border of the carpal tunnel, to supply the abductor pollicis brevis (APB), the opponens pollicis (OP) and the superficial head of the flexor pollicis brevis (FPB) muscle.



(a) (b)

Figure 24.11(a,b)
Supracondylar process of Struthers.

The PT, FCR, and PL are innervated mainly by fibers from the C6–C7 roots, whereas the FPL, FDS, $FDP_{II/III}$, PQ, and the median-innervated thenar muscles receive fibers from C8–T1.[155,156] C6–C7 sensory fibers supply the thenar eminence, the thumb, and the index, and middle fingers, and C8 fibers supply the radial half of the ring finger.[155]

Motor deficits usually arise from entrapment at the following levels.

In the arm, tumors, vascular abnormalities, and the Struthers' process or ligament may compress the nerve.

At the elbow and in the proximal forearm, the MDN may be compressed as it traverses the anatomic passages of this area (LF, PT, and the fibrous arch of the FDS). Anatomic aberrations may predispose to this (a double LF,[157] passage deep to both heads of the PT, a high origin of the humeral head of PT, a fibrous deep head of the PT, various fibrous bands, an accessory head of the FPL, a voluminous bicipital bursa, vascular leashes, tumors, etc.). Compression of the AION may occur by essentially the same causes, either inside the MDN (as fibers destined to form the AION are individualized well proximal to the elbow[158]) or just after the AION is given off (as the two nerves have a closely related course). Thus, one cannot predict the exact level of compression from the type of entrapment (MDN, an isolated AION).

At the wrist, MDN compression in the carpal tunnel is the most frequent entrapment syndrome of the upper extremity.[155] 'Idiopathic' MDN compression is more common. Secondary compression occurs in case of anomalous muscles in the tunnel, tumors, synovial hypertrophy (in rheumatoid arthritis, renal insufficiency on hemodialysis, etc.), acute displaced or old malunited fractures, vascular anomalies, endocrine diseases, etc.

Isolated compression of the recurrent motor branch of the MDN may rarely occur, due to the presence of a nearby ganglion,[114,159] a tumor,[160] anomalous muscles[161] or entrapment by the transverse carpal ligament (TCL) itself.[162]

Symptoms and signs of MDN entrapment

The pronator teres syndrome

This results from compression of the MDN at the elbow and the proximal third of the forearm. Symptoms appear mainly during repetitive pronation/supination of the forearm or flexion/extension of the elbow and disappear at night.[163] Paresthesias and hypesthesias appear in the hand with MDN distribution,[164] and may also involve the territory of the PCB-MDN. A deep aching pain is felt in the proximal forearm.[164] Subjective weakness and fatigability of the forearm muscles may also present. Symptoms arise insidiously and appear progressively.[151]

Digital compression over the proximal third of the forearm for 30 seconds may trigger pain and paresthesias.[164] A positive Tinel's sign here is frequent, but is not the rule.[163] Objective sensory or motor deficits are exceptional.[151] Repetition of active pronation and supination against resistance may trigger the symptomatology.[163]

Spinner's tests should be performed:[151] (a) active elbow flexion and forearm supination against resistance detects compression from the LF; (b) active forearm pronation against resistance may compress the MDN at its passage through the PT; (c) active flexion of the PIP joint of the middle finger against resistance may compress the MDN as it traverses the fibrous arcade of the FDS. A positive test is characterized by reproduction of pain and less frequently of sensory symptoms.

The anterior interosseous nerve syndrome

The AION syndrome is characterized by a motor deficit concerning all AION-innervated muscles (FPL, $FDP_{II/III}$, and PQ) or only some of them. There may be complete paralysis or muscle weakness.

The onset of motor deficits is frequently preceded by a short period of pain in the proximal forearm,[151] sometimes diffuse. Pain diminishes or disappears with the onset of deficits. Some patients report a history of unusual muscular contraction, lifting heavy weights,[154] falling asleep with the head over the forearm, or some contusion or hematoma in the area.[154] The patient at presentation may exactly describe the deficits, or may complain of weakness, clumsiness, and loss of dexterity in handwriting.[165] Typically, pain and sensory deficits are absent,[151] but deep palpation in the proximal forearm may trigger pain.[166] Seldom, AION compression presents with pain and only occult deficits,[167] overt paralyses appearing tardively.[165,168]

PQ deficit is felt as a weakness of pronation with the elbow flexed (neutralizing the action of PT). Deficit of FPL is manifested by weakness of flexion of the interphalangeal (IP) joint of the thumb. Deficit of $FDP_{II/III}$ provokes weakness of flexion of the DIP joints of the index and middle fingers. The type of thumb–index pinch is a clue to the diagnosis (Figure 24.12). Isolated deficit of the FPL or the $FDP_{II/III}$ may be present; sometimes, only the FDP_{II} is affected clinically. Possible explanations for these incomplete forms are variations in innervation of the FDP (by the ULN or AION),[154,169] compression of a branch of the AION,[154] and incomplete recovery and compression of the trunk of the AION affecting selectively some of its fibers.[169] In the presence of a Martin–Gruber anastomosis, AION palsy may result in intrinsic weakness.[151,170] Typically, other muscles of the flexor–pronator group are not affected and there is no sensory deficit.

Carpal tunnel syndrome

MDN entrapment in the carpal tunnel provokes pain in the wrist, sometimes radiating proximally and distally. Paresthesias are reported in the territory of the MDN in the hand except over the thenar (supplied by the PCB-MDN). Symptoms are intermittent in mild compressions, appearing during night sleep or some occupational activities. A positive Tinel's sign may be found over the TCL. The Falen's and reverse Falen's maneuvers (appearance of paresthesias after keeping the wrist in flexion or extension, respectively, for 30 seconds–2 minutes),[155] as well as application of

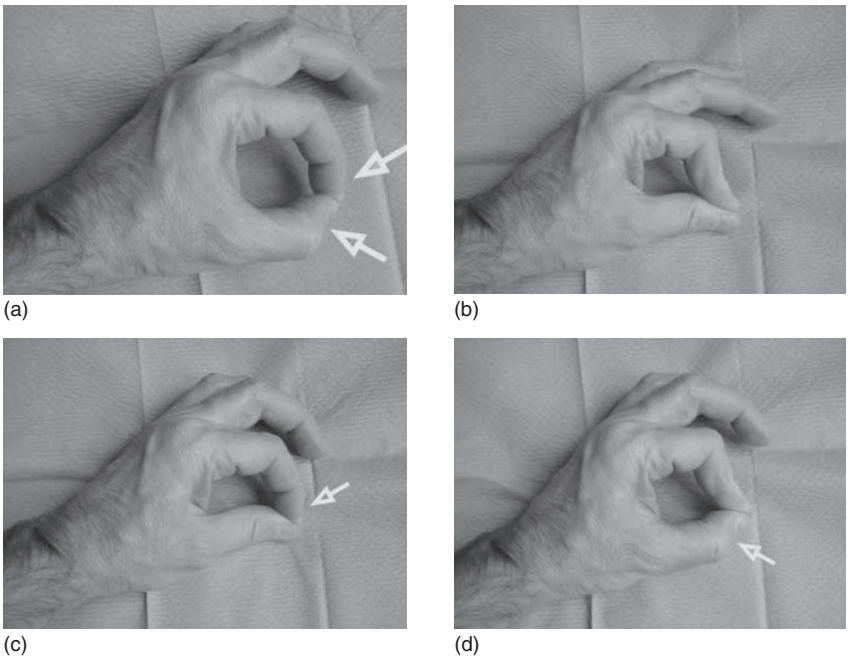


Figure 24.12

Types of paralysis from AION compression. (a) Normal hand. The FPL and the FDP to the index are functional. (b) Paralysis of the FPL and FDP to the index finger. (c) Paralysis of only the FPL. (d) Paralysis of only the FDP to the index finger. Note that the FDS to the index finger is intact.

pressure over the TCL, are the most widely applied provocative tests.

Constant hypesthesia as well as loss of dexterity, weakness in thumb abduction and opposition, and thenar muscle wasting are late signs, resulting from a severe or prolonged compression.[155]

In young patients with symptoms of carpal tunnel syndrome, an etiologic factor (e.g., a mass) should be sought.

Other types of MDN entrapment producing motor deficits

Entrapment of the MDN in the arm may be provoked by Stuthers' process or ligament or by other causes.[171] The clinical setting is similar to that of a pronator teres syndrome. Weakness of the flexor–pronator group may be present. Pain may be felt on palpation of the lesion in the arm, and symptoms may be triggered by extension of the elbow. A positive Tinel's sign may be elicited at the level of compression.

Isolated compression of the recurrent motor branch of the MDN gives muscle wasting and hand weakness. Abduction/opposition of the thumb is affected and sensory deficits are absent.

Electrophysiologic studies

In the pronator teres syndrome

The percentage of positive studies varies among series.[163,164,170] Performing the study after some cycles of repetitive motion may increase its sensitivity. Serial evaluations may be necessary.[168] Motor and sensory NCS in the proximal forearm may be affected,[172] while they stay within normal range across the carpal tunnel, except when a severe proximal compression has provoked axonal loss.

Findings on EMG studies may be more conclusive.[168,172] Needle EMG sampling of the APB may be positive,[170] and some muscles of the flexor–pronator group may also show involvement.[168] As some branches to the PT may be given off under its humeral head,[150,173] MDN compression at the PT level may influence the PT itself.[170,173]

In the anterior interosseous syndrome

Motor NCS are performed recording from the PQ muscle.[174] Sampling of the PQ, FPL or rarely FDP_{II} will show spontaneous activity and/or neurogenic recruitment patterns.

Findings in other MDN-innervated muscles not supplied by the AION may be normal. However, (a) in a setting of AION palsy, careful clinical evaluation or electrophysiology will sometimes reveal a more global involvement (sensory[167,175] or motor[157,170,171]); (b) a compression of the MDN proximal to the emergence of the AION may preferentially influence fibers destined to later form the AION;[157,171] and (c) the PT arch, the arch of the FDS, and the emergence of the AION from the MDN are found practically at the same level, with some individual variations.[152] Thus, entrapment at the level of the arches of the PT/FDS or proximally can produce AION, main trunk of the MDN, or combined involvement, clinically and/or electrophysiologically.

In the carpal tunnel syndrome

Motor and/or sensitive NCS across the carpal tunnel may be influenced. Needle EMG can detect denervation or reinnervation signs in the APB. Normal electrophysiologic studies do not exclude this syndrome. Incremental

NCS and internal comparison studies (between the MDN and ULN) are more sensitive in revealing subtle compression.[155]

Demyelination as well as axonal loss provoke a decrease of SNAP and CMAP amplitudes across the carpal tunnel. When stimulating distal to the carpal tunnel, potential amplitudes return towards normal levels in case of demyelination, as fibers demyelinated at the level of the carpal tunnel can still be stimulated distally;[155] on the contrary, in axonal loss, potential amplitudes remain low.

Isolated entrapment of the recurrent motor branch of the MDN will show pathologic NCS and needle EMG findings in the MDN-innervated thenar muscles (APB).

Differential diagnosis

Clinical evaluation and electrophysiology help in defining the level of compression (plexus root, plexus trunk, plexus cord, MDN trunk, or branches). Imaging studies may detect a structural lesion (cervical disk hernia, cervical spondylosis, supracondylar process, tumors, ganglia, anomalous muscles, etc.).

The AION syndrome has a particular differential diagnosis. Parsonage and Turner's neuralgic amyotrophy may involve deficits in the AION territory.[58] Examination of shoulder girdle and arm muscles is imperative. It should be kept in mind that no absolute criterion exists to differentiate the two entities and that neuralgic amyotrophy is, at present, of unknown etiology.

Besides, 'hourglass fascicular nerve compression'[176–179] can affect the trunk of the MDN. The lesions encountered are intra-epineurial, uni- or multilevel, they may be found proximal to the elbow, and may involve the fascicles destined to form the AION. Some fascicular torsion of mechanical or inflammatory etiology seems to play a role.

Rupture of the FPL tendon (e.g., in a rheumatoid patient) can mimic AION syndrome with isolated paralysis of the FPL. A simple test,[180] making use of the 'tenodesis effect' of the FPL tendon, can help in differential diagnosis. In borderline cases, MRI may be used to check tendon integrity.

Another pathology to consider in the differential diagnosis of AION is hereditary neuropathy with liability to pressure palsies (HNLPP).[181] History, electrophysiologic findings across other (probably silent) sites of compression, and sometimes histology will help in the diagnosis.

Treatment of MDN entrapment

Pronator teres syndrome

Some 50% of cases may recover with conservative treatment. Avoidance of any physical activity that triggers the symptoms, brief immobilization, local infiltration with a combination of an anesthetic and steroid drug, as well as oral administration of non-steroidal anti-inflammatory drugs (NSAIDs), may all be tried.

Surgical treatment is proposed where conservative treatment has failed and in the rare cases with overt sensory or motor deficits. Excellent or good results have been reported in some 75% of patients.[163,164] However, residual sensory symptoms or weakness of non-disabling character are frequently found among operatively treated patients.[163,164] For some authors, confirmatory electrophysiology is a positive prognostic factor for good results after surgical treatment.[168] Others find a trend towards better results in patients with normal preoperative NCS.[164] Finally, some consider the failures as resulting from misdiagnosis or inadequate decompression.[163]

Anterior interosseous nerve syndrome

Treatment of this syndrome is a very controversial issue. Some publications advance operative treatment,[151,154,182,183] while others favor a more conservative attitude.[174,184–186] Additionally, some authors advance the idea that an important percentage of these cases are forms of neuralgic amyotrophy.[174,184,187–189] Thus, waiting periods from as short as some weeks to as long as 16 months have been proposed. Somehow, the type of treatment also depends on the specialty of the physician (neurologist or surgeon) who proposes it.[188,189]

As no prospective controlled trial has been performed, one should try to differentiate between deficits resulting from mechanical compression and those resulting from neuritis, although no absolute clinical or electrophysiologic criteria exist. AION palsies where a mechanical factor has plausibly played a role can be treated with a short observation period (3–4 months) and then explored, if no recovery is documented clinically and/or electrophysiologically. In contrast, cases with no mechanical compression and with evidence of involvement of the shoulder girdle or other non-MDN innervated muscles should be observed for prolonged periods. For cases falling in between, no evidence-based treatment algorithm can be proposed at present. Risks from surgery (including poor aesthetic results) should be balanced against the risk of irreversible amyotrophy.

Although large series of patients are not available, results of operative treatment of the AION palsy seem gratifying. The time to recovery is very variable, possibly reflecting different grades of nerve lesion. Non-recovery is attributed to long-persisting compressions.

Exploration of the MDN and the AION in the elbow and proximal forearm is performed through a longitudinal, oblique or transverse incision (Figure 24.13). The LF is sectioned. Careful dissection, staying lateral to the MDN, will avoid harming its branches to the flexor–pronator mass.[168] The arch of the PT is widened to ease exploration of the MDN and AION; this is done by detaching the superficial head of the PT from the conjoined tendon of the two heads (and resuturing it at the end with lengthening) or by just cutting the most fibrous part of this muscle. Then the arch of the FDS is sectioned. Any fibrous bands or

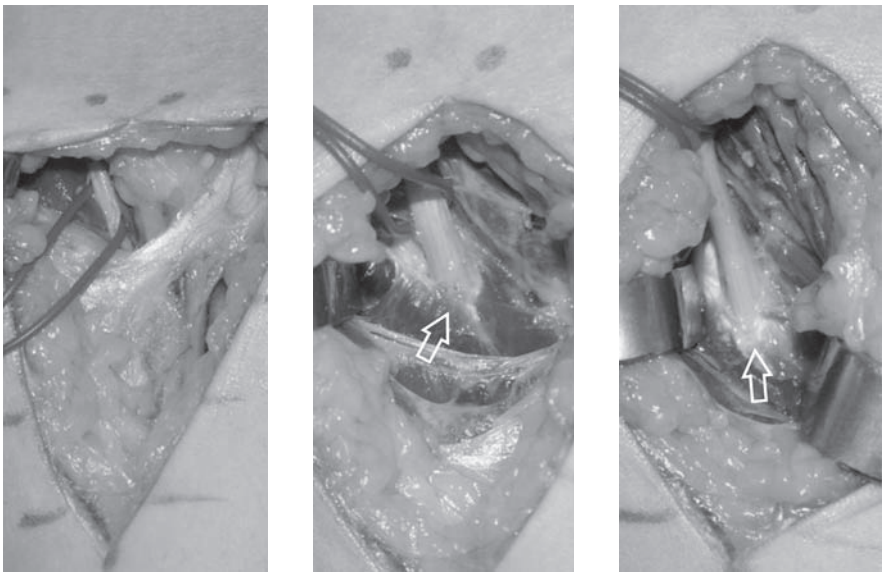


Figure 24.13

Decompression of the median nerve (MDN) at the elbow and in the proximal third of the forearm. (a) The lacertus fibrosus. (b) The MDN dips between the two heads of the pronator teres muscle. (c) The MDN is seen passing under the arcade of the FDS, after the passage between the two heads of the pronator teres has been enlarged.

vascular leashes found in the area are excised and the MDN and AION are followed as distally as possible, to ensure complete decompression. Sometimes, a pseudoneuroma is seen in one of these nerves. Through digital palpation or proximal extension of the skin incision, if needed, compression by a ligament of Struthers can be controlled.[164] The participation of 'hourglass fascicular nerve compression' in the etiology of the AION syndrome and other proximal MDN neuropathies cannot be estimated at present; thus, indications for exploration of the MDN in the arm and intra-epineurial decompression of constricted fascicles via perineurial incision are not presently well defined.[176-179]

Tendon transfer can substitute for lost function when there is no recovery after release, or as the first operative choice when paralysis has persisted for more than 12–15 months. Thumb function can be restored by transfer of the BR tendon or the tendon of the FDS_{IV} to the tendon of the FPL.[151,154,182] Active flexion of the DIP joint of the index finger can be restored by transfer of the distal part of the tendon of FDP_{II} to the tendon of the functioning FDP_{III} or FDP_{IV}[151,154] at wrist level. When paralysis affects the FDP_{II} and the FDP_{III}, the function of both might be restored by transfer of the ECRL tendon.[151]

Carpal tunnel syndrome

Conservative treatment of carpal tunnel syndrome comprises modifications in occupational or recreational activities as well as night splinting of the wrist.[155] Local infiltration of corticosteroids may bring transient or long-lasting remission.[155] Symptom relief after local steroid injection is a predictor of good outcome should surgery be offered later to these patients.[190] Oral administration of NSAIDs may also help.[155]

The criteria for surgical treatment of the carpal tunnel syndrome are clinical and electrophysiologic: a symptomatology recalcitrant to conservative measures or recurring after remission, thenar atrophy, hand weakness, and loss of dexterity,[78,155] permanent sensory deficits,[155] and axonal loss. Any compressive mass should also be treated surgically.

Operative decompression is performed with open or endoscopic techniques (monoportal or biportal). Although endoscopic methods seem to offer slightly faster convalescence and return to activity, there are only a few papers that have directly compared these methods in a prospective, randomized blind manner.[78] Issues such as cost[191] and learning curves should also be considered. Besides, particular contraindications exist to endoscopic release: any patient with a solid mass or synovitis (e.g., rheumatoid patients) or a previous operation in the area should be treated with open surgery. Complications (nerve and vascular lesions, reflex sympathetic dystrophy, and infection) occur at similar rates.

Relief from sensory complaints is generally speedier than recovery from motor involvement. As anywhere in the body, recovery after releasing the compression depends on the grade of the nerve lesion, the duration of compression, the distance between the level of compression and the target organs (which is here a few centimeters only), and the age of the patient. Subjective improvement, however, as judged by patient self-satisfaction, is high, even in longstanding cases with severe compression.[192,193]

When thenar atrophy is irreversible, a Camitz transfer might be tried[194] concurrently with surgical decompression, or secondarily. It consists of using a part of the palmar aponeurosis to lengthen the PL tendon and transfer it to the tendon of the APB, near its insertion. This transfer aims at restituting abduction of the first metacarpal and re-establishing thumb opposition (the AdP and the deep head of the FPB are functional, innervated by the ULN). Patients with thenar atrophy treated with decompression and transfer report increased performance of their hands in activities of daily living.

In case of isolated paralysis of the recurrent motor branch of the MDN, exploration is the choice, in order to excise any mass compressing the nerve or to release the nerve, when it runs through the TCL or around its distal edge.

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25 Sensory palsy

Göran Lundborg and Birgitta Rosén

Hand sensibility

The prime purposes of the sense of touch are protection and to give feedback to motor programs, but the tactile input is also a code to describe the character of the environment.[1] The richness in specific tactile information from the hand, in combination with the dynamic processing capacity of the brain, has made the human hand a delicate instrument with an enormous capacity to perceive, to execute, and to express – simultaneously, in the explorative act of touch.[2]

There is a continuous flow of impressions and experiences from the perceptual organs, which, after cognitive processing, gives us information about the world. Descartes described the hand as *'the outer brain'*, and the hand is indeed intelligent – it can learn, it can remember, it is creative, and it can improvise. The hand is strongly linked to the personality, and is projected in large areas of the cortical maps of the body.[3–6]

It takes all senses to fully perceive the world, and the interplay between the senses, especially between vision and touch, to facilitate perception is widely described.[7] The hand obtains information on the innards of an object, i.e., the microstructure, while the eye remains at the surface, i.e., the macrostructure, and plays a lesser role in developing a belief in the reality of the external world.[8]

There is a multi- and cross-modal activity of the brain based on multisensory neurons that receive more than one type of sensory signal from all senses, thus keeping the specialized areas in the brain simultaneously informed, and we are able to extract information from one sensory modality and use it in another by using polymodal association centers.[9,10] The flow of impulses from the senses influences the number of activated nerve cells, so it seems that conscious use of additional perceptual input might strengthen the experience of touch, especially at a time when the sense of touch is weakened, such as after an injury to a peripheral nerve.

Moberg[11] established the term *tactile gnosis* for the specific aspect of functional sensibility representing the interplay between the peripheral function of the nerve and the interpretation of sensory impressions in the somatosensory cortex of the brain. This is a function that enables localization of touch, discrimination of touch, and recognition of the qualities and the character of objects without using vision. Hand surgeons often describe the hand as a sense organ, strongly linked to the brain.[12–14] This approach has in recent years also gained importance in discussions of surgical techniques to improve poor results after nerve lesions. The emphasis in these discussions is not only on the importance of advanced surgical techniques, but also on the rapidly expanding knowledge in neurobiology, with several tools available to influence injured neurons, and to use the inherent plasticity of the neurons in further development of rehabilitation after surgical interventions.[5,6,15]

The well-developed feedback system between the hand and the brain, with continuous proprioceptive information of position, and tactile input that is coordinated with memory systems in the brain, is a prerequisite for regulation of grip force and grip speed.[16] The feedback from a functioning sense of touch is also essential for body awareness.[17]

Physiology

Sensory perception is based on stimuli of mechanoreceptors and free nerve endings in the skin, resulting in an afferent signal pattern that has to be interpreted and processed in brain cortex. Sensory palsy may be a result of lesions or pathologic processes at many levels, such as sensory receptors in the hand, peripheral nerves, spinal cord, and brain cortex. The discriminative touch, one aspect of *tactile gnosis*, is dependent on cutaneous mechanoreceptors sensitive to pressure, vibration, or stretching. The physiology, function, and distribution of the mechanoreceptors in subepidermal, dermal, and subcutaneous layers of the volar glabrous skin of the hand have been well defined and described in numerous investigations.[18–24]

Fast-adapting (FA) mechanoreceptors are sensitive to fast mechanical changes in the skin such as vibration and friction, while *slowly adapting (SA)* mechanoreceptors respond to sustained stimuli and skin stretch. Among FA mechanoreceptors, vibrations below 50 Hz are mediated mainly by Meissner's corpuscles (FA I) located superficially in the dermal papillae. Meissner's corpuscles have small peripheral, well-defined receptive fields in the fingers, with sharp borders. Vibrations above 50 Hz are mediated mainly

by the Pacinian corpuscles, which are located in the subcutaneous layers. Pacinian corpuscles represent a system of FA mechanoreceptors type II (FA II). In humans, the Pacinian corpuscles cluster close to nerves and vessels at the metacarpophalangeal joints, the total mean number in a human hand being around 300.[24]

Among SA mechanoreceptors, Merkel's end-organs SA type I, SA I) respond to sustained stimuli in the skin, such as pressure and stretching. These receptors respond especially to indentations that are produced when an edge cuts through the receptive fields.

Ruffini's end-organs (SA receptors type II, SA II) are located in deep dermal layers and possess large diffuse receptive fields.[23,25,26] Ruffini endings respond to stretching forces associated with changes in joint position and skin displacement during movements. The Ruffini endings are situated at deep dermal levels and represent an important type of proprioception mechanoreceptors.[27,28]

When a mechanoreceptor is stimulated, signals travel to the somatosensory brain cortex after passing the dorsal root ganglia, up the dorsal column of the spinal cord via the medial lemniscus pathway and intermediate relay stations situated in the cuneate nucleus in the brainstem and the ventroposterior nucleus in the thalamus.[5] Signals elicited by touch reach primarily the contralateral hemisphere, but to a lesser extent also the ipsilateral somatosensory cortex.[29–32] The pattern of sensory inputs preserves the spatial relations of the receptors of the body surface, thereby creating a *somatotopic map*, also called a *neural map,* of the body surface in the somatosensory brain cortex. In the brain cortex, the body parts have representational areas in relation to their innervation density and their relative importance in sensory perception. Thus, the hand, face, and lips are huge compared with the feet, legs, and trunk.[3] In primate experiments, based on direct recordings from brain cortex, the exact finger and hand representations have been meticulously outlined.[33–36] These primate studies have demonstrated individual finger representations in well-defined cortical band-shaped areas.[33,35–38] The size of the hand representational area in primary somatosensory cortex (S1) has been assessed as about 10 mm.[37,39,40]

Sampling and processing of tactile shape is complicated and not fully understood. For instance, brain imaging studies have shown that the microdiameter (the texture of objects) and the macrodiameter (the shape of objects) are processed in different somatosensory areas of the brain. The processing of sensory input is diverse and hierarchic, and it appears that information about an object that is touched is sampled sequentially and not in single grasps:[41,42] when discriminating the shapes of objects, including edges, corners and curvature, sampling of information is mainly focused on sequential palpation of object properties.[42]

Defining the problem

Sensory palsy, here defined as loss of sensory functions in the hand, may have disastrous consequences. Loss of *protective sensibility*, as seen in patients suffering from leprosy, may result in injury, infection, and spontaneous amputation of fingers. Loss of protective sensibility may be a serious problem also in the early phase following nerve repair and in metabolic neuropathies such as diabetic neuropathy. Impaired tactile discrimination and tactile gnosis is also seen in compression neuropathies and vibration injury. Severe sensory palsy, combined also with motor palsy, is common in spinal cord injuries and stroke.

In hand surgery, injuries to the major nerve trunks of the upper extremity represent major reconstructive problems, with limited possibility to regain normal sensory functions following nerve repair in adult patients.

Regeneration and repair – physiology

Restoration of sensory functions in the hand after nerve repair is a complex process based on cellular, chemical, and functional changes at many levels – from the fingertips to the brain cortex.[5] The role of the surgeon is limited to coaptation of the nerve ends by placing sutures in the epineurium or perineurial tissue. However, the surgeon can never work at the level of axonal sprouts, less than 1 µm in size. Axonal outgrowth and orientation are dependent on molecular mechanisms in the microenvironment, with various types of attractive or repulsive mechanisms stimulating or inhibiting the advancement of axons.[5]

Transection of a nerve is followed by prominent functional and structural changes in the corresponding nerve cell bodies.[43–46] There is an increase in cell body volume and a shift in metabolism from a mode of maintenance to a mode of growth.[47] As a result of trauma, there may be a 20–50% loss of neurons in the dorsal root ganglia,[48–50] and also motor cells may die, although to a lesser extent.[51,52] Several factors may have an influence on post-traumatic neuronal loss in the dorsal root ganglia, such as age, time elapsed from injury to repair, and proximity of the injury. Immediate repair of the nerve may reduce post-traumatic cell death.[53]

At the site of injury, there is – regardless of the surgical repair technique – a great extent of axonal misdirection, so that sensory axons grow into motor Schwann cell tubes, and vice versa.[5,54,55] The distal segment undergoes Wallerian degeneration,[56] and the Schwann cells start to proliferate and produce a number of various growth factors, such as nerve growth factor (NGF),[57,58] ciliary neurotrophic factor (CNTF), brain-derived neurotrophic factor (BDNF), and NT-3, NT-4/5, and NT-6.[5,6,59] The Schwann cells increase their expression of NGF receptors.[60,61] Invading macrophages release interleukin-1 (IL-1), which triggers increased NGF transcription and NGF receptor density in the Schwann cells.[60,62] For details of the physiology of regeneration and the importance of growth factors, see, for instance, Fu and Gordon,[47] Terzis et al,[63] Frostick and Kemp,[64] Yin et al,[65] McAllister et al,[66] Terenghi,[67] and Lundborg.[68]

There are various opinions about the extent of *specificity* in axonal growth. In a number of articles, Brushart and co-workers[69–73] have stressed the existence of 'preferential motor re-innervation', indicating that motor fibers preferentially innervate distal motor Schwann cell tubes in contrast to distal sensory Schwann cell tubes. The basis for this is believed to be the presence of specific 'recognition molecules' in motor Schwann cell tubes, as opposed to sensory Schwann cell tubes.[74,75] However, according to Maki and co-workers,[76–80] there is a specificity in *sensory* regeneration, but not in motor regeneration. Maki feels that outgrowth of Schwann cells from the distal segment has an important role in this context. Thus, opinions differ regarding specificity in growth of sensory and motor axons. The reason for the disagreement may be utilization of different experimental models and various techniques for assessment of growth.

At the level of the central nervous system, nerve transection induces immediate and longstanding reorganizational changes in somatosensory cortex.[5,6,68,81–86] First, deafferentiation is followed by a silent 'black hole' in the somatosensory cortex corresponding to the representational area of the deinnervated peripheral body part. The cortical area rapidly becomes occupied by expanding adjacent cortical areas. When reinnervation of the hand occurs, the previously well-organized cortical hand representational area (the 'cortical hand map') is again re-established, but is changed into a mosaic-like, completely disorganized pattern.[38,83,87,88] Cortical regions that receive tactile signals from reinnervated skin regions then contain multiple recording sites with abnormally located or multiple cutaneous receptive fields[83] – the hand 'speaks a new language to the brain'.

Factors influencing sensory recovery after nerve repair

Type of nerve

The type of nerve that is injured influences the outcome considerably. Injury to a pure sensory nerve eliminates the risk of mismatch between sensory and motor pathways, thereby optimizing accuracy in sensory reinnervation. An illustrative example is repair of digital nerves, containing only sensory fibers. However, with mixed nerves (e.g., the ulnar nerve), the situation is quite different, with obvious risks of motor–sensory mismatch.

Level of injury

Following nerve transection, there is an initial delay, followed by sprouting and axonal outgrowth. An average outgrowth rate of at most 1–2 mm/day in humans has been suggested as likely.[89,90] In distal nerves, the functional outcome is not influenced by long regeneration distances, while more proximal lesions create a different situation. A median nerve lesion at wrist level may require 3–4 months before the first signs of reinnervation in the hand occurs. In brachial plexus lesions reinnervation of the hand seldom or never occurs, because of the long regeneration distances.

Timing of repair

It is generally agreed that freshly transected nerves should be repaired acutely with no or minimal delay.[91–99] There are several reasons for this. The postoperative death of nerve cells in dorsal root ganglia can be substantially reduced if the repair takes place within 24 hours after injury.[53] With increasing preoperative delay, the result becomes progressively worse as a result of fibrosis of the distal nerve segment, atrophy of Schwann cells, and progressive loss of neurons. While irreversible changes have usually occurred in denervated muscle cells after 18–24 months,[100,101] sensory organs seem more resistant to denervation than muscle, and the final extent of sensory recovery has been reported to have little correlation with the time between injury and nerve repair.[102] However, denervated skin receptors can cause a poor result. Denervated Meissner's corpuscles in monkeys progressively degenerate over a period of several months, but never disappear.[103,104] Merkel's cell are reduced in number, become atrophic, and may be differentiated into transitional cells or keratinocytes after deinnervation. In Pacinian corpuscles, the outer lamellar layer remains intact, though atrophied.[105,106]

Age of the patient

Transection of a nerve results in functional reorganizations within the central nervous system, due to misdirected axons, at cortical as well as subcortical levels.[86] In primate experiments, young individuals have a greater capacity for normalization of the cortical hand map after nerve repair compared with adults, and in the developing brain there are mechanisms that can create a normalized cortical representational topography despite a disordered sensory input.[87,107] The young brain is more plastic to changes, is programmed for learning, and easily absorbs new knowledge. That is one reason for the excellent functional sensibility after nerve injury usually seen in very young children in contrast to adults.[96,102,108,109]

Age is also the main explanatory factor for the variance in the outcome after nerve repair,[6,85] and since axonal regeneration, nerve maturation, and conduction velocity after nerve repair do not differ between children and adults,[108] the variance is probably due to the better adaptation in the children to relearn sensation, and quickly interpret the reorganized impulse patterns.[107] Adults can compensate to some extent for the age factor with sensory

re-education programs utilizing the capacity for functional remodeling that is possible also in the adult brain.[14,110] However, clinical experience as well as the literature show that functional sensibility after a nerve repair often is very limited in adults.[111,112]

Cognitive capacities

We have previously demonstrated that cognitive capacities such as verbal learning capacity and visuospatial logic capacity explain variations in the recovery of functional sensibility in adults after nerve repair.[113] Regarding verbal learning in general, a critical age period for acquiring a second language has been presented by Johnson and Newport,[114] demonstrating that, following a maximum at the age of 5–10 years, there is a rapid decline, leveling out after the age of 18, with a temporary increase in the late 20s. We have compared these data with the age-related capacity to recover tactile gnosis in the hand after surgical repair of median or ulnar nerves.[115] There is a striking analogy between these data and the data describing the acquisition of the second language as related to age. The data indicate that principles for learning a new language may also be applicable to sensory relearning, and future sensory re-education programs should use protocols based on experience and knowledge gained from strategies to improve learning of a new language at the adult stage. This is a challenging and important area for multidisciplinary clinical research.

Sensory re-education and brain plasticity

Current protocols for sensory re-education

Sensory re-education programs are used in the rehabilitation phase to facilitate and positively influence the relearning process that is required to allow adaptation to the new synaptic organization associated with cortical reorganization, and to improve the recovery of functional sensibility – tactile gnosis.

Much of the relearning process is gained by use of the hand in common manual tasks, using vision as a guide and a code for impaired hand sensibility. To facilitate and enhance this process, specific programs developed in the 1970s for sensory re-education are used as a clinical routine in adult patients for regaining tactile gnosis.[4,14,116–118] According to these strategies, the brain is reprogrammed on the basis of a relearning process: first, the perception of different *touch modalities* and the capacity to *localize* touch is trained; this is followed by touching and exploration of items presenting *shapes* of varying and increasing difficulty – with the eyes opened or closed. In this way, an alternate sense (vision) trains and improves the deficient sense (sensation[14]). However, the outcome after nerve repair in adults is often disappointing. Sensory re-education programs are normally used in the second phase after the repair (when the hand is re-innervated by axons to a large extent, however misdirected), to facilitate and positively influence the relearning process that is required to make possible an adaptation to the new synaptic organization, and to improve the recovery of functional sensibility.

This second phase occurs when some perception of touch can be demonstrated in the distal vola or once the patient has some return of sensation in the fingers,[14,116] that is, roughly 3 months after repair at wrist level. At this point, the reorganizing brain presents a distorted pattern that may not be possible to reverse and that can be very difficult for the patient to learn to interpret.

Timing of sensory re-education

One explanatory factor for the poor functional result after nerve repair may be the all too late onset of sensory re-education, and the fact that the design of the sensory re-education programs has not changed over the last few decades. Evolving concepts in neuroscience and cognitive science open up new perspectives and potential possibilities for the future to improve sensory recovery after nerve repair.[118]

In recent years, rapidity of functional reorganization has been emphasized.[85,119] Due to the deafferentiation such as amputation or transection of a single nerve, the representation of that specific hand area in the sensory cortex is rapidly invaded by expanding adjacent areas.[35,120] Synaptic reorganization may also be use-dependent, occurring when the input to brain cortex is not interrupted but merely changed. Frequently used fingers expand their cortical representations, for example in musicians.[121] Such synaptic reorganization changes may also be activity-dependent, based on alterations in hand activity and tactile experience.[122,123] An analogous phenomenon can also be seen after local anesthetic blocks that quickly and temporarily can induce shifts in neuronal receptive fields with cortical reorganization.[124,125]

The holistic organization of the brain

The use of vision to guide the retraining of sensation is the basis for classic sensory re-education, but there is a continuous interplay between *all* senses. There is multi- and cross-modal activity of the brain based on, for example, multisensory neurons that receive more than one type of sensory signal from eyes, ears, nose, tongue, and the skin, thus keeping the specialized perceptive areas in the brain simultaneously informed about what is happening, and it is demonstrated that we are able to extract information from one sensory modality and use it in another by means

of polymodal association centers. It has been proposed that brain areas should be regarded as not being dedicated to one sense specifically but rather to various tasks that require the interaction of several senses at the same time, for example, judgment of distance requiring information of visual, tactile, and perhaps acoustic nature.[9,10,126]

This holistic concept, focusing on functional co-work between the different association areas of the brain justifies re-evaluation of traditional territorial thinking of the brain. The concept opens for new possibilities to use the plastic potential of the brain in the rehabilitation after nerve repair at a very early stage. Such alternate strategies, which might improve the effects of sensory re-education, could be made possible with artificial sensibility before there is a 'real' sensibility after nerve repair.

Artificial sensibility and sensor glove

Hand sensibility is a prerequisite for good hand function, and decreased or absent sensory feedback may be a major problem for patients with injury to the peripheral or central nervous system, as well as for amputees using hand prostheses. The lack of sensory feedback in hand prostheses is probably one reason for the common rejection of prostheses, despite the existence of sophisticated electromechanical devices. Various concepts of artificial sensibility have also been discussed since the introduction of myoelectrically controlled hand prostheses.

We have recently presented a model for artificial sensibility based on sense substitution, using hearing as a substitute for sensibility. Miniature microphones are mounted on a glove at the fingertips.[127] The stimuli generated by active touch of various structures (the friction sound) can thereby be picked up, amplified, and transposed to stereophonic acoustic stimuli. It is thus possible to train, for example, localization of different fingers and identification of different textures, and to use the alternate sensory feedback in activities of daily living. This principle is used to provide the sensory brain cortex with an alternate sensory input at a time when regenerating nerve fibers have not yet reached their peripheral targets. The resemblance in perceptual experience between sound and touch is bridged by the vibratory sense. This method allows initiation of sensory re-education the day after the operation – long before any reinnervation can be identified – in order to feed the sensory cortex with 'relevant' information, the purpose being to maintain the cortical hand representation from the affected hand until 'real' sensibility is present. Pilot cases have shown very promising functional results with the sensor glove system (SGS).[128–130]

Interhemispheric plasticity

Sensory input is processed in the contralateral hemisphere, but recent research on perception of touch in humans, with, for example, functional magnetic resonance imaging (fMRI) techniques, and positron emission topography (PET) demonstrates that there is also an ipsilateral activation – to a lesser extent – of the sensory cortex on sensory stimulation of the fingers.[29,30,131] fMRI investigations have also shown a different emphasis regarding laterality in patterns of cortical activity during precision versus power grip.[32] Interhemispheric plasticity has also been demonstrated, with gain in tactile discrimination and perception of touch in the contralateral hand that was identified during deafferentation, which suggests that existing neural substrates are involved.[125,132] Understanding the mechanisms of interhemispheric plasticity is necessary to design appropriate strategies to up- and downregulate plasticity changes to promote sensory re-education following nerve injury.

Learning and importance of enriched environment

Sensory re-education is an issue of learning, and it has been demonstrated that an enriched environment influences learning by stimulating the formation of new synapses.[133,134] Factors such as a stimulating environment, meaningful activities, and encouragement influence the molding of the brain in a positive direction.[135,136] It has also been demonstrated that passive unattended and repetitive tasks lead to negative changes of the territories in the sensory cortex. These observations have been discussed in particular in the context of dystonia and repetitive strain injury problems.[122,137,138]

Very little is known about the effects of rehabilitation strategies on brain organization. The neural basis for these central events that are triggered by sensory re-education has been little investigated, but Florence et al[139] have shown that monkeys with median nerve repair in an enriched sensory environment developed small and well-localized receptive fields in cortical area 3b compared with animals without the sensory enrichment.

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26 Re-education after sensory nerve transfer

Giorgio A Brunelli

The defects of sensory function of the hand are very disabling conditions, especially those of the median nerve that deprive the thumb–index pliers of sensation, or of the ulnar nerve, that render the ulnar side of the little finger insensate.[1–5] Loss of sensation is often part of a much more severe lesion, as for instance a palsy of the brachial plexus or a devastating wound of the upper limb. In such cases, very few surgeons try to restore sensation to a paralyzed hand that has regained only very poor motor function.

Reconstructive surgery is essentially based on motor function restoration.[6] Leffert says that there is absolutely no justification for surgical reconstruction of the flail anesthetic arm, and that amputation must be considered.[6] However, in contrast even in avulsion of the brachial plexus some motor and sensory recovery may be obtained by combining transfers of motor nerves (XIth cranial nerve, intercostals, branches of the 3rd cervical ansa) with the transfer of the supra-acromialis and supraclavearis sensory nerves to the medial sector of the median nerve.[1,7–9] Sometimes the recovery of sensation, even if marginal, may be integrated in the general re-education and also improve the motor function.[6]

Restoration of sensation of the most important digits is a prerequisite for the recovery of motor function.[2,10] While numerous techniques have been described for the restoration of motor function, very few have been published on sensory reconstruction.[1,4,8,10–15] A nerve transfer comprises the sacrifice of a normal, relatively unimportant donor nerve and its transfer to reinnervate the distal stump of a more important nerve that has suffered an otherwise irreparable lesion. Obviously the damage caused by the transfer of the donor nerve must be considerably less than that in the receiving area. A satisfactory result can only be expected if the donor nerve is sound (S4). In fact, part of the sensory function will be lost after the transfer and an S4 score will never be regained.

It is very rare to have an S4 nerve available. In fact, sensory nerve transfers are mostly done in cases of proximal lesions (as, for instance, brachial plexus injury) where it is very difficult to find S4 nerves. In such cases, therefore, it is difficult to restore a discriminating function, and only restoration of a protective sensation can be expected, with partial recovery of gnosis in exceptional cases.

Indications for sensory nerve transfer are mainly loss of sensation in the thumb–index pliers or the ulnar side of the hand and little finger. In a totally insensate hand, as in cases of total palsy of the brachial plexus by avulsion of the roots from the cord, when transfers of motor nerves (the XIth cranial nerve, the intercostals, or the cervical plexus branches) are performed, the supraclavearis and supra-acromialis nerves can be transferred to the medial sector of the median nerve to gain some protective sensation in the hand.

Sensory nerve transfers are also used to restore protective sensation in the sacral area in paraplegics, to avoid pressure sores, by transferring intercostal nerves, and to restore sensation to the sole of the foot by transferring the sural nerve on the sensory branches of the peroneal nerve.

If the loss of sensation is due to loss or alteration of the skin, sensory skin flaps are preferentially done, as suggested by Moberg in 1955 and popularized by Littler. The island flaps are taken mainly from the ulnar pulp of the ring finger, innervated by the ulnar nerve.[2,11,13] The results are often moderate, and are frequently complicated by disethesia or parestalgia due to the stretching and kinking of the nerve or the vessels – drawbacks that do not occur in nerve transfers.

Sensory flaps can only be carried out in particular sites and conditions. The donor site, covered with a free skin graft, is often disesthesic. Free sensory flaps from the toe have been used, with good and very good results, but they can only be done if a sensory recipient nerve is available.

The-pulp-of the big toe has a very low discrimination because existing sensory corpuscles are not fully used. Re-education is able to restore a higher discrimination than in the foot, because the sensory corpuscles of the big toe are connected to the receiving cortical area that corresponds to the thumb. This is well demonstrated by the astonishing capacity of patients with congenital aplasia of the upper limbs to knit and sew by using their toes.

Only a normal donor nerve (S4) can restore a reasonable sensivity to the receving area. Nonetheless, in cases of severe

brachial plexus lesions, S3+ or S3 nerves may also be transferred to restore protective sensitivity to the thumb–index pliers.

The preoperative evaluation of the donor skin is relatively easy for the glabrous skin of the digits, but it can be difficult for skin not specifically used for tactile function (e.g., the dorsal ulnar skin of the hand). Electromyography (EMG) can test the preservation of sensory potential.[16] Better results are obtained when the transfer is done from one branch to another branch of the same nerve (e.g., a collateral nerve of the long finger to the thumb) or when sensory branches of different nerves are used at the same digit (rather than at different digits) (e.g., transfer of a dorsal to a collateral nerve of the thumb radial and median innervation of the same digit).

Sensation can be classified into protective, tactile, discriminative, and gnostic:

- protective sensation perceives pain (S1)
- tactile sensation perceives (to a different extent) the touch (S2-S2+S3)
- discriminative sensation recognizes the size of the object and the distance between two points not more than 10 mm apart (S3+)
- gnostic sensation allows the subject to recognize two points closer than 10 mm, and also to identify the nature of the object by means of activation of brain circuits involving perception, memory, and attention.

In sensory recovery after nerve transfer, gnosis requires the formation of new circuits of neurons, new synapses, and their long-term potentation to create new memories. Gnosis should be the target of every sensory reconstructive surgery. Unfortunately, often only tactile sensation, and sometimes even only protective sensation, is feasible due to the poor condition of both the receiving and donor nerves.

In nerve transfers, after the regenerating axons have reached the skin and recolonized the sensory corpuscles, the sensory cortex of the brain will perceive sensations coming from areas different from the original ones. At the beginning this generates 'confusion'; however, through proprioceptive fibers and eye control the brain receives information on the 'new' sensory area and is able to integrate the new functions into the scheme of sensory motor function of the hand.

Nerve transfers must be considered strictly microsurgical operations. The donor nerve has to be manipulated very gently, to preserve its blood supply, and it must be laid on a soft and sound bed. Suture must be performed as distally as possible, in order to have a shorter tract of Wallerian degeneration to reinnervate. If these rules are followed, sensory nerve transfers are very useful, albeit some of them must still be considered experimental.

The most frequently used transfers are those of the dorsal branches of the radial nerve to the opposing pulps of the thumb and index finger (Figure 26.1a). The suture is preferentially done at the first web. Only where the surrounding skin is bad should the connection be done at the wrist (Figure 26.1b). Other transfers in the hand include the dorsal branches of the ulnar nerve to the ulnar skin of the hand and little finger, or to the opposing collaterals of the thumb and index finger where the radial branches are not available (Figure 26.1c) the collaterals of a normally innervated finger to an insensate finger or the thumb (Figure 26.1d–f).

Evaluation of the sensory function may be done by various methods:

- the slowly adapting fiber receptor group by the classic Weber test (2 point discrimination) (Figure 26.2)
- the quickly adapting fiber receptor system by the moving 2 point discrimination test
- the Semmes Weinstein monofilament test (less frequently used)
- the vibration threshold (mainly used in experimental research)
- the sweating test, which is an indirect test for sensitivity.

Sweating, in fact, is correlated with sensation if the lesion is below the gray communicating branches of the autonomic nerve system. If the lesion is above this connection, there is dissociation of the sensory from the sweating function. Sweating may be tested and recorded by means of the ninhydrin test (Figure 26.3), but an easier and quicker method is observation of the small sweat droplets among the pulp glyphs by a simple otoscope (Figure 26.4).

For the evaluation of gnosis, only gnostic rings may be used. These are a set of three rings (Figure 26.5), each of which has inset about its periphery an alphabet letter (A), an Arabic numeral (3), a geometric figure (0), a rough level surface, and a smooth convex surface. The three rings are identical, with the exception that they differ in size. On one ring the dimensions of the symbols are 15 mm on each side; on the second ring they are 10 mm on each side; and on the third ring they are 5 mm on each side. For the evaluation, the subject is blindfolded then asked to identify each of the five different symbols on each of the three rings. The recognition of each symbol is given a score. On the largest ring, each symbol recognized is given 12 points, for a total of 60 points with this ring. On the of intermediate size ring, each symbol recognized is given 6 points, for a total of 30 points. For the smallest ring, each symbol recognized is given 2 points, for a total of 10 points. Thus, if each symbol is recognized correctly, the subject receives a total score of 100 points. Only volunteers with normal intelligence can achieve a score of 100. However, in manual workers with calloused fingers, or in older individuals, a score of 90–84 would still be considered very good.

The first goal of sensory re-education is protective sensitivity, but re-education cannot be started until the regenerating axons in the receiving nerve have reached the sensory corpuscles. Therefore there is a time lapse (between the operation and the arrival of the regenerating axons at the skin), during which re-education is worthless and protective measures have to be taken to prevent injuries to the anestatic area. During this period, both hot and ice-cold objects should be avoided, the motor strength should be modulated, pressure on the handles should be reduced by

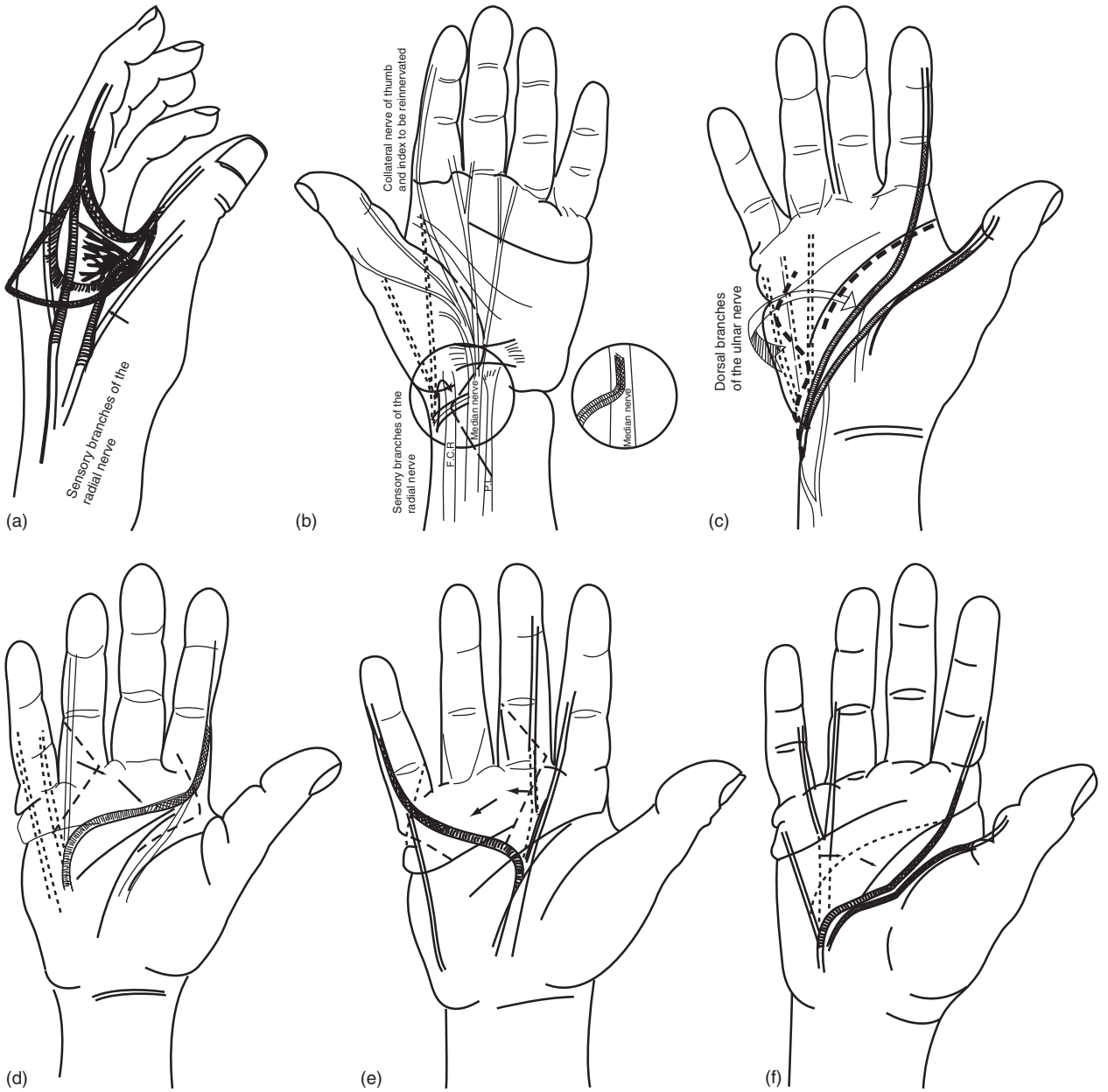

Figure 26.1(a–f)

Different types of sensory nerve transfers (see text).

enlarging their contact surface, and any skin alteration should be taken care of every evening.

Favorable conditions to start sensory re-education are a very distal tinel sign, initial pain (protective) sensation, return of sweating (visible by the otoscope), motivation, and the patient's capacity to learn and concentrate. First the patient is asked to locate the stimulus. At first he is able to perceive but not to pinpoint it. The rehabilitator must patiently touch a single zone of skin at a time, and the patient, with eyes closed, is asked to identify the zone. The patient is then asked to open his eyes to see whether he was right or not. In this way the attention of the patient is stimulated, and by repeating this eyes closed/eyes open test, long-term potentiation of the synapses of the brain cells is achieved, which introduces new pieces of memory necessary for new circuits.

The patient is then asked whether two stimuli are identical or different, and in which manner they differ. Different materials – e.g., sand, stone, wood, plastic, sponge – are then offered to the patient for identification. Each session should not exceed 20 minutes, in order not to tire the patient, and should be repeated at last 3 times a day.

It is important to insure good pain control and neutralize hyperestesia or paraestalgias because they compromise the treatment and the result. The cooperation of the patient is a prerequisite for a good rehabilitation. In addition to the use of pain-relieving and tranquilizing drugs, local stimulation

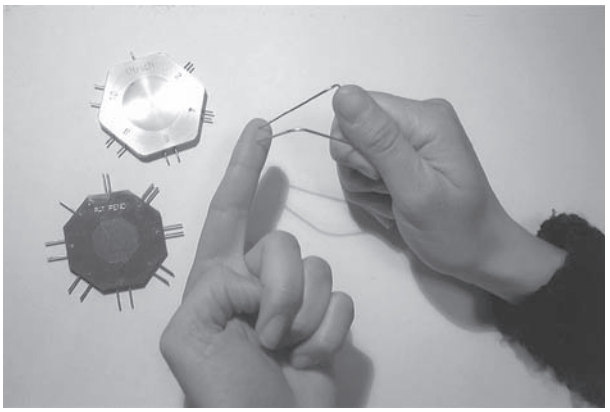

Figure 26.2
Two-point discrimination test.

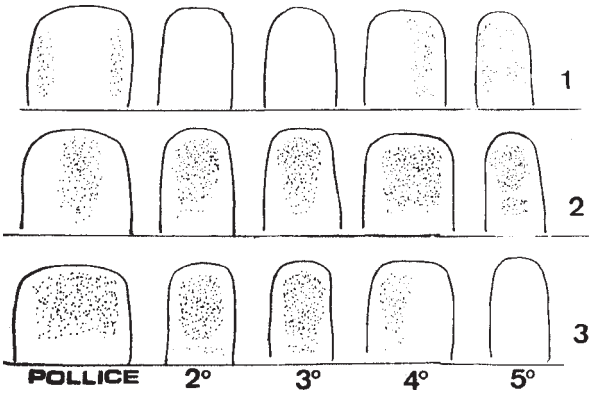

Figure 26.3
Ninhydrin test: in row 1 sweat droplets are missing in the region of the median nerve; in row 2 droplets are lacking in the skin innervated by the radial nerve; in row 3 denervation of the skin depending on the ulnar nerve is shown.

Figure 26.4
Examination of sweat droplets by the otoscope.

with various instruments can be helpful – increasing the force of application and duration, but remaining below the pain threshold. This approach may be more effective if incorporated into a game.

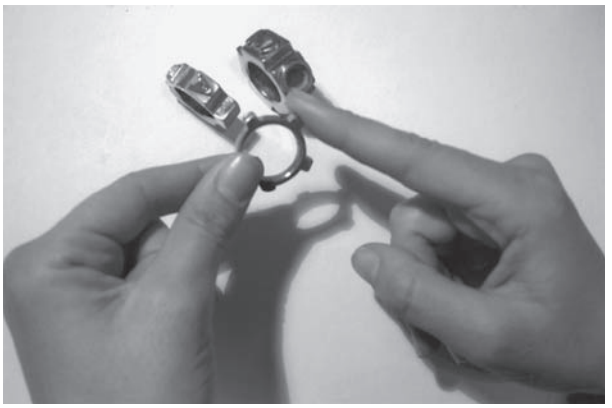

Figure 26.5
The gnostic rings test (explanation in the text).

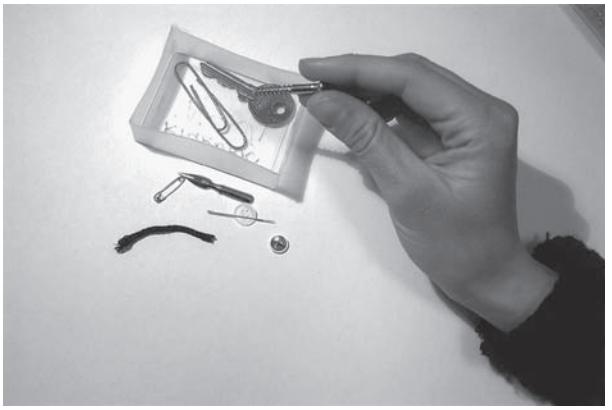

Figure 26.6
Pick-up test.

Mobility and motility are paramount for sensory re-education. Immobilization hinders the sensory recovery, and even if it becomes necessary for other causes during the re-education, it is still a setback for the progress achieved. Therefore gentle mobilization of all the joints of the hand should be done daily, and active motion encouraged.

As mentioned before, the results depend on the severity of the global damage, and only in a few cases can re-education aim to regain discrimination and gnosis. The latter may also be recovered by the transfer of a nerve with an S3 score, due to the brain's ability to incorporate new memories and neuronal circuits when tactile sensitivity has been regained.

Sensory re-education includes:

- the recognition of different materials
- the pick-up test and exercises (Figure 26.6)
- the gnostic rings (Figure 26.5).

The patient is instructed to work with the gnostic rings several times a day. First the patient must be familiar with the symbols on the rings, and able to recognize them when

blindfolded. The patient checks whether he identified the ring correctly by opening his eyes. The exercise begins with the largest ring and progresses to the smallest ring, and should be repeated many times a day. The great advantage of the gnostic rings in sensory re-education is that the patient can keep one in his pocket and carry out the exercise many times a day.

The superiority of the gnostic rings in sensory re-education resides in the 'exertion' by the neuronal assemblies, that is much more significant than required by the 2 point test or the pick-up test. This 'exertion' introduces the formation of new neuronal circuits, with new synapses. Repetition of this effort leads to the long-term potentiation of these synapses. The results are far better than those obtained by traditional methods.

The scar must be taken into consideration and treated. The superficial scar is less important, and is treated by gentle and prolonged compression and massage. The most important scar is that occurring at the level of the nerve connection, because the proliferation of fibroblasts may lead to an internal fibrosis which will hinder and even prevent the recovery. X-ray therapy is recommended, at an antifibroblastic dose, above the nerve suture, starting 10–12 days postoperatively. Unfortunately, this therapy almost entirely ceased due to the severe damage it caused when used at high dosage to treat cancers (7000 rads or more). However, in my experience using only 800 rads no collateral effects have been seen in more than 3000 treatments used routinely in all nerve sutures over a period of 30 years. Experimental research on guinea pigs also demonstrated significant reduction of intraneural fibrosis in the treated animals.

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27 Volkmann's contracture

René Malek

Introduction

It is interesting to see how a medical chapter becomes clearer once a better understanding of its pathogeny has been achieved. After having reported the first case of the syndrome that was to bear his name in 1869, R. Volkmann[1] gave the following definition in 1881: 'contracture of the muscles of the anterior region of the forearm, associated with more or less extensive paralysis of the hand'. He had already introduced the way ischemia affects different tissues. However, this definition corresponded to the late stage of a disease, for which the physiopathology was not fully understood. Indeed the term 'Volkmann's disease', often used in textbooks, clearly reflected the mystery with which this condition was viewed by surgeons until very recently.

Many interpretations of the cause of this ischemia have been proposed, leading to different therapeutic attitudes which were as varied as they were ineffective, and mostly not capable of identifying the stage of the disease.

The modern understanding of the onset of the trouble is now widely accepted. It is an example of a *compartment syndrome*, and the classic description of Volkmann corresponds to an untreated or very late evolution. The anterior region of the forearm, as some other parts of the body, has the unusual feature of being an almost completely closed unit, because of the bony insertions of the aponeurotic or fascia tissues. These are not extensible and so hyperpressure can develop inside for many reasons (Figure 27.1). This hyperpressure interferes with the local circulation, affecting the small vessels and the capillaries in particular: edema occurs and creates a vicious circle that causes progressive aggravation of the condition, leading to ischemia.

In time a muscle contracture results, caused by the necrosis of scattered fibers, which can be further extended to the entire belly. Paralysis is due either to the lost function of the necrotized muscle or to ischemia of the nerve trunks that pass across, leading to necrosis of the fibers. Moreover, at the same time, compression of the main nerves can occur – in some existing anatomic narrow passages or under a fibrous and compressive mass that has developed in the area of tissue necrosis (Figure 27.2).

Thus the therapeutic action seems clear:

- First it is necessary to prevent the compartment syndrome which can develop in numerous pathologic situations, and so avoid Volkmann's contracture.
- Second, it is relatively easy to recognize the incipient compartmental syndrome if the practitioner measures the pressure inside the anatomic and closed zones of the forearm. Medical treatment can be used at that time, for example mannitol[2] or hyperbaric oxygenation.[3]

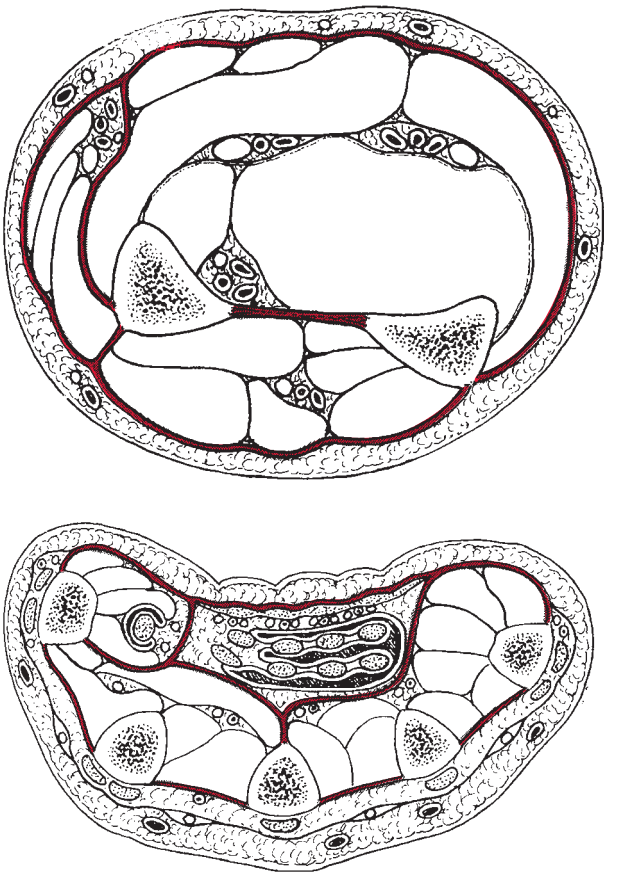


Figure 27.1

The closed areas within which a compartmental syndrome can occur. Reproduced from Tubiana.[4]

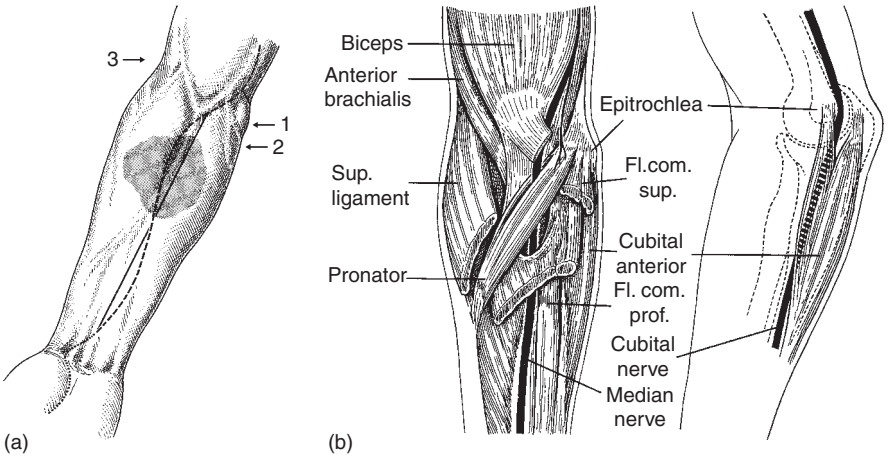


Figure 27.2

The narrow passages where the nerve trunks may be damaged. Reproduced from Tubiana.[5]

- Surgery, when necessary, should be performed as soon as possible to relieve compression, mainly using large fasciotomy. This is one example of a real surgical emergency.

If surgery is performed early, a true Volkmann's syndrome (see definition) fortunately becomes extremely rare in developed countries. However, it remains frequent in many regions, where the teaching of practitioners must be pursued. In the case of an established syndrome, ischemia and necrosis are the major problems. Treatment will depend of the proximity of the onset of the syndrome. But even if there has been some delay, it is still necessary to operate quickly, to eliminate the residual hyperpressure. Moreover, it is necessary to intervene because an extensive dissection of almost the entire forearm is necessary to take off the eventual necrosed tissues (mainly of the muscular bodies) and also to prevent or to treat the associated paralyses, given that there is a short period of possible recuperation after a surgical neurolysis.

Treatment

Treatment of the sequelae involves various operations, depending on the severity of the lesions. We shall deal with this in three stages:

1. the compartment syndrome
2. the established Volkmann's syndrome
3. treatment of Volkmann's sequelae.

The compartment syndrome

Locations

There are many sites in the upper limb where a compartment syndrome can occur:

- in the forearm
  - the two anterior compartments of the flexor digitorum superficialis
  - of the flexor digitorum profundus
  - the posterior compartment of the extensors
  - the compartment on the ulnar side
  - under the laceratus fibrosus
- in the hand in the interosseous spaces.

Causes of the compartment syndrome

This pathologic state is usually acute, but it can also be chronic.

The acute syndrome In the upper limb, under the arm, there are many reasons for hyperpressure to develop:

- The main cause is traumatic. It has been reported that Volkmann's syndrome occurs frequently in children aged 3 to 13 years, especially in boys, who can be considered overactive and fearless. Supracondylar fracture of the humerus with significant displacement is common, and a precipitating factor is a difficult or repeated reduction, as well as the use of a circular plaster with immobilization in flexion to stabilize the fragments. Other traumatic conditions are also risky: a comminuted fracture of the elbow, dislocation of the elbow, fracture of one or both forearm bones, contusion without fracture, and a penetrating wound of the humeral vessels.
- External compression can also be the cause, such as an individual caught in a landslide (frequently with an associated crush injury), or a comatose patient who has been lying for a long time on his forearm.
- Many cases have been reported after surgery on the forearm bones. Great care must be taken when a torsion movement is necessary, such as in the palliative treatment of congenital radioulnar synostosis, or in the correction of other pronosupination fixed deformities (Figure 27.3).
- The soft tissues can be involved: for example in burns, too tight closure of the skin in large wounds, abcesses and their local inflammation and edema, angioma, and amniotic band.
- Anesthetists are aware that they must avoid any compression of the upper limb in surgery performed on a

patient lying on the lateral side. They also have to be wary of arterial cannulations with the risk of local hematomas.

- Vascular causes include spontaneous hematoma in the hemophilic patient,[6] intravenous injections at the elbow crease of some pharmaceutical products, such as nitrogen mustards, and upper limb snake envenomations.

However the list is not closed.

The chronic syndrome A chronic compartment syndrome can occur in some conditions (mainly sporting activities[7]) and their clinical symptoms are various; diagnosis can be made by pressure measurement during effort (see below). Repeated and significant muscular exercise can expose the patient to rhabdomyolysis, with local compression and its specific renal complication.

Clinical findings

Clinical findings are important to obtain a compartment syndrome diagnosis as early as possible. It must be suspected whenever significant pain occurs in an extremity following injury. Nerve impairment will often cause the patient to complain of severe pain, which is out of proportion to the results of examination; the pain is often accompanied by a sensation of burning or tightness.

The classic '5p' symptoms – pain, paresthesia, pallor, poikilothermia, and pulselessness – are not reliable. Moreover, it must be remembered that these symptoms assume a conscious patient without additional injury. Also, that it is difficult to gather a history of complaints in young children.

Pain resulting from certain movements (e.g., passive stretching) can be the earliest clinical indicator of compartment syndrome. Another one is when the patient finds it difficult to move his fingers actively. During palpation the affected limb may feel tense or hard, as if it is filled with fluid, compared with the other side.

It is necessary to find out whether the patient is under anticoagulation drug therapy.

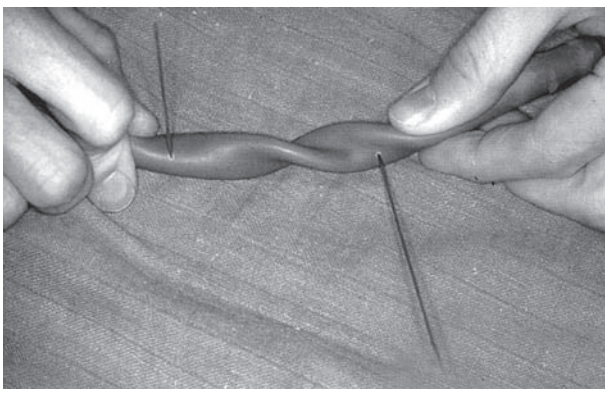


Figure 27.3

The torsion movement necessary to correct the pronated forearm in radioulnar synostosis leads to a stricture and compression of the vessels. Reproduced from Tubiana.[4]

Compartmental pressure measurement

In recent years, after the publications by Owen et al[8] and Matsen,[9] measurement was performed with a needle or a catheter implanted in the site and connected to a mercury manometer (Figure 27.4). Nowadays modern transducers (or tonometers) are usually easy to use and allow more reliable results. In fact this measurement gives the pressure of the tissues (muscles) rather than of the compartment. Tissue perfusion is determined by measuring capillary perfusion pressure (CPP), minus the normal interstitial fluid pressure. Elevated perfusion pressure is the physiologic response to elevation of the intracompartmental pressure. Normally the CPP is around 25 mmHg. Matsen[9] demonstrated that, as intracompartmental pressure rises, venous pressure also rises; and when venous pressure is higher than the CPP, the capillaries collapse. It is generally agreed that a pressure of 30 mmHg or more requires an emergency operation (fasciotomy).

It appears that the difference between the diastolic pressure and the CPP is a very important factor. One must

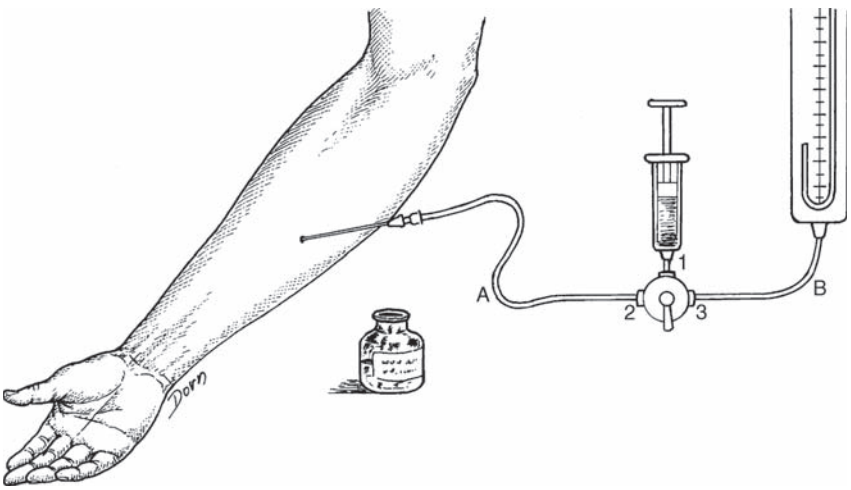


Figure 27.4

A simple method of measuring tissue pressure. Reproduced from Tubiana.[4]

avoid elevating the limb, which reduces the diastolic, venous pressure. Measurements must be taken in different zones of the forearm to find where the hyperpressure is maximum.

Imaging studies

Imaging studies have been proposed:

- Radiography is always necessary for bone lesions, but it is not useful for the diagnosis of compartment syndrome.
- Ultrasonography can help in evaluating arterial flow as well as visualizing any deep venous thrombosis, but it is not helpful in the diagnosis of compartment syndrome. However, it can help to eliminate differential diagnoses.
- Magnetic resonance imaging (MRI) allows the study of water distribution in the tissues, and can then show edema or necrosis, but it is difficult to base an emergency surgical indication on it, unless the patient is being treated in a center of surgery where MRI testing is available.

Treatment of compartment syndrome

At the early stage fasciotomy is the definitive therapy, with good results. There is a debate regarding the threshold for fasciotomy: as we have seen, 30 mmHg is recommended by numerous authors. Some authors cite 45 mmHg, and in contrast, many of them urge prophylactic fasciotomy even when there is a normal pressure, in some risky conditions, to prevent compartment syndrome.

The difference between the general diastolic pressure and intracompartmental pressure (called delta-P) is an important criterion for McQueen et al[10] for surgical fasciotomy.

During surgery, the use of a tourniquet is not recommended. The incision is extended from the elbow to the palm and fasciotomy must involve all the fascia – superficial, profundus, and lateral – on the ulnar side, and also on the posterior, when the pressure is elevated there (Figure 27.5).

The eventual lesion of the vessels must be confirmed at the time of surgery, and then any lesion should be treated. (However, excessive importance should not be given to the restoration of vascular permeability of the humeral artery at the elbow, since there is a rich vascular anastomotic collateral network.)

As a rule the skin is not closed – it is left open to avoid any tension, covered by a greasy dressing (Figure 27.6). The wound should be closed after a few days, when the edema has disappeared. Recently, the timing and use of fasciotomy have been questioned in this regard. It is said that to leave the wound open extends the hospital stay, and also has the disadvantage of changing a closed injury to an open one, thereby increasing the chance of infection. However, the severity of an established Volkmann's syndrome must counterbalance these comments. Medical treatment is normally utilized, but is only adjuvant: antibiotics, antispasmodics, vasodilatators, or corticosteroids, as well as mannitol[2] and hyperbaric oxygenation.[3]

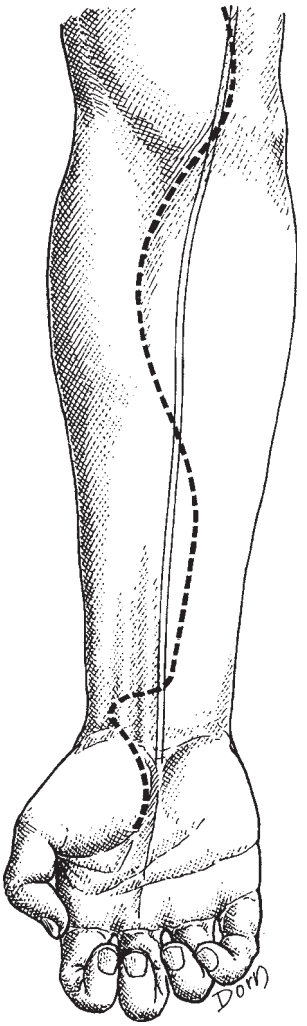


Figure 27.5

The incision is extended from the arm to the palm. Reproduced from Tubiana.[4]

The established Volkmann's syndrome

Volkmann's syndrome becomes established rapidly, in a matter of days, and often despite emergency treatment, characterized by its two classic and main signs:

- muscle contracture and
- paralysis.

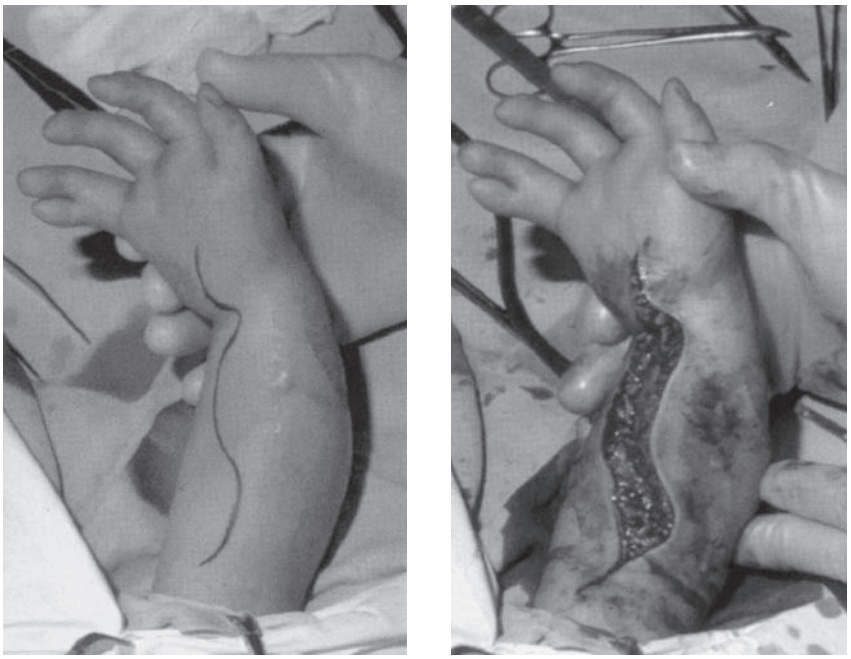
The two are localized in the forearm.

General clinical features

The vascular changes can persist for some time, and edema remains for many days. Large blisters appear on the skin and may become necrotic and ulcerate. The distal pulse may be absent or weak, but is still found in most cases. Globally, the appearance of the hand does not reflect the circulatory changes in the forearm.

Muscle contracture

This involves the muscles of the anterior compartment. The hand is in a pronation position and the wrist stays flexed.



(a) (b)

Figure 27.6

In acute compartment syndrome the incision is left open for some days to eliminate hyperpressure. Reproduced from Tubiana.[5]

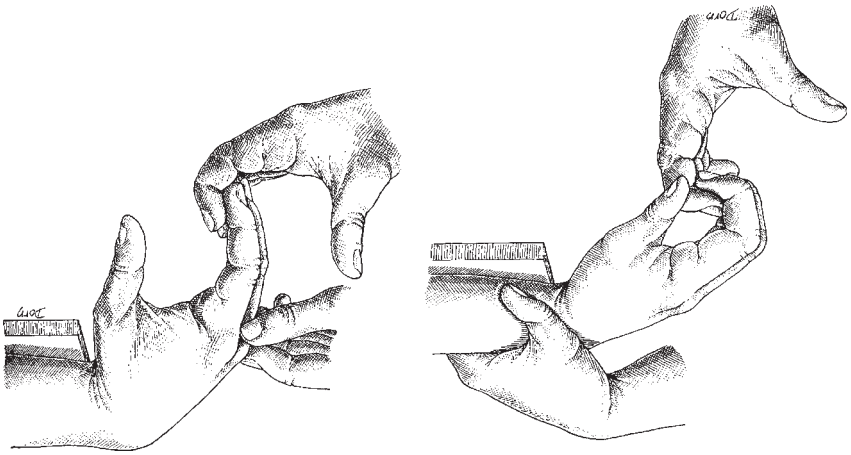


Figure 27.7

Pathognomonic maneuver of muscular contracture in Volkmann's syndrome: when the wrist is extended the claw of the digits is irreducible. When the wrist is flexed the fingers can be extended. Reproduced from Tubiana.[4]

Usually the matacarpophalangeal (MP) joints are extended and the other phalanges are in flexion. The thumb can be untouched, but is commonly in adduction with the distal phalanx flexed.

A classic and *pathognomonic sign of ischemic contracture* is when maximal flexion of the wrist allows some passive extension of the fingers, whereas wrist extension results in flexion of the fingers, and then attemps to straighten them causing pain (Figure 27.7). Active mobility is reduced and also depends on the extent of the paralysis.

Histologically, the fibers first retain their structure, but are grossly increased in volume by the edema that destroys them. Then dedifferentiation occurs and fibroblasts take their place.

The extent of the lesions is variable: they are usually found deep, close to the skeleton and of the interosseous membrane. Within the same muscle, some fibers may undergo necrosis while others retain their contractility. Seddon[11] has described three types of Volkmann's syndrome which may have a prognostic value (Figure 27.8):

- type 1: with diffuse and mild ischemia, and good recovery
- type 2: severe, with a localized ischemia and loss of function of the muscles involved
- type 3: the most severe, with diffuse damage and destruction of a high proportion of the long forearm muscles.

Contracture is interpreted as an accumulation of toxic metabolites which induces rigidity. The fibrosis follows the necrosis, and fixes this retraction. It is only at the beginning of the syndrome that muscle spasm exists, and fortunately this is reversible.

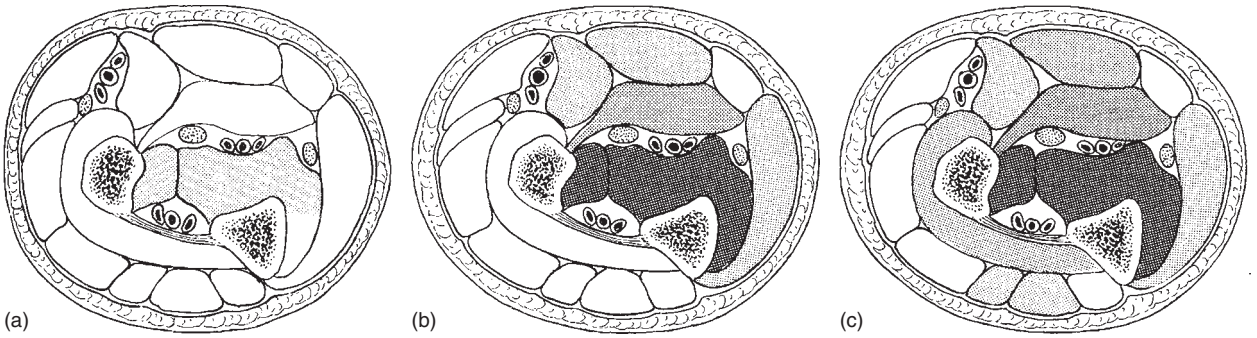


Figure 27.8

The extent of the necrosis according to Seddon[12], and the three degrees of gravity: (a) necrosis of the profundus muscles, (b) necrosis extended to all the muscles of the anterior forearm; (c) in very severe cases the posterior muscles are also involved. Reproduced from Tubiana.[5]

Paralysis

Palsies mainly affect the territory of the median nerve motor fibers leading to loss of opposition, and also sensitive fibers in the classic territory. This lesion of the median nerve is well understood, lying as it does in the ischemic area of the anterior forearm.

In contrast, the ulnar nerve suffers less frequently because it runs peripherally under the flexor carpi ulnaris, usually spared by the necrotic area. However, extended ischemia leads to sensory trouble in the two ulnar digit territories, associated with palsy of the intrinsic muscles of the hand. Testing must be done very carefully, and recorded well, to follow the course of the syndrome after treatment.

There are two possible causes of the nerve lesions:

1. The interference of their blood supply secondary to the local ischemia, which affects their conductibility. All the stages are possible: neuropraxia, axonotmesis (usually in Seddon's types 1 and 2, where recovery can occur), and necrosis, which is the rule in type 3.
2. The other possibility is direct compression of the nerve (in which compression of the specific artery of the median nerve can play a role leading to ischemia). However, direct compression of the trunk of the nerve is often the main trouble, and the physiologic consequences are well known in traumatic lesions.

The location is variable, in a number of specific anatomic sites (Figure 27.9): under the pronator teres, under the arch of the flexor digitorum superficialis, under the belly of the flexor pollicis longus, or in the carpal tunnel. This compression can occur under a mass of necrotic fibrotic tissue at a later period.

Motor and proprioceptive fibers are much more vulnerable than tactile and sympathetic fibers. Prognosis is severe when Wallerian degeneration occurs, or when the continuity of the trunk of the nerve no longer exists.

Electrophysiologic testing is very useful. Nerve conduction tests, when performed without delay, and during surgery,[12] can measure the extent of the denervation and can guide the technique. After surgery it is useful for assessing nerve recovery.

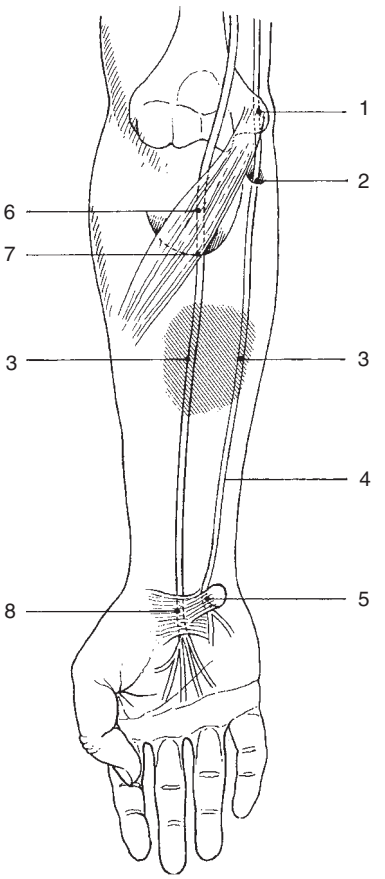


Figure 27.9

The possible locations of nerve compression: for the ulnar nerve (1) in the epicondylar tunnel; (2) at the level of the flexor carpi ulnaris arch; (3) under the necrotic mass; (4) at Guyon's tunnel; for the median nerve; (6) under the pronator teres; (7) under the flexor sublimis arch; (3) under a fibrotic necrotic mass; (8) and at the level of the carpal tunnel. Reproduced from Tubiana.[4]

Treatment

Even after the onset of the compartment syndrome, the treatment of the established syndrome remains an emergency. Decompression can be still useful for a few

days. It allows an objective exploration and appreciation of the lesions. It is obvious that appearance of muscle contracture and nerve problems demands specific procedures:

1. *Treatment of nerve lesions* Even if the local vascular aspect seems to improve spontaneously it is not reasonable to wait, because compression occurs frequently and early. Exploration of the median nerve must be systematic and a neurolysis is always performed, with preventive section of the muscular zones which are susceptible to compression. In some cases when necrosis seems significant it is difficult to decide whether the nerve damage is reversible. In these cases it is very useful to test the nerve conductivity during surgery and to extend the neurolysis to a level where conductivity is poor or absent. However, it does not seem reasonable to do a more radical procedure on the nerve (such as a resection with a suture or a graft) at this stage.
2. *Treatment of muscular lesions* If necrosis of muscles is obvious, dead tissues must be excised: indeed, there is nothing to be gained by leaving behind necrosed fibers that may be invaded by sclerosis and cause nerve compression. If a muscle is completely affected it is better to remove it and to restore its action later by the use of tendon transfers.

When retraction exists but is only moderate, a simple desinsertion of the epitrochlear muscles, as advocated by Goldner,[13] may be a simple procedure which can be associated with the extensive neurolysis (Figure 27.10). When the muscles are extremely retracted but still active (and this can be observed in late cases) the major desinsertion of the forearm muscles as described by Page[14] (publicized by Scaglietti[15] and introduced in France by Gosset[16])

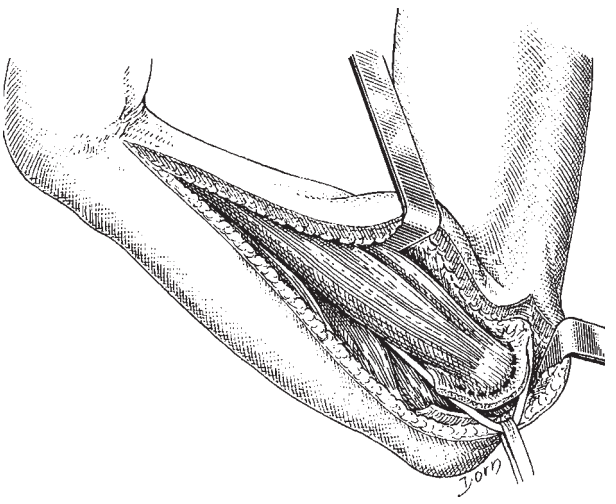


Figure 27.10

Technique of Goldner[13] to release contracture of the epicondylar muscles. Reproduced from Tubiana.[4]

may be indicated (see later). The good results of this technique when applied systematically by some authors in the early cases could probably also be obtained by the major exploration and neurolysis already described. And so, for us, it is a procedure only to be used in some sequelae of the syndrome.

In every case of Volkmann's contracture, early *physical therapy* is necessary to counteract retraction: the wrist is maintained in extension with the MP joints flexed, and attempts are made to straighten the proximal interphalangeal (PIP) joints by means of manipulation and also by elastic splints. Adduction of the thumb must also be avoided.

Treatment of Volkmann sequelae

The same two questions remain: how to treat the muscular retraction and the paralytic hand.

Muscle retraction

When flexors are still active It is always worthwhile increasing their range of motion and therefore their useful mobility. Bone shortening, whether at the forearm or at the wrist, has the disadvantage of also relaxing the extensors. Tendon lenghtening should be selective and must be carried out in such a way that the suture zones do not lie in the carpal tunnel, where the risk of adhesions is enormous. However, its disadvantage is that it may weaken the muscles involved.

The desinsertion of the anterior forearm muscles (Page–Scaglietti–Gosset)[14–16] seems to have its best indication at this late stage (Figure 27.11). The incision starts some 5 cm above the humeral trochlea, runs along the radial border of the forearm to the flexion crease of the wrist, and, if necessary, is continued into the hand, with the usual precautions at the palmar creases. The ulnar nerve is largely exposed. The epitrochlear muscles are desinserted, the biceps expansion divided, and the tendon of the brachioradialis is exposed. Then the median nerve is identified and the desinsertion of the antebrachial muscles can be performed – they are detached from the bones and the interosseous membrane. The only danger is the posterior interosseous artery, which is divided betwen ligatures. The muscle mass can then be pulled distally by several centimeters.

Gosset[16] advocated a lateral antebrachial incision which allows section of the fascia at the lateral border of the pronator quadratus and gives access to the brachioradialis and the flexors. Liberation of adhesions of the tendons in the carpal tunnel is sometimes necessary.

When long muscles are inactive It is possible, when they act as tenodesis, to change their effective sector. Arthrodesis of the wrist can be considered if the extensors are weak, and it is combined with resection of the proximal row of carpal bones. Modern authors propose microsurgical transfers of

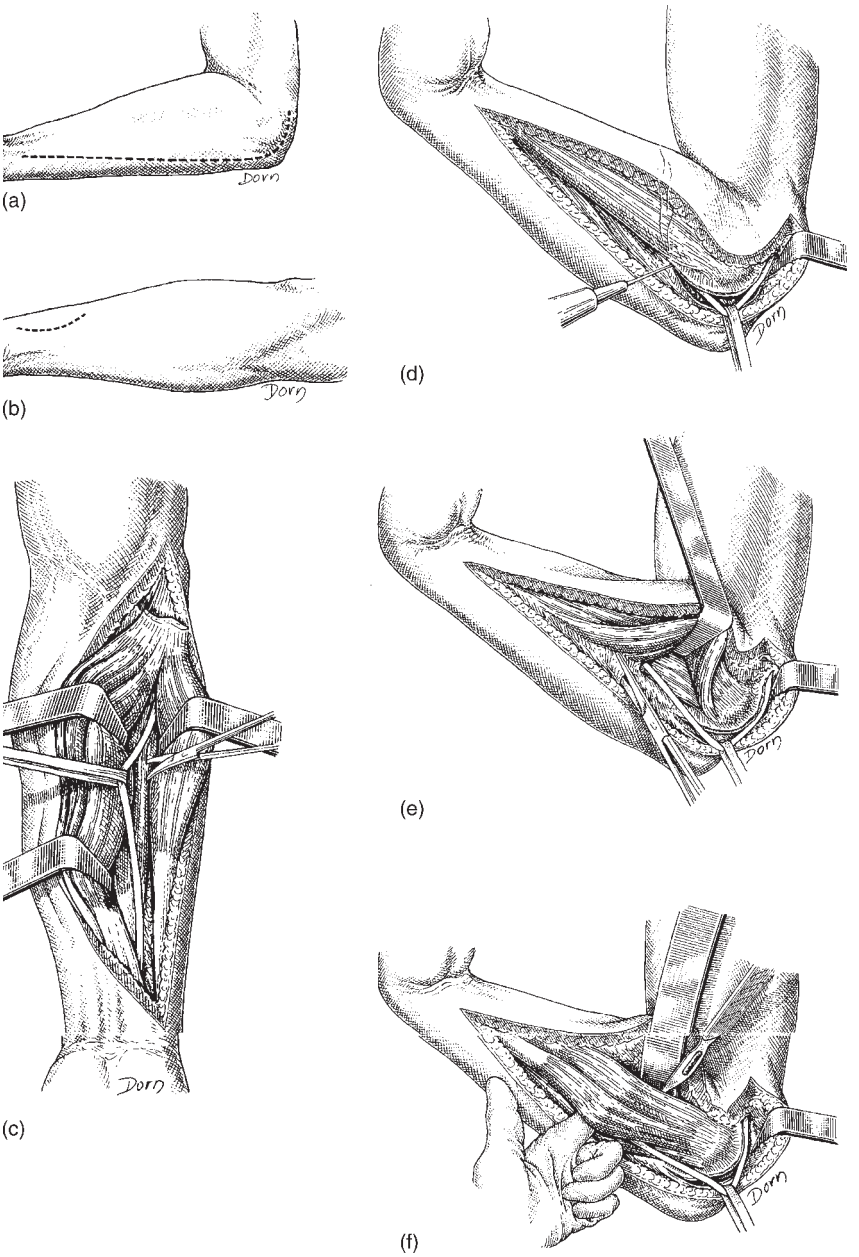


Figure 27.11

Technique of Page-Scaglietti-Gosset. Reproduced from Tubiana.[4]

foot muscles to replace the extrinsic muscles, mainly the flexors digitorum.

Paralyzed hand (see Chapter 7)

In brief, extrinsic muscles can be reactivated by transferring the extensor carpi radialis longus and brevis to the flexor pollicis longus and flexor digitorum communis respectively. However, in severe cases, these muscles may also be weakened. As for the intrinsic muscles, priority lies with restoration of opposition of the thumb. The long muscles often being unusable, in the case of a median isolated palsy a possible solution is to transfer the abductor digiti minimi, as described by Littler.[17] If there is no muscle available for transfer, intermetacarpal arthrodesis is worth considering.

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28 Compartment syndromes of the upper limb: management of initial and sequelae stages

Eduardo A Zancolli

The compartment syndrome can be defined as a clinical condition produced by a high fluid pressure within the inextensible fascial muscular spaces of a limb, sufficient to reduce blood supply and tissue viability of muscles and nerves. Severe cases of tissue compression may lead to muscle scarring and shortening, manifested by typical hand deformities, and even limb amputation may be required in cases of irreversible and extreme ischemia.

The two principal compartment syndromes of the upper limb are:

- Volkmann's compartment syndrome of the forearm,[1] in which the forearm muscles and nerves are affected, and
- Finochietto's compartment syndrome of the hand,[2] in which the interosseous and intrinsic muscle of the thenar eminence are principally involved.

Occasionally both syndromes may be associated. Our main purpose is to study the physiopathology and the surgical management of these compartment syndromes during their initial (vascular crisis) and established deformity stages.

Physiopathology and etiology of compartment syndromes

Initial interpretations of the mechanism of muscle ischemia in compartment syndromes were classically referred to as Volkmann's compartment syndrome of the forearm. In 1869, Richard von Volkmann of Halle, Germany, identified ischemia and paralysis of the forearm muscles to be the cause of certain post-traumatic hand deformities. In the *Zentralblatt für Chirurgie* of 1881[1] he wrote:

> A number of years ago I drew attention to the fact that the paralyses and contractures of the limbs which occasionally occur following bandaging that is too tight are not – as previously assumed – due to paralyses of the nerves through pressure, but arise due to a rapid and large-scale desintegration of the contractile substance, and the reactive and regenerative processes that follow it. The paralysis and contracture is to be regarded as purely myogenic.

He added that

> the prognosis for ischemic muscle paralysis and contracture depends entirely on the quantity of dead and disintegrating muscle fibers

and that

> severe and old cases involving the muscles of the hand and fingers are to be regarded as incurable, even under forceful passive extension of the shortened muscles, under chloroform narcosis.

Hildebrand (quoted by Fevre[3] was the first to use the term *Volkmann's ischemic contracture*, considering that muscle damage was the most important factor in hand deformity but that nerve damage was also a part in the clinical condition of the hand. Bardenheuer[4] and Murphy[5] recommended splitting the fascia of the forearm from the elbow distally in the initial phase of 'cyanosis, coolness and pain'. Griffiths,[6] during the era of World War II, proposed the etiology of Volkmann's contracture to be 'an arterial injury with reflex spasm of the collateral circulation', and that 'nerve compression were purely a secondary cause'. Griffiths' greatest contribution was the description of the so-called *four Ps* for diagnosis of acute compartment syndrome: '*pain*' – with passive extension – *painful or painless* onset, *pallor*, and *puffiness*'. Initial arterial injury was also mentioned by Foisie,[7] Holden,[8,9] Matsen,[10,11] Mubarak and Carroll,[12] Louis,[13] and Sarokhan and Eaton.[14]

Ottolenghi of Argentina wrote, in 1956,[15] an excellent paper on the prevention of Volkmann's ischemia after supracondylar fractures in chilhood. His program of treatment,

in cases with arterial injury or spasm and pulse deficit, was initial olecranon traction followed by surgical exploration of the brachial artery in the antecubital fossa if circulation did not recover in 30 minutes. His experience was based on 19 cases of arterial injury in a series of 372 distal humerus fractures (5.10%). Eichler and Lipscomb[16] concurred with Ottolenghi's principles, adding the division of the fascia overlying the flexor muscles of the forearm, but this approach was only indicated in 2 of their 64 cases.

Seddon[17] described the muscular ischemic area as a 'massive infarct' about the middle of the forearm, with the greatest circulatory deficit in the center of the lesion, while in the periphery the ischemia may be comparatively slight due to collateral circulation. The center of the infarct was described as a 'greenish-yellow pulpy mass', without fibrotic transformation for years; the periphery may show some partial muscle recovery, but develops diffuse fibrosis and progressive contracture. Seddon also referred to the frequency of nerve involvement, particularly the median nerve due to its course through the center of the infarct. Two types of nerve lesion were considered: (1) the irreversible lesion due to severe ischemia, and (2) the reversible lesion with Wallerian degeneration.

Ricardo Finochietto of Argentina described, in 1920,[2] the compartment syndrome localized in the intrinsic muscles – interossei and thenar – of the hand. He studied a case with multiple metacarpal fractures (see Figure 28.1). He interpreted the ischemia of the interosseous muscle as a consequence of vascular compression produced by edema and hematoma into the intermetacarpal spaces.

The modern concept of compartment syndrome physiopathology establishes that the principal cause of ischemia depends on an increase of fluid pressure within inextensible fascial spaces, sufficient to cause anoxia by occlusion of the circulation of the small vessels and consequent tissue damage. Postischemic swelling has been identified as an ischemia–edema cycle, initiated by different factors that increase the compartmental volume and reduce the blood supply to muscles and nerves.[18–20]

The blood supply deficit can be produced by one of two mechanisms.

- The first mechanism is related to an initial direct injury or disease of the principal vessels of the upper limb. Here the reduction in blood circulation causes anoxia that increases vascular endothelial permeability, which leads to progressive intramuscular edema with muscle compression and further anoxia by arterial and venous compression. Examples of this mechanism are all the factors that initially affect the arterial and venous circulation, such as displaced supracondylar fractures of the distal humerus in childhood (producing the occlusion or spasm of the brachial artery); prolonged limb compression, due to constricting casts or dressings,

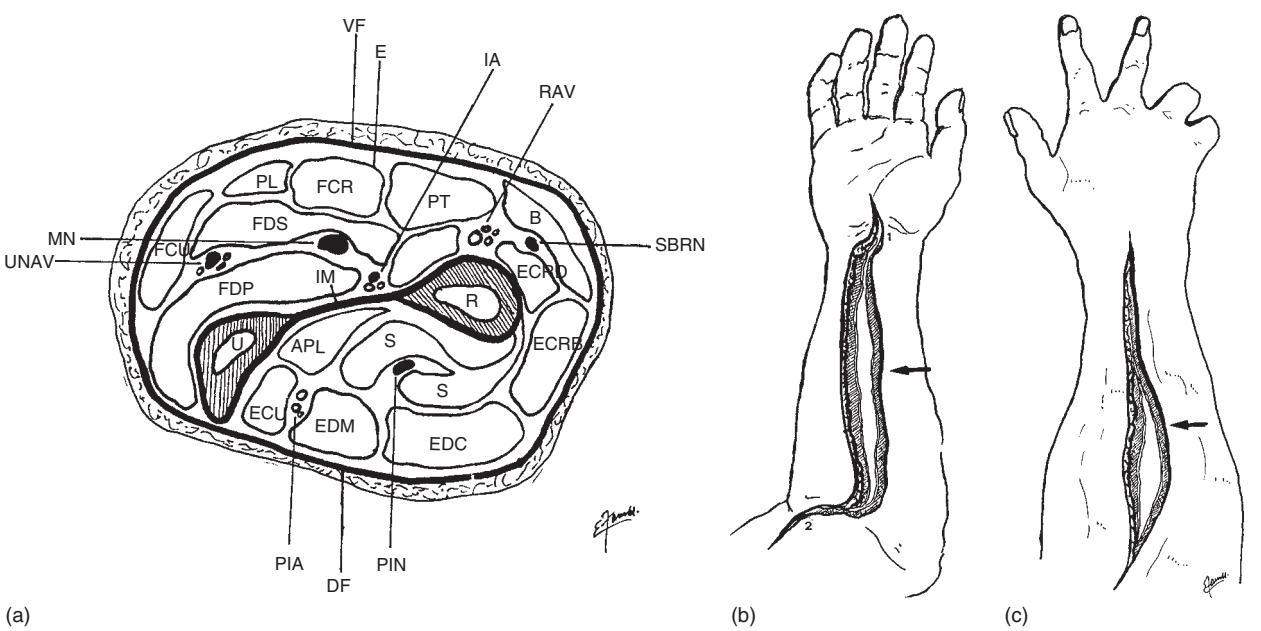


Figure 28.1

(a) Forearm compartments: cross-section of the proximal half of a right forearm. *Volar compartment*: superficial volar antebrachial fascia (VF) and epimysium of a muscle (E). Superficial muscles: flexor carpi ulnaris (FCU), palmaris longus (PL), flexor carpi radialis (FCR), pronator teres (PT), and brachioradialis (B). Deep muscles: flexor digitorum superficialis (FDS) and profundus (FDP). Superficial branch of the radial nerve (SBRN), median nerve (MN), radial artery and veins (RAV), ulnar nerve, artery and veins (UNAV). *Dorsal compartment*: thick superficial dorsal antebrachial fascia (DF). Superficial muscles: extensor carpi radialis longus (ECRL) and brevis (ECRB), extensor digitorum communis (EDC) and minimi (EDM), extensor carpi ulnaris (ECU). Deep muscles: supinator (S), abductor pollicis longus (APL). Posterior interosseous nerve (PIN). Posterior interosseous artery (PIA). Ulna (U). Radius (R). Interosseous membrane (IM). (b) Surgical approach to the volar compartment of the forearm extended to expose the carpal tunnel distally (1) and the antecubital fossa proximally (2). (c) Surgical approach to the dorsal compartment of the forearm.

or due to an unconscious or paralyzed patient lying on a limb (commonly associated with drug overdose); and arterial injections.[21,22]

- The second mechanism depends on a severe and persistent primary edema in close and inextensible fascial compartments, that initiates the ischemia–edema cycle with progressive muscle anoxia and compression of the small vessels. Examples of this mechanism are edema produced after severe crush injuries; progressive tissue infiltration in intravenous or arterial infusions (particularly in obtunded patients); exertional (exercise related); thermal injuries; hemorrhage accumulation due to hereditary bleeding disease; venous disease; frostbite; and fractures and bone surgery.

It is very important to note that, if the ischemia–edema cycle is not stopped by early fasciotomy, muscle and nerve ischemia will end with a Volkmann's contracture of the forearm, an intrinsic-plus deformity of the digits, or both. It is relevant to note that fasciotomy needs to be indicated before all the Griffiths' symptoms (four 'Ps') are present.

Much work has been done on the tolerance of muscle and nerve tissue to ischemia. It has been reported that nerve ischemia produces altered sensation within 30 minutes. Muscle ischemia produces functional changes after 4 to 6 hours, and compartment syndromes lasting more that 12 hours usually lead to permanent damage.[10,12,23–26] Recently compartment syndromes have been produced in animal experiments by intracompartmental infusion of blood or plasma to simulate a compartment syndrome. These studies have demonstrated peroneal nerve conduction block after 2.5 hours at 40 mmHg, and muscle necrosis after 8 hours at 40 mmHg.

Volkmann's compartment syndrome of the forearm (clinical stages)

Classic Volkmann's ischemic contracture of the forearm may pass through three stages:

1. initial or acute (vascular crisis)
2. evolutive or subacute (paralytic)
3. final or chronic (established deformity).

Initial or acute stage (vascular crisis)

This stage represents a vascular crisis where the most typical symptoms are:

- severe pain that increases with time, is located at the site of trauma and exacerbates with passive extension of the digits
- tense and tender compartments, edema preferentially located at the volar aspect of the forearm
- progressive hypoesthesia on the volar aspect of the digits
- cool and cyanotic forearm and hand, and weakness or paralysis and flexion contracture of the wrist and digit muscles.

Muscle contracture may be mild, moderate, or severe.[27] Absence of the radial pulse is an unreliable sign and has far less significance than progressive pain, paresthesia, and forearm swelling, because the radial artery passes relatively superficially through the forearm.[28]

The muscles most commonly affected by ischemia are those located at the deep part of the volar compartment of the forearm (flexor digitorum profundus (FDP) and flexor pollicis longus (FPL)), which are supplied by 'end arteries', branches of the ulnar and anterior interosseous artery. These are the muscles where the classical infarct described by Seddon[17] normally localizes. Usually, the flexor digitorum superficialis (FDS) and flexors of the wrist are involved. The nerves most commonly affected are the median and ulnar nerves. The median nerve may be compressed at the carpal tunnel. The muscles of the dorsal compartment of the forearm can also be involved (see Table 28.1). It is vital that all patients with swollen painful extremities and palpably tense compartments are closely controlled.

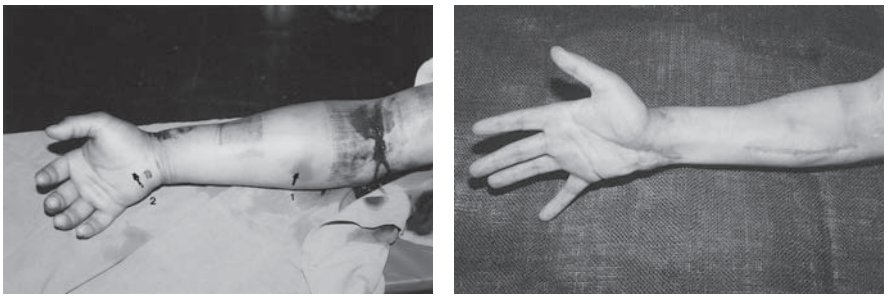
Most compartment syndromes can be diagnosed clinically, but tissue pressure measurement is a useful adjunct in an acute compartment syndrome, particularly in equivocal cases or in those in which the patient is comatose or unable to cooperate. The method used by the author is that advocated by Whitesides et al.[29] Others methods include the wick-catheter technique developed by Mubarak et al[30,31] and the continuous technique of Matsen.[11] The injection method of Whitesides et al consists of a needle, plastic tubing filled with air, and saline attached to a mercury manometer to measure the pressure – thus it uses inexpensive equipment.

Normal tissue pressure is 0 to 10 mmHg, it rises with muscle contraction and almost immediately drops with relaxation. A resting compartment pressure of over 30 mmHg has been shown to cause pain, paresthesias, and interference with capillary circulation; between 10 and 30 mmHg of diastolic pressure produces inadequate perfusion and relative ischemia. There is general agreement that a tissue pressure from 30 to 50 mmHg, maintained for 4 to 8 hours, produces muscle necrosis.[20] Mubarak and Hargens[25] consider that a compartment pressure of 30 mmHg or greater is indication of compartment syndrome and fasciotomy.

Knowledge of limb compartment anatomy is of great value in the diagnosis and treatment of acute compartment syndromes. The forearm has two principal compartments, volar and dorsal (Figure 28.2a). The volar compartment is divided into two parts: superficial and deep. The superficial part contains the flexor carpi ulnaris (FCU) and radialis (FCR), pronator teres (PT), palmaris longus (PL) and brachioradialis (B) muscles. The deep part contains the FDS and FDP muscles. These muscles are supplied by the radial

Table 28.1 *Grouping of established deformities following compartment syndromes of the upper limb. Based on a study of 85 cases*[42]

GROUP A	Volkmann's compartment syndrome of the forearm (33 cases)	Ventral muscles (29 cases)	Classical (28 cases)	1. Normal intrinsic muscles 2. Intrinsic paralysis (fingers and/or thumb) 3. Intrinsic contracture (fingers and/or thumb) 4. Associations
			Localized (flexor digitorum profundus and flexor pollocis longus) (one case)	
		Ventral and dorsal muscles (4 cases)		
		Interosseous muscles (subtypes I, II, III)		
GROUP B	Finochietto's compartment syndrome of the hand (52 cases)		medial thenar muscles both lateral	
		hypothenar muscles		



(a) (b)

Figure 28.2

(a) Combined acute compartment syndromes of the upper limb: forearm (1) and hand (2) in a 32-year-old male following a crush injury in a traffic accident. The patient was treated 3 hours after the initial injury. Fasciotomies were performed in the volar aspect of the forearm and hand. The carpal tunnel was opened. The wounds were closed 9 days after surgery.
(b) Complete recovery of hand function after 4 months.

and ulnar arteries. The anterior interosseous artery supplies the deep muscles in particular, and it can be compressed to elevate forearm pressure and explain the frequent ischemia of the deep volar muscles. The dorsal compartment of the forearm contains superficial (extensor carpi radialis longus (ECRL) and brevis (ECRB), extensor carpi ulnaris (ECU), extensor digitorum communis (EDC), and extensor digiti minimi (EDM)) and deep muscles (abductor pollicis longus (APL), extensor pollicis brevis (EPB) and extensor indicis (EI)) (Figure 28.2a).

The hand at the mid-palmar part has six compartments that are described below.

The goal of treatment during the acute stage is to re-establish the circulation of the affected muscles and nerves. In the case of a displaced supracondylar fracture, the principal corrective measures to be instituted are: (1) removal of all constricting bandages to decrease pressure and (2) reduction of the fracture by gentle manipulation or by Dunlop's traction. These measures frequently restore circulation and radial pulse, and reduce symptoms. The elbow is inmobilized to prevent acute flexion for the first few days. Pinning is an excellent indication to maintain fracture reduction. The limb is not elevated but rather is placed at heart level. Elevation significantly increases anoxia in the face of high intercompartmental pressures.[10]

If these measures do not improve circulation in less than 30 minutes (Ottolenghi's protocol), bringing relief of symptoms, surgical exploration of the brachial artery and median nerve at the elbow level and a fasciotomy of the volar forearm compartment are undertaken. If a lesion of the brachial artery is present, the damage is located and lacerations are repaired. Localized narrowing in the artery may indicate a tearing of the intimal lining, and a clot is frequently palpable. In this case, the artery is opened,

the clot removed, and blood flow re-established by graft or repair. Mechanical spasm may frequently be overcome by injecting a bolus of fluid through the narrow segment from above.[8,9,32]

In all compartment syndromes with severe forearm symptomatology, and with an intracompartment pressure greater than 30 mmHg, an extensive fasciotomy is indicated to prevent irreversible tissue damage. Both the superficial and deep flexor fascial spaces are opened through a long incision from the flexor crease of the elbow distally to the base of the hand (Figure 28.2b and c). The carpal tunnel is opened. Individual muscle spaces are palpated, and the epimysium is opened if there is any doubt as to increased pressure under it.[17] The median and ulnar nerves are decompressed. If the dorsal and lateral fascial spaces are tight, they are also opened. About 10 to 15 minutes after muscle and nerve release, small vessel bleeding will increase as circulation returns. The wound is left open and moistened dressing is applied. In those cases with severe acidosis, hyperkalemia and impending myoglobinuric renal failure must be corrected before fasciotomy.[21,22]

Delayed direct skin closure or grafting is done in a few days, after edema has subsided. Conservative treatment – rehabilitation – is indicated immediately after the end of the vascular crisis. In the most favorable cases, recovery depends on the reversible damage suffered by nerves and muscles (Figure 28.3).

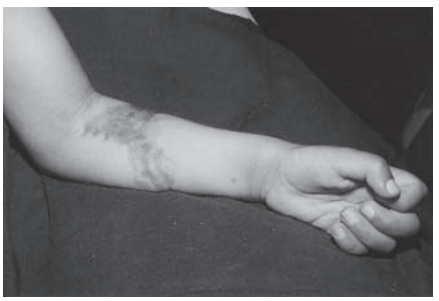
In cases without fracture and with mild intracompartment pressure, and if the initiating event has occurred within a few hours, a manual 'pumping' of the upper limb for a few minutes may alleviate muscle compression and obviate fasciotomy.[22]

Evolutive or subacute stage (paralytic)

This stage has somewhat vague boundaries, ranging from 1 to 2 days when muscular and nerve sequelae are present, up to 3 to 6 months, when spontaneous recovery of muscles and nerve ends and established final deformity appears.[17]

The main goals during the evolutive period are: (1) nerve release; (2) excision of deep forearm scar tissue or muscle infarct;[17,33] (3) orthosis with rubber band traction; and (4) movement of all joints to minimize muscle contracture, digital joint stiffness, and nerve function.

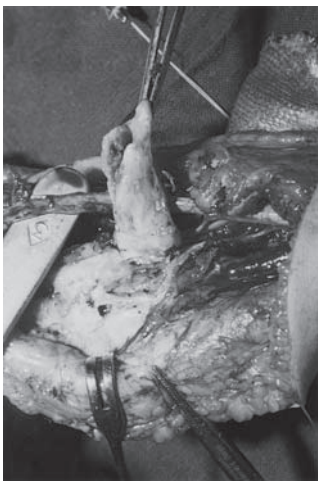
Nerve release is indicated – particularly for the median nerve – when spontaneous recovery does not occur. In this situation, excision of the scar tissue and the muscle infarct is performed simultaneously with neurolysis. The nerves must be displaced from within the flexor muscle mass and allowed to lie in tissues free from any fibrous incarceration.[34] The median nerve is usually compressed beneath the pronator teres or the arcade of the FDS muscle (Figure 28.4). In very severe lesions (axonotmesis III and



(a)

(b)

(c)



(d)

Figure 28.3

(a) Supracondylar humerus fracture in a 3-year-old boy complicated by Volkmann's compartment syndrome and compression of the median nerve, 4 months after injury (evolutive period). (b) The forearm was surgically explored. The median nerve (1) was found compressed by scar tissue at the middle of the forearm (arrow). (c) Severe compression of the median nerve is observed after neurolysis (arrow). (d) Excision of the deep scar tissue. Tendon transfers to restore digital function were performed 5 months later.

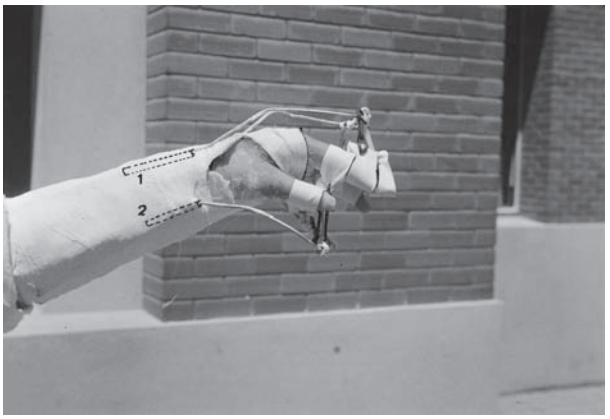


Figure 28.4

Plaster orthosis with rubber bands to extend the digits during the evolutive period in a Volkmann's ischemic contracture of the forearm. Plaster of Paris can be molded to obtain an exact fit. The outriggers are placed with the correct angle of pull. They can be removed to facilitate sleep and dressing. The dotted lines (1 and 2) indicate aluminum tubes – included in the plaster – to allow remotion of the traction wires when necessary.

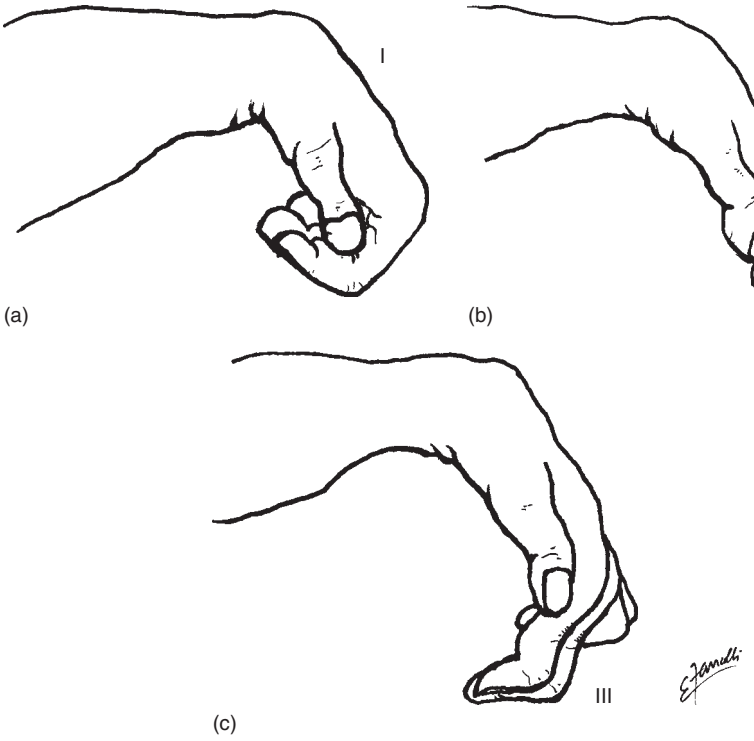


Figure 28.5

Types of Volkmann's ischemic contractures of the forearm. (a) Type I with normal intrinsic muscles (see Figure 28.8). (b) Type II with intrinsic muscle paralysis of all the digits (see Figures 28.10 and 28.11) or exclusively the ulnar fingers because of isolated ulnar nerve paralysis. (c) Type III with combined contracture of the forearm muscles and hand intrinsic muscles in the radial digits and clawhand in the ulnar fingers (see Figures 28.12 and 28.13).

neurotmesis), the nerve should be replaced by nerve grafts. After this procedure perfect tactile gnosis cannot be expected, but in a child the prognosis for a return of useful sensibility is very favorable.[28]

The second indication for early surgery during the evolutive period is a severe flexion contracture of the digits, for which stretching exercises and splinting have been ineffective (Figure 28.5). In these cases, all fibrotic tissues and the muscle infarct are excised, but no tendon transfer should be performed.[17] It is preferable to delay tendon transfer for a later stage.

Final or sequelae stage: classification and surgical treatment

Different classifications of final Volkmann's deformity have been published. In 1944 Bunnell (quoted by Boyes[35]) classified Volkmann's contracture sequelae into *simple* and *severe*, according to the characteristics of the deformity at the forearm and hand. Pedemonte,[36] of Uruguay, divided the hand deformity into two types: (1) classic or favorable and (2) with clawhand. Seddon[17] suggested three groups, in accordance with the severity of the ischemia: group I, with diffuse tissue ischemia but without infarct of the deep muscles of the forearm, in which spontaneous recovery frequently occurs; group II, with the typical muscular infarct, and with or without nerve damage; and group III, with diffuse fibrosis and severe paralysis and hand deformity.

The author[37–44] has subclassified the established deformity of Volkmann's contracture into three types, based on the condition of intrinsic muscles of the hand: type I, with normal intrinsic muscles; type II, with paralyzed intrinsic muscles, and type III, with intrinsic muscle contracture (Table 28.1 and Figure 28.5). These types of established deformities may be combined, usually type I with type II, and type II with type III (associated type) (Figures 28.6–28.8).

(a)

(b)

Figure 28.6

(a) A 12-year-old girl with an established deformity of the hand due to an ischemic contracture of the volar and dorsal forearm muscles caused by a crushing injury of the forearm. The intrinsic muscles of the hand are preserved. Volar view of the hand. (b) Ulnar view of the deformity: extension contracture of the wrist and flexion contracture of the digits.

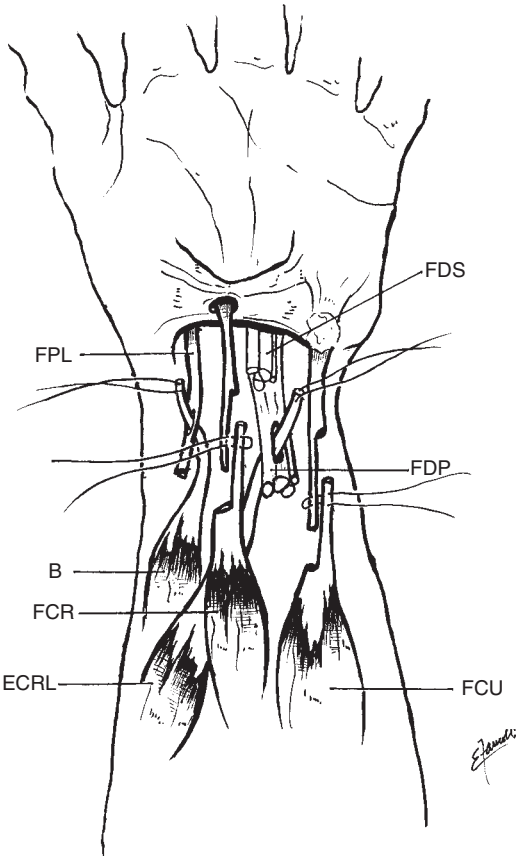


Figure 28.7

Excision-reconstruction surgical procedure to correct a type I Volkmann's ischemic contracture of the volar muscles of the forearm. The usual technique is as follows: 1, excision of the fibrotic or muscle infart of the deep muscles of the forearm; 2, Z lengthening of the flexor carpi radialis (FCR) and flexor carpi ulnaris (FCU) tendons; 3, tendon transfer of the brachioradialis (B) to the flexor pollicis longus (FPL) tendon, and 4, tendon transfer of the extensor carpi radialis longus (ECRL) to the distal stumps of the flexor digitorum profundus (FDP) tendons. FDS, flexor digitorum superficialis.

We consider this classification to be a great help in the diagnosis of muscle and nerve pathology, and in the program of surgical reconstruction. Uncommonly, the ischemia of the volar muscles of the forearm is associated with ischemia and contracture of the dorsal muscles (Table 28.1 and Figure 28.6).

In a series of 85 cases of established deformities following compartment syndromes of the upper limb investigated by the author,[42] 33 cases (38.8%) belonged to Volkmann's ischemic contracture of the forearm. In the great majority of these cases (28 cases) (96.6%), the pathology was localized in the ventral muscles of the forearm – FDP, FDS, FPL, PT, and flexors of the wrist (classic type). In only one case (3.4%) were the FDP and the FPL the only ventral muscles involved (localized type). In four cases (12%), the dorsal muscles of the forearm were associated with contracture of the ventral muscles. Of the total series, 52 cases belonged to Finochietto's ischemic contracture of the intrinsic muscles of the hand. Usually the interossei and the thenar muscles are involved. In only one case was the abductor digiti quinti was contracted. In this case, the little finger was in the permanent abduction position.[42]

Type I (contracture of forearm muscles with normal intrinsic muscles)

In this type of Volkmann's deformity, the volar deep muscles of the forearm are most commonly affected. The wrist and fingers are deformed in flexion, and the forearm is fixed in pronation. The median and ulnar nerves are in good condition. Usually there is no finger joint stiffness. Surgery is indicated after maximum spontaneous muscle recovery, but before definitive articular stiffness of the finger occurs. Preserving passive mobility of the finger joints during the evolutive stage is of paramount importance to permit surgical reconstruction of the established deformity. Type I deformity is a most favorable condition to correct,

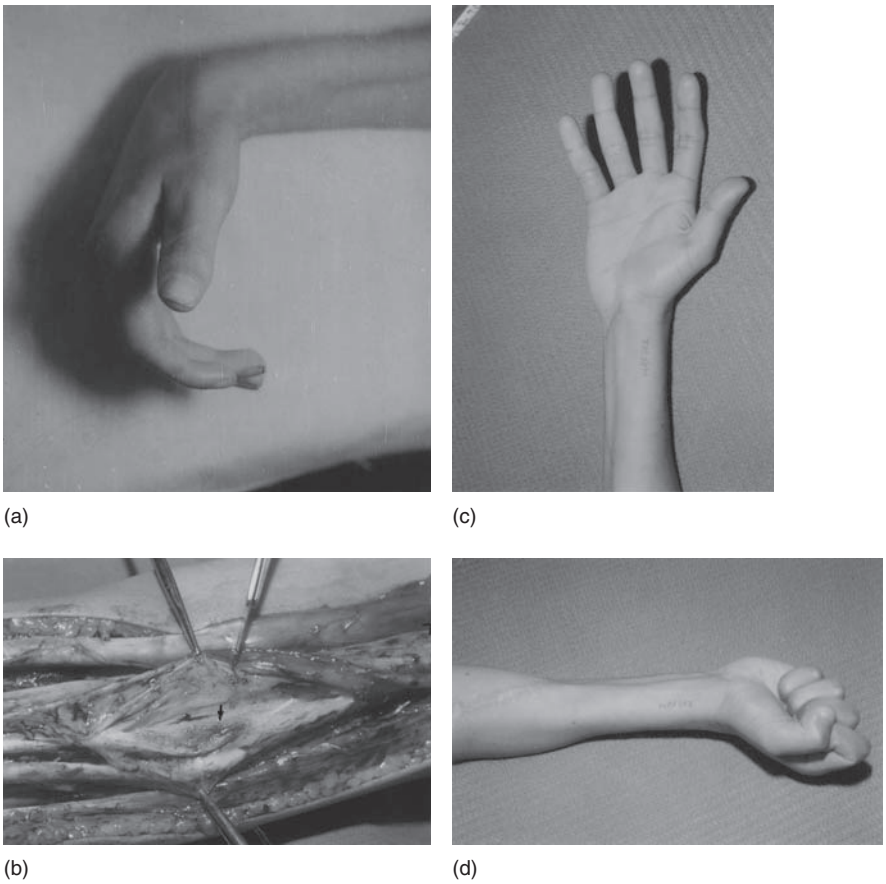


Figure 28.8

(a) Established flexion contracture of the wrist and digits in a type I Volkmann's ischemic contracture, in an 18-year-old boy, caused by a displaced fracture of the distal humerus 8 months earlier. (b) Excision of the deep volar fibrotic forearm muscles followed by a reconstruction procedure, as shown in Figure 28.7. Release of the median nerve (arrow). (c,d) Excellent hand function 2 years following surgery.

since intrinsic function of the hand and sensation of the digits are preserved.

Surgical reconstruction is directed to the forearm muscles. The surgical procedure the author[37,39,41,42,44] prefers is excision of the retracted digital muscles associated with lengthening of wrist flexor tendons and tendon transfers to restore digital flexion. This method was developed by Parkes[45] and Seddon.[17] The author[42] advocates excision-reconstruction of the forearm muscles when the following conditions are present:

1. mild or severe retraction of the long flexor muscles
2. available muscles for transfer
3. little or no voluntary activity of the digital flexor muscles after sufficient time for recovery has been given.

The technique consists of 'Z' lengthening of the principal flexor tendons of the wrist (FCR and FCU) and the FPL. When this last muscle is completely fibrotic or paralyzed, it is sectioned and activated by the transfer of the brachioradialis. The palmaris longus is lengthened or sectioned. The flexor digitorum sublimes are excised completely to the limits of the forearm incision. The ischemic profundus tendons are excised at their musculotendinous junction, leaving the distal stump tendons with sufficient length to permit their activation by tendon transfers. If the flexor tendons of the fingers are only divided, and not excised, partial recurrence of the deformity is possible.

After flexion deformity of the wrist and digits has been corrected the lengthened wrist flexor tendons are repaired and the ECRL is transferred to the distal stumps of the FDP tendons. The brachioradialis is transferred to the FPL if this tendon has been excised or paralyzed (Figures 28.9 and 28.10).

Postoperative re-education is usually simple after the procedure of excision-reconstruction in type I deformity, since the transferred ECRL is a synergistic motor of finger flexion and the intrinsic function of the hand and digital sensation are preserved.

The author considers that there is little indication for the classic proximal muscle slide operation,[46] as it allows for little control of the release and may lead to muscle weakness. It may also affect the blood supply of the released flexor muscle bellies. The only indication for this procedure could be in a mild deformity with preserved good function of the retracted flexor muscles, but even in this case the author has obtained better results with the excision-reconstruction procedure.

Occasionally the FDS muscles are partially retracted with good voluntary contraction. In this situation their tendons are divided well distally in the forearm and transferred to the distal tendons of the FDP, which have been divided proximally. This technique represents an

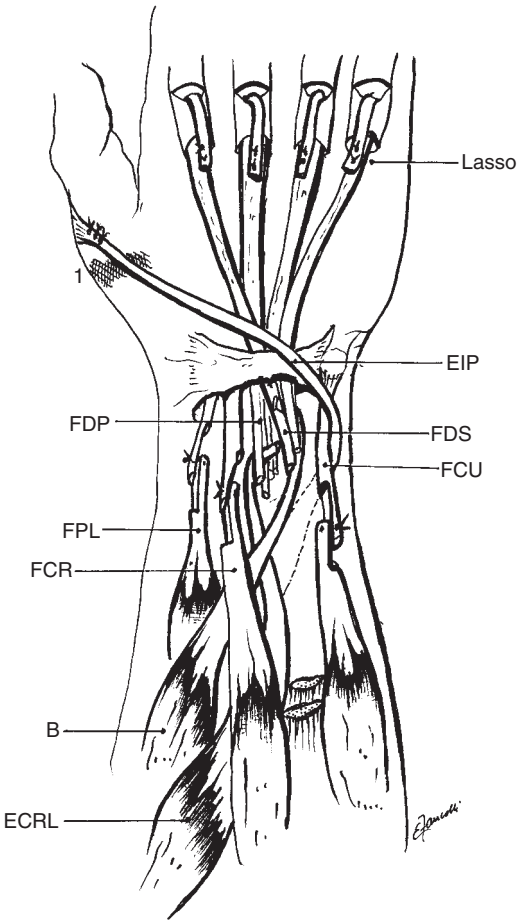


Figure 28.9

Surgical procedure for a type II established Volkmann's contracture, with opposition paralysis and intrinsic 'clawhand' – without finger joint complications. Z lengthening of the flexor pollicis longus (FPL) (if active), flexor carpi radialis (FCR), and flexor carpi ulnaris (FCU) tendons. Tendon transfer of the extensor carpi radialis longus (ECRL) to the distal stumps of flexor digitorum profundus (FDP) tendons. Transfer of brachioradialis (B) to flexor digitorum superficialis (FDS) tendons, which are fixed to an A1 pulley in each finger to correct the clawhand deformity (indirect 'lasso' procedure). Opposition is restored by transferring the extensor indicis propius (EIP) to the extensor apparatus of the thumb with fusion to its metacarpophalangeal joint (1).

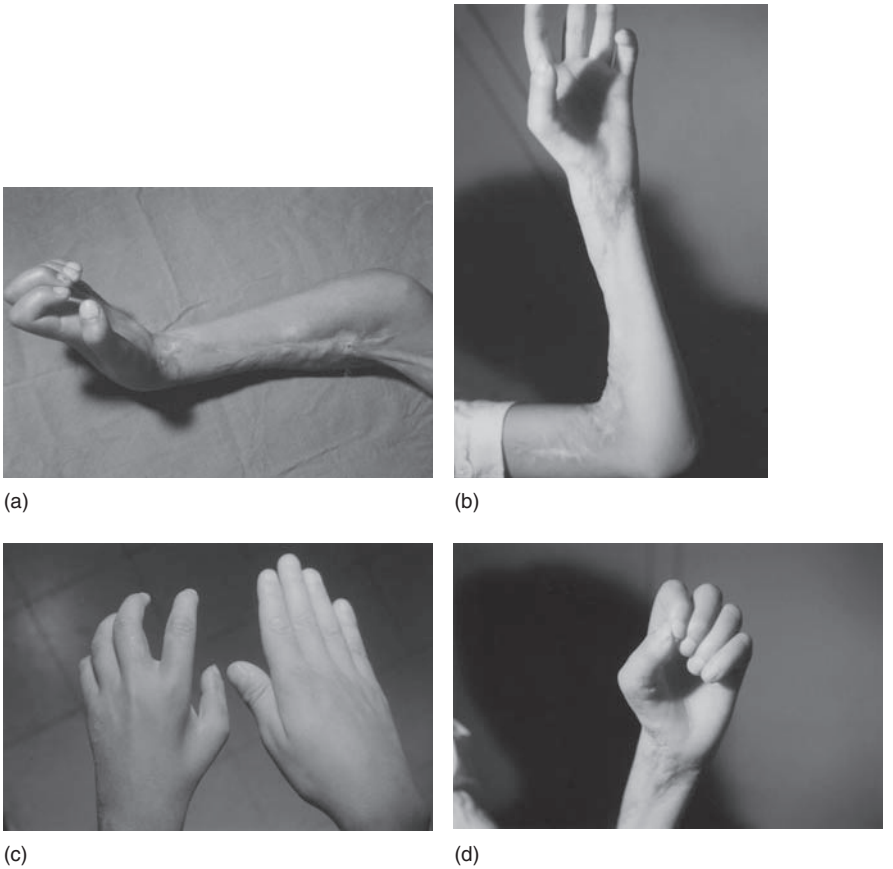


Figure 28.10

Severe established deformity of a type II Volkmann's ischemic contracture in a 15-year-old boy due to a crush injury at the forearm and elbow, with fracture of both forearm bones, 4 years previously. Skin scarring consecutive to the injury. Complete paralysis of the intrinsic muscles of the fingers – clawhand – and opposition paralysis. (b) Active opening of the hand 2 years after an excision-reconstruction procedure and release of the median and ulnar nerves, surrounded by fibrotic tissue. Clawhand corrected by metacarpophalangeal capsulodesis and opposition using a transfer of the extensor indicis propius around the ulnar border of the wrist. (c) Dorsal view of the hand. (d) Opposition obtained.

'intertendinous lengthening', described by the author in 1957[47] and 1979.[42]

Type II (contracture of forearm muscles with intrinsic paralysis of the hand)

Flexion contracture of the wrist and digital flexor muscles with fixed pronation of the forearm and intrinsic paralysis of the hand – clawhand and thenar paralysis – represents the classic clinical picture of this type of sequelae. Volar nerve trunks – median and ulnar – are frequently involved. Intrinsic clawing may be simple or complicated – finger stiffness of the metacarpophalangeal and proximal interphalangeal joint or attenuation of the extensor aponeurosis over the proximal interphalangeal joints. Opponens paralysis of the thumb may or may not be associated with the clawhand. In general, it is preferable to deal with the motor problem of an ulnar nerve clawhand rather than with a severe median nerve lesion, as the latter generally presents difficulty in the restoration of complete hand sensation.

For patients with type II deformity, treatment is divided into two surgical stages. During the first stage, the retracted forearm muscles are corrected by the excision-reconstruction procedure, as described in type I deformity. During the postoperative period, finger re-education is encouraged for several months and splinting is employed to prevent stiff 'clawing' of the fingers. During the second stage, intrinsic muscle paralysis is corrected. The surgical method to employ depends on available muscular motors for transfers and the clinical condition of the fingers and thumb.

Clawhand, without significant joint complications, is treated like any other intrinsic paralysis by active or passive surgical procedures. The 'lasso' operation[42,48] and metacarpophalangeal capsulodesis with bone fixation[41,42,49,50] are the author's procedures of choice. If a motor is available – usually the brachioradialis – the indirect lasso technique is the preferred method. In this technique the brachioradialis is lengthened, using the distal stumps of the four flexor superficialis tendons or tendon grafts, and fixed distally around the A1 pulley of the flexor tendon sheath of each finger (Figures 28.7, 28.11, and 28.12). When no motors are available, a metacarpophalangeal joint capsuloplasty, with bony fixation, is indicated. Capsulectomy of the metacarpophalangeal joints is contraindicated when the interosseous muscles are paralyzed.

Paralysis of the intrinsic muscles of the thumb is corrected by tendon transfers. The extensor indicis proprius around the ulnar border of the wrist is an excellent motor to restore opposition[39,42,51] (Figure 28.11). This transfer is usually combined with fusion of the metacarpophalangeal joint of the thumb. If there is an adduction contracture of the thumb, it must be corrected simultaneously with the opponens transfer. In these cases the adductor and flexor pollicis brevis muscles are released, and occasionally the retracted dorsal trapeziometacarpal ligament is divided. A thenar web release requiring major skin flaps demands a previous surgical procedure.

Type III (contracture of forearm muscles with combined intrinsic contracture and paralysis of the intrinsic muscles of the hand)

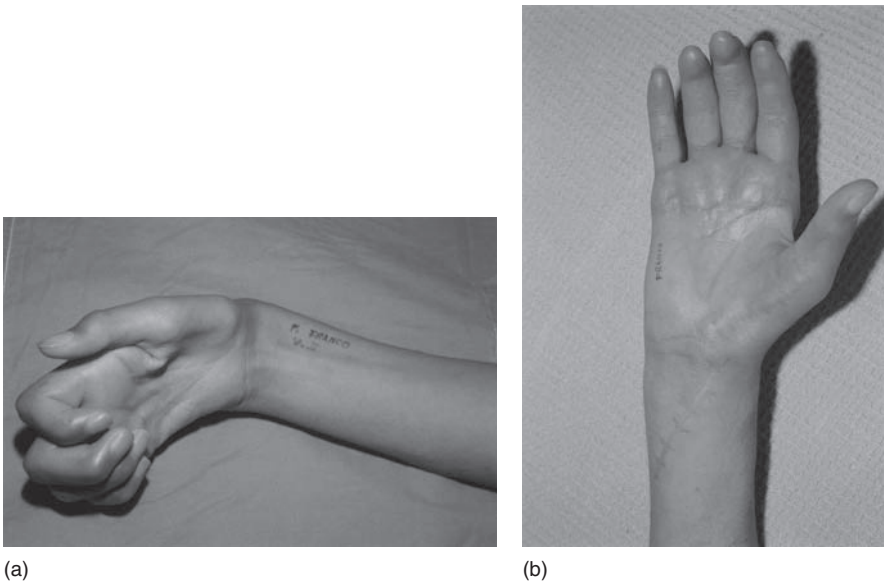
This type of deformity is uncommon. The intrinsic contracture of the hand is frequently localized in the radial digits. The fingers show the metacarpal joints contracted in flexion, the proximal interphalangeal joints contracted in extension, and the distal interphalangeal joints in flexion due to contracture of the FDP muscle. The thumb may be adducted into the palm with the metacarpophalangeal joint flexed and the interphalangeal joint hyperextended. The ulnar fingers usually present a clawhand deformity due to ulnar nerve paralysis (combined type II and III) (Figures 28.6, 28.8, and 28.13).

In this type of deformity, the contractures of the forearm and hand intrinsic muscles are corrected during the same surgical procedure. It would be impossible to obtain finger function with only the forearm excision-reconstruction procedure if the interosseous muscles of the fingers are contracted.[39] Forearm excision-reconstruction is performed as previously described (Figures 28.9 and 28.11). The technique used to release the interosseous muscles depends on the severity of the contracture. In mild cases the Littler technique,[28] consisting of the excision of the intrinsic lateral brands and oblique fibers of the extensor apparatus, is indicated (distal intrinsic release) (Figure 28.14a). In cases with flexion contracture of the metacarpophalangeal joints, the retracted and paralyzed interosseous tendons are excised at the level of the neck of the metacarpal bones (proximal intrinsic release)[42] (Figure 28.14b). In thumb adducted contracture the retracted intrinsic muscles are released. A skin flap may be associated.

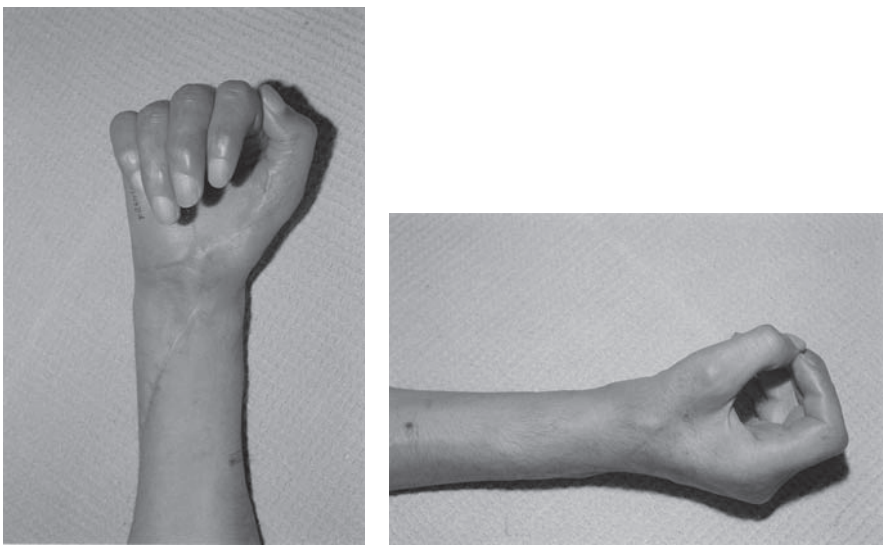
To restore sensation, nerve grafts may be needed to bridge defects. With major segmental loss in both median and ulnar nerves, the pedicle nerve graft – a technique devised by Strange[52] – may be indicated. This is a two-stage procedure that uses the ulnar to replace the median nerve in such a way as to preserve neural vascularization. In our experience, this technique can yield enough sensation to protect the digits during daily activities. It represents a suitable procedure for severe nerve graps. Similar nerve procedures are employed in type II established Volkmann's contracture.

Useful signs in the diagnosis of the different types of Volkmann's established deformities

Specific signs are helpful for each type of Volkmann's deformity. In type I, some finger extension is obtained by increasing wrist flexion. In type II, the fingers obtain interphalangeal active extension when the wrist is passively flexed, and hyperextension of the proximal phalanx is prevented (Bouvier's maneuver).[42,53] In stiff clawfingers Bouvier's maneuver is negative. In type III deformity, proximal interphalangeal flexion increases only after the metacarpophalangeal joints are passively flexed – as in other



(a) (b)



(c) (d)

Figure 28.11

(a) Established deformity of a Volkmann's type II ischemic contracture after a bycicle accident, with fracture of radius and ulnar bones, in an 18-year-old male. Severe intrinsic clawhand and opposition paralysis of the thenar muscles. (b) Hand release after an excision-reconstruction procedure as shown in Figure 28.9. Neurolysis of the median and ulnar nerves – 8 months after surgery. (c) Digital flexion. (d) Opposition.

intrinsic-plus contractures (Finochietto's test).[2,42] In these cases, passive extension of the proximal phalanx of the fingers increases hyperextension of the middle joint and flexion of the distal joint.

Finochietto's compartment syndrome of the hand

Contracture of the intrinsic muscles of the hand is the result of a compartment syndrome of the hand with ischemia. Whether a particular case is in the initial ischemic or the established deformity stage should be clearly determined, because appropriate treatment is specific for each stage. The most frequent causes of an intrinsic compartment syndrome of the hand are multiple metacarpal fractures, fractures of the distal radius or carpal bones, direct arterial or burn injuries, prolonged hand compression, severe and persisting edema or hematoma in the deep fascial spaces, and crush injuries.

Post-traumatic ischemic contracture of the intrinsic muscles of the hand was initially described by Ricardo Finochietto in 1920.[2] He presented his first communication on the subject to the Buenos Aires Surgical Society with the name of 'Volkmann's retraction of the intrinsic muscles of the hand'. In his presentation (concerning one clinical case with multiple metacarpal fractures), Finochietto described:

1. the ischemic origin of the intrinsic muscular anoxia and contracture
2. the precise clinical picture of the deformity
3. the specific clinical sign (intrinsic-plus test) to demonstrate the interosseous muscular contracture
4. the surgical procedure, consisting of excision of the retracted interosseous and thenar muscles to correct the deformity (Figure 28.14).

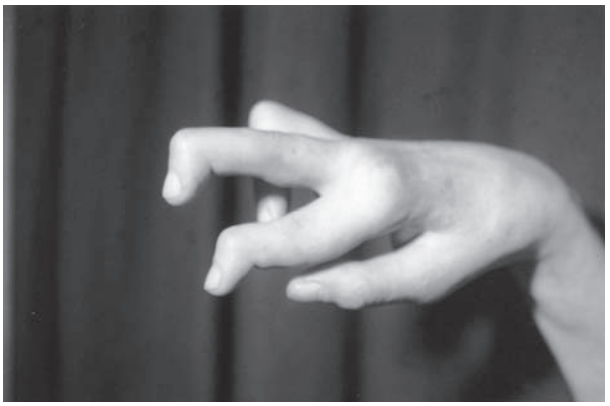


Figure 28.12

A type III Volkmann's established deformity of the right hand in a 22-year-old male. The deformity shows contracture of the wrist flexors. Intrinsic-plus deformity of the index and middle fingers with permanent extension of the proximal interphalangeal joints and fixed flexion of the distal interphalangeal joints due to contracture of their flexor digital profundus muscles. Mild contracture of the thumb interphalangeal joint. Clawhand deformity of the ring and fifth fingers due to ulnar nerve intrinsic paralysis and opposition paralysis(6). This case represents a combination of types II and III ischemic contracture (forearm and hand).

Before Finochietto's presentation, the deformity had been variously attributed to a lesion of the spinal cord,[54] to an 'irritation' or traumatic lesion of the ulnar nerve,[55,56] and to hysteria.[57]

Twenty-eight years after Finochietto's presentation, Bunnell et al[58] remarked on the significant incidence of contracture of the intrinsic muscles in the hand of soldiers wounded in World War II. They described the release of the interosseous muscles at their proximal metacarpal attachments as an 'interosseous slide procedure'.[35] In 1949, Littler[59] described the excision of the lateral bands and the oblique fibers of the extensor apparatus to correct interosseous muscle contracture of the fingers. In 1979, the author[42] presented the technique of excision of the distal interosseous tendons to correct the flexed metacarpophalangeal joint in severe cases. More recently, other authors have referred to this syndrome.[60,61] Thermal injuries of the upper limb have been considered a very frequent cause of contracture of the interosseous muscles and hand dysfunction.[19,40,42,62,63] Necrosis of the interosseous muscles after unaccustomed work has also been mentioned as a cause of the intrinsic contracture syndrome (Vogt, quoted by Reid and Travis[64]).

Material

The author's clinical material consists of a series of 68 cases of intrinsic muscle contracture observed over more than 30 years, published in 1990[43] – 52 cases were presented in 1979[42] (see Table 28.1). In the last series the etiology of muscular ischemia of the intrinsic muscles was referred to crush injuries with or without carpal or metacarpal fractures (10 cases); mild trauma but with persisting edema of the hand (9 cases); forearm and Colles' fractures, particularly in patients of advanced age (9 cases); thermal injuries involving all or almost all the upper limb (25 cases); tight plaster casts or constricting bandages applied to the upper limb or hand (7 cases); injury or obstruction of the principal arteries of the upper limb, subclavian (3 cases), axillary (1 case); collagenous diseases (2 cases); and tetraplegia after spinal cord lesion (2 cases). This series shows that the most frequent causes of ischemia of the intrinsic muscles of the hand were in severe thermal injuries of the hand and that the principal mechanism was reduction of the blood supply of the interosseous muscles due to persistent and severe intermetacarpal edema.[62,63]

In the majority of the author's cases, the interosseous muscles of the radial intermetacarpal spaces and the muscles of the thumb were involved (45 patients). In 8 cases, the only digit affected was the thumb, usually of traumatic origin. Interosseous muscle contracture, without thumb involvement, was present in 14 cases. In only one case was the muscular ischemia localized in the hypothenar area (abductor digiti quinti).[42] Of all the patients of the author's series, 81% were males. The youngest was 18 years and the oldest 73 years of age.

Physiopathology

Here, as in Volkmann's contracture of the forearm, the blood deficit of the intrinsic muscles can be produced by either of two mechanisms:

- direct injury to or disease of the principal vessels of the upper limb, that reduces circulation and produces tissue anoxia, with increase in endothelial permeability and progressive intramuscular anoxia by arterial and venous compression.
- persisting edema, occasionally associated with deep hematomas, causing progressive compression of the small vessels in the inextensible intermetacarpal spaces with consecutive muscular ischemia and shortening due to fibrosis. This theory of an ischemia–edema cycle with consequent muscular anoxia and necrosis is related to the presence of inextensible fascial spaces and the anatomic characteristics of the principal vessels of the hand that perfuse the interosseous and thenar muscles.

Anatomy of hand muscle compartments

According to the author's anatomic investigations[44,65] and clinical observations, there are six fascial compartments where the intrinsic muscles of the hand and their supplying

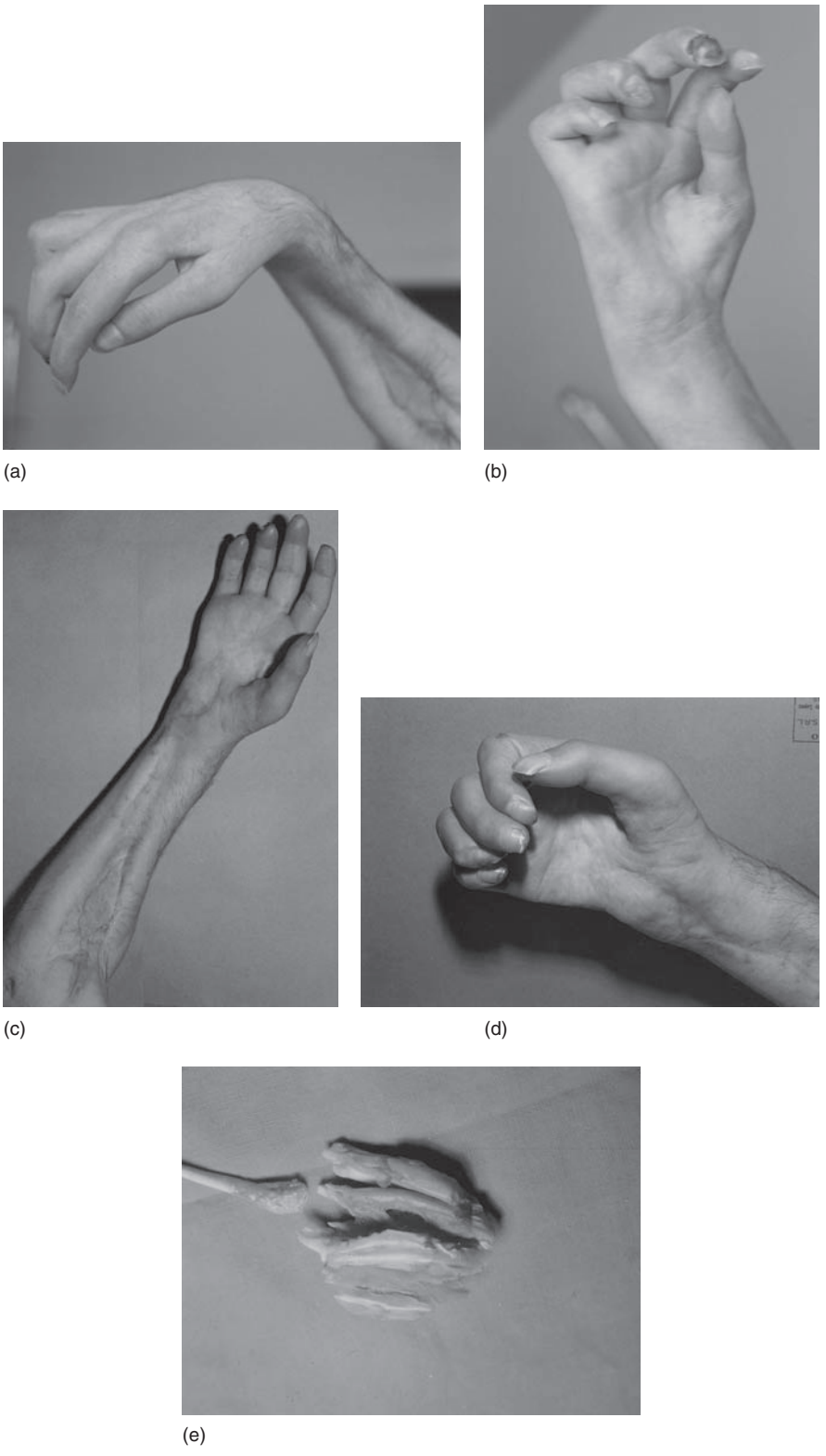


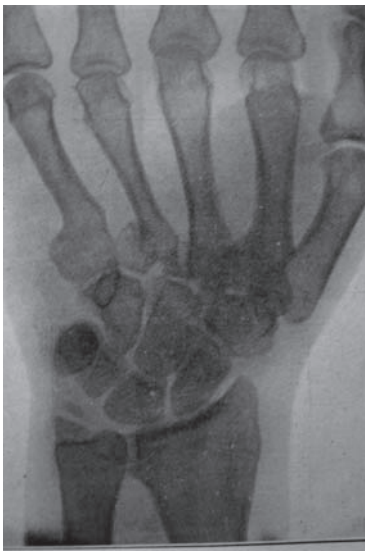
Figure 28.13

(a, b) Severe sequela of a compartment syndrome of the right forearm and hand (combined syndrome) in a 30-year-old male (type III) after an automobile accident. Contracture of the forearm and interosseous muscles of the index and middle fingers associated with contracture and paralysis of the intrinsic muscles of the thumb. Intrinsic paralysis – clawhand – of the two last fingers. Trophic changes in the middle finger. Anesthesia of all the digits. The patient was seen 6 years after a compound fracture of the elbow. Initially the patient was treated by fasciotomies of the forearm and free skin graft in the volar aspect of the forearm 4 weeks after the accident. (c, d) Hand motor function obtained after an excision-reconstruction procedure similar to the one shown in Figure 28.9, combined with interosseous proximal release of the radial fingers and thumb muscles (adductor pollicis and flexor pollicis brevis). The first web was reconstructed from a rotatory flap. (e) Excision of unchanged deep necrotic muscles of the forearm – infarct – found 6 years after the initial injury. The infarct excised was 5 cm in length.

vessels can be easily compressed by edema and/or tense hematomas: (1) adductor, (2) retroadductor, (3) first intermetacarpal, (4) second, third, and fourth intermetacarpals, (5) thenar, and (6) hypothenar (Figure 28.15a).

1. The adductor space contains the adductor pollicis muscle and is limited by the volar interosseous fascia dorsally and by the deep thenar fascia and oblique septum of the midpalmar fascia, volarly.
2. The retroadductor space is limited volarly by the adductor pollicis muscle and its fascia, and dorsally by the interosseous fascia that covers the second and first intermetacarpal spaces volarly. The radial part of the deep palmar arch runs through this space.

(a)



(b)

Figure 28.14

(a) Finochietto's illustration in his initial publication of 1920.[2] This pathology was described in the case of a 17-year-old male with an intrinsic-plus deformity of the radial fingers and an adduction contracture of the thumb, after a severe crush injury of the left hand in 1918. (b) A radiographic study showed a fracture-dislocation of all the carpometacarpal joints. Reconstructive surgery was indicated on 3 January 1919, consisting of a partial excision (1.5 cm) of the distal interosseous tendons of the first and second intermetacarpal spaces and 2 cm of the third metacarpal insertion of the adductor pollicis muscles. Partial excision of the metacarpophalangeal collateral ligaments of the first two radial fingers was also performed. The excised muscles were shown to be fibrotic. A plaster splint was indicated postoperatively, with dorsal traction (rubber bands) of the fingers and thumb. Nine months after surgery the strength was almost similar to the opposite hand. Active thumb abduction was 30°. Complete active finger flexion was obtained. The two radial fingers showed 10° of residual metacarpophalangeal flexion contracture.

3. The first intermetacarpal space is limited volarly by the volar interosseous fascia and dorsally by the fascia that connects the extensor tendons of the fingers with the extensors of the thumb (Anson's 'intertendineal fascia').[66] This space contains the first dorsal interosseous muscle.
4. The second, third, and fourth intermetacarpal spaces are limited volarly by the volar interosseous fascia and dorsally by the dorsal interosseous fascia. The vessels contained in these compartments are branches of the deep palmar arch of the hand (see below).

5 and 6. The thenar and hypothenar spaces are limited by their respective surrounding fascias.

The study of the deep palmar arch of the hand and its branches gives the surgeon a better understanding of the mechanism of intrinsic muscular ischemia. The author's conclusions[65] as to the anatomy of the deep palmar arch and its branches coincide with those of Fracassi[67] and Coleman and Anson.[68] The radial artery, which basically forms the deep palmar arch, penetrates the retroadductor space at the angle formed between the two first metacarpal bones and through a tendinous ring formed by the two heads of the first dorsal interosseous muscle. From this point, the artery runs across the bases of the second and third metacarpals volarly to the interosseous fascia and emerges from the retroadductor space through another tendinous ring formed by the transverse and oblique fascicles of the adductor pollicis muscle (Figure 28.15a). During this section (Delorme's oblique part)[65] the deep palmar arch yields two groups of branches: (1) proximal or recurrent volar branches of the carpus, and (2) distal or metacarpal branches (Figure 28.16a).

The proximal or recurrent branches might be connected to the anterior interosseous artery of the forearm. Anastomosis may compensate the circulation of the hand in cases with combined lesions of the radial and ulnar arteries. The distal metacarpal arteries are volar and dorsal.

The first volar metacarpal artery divides into two, the princeps pollicis and the radialis indicis. These arteries may emerge separately from the deep palmar arch. The volar metacarpal arteries of the three ulnar intermetacarpal spaces anastomose with the dorsal metacarpal arteries, and distally with the digital branches of the superficial palmar arch (Figure 28.16a). Irrigates the interosseous muscles and ends distally close to the metacarpophalangeal joints of the finger, with branches to the volar plate and metacarpal metaphysis and epyphysis. The second volar metacarpal artery irrigates particularly the interosseous muscles of the second intermetacarpal space and the adductor pollicis and flexor pollicis muscles. This group of muscles is the most frequently involved in Finochietto's compartment syndrome.[65]

The dorsal metacarpal arteries of the three ulnar intermetacarpal spaces – perforating arteries of the classic anatomists – penetrate the intermetacarpal compartments at their proximal ends after perforating the volar interosseous

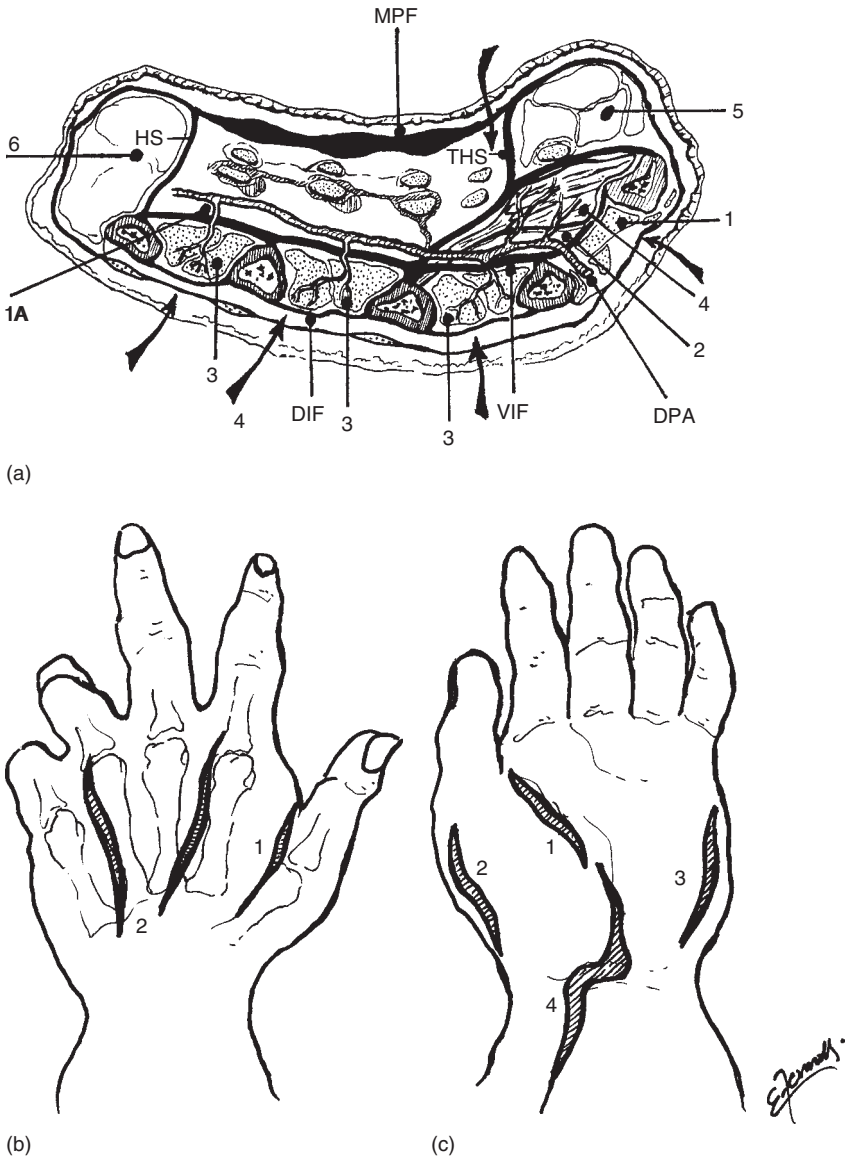


Figure 28.15

(a) Cross-section of the hand at the deep palmar arterial arch showing six fascial spaces – related to the location of the usual compartment syndromes – and the principal neurovascular structures. 1, first intermetacarpal; 2, retroadductor; 3, second, third and fourth intermetacarpals; 4, adductor; 5, thenar and 6, hypothenar compartments. MPF, midpalmar fascia; DPA, deep palmar arterial arch showing the emergence of its volar branches that divide into the volar and dorsal metacarpal arteries and irrigate the interosseous and deep thenar muscles; VIF, volar interosseous fascia, and DIF, dorsal interosseous fascia. (b) Dorsal aspect of the hand demonstrating the ideal skin incision for releasing the first intermetacarpal and retroadductor spaces (1) and the second, third, and fourth (2) intermetacarpal compartments. (c) Palmar aspect of the hand showing the incision for the adductor (1), thenar (2), hypothenar (3), and carpal tunnel (4) compartments.

fascia. Arriving at the dorsal aspect of the hand, these vessels perfuse the dorsal interosseous muscles and end at the metacarpal metaphysis (metaphyseal arteries). The second dorsal metacarpal artery may anastomose with the palmar arteries. In accordance with this description, the dorsal metacarpal arteries originate from the deep palmar arch, and not from the dorsal carpal arch, as described by many classic anatomists. Ulnarly, the deep palmar arch connects with the deep volar branch of the ulnar artery, which passes through the hiatus formed by the origins of the abductor and short flexor muscles of the little finger.

On the basis of this description, the deep palmar arch and its metacarpal arteries run in close fascial compartments – retroadductor and intermetacarpals – and end by irrigating the interosseous and deep thenar muscles. These two anatomic characteristics facilitate intrinsic muscle ischemia when edema or hematomas infiltrate the deep inextensible compartments of the hand.

The described anatomy indicates the importance of early fasciotomies to eliminate severe and persisting edema or deep hematomas, when the clinical picture of the hand presupposes anoxia and ischemia of its deep intrinsic muscles.

It is of interest to point out that the lumbrical muscles are not affected by the mechanism of compartment syndrome, since these muscles are not included in close fascial spaces. The lumbrical muscles may produce intrinsic-plus deformities of the fingers by mechanisms other than ischemia.

Finochietto's compartment syndrome of the hand may course through an acute and a sequelae stage.

Initial or acute stage

The initial or acute stage is characterized by arterial circulation impairment and muscular anoxia. The most common symptoms are:

- Pain that increases with muscle stretching – pain is characteristic because it is out of proportion to the

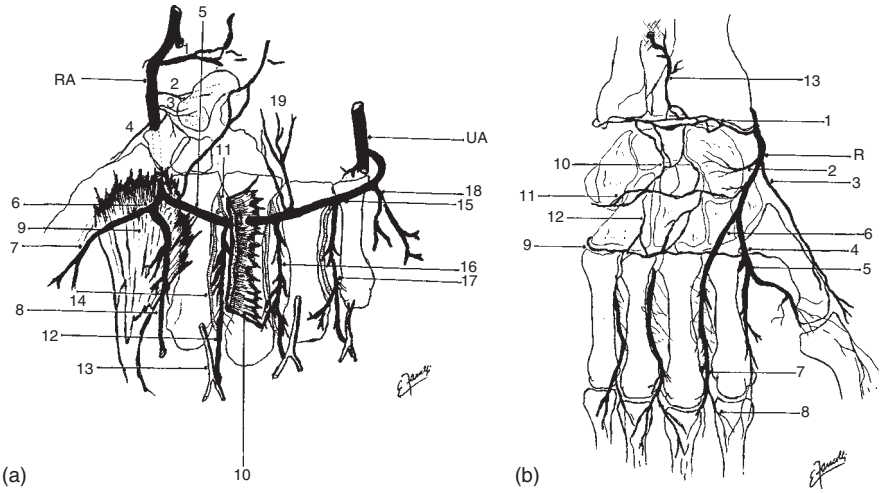


Figure 28.16

(a) Drawing from a cadaveric dissection of the deep palmar arch and its branches –most common vascular pattern (volar view). RA, radial artery and its carpal branches: anterior carpal (1) scaphoid arteries, to its dorsal aspect (2) and its distal tubercle (3). Dorsal pollicis artery (4). Deep palmar arch with its descending or metacarpal branches (5, 15): first volar metacarpal (6) divided into the princeps pollicis (7) and the volar radialis indicis (8). First volar interosseous muscle (9). Adductor pollicis muscle (third metacarpal insertions) (10). First perforating artery in the second intermetacarpal space (11) forming the second dorsal metacarpal artery (14) that anastomoses with the second volar metacarpal artery (12). Anastomosis of the second volar metacarpal artery with the second common volar digital artery (13). Volar and dorsal metacarpal arteries of the third (16) and fourth (17) intermetacarpal spaces. Arterial branch to the hypothenar muscles (18). UA, ulnar artery. (b) Dorsal view of the dorsal metacarpal arteries. Radial artery (R) with branches emerging at the snuff-box: dorsal carpal (1), dorsal scaphoid (2) and dorsal pollicis (3). Origin of the deep palmar arch (4) branching the first dorsal metacarpal artery (5). Second dorsal metacarpal artery (6). Anastomosis between the second dorsal and second volar metacarpal arteries (7). Branches to the dorsal aspect of the fingers (8). Basal metacarpal arch (9). Descending branches of the anterior interosseous artery (13) and dorsal carpal arch (10). Dorsal intercarpal arch (11). Anastomosis (12) between the dorsal arches (9, 11).

initial injury. The intermetacarpal spaces are examined for pain by passively extending the metacarpophalangeal joint and flexing the interphalangeal joints (Finochietto's maneuver). The adductor compartment of the thumb is tested by passive adduction of the thumb. Likewise, the lateral thenar muscles are stretched by abduction of the thumb. The hypothenar muscles are tested by radial deviation of the small finger.

- Swelling with tense and hard fascial spaces – this is the earliest sign of compartment syndrome of the hand. Each compartment should be examined individually. The fingers tend to rest in the intrinsic-plus position.
- Paresthesia – this depends on the compression of the median and/or ulnar nerves. Severe paresthesiae and pain of the radial digits may relate to an acute carpal tunnel syndrome, produced when the canal pressure rises to a level that reduces the capillary blood flow of the median nerve and its viability. This condition demands an early release of the carpal tunnel.
- Capillary refill slowness and occasional pulse deficit.

The fundamental concept in an acute compartment syndrome in the hand is to achieve early reduction of high tissue pressure within the involved fascial spaces. This is accomplished by reducing hand fractures or dislocations and by enlarging fascial compartments by fasciotomies. The release of fascias is directed to the compartments where clinical examination indicates a tissue compromise (Figure 28.15b and c). Measurement of tissue pressure within the fascial spaces at risk may help the clinical decision on whether to perform fasciotomies.

Final or sequelae stage intrinsic-plus deformity classification and surgical treatment

Sequelae of Finochietto's ischemic compartment syndrome may show different clinical manifestations of the fingers and the thumb (intrinsic-plus deformity), according to the severity of muscle contracture and its localization.

The author[38,41,42] divides the intrinsic-plus deformity of the fingers into three types, according to the severity of the interosseous muscle contractures and to their clinical manifestations (Table 28.2).

Intrinsic-plus deformity of the fingers

Type I This type of intrinsic-plus deformity of the fingers relates to a mild interosseous muscles contracture. It may be overlooked since the patient can open and close the

Table 28.2 *Types of intrinsic-plus deformities of the fingers and thumb*

Muscle ischemia localization	*Type of deformities*	*Clinical features*
A Fingers (contracture of the interosseous muscles)	I	Complete active finger extension and flexion, but inability to place the fingers in the 'hook' position
		Delayed flexion of the proximal phalanx
		Positive intrinsic-plus test
	II	Type I plus metacarpophalangeal flexion contracture
	III	Type II plus metacarpophalangeal and proximal interphalangeal joints stiffness
B Thumb (thenar muscles)	I	Adduction contracture (contracture of the medial thenar muscles)
	II	Adduction and palmar abduction contracture (contracture of the medial and lateral thenar muscles)

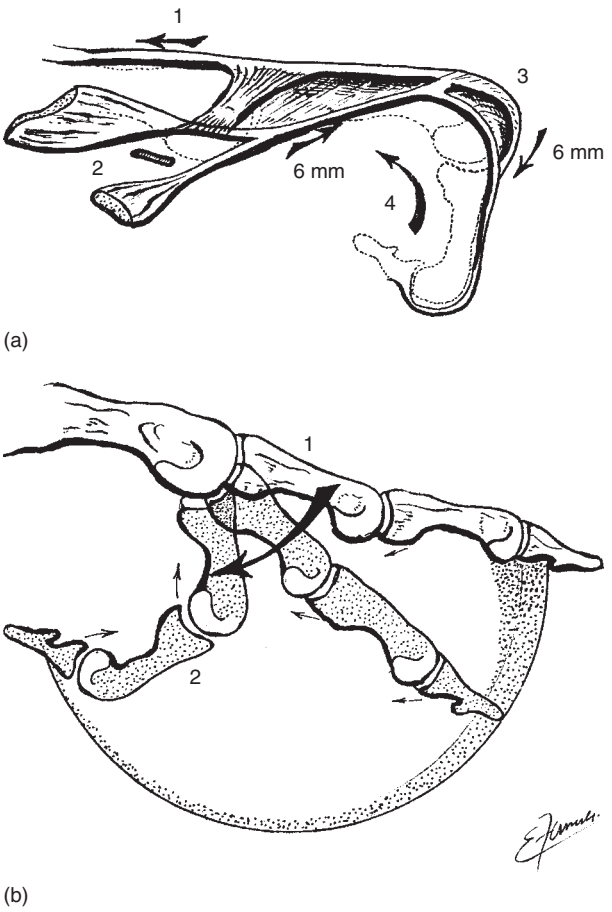


Figure 28.17

(a) Normally during the position of 'hook' flexion of a finger the extensor tendon contracts (1) and the intrinsic muscles – interossei and lumbrical – relax (2) to allow distal gliding of their intrinsic tendons and the extensor apparatus over the proximal interphalangeal joint (3). This balance allows active interphalangeal flexion by the action of the long flexor tendons (4). In ischemic contracture of the interosseous muscles, 'hook' finger cannot be produced since distal gliding of the intrinsic tendons is blocked. (b) In Finochietto's intrinsic contracture, finger flexion begins by flexion of the proximal phalanx (1) followed by flexion of the interphalangeal joints (2).

digits completely. Muscle contracture can be demonstrated by three clinical signs:

1. *Inability to place the affected fingers in a 'hook' position.* Normally, to actively place the fingers in the 'hook' position (metacarpophalangeal extension and interphalangeal flexion) it is necessary that the intrinsic tendons – interosseous and lumbricals – shift distally approximately 6 mm. This displacement allows distal shifting of the entire extensor apparatus, which in turn permits interphalangeal flexion without simultaneous flexion of the metacarpophalangeal joint (Figure 28.17a). When the interosseous muscles are contracted, their tendons cannot be distally displaced, and thus it is impossible to flex the interphalangeal joints while the metacarpophalangeal joint remains in complete extension. Owing to this pathophysiology, complete interphalangeal joint flexion is only possible with some metacarpophalangeal joint flexion. The greater the interosseous contracture, the greater the flexion position of the metacarpophalangeal joint to allow interphalangeal flexion.
2. *Delayed flexion of the proximal phalanx during active complete finger flexion (intrinsic-plus type of finger flexion).* The delay of active interphalangeal flexion during complete finger flexion produces a rolling rather than the normal simultaneous type of finger flexion (Figure 28.17b). This rolling flexion differs from that of the clawhand due to intrinsic paralysis of the hand. In this case flexion is initiated at the interphalangeal joints, while in intrinsic contracture finger flexion is initiated at the metacarpophalangeal joint.
3. *Positive Finochietto's intrinsic-plus test.* Finochietto's intrinsic-plus test is positive when passive flexion of the interphalangeal joints is blocked, while the metacarpophalangeal joint is kept in passive extension. Interphalangeal flexion is only possible if the metacarpophalangeal joint is permitted to flex (Figure 28.18). The intrinsic-plus test can be masked when intrinsic contracture is combined with adhesion of the long extensor tendons or with interphalangeal stiffness.

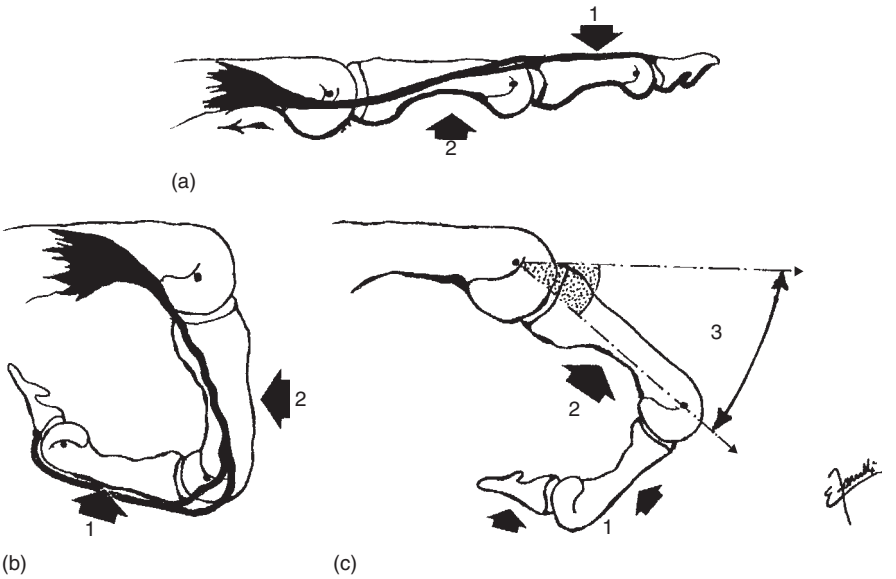


Figure 28.18

Two parts of Finochietto's intrinsic plus test. (a) The first part consists of the impossibility of producing passive interphalangeal flexion (1) if the metacarpophalangeal joints are held in passive extension (2). (b) The second part consists of the possibility of obtaining passive interphalangeal flexion (1) if the metacarpophalangeal joints are permitted to flex (see Figure 28.20). (c) In an interosseous ischemic contracture syndrome, passive initial flexion of the interphalangeal joints (1) automatically flexes the proximal phalanx (2). The magnitude of this angle of flexion depends on the severity of the contracture of the interosseous muscles (3).

Under the latter conditions the position of the metacarpophalangeal joint will not influence passive interphalangeal motion. It is important that investigation of the intrinsic-plus test be routine in all suspicious cases because only through this test can mild interosseous muscle contractures be demonstrated.

Type I fingers' intrinsic-plus deformity can be corrected by excision of the intrinsic lateral band and the oblique fibers of the dorsal aponeurosis on both sides of each affected finger (Littler technique)[28] (Figures 28.14 and 28.19). The procedure represents a distal intrinsic muscle release, and has advantages over the isolated excision of the lateral bands of Bunnell (quoted by Boyes[35]) because it eliminates the more medial fibers of division of the interosseous tendons, which reach the long extensor tendons in their course over the proximal phalanx. This procedure is performed through a unique longitudinal excision over the proximal dorsal half of the proximal phalanx of each finger (Figure 28.19). Surgical damage to the transverse hood fibers of the extensor apparatus must be avoided in order to prevent dislocation of the long digital extensor tendon over the metacarpal head.

Once the intrinsic release has been performed, the interphalangeal joints are flexed passively – very gently – while the proximal phalanx is held extended. Resistance to passive flexion of the middle joint usually means that the collateral ligaments are retracted. If, under these circumstances, the surgeon were to force interphalangeal passive flexion, an irreversible stretching of the extensor apparatus would be produced, leaving a definitely flexed, painful, semirigid, and swollen middle joint. Other causes of resistance to passive flexion of the middle joint may depend on adherences or retraction of the extensor apparatus over the middle joint. This technique cannot correct flexion contracture of the metacarpophalangeal joint and therefore is not indicated in types II or III intrinsic-plus deformities.

During the postoperative period, the hand is immobilized by means of a sufficiently padded circular plaster cast that keeps the wrist and metacarpophalangeal joints in neutral position. The interphalangeal joints are left free to allow early active and passive flexion–extension movements during the immediate postoperative period. The initial postoperative plaster cast is removed after 10 days, and re-education is started with complete digital movements. The patient uses the opposite hand to repeatedly produce the intrinsic-plus maneuver in order to distend the intrinsic tendons. The dynamic splint conceived by Bunnell[35] – the metacarpophalangeal extension splint or 'severe knuckle-bender splint' – or a special plaster splints[41] is very useful during the re-education period. Re-education of the hand movements must continue for several weeks, or even months, owing to the great and particular tendency of the deformity to recur.

Postoperative re-education is considered to be complete when the patient affirms that, on waking up in the morning, the hand is the same as it was when going to bed at night, and that the fingers rapidly regain complete movements. The ability to actively place the operated fingers into a complete 'hook' position is evidence of an excellent correction.

Type II In type II intrinsic-plus deformity the interosseous contracture is greater than in type I, and, consequently, the metacarpophalangeal joints of the fingers present with some flexion, and contracture proportional to the fibrotic contracture of the interosseous muscles. The metacarpophalangeal collateral ligaments are not contracted in type II deformity. The interphalangeal joints remain in a position of extension. Occasionally they may be deformed in recurvatum.

The principal functional deficits in this type of deformity are (1) an inability to completely open the base of the fingers, and (2) active flexion of the interphalangeal joint is more delayed than in type I, when complete

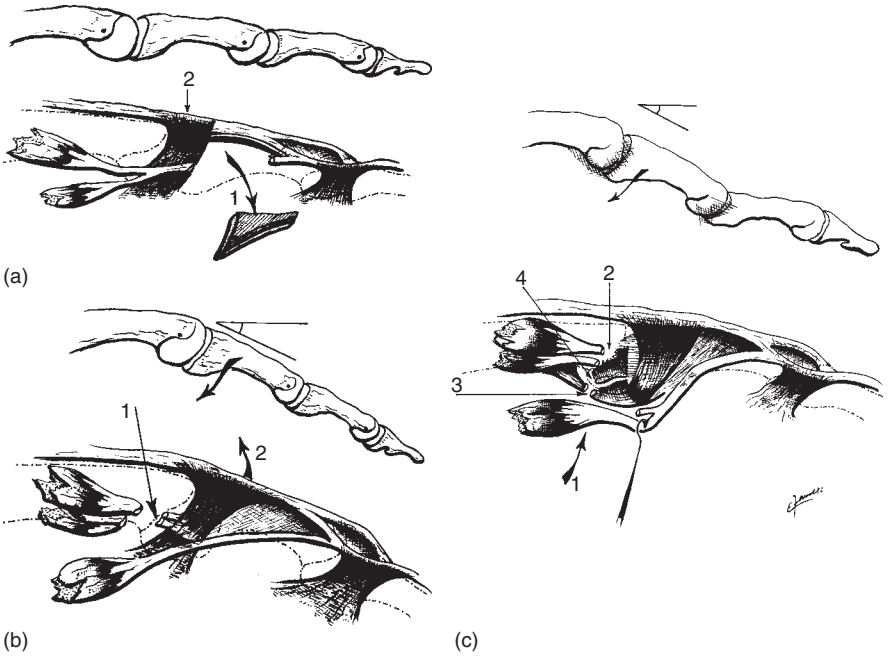


Figure 28.19

Surgical procedures to correct intrinsic-plus deformity of the fingers due to ischemic contracture of the interosseous muscles. (a) Type II: bilateral excision of the lateral bands and oblique fibers of the extensor apparatus (1). Transverse fibers of the extensor hood are preserved (2) (distal interosseous release on both sides). (b) Type II: bilateral excision of the distal tendons of the dorsal and volar interosseous muscles (1) – dorsally to the deep transverse metacarpal ligament. Longitudinal dorsal intermetacarpal approaches are employed. This technique allows complete passive metacarpophalangeal extension (2) (proximal interosseous release). (c) Type III: Through a transverse palmar approach, following the distal palmar crease (1), metacarpophalangeal joint and interosseous muscle contractures are corrected by excision of the distal interosseous tendons (2); transverse release of the volar plate (3) and excision of the glenoid part of the collateral are performed (extended proximal release) (both sides). Excision of the phalangeal bundles of both collateral ligaments in stiff proximal interphalangeal joints may be necessary.

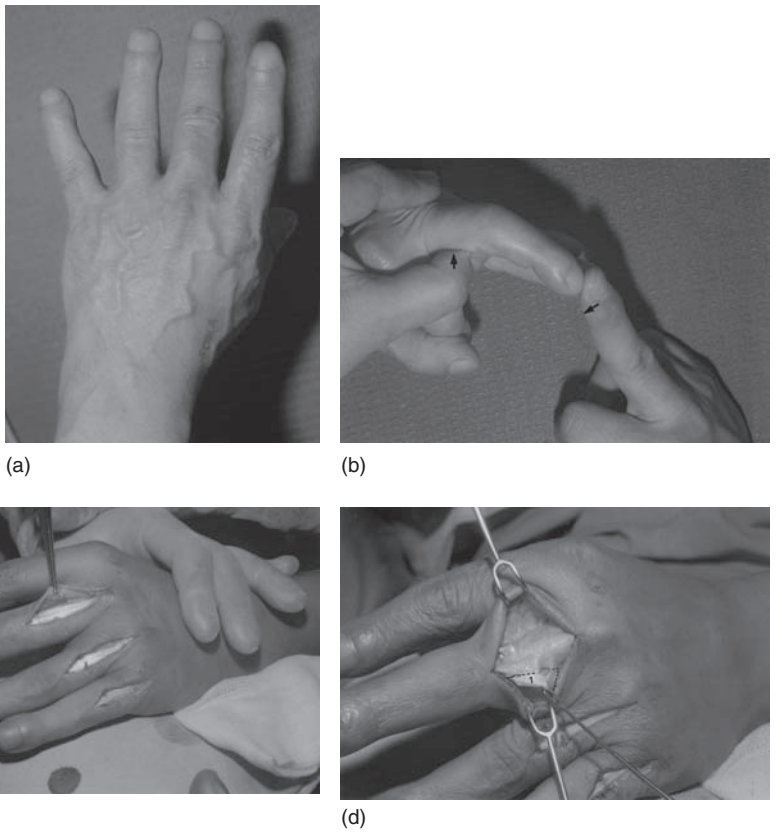
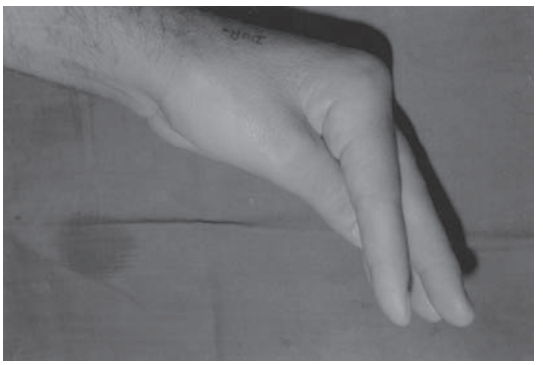


Figure 28.20

(a) Interosseous ischemic contracture of all the fingers with intrinsic-plus deformity type I. Sequela of a compartment syndrome after the fracture of both bones of the forearm – distal third – and persisting hand edema. (b) Positive Finochietto's intrinsic plus test. (c) Longitudinal incision over the proximal phalanges – proximal part. (d) Bilateral excision of the lateral bands and oblique fibers – dotted lines – of the extensor apparatus (1).

active finger flexion is attempted. The intrinsic-plus test is positive.

Intrinsic plus deformity in type II is corrected by release of the interosseous muscles proximal to the metacarpophalangeal joint (proximal intrinsic muscle release).[42] In this technique, the interosseous tendons are excised at their musculotendinous junction, dorsally to the deep transverse intermetacarpal ligament.[41,42] Finochietto[2] employed a similar procedure for the case he had the opportunity to treat (Figure 28.1). The author prefers this technique to the



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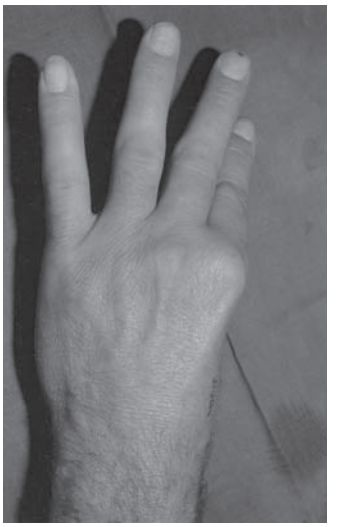

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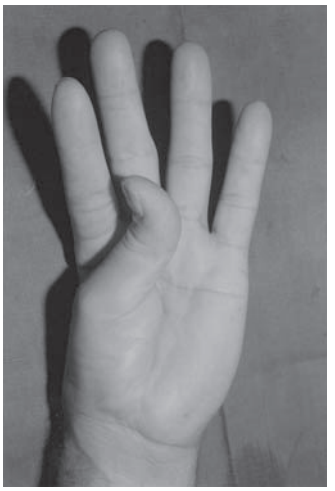

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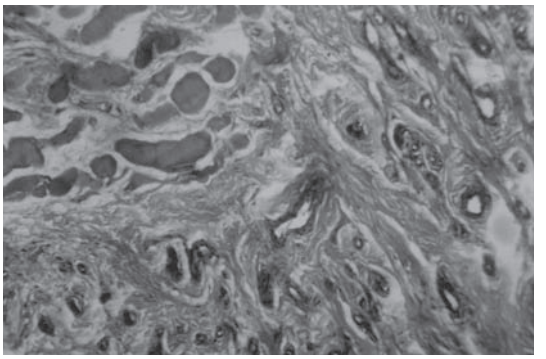

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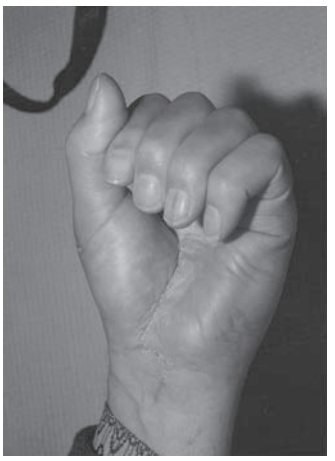

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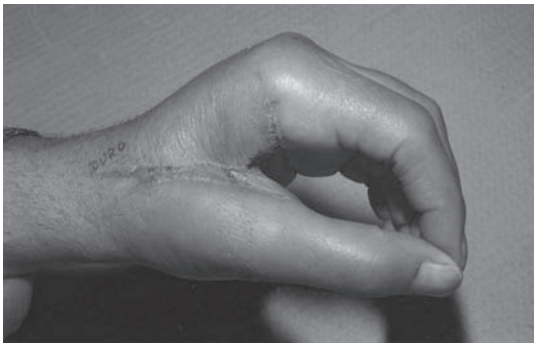

(f)

Figure 28.21

(a–c) Severe ischemic intrinsic deformity of all the digits in a 38-year-old male following thrombosis of the axillary artery. Intrinsic-plus deformity of all the fingers – index and middle type II, ring and little finger type I – and thumb with mild contracture of the web. The surgical program was: 1, excision of the distal interosseous tendons of the two radial fingers through a transverse volar approach (proximal intrinsic release); 2, distal intrinsic release of the last two fingers – Littler's technique; 3, intrinsic release of all the thenar muscles; and 4, Z plasty of the thumb web. (d) Microphotography showing fibrosis of the interosseous muscles. (e, f) Satisfactory hand closure and thumb function. Finger extension was complete and thumb intrinsic-plus deformity was corrected.

interosseous slide procedure described by Bunnell.[58] Proximal interosseous muscle release is indicated not only to restore the normal rhythm of digital flexion, but also to allow complete extension of the fingers.

The procedure is performed through longitudinal incisions on the dorsum of the hand, at the distal ends of the intermetacarpal spaces. Excision of both interosseous tendons must be complete, with the precaution taken to eliminate all fibers of the volar interosseous muscles, which may remain hidden at the depth of the wound (Figure 28.14b).

If, after excision of the interosseous tendons, the metacarpophalangeal joint does not passively extend completely, the volar plate and collateral ligaments are retracted, indicating a type III deformity. Occasionally proximal and distal releases are indicated simultaneously.[41]

Type III This type of intrinsic-plus deformity of the fingers is complicated by joint stiffness, principally at the metacarpophalangeal joint, where the volar plate and the glenoid bundles of the collateral ligaments are retracted. Stiffness of the proximal interphalangeal joint may be associated. Occasionally, the affected fingers present in a 'swan-neck' deformity. In this case, hand function is severely affected. The thumb is usually included in the pathology (Figure 28.20).

Type III intrinsic-plus deformity represents a very difficult clinical condition to correct. The possibility of restoring acceptable digital function varies according to the severity of the case. The most favorable situations are those in which the metacarpophalangeal joints are stiff but the interphalangeal joints are flexible.

In type III deformity, the author advocates a proximal interosseous muscle release associated with a metacarpophalangeal joint release.[42] The procedure is performed at the same surgical stage, through a unique transverse approach at the level of the distal palmar crease (Figure 28.14c). After dissection of the cutaneous flaps, and retraction of the lumbrical tendons, and vessels and nerves of the palm, each intermetacarpal compartment is approached. The deep aponeurosis of the palm is opened, proximally to the deep transverse intermetacarpal ligament, and the distal tendons of the interosseous are exposed and excised. Next, the sheath of the long digital flexor tendons is opened longitudinally and the flexor tendons retracted, permitting a transverse division of the metacarpophalangeal volar plate at its middle part, and excision of the glenoid fascicle of the collateral ligaments. This technique allows passive extension of the metacarpophalangeal joint. Should the middle joint be stiff in extension, it must be either mobilized by excision of its collateral ligaments or, in irreversible cases, fused in the position of function.

In cases with marked circulatory deficit of the hand, this surgical procedure must be performed with great precaution to preserve tissue perfusion.

Intrinsic-plus deformity of the thumb

The thumb may present two types of deformities based upon which intrinsic muscles are affected: medial, lateral, or medial-lateral (Tables 28.1 and 28.2). The most frequent deformity depends on the ischemic contracture of the deep medial thenar (flexor pollicis brevis, adductor pollicis) and first dorsal interosseous muscles, producing an adduction contracture of the thumb. The first web and the first intermetacarpal ligament may be contracted. Passive flexion of the interphalangeal joint may be limited when the metacarpophalangeal joint is kept in extension (positive intrinsic-plus test).

When the radial and lateral thenar muscles are affected by ischemia, the thumb contracts in flexion and palmar abduction. The metacarpophalangeal joint is contracted in flexion and the interphalangeal joint in hyperextension (intrinsic-plus deformity of the thumb). In this position, the thumb may block flexion of the index and middle fingers (Figure 28.21).

Thumb deformity is corrected by muscle release at the same surgical stage with the correction of the intrinsic-plus deformity of the fingers. Contracture of the first web may demand a Z plasty, a local cutaneous flap, or an island forearm pedicle flap.[69–71]

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29 The tetraplegic upper limb

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Introduction

Prior to the 1950s, relatively few patients suffering transection of the cervical spinal cord survived their injury. Improved medical care and better coordination of the multiple levels of care have resulted in greater numbers of patients surviving their spinal cord injury and leaving hospital.

In the 1940s, occasional reports appeared in the literature, that described the application in tetraplegics of surgical procedures developed for the poliomyelitic upper limb. Bunnell,[1] in his textbook on hand surgery first published in 1944, stated that the reconstruction of thumb opposition was an important goal in tetraplegic patients and described a reconstructive procedure associating a tendon graft for thumb opposition and tenodeses of the flexor and extensor tendons to the radius. Subsequently, a few authors reported on limited series of tetraplegic patients operated with various techniques.[2–4] At the time, upper limb surgery was not felt to be particularly beneficial by the physiatrists and others in whose care the tetraplegic patients remained. This lack of enthusiasm was more than likely related to a number of partial results, or worse, failures of the early procedures, which in some cases resulted in an aggravation of the patient's upper limb function.

An attempt at creating a fully automatic grasp and release hand was developed by Nickel and co-workers at Rancho Los Amigos (USA), by multiple fusions of the thumb and finger joints. This procedure, known as the 'flexor hinge hand', only gained local popularity because of the frequent inability of the tips of the digits to meet, and because a number of patients were dissatisfied with the resulting stiff and inflexible hand. This further increased pessimism regarding the usefulness of surgery for the tetraplegic patient, and greater emphasis was subsequently placed on the development of adaptive aids and external orthoses. In 1963, Nickel and co-workers[5] reviewed their philosophy and design for a wrist-driven 'flexor hinged splint', a metallic device which constrained all joints of the hand, save the metacarpophalangeal joints of the fingers, in a position that allowed wrist motion to generate flexion and extension of the fingers, and tip to tip contact between the thumb, and the index and middle fingers. Only a few patients accepted to wear these devices permanently. Complaints included discomfort, and the need for others to put the device on.

During the 1960s and the 1970s, a few surgeons continued to perform and recommend surgical reconstruction of upper limb function in the tetraplegic patients, mainly in those patients in whom wrist extension had been spared. In 1967, Freehafer and Mast[6] reported on the use of the brachioradialis (BR) to improve wrist extension in six patients. In 1971, Lamb and Landry[7] reported on tendon transfers performed on 25 tetraplegic patients, and were the first to advocate preferential restoration of a side (or 'key') pinch rather than opposition pinch. The French experience of Maury and co-workers was published in 1968[8] then 1973,[9] but they acknowledged 25% failures, 25% partial improvements, and only 50% significant improvements.

In 1975, Eduardo Zancolli published his experience on 76 tetraplegic patients operated between 1947 and 1974, setting up a number of the rules which are now felt as essential to this type of surgery.[10] He pointed out that surgery should be delayed until the patient can sit in bed, and thus pronate his forearms. If he cannot achieve pronation in the sitting position, then this should be restored by rerouting the biceps. He also stated that two key muscles must be left undisturbed: the extensor carpi radialis brevis (ECRB) and the flexor carpi radialis (FCR), since wrist motion is the fundamental motion to produce finger function. He recommended a two-stage approach for each hand, the first stage for extension and opening of the hand, and the second for closure.

The individual who is probably regarded by all as the champion of surgical restoration of function in the upper limbs of tetraplegic patients is Eric Moberg from Sweden. When faced with retirement from his university position at the age of 65, Moberg began concentrating on the tetraplegic patients, and especially those with high-level injuries, who have very few functional resources left, and for whom surgery at the time had little if anything to offer in terms of function. He developed a very conservative surgical approach, based on a philosophy of doing less rather than more, in this particular group of patients where it is generally felt that any loss of function due to a complication of surgery is intolerable. He concentrated on restoring a simple function, a key-type of pinch between the pulp of the thumb and the lateral aspect of the index finger, and demonstrated how to restore another useful function in this population, by transferring the posterior half of the deltoid muscle into the triceps.

Moberg's philosophy remains the foundation of surgical decision-making today, almost 30 years after he published his views in 1975,[11] then expanded them in his monograph published in 1978.[12] For the rest of his life he travelled the world over, promulgating his strong opinion that the majority of tetraplegics can benefit from carefully chosen and performed procedures. He must be given credit for stimulating the interest of surgeons from many countries, including the two authors of the present chapter, and thus creating renewed surgical interest in this area.

Since the publication of Moberg's monograph, more than one hundred articles devoted to surgical restoration of the upper limb have been published. Under Moberg and others' stimulus, a first International Conference on surgical rehabilitation was held in Edinburgh in 1978.[13] During the Second International Conference, held in Giens (France) in 1984, the international classification of tetraplegic upper limbs was established,[14] and seven more conferences have been held, one every three years, with constantly rising attendance and interest.

In most developed countries nowadays, surgical improvement of the upper limbs is regarded as one of the options which should be offered to tetraplegic individuals in order to improve their function. Indications of these procedures are well codified, techniques are well mastered, and predictable results can be expected.[15]

Classification

Several systems to classify spinal cord injured patients have been used by various professionals involved in the care of these patients. Some were based on the location of the bony injury as determined radiographically. Others were based on the evaluation of residual motor and sensory function, the results of which could then be related to specific areas of the spinal cord itself. Still others were based on commonly recurring patterns of injury, e.g., the older Frankel and newer American Spinal Injury Association (ASIA) scale.[16]

Most systems of classification prior to the ASIA scale failed to differentiate between the two arms, although half the patients have significant interarm differences and, frequently, the motor level does not coincide with the sensory level, especially in higher-level injuries. These deficiencies, suggesting the need for a system that separately classifies the type and level of injury for each patient and each limb, were recognized by the ASIA, which, in 1982, published standards (Figure 29.1) for neurologic classification of spinal cord injuries, which have since been periodically revised.

The ASIA impairment scale assigns patients with spinal cord injuries into one of five categories:

- A: *Complete* No motor or sensory function is preserved in the sacral segments S4–S5.
- B: *Incomplete* Sensory but not motor function is preserved below the neurologic level and extends through the sacral segments S4–S5.
- C: *Incomplete* Motor function is preserved below the neurologic level, and the majority of key muscles below the neurologic level have a muscle grade less than 3.
- D: *Incomplete* Motor function is preserved below the neurologic level, and the majority of key muscles below

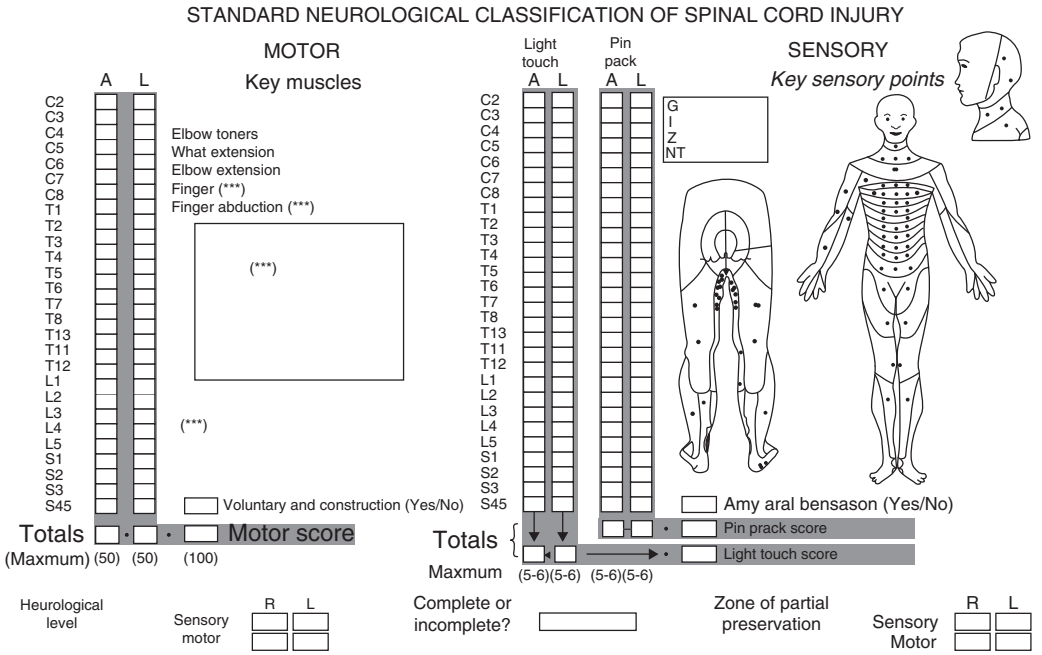


Figure 29.1

The ASIA scale.

the neurologic level have a muscle grade greater than or equal to 3.
- E: *Normal* Motor and sensory function is normal.

The *neurological level* refers to the most caudal segment of the spinal cord, with normal sensory and motor function on both sides of the body. The *sensory level* refers to the most caudal segment of the spinal cord with normal sensory function. There is one key point for each of the 28 dermatomes on the right and on the left side of the body. For the upper limb, these key points include:

- C4: the top of the acromioclavicular joint
- C5: the lateral side of the antecubital fossa
- C6: the thumb
- C7: the middle finger
- C8: the little finger
- T1: the medial side of the antecubital fossa.

The *motor level* refers to the most caudal segment of the spinal cord with (preserved) useful motor function. A key muscle (on the right and on the left side of the body) is tested in each of the 10 paired myotomes of the upper and lower limbs. For the upper limb, the key muscles are:

- C5: the elbow flexors (biceps and BR)
- C6: the wrist extensors (extensor carpi radialis longus (ECRL) and ECRB)
- C7: the elbow extensors (triceps)
- C8: the finger flexors (flexor digitorum profundus, middle finger)
- T1: the abductor digiti minimi.

The strength of the muscle is graded on a 6-point scale (0–5), according to MRC recommendations. The ASIA classification is thus comprised of one motor and one sensory level for each side of the body (R-motor, R-sensory; L-motor, L-sensory) as well as two sensory scores (one for pin prick and one for light touch) and one motor score.

Even when accurately applied, the ASIA classification provides only minimal information about each muscle of the upper limbs in tetraplegia. As rehabilitative surgery of the tetraplegic's upper limb is based primarily on tendon transfer of muscles graded 4 and above, many of the surgical pioneers such as Lamb, Zancolli, Freehafer, and Moberg introduced personal systems based on residual muscle power.

The international classification

At a formal gathering of experts in Edinburgh in June 1978, at what was to become the first of a series of International Conferences on Surgical Rehabilitation of the Upper Limb in Tetraplegia, the foundation for a surgical classification was established, based on the classification developed by Moberg in 1975,[11] and the International Classification (IC) was finalized during the Second International Conference, held in Giens (France) in 1984[14] (Figure 29.2). This classification was later adopted by the International Federation for Societies for Surgery of the Hand (IFSSH) and revised at subsequent International Conferences on Tetraplegia.

Similar to, but also differing substantially from the ASIA system, there is a sensory and a motor component to the IC. The sensory component differentiates the presence/absence of sufficient proprioception, defined by two-point discrimination (2 Pt) less than 12 mm. If absent, the eyes substitute for the hand as regards sensory afferent

International Classification for Surgery of the Hand in Tetraplegia (Giens 1984)

Group	Motor characteristics	Description of function
0	No muscle below elbow suitable for transfer	Flexion and supination of the elbow
1	BR	
2	ECRL	Extension of the wrist (weak or strong)
3	ECRB	Extension of the wrist
4	PT	Extension and pronation of the wrist
5	FCR	Flexion of the wrist
6	Finger extensors	Extrinsic extension of the fingers (partial or incomplete)
7	Thumb extensor	Extrinsic extension of the thumb
8	Partial digital flexors	Extrinsic flexion of the fingers (weak)
9	Lacks only intrinsics	
X	Exceptions	

BR, brachioradialis; ECRL, extensor carpi radialis longus; ECRB, extensor carpi radialis brevis; PT, pronator teres; FCR, flexor carpi radialis.

Figure 29.2
The IC scale.

information, and this is termed 'ocular' and designated 'O'. If, in addition to ocular afferent sensation, proprioception is present, this is termed 'oculo-cutaneous' and is designated 'O, Cu'.

The motor classification is based on residual motor function and enumerates only muscles originating around or below the level of the elbow that are graded MRC 4 or 5 strength. Each limb is individually classified. Group O includes cases where there is no muscle below the elbow suitable for transfer. A new muscle is then added for each successive muscle. Typically, for IC group 1, this is the BR muscle; for IC group 2, the extensor carpi radialis longus (ECRL) is also at the MRC 4–5 level, and so on. Group 9 signifies those lacking intrinsics only, and group 10 includes all exceptions. The system recognizes that it is frequently difficult to determine whether both radial wrist extensors are grade 4 or 5. Later additions to this classification include indicating the presence/absence of antigravity elbow extension (triceps) and a system to classify spasticity is being proposed for inclusion. The IC has been widely adopted by upper limb surgeons as the methodology is simple, reasonably intuitive, and easily learned.

Examination and selection of patients

The therapeutic plan for each tetraplegic individual is developed together with several professionals: physician, occupational therapist, physical therapist, nurses, psychologist, and social worker. The decision to undertake upper limb surgery to restore lost function is made by this team on the basis of several factors. Several meetings are necessary for a complete assessment, and adequate time must be provided for the examination, which should be performed at a time when the patient is not fatigued from prior therapy or testing.

General evaluation of the patient

Assessment of the psychologic status of the patient, his/her motivations, and intelligence must be performed. Lack of psychologic stability, poor motivation, unrealistic expectations, and limited intelligence are potential contraindications, that must be carefully identified and assessed. Once they have been identified, the team is better able to plan further strategies.

History of medical problems affecting the upper limb

Questions should be asked about any previous upper limb injury or pathology. Several pre-existing conditions, such as lesion of the shoulder rotator cuff, may predispose the patient to pain once more vigorous physical therapy begins. Other injuries may have been contemporary to the spinal cord lesion, such as gleno-humeral dislocation, brachial plexus injury, or wrist trauma.

Current functional status

The patient's functional status must be analyzed in the light of the neurologic level of injury and time since injury. It must be conducted with the patient sitting in his/her wheelchair, and with all orthoses and adaptative devices currently used. By examining how the patient uses his/her hands in performing tasks, and what objects he/she has found helpful, the rehabilitation team can gain perspective regarding how the individual patient has learnt to adapt to the environment. The patient may bring up points of view regarding his function that may differ greatly from those of the therapist or surgeon, and this must be taken into consideration.

The global functional status is assessed with a standard scheme. Several of these are available, such as the Functional Independence Measurement (FIM)[17] (Table 29.1).

Physical examination of the upper limb

Passive and active motion of each joint of both upper limbs are recorded, along with pain with motion. The wrist is specifically assessed for the tenodesis tone (automatic flexion of the fingers and thumb-to-index pinch with wrist extension).

Joint stability is assessed by stress testing. Key joints include the carpo-metacarpal (CMC) and metacarpophalangeal (MP) joints of the thumb, as well as the MP joints of the fingers. Extreme laxity of any of these joints will need to be addressed at the time of surgery.

Motor assessment is critical for surgical planning. All muscles of the upper limb should be tested and recorded with the MRC grading scale, with special attention to the three parts of the deltoid, the triceps, BR, and the radial wrist extensors (ECRL and ECRB).

Sensory examination: the basic sensory modalities such as light touch and temperature are tested for each spinal segment. Proprioception is also included. Moberg has championed the use of two-point discrimination as a measure of retained proprioception and finer discrimination. This is a simple and inexpensive evaluation, easily performed with a paper clip. The normal digit can determine two points at an interval of 2–3 mm. Moberg believed that proprioception is retained in a digit still capable of discrimination at 12–15 mm.

Pathologic features likely to compromise functional recovery are sought. Hypersensitivity of the digits or hand may make it impossible for the patient to touch or hold objects. Contractures may interfere with opening or closing of the fingers. Spasticity may be useful when moderate, but compromises function when it is severe (which is more frequent in incomplete forms of tetraplegia).

Table 29.1 *The Functional Independence Measurement (FIM)*

LEVELS		
	7 Complete Independence (Timely, Safely)	No Helper
	6 Modified Independence (Device)	
	Modified Dependence	Helper
	5 Supervision	
	4 Minimal Assist (Subject = 75%+)	
	3 Moderate Assist (Subject = 50%+)	
	Complete Dependence	
	2 Maximal Assist (Subject = 25%+)	
	1 Total Assist (Subject = 0%+)	

	ADMIT	DISCH
Self Care		
A. Eating		
B. Grooming		
C. Bathing		
D. Dressing – Upper Body		
E. Dressing – Lower Body		
F. Toileting		
Sphincter Control		
G. Bladder Management		
H. Bowel Management		
Mobility		
Transfer:		
I. Bed, Chair, Wheelchair		
J. Toilet		
K. Tub, Shower		
Locomotion	W C	W C
L. Walk/WheelChair		
M. Stairs		
Communication	A V V N	A V V N
N. Comprehension		
O. Expression		
Social Cognition		
P. Social Interaction		
Q. Problem Solving		
R. Memory		
Total FIM		

NOTE: Leave no blanks; enter 1 if patient not testable due to risk.

Selection of patients

After completion of the basic examination, the evaluating team should have gathered sufficient information upon which to decide which patients are candidates for rehabilitative surgery of the upper limbs. Almost every tetraplegic patient who retains control over one or more forearm muscles (C5 or more distal) is a potential candidate for functional surgery of the upper limbs. However, not every patient with this prerequisite will need or choose to undergo surgery.

Some patients are not ready to devote the time necessary for postoperative immobilization in a cast and physical therapy. Patients with high level injuries (IC 1), who are barely functioning, may see little inconvenience from surgery, whereas patients who retain some active flexion of the fingers and digits (IC 8) will be less willing to give up their independence, even briefly, unless absolutely convinced that they will get a substantial benefit from surgery.

General contraindications include:

- patients whose current health status is not satisfactory
- patients who have not reached neurologic and emotional stability
- patients whose motivation is poor, or whose expectations are unrealistic
- associated features which cannot be improved preoperatively, such as severe spasticity, hypersensitivity of the fingers.

In every case, surgery should be performed in or in conjunction with a medical center specialized in the care of spinal cord injury patients, where the postoperative rehabilitation will take place.

Early (preoperative) care, patient education

Preserving the health of the upper limbs becomes a top priority in the acute postinjury and early rehabilitation period. Key is early education of the patient and family. The initial treatment plan will differ according to the level of injury; however, the principal therapy goals for all upper limbs in the initial weeks to months following injury include the following.

Prevention and treatment of hand edema This is achieved by early elevation, and by controling the position of the wrist with hand splints that maintains the wrist in near full extension and the metacarpophalangeal joints in near full flexion. Massage, compression, and exercise are key components to preventing edema.

Maintaining supple joints Correct positioning of the joints, and avoiding and treating spasticity-induced abnormal limb postures that increase the risk of developing edema-induced stiffness leading to contractures, pain, and additional spasticity help to maintain suppleness. Early, and on a regular basis, splints should be removed and *all* the joints

of the upper limb moved through as complete a range of passive motion as pain and edema allow.

Pain control In the early postinjury period, if upper limb pain interferes with proper positioning, splint wear, or exercise, an integrated, comprehensive approach to pain management should be followed. This includes a careful examination for the source of pain, such as an overlooked wrist or shoulder injury, or a concomitant brachial plexus injury. Once the likely cause of persistent pain has been established, then an orderly trial of non-pharmacologic strategies should begin, including transcutaneous nerve stimulation, traction (where spinal stability is not an issue), acupuncture, massage, and prudent use of mobilization techniques. If unsuccessful, pharmacologic measures will be needed, beginning with non-opioid analgesics such as non-steroidal anti-inflammatories, followed by mild opioid agents such as codeine. Adjuvants, including tricyclic antidepressants or selective serotonin re-uptake inhibitors, or anticonvulsants such as gabapentine or tegrotol may be beneficial. Sympathetic or peripheral nerve blocks are of limited benefit in this population.

Spasticity control Uncontrolled upper limb spasticity interferes with rehabilitation and must be treated aggressively. While destructive agents, e.g., phenol, are less often used today, botulinum toxin has gained wide use as a means of controling spasticity since its effect is essentially completely reversible within two to three months following injection. Other problems frequently requiring special treatment include the spastic biceps causing elbow flexion contracture, and the persistent supination deformity of the elbow, refractory to botulinum toxin injection. These problems may require surgical treatment including elbow contracture release, and biceps rerouting for a persistently supinated forearm. This procedure involves detaching the biceps tendon from its insertion site and then redirecting the course of the tendon so that contraction of the biceps produces pronation of the forearm rather than the normal supination. It is essential to determine preoperatively that the patient possesses an adequate function of the supinator muscle otherwise all ability to supinate the forearm will be lost. This can be done by electromyographic (EMG) analysis or, if doubt still exists, by temporarily anesthetizing the musculocutaneous nerve.

Rehabilitation Once the patient is able to sit for prolonged periods, they begin functional re-education. At this point, the patient begins to learn how to manage his or her life within the confines of the wheelchair. The patient must be taught methods of transfers and weight shifts, and methods to propel the wheelchair that do not put pathologic stresses on the important joints of the hand, including the thumb CMC joints and the MP joints of the fingers.

Functional splinting becomes an integral part of this early rehabilitation to prevent overstretching of ligaments, maintain functional position, prevent deformity, i.e., a claw deformity, protect and stabilize flail joints, and enhance residual upper limb function. The choice of splint depends on residual motor resources, e.g., patients with absent or weak wrist extension should be fitted initially with a long opponens type splint (Figure 29.3) that stabilizes the wrist, and should spend some time with their fingers taped into flexion. This is termed 'tenodesis splinting', the finger flexors purposefully permitted to become somewhat shortened to assist the patient in developing a functionally useful tenodesis grasp (Figure 29.4). For patients with stronger wrist extension, forearm pronation, and some wrist flexion, the goal of initial hand splinting and therapy should be the maintenance of normal balance between paralyzed flexors and extensors. A much shorter splint such as a short opponens splint (Figure 29.3) fitted with adaptive devices may be used. As wrist extension increases, patients may be fitted with a dynamic orthosis that uses wrist movement to effect simple grasp (Figure 29.5). The long-term use of this device varies widely.

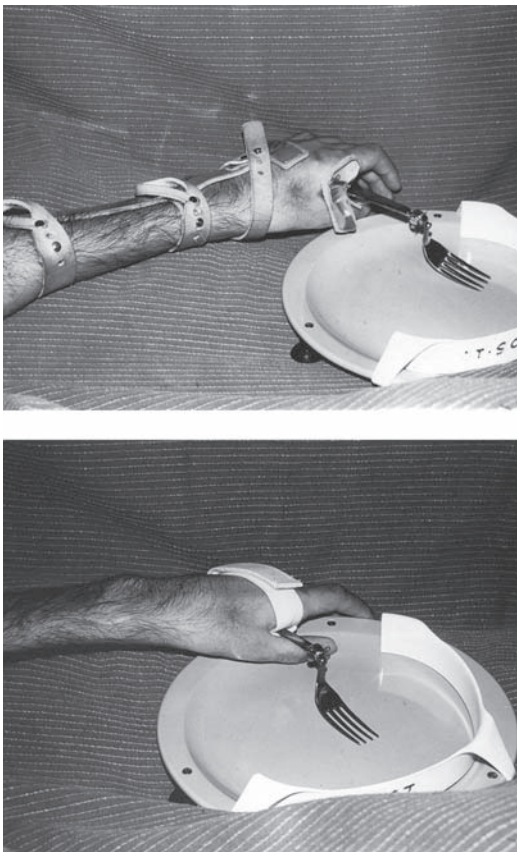


Figure 29.3
Long versus short opponens type splint.

Elbow extension

Erik Moberg stressed the importance of restoring active elbow extension for the spinal cord injured patient in terms of locomotion, transfers, and accurately positioning the

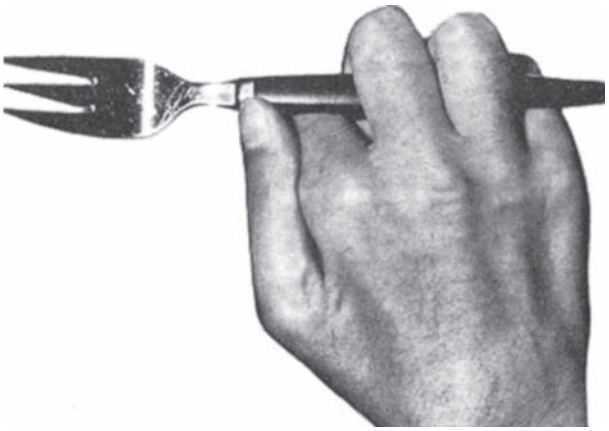


Figure 29.4
Tenodesis grip.

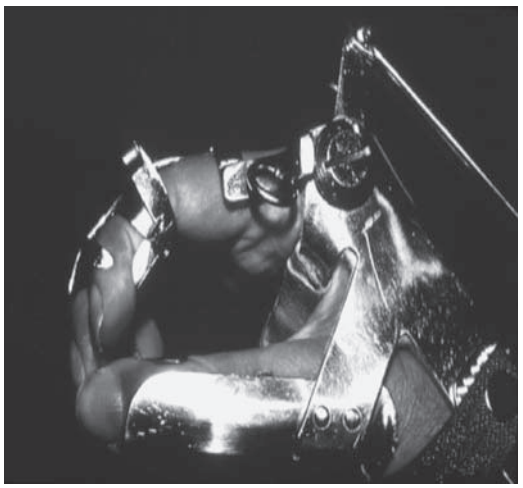


Figure 29.5
Illustration of the wrist-driven flexor-hinge splint (WDFHS).

arm in space. Additionally, Moberg stressed the beneficial effect of restoring elbow stability on the functional outcome of procedures that utilize either the BR or ECRL for muscle-tendon transfers. Since these muscles cross the flexor side of the elbow, once transferred to provide more distal function, e.g., wrist extension or finger or thumb flexion, if the elbow can be stabilized in some extension, the potential power of the muscle in achieving its new function is enhanced. Today, there are three surgical procedures advocated for restoring active elbow extension. The most commonly performed is transfer of a strong, typically posterior portion of the deltoid muscle into the triceps, termed deltoid to triceps transfer. The second commonly performed procedure is transfer of the biceps muscle into the triceps (biceps to triceps transfer). Functional neuromuscular stimulation of an electrically stimulable triceps is a newly described, third option.

Deltoid to triceps transfer (Figure 29.6)

Indications

The procedure is potentially indicated in the tetraplegic patient who has maintained a good passive range of motion at the elbow, and who has sufficient strength in the posterior half of the deltoid. An absolute contraindication is inadequate strength of the deltoid muscle. A relative contraindication is a fixed elbow flexion contracture greater than 45°. Other relative contraindications are associated with the particular rigorous time demands necessary for a good outcome following surgery, including up to 6 weeks in a cast and an equal number of weeks of progressive, protected exercise.

Operative technique

Many different techniques and technical variations have been described. In terms of principles, the following should apply to all techniques:

1. Dis-insertion of the posterior half of the deltoid to include as much periostium and fascia as possible and proximal separation from the anterior deltoid until the neurovascular leash.
2. Joining the detached deltoid to the appropriate attachment site using one of many described methods, e.g., autogenous interpositional graft (fascia lata, toe extensors, tibialis anterior tendon), synthetic interpositional material (Dacron, prosthesis), or turning proximally the central third of the triceps tendon.
3. A careful postoperative immobilization and remobilization regimen that avoids resistance forces that will elongate the transfer and weaken its power.

Biceps to triceps transfer (Figure 29.7)

Indications

While some surgeons utilize biceps to triceps transfer as their primary means of helping the patient achieve improved elbow control, others prefer to perform this transfer when there is a pre-existing fixed flexion contracture of the elbow greater than 30–45°, or when the posterior deltoid is determined too weak to permit deltoid to triceps transfer. A potential contraindication to biceps to triceps transfer is the absence of any function in the supinator muscle of the forearm.

Operative technique

While many techniques have been described, the general principles include:

1. Sectioning the primary tendon of the biceps to its point of insertion on the radius.

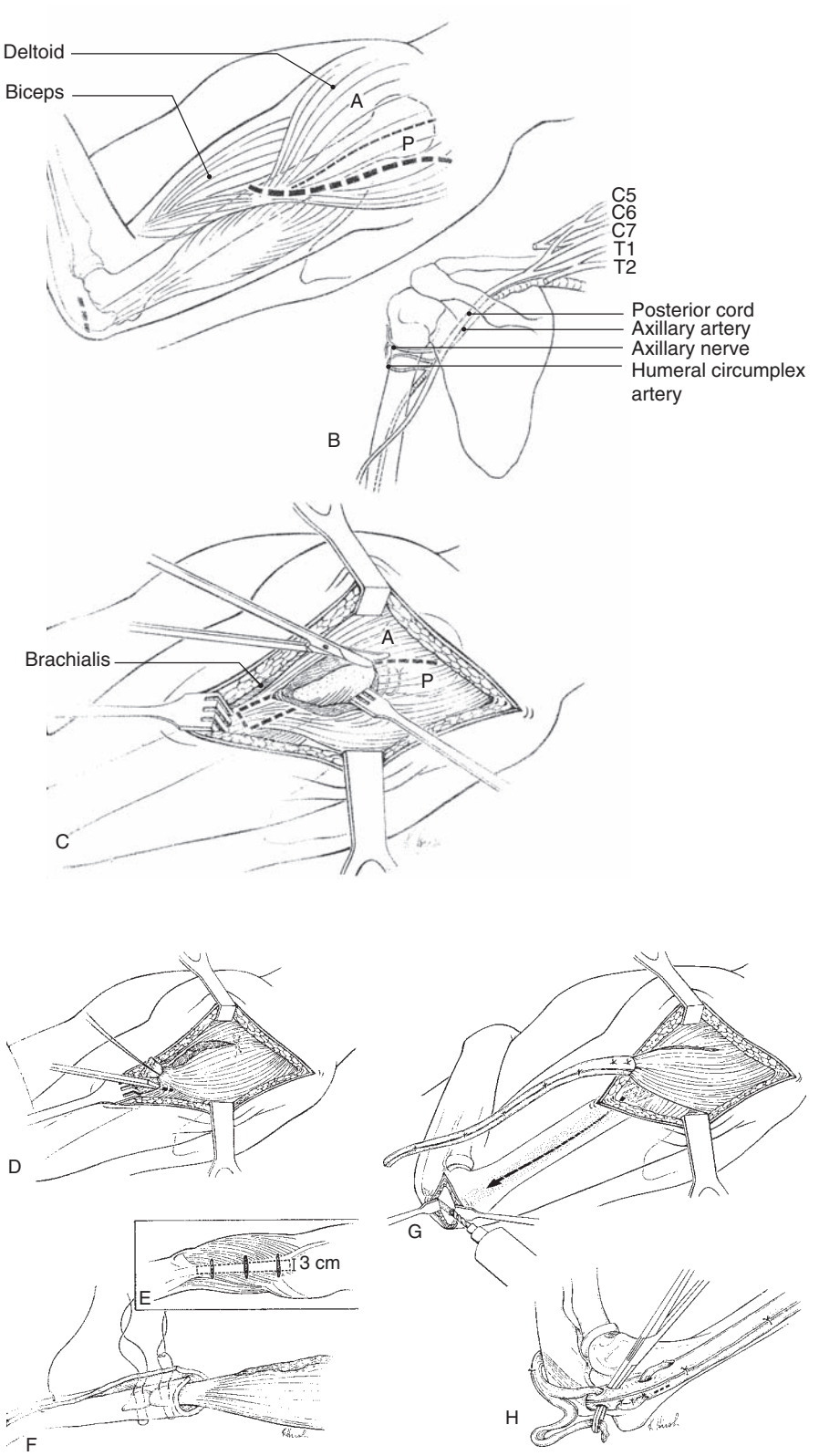
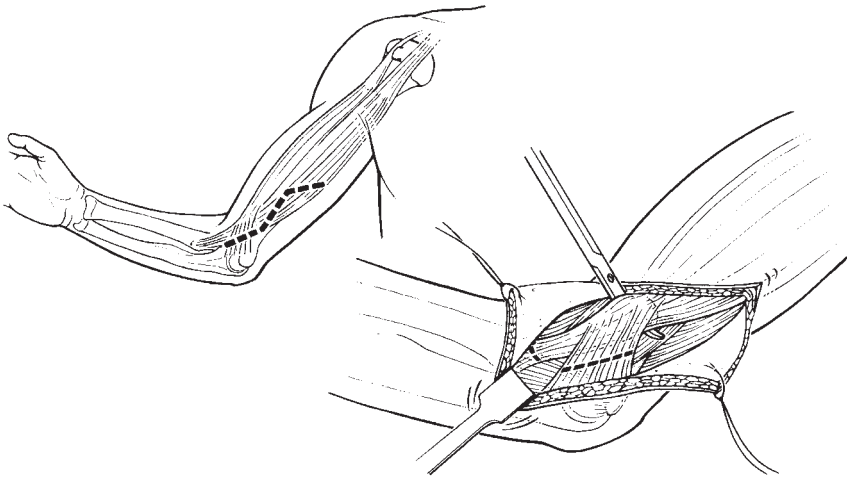


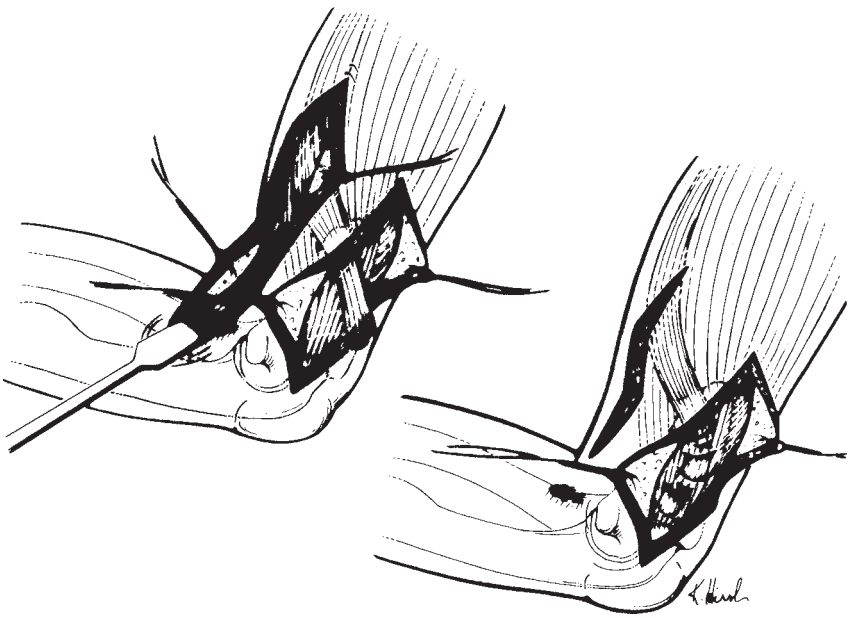
Figure 29.6

Operative steps of deltoid to triceps transfer.

2. While both medial and lateral routing of the transfer have been described, because the ulnar nerve is typically non-functional in this population, while the radial nerve is functional and thus at risk if the lateral route is chosen, the medial route is preferred by most.
3. Various methods to attach the transferred tendon have been described, including weaving into the triceps tendon, bone anchor attachment to the olecranon, and transolecranon drill holes.
4. Postoperative splinting is similar to that employed for the deltoid to triceps transfer, although most authors



(a)



(b)

Figure 29.7

Operative steps of biceps to triceps transfer.

advocate recovery of elbow flexion more rapidly after biceps to triceps transfer since there is only one tendon junction.

Surgery for the weaker patients (IC groups 0, 1, and 2)

Group 0

Patients whose injuries result in no functionally useful muscle strength (less than MRC grade 3) distal to the elbow have limited potential for improvement of upper limb function by surgery. While they may retain some shoulder and elbow strength, most must depend on various orthoses to enhance hand function. Some of these patients might benefit from functional neuromuscular stimulation, discussed below. Nearly all are candidates for improved elbow control procedures such as deltoid to triceps or biceps to triceps, described above. Many of these weaker patients will exhibit a persistently supinated forearm position, with or without a persistently flexed elbow. Biceps rerouting to treat this deformity has been discussed above.

Group 1

The group 1 patient has one MRC grade 4 or 5 forearm muscle, typically the BR. Lack of antigravity wrist extension (MRC grade 2+ or 3–) renders the hand essentially incapable of performing any but the simplest single-handed activities. The isolated hand serves primarily as a weight used to

stabilize, balance, or push light objects. The principal functional goal is active wrist extension, perhaps permitting the IC group 1 patient to be fitted with a dynamic orthosis such as a wrist-driven, flexor-hinge splint. Finally, restoring adequate wrist extension power may allow the IC group 1 patient the possibility of having the strength of any pre-existing 'tenodesis' grasp between thumb and fingers enhanced by a surgical tenodesis of the flexor pollicis longus to the radius (Moberg procedure, discussed below).

Group 2

Besides the BR, the IC group 2 patient has an additional MRC grade 4–5 muscle, typically the ECRL.

For the weaker IC group 2 patient, defined as having barely MRC grade 4 wrist extensor strength, the principal reconstructive goals are to surgically augment the power of active wrist extension and, at the same time, surgically enhance a tenodesis grip between thumb and digits. For the fortunate IC group 2 patient who has stronger active wrist extension, the reconstructive options include a surgically created pinch activated by wrist extension (tenodesis) or by tendon transfer.

In IC group 1 or 2 patients, the BR muscle, an accessory elbow flexor, is the only available transferable muscle. Wrist extensor strength can be augmented by transferring the BR into the tendon of the ECRB.

Absolute prerequisites include an adequately strong BR, a functional range of passive wrist extension and flexion, and shoulder control to assist forearm pronation. A relative prerequisite for optimum function following BR transfer is active elbow extension. For this reason, it is preferable first to reconstruct active elbow extension if there is no antigravity triceps function remaining.

Surgical procedure: BR to ECRB transfer (Figure 29.8)

The surgical technique has been reasonably standardized. Key components include:

1. mobilization of the BR sufficiently proximal (almost to the elbow) to provide adequate passive stretch of the muscle tendon unit

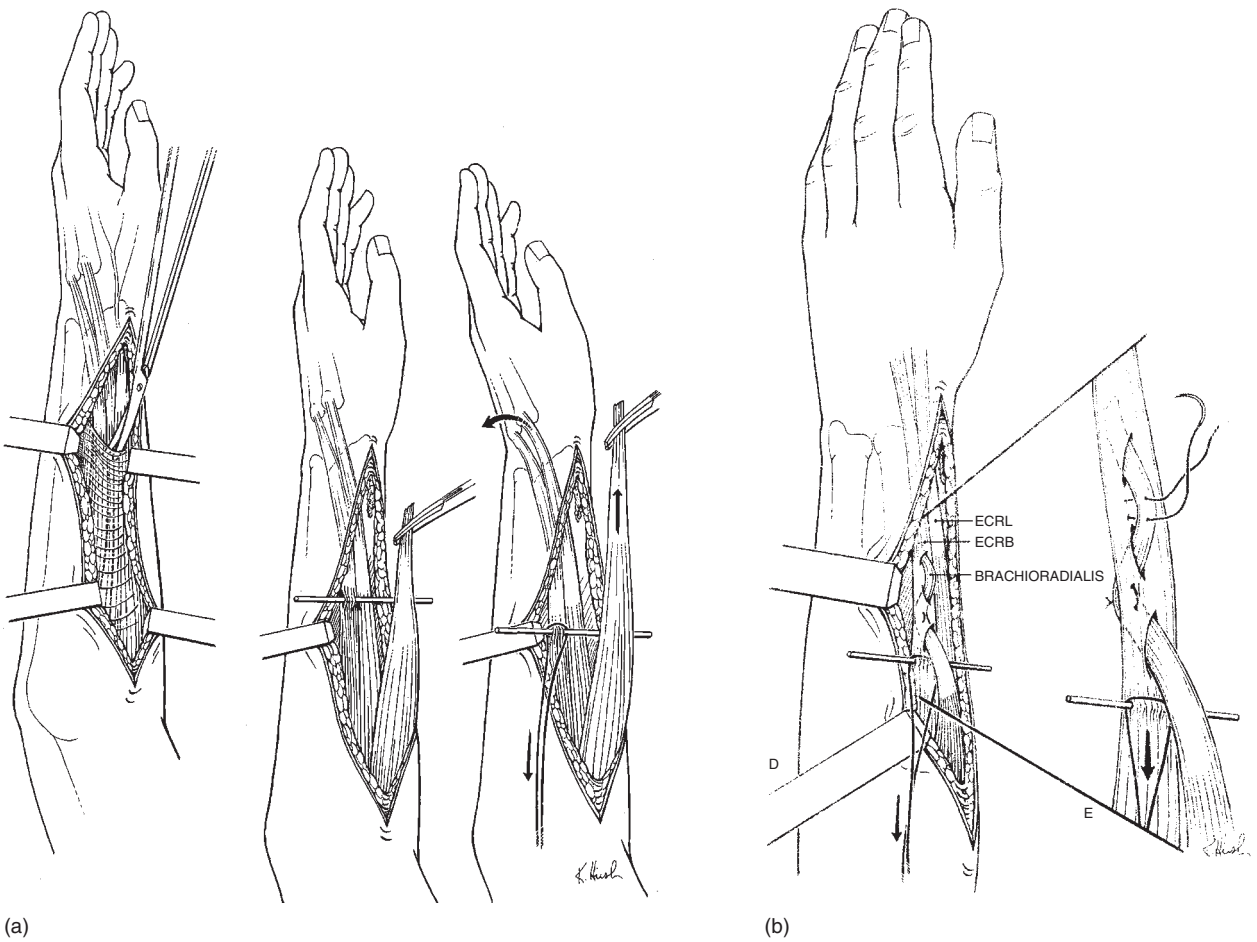


Figure 29.8

Operative steps of BR to ECRB transfer.

2. Pulvertaft[18] type weave of BR into the tendon of the ECRB
3. adjustment of tension of transfer with the elbow in approximately 30–45° of flexion
4. postoperative immobilization with the wrist in moderate extension
5. cautious rehabilitation to teach the patient how to trigger the transfer while avoiding undue stress on the transfer, followed by careful reintroduction of transfers and pressure relief activities.

Reconstruction of key pinch by FPL tenodesis[11]

If patients in IC group 1 achieve MRC grade 3+ or greater wrist extension by Br to ECRB transfer, they become candidates for creation of a lateral or key pinch. The same is true for the weaker (and stronger) IC group 2 patient. Erik Moberg is credited with demonstrating that this is a reliable and attainable function, and that the procedure has a predictable outcome if several tenets are kept in mind. Conceptually, this is a very simple operative procedure and, importantly, is essentially totally reversible should the patient decide that he or she was more functional before surgery. It represents an automatic pinch in that the tendon of the flexor pollicis longus (FPL) is anchored to the palmar surface of the radius so that, as the wrist extends, the thumb tip is pulled against the side of the index finger, termed lateral (termino-lateral) or key pinch. The other fingers are usually left supple and, unless these digits automatically flex as the wrist extends, the patient must learn to roll these digits into some flexion in order to provide a platform against which the thumb can act by the tenodesis effect. Gravity is needed to flex the wrist, thus relaxing the FPL tenodesis and allowing opening of the grip. The key pinch is most successful when neither the interphalangeal nor the metacarpophalangeal joint of the thumb flexes excessively and most motion occurs at the carpometacarpal joint. Therefore, Moberg recommended stabilizing the interphalangeal joint of the thumb with a large Steinman pin to be left permanently in place. Several additional steps were suggested, depending on the preoperative assessment of the patient's thumb, particularly the stability and passive range of motion of the thumb metacarpophalangeal joint.

Prerequisites

1. stability in a wheelchair, i.e., the ability to sit for extended periods in a wheelchair
2. sufficient control of the elbow
3. the ability to pronate the forearm so that gravity can affect wrist flexion
4. MRC 3+ or greater power in the extensors of the wrist (see above)
5. sufficient passive flexion of the wrist aided by gravity when the patient pronates the forearm
6. at least one thumb joint (usually the CMC joint) with essentially normal passive range of motion
7. the ability to position the index and preferably the middle fingers so that they serve as a platform against which the thumb can push during key pinch
8. preoperative assessment of wheelchair propulsion, transfers, and weight shifts so that postoperatively the tenodesis is not unduly stressed.

Procedure

The procedure first described by Moberg[11] has undergone many subsequent variations. One variation is illustrated in Figure 29.9, including a procedure termed the 'New Zealand split FPL to EPL transfer' that has been widely accepted as it avoids the need to fuse the thumb's IP joint and the problems associated with long-term pinning of this joint.[19]

Postoperatively, the thumb ray and wrist are immobilized for 4 weeks. If the split FPL to EPL transfer has been performed, the Kirschner wire is usually left in place for 1 to 2 more weeks and then removed. A small taped-on aluminum splint is substituted. This is removed frequently during the day and gentle pinching exercises begun. The joint is supported against significant stress for a total of 8 weeks.

Active key pinch by BR to FPL transfer

If very strong wrist extensor strength exists preoperatively, the patient is a candidate for either a Moberg key pinch procedure, or an active pinch achieved by transfer of the BR to the FPL along with thumb carpometacarpal fusion and interphalangeal (IP) joint stabilization (FPL to EPL transfer). The prerequisites are essentially the same as for FPL tenodesis.

Operative technique (Figure 29.10)

The key operative decisions include the position of CMC fusion to preposition the thumb ray for accurate pinch, tenodesis of the EPL to the radius so that the grip opens with wrist flexion (either by gravity or some residual weak function in the FCR), mobilization of the BR followed by a Pulvertaft type weave into the tendon of the FPL, and thumb IP stabilization by the New Zealand technique discussed above.

The limb is casted for approximately 4 weeks. Various removable splints are then used to assist in rehabilitation of the grip and protection against excessive force. Transfers may begin between 8 and 12 weeks. Since some patients have difficulty in activating the transferred BR in its new task (thumb flexion), various rehabilitation exercises such as biofeedback may be useful.

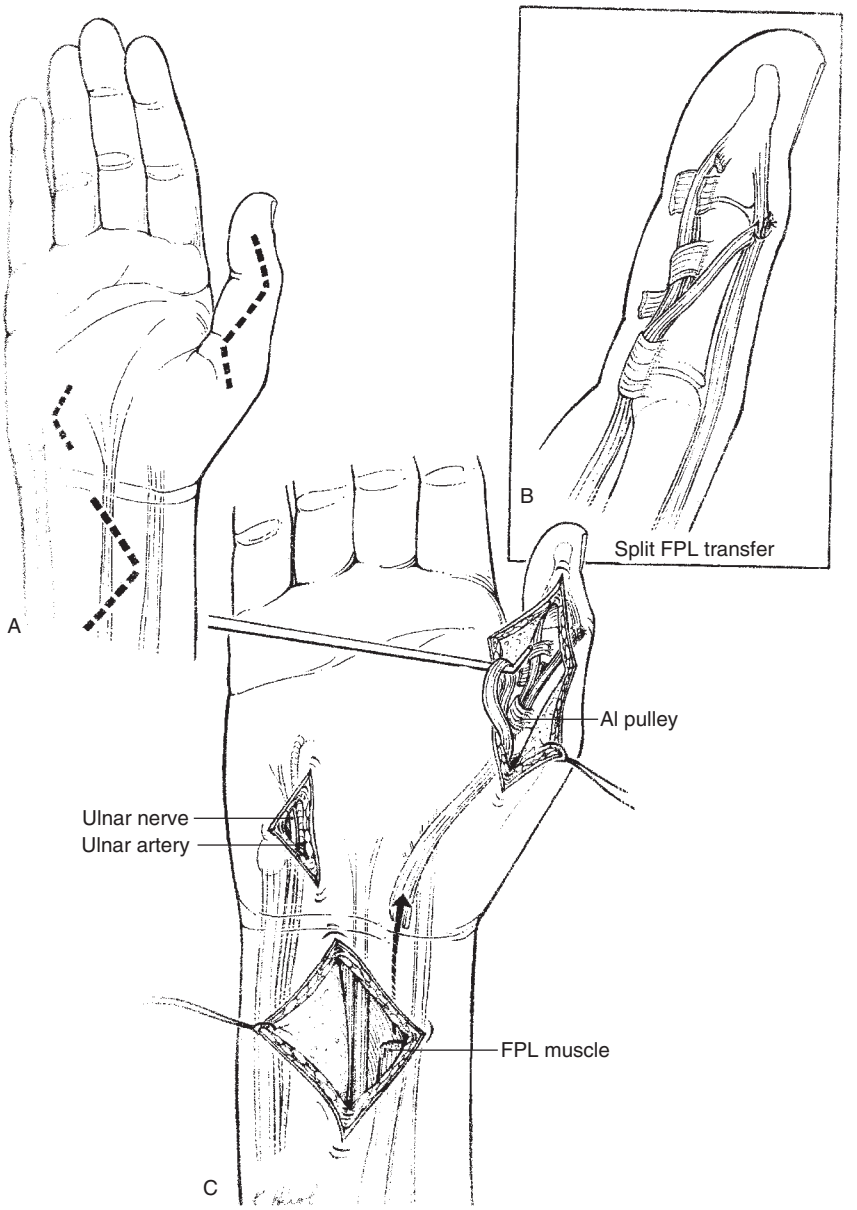


Figure 29.9

Operative steps of FPL tenodesis plus New Zealand split transfer.

Functional neurologic stimulation[20]

While the initial goal of functional neurologic stimulation (FNS) research was to enable paraplegic patients to stand and walk, other investigators began to consider the application of these technologies to the paralyzed upper limb of the tetraplegic population. Currently, FNS technology for activating thumb and digital grasp and to restore elbow extension has been clinically introduced and is being investigated to assist shoulder function in the higher C4 level tetraplegic.

FNS takes advantage of the existence of muscles no longer under voluntary control but whose spinal reflex arcs still remain intact, termed upper motor neuron paralysis, muscles that can be directly stimulated with relatively weak direct electrical currents. If, in the same arm and hand, sufficient numbers of key upper motor neuron paralyzed muscles exist, these muscles can be recruited via FNS to perform useful functions. These patients also need a means of controling the stimulation parameters, i.e., which muscles contract and when, and at what proportion of their potential contractile force. Currently, movement of the opposite shoulder is used for control.

FNS has also led to an interesting and natural conceptual leap. If a key muscle, e.g., the extensor of the digit (EDC), has a lower motor injury and is not electrically excitable, one may be able to identify any other non-key muscle in proximity, e.g., the extensor carpi ulnaris (ECU), and substitute its function, generated via FNS for the worthless key muscle by combining an old procedure, muscle-tendon transfer with new technology, FNS. In this case, the tendon of the ECU is detached from the base of the fifth metacarpal and woven into the combined tendons of the EDC.

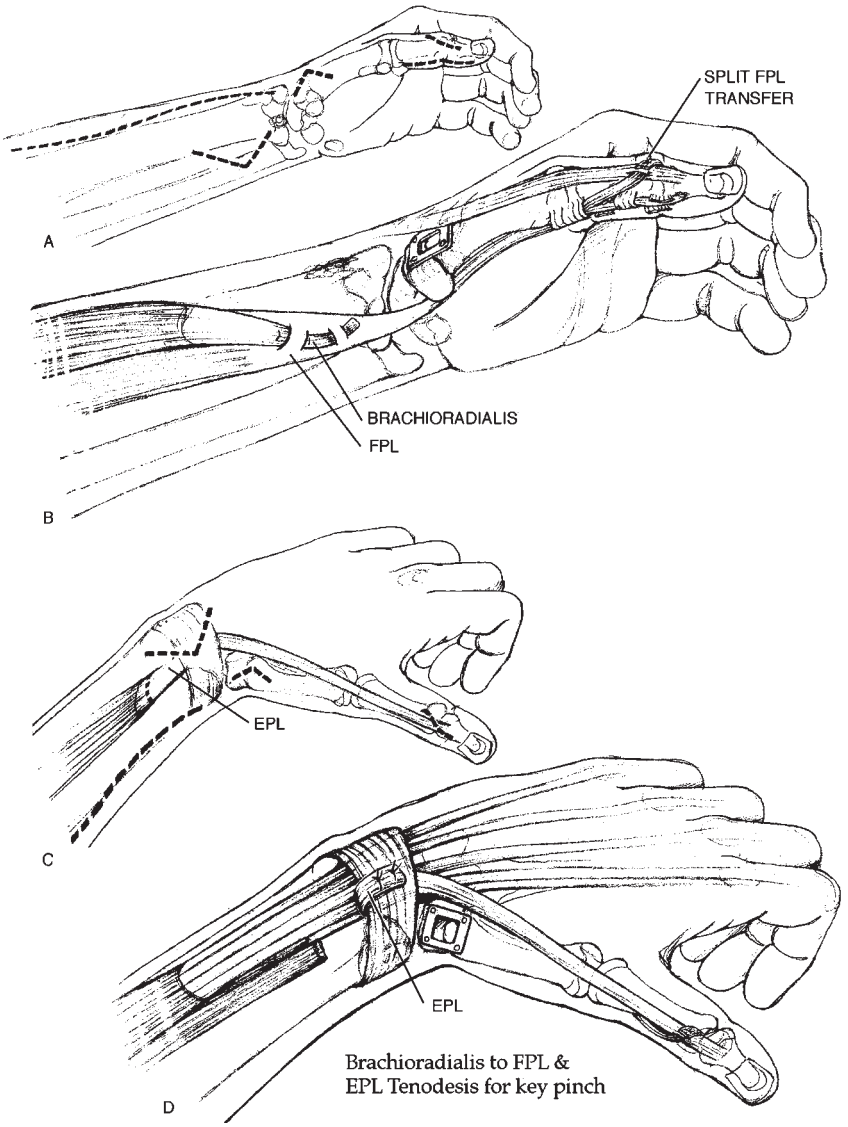


Figure 29.10
Operative steps of BR to FPL transfer.

Electrical stimulation of the ECU will now bring about digital extension.

Since the total number of muscles that can be stimulated is limited by the number of available stimulation channels, and the potential number of muscles that might respond to stimulation is vastly greater, it has been necessary to simplify the mechanics of the hand. For example, by suturing together all of the profundus tendons, stimulation of one of the profundus muscles will affect flexion of all the digits. This same simplification can be carried out for the tendons that effect extension of the fingers.

Current indications

The most widely used system, which has been implanted in patients at several IC levels including IC groups 0–3, has eight programmable stimulators used to control both extrinsic and intrinsic muscles (as available) in a manner that permits two different patient-selectable grasp patterns, a key and an opposition type grasp. The current algorithm includes the following steps:

1. preoperative electrical stimulation to determine which muscles are upper motor paralyzed (and thus able to be stimulated)
2. a period of preoperative conditioning of these muscles using an external stimulator
3. operative placement of electrodes, leads, and stimulator, and intraoperative testing to confirm proper placement (Figure 29.11)
4. several weeks of healing to allow electrode stabilization, followed by an additional period of 'muscle conditioning'
5. programming of electrodes and patient training
6. reprogramming as necessary to achieve and maintain optimum hand function.

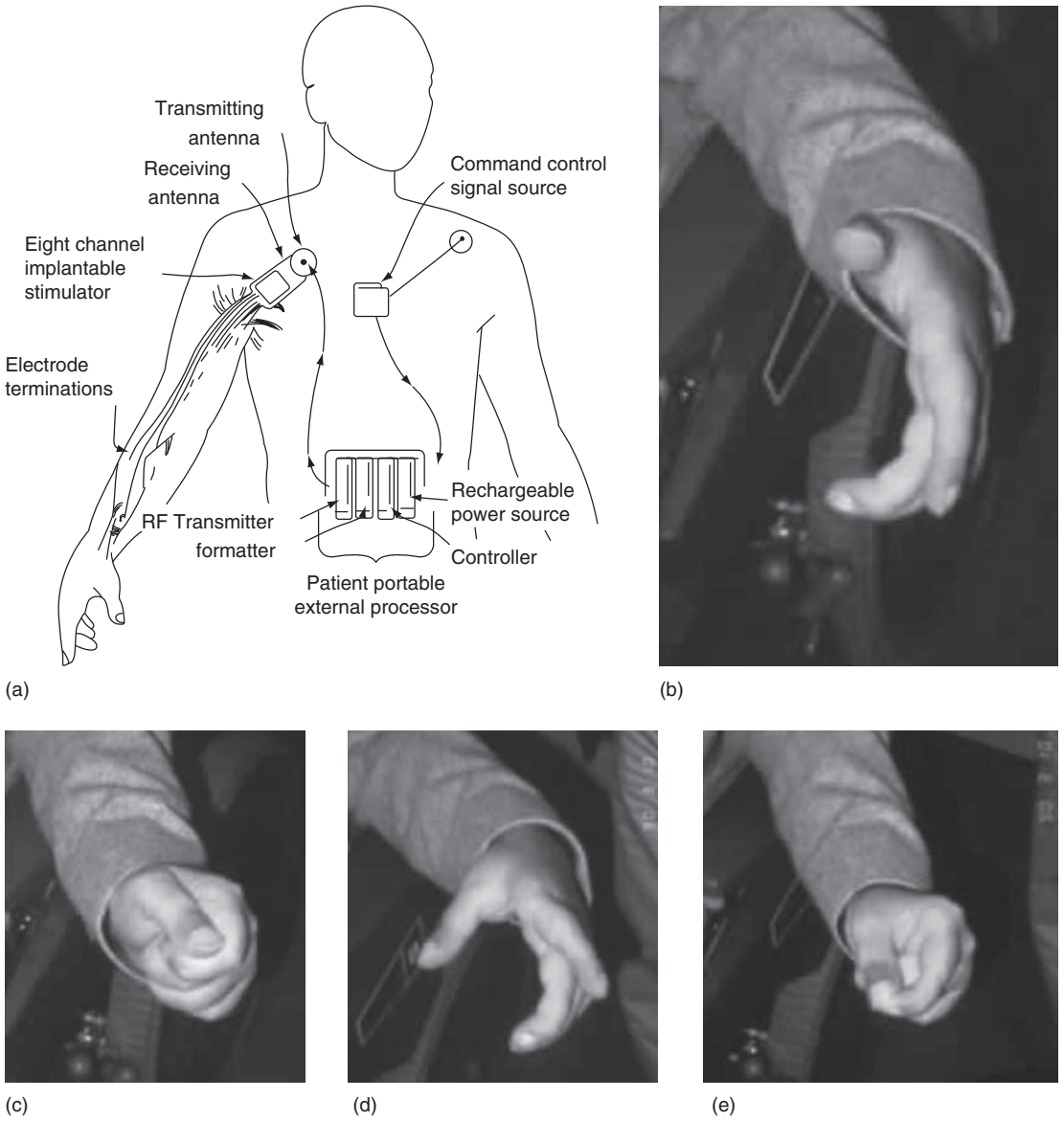


Figure 29.11

(a) Diagram of the freehand FNS system; (b and c) key grip; (d and e) opposition grip.

Current results

At the time of writing, more than 250 tetraplegic patients have been implanted worldwide,[21] including several tetraplegic children.[22] The functional outcome from the use of these devices has been well documented by means of specifically designed functional tests and by subjective patient assessment. There is general agreement among clinicians that the device extends the possibility for improved upper limb function to groups of patients, such as the IC group 0 or 1 patient, for whom standard surgery or braces offers little in the way of functional gain. Patient acceptance has been high. Unfortunately, the expense of the device and the associated implantation costs have restricted its application. Unfortunately, at the time of writing, the company that had obtained the rights to commercialize this device has decided to no longer sell or support new device implantation, because of economic concerns. It remains to be seen what the future holds for this very promising technology.

Surgery for the intermediate patients (IC groups 3, 4, and 5)

The IC group 3 patient is characterized by normal BR strength, and a strong wrist extension with both ECR muscles graded MRC 4 or above. The IC group 4 patient has, in addition, a strong pronator teres (graded 4 or above), and the patient graded IC group 5 also has a strong FCR muscle.

We have gathered these three groups of patients in the same chapter, as the basic recommendations for surgical

rehabilitation are similar, even though there are more surgical options in IC groups 4 and 5 secondary to the presence of the pronator teres.

Evaluation of IC groups 3, 4, and 5 patients

Determination of IC group 2 versus 3 solely by manual muscle testing is probably the most challenging aspect of applying the International Classification system to the tetraplegic population. The difficulty lies in assessing the strength of the ECRB in isolation. It has been our experience that when there is MRC grade 4+ active wrist extension, i.e., extension against strong but not full resistance, ECRB is at least MRC grade 4. In contrast, if wrist extension prior to surgery is graded only MRC 4, and if it is easily fatigued, then the upper limb is considered an IC group 2, and using the ECRL as a transfer should not be considered.

The triceps may or may not be functioning in IC group 3. In Moberg's experience from examining about 1000 tetraplegic patients, 25% of IC group 3 patients have a useful triceps, though usually less than MRC grade 4.[23]

There is a drastic difference both in presentation and in function between IC groups 1 and 2 patients, and groups 3, 4, and 5. In the latter, there is good muscular control of the shoulder girdle, with strong abductor and rotator muscles, and generally good adductor muscles.

By virtue of active wrist extensors, the wrist does not need the support of a static splint. Active extension of the wrist also produces an automatic pinch between the thumb and the index finger, through the so-called 'tenodesis effect'. As a consequence, the patient can grasp a number of light objects, although this pinch is not strong.

When in bed, active wrist extension helps the patients to lift their bodies out of bed using an overhead frame. When sitting, it makes the use of a manual wheelchair possible. Muscle power transmitted from the shoulder girdle and the elbow flexors allows the patient to push on the wheels with the hypothenar eminence, and self drive the wheelchair on a flat surface.

Resources for tendon transfers

In IC group 3 patients, active elbow flexion is powered by three strong muscles: the biceps brachii, the brachialis, and the BR. There is also active wrist extension, powered by two strong muscles, the ECRL and ECRB. Therefore two muscles can be spared for transfer to power the fingers and thumb: the BR and one of the ECR muscles.

The triceps muscle must be evaluated prior to planning a BR transfer. If no active elbow extension is present, the use of the BR as a transfer may lead to an unsatisfactory outcome, because the muscle may waste some of its excursion and power in flexing the elbow rather than performing its newly intended function. In those cases, active elbow extension should be reconstructed first. Regarding which ECR should be transferred, the preferred choice is the ERCL, because the extensor moment of the ECRB is greater than that of the ECRL. Therefore it produces almost pure wrist extension, whereas the ECRL also produces radial deviation.

In IC group 4 patients, the pronator teres is functioning at an MRC grade 4 level, and is available for transfer. This brings the number of transferable muscles to three.

In IC group 5 patients, the FCR is also functioning. However, this muscle should not be used as a transfer as it stabilizes the wrist, and contributes to the tenodesis effect by increasing passive extension of the fingers as the wrist flexes. Furthermore, it will enhance future tendon transfers to the fingers, because it stabilizes the wrist.

Functional goals for IC 3–4–5 patients

Pinch and grasp

With two or three muscles available for transfer, one may be more ambitious in terms of functional rehabilitation of the hand than in IC groups 1 and 2. In the past, some surgeons felt that restoring a pinch between the thumb and the index finger was a sufficient goal, whatever the level of injury.[11] However, after gaining experience, most authors became convinced that it was possible to restore both pinch and grasp in these patients;[24–28] however, today some still prefer not to restore finger flexion in IC group 3, instead performing a one-stage key pinch and release.[29,30]

Finger extension

A related issue was whether or not it was necessary to restore finger extension, along with finger flexion. Some authors[26,31,32] felt that this was too ambitious a goal, and relied on gravity-induced finger extension via the tenodesis effect. Others felt it was important to restore finger extension, in order to avoid the risk of a closed hand posture after restoration of the finger flexors. Some[24] advocate an active restoration of finger extension using the BR, whereas others[33,34] prefer to achieve finger extension through a tenodesis of the finger extensors to the radius. Currently, there is no consensus regarding this issue, and both schemes are still advocated.

We[15] have often been dissatisfied with the transfer of BR to the finger extensors when the FCR is paralyzed (IC groups 3 and 4) as the lack of anterior wrist stabilization makes this transfer much less effective. We use it only when FCR is strong (IC groups 5), and we prefer to restore finger extension by passive procedures in IC groups 3 and 4, and save the BR for a more important purpose. House,[34] who has done both on several patients (one on each side), noted that active finger extension provided by the BR in the first method was not subjectively better than the extensor

tenodesis in the other method, and currently cannot justify the use of a good motor to restore finger extension.

Thumb flexion

All surgeons agree on the value of constructing a lateral pinch between the thumb pulp and radial side of the index finger, but there is no consensus on how to achieve this. Moberg[35] used a passive tenodesis of FPL to the radius. Zancolli[10] favors either transfer of a supernumerary ECR when present, or an active tenodesis of the FPL by a side-to-side suture between the FPL and the ECRB at the distal forearm level. This produces thumb flexion by contraction of the ECRB. Others have used the BR, or the pronator teres (PT) when it is strong enough in order to activate the FPL. Still others[36] have used two simultaneous transfers for the thumb: one for thumb flexion (PT to FPL) and one for thumb adduction-opposition. The latter is done with the paralyzed flexor digitorum (FDS) to the 4th finger, left as an 'in situ tendon graft', sutured proximally to the BR as an active donor, rerouted around a palmar fascial pulley and sutured around the MP joint of the thumb distally.

Thumb posture

As the thumb can be activated by only one or two muscles, as opposed to its normal complement of nine or ten, it is likely to assume a pathologic posture, especially if the patient's joints are lax, which is likely to impair the quality and strength of the lateral pinch. The thumb needs to be stabilized either by soft tissue procedures (tenodesis), or bony procedures (fusion). When only the FPL is to be activated, most authors prefer to fuse the thumb's CMC joint. If both thumb flexion and thumb adduction-opposition are to be restored by tendon transfer, then a tenodesis is preferred. Every time the FPL is activated, there is a need for IP joint stabilization, which would otherwise assume a hyperflexed posture. The preferred procedure is now a 'split FPL to EPL' stabilization, rather than arthrodesis. Less often does the MP joint need stabilization. If required, one can perform an arthrodesis or a tenodesis.

Intrinsic substitution

In the absence of the synchonizing effect of the intrinsic muscles, active muscle transfers to the finger flexors will result in digital flexion, beginning at the distal IP joint and the finger tip rolling into flexion rather than sweeping broadly along the spiral that the normally innervated finger tip follows. This will tend to push large objects out of the grasp (Figure 29.12). Also in the absence of intrinsic muscle activity, the action of the extrinsic extensor muscles at the metacarpophalangeal joint is unopposed. In hyperextending the metacarpophalangeal joint, the extrinsic extensor forfeits the excursion that might allow it to extend the IP joint. This imbalance gives rise to the 'claw hand', characterized by metacarpophalangeal hyperextension and IP joint flexion.

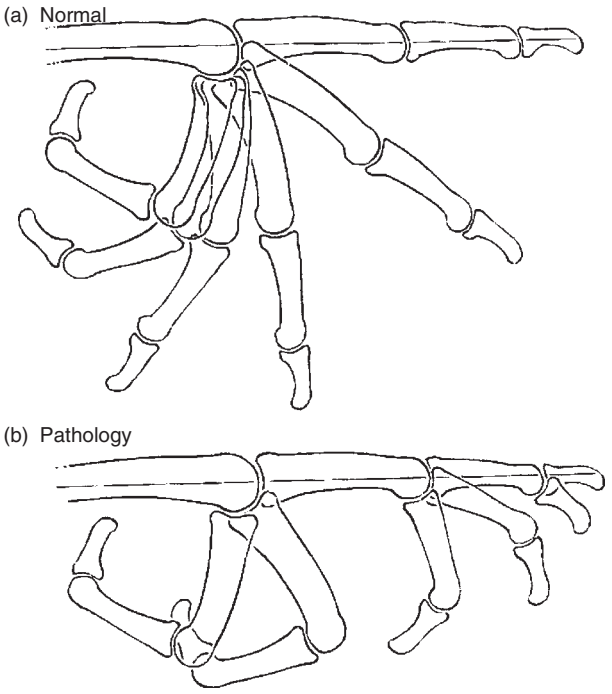


Figure 29.12

(a) In the normal hand, finger flexion sweeps through a large arc, and the hand is able to grasp large objects. (b) Absence of the intrinsic muscles causes the fingertips to roll into flexion and push larger objects out of the hand.

Since most tetraplegic patients do not possess enough transferable muscles to allow intrinsic substitution by standard tendon transfer procedures, static procedures must typically suffice. One of two procedures, the first attributed to Zancolli[37] and termed the 'lasso procedure', and the other described by House et al,[38] may be utilized for the tetraplegic patient.

Specific preparation of IC groups 3, 4, and 5 patients

The main goal of the preoperative management of the upper limb should be to prevent deformities and keep all joints supple. Previously, it was considered too ambitious to restore both types of grip (pinch and grasp), and a number of authors focused on restoration of lateral thumb to index (key) pinch. In order for this pinch to be effective, the index (and middle) finger had to assume a rather flexed position, so the thumb would not tend to escape under the index finger. Therefore, therapy recommendations included allowing the fingers to 'stiffen' somewhat. They were splinted in a position which would favor tightening of the flexor tendons, and passive ranging was performed with this goal in mind.

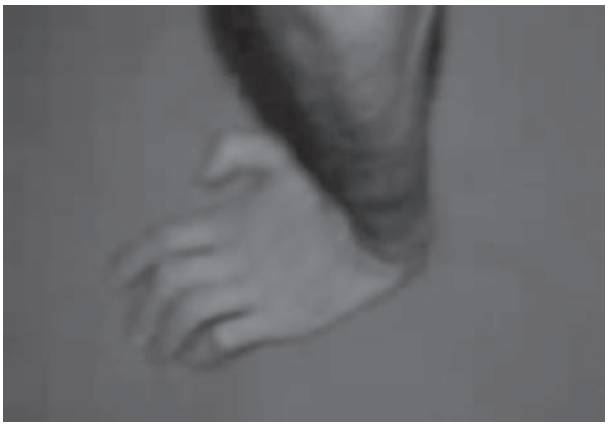


Figure 29.13
Body transfer on flat hand.

More recent acceptance of the benefits of surgical procedures have led many rehabilitation teams to reassess the goals of early splinting and therapy in these patients. The main goal for IC groups 3 and above should be to keep all joints of the hand absolutely supple, and to maintain a normal balance between the paralyzed flexors and extensor tendons. This is performed through passive mobilization of the joints through their entire range, and splinting, both static and dynamic, as required according to existing or potential contractures.

Another goal is to teach the patients, prior to surgery, how to protect their hands so as not to compromise the results of surgical procedures. This is especially true of passive tenodeses, which will slacken with time, if submitted to regular overstretching. These patients are often initially taught to perform transfers by using the flattened hand to support their body's weight on the fully extended wrist (Figure 29.13). This position may be one of stability, but it does endanger tendon transfers to the thumb and digit flexors, and will predictably elongate intrinsic reconstruction procedures.

Therefore the patients must be taught more protective methods of transfers. One method consists in rolling the fingers into near full flexion, as in forming a closed fist, and then using the dorsal surface of the flexed proximal phalanges as the platform for weightbearing. Another method, less protective but still better than a flat hand transfer, consists in rolling the fingers into flexion, and using the dorsal surface of the middle and terminal phalanges as a platform, with the wrist hyperextended.

Preferred procedures

Each patient is different both in terms of presentation and in terms of needs, and the surgical planning is adapted according to these parameters. There is no such thing as a 'standard procedure' for a specific group of patients; several options are usually discussed with the patient and the final surgical planning is matched to the patient's unique presentation and specific requirements.

However, we usually plan in all three groups to restore both key pinch, and digital grasp and release. This is usually performed in two stages, the extensor phase first, then the flexor phase. Each stage requires 4 weeks of postoperative immobilization, then physiotherapy for 6 to 8 weeks. Therefore reconstructive surgery for one hand is usually completed in 6 months (provided the triceps does not need previous surgical rehabilitation). The patient must be fully aware that the first operative step will not bring any functional improvement, and that the result will only be achieved after the second step.

Group 3

The first surgical stage, referred to as the 'extensor phase', focuses on rehabilitation of the extensor tendons. This phase includes:

- passive tenodesis of the extensor tendons of the fingers (EDC) and of the thumb (EPL)
- thumb CMC joint arthrodesis
- intrinsic tenodesis (lasso or House procedure).

The second phase, consisting of muscle transfers to the flexor tendons to the thumb (FPL) and fingers (FDP), is termed the 'flexor phase'. It includes:

- transfer of ECRL to the finger flexors
- transfer of BR to FPL
- 'split' FPL to EPL stabilization of the thumb IP joint
- if there is an accessory ECR muscle, transfer to FPL, with BR used for another function.

Extensor phase

A single longitudinal incision is performed on the lateral aspect of the forearm, prolonged as needed for thumb procedures. Alternatively, multiple incisions may be used over each operative site (volar forearm, dorsal forearm, CMC joint).

The preferred sequence for the operative steps is as follows.

Examine for supernumerary ECR muscle The presence and size of a supernumerary ECR muscle is first evaluated. If the muscle is large enough it will be used as a transfer, usually to FPL during the second phase. In this particular circumstance, we may consider using the BR to activate finger and thumb extensors during the first phase.

Tenodesis of extensor tendons to radius When no supernumerary ECR is available, the tendons of the EDC and EPL are tenodesed by implantation into the radius. The tendons are dissected proximal to the dorsal retinaculum, sutured together side by side, severed proximal to the suture, then

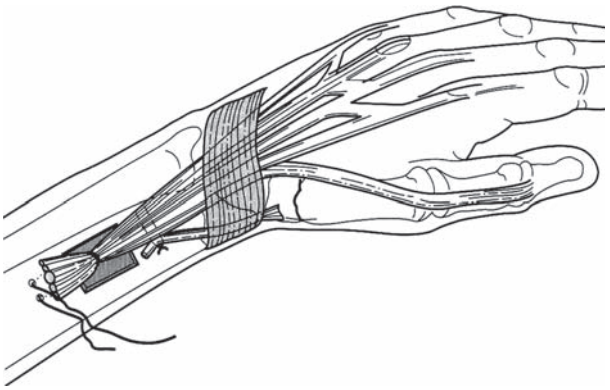


Figure 29.14
Tenodesis of extensor tendons to radius.

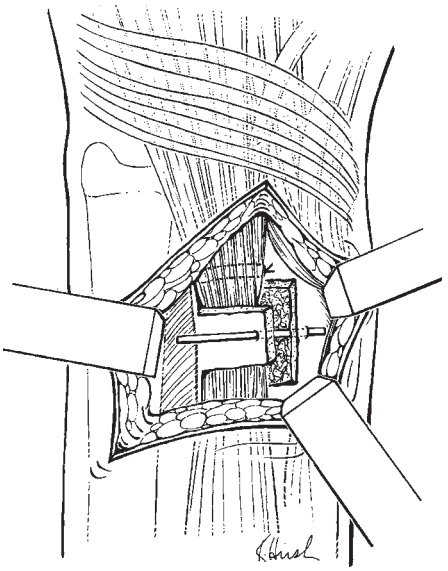


Figure 29.15
House's technique of tenodesis.

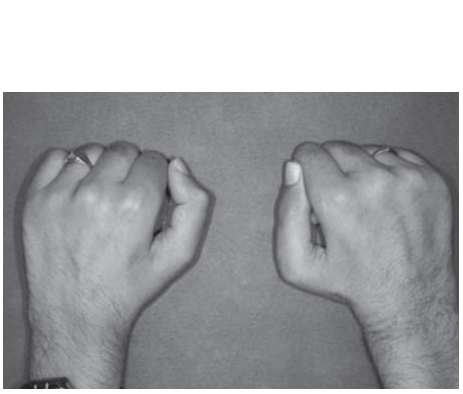
drawn into a dorsal cortical window created in the radius (Figure 29.14). The tension, adjusted by passive maneuvers of the wrist, must be such that the fingers fully extend at the MP joint when the wrist reaches 30° of flexion.

Alternatively, the technique described by House et al[38] may be used, as illustrated in Figure 29.4. It consists in elevating a C-shaped piece of the dorsal cortex of the radius, and sliding the extensor tendons into the channel under the remaining bony ledge. A fine Kirschner wire closes the dorsal gap to help retain the tendon slips within the bone channel (Figure 29.15). The tendon of the EPL may alternatively be tenodesed separately, around its retinacular compartment.

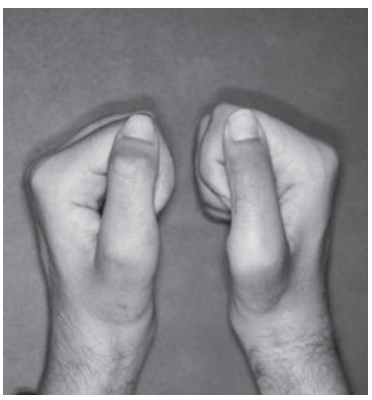
Arthrodesis of the thumb CMC joint Only the cartilage is removed from the trapezial and metacarpal articular surfaces, and care is taken to keep the whole bone stock. This allows the surgeon to choose the exact position in which the joint will be fixed, by moving the joint around until the desired position has been obtained. The joint is then temporarily fixed with K-wires, and the position of the thumb evaluated throughout the whole passive range of flexion–extension of the wrist. The usual arthrodesis position averages 25 to 30° of anteposition (palmar abduction) and 30° of abduction (Figure 29.16), in order to position the pulp of the thumb at the level of the PIP joint or distal part of the proximal phalanx of the index finger. We usually fix the arthrodesis with pneumatic staples or a rectangular mini-plate.

In cases where the CMC joint is spontaneously stable, one may decide not to fuse it. Additional tenodeses will be needed in these cases in order to keep the thumb abducted (see alternative procedures below).

Intrinsic tenodesis Two passive procedures may be utilized in these groups of patients, the 'lasso procedure' of Zancolli,[37] and the intrinsic tenodesis described by House et al.[38] The former is used when tension on the digital extensors results in good extension of the PIP joints, provided that the metacarpophalangeal joints are kept from hyperextending. House's procedure is used when this maneuver does not effect good extension of the PIP joints, indicating that the



(a)



(b)

Figure 29.16
(a and b) Position of CMC joint arthrodesis.

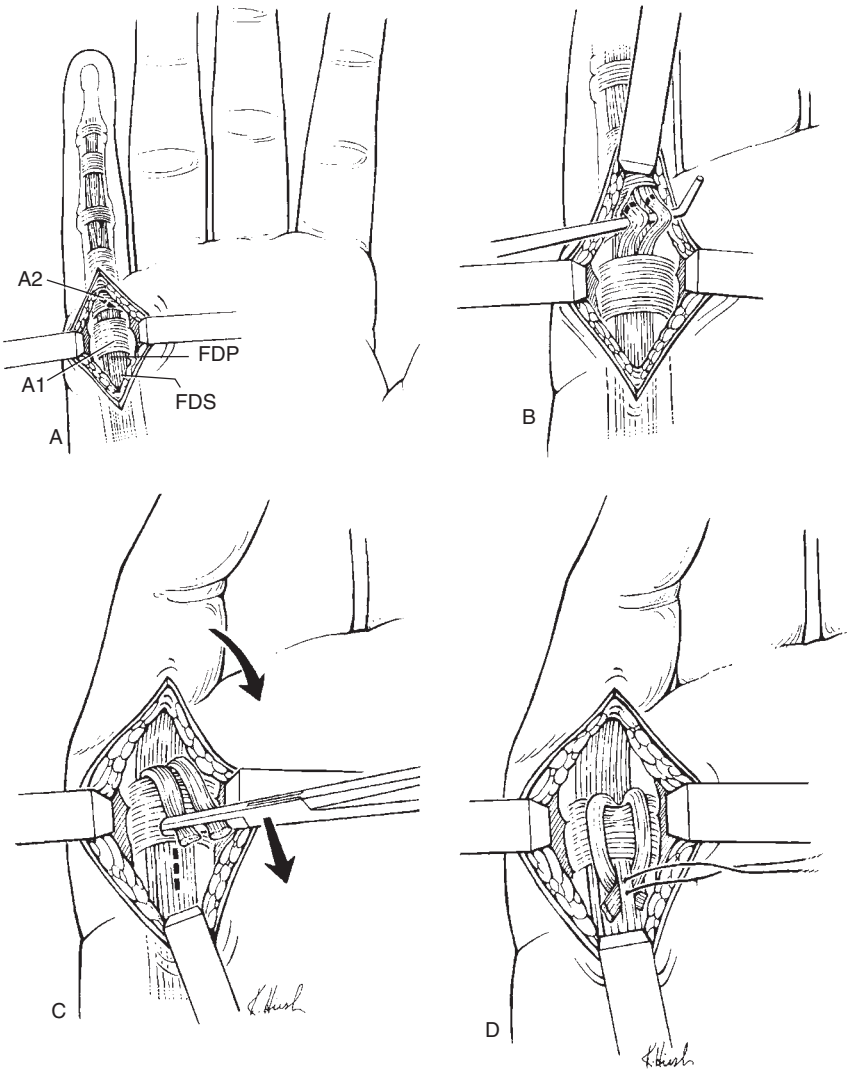


Figure 29.17

Operative steps of the lasso procedure. FDP, flexor digitorum profondi; FDS, flexor digitorum superficialis.

extensor mechanism over the joint has elongated, in which case the lasso procedure will not augment interphalangeal extension.

Lasso procedure (Figure 29.17) The flexor digitorum superficialis (FDS) muscles, which are paralyzed at the upper motor neuron level, retain some stretch reflexes through the intact spinal reflex arc and relatively normal viscoelastic properties. Zancolli[37] proposed using them as an elastic tenodesis: the superficialis tendons are inserted into the flexor sheath at the distal margin of the A1 pulley, thus becoming flexors of the metacarpophalangeal joints.

Through either a single transverse incision in the distal palmar crease or four longitudinal incisions, the flexor sheath and the margins of the A1 pulley are located. The proximal third of the A2 pulley is opened via a short L-shaped incision.

Next the flexor superficialis is retracted proximally, and its two tails cut as distally as possible. The tendon is pulled proximally around the distal margin of the A1 pulley and it is reflected over itself under tension. A braided synthetic non-absorbable suture is then performed, either by weaving the two slips of the superficialis through the substance of the superficialis tendon proximal to the A1 pulley, or by suturing the tendon to itself and to the A1 pulley. It is important to leave the flexor profundi undisturbed throughout the whole procedure so as to avoid creating adhesions between the two tendons.

This tenodesis is subject to slackening if the MP joint is inadvertently passively extended. It must therefore be protected during the rest of the procedure, as well as postoperatively.

House procedure (modified) This procedure is a modification of Riordan's[39] tenodesis procedure. It is often performed for only the index and middle fingers. A tendon graft is obtained from the plantaris tendon, the palmaris longus, or a strip of fascia lata. Through three small skin incisions it is passed under the extensor digitorum tendon at the level of the metacarpal neck. By fully flexing the metacarpal, a small straight hemostat is introduced palmar to the intermetacarpal ligament, and along the course of the intrinsic muscles, to exit in the dorsal digital incision. By means of an intermediate suture, and a second hemostat running in the opposite direction, the tendon is grasped and pulled distally to emerge in the distal incision site.

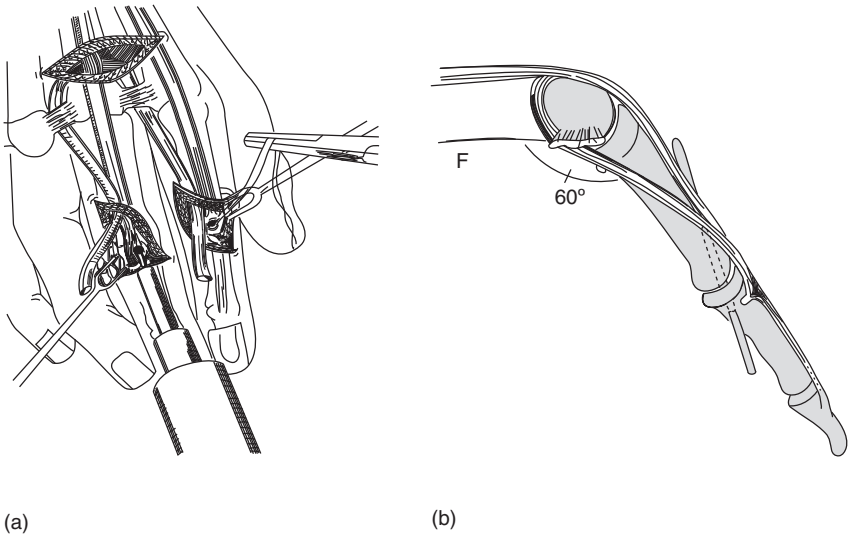


Figure 29.18
(a and b) The House procedure.

This maneuver is carried out on both aspects of each finger. A suture anchor is then used to fix the tendon graft to the bone at the base of the middle phalanx, and a Kirschner wire holds the PIP joint in near full extension (Figure 29.18).

Postoperatively, the metacarpophalangeal joints are maintained in 60° of flexion using a dorsal plaster slab.

Postoperative rehabilitation The cast is removed after 4 weeks. If a lasso procedure was performed, a light dorsal blocking splint is applied, maintaining the MP joints in 60° of flexion; it is removed only during therapy sessions. The MP and IP joints are gently mobilized into flexion by passive exercises, but at no time are the MP joints passively extended. The splint is continued at least until the second operative phase.

If a House intrinsic procedure was performed, the Kirschner wires are removed at the time of cast removal. A dorsal blocking splint that prevents the metacarpophalangeal joints from fully extending is fitted and the patient begins gentle exercises. The rehabilitation for this procedure must be carried out in a non-aggressive fashion to avoid overstretching the tenodesis.

Fusion of the CMC joint of the thumb is monitored with regular X-rays. It is usually sound by the time the patient is ready for the second phase, and if temporary removable fixation has been used, it will be removed at this time.

The second operative step can be performed when joints have recovered full passive motion. The only joints where passive motion is not encouraged are the thumb CMC joint which underwent arthrodesis, and extension of the finger MP joints if an intrinsic substitution procedure has been performed. Active muscles must also be exercised back to their preoperative strength, especially those which will be transferred during the second phase (BR and ECRL).

Flexor phase

This phase consists in activating the finger flexors profondi (FDP) and the thumb flexor (FPL). In IC group 3 as well as in groups 4 and 5, the FDPs are activated by transfer of ECRL. The FPL is activated differently in IC group 3 than in groups 4 and 5. In the rare occurrence of a supernumerary ECR muscle available for transfer, it is employed to activate FPL. Otherwise, the BR is used for this transfer.

In all cases, the thumb IP joint needs stabilization because the unopposed action of the new flexor tends to cause the IP joint to assume a permanently flexed position. The flexed thumb is then likely to come underneath the index during attempted pinch, resulting in a pinch posture between the dorsal aspect of the thumb and the volar aspect of the index finger (Figure 29.19) that is far less functional. Rather than performing a permanent bony fixation of the IP joint, as advocated by Moberg,[11] or a conventional arthrodesis, we have adopted the procedure described by Mohammed and co-authors.[19] This procedure, termed the 'New-Zealand split of FPL to EPL transfer', preserves some IP joint movement while stabilizing the IP joint.

Surgical exposure Surgery is performed through the same incision as in the extensor phase, prolonged proximally over the BR muscle belly. First the BR is dissected: the distal tendon is divided from the radius as far distally as possible. Then the muscle is elevated from distal to proximal, with care to leave the radial sensory nerve and the radial vascular bundle undisturbed. The muscle must be dissected as far proximally as the elbow crease, so as to free it from its fascial attachments. This is essential so as to obtain adequate muscle excursion.

Next the ECRL is exposed distal to the dorsal retinacular ligament. After the ECRB has been identified, usually 1 or 2 cm ulnar to the ECRL, the terminal tendon of ECRL is severed from its bony insertion, then dissected free from its investing fascia, as far proximal as its main neurovascular bundle in order to gain maximal tendon excursion.

A heavy suture is looped through the terminal part of both tendons for further manipulations, and the two muscle-tendon units are protected and kept moistened with wet gauzes throughout the rest of the procedure.

Figure 29.19
The overflexed thumb reaches under the index, creating a less functional pinch.

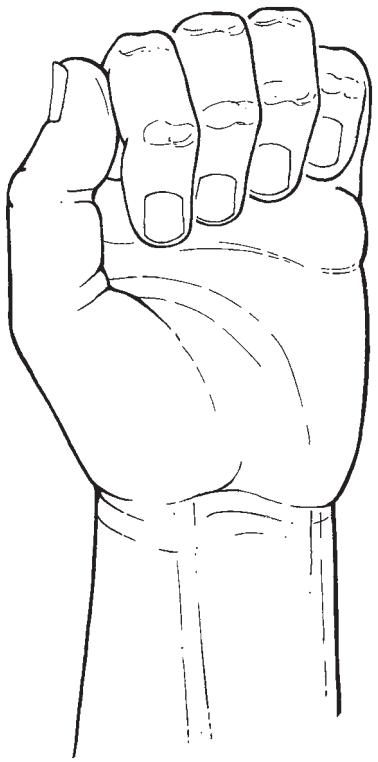


Figure 29.20
The reverse cascade.

Stabilization of the thumb IP joint The 'New-Zealand split transfer' consists in transferring half of the distal end of the FPL to the EPL over the IP joint (see Figure 29.9b). After FPL has been identified, a blunt probe is used to find the small midline split in the tendon's structure. The radial half of the split FPL tendon is then divided distally, delivered proximal to the distal flexor pulley, and routed dorsally to the EPL. The tendon slip is woven through the EPL, and the IP joint is temporarily stabilized in slight flexion with a Kirschner wire. The pin is either removed at one month, and replaced by a light IP protection splint for one more month, or removed at 2 months postoperatively.

Transfer of ECRL and BR Next the donor muscles are transferred to the recipients. The FDPs are identified above the wrist. The tendons of all FDPs are sutured together with a strong suture running obliquely so as to set more tension on the radial fingers than on the ulnar ones, setting them in a 'reverse cascade' as opposed to the normal physiologic cascade of the fingers at rest (Figure 29.20).

The ECRL is then routed palmarward, deep to the radial vascular bundle and the FCR, and woven back and forth through the now combined tendons of the FDP. Multiple non-absorbable sutures are used to anchor donor and recipient tendons together. Tension of the transfer is set with the wrist in neutral position, such that the little finger remains in the normal resting posture, and flexion increases for each finger from ulnar to radial, displaying the reverse cascade described above.

Then the BR is transferred to the FPL. The latter may have a short tendon, in which case the BR must be inserted as proximally as possible in the FPL tendon so as to be able to interweave these two tendons once or twice.

Setting the tension of this particular transfer is probably one of the most critical of all transfers in tetraplegia. If the tension is too tight, it will prevent adequate opening of the first web even with the wrist fully flexed, making it difficult to obtain and hold larger objects. If the tension is not tight enough, the result is worse because either the thumb pulp will not come into contact with the index finger even with a fully extended wrist, and the patient can only seize large objects, or the thumb does reach the lateral side of the index finger but with no strength, because the transferred tendon is at maximum course, and the patient cannot hold small objects strongly. For optimal results, the suture is best performed with the elbow slightly flexed (40°) and the wrist in neutral position. A temporary suture is inserted, then the position of the thumb is tested through the whole range of

wrist passive motion, and the final tension is adjusted accordingly.

Postoperative care A well-padded cast is applied below the elbow, maintaining the wrist in slight flexion, and the whole length of the fingers and thumb, slightly more flexed than their spontaneous resting position. As the elbow is left free, the patient and his attendants must be carefully instructed to avoid strong contraction of the BR during the first 4 weeks. This is particularly important for those patients who are used to lifting their body by contracting their elbow flexors. Fearing such ruptures, some authors do recommend immobilizing the elbow after transfer of the BR.

The cast is removed after 4 weeks and the patient begins exercising his transfers with the therapist. Only active flexion of the fingers and thumb is encouraged for the first 2 weeks, then resisted flexion is progressively initiated, and active and passive extension are reinitialized. It is important to protect the sutures against great forces for an additional 4 weeks after removal of the cast, and the patient should preferably be returned to an electric wheelchair for this period.

When a transferable accessory ECR muscle is present In this rare situation, the accessory muscle is transferred to the FPL. The BR is then available for another function. We have used it to restore active extension of the fingers, but this procedure has not been reliable in IC group 3 patients. This may be due to the absence of a volar wrist stabilizer, as all wrist flexors are paralyzed in this group. Instead, we now tend to use the BR for restoring active wrist flexion by transferring it to the FCR. The goal of this procedure is to increase finger opening through the tenodesis effect. We use it more specifically in those patients whose finger opening is not yet sufficient after the extensor phase. Otherwise we may decide to save the BR for a potential other function at a later stage, depending on the functional results and the patient's needs.

Groups 4 and 5

In IC group 4, the PT is strong and available for transfer. In IC group 5, the FCR is strong, but experience has shown that it should not be used as a transfer. Therefore the surgical options are similar for both IC groups 4 and 5. The PT is the only functioning pronator muscle in these two groups. However, it can be transferred and yet retain most of its pronator function if the direction of the transfer does not differ much from its original direction. In this respect, it can be safely used to activate the FPL.

The BR is then available for another function. As stated above, it can be transferred to the finger extensors provided there is enough volar stabilization of the wrist (FCR graded MRC 3 or above). Other options include increasing the number of transfers directed to the thumb.

Surgery is performed in two stages, as in IC group 3, with the extensor phase performed first, then the flexor phase.

Extensor phase

If FCR is less than MRC grade 3, the extensor phase is identical to group 3:

- tenodesis of EDC and EPL to the radius
- thumb CMC joint fusion
- intrinsic procedure (lasso or House), preferably on all four fingers.

If FCR is graded MRC 3 and above, the finger and thumb extensors can be activated by the BR. The procedure then includes:

- transfer of BR to EDC and EPL
- thumb CMC joint fusion
- intrinsic procedure.

BR to EDC and EPL After fusion of the thumb's CMC joint as described for IC group 3, the extensor tendons are exposed proximal to the dorsal retinaculum. After the tendon slips of the EDC have been sutured together and to the tendon of the EPL, the BR is dissected and transferred to the extensors. Rather than passing the tendon of BR through each individual EDC tendon slip, it is buried in the middle of the tendinous mass, and sutured to it with two or three strong horizontal mattress sutures. This makes tension adjustment easier.

Tension is set with the elbow in 40° flexion, the wrist in neutral, and the MP joint in 20° of flexion. Tension should be such that, during passive flexion of the wrist, the MP joints start extending when the wrist reaches neutral from an extended position. The tension on the EPL tendon is typically set slightly looser than that of the EDC.

Associated procedures are performed as in group 3: stabilization of the thumb with CMC joint fusion (or abduction tenodesis if the joint is stable), and intrinsic tenodesis (lasso or House procedure).

Postoperative immobilization and rehabilitation The typical position of immobilization for one procedure may compromise the other. A mid-position is thus required, with the wrist in 30° of extension, the thumb fully extended at the MP joint, and the finger MP joints flexed to 60°.

After 4 weeks the cast is removed and rehabilitation is initiated. This phase is challenging, as again one seems to pursue irreconcilable goals. Rehabilitation of active finger extensors is directed toward developing active extension of the MP joints, whereas protection of the intrinsic substitution procedure precludes early or vigorous MP joint extension. However, the problem is usually satisfactorily addressed if the therapist prevents full extension of the patient's MP joints, manually during rehab sessions, and with a dorsal MP splint called a 'lumbrical bar' between sessions. As stated earlier, the patient must not thereafter transfer his body weight on his extended MP joints.

Flexor phase

The second stage is performed once the patient has been able to demonstrate active thumb and finger extension. The operative steps include:

- transfer of ECRL to finger flexors (FDP)
- transfer of PT to FPL
- thumb IP stabilization.

Thumb IP stabilization The 'New Zealand split transfer' is performed first before adjusting the tension of the other transfers, so the tension can be set according to the position of the IP joint.

Transfer of ECRL to FDP This is performed as described in group 3.

Transfer of pronator teres to FPL The pronator teres is detached from the radius together with a strip of periosteum so as to extend the length of its broad but short tendon. It is then mobilized proximally from its investing fascia. Next the muscle–tendon junction of the FPL is identified. As much tendinous material as possible is dissected out of the muscle–tendon junction in an attempt to make a direct suture with the PT. However, in a number of cases the tendon of FPL is too short and an interpositional tendon graft is needed. The tension is adjusted so that with the wrist in neutral the thumb rests against the lateral aspect of the index finger.

Postoperative care The wrist and fingers are immobilized with the wrist in slight flexion, and the fingers in 60° of MP and 45° of PIP flexion. At 4 weeks the cast is removed, and physiotherapy is conducted in the same manner as in IC group 3. The use of a manual wheelchair and shifting of the body weight onto the hands are restricted for one more month.

Alternative procedures

Alternative procedures may be discussed with regard to variations in patient presentation and individual functional objectives. For example, one may decide to perform different procedures for each arm (i.e. one for grasping large objects and the other for more precise tasks). Another discussion concerns the management of the thumb's CMC joint. A fused CMC joint, while predictably prepositioning the thumb, does limit the size of objects easily grasped within the first web space. If the CMC joint is left mobile, the postoperative position of the thumb is less predictable, but larger objects can be pushed into the first web space and held. If the joint is completely unstable, we prefer to fuse the CMC joint and carry out the reconstruction as described above, but if it is stable, a different scheme can be proposed, including thumb positioning with tenodesis of the rerouted EPL in the extensor phase, and transfer of the BR for thumb opposition-adduction in the flexor phase.

Tenodesis of the rerouted EPL (Figure 29.21) The EPL is divided at its muscle–tendon junction and withdrawn into the incision made over the thumb MP joint. It is passed subcutaneously around the abductor pollicis longus (APL), and then wrapped around the extensor retinaculum at the third compartment, and sutured to itself. This maneuver gives the thumb an extension-radial abduction vector as the wrist is flexed, which provides additional CMC stability.

BR transfer for thumb flexion and abduction This transfer is essentially House's[38] modification of the transfer described by Royle[40] and refined by Thompson.[41] It is performed after dissection and passage of the ECRL and PT transfers. The BR tendon must be elongated by a tendon graft, and one of the paralyzed superficialis tendons is used for this purpose. The BR is passed into the palmar forearm. It is joined to the cut end of the ring finger superficialis tendon by a weaving technique and non-absorbable sutures. The superficialis tendon is then exposed distally at the base of the ring finger and divided. It is rerouted, through an intermediate palmar incision, towards the tendon of the APL at the thumb MP joint level. One slip of the superficialis is anchored to the tendinous insertion of the abductor pollicis brevis. The second slip is passed dorsally and anchored to the tendon of the EPL (Figure 29.22). Postoperatively the wrist and fingers are splinted with the wrist in slight flexion; the fingers fully flexed at the metacarpophalangeal joints and the thumb in anteposition.

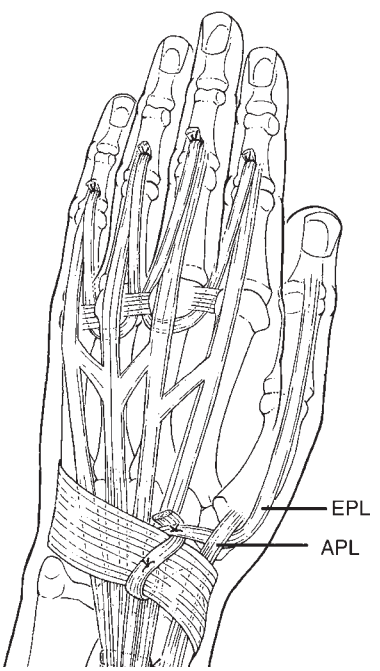


Figure 29.21

Tenodesis of the rerouted extensor pollicis longus (EPL). APL, abductor pollicis longus.

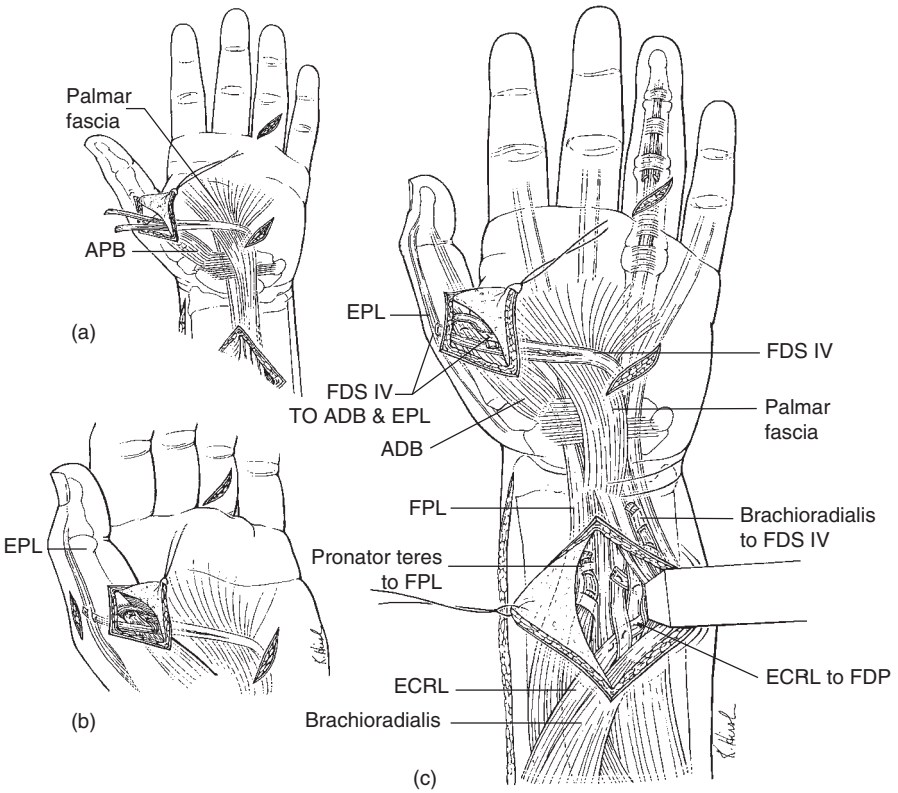


Figure 29.22

Operative steps of the brachioradialis transfer for thumb flexion and abduction.
ADB, abductor pollicis brevis;
APB, abductor pollicis brevis;
EPL, extensor pollicis longus;
FDS, flexor digitorum superficialis;
FPL, flexor pollicis longus;
ECRL, extensor carpi radialis longus.

Common complication and salvage procedures

Suture breakage/rupture of transfer Suture breakage occasionally occurs. This complication can be related to a technical problem such as inadequate suture technique, or inadequate suture material. It can also be due to inappropriate positioning of the involved joint(s) postoperatively, or too early removal of the cast. We believe that it is more often related to an inadvertent contraction of the transferred donor muscle, putting the suture area under such great tension that it gives way. This has been the case mainly with the BR transfer. As the elbow is not immobilized, if the patient and his/her attendants have not been warned or cautious to avoid strong contraction of the elbow flexors, the suture is likely to rupture in the situation where the patient lifts his/her body weight with his/her elbow flexors. It would seem an easy solution to immobilize the elbow within a cast during the first 4 to 6 weeks. However, this would further impair function and nursing of the patient during this period where he is already much more dependent on others, and immobilization of the elbow does not prevent isometric contraction of the muscle, which may also lead to a rupture.

A serious complication has been described by Revol[42] after active tenodesis of FPL with ECRB. In four cases, his patients experienced rupture of ECRB after side-to-side suture to FPL. Secondary repair led to suboptimal results in all cases, especially since the ECRL had been transferred during the same operative phase to the FDP. This resulted in a weak residual wrist extension.

Another cause of failure of the transferred tendon to activate the recipient tendon may be tendon adhesions. This usually occurs at the suture site, and is more likely to happen if the part has been immobilized longer. We have performed secondary tenolysis of the BR to EDC donor–recipient tendon unit in two patients with satisfactory results as reported.[31]

Loosening of transfer A more subtle complication of these procedures is tendon/suture slackening. This is more often related to the patient's lack of education or deleterious habits than to the operative procedure itself. It is more likely to occur with tenodeses than with tendon transfers, and more with flexion procedures than with extension procedures. A typical example of this complication is slackening of the intrinsic substitution procedures ('lasso' and House technique). These are passive procedures, e.g., the tendons are thus subject to slackening if submitted to repeated stretching. If the patient has not been taught or does not comply with the specific ways to protect his transfer, then after a variable period of time the initially flexed posture of the MP joints moves toward full extension, then to hyperextension, and clawing of the fingers is likely to occur. This will lead to a functional impairment of grasp.

It is therefore critical to teach the patient, prior to upper limb surgery, how to protect his future operations. The MP joints should never be pushed by force into passive extension. At rest the hands should not lie flat on the thighs or

chair arms; rather, the fingers should be rolled into flexion. During body transfers the weight of the body should never be borne on the flattened palm of the hand. Rather, the fingers must be rolled into flexion as in forming a fist, and the weight supported either by the dorsal aspect of the proximal phalanges, or, if the wrist is not stable enough, by the dorsal aspect of the flexed middle and distal phalanges and the proximal palm.

Thumb position and CMC fusion Evolution of the thumb CMC joint fusion to a non-union has been described. It has been reported to be as frequent as 24%,[29] and does not seem to be related to the type of fixation which has been used. However, these fibrous unions may remain painless and stable with time.

Thumb positioning remains one of the most challenging technical parts of these procedures. The thumb is a complicated articular complex, made of three joints, one of which has a wide range of motion, activated by four extrinsic and four intrinsic tendon/muscles. Although in the tetraplegic patients one tries to restore only a simple type of motion (lateral pinch), all joints must be either activated or stabilized, and sometimes both, in order to make this complex chain into a simple one.

IP joint hyperflexion
This occurred regularly in our early experience after FPL activation, but is now effectively prevented by either IP fusion or preferably by stabilization with the so-called 'New-Zealand split-FPL transfer'.

CMC joint hyperlaxity
Hyperlaxity requires a fusion. If it is not performed, it is very difficult to predict and control the final position of the thumb pulp after activation has been performed. The EPL extends and adducts the thumb dorsally and tends to create an inefficient lateral pinch between the thumb and the proximal phalanx of the index finger.

MP joint stabilization
This joint may be or become unstable both in excessive flexion or hyperextension. Occasionally, thumb MP hyperextension is functionally beneficial even if esthetically unbecoming, as it allows a wide opening, making it easier to grasp larger objects. An excessively flexed MP joint may cause the thumb tip to pass under the index and requires either fusion of the joint or passive stabilization by tenodesis of the extensors just proximal to the MP joint.

Surgery for the stronger patients (IC groups 6 and 7)

Group 6

IC group 6 represents a minority of tetraplegic patients (less than 5% of the operated series). The International Classification defines those patients as possessing a strong extensor digitorum communis muscle (graded MRC 4 and above). Functional assessment of their hands, however, reveals that they are not significantly better performers than those in IC 4 and 5, and sometimes less, because active digital extensor tendons without active antagonist finger flexors are more a handicap than a benefit. This group of patients usually benefits greatly from surgical rehabilitation of their upper limbs, directly through tremendous improvement in their ability to grasp and pinch, and indirectly because they have a greater potential to make use of these improvements.

Functional goals for IC 6 patients

- The primary goal, in IC group 6 patients, is to restore a strong pinch and grasp, as well as thumb extension. Many muscles are available for transfer and one will usually choose strong donor muscles for fingers and thumb flexion (typically ECRL and BR, or PT), and a smaller muscle for thumb extension. The remaining potential donors are used for restoring some intrinsic function, not only in the fingers, but also in the thumb. Substitution of finger intrinsics may be performed either with the 'lasso' procedure, or with House's procedure, as described earlier. Good extension of the PIP joints occurs more often in this group of patients, because the active digital extensors have kept the PIP joints from flexing, and thus the extensor mechanism from slackening. Therefore a 'lasso' is performed in most instances. In the thumb, some form of opposition can be restored to provide a stronger pinch; alternatively, one can restore some antepulsion, commonly referred to as palmar abduction, in order to achieve a pulp to pulp (subterminal) pinch. This pinch is very useful, as a complement to the lateral pinch, for precision pinch.
- Thumb stabilization in this group of patients can usually be achieved through tendon transfers and tenodesis, rather than arthrodesis. At the CMC level, an arthrodesis would be considered only in cases where the trapeziometacarpal joint is very unstable, but if it is performed, it will not be possible to restore any form of opposition, adduction, or antepulsion of the thumb. Rather, if it seems mandatory to stabilize one joint, in cases when the thumb is very lax, one will prefer to fuse the MP joint. Any tendency to hyperextension of the MP joint must be treated, otherwise it is likely to increase when the extrinsic muscles of the thumb are activated, resulting in a 'thumb swan-neck' deformity with a poor, unstable pinch. Therefore some form of volar stabilization is necessary, by means of muscle transfer, capsulodesis, or sesamoido-metacarpal arthrodesis. The necessity to stabilize the distal joint of the thumb has already been stressed. The best procedure to achieve stability is the 'New Zealand split-FPL' transfer described previously.
- In IC group 6 patients, a two-stage procedure is still usually necessary, as both the EPL and the FPL need to be motored. These two procedures are not readily compatible, as they require opposite positions of

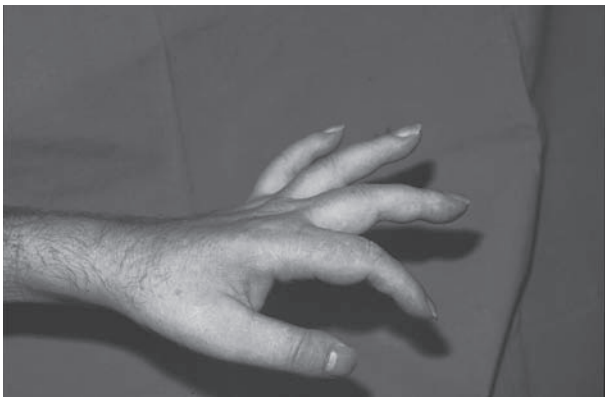


Figure 29.23

This patient demonstrates strong extension of the ulnar fingers, and weak extension of the radial fingers.

immobilization, and their physical therapy is contradictory.

- A specific group of patients has strong extensors to the ulnar fingers (IV and V, or III, IV, and V), and weaker extensors in the radial fingers (I–II, or I–II–III) (Figure 29.23). These patients, who might be classified as intermediate IC group 5–6, are included in group 6 as their treatment scheme is similar.

Surgery is performed in two stages. The first stage is referred to as the extensor phase, and the second stage as the flexor stage.

Extensor phase

Surgery is performed through a lateral antebrachial incision. Alternatively, one may use a dorsal incision during the first phase, and a volar one during the second phase.

Thumb (and radial finger) extension If the radial digital extensors are weak, a side-to-side suture is performed between all tendons of the extensor digitorum communis (EDC). The tendons are exposed above the dorsal retinaculum, and a heavy non-absorbable transverse mattress suture is passed through each of the tendons. The tendon of EPL may or may not be included in the suture. If it is, tension should be slightly less on the thumb than on the finger extensors.

If only the thumb extensor (EPL) needs augmentation, two methods can be used. One is as described above, with a side-to-side suture to the tendons of the EDC. The other method is to transfer the extensor digiti minimi (EDM) to the EPL. Prior to harvesting the EDM, the tendons are checked for anatomic variations, knowing that in a limited number of cases there is no tendon of the EDC for the little finger. In these cases, EDM ensures MP joint extension, and cannot be utilized, unless it consists of several tendon slips.[43,44] The most ulnar slip is detached at the level of the MP joint. It is retrieved proximal to the dorsal retinaculum through a small incision, then rerouted toward the EPL. This can be done through the 3rd compartment, or through the 2nd compartment, depending on the spontaneous position of the thumb.

The tendon of the EDM is then woven through the EPL distal to the dorsal retinaculum. The tendon juncture must be made distal enough so that it does not impinge on the distal edge of the retinaculum in full extension. Tension of the suture is set by moving the wrist passively: the thumb should start to extend when the wrist starts to flex from the neutral position.

Thumb stabilization APL tenodesis: the various transfers which are performed during the first or the second surgical stages stabilize the CMC in flexion, extension, and opposition. However, there remains the need for stabilization in radial abduction, in order to avoid a progressive contracture of the first web space. This is best performed with a passive tenodesis of the APL, which, besides contributing to stability, will maintain the thumb in a slightly abducted posture.

The tendon of the APL is severed proximally, at the muscle–tendon junction. The proximal end is looped around the distal edge of the extensor retinaculum, and the tendon is sutured to itself around the retinaculum. Tension is adjusted so that the thumb assumes a mildly radially abducted position. As this tenodesis lies along the flexion/extension axis of the wrist, it is not influenced by the position of the wrist.

Intrinsic substitution This step can be performed either during the extensor or the flexor phase. We prefer to include it in the first stage because there are usually more procedures to be performed during the flexor phase and tourniquet time may become a limiting factor.

As stated earlier, a 'lasso' procedure is indicated more often than the intrinsic substitution described by House. If extra donor muscles such as the extensor carpi ulnaris (ECU), or the flexor carpi ulnaris (FCU), or even the BR are available, they may be used to produce an 'active' lasso: after each flexor superficialis has been severed distally and looped around the A1 pulley, the ECU is dissected, then passed through the interosseous membrane to the volar aspect of the forearm where it is sutured to the four tendons of the paralyzed FDS. The tension of the transfer is set so that the resting position of the finger MP joints, with the wrist in neutral, should average 30° of flexion after the suture has been completed.

Flexor phase

Besides restoration of finger and thumb flexion, which may be performed as described above (ECRL to FDP, and BR to FPL), some intrinsic function of the thumb should be restored. Among the many procedures available, we have used two different techniques in the tetraplegic patients, which restore somewhat different functions.

Restoring palmar abduction of the thumb This technique is best performed in cases when the EDM has not been utilized during the first phase. The tendon is harvested

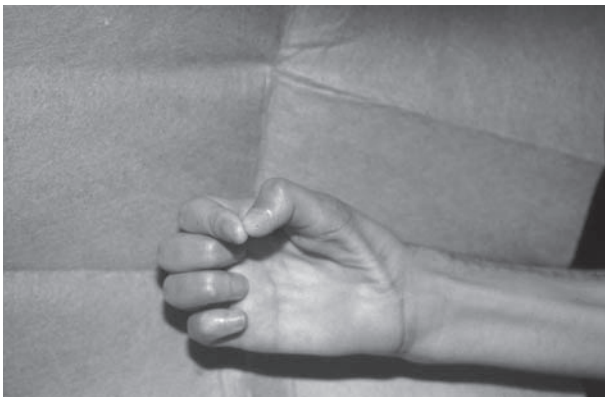


Figure 29.24

Restoration of a subterminal pinch by means of an extensor digiti minimi transfer.

as described above, taking care to divide it as far distally as possible. It is retrieved proximal to the dorsal retinaculum, passed around the ulnar border of the forearm, then transferred subcutaneously to the thumb, with a direction parallel to the abductor pollicis brevis (APB) (Figure 29.24).

If the thumb MP joint does not need stabilization, the tendon is woven into the distal tendon of the APB. If stabilization is needed, the distal part of the EDM is divided into two equal strips, and inserted as described by Brand:[46] the proximal slip is passed dorsally around the metacarpal neck and sutured to the terminal tendon of the adductor muscle, and the second slip is woven through APB, then passed dorsally and sutured to the EPL, distal to the MP joint, in slight flexion of the MP joint.

Alternatively, if the EDM has been used during the first stage, one may transfer the palmaris longus, prolonged by a strip of the superficial palmar fascia, as advocated by Camitz.[45]

Restoring thumb opposition This technique, often referred to as the 'triple transfer', uses the ECRL for finger flexion, PT for thumb flexion, and the BR for thumb opposition, as advocated by House (see Figure 29.22 for brachioradialis transfer for thumb flexion and abduction).

This technique provides an increase in pinch strength, but this pinch remains essentially a lateral one. On the other hand, the BR, when attached (via the superficialis) into the tendon of the EPL as part of the thumb intrinsic substitution procedure, has proven strong enough to stabilize the thumb's IP joint against excessive flexion, even when the FPL is strongly motored by PT transfer.

By whatever means, intrinsic extension of the IP joint must be substituted for, in order to avoid a Froment's sign, either by the transfer described above or by the 'New Zealand split FPL' procedure.

It may be useful, in a bilateral group 6 patient, to perform a different technique on each side, so as to provide the patient with two different types of pinch, one for precision, and one for strength.

Group 7

Even patients who are classified as IC group 7, 8, or 9 (ASIA level C8) may experience increased upper limb function from surgical procedures for their upper limbs. Few will benefit from or accept orthoses because, compared to the majority of tetraplegic patients, they are already functioning at a high level. Most possess normal shoulder, elbow, and wrist control and, by definition, they have strong thumb and finger extension. Many IC group 7 patients will also have some useful finger flexion. The IC group 8 patients will have strong finger flexion and some active but weak thumb flexion. The hands of IC group 9 patients are similar to those with a distal laceration of the median and ulnar nerves, referred to as the 'intrinsic minus hand.'

Because they are functioning at relatively high levels, they are more likely to be involved in full-time employment or school. It may be difficult for these patients to set aside enough time to undergo surgery and the follow-up therapy. They are typically using a manual wheelchair and do not like the idea of having to use a powered chair, even for a short period of time. They are far more independent and see even a short period of time in a more dependent state, i.e., having one arm in a cast, as a huge liability. Finally, because they are already functioning at a relatively high level, compared to the IC groups 3, 4, 5, and 6 patients just discussed, decisions regarding surgery must be carefully considered.

There are many potential combinations of active muscle-tendon transfers that might be considered for thumb and digital intrinsic substitution for the IC group 8 or 9 patient. Technical details regarding these procedures can be found in most general hand surgery texts and will not be discussed here.

Reconstructive options for IC group 7 patients

The IC Group 7 patient, by definition, possesses at least MRC grade 4 finger and thumb extension. In addition, a number of other muscles such as the extensor carpi ulnaris and the extensor indicis may be graded 4 or 5, varying from patient to patient. The variety of potential reconstructive surgical options differs accordingly.

Surgical rehabilitation of the hand can usually be performed in a single operative stage. It should include provisions for:

- finger flexion
- thumb flexion/adduction and abduction
- digital intrinsic balance.

Finger and thumb flexion These patients possess a number of potentially transferable synergistic muscles. The muscles most typically transferred to achieve finger and thumb flexion include the ECRL, the BR, and the PT. Usually, one donor is transferred to the FDP, and one to the FPL, as described previously. Rarely, function of both superficial and deep flexors can be restored. For example, in some IC group 7 patients who have MRC grade 3 deep flexor strength, one may choose to restore power to the superficial flexors.

Thumb intrinsic reconstruction The IC group 7 patient will benefit from reconstruction of thumb opposition, either by standard opponensplasty procedures or various modifications. There are several potential sources such as extensor indicis, EDM, or flexor carpi ulnaris to restore this function, and more than one effective method of routing the transfer. Surgical choices must be individualized to the specific presentation of the thumb and available accessory muscles.

If the extensor indicis proprius (EIP) is functioning at an MRC 4+ level, we prefer to transfer this muscle. The tendon is rerouted around the ulnar side of the distal forearm and across the base of the palm, then inserted at the thumb MP joint as described by Brand[46] (see group 6, flexor phase). An alternative is to stabilize the IP joint of the thumb against hyperflexion (Froment's sign) by performing the 'New Zealand split FPL' transfer and perform only a single insertion into the tendon of the APB.

Other potential sources of motor power for restoring thumb flexion/abduction or opposition include the ECU, the FCU, the palmaris longus and the EDM. The first two of these will require an interpositional tendon graft.

Digital intrinsic reconstruction The strong digital extension enjoyed by these patients, when combined with transfers for restoring strong finger flexion, creates a significant risk of developing a 'claw' finger deformity as the patient attempts to open the hand. Therefore, these patients are candidates for one or another intrinsic substitution procedure.

Many surgical procedures have been described as effective in restoring intrinsic function. These include static procedures including various tenodesis or capsulodesis procedures and many active muscle-tendon transfer procedures. The technique and indications of the tenodesis procedure described by House et al[38] and the 'lasso' procedure described by Zancolli[37] have been discussed earlier. It has been our experience that the majority of IC group 7 patients retain good IP extension when metacarpophalangeal joint hyperextension is prevented and the 'lasso' procedure is more often performed. Furthermore, in contrast to IC group 4 and 5 patients, where only the index and middle finger may need intrinsic substitution, the IC group 7 patients benefit from intrinsic substitution to all four digits.

Some IC group 7 patients may have sufficient numbers of transferable motor resources so that one could perform an active intrinsic substitution procedure. For example, the PT could be used to power the thumb by transfer to the FPL, and the BR could be passed into the volar forearm and its tendon woven into one or several of the paralyzed FDS tendons in the forearm.

Results

Patients from IC groups 6 and 7 usually benefit greatly from surgery. Their hands evolve from a poor and weak pinch, and no grasp, to a strong lateral pinch (usually with a precision subterminal pinch on one side) and a strong and effective grasp.

These patients usually make the best of these transfers, as they also possess strong wrist stabilizers, which potentiate the strength of the transfers, and a number of proximal muscles around the shoulder and elbow, which allow them to reach out into a much greater functional space. Following restoration of grasp and pinch in IC groups 6 and 7, these patients begin functioning more as a high-level paraplegic than a tetraplegic. Our patients refer to themselves as 'para-quads'.

Conclusions and future perspectives

Not every tetraplegic individual *needs* to undergo surgery, but essentially all who retain control of the shoulder and elbow could *benefit* from surgery to restore or improve function of the wrist and/or hand. At one end of the spectrum is the IC group 1 tetraplegic patient who lacks MRC grade 3 wrist extension, but who has a strong BR. He or she can, by surgical transfer of BR to the wrist extensor, achieve better than antigravity wrist extension. In itself, little that is measurable by dynamometer or manual muscle testing has been gained. However, this small gain in wrist extension strength may make it possible for this patient to use a wrist-driven, flexor-hinged orthosis and achieve useful grasp and release, or to secondarily undergo surgical tenodesis of the tendon of the FPL to the radius (Moberg key grip procedure) and achieve functionally useful key pinch without the need for any external orthosis. This is an example of the old adage 'When you have nothing, a little (gained) becomes a lot.'

At the opposite end of the spectrum is the IC group 6 or 7 patient. This patient has many functionally strong forearm muscles under excellent central control, including flexion and extension of the wrist. Because they are so strong, these patients are functioning, in comparison to a group 1 or 2 patient, at a relatively high level. When compared to normal, however, this patient's weak digital grasp and pinch pales in contrast. Upper limb surgery for this patient may move a hand that, in comparison to the hand of the IC group 2 patient, is functioning 'well' to a hand with truly dramatically increased function.

More than two decades ago, one of the pioneers of this discipline, Erik Moberg, often stressed that, 'aside from the brain, the tetraplegic patient's upper limbs represent their most important functional resource.' Today, greater numbers of tetraplegic patients are demanding the possibility of functional upper limb surgery and greater numbers of spinal cord rehabilitation centers are offering this possibility. The many outcome studies performed and published support the contention that upper limb surgical rehabilitation for the tetraplegic patient is now a discipline that has achieved relative consensus regarding which tetraplegic

individuals are the best candidates for surgery in both adult and pediatric age patients, one possessing an armamentarium (albeit somewhat limited) of relatively predictable surgical procedures, procedures that can be readily taught to residents and fellows and, provided they understand the special environment in which the tetraplegic patient exists, one can expect predictable outcomes without the necessity that residents and fellows become 'experts' in the care of tetraplegic patients. Nonetheless, there still exist, within the minds of many physiatrists and spinal cord medicine specialists, doubts regarding the role of upper limb surgery for their tetraplegic patients. Overcoming remaining doubts will require further efforts at educating non-surgical colleagues of the value of this surgery.

As mentioned earlier, few of our patients perform many new activities. Typically, a good or excellent result means that the patient performs many of the same functions but with much greater efficiency. The rewards for surgeons, rehabilitation medicine specialists, and therapists are best expressed by one of our patients who replied to a question requesting his feelings on his outcome, 'it's not as much as I hoped for, but it's much more than I ever had.'

The last 10 years have witnessed a surge of interest in spinal cord regeneration research into the molecular transformations associated with injury, molecular manipulations to reduce the extent of injury, and how to alter the molecular events that impair regeneration and ultimately promote spinal cord regeneration. Many unknown factors are at play, not the least of which is the outcome of the current debate, across many countries, of the ethical uses of genetic and stem cell research.

Today, many tetraplegic patients are strongly influenced by the 'promise' that a cure is just around the corner and decline surgical upper limb rehabilitation. The near-term effect of this sense that a cure is near has altered the practice patterns for some surgeons.

The role of technology in improving the function of the tetraplegic patient can only expand, the recent experience with the Freehand® upper limb functional electrical stimulation system notwithstanding. Similarly, it has become clear to surgeons involved in this field that there is an enormous need to quantify the functional gains achieved by tetraplegic patients undergoing upper limb surgery. The result is greater effort on the part of surgeons and therapists to design and implement a tetraplegic-specific assessment methodology that can document gains made.

Finally, if results of the relatively few operative procedures available for this population are to be optimized, there is the need to involve basic scientists and bioengineers in collaborative research. Today, this collaboration is producing exciting research into technologies that allow surgeons to tension tendon transfers in an individualized, need-specific manner, that permit earlier postoperative remobilization, and that drive more productive postsurgical rehabilitation protocols. Ultimately, the future (short of the 'cure') includes a treatment algorithm (clinical pathway) that includes the premise that functional upper limb surgery is as integral a part of rehabilitation as training in activities of daily living, bladder and bowel stabilization, transfers, and locomotion. In the future, an assessment of the status of the tetraplegic patient's upper limb will become an integral part of every interval examination. The future involves a paradigm shift, if not a 'sea change' in the role of upper limb functional surgery in the rehabilitation of the tetraplegic patient, and the recognition (on the part of other caregivers) of the value of restoring better function for the upper limbs of the tetraplegic patient.

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30 The spastic upper limb and its surgical rehabilitation

Caroline Leclercq

Spasticity is characterized by muscle hypertony, linked to a central neurologic impairment involving the pyramidal tract. It may occur in varied circumstances:

- In children, *cerebral palsy* is caused by brain anoxia, which is usually secondary to fetal or perinatal problems, and less frequently to brain damage during childhood.
- In adults, spasticity is usually related to *hemiplegia*, whether vascular (stroke) or traumatic (head injury).
- *Tetraplegia* is often associated with spasticity of the lower limbs. Involvement of the upper limbs is rare, and it occurs mostly in incomplete cases.

Spasticity is rarely an isolated feature. The clinical picture generally includes other neurologic and orthopedic impairments which need to be carefully assessed together with spasticity.

Cerebral palsy

Cerebral palsy is a general term that includes all the sequelae of infantile encephalopathies occurring during pregnancy, delivery, or infancy. Since the time of Little,[1] in the nineteenth century, it has been thought to be mostly linked to perinatal asphyxia, but current studies indicate that this factor is responsible for only 5 to 10% of cases of cerebral palsy. Other causes may be related to the fetus (gestational age at birth, birth weight, growth restriction), or to the mother (neurologic disorders, infertility treatment, antepartum infection, thyroid disease).[2] Postnatally acquired cerebral palsy accounts for 15% of cases, and is mostly due to meningoencephalitis, head injury, and cerebrovascular accidents.

This section will discuss infantile cerebral hemiplegia, which is the main type of cerebral palsy possibly amenable to surgical functional improvement in the upper limb. It is characterized by unilateral cortical and subcortical involvement, particularly in the pyramidal tract, which causes a motor deficit and various degrees of spasticity in the contralateral limbs. It can be associated with epilepsy, mental retardation, and speech, vision, or hearing deficits. It manifests progressively during growth, but, once established, follows a non-progressive course, which makes it amenable to surgical treatment.

Clinical examination

The clinical picture may vary greatly from one individual patient to the other, depending on the nature and location of the initial brain insult. Clinical examination is a critical part of the assessment. Its goal is fourfold:

- evaluate spasticity
- evaluate motor and sensory impairment in the upper limb
- evaluate existing function, and functional needs of the upper limb
- perform a complete general examination in order to seek associated neurologic disorders and potential contraindications to surgery.

This examination is lengthy, and requires detailed knowledge in neurology, pediatrics, and physiatry. It is best performed as a team procedure including all the specialists involved in the child's care (physiatrist, physical therapist, occupational therapist, pediatrician, and surgeon). This should ideally be done in a warm, quiet, and friendly environment, ensuring that the child is comfortable and confident. If painful procedures (i.e. injections) are necessary, they should be performed last. This is of paramount importance since the child's co-operation is essential for sensory and motor evaluation, and because spasticity may increase considerably if the child is frightened or recalcitrant.

Generally speaking, the clinical picture may vary greatly with the child's emotional state and fatigue level. Some of these children also have limited concentration capacity, and cannot co-operate throughout the entire examination. Therefore it is not wise to decide on surgery after a single session, and assessment should be repeated before making a decision.

Video-recording of each clinical session is most helpful, for initial evaluation, for the decision-making, and for evaluation of surgical outcome.

Resting posture of the upper limb

Inspecting the limb at rest prior to examination provides information on the amount of spasticity. If predominant, spasticity leads to a typical resting posture in shoulder adduction and internal rotation, elbow flexion, forearm pronation and wrist flexion, and ulnar deviation (Figure 30.1). The fingers may assume varied positions. Most frequently they are curled into a clenched fist, as a result of spasticity of the finger flexor muscles. Less typically, they assume a swan-neck deformity, resulting either from excessive traction on the extensor tendons due to excessive wrist flexion (extrinsic swan neck) or from spasticity of the interossei muscles (intrinsic swan neck), or a combination of both (Figure 30.2). The fingers may also assume an 'intrinsic plus' deformity with flexion of the metaphalangeal (MP) joints and hyperextension of the interphalangeal (IP) joints, due to spasticity or contracture of the intrinsic muscles (Figure 30.3), or the opposite 'intrinsic minus', or claw-type deformity, with the MP joints hyperextended and the proximal interphalangeal (PIP) joints flexed, due to a combination of excessive traction on the extensor tendons and paralysis of the intrinsic muscles. A boutonnière deformity is less common.

The thumb can assume either an adducted posture or an adducted and flexed posture. The adducted thumb is tightly clenched to the lateral aspect of the index finger, with the MP and IP joint extended (Figure 30.4). The 'flexus-adductus' thumb, often referred to as 'thumb-in-palm', is embedded in the palm with full opposition and full flexion of both MP and IP joints. Often the clenched fingers are curled around the thumb (Figure 30.5). Any factor that aggravates spasticity will increase these deformities.

Figure 30.1
Usual posture of the spastic upper limb.

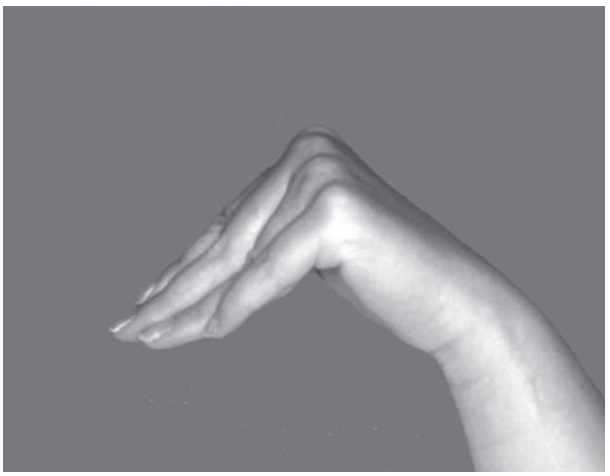

Figure 30.3
'Intrinsic plus' hand.

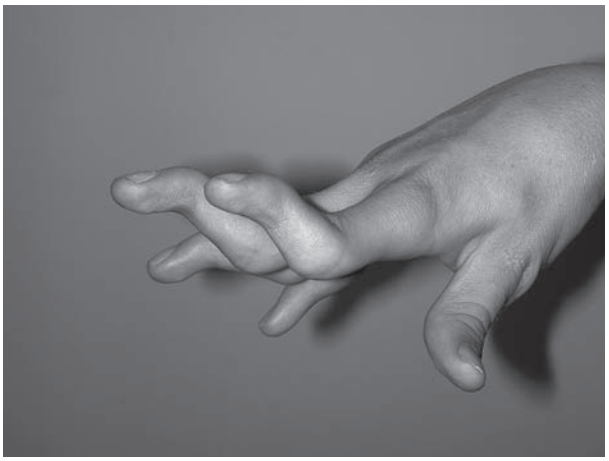

Figure 30.2
Multiple swan-neck deformities of the fingers.

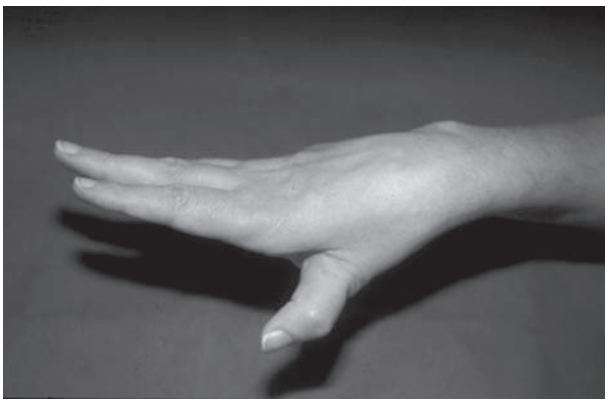

Figure 30.4
Thumb adducted.

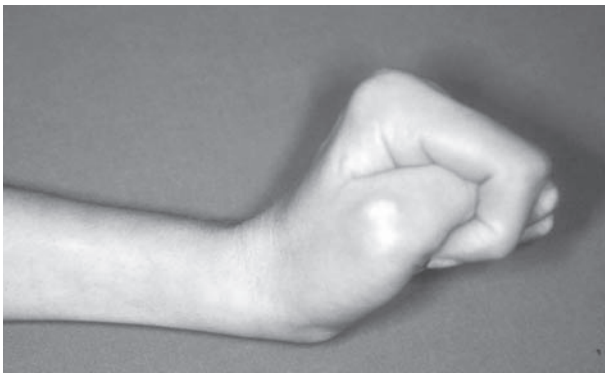

Figure 30.5
Thumb 'flexus-adductus'.

Evaluation of spasticity

Spasticity is a muscle hypertony, characterized by five classic clinical features:

1. It is selective, and is predominant on flexor, adductor, and pronator muscles, responsible for the characteristic 'flexion-pronation' deformity of the upper limb described above.
2. It is elastic. Attempts at reducing the deformity meet with a resistance, which increases with the strength applied. Unlike 'plastic' contractures, the limb returns to its initial position as soon as the imposed movement is stopped. However, if the opposing force is maintained long enough, the limb usually yields, sometimes abruptly.
3. It is present at rest, and exaggerated with voluntary movement, emotion, fatigue, and pain.
4. Osteotendinous reflexes are exaggerated, brisk, diffuse, and polykinetic. Clonus is infrequent in the upper limb.
5. There may be associated synkineses, described as 'the phenomenon whereby paralyzed muscles incapable of a certain voluntary movement, execute this movement in a voluntary fashion by accompanying intact muscles' (Lhermitte sign). For example, active shoulder abduction may be accompanied by synkineses of the finger extensors and abductors (Souques synkinesis).

Motor assessment

Motor examination of the upper limb is not easy in children, especially in those less than 5 years of age. The child should be provided with toys of different forms and colors, and be observed at play.[3] Video-recording is extremely helpful at this stage, as it avoids lengthy repetitions of tasks. Each muscle or muscle group is evaluated for (1) voluntary control, (2) fibrous contracture, and (3) joint motion.

Voluntary motor control The palsy (or 'pseudo palsy') usually predominates in the extensor and supinator muscle groups, and in the distal part of the upper limb (wrist and finger extensors, abductor pollicis longus, extensor pollicis longus and brevis, and supinator muscle). Motor examination of these muscles may be difficult when the antagonist flexors and pronators are severely spastic. Proximal muscles (shoulder external rotators and elbow flexors) are involved to a lesser degree. More than a true paralysis, this is typically a deficit in voluntary control linked with the pyramidal tract involvement. This lack of control varies with limb position. For instance, voluntary movement of the thenar muscles is often facilitated by elbow extension. In some cases these muscles may be present, but made ineffective by the spastic antagonists or by elongation caused by a severe deformity. The flexor, adductor, and pronator muscles, mostly spastic, usually retain some voluntary control. Their examination is made difficult when synkineses or co-contractures are present. In difficult cases, botulinum toxin and electromyographic (EMG) studies may be helpful in evaluating both the pseudo-paralytic and the spastic muscles (see below).

Fibrous contracture Fibrous contracture may involve spastic muscles. Unlike spasticity, fibrous contracture is permanent and cannot be overcome, but shortening the involved articular segment can alleviate it. For example, posturing the wrist in flexion relieves contracture of the finger flexors. Intrinsic contracture of the fingers is assessed by the Finochietto test. Clinical distinction between contracture and spasticity may be difficult to establish. In such cases, nerve blocks with lidocaine are useful.[4] The anesthetic may be injected either in the nerve trunk or in the motor point of the involved muscle(s). Spasticity yields completely, whereas contracture persists.[5] Botulinum toxin yields the same result, with a much longer lasting effect.

Joint range of motion Passive range of motion of the involved joints may be difficult to assess, not so much because of spasticity but because of muscle fibrous contractures. It can only be tested with the involved muscles fully relaxed. Motor blocks are not very helpful here, as they do not alleviate muscle contracture. Sometimes it is so difficult that it is not until the preoperative examination under anesthesia that the actual range of passive motion can be evaluated. Some joints of the fingers and thumb may have an increase in passive extension, resulting in joint instability. This occurs mainly at the thumb MP joint, and at the finger MP and PIP joints.

General motor assessment Finally a general motor assessment is performed, in order to evaluate the global motor control of the upper limb. Standard tests are performed, such as the 'head-to-knee' test, where the patient is asked to place his hand on his head and then to move it to the contralateral knee. The speed and precision of the movement are recorded. These non-specific tests involve many of the elements susceptible to perturbation (hypertonia and muscle contracture, ataxia, apraxia, and extrapyramidal lesions). Primitive reflexes are also sought. They are due to an abnormal sensory motor development, and may greatly impair the functional capacity of the limb. The classic 'asymmetric neck reflex' is triggered when the head

is turned actively or passively to one side, which produces abduction of the shoulder, and extension of the elbow, wrist, and fingers of the ipsilateral upper limb, while the contralateral limb flexes in all joints.

Classification

Once the motor examination has been completed, an attempt at classification can be made, using, for instance, Zancolli's classification,[6] where:

- Type I includes the spastic 'intrinsic plus' hands, in which spasticity of the interossei and lumbrical muscles causes flexion of the MP joints and extension of the IP joints, sometimes associated with a swan-neck deformity. In this type a wrist flexion deformity is rare.
- Type II includes the spastic 'flexion-pronation' hands with (hyper) flexion of the wrist and pronation of the forearm. Three groups are individualized within type II depending on the degree of active finger extension. In the first group, with the wrist in neutral or near neutral, there is full active extension of the fingers. In the second group there is nearly complete active extension of the fingers, but with some degree of wrist flexion. This group is further subdivided based on the presence (subgroup A) or absence (subgroup B) of an active wrist extension. In the third group there is no active finger extension, even with maximum wrist flexion.

Goldner[3] has produced another classification:

- Group I, in which the wrist and MP joint can be extended at least to neutral. There is active grasp and release. The main deficiencies are delayed speed, slow co-ordination, and minimal dexterity.
- Group II, in which there is weakness of wrist and finger extension, with mild contracture of the wrist, fingers, and thumb flexors. The thumb remains in the palm during hand extension. The hand is used only as an assist and a stabilizer.
- Group III, in which the wrist and finger flexors are severely contracted. The primary goal of surgery is cosmetic improvement.
- Group IV, in which the hands are both spastic and athetoid.

Aside from Tonkin's classification of thumb deformities,[7] which is purely descriptive, we do not find any of the available classifications particularly helpful, since there is such a wide range of clinical pictures, depending on the amount and extent of the initial brain damage. There are no two identical cases and many of them do not fit accurately into any of the described categories.

Sensory examination

Sensory examination is practically impossible in the small child; it becomes feasible at age 4 to 5 (and two-point discrimination at age 6 to 7). It requires, besides the child's co-operation, a certain level of intellectual capacity and language ability.[8] In cerebral palsy the basic sensory functions (light touch, pain, temperature) are essentially intact, while complex sensations (epicritic sensibility, proprioception, gnosis) are more readily affected:

- Light touch is explored with a smooth point or a finger, pain with a needle, and temperature with tubes of hot (40°C) and cold (melting ice) water.
- Epicritic sensibility is explored with discriminatory tests such as two-point discrimination.
- Proprioception is tested by vibration (tuning fork) and by the sense of position of the limb: the patient is blindfolded, the unaffected limb is placed in one position, and he is asked to reproduce it with the affected limb. Proprioception is usually more disturbed in the distal part of the limb than in the proximal part.
- Gnoses are the most affected. Placing an object in the child's hand and asking him to identify it tests stereognosis. Graphesthesia is tested by drawing figures or forms in the patient's palm.

On the whole, sensation is considered satisfactory when the child identifies at least three out of five objects, can recognize large figures drawn in the palm, and has a two-point discrimination test no greater than 5–10 mm (according to the child's age).[9] Pain may be present, but is difficult to evaluate, especially in children, who may not report it, and may not know how to describe it. It may be linked to severe contractures, or to a deformed joint, or, occasionally at the wrist to a Kienböck disease secondary to a severe flexion deformity.[10]

Functional assessment

This part of the evaluation tests the actual functional capacities of the upper limb. It is done with standard reproducible tests, using a pre-established scenario, with simple objects. It should be video-recorded and this usually proves to be a great assessment tool, as it can be visualized as many times as necessary without necessitating the child's presence and co-operation. The same tests, and video-recording, will be repeated after surgery, and will then serve as a comparison for evaluation of the results of surgery.

The pick-up and release test evaluates not only hand prehension, but also the contribution of the whole limb to that function. Several objects of different sizes and volumes are displayed on a table in front of the child; the child is asked to pick each one up, and to displace it to a different spot. The pollicidigital pinch is often limited to a lateral 'key-grip' type of pinch because of the lack of fine voluntary control and because of the adducted thumb. Grasp is generally preserved, although not always functional because of contractures of the finger flexors and weakness of the finger extensors. Release often proves difficult because of weakness of finger extensors.

In the 'box and block test', the patient is asked to move as many wooden blocks as possible from one compartment to another in 1 minute.

Computerized systems have been used more recently in an attempt to quantify hand grasp. They allow analysis of the movement, and captors integrated in gloves measure the forces generated by grasp.[11,12]

Bimanual activities (such as carrying a container with two handles, cutting meat, holding one object into which another one should be placed, or holding a ruler while drawing a line with the unaffected hand) give accurate information on the child's actual functional ability.

Questionnaires The child and family are also asked to describe precisely how the hand is used in activities of daily living such as dressing, self-care, and eating. This can be done in the form of a questionnaire adapted to the child's age, which can be completed during the session, or at home (self-questionnaire). In a number of cases, the child neglects the upper extremity in spite of a true functional capacity. In these cases, the child may persist in ignoring it whether functional ability can be improved through surgery or not. There are several validated non-specific questionnaires evaluating hand function. But to the best of our knowledge, there is no validated questionnaire adapted to the cerebral palsied child.

Evaluation tools Many standardized tests have been designed to assess the functional value of the upper limb, but few of them are specifically designed for the spastic upper limb.[3,8] We have chosen to use that described by Hoffer,[9] which tests dressing, personal hygiene, feeding, bimanual activities, grasp and release, and the lateral pinch.

General preoperative assessment

The aim of this general examination is to evaluate the real benefice the child could get from surgery, taking into account other neurologic impairments, the patient's age, intelligence, motivation, and environment.

Other neurologic impairments As these children are usually hemiplegic, the lower limb deficit must also be assessed, and it is especially important to evaluate the child's walking ability, and the possible need for walking aids (wheelchair, crutch). If operations are necessary for improvement of the lower limb, they are usually planned before any upper extremity surgery. Associated extrapyramidal signs should also be detected. These include the following:

- Athethosis, which is characterized by unexpected, non-voluntary movements causing a slow oscillation of the limbs. It is reduced at rest, abolished at sleep, and increased by noise, fatigue, and emotions.
- Chorea is made of brisk rapid and anarchic non-voluntary movements, of variable amplitude, which can involve all territories. In the upper limb, these contortions of the forearm, hand, and fingers make activities of daily living impossible.
- Parkinson syndrome is characterized by the classic triad: resting tremor, plastic hypertonia (predominant in the proximal muscles) with the cogwheel sign, and akinesia.

If these extrapyramidal signs are predominant, they preclude surgical treatment, because the child is unable to use his hand because of these non-voluntary movements. But when they are mild, they do not contraindicate surgery.

The capacity of the child to communicate must be evaluated, seeking for visual, hearing, and language problems. Behavioral problems such as irritability, inability to concentrate, and emotional instability may also constitute contraindications to surgical treatment if they predominate.

Intelligence is evaluated through the intelligence quotient (IQ). It is usually stated that rehabilitation surgery is not indicated when IQ is lower than 70, but this is not absolute, and a number of surgical procedures aimed at improving comfort, cosmesis, and personal hygiene are still indicated.[13]

Age Because the neurologic deficit in cerebral palsy is not evolutive, early surgery can be planned. Sometimes it is necessary to operate very early because of an increasing deformity. However, in most cases one can wait until the child is old enough so that motor and sensory capacities can be evaluated accurately and he or she can co-operate through surgery and postoperative rehabilitation.

Sequelae of cerebral palsy in adults should be evaluated very cautiously before surgical planning, because usually the patients have already adapted functionally and socially to the handicap, and surgery may be more disturbing than beneficial.

Motivation and environment Evaluation of motivation should take into account the patient's ability to understand the modalities and benefice of the proposed treatment, and to participate actively in the postoperative regimen. Understanding and motivation from the child's parents' part are also mandatory. Environmental factors during the surgical period are also important, such as a rehabilitation center with an integrated school system and physiotherapists experienced with cerebral palsied children.

According to Tonkin,[14] 'the ideal candidate is a cooperative 6-year-old child, with stable family support, who has a predominantly spastic upper limb deformity, with satisfactory hand sensibility, hemiplegic or monoplegic and without significant neurological deficits.'

X-rays and electromyography

X-rays are part of the preoperative evaluation. They are aimed at assessing any growth disturbance and joint deformity linked to the spasticity; however, satisfactory views may not be easy to achieve when there is a severe deformity such as wrist hyperflexion. Contralateral views in the same position may then be helpful. They may reveal growth disturbances of the distal radius, ulna, and carpus,

and occasionally avascularity of the lunate[10] or a rare dislocation of the radial head.[15]

Electromyographic (EMG) studies may be helpful. Both static and dynamic studies are necessary, which requires co-operation on the child's part.[16] This may be difficult to achieve in children younger than 5. In spastic muscles, EMG studies give information on the voluntary control, the possibility of relaxation, as well as possible co-contractures of the antagonist muscles. In pseudo-paralytic muscles, they may be able to identify voluntary control that is not clinically detectable (spastic and/or retracted antagonists, joint deformity, and/or stiffness). EMG studies may be particularly useful in determining the most appropriate donor muscles for transfer. Most of the potential donors are spastic to some degree. But they can be utilized only if they are capable of relaxation at rest or during the antagonist movement (phasic control). A muscle that fires continuously with no phasic control is not a good candidate for use as a transfer.[16,17]

Stroke

In adult hemiplegia related to a vascular stroke, the clinical picture is quite different. Patients are usually relatively old. Spasticity occurs after a few weeks of a flaccid phase. There are usually few active muscles, and the wrist is often paralytic, both in flexion and in extension. Sensory impairment is often severe in the hand, involving mainly deep sensation and stereognosis. It can persist despite dramatic motor improvement, thus preventing functional recovery. Trophic changes, such as reflex sympathetic dystrophy and vasomotor changes, are frequently associated. Surgery is less often indicated in this group of patients.

Head injury

The initial trauma may have included various portions of the encephalon and cerebral trunk, and the clinical features will vary accordingly. Thus the clinical picture is extremely polymorphous in this group of patients. Motor impairment depends upon the extent of brain damage. It may regress rapidly in some patients, or remain important in others. Other neurologic disorders are usually predominant, e.g., cerebellous syndrome, frontal impairment, and often contraindicate surgical attempts at improving function of the upper limb.

Tetraplegia

Spasticity in tetraplegic patients usually predominates in the lower limbs. According to Zancolli,[18] it affects the upper limbs in 15% of patients only, mainly those with an incomplete tetraplegia. In such patients, it involves mostly the wrist and finger flexors. It can be extremely useful to the patient, who, by triggering the stretch reflex, can initiate a pinch or a grasp.[19] Spasticity, when moderate, does not interfere with surgical rehabilitation of the tetraplegic upper limb. When it is impairing, the spastic muscles may be severed proximally before rehabilitation by a tendon transfer.

Treatment

Surgery has a limited place in the treatment of spasticity of the upper limb. It is only one element of the rehabilitative care, which includes primarily physiotherapy and splinting, occupational therapy, and pharmalogic treatment as needed. Decision-making is discussed with the patient and his (her) family, and all the physicians and caregivers involved in the treatment, typically after several assessment sessions, and video-recording of the patient's functional achievements. As mentioned earlier, surgery seems more effective if performed early during the patient's life, preferably during childhood.[14] Later on, the patient develops new functional schemes with the contralateral limb, and will often have great difficulty in using the operated limb, even though it is analytically improved.

Three types of procedures may be involved, in isolation or associated:

- those which aim at reducing spasticity
- those which aim at reducing muscle and/or joint contracture
- those which aim at reinforcing paralyzed muscles.

Reducing spasticity

Local pharmacologic agents Besides systemic medications such as baclofen and benzodiazepines used in severe and generalized spasticity, some agents are effective locally. Before the era of botulinum toxin, nerve blocks were often used. Lidocaine blocks have a temporary effect, they are mostly used as a diagnostic tool in order to differentiate those difficult cases between spasticity and contracture.[4,5] Some authors have acquired considerable experience with alcohol blocks,[20,21] injected either in the nerve trunk or in the motor point of the involved muscle(s), with a reduction of spasticity which can last up to several months, and even longer if the antagonist muscles are active.[4] Phenol, which is more effective, may be toxic for the surrounding tissues, and must be applied directly within the epineurium through a surgical approach.[22]

Botulinum toxin A

BT is a neurotoxin produced by the bacterium *Clostridium botulinum*. When injected in a muscle, it blocks the release of acetylcholine at the neuromuscular junction, thus yielding a denervation of the involved muscle. This denervation is dose-dependent and reversible. Its effect starts 10 to 15 days after the injection, is maximum at 2 to

3 months, and usually lasts for 4 to 6 months. Used initially for blepharospasm and strabismus, BT has been used routinely in spastic limbs for the last 10 to 15 years,[23–25] with measurable and reproducible effects.[26] However, it is not approved in all countries yet for use in cerebral palsied children.

Although BT yields the same results as the other pharmacologic agents mentioned, it is much easier to use, because it is applied inside the body of the muscle instead of at the motor point, which often proves difficult to locate. Cannulated stimulation needles make localization of the injection even easier. Charts are now available which indicate the effective dose for each individual muscle.[27]

Besides its diagnostic use previously mentioned, BT may be used in isolation to reduce spasticity of a muscle or a group of muscles (usually wrist flexors and/or extrinsic finger flexors). During its temporary effect the antagonist muscles may be exercised. The stronger they become, the longer the effect of BT will be. Muscle contractures and stiffness of the involved joint(s) may also be efficiently exercised during that period, enhanced by passive and/or dynamic splints. Cosmesis and, pain, if present, are also improved.

BT may be repeated as required, possibly yielding a permanent improvement if the antagonist muscles are active and progressively made strong enough to overcome spasticity of the agonists. For instance, a patient with moderate spasticity of the wrist flexors associated with active extensors (even if weak prior to therapy) is a good candidate for BT injections. If spasticity recurs after each injection, a more definitive procedure can subsequently be performed. In such cases, BT also plays an educational role in simulating the effect of surgery. This is usually very much appreciated by the patient, who knows exactly what to expect from the planned procedure.

BT is also indicated in cases of spasticity of the upper limb secondary to a head injury, where spasticity may be temporary. It is particularly useful in spasticity of the elbow flexors, which may be quite severe, resistant to other types of conservative treatment, and yield severe and permanent muscle and joint flexion contractures of the elbow if left untreated. In such cases, BT should be used early, in association with rehabilitation and splinting as needed, and repeated until spasticity decreases.

Finally BT may be used pre- or immediately postoperatively to attenuate the spastic muscles when performing a tendon transfer to the antagonists, in order to facilitate education of the transferred muscle.

Tenotomy Tenotomy of a spastic muscle will obviously permanently relieve spasticity. However, it will also permanently suppress the muscle function. In the upper limb, it is indicated mainly for severe isolated or predominant spasticity of the FCU, with an active FCR.

Technique

Simple section of the spastic FCU has, on occasion, been associated with recurrence of the spasticity by reattachment of the tendon ends through a fibrous scar band. It is advisable to resect at least 1 cm of the distal tendon, then apply a wrist splint in slight extension for 2 weeks.

Hyponeurotization Neurectomy, like tenotomy, suppresses both spasticity and function. It may be indicated in non-functional upper limbs with severe spasticity, in order to facilitate hygiene and nursing care, and improve limb appearance. A specific indication is spasticity of the intrinsic muscles where a neurotomy of the motor branch of the ulnar nerve will improve cosmesis and self-care, and sometimes function. Selective neurectomy involving only part of the nerve fascicles, in an attempt to retain some function, was suggested as early as 1913 by Stoffel.[28] This technique gained some popularity after Brunelli and Brunelli[29] published a series of clinical cases in 1983, coining the term 'hyponeurotization'.

Technique

The motor nerve is approached at its entry point into the muscle, where it usually divides into three or four fine fascicles. Under magnifying loops, between two-thirds and three-quarters of the fascicles are resected, from the nerve division to the motor plate. Brunelli and Brunelli initially advocated resecting 50% of the fascicles, but they experienced some recurrence of the spasticity, attributed to a phenomenon of 'adoption' of the orphan muscle fibers by the remaining motor fascicles. When applied to all flexor muscles of the wrist and fingers, this procedure is lengthy and requires a wide exposure of the anterior forearm compartment. In any event, it is best performed with the help of a nerve stimulator and magnifying loops.

In order to simplify the procedure, some have chosen to perform a 'partial' neurectomy at the level of the nerve trunk, without approaching the motor plate. The motor fascicles are identified with a stimulator before being resected. While faster and more limited in exposure, this simpler technique is quantitatively less accurate, with potential injury to sensory fibers.

Indications

Hyponeurotization is indicated each time a decision is made to reduce spasticity permanently. Resection of the motor fibers must include at least two-thirds of the fascicles, knowing that it will reduce the strength of the muscle in the same proportion as its spasticity. When faced with recurrence due to fiber adoption, reoperation is usually technically more demanding because of the scar tissue surrounding the area.

In our hands this technique has proved more effective on large muscles with a single or mostly predominant motor pedicle. It has not been very satisfactory in small intrinsic muscles such as the thumb adductor and first dorsal interosseous muscles.

Hyponeurotization and partial neurotomy have no effect on muscle or joint contractures; if either are present, these must be addressed by a distinct procedure, preferably during the same operative session.

Neurosurgical procedures Treatment of spasticity by posterior rhizotomy was already mentioned in the late nineteenth century literature. It is now frequently used for spasticity of the lower limbs. However, in the upper limb results have been very partial, with a number of respiratory complications.[30] Bertelli[31] reported a significant reduction of upper limb spasticity with a new technique of brachial plexus dorsal rhizotomy in 61 children or adolescents with spastic hemiplegia.

Muscle contracture

Several types of procedures can be employed to overcome muscle contracture.

Tenotomy As mentioned above, this technique is useful in severe contractures occurring in non-functional upper limbs, when there is no hope of functional recovery, with the exception of isolated and predominant contracture of the FCU with a functioning FCR.

Flexor pronator release This classic procedure was described by Page[32] in 1923. It consists in releasing the proximal insertion of the flexor-pronator muscles from their medial epicondylar origin. Through a longitudinal medial elbow incision, the ulnar nerve is dissected and protected, as well as the humeral neurovascular bundle, then all the medial epicondylar muscles are freed from their insertion on the epicondyle, the joint capsule, and more distally the intermuscular septum. The muscles are allowed to slide distally through gentle passive extension and supination of the wrist, allowing usually a 4 to 5 cm distal slide. Although classically left to spontaneous reinsertion distally, it has been advocated to suture the proximal extremity of the muscles to the ulnar periosteum in order to prevent a permanent supination deformity postoperatively.[33]

This procedure can be extended to the finger flexor muscles, as described by Scaglietti.[34] The skin incision is expanded distally, and all the finger flexor insertions are freed from the anterior aspect of the ulna and radius. Care must be taken to protect the anterior interosseous artery during the procedure. This procedure necessitates a wide dissection of the anterior forearm compartment, which makes careful hemostasis and postoperative suction drainage mandatory.

Zancolli[35] has described an ingenious and much more limited release of the medial epicondylar muscles consisting of a transverse resection of the inter- and perimuscular fascia of all the involved muscles, performed 6 cm distal to the medial epicondyle ('flexor aponeurotic release').

Tendon lengthening Goldner[3] advocated performing a Z lengthening of each individual tendon. This procedure, simple when there is only one or a few tendons requiring lengthening, becomes fastidious and time consuming if it is to involve all the flexors of the wrist and fingers, not to mention the resulting amount of scar tissue potentially impairing tendon gliding. When multiple lengthening is required, this is preferably performed at the muscle–tendon junction. The red muscle fibers are carefully detached from the white tendinous bands, and allowed to slide distally by passively extending the wrist and fingers.

STP Transfer of the flexor digitorum superficialis (FDS) to the profundi is a special type of tendon lengthening. Described by Braun[36] in 1974, it consists of sectioning all the finger FDS distally at the wrist, sectioning all the flexor digitorum profundi (FDP) 5 to 7 cm more proximally, and suturing the proximal stump of the FDS to the distal stump of the FDP. This predictable procedure reduces finger flexor contracture and pain, while improving hygiene and easing daily activities.[37]

Bone shortening Shortening the skeleton of both bones of the forearm does reduce muscle contracture, and has been advocated in non-cooperative adults. Omer[38] has performed proximal row carpectomy together with muscle transfers. This procedure slightly reduces muscle contracture, while improving wrist mobility in cases of joint contracture. It is also indicated in those patients who develop a symptomatic Kienböck's disease.[10]

Indications Muscle contracture involving only one tendon, or a limited number of tendons, may be treated by Z lengthening of the individual tendons. Mild contracture of the flexor and pronator muscles will be relieved by Zancolli's flexor aponeurotic release. This procedure has been reported as less effective in adults, where there is a component of myostatic contracture, than in children.[39] More severe cases require a conventional Page release. If the finger flexors are also contracted, the release can be extended in the manner of Scaglietti. It is technically easier not to include the FPL in the release, and to perform an individual Z lengthening of its tendon through a separate incision in the distal forearm.

In some adult patients unable to cope with the postoperative requirements, one might prefer to perform a bone shortening fixed with a strong plate and screws. In non- or poorly functioning hands, where contracture interferes with whatever function remains and/or with nursing care, it may be relieved by the simple STP procedure. None of these procedures (with the exception of first row carpectomy) has any effect on joint contracture. If, after all muscle contractures have been eliminated there is still a limitation of passive motion, joint procedures must be added.

Joint contracture

The classic techniques of arthrolysis may be applied to the spastic upper limb, taking into account that there are often combined muscle and joint contractures, both of which need to be addressed, and that the contracture is likely to recur if spasticity is not relieved and if the antagonist muscles are not active (or activated).

Fixed pronation deformity may require a release of the interosseous membrane (usually in conjunction with a Page procedure). Longstanding deformities in adults may only respond to osteotomy of the forearm.

Tendon transfers

Tendon transfers are required when the antagonist muscles are paretic or paralyzed. They are usually performed to improve forearm pronation, wrist extension, or thumb extension-abduction. They differ from classic tendon transfers in several points:

- Muscles available for transfer vary with each patient. They are often difficult to select because spasticity, co-contracture, muscle weakness, and lack of co-ordination render individual muscle assessment extremely difficult. Careful and repeated muscle evaluation, EMG study, and BT may all be helpful in that decision.
- The transferred muscle is often spastic to some degree. It has been pointed out that a spastic muscle that does not have a phasic control should not be used as a transfer.[16,17] Dynamic electromyographic (EMG) studies are very helpful in selecting adequate muscles.
- Tendon transfers have the double role of activating a paralyzed muscle, and of overcoming spasticity of the antagonists. However, if these are very spastic, they should be attenuated prior to or at the time of tendon transfer, by temporary (BT) or permanent (hyponeurotization) means.

One should also remember the possibility of an apparent palsy due to an excessive tendon length (i.e., ECR in severe flexion deformity of the wrist), in which case a mere muscle shortening alleviates the need for a tendon transfer.

Most frequent procedures

The extreme diversity of clinical pictures makes it impossible to describe all the surgical combinations of procedures performed in the spastic upper limb. The surgical planning adapts to each specific situation, bearing in mind that all deforming elements should be treated, and preferably at the same time, for a satisfactory result.

At the shoulder level Shoulder retraction in adduction and internal rotation, which impair function of the hand, should be treated. If necessary, a prior injection of BT will differentiate a true muscular contracture from spasticity. Muscle contracture is released by liberation of the subscapularis and tenotomy of the pectoralis major. Postoperative physiotherapy is initiated immediately, and continued for 8 weeks at least.

At the elbow level Surgery at this level is indicated mostly if one anticipates an improvement in hand function. However, elbow retraction may be so severe that it may require surgery even though the upper limb is not functional. Surgery is usually performed only after a prolonged period of rehabilitation and splinting.

In head injury patients, spasticity of the biceps may be isolated and temporary. In these patients, severe muscle hypertony may lead to early and irretractable flexion of the elbow.[40] If conservative therapy fails to improve it rapidly, BT is indicated. The triceps is then strengthened by physical therapy, and this regimen may permanently overcome spasticity. If spasticity recurs, hyponeurotization is indicated. It is particularly effective in this large muscle with a single and easily accessible motor branch.

In a number of cases, however, the other elbow flexors may also be spastic. EMG studies by Keenan[41] have shown that the brachioradialis may be severely affected. Each spastic muscle must be treated, whether BT or hyponeurotization is used.

In some cases, the flexion deformity is a complex one, combining spasticity, muscle (and joint) retraction, and paresis of the triceps muscle. BT facilitates assessment of the degree of muscle contracture. If it is mild (less than 40° of extension lag), it does not usually require surgical correction. More severe cases are treated by lengthening of each contracted muscle:

- The biceps may be lengthened by multiple transverse myofasciotomies at the muscle–tendon junction if the contracture is mild.[42] If it is severe, it is more appropriately lengthened by a long Z plasty of the biceps tendon.
- The brachialis is lengthened by multiple transverse fasciotomies, after dissection and protection of the radial artery.[43]
- The brachioradialis may require release from its proximal insertion, after dissection and protection of its nerve supply.
- Release of the flexor-pronator muscles, which are accessory elbow flexors, may also improve elbow extension.

Contracture of the elbow joint can be demonstrated only after muscle contractures have been released. If it is severe it requires a conventional anterior arthrolysis. Results of this surgery may be disappointing in the long term, especially if the triceps muscle is weak.

Tendon transfers are rarely indicated at the elbow, and concern only the triceps. The choice of motor is large, as many proximal muscles are usually active.

In non-functioning limbs with a severe elbow deformity, one may decide upon a simple procedure, such as a neurectomy of the musculocutaneous nerve,[44] or a biceps tenotomy, extended as required to the other elbow flexors, which usually improves elbow extension immediately by 40°.[40] Successive postoperative plaster postures usually further improve the result.

At the forearm level An impairing pronation deformity can be improved surgically. Available procedures include:

- hyponeurotization or tenotomy of the pronator teres if it is purely spastic
- lengthening of the pronator teres if the muscle is contracted, and release of the interosseous membrane when required
- rotational osteotomy of the forearm bones if the deformity is fixed
- tendon transfer, usually by pronator rerouting,[45,46] if the supinators are paretic or paralyzed.

Gschwind and Tonkin[47] have established a classification of pronation deformities and a proposed surgical plan for each group:

- Group I: there is active supination beyond neutral; it does not require surgery.
- Group II: active supination is limited to neutral or less; it may be treated by pronator quadratus release, associated with a flexor-pronator release if the flexor-pronator group of muscles is contracted. The quality of the result in this group will depend on the ultimate strength of active supination.
- Group III: patients display no active supination, but have full passive supination. This group is treated by muscle transfer of either the pronator teres (pronator teres rerouting) or the FCU.
- Group IV: there is no active supination, with a fixed pronation deformity. A release of the spastic pronator and pronatory-effect muscles is indicated to allow possible active supination to be unmasked. If this does not occur, a pronator teres transfer is indicated.[39]

At the wrist level The wrist is the most frequent site of operations in spasticity of the upper limb. Here again, the deformity (problem) results from a combination of spasticity and muscle contracture involving, to various degrees, the wrist and finger flexors, and muscle imbalance due to paresis or paralysis of the antagonist wrist extensors.

If there is no muscle contracture, one usually starts the therapeutic program with injection of toxin in the spastic muscles (FCU, FCR, PL), followed by a regimen of strengthening of the extensor muscles. This protocol may be sufficient in mild spasticity, provided rehabilitation and splinting are pursued for many months. If the extensor muscles do not respond to strengthening, the usual procedure is then to perform hyponeurotization of the spastic muscle at the same time as tendon transfer to the paralyzed extensors (usually to the ECR muscles). Relieving spasticity in the wrist flexors may unmask spasticity of the finger flexors, which will then tend to perpetuate the wrist flexion deformity. They must be treated accordingly.

Contracture in the flexor-pronator muscle group should be treated as described earlier. It has been our experience that this release usually attenuates spasticity of the involved muscles to a point where there is usually no need for complementary procedures aiming at reducing spasticity.

Paralysis or paresis of the wrist extensors is treated by tendon transfers, usually directed at the ECR muscles. The FCU is the most appropriate muscle when it demonstrates adequate relaxation at rest, provided the FCR is active. Other motors may include the brachioradialis, the pronator teres of a finger flexor superficialis.

In cases of a non- or poorly functioning hand with a severe wrist flexion deformity, a reasonable option for improving cosmesis and nursing care may be to fuse the wrist.[48,49]

At the finger level

Flexion deformity

Flexion deformity of the fingers should be treated in conjunction with the wrist deformity.

Isolated spasticity of the finger flexors theoretically responds to BT and hyponeurotization; however, the results of these two procedures are less predictable in the fingers than in the wrist, given the number of muscles and motor branches involved. Muscle contracture responds to flexor muscle slide (Scaglietti), tendon lengthening, or STP (see above for indications). Release of the finger flexion deformity may unmask an intrinsic spasticity, which will require subsequent treatment.

Tendon transfers to augment active finger extension are not frequently indicated, although there is no consensus in the literature.[50,51] Smith has recommended not to perform them until a minimum 6 months after the release procedures at the wrist and fingers, in order to allow spontaneous recovery of the tone of the stretched extensors.

Swan-neck deformity

As swan-neck deformity may be secondary to wrist hyperflexion, the latter must be corrected first. If the swan neck persists and interferes with function (locking), it should be corrected surgically.

If the swan neck is due to muscle imbalance, combining intrinsic muscle spasticity and overactivity of the finger extensors, this may be corrected by one of the following procedures: tenodesis of the flexor superficialis,[52] tenotomy of the central band of the extensor tendon,[53] lateral band tenodesis as performed by Littler[54] or by Zancolli, or spiral oblique ligament reconstruction.[55]

In spastic patients, one must be extremely careful not to overcorrect the deformity, as this may lead to the opposite boutonnière deformity.

Intrinsic contracture

Intrinsic contracture may be isolated, or associated with a swan-neck deformity. If the deformity is mild, the contracture may be released by resection of the triangular laminae, as advocated by Zancolli.[6] If the swan-neck deformity is severe, interphalangeal (IP) joint hyperextension must be treated as described above at the same time as the intrinsic release. Severe spasticity of the intrinsic muscles may be treated by neurectomy of the motor branch of the ulnar nerve in Guyon's canal.[56]

At the thumb level Available procedures aim at reducing spasticity, releasing contracted muscles, stabilizing thumb joints, and augmenting paretic or paralytic extensor muscles by tendon transfers.

Reduction of spasticity and muscle contracture

As mentioned earlier, nerve procedures do not seem to be effective enough in intrinsic muscles. An isolated contracture of the adductor muscle may be corrected by distal tenotomy at the sesamoid level, associated with a stabilization of the MP joint to avoid the development of a hyperextension

deformity of the joint. However, most frequently there is a combined contracture of the other thenar muscles (abductor pollicis brevis (APB), flexor pollicis brevis (FPB)), and of the first dorsal interosseous (1st DIO) together with the adductor muscle. In such cases, Matev[57] has described an extended palmar release including adductor pollicis (AP), FPB, and the distal two-thirds of APB. The first dorsal interosseous muscle may be released as needed through the same incision. This procedure is technically demanding as one must release these muscles completely while protecting their motor branches.[58]

When the flexor pollicis longus is contracted, it can be released through a tendon lengthening at the forearm. Muscle contracture, especially in cerebral palsy, may be associated with a skin shortage, which is treated by a Z plasty.

Joint stabilization

MP joint hyperextension deformities may be treated by a volar tenodesis or capsulodesis, but both of these have a tendency to slacken with time. One will usually prefer a sesamoid–metacarpal fusion described by Zancolli, or a conventional MP joint fusion in severe cases.

Tendon transfers

Tendons transfers are necessary when extension-abduction is paralyzed or weak. Suggested motors have been the extensor carpi radialis if the wrist extensors are spared,[57] the brachioradialis (BR),[35,52] or the flexor carpi radialis,[59] or a flexor superficialis.[60]

Inglis suggests rerouting the abductor pollicis longus (APL) through the APB in order to increase thumb abduction. Manske rerouts the extensor pollicis longus (EPL) when active, through the first dorsal compartment, so as to change its adduction component into abduction.

Classification

Tonkin[7] has described a classification into three types, modified from House,[61] with specific indications:

- Type I (intrinsic deformity) where spasticity of the medial thenar muscles (AP, FPB, and 1st DIO) associated with paresis of the thumb abductor and extensor muscles causes adduction of the 1st metacarpal, flexion of the MP joint, and extension of the IP joint. Tonkin advocates treating these patients with a combination of adductor/FPB release, and 1st DIO release if necessary, rerouting EPL to extensor pollicis brevis (EPB), BR transfer to APL, and stabilization of the MP joint by capsulodesis or sesamoido-metacarpal fusion, or MP fusion when required.
- Type II (extrinsic deformity), where the dominant deforming force is the FPL, opposing a weak EPL. Metacarpal adduction is less marked, but there is hyperflexion of the IP joint.These patients are treated by FPL tendon slide, and release of the other contractures as required.
- Type III (combined deformity), where there is a combination of spasticity of the adductor muscles and of the FPL, with weakness of the abductor and extensor muscles. This results in the typical 'thumb in palm' deformity, with adduction of the thumb metacarpal and flexion of the thumb MP and IP joints. Treatment includes the same procedures as in type I, associated with FLP tendon slide, and IP joint fusion in recalcitrant flexion deformities.

Conclusion

Not many spastic patients are candidates for surgery of their upper limb, because of the many other neurologic problems frequently associated. Surgery should be decided upon only after several sessions of examination and evaluation of the upper limb. One should take into acount the other neurologic impairments, the patient's functional achievements, and his (or his parents') wishes. Surgery of the spastic upper limb is complex, involving reduction of spasticity, release of contracted muscles and joints, augmentation of weak or paralyzed muscles, and stabilization of unstable joints. There is no such thing as 'standard procedures' in this type of patient, because each case is different.

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31 Hand reconstruction in leprosy

Francis Chaise

Introduction (Figure 31.1)

Leprosy, or Hansen disease, a chronic infectious disease caused by *Mycobacterium leprae*, affects an estimated 700 000 persons each year. Clinically, leprosy can be categorized as a paucibacillary or multibacillary disease. The immunologic status of the patient, as evidenced by the degree of resistance to the organism, appears to determine the type of disease that develops. Leprosy is important largely because of the deformities, disabilities, and handicap it causes in a proportion of those affected by the disease. There are surgical procedures and techniques to correct or limit the deterioration of these conditions. In the past these surgical procedures were only performed in special institutions for treating leprosy and their complications. However, with the widespread use of multidrug therapy (chlofazymine, rifampicin, and dapsone; PCT) and the consequent reduction in the prevalence of leprosy, there is progressive integration of the care of people affected by leprosy into the general health services.

Surgery, as in intervention in the management of leprosy and its complications, is used in patients who are already under antileprosy treatment, or after they have completed it satisfactorily. Therefore preventive surgery like nerve decompression and corrective surgery should not be practiced in places where there is no leprosy program.

This chapter describes nerve decompression for preventing paralytic deformities and procedures for the correction of claw deformities of finger and thumb resulting from ulnar or combined ulnar and median nerve paralysis, commonly seen in people affected by leprosy.

In order to carry out these procedures, many involving tendon transfers with or without tendon grafting, the surgeon has to be well versed in the structural and functional anatomy of the hand and should be trained in hand surgery. Furthermore, supportive physiotherapy and, if possible, occupational therapy services for pre- and postoperative management of the hand should be available. If the corrective procedures are carried out in the absence of any of these requirements, the venture is bound to result in failure, worsen the hand disability, as well as make any subsequent correction very much more difficult.

Surgical procedures for correcting deformities in leprosy patients, as well as those for preventing disabilities or their worsening, began to be practiced when leprosy became a curable disease with the use of dapsone, about five decades ago. These procedures were developed and carried out mostly in special institutions treating leprosy patients.

Since the 1980s, with the advent of the curative multidrug therapy (MTD) and its widespread implementation, increasing numbers of leprosy patients are being cured outside these special institutions, and are released from treatment. A great proportion of them have residual problems, mainly loss of sensibility in hands, feet, or eyes, with or without motor paralysis and associated deformities. The affected parts are certain to develop further complications and secondary problems like ulceration of soft tissue, stiffness of joints, and destruction of skeletal architecture if they are not well cared for. Inevitably these complications will worsen the disability and make correction of the deformities very much more difficult or impossible, and eventually cripple the patient severely. However, the occurrence of these secondary complications can be prevented by taking protective measures.[1] Furthermore, even when they have occurred, their late consequences can be prevented by detecting the conditions early and treating them quickly and properly using appropriate surgical and non-surgical methods.

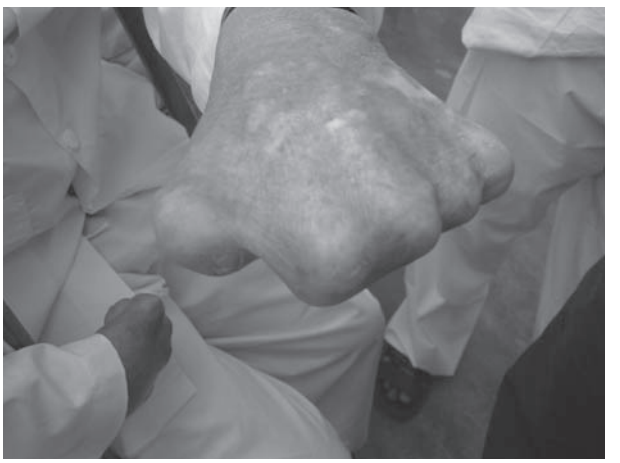


Figure 31.1
Mutilations of the hand.

General considerations on hand surgery in leprosy

Leprosy patients develop deformities of three types:

1. deformities due to the multibacillary type of leprosy (loss of eyebrows)
2. deformities resulting from paralysis of some muscles because of leprosy-induced damage to peripheral nerve trunks (claw hand, foot drop, lagophthalmos)
3. deformities resulting from injuries and infections to hand and feet that have lost sensibility because of damage to peripheral nerves (scar contractures of finger, ulcerations of feet and hands, shortening of digits, mutilations of hands and feet, corneal ulcerations).

Aims

Surgery is required in leprosy patients for many reasons:

- To treat acute infections of the hand.
- To prevent permanent paralysis of a nerve trunk and thus prevent paralytic deformities. Decompression of the nerve trunk is performed in order to eliminate the mechanical factors that accelerate nerve damage and thus to help in recovery of the nerve.
- To correct paralytic deformities and disabilities resulting from muscle paralysis in the hand. This often involves tendon transfer.

With surgery we can only improve the form and function in paralytic deformities, but sensibility cannot be restored. Therefore it is absolutely essential to educate and train the patient to take care of the part which has loss of sensibility, to prevent injuries, infections, and secondary deformities.

Timing

Surgery for acute infections of the hand depends on the local condition and not on the leprosy disease status. If the local condition warrants a particular procedure, it is done provided the general condition of the patient permits it.

As for other paralytic deformities, in leprous deformities it is necessary to be sure that no further muscle paralysis is likely to occur. One should also be sure that the muscle paralysis is not likely to recover. Both conditions are met if the patient has been under treatment, if the deformity has been present for 6 months or longer, or if the muscles concerned are atrophied. It needs to be stressed that no nerve trunk should be tender, which indicates disease activity and possible paralysis in the future.

In summary, corrective surgery for paralytic deformities may be done if the patient:

- has shown good clinical and bacteriologic response with PCT (multidrug therapy)
- has had no reaction or neuritis within the previous 6 months
- has no tenderness of the nerve trunk
- and has had the deformity for at least 6 months.

Decompression of the nerve in leprous neuritis[2–7]

In leprosy, the nerve trunks of the upper and lower limbs are maximally affected where the nerve lies superficial to or passes through an osteofibrous tunnel close to a joint. The inflamed, swollen nerve is vulnerable to physical trauma, compression, stretch, and friction at these sites. These mechanical factors accelerate nerve damage. Further, the swollen nerve trunk may become ischemic due to pressure from structures outside the nerve (e.g., the unyielding walls of the tunnel) or by its own thickened outer sheath. The drugs given to alleviate the neuritis do not reach the strangulated part of the nerve. These factors contribute to the accelaration of nerve damage. So, the aim of decompression of a nerve trunk is to aid functional recovery by removing these mechanical factors. Decompression is only one part of the treatment of nerve damage, the other comprising the medical and physical measures of therapy. In the majority of cases, nerve damage is reversible if detected early, and treated medically and with physiotherapy. In some cases, neuritis and nerve damage continue to progress in spite of adequate medical treatment, and it is in these cases that surgical decompression of the nerve is indicated. Surgical decompression also helps to relieve chronic and severe pain. It is usually performed to arrest progressive damage and possibly aid in the recovery of ulnar and median nerves. Two types of surgical decompression are possible:

- *In external decompression*, compression of the nerve trunk by external structures is relieved. An example is decompression of the median nerve in carpal tunnel syndrome by dividing the roof of the tunnel.
- *Internal decompression* with epineurotomy is the procedure whereby the nerve bundles are released from compression by the epineurium which has become thickened, fibrosed, or scarred. This a difficult and sophisticated procedure needing good training in hand and nerve surgery. The main indication for epineurotomy is when fascicles are not visible through the thickned sheath.

When performing nerve surgery in leprous neuritis, the following points should be borne in mind:

- Surgery is the deliberate infliction of a wound, in leprosy the nerve trunk is already damaged and diseased.

- The nerve trunk gets its blood supply through vesssels in the mesoneurium, which conveys these vessels to the nerve and is attached to the nerve on its deeper side. Therefore the mesoneurium has to be fully respected.
- The perineurium is an important structure covering and protecting the nerve fascicles and should never be damaged during surgery.

Surgical decompression of the ulnar nerve in the arm (Figure 31.2)

Indications Surgical decompression of the ulnar nerve is required when signs of ulnar damage appear or increase while under adequate treatment for neuritis (steroid administration, splinting, physiotherapy for 2 or 3 weeks). The supplementary indications for surgical decompression of the ulnar nerve are as follows:

- persistence or increase of pain or tenderness of the nerve
- appearence of pain at the elbow when the elbow is forcibly flexed passively beyond the limit of active flexion
- continued restriction of full active elbow flexion even under adequate treatment.

Anesthesia Axillary block of the brachial plexus may be used. A tourniquet is used when possible (neuritis only around the elbow and not at the proximal part of the arm).

Aim of the procedure The procedure aims to divide all structures that may be constricting the ulnar nerve in its course through the posterior compartment of the arm, from its point of entry in the upper arm to below the elbow.

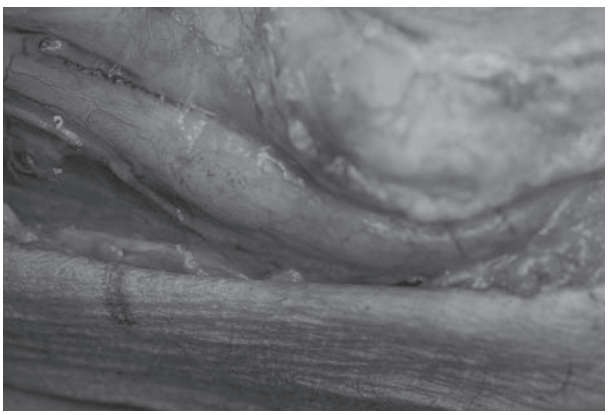


Figure 31.2
Neurolysis of the ulnar nerve.

Procedure Three steps are needed:

- a long incision is made around the medial epicondyle (10–12 cm)
- the compression is released by dividing the structures constricting or compressing the ulnar nerve (external decompression)
- the medial epicondyle of the humerus is resected as part of the external decompression operation in order to eliminate all mechanical stresses on the ulnar nerve consequent to elbow movements.

Internal decompression (epineurotomy) should be performed when the epineurium is found to be very thickened or scarred. The epineurium should be split longitudinally, and the underlying nerve fascicles should be carefully protected.

Postoperative care Bulky dressings are applied and the arm is bandaged with the elbow in the extended position. After 10 days the sutures are removed and the patient starts to practice active elbow flexion exercises.

By the time of surgery the dosage of steroids will have been decreased to 10 or 5 mg prednisolone daily. The same dosage should be continued after the nerve decompression, the duration depending on many factors: involvement of other nerves, type of leprosy, and recurrence of neuritis. No antibiotics are ordinarily required.

Surgical decompression of the median nerve at the wrist region

Indications Surgical decompression of the median nerve is required when signs of median damage appear or increase while under adequate treatment for neuritis (steroid, splinting, physiotherapy for 2 or 3 weeks). Persistence or increase in pain or tenderness in the nerve, or pain in the nerve during thumb–middle finger pinch, even while under adequate treatment, are supplementary indications for decompression of the median nerve.

Anesthesia Axillary block of the brachial plexus may be used. A tourniquet is used when possible.

Aim The procedure aims to divide all structures that may constrict the median nerve in its course through the wrist and the lower quarter of the forearm.

Technique A 10 cm long longitudinal incision is made over the middle of the lower part of the front of the forearm and the anterior part of the wrist. The deep fascia and the flexor retinaculum are divided carefully to open the carpal tunnel and perform external decompression of the median nerve. Internal decompression (epineurotomy) should be performed when the epineurium is found to be very thickened or scarred. The epineurium should be split

longitudinally, insuring that the underlying nerve fascicles are carefully protected.

Postoperative care Bulky dressings are applied with the wrist bandaged in the extended position. After 10 days the sutures are removed and the patient starts to practice active wrist flexion–extension exercises. In acute neuritis a splint may be given for 20 days to insure that the wrist is rested in the neutral position. The corticosteroids must be continued for 10 days unless there are other indications. No antibiotics are ordinarily required.

Nerve abscess

In leprosy, acute and chronic abscesses can develop in the nerve. Acute abscesses are usually very small and are frequently seen in acute neuritis. Chronic abscesses are 'cold abscesses', as in tuberculosis, resulting from caseation and liquefaction of the affected nerve fascicles, and are not painful. The abscesses may be wholly intraneural with nodular swelling of the affected segment of the nerve. Quite often the abscess breaks out of the nerve to form a 'collar stud' abscess around the nerve. Decompression of the nerve is required when there is partial paralysis of the nerve or complete paralysis of recent onset (2 months or less), or when the abscess becomes adherent to the overlying skin and threatens to break open to form one or more sinuses. In these cases the abscess should be carefully removed.

Surgical procedures for correction of paralytic deformities of the hand (Figures 31.3–31.7)

General considerations

Careful preoperative assessment, physiotherapy, and muscle training, as well as postoperative physiotherapy, are equally essential for the success of the operations described. This also requires some experience because the situation in leprosy is complicated by loss of sensibility, consequently any postoperative complications will not be signaled by pain in the area. Only careful daily monitoring of the patient will detect such adverse events at an early stage (e.g., infection, ischemia). It would be totally unethical to perform this kind of surgery without experienced personnel available for pre- and postoperative management. This is because these operations are elective procedures aimed at improving the quality of life of the patient, and failure of surgery leaves the patient worse off and very much more disabled than before.

Nerve trunks of the upper extremity, especially the ulnar and the median nerve, are frequently affected by leprous neuritis, resulting in paralysis of these nerves. Thus many

Figure 31.3
Claw fingers.

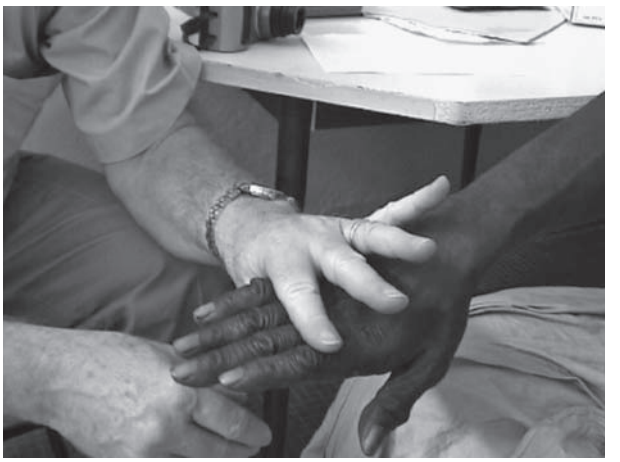


Figure 31.4
Claw fingers with no stiffness.

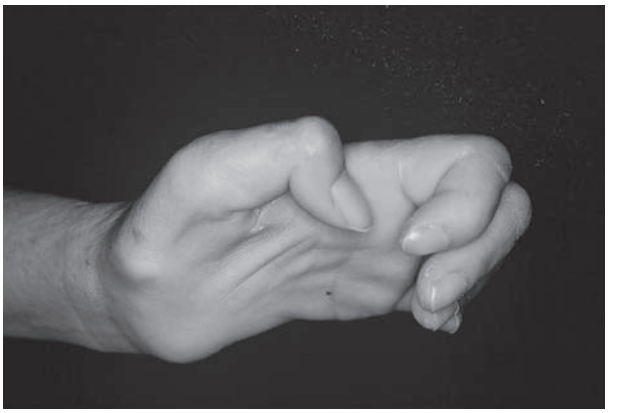


Figure 31.5
Typical claw hand.

patients develop ulnar nerve paralysis alone, median paralysis alone, or combined paralysis of ulnar and median nerves. The first type of paralysis (ulnar isolated) gives rise to claw-hand involving the fingers, while the second type

Figure 31.6
Claw hands.

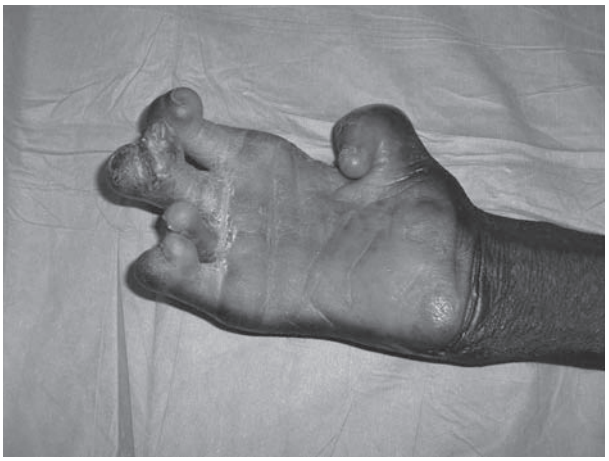


Figure 31.7
Paralytic deformities.

(median and ulnar) gives rise to total claw-hand involving the thumb as well as the fingers. These deformities stigmatize those affected as leprosy patients. Further, because the hands are involved, these people also experience certain disabilities in the use of their hands. These deformities and disabilities occur because the intrinsic muscles are paralyzed and the normal balance of forces around the joints of the fingers and thumb is upset. This renders useful postures and movements impossible for these digits.

A number of operative procedures, many involving tendon transfer with or without tendon grafting operations, are available to restore the balance of forces and so correct the deformity and disability. For the correction to be successful, the muscles of relocated tendons should act in the proper sequence and in coordination with the other muscles. This is possible only when the brain recognizes the altered anatomic situation and learns to initiate appropriate movement patterns. This is achieved by a planned pre- and postoperative training program utilizing physiotherapy and occupational therapy. Corrective surgery for paralytic deformities of the hand should not be undertaken when facilities for such training are not available.

Selection of subjects

The first requirement is a well-motivated patient; it is important that the surgeon understands exactly which function the patient would like to be restored, or the particular deformity correction that the patient considers most important. What is expected of the patient (e.g., physiotherapy, program, duration of stay in hospital) should be explained in simple language. One should also explain what will not be restored (sensibility, adduction–abduction of fingers). These explanations are needed because surgery is only one step in the treatment of the patient, and the other steps (re-education, protecting the corrected hand from injury and infection) depend entirely on cooperation and understanding by the patient. The best results are obtained in young people with recent deformity and a strong motivation to undergo corrective surgery. It is also necessary for the success of corrective surgery that the fingers are supple. In the case of flexion contracture, the stiffness should be overcome first and the digit made straight and supple by physiotherapy and splinting before surgical correction.

Timing of surgery

Several requirement are needed for a successful correction:

- a good reponse to antileprosy treatment
- no reaction or neuritis during the last 6 months
- no tenderness of any nerve trunk
- no joint or soft tissue contracture or damage to underlying bones or joint
- a record of the assessment of the paralyzed hand
- preoperative training: learning to achieve isolated contraction of the muscle to be transferred, and to eliminate the compensatory abnormal movment using techniques of physiotherapy and occupational therapy: e.g., wax bath, oil massage, exercises, and splinting.

Anesthesia and tourniquet

The procedures on the hand are best done under local anesthesia using the technique of axillary block of the plexus brachial and tourniquet. Surgery of the hand in leprosy requires gentle handling of the tissue, minimal handling of tendons, hemostasis, and perfect asepsis. It is essential to minimize the time the wound is exposed to the atmosphere. Finally, the surgeon himself must apply the dressings, bandages, and the plaster slabs or casts at the end of the operation to ensure that the operated part is

positioned correctly and the bandaging and plaster cast are not too tight.

Postoperative care

Postoperative care is the same for all the procedures described:

1. The operated hand should be kept well elevated for 72 hours.
2. Mild analgesics may be needed for 2 or 3 days and no longer.
3. There is no need for routine antibiotic therapy postoperatively. If the correct procedures are followed for preoperative preparation of the skin and limb, aseptic precautions and sterility in the operating room, and gentle surgery, perfect hemostasis will ensure primary wound healing with no infection.
4. The sutures are removed after 3 or 4 weeks. Postoperative physiotherapy and training are started by the therapist or the surgeon himself and continue for 4 weeks. It must be emphasized once again that corrective surgery should not be undertaken if such postoperative training is not possible.

Surgery for correction of paralytic claw fingers[8–24]

Claw-finger deformity is commonly seen in leprosy. Ulnar paralysis alone results in 'partial claw hand', in which the little and ring finger are maximally clawed because both the lumbrical and interosseous muscles of these fingers are paralyzed, while the index and the middle fingers show only mild or no clawing because only the interosseus muscles are paralyzed, not the lumbricals. Damage to both nerves results in 'complete claw hand' and all four fingers show severe claw deformity. The claw deformity stigmatizes the person as a leprosy patient and the functional disability affects the skillful use of the hand, both in the activities of daily life and at the workplace.

Preoperative evaluation

Preoperative evaluation is necessary for selection of one procedure, and for assessing the result of surgery later.

Assessment of claw deformity With the wrist in a neutral position the patient opens out all the fingers, then the angle of flexion at the proximal interphalangeal (PIP) joint is measured for each finger.

Assessment of the extensor apparatus The patient must straighten the fingers at the PIP joint when the metacarpophalangeal (MCP) joints are held straight. When there is no flexion contracture at the PIP joint, when the extensor muscles are normal and when the extensor expansion of the fingers is not damaged, the PIP is found to be fully straight. If there is an angle at the PIP it must be measured. If a procedure such as capsuloraphy, flexor pulley advancement, or extensor diversion graft is selected it is necessary to evaluate the minimum amount of flexion in which the MCP joint must be held to attain a straight finger. If this angle exceeds 30°, these operations must not be done.

Assessment of the state of the PIP joint If the PIP joint cannot be extended passively, the angle of contracture must be evaluated. When this contracture angle is more than 45°, tendon surgery is not suitable, if it is less, physiotherapy measures can correct such mild contracture.

Assessment of the digital flexors The status of each finger must be tested separately (actively and against resisting movement) for the flexor superficialis and flexor profundus. The strength of the flexor profundus of the ring or little finger must be carefully evaluated. In case of weakness of the flexor profundus of the ring finger, the flexor superficialis will be not removed for transfer. In a hand with longstanding claw-finger deformity, as in Volkmann's ischemic contracture, the finger cannot be extended, even passively, at the PIP joint. However, when the wrist or MCP joint is held in flexion it becomes possible to straighten the PIP joint fully, at least passively. Patients with this condition need stretching of the flexor muscle mass, splinting, and physiotherapy.

Other assessments Sensory charting, general examination of the hand, muscle testing, assessment of power grip and pinch, and palpation of the nerve trunks should be done routinely. Corrective surgery must be postponed when there is tenderness of nerve trunks.

Selection of patients

Best results are obtained in the hands of well-motivated young patients, with recent deformity, no stiffness of the finger joints or soft tissue contractures, with no or very little hyperextension at the PIP joint, no or very little ulnar deviation of the fingers, no loss of PIP extension when the MCP is straight, and no weakness of the forearm muscle.

Preoperative physiotherapy

Routine physiotherapy with hot wax baths, oil massages, and exercises to isolate the muscle to be transferred should be given for at least 2 or 3 weeks before surgery.

Procedures

Many procedures requiring different levels of expertise have been described for the correction of claw deformity of

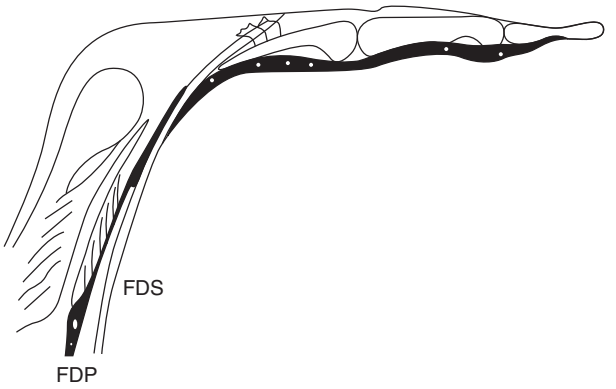


Figure 31.8

Transfer of the flexor digitorum superficialis (FDS) and insertion of the slip. FDP, flexor digitorum profundus.

the fingers. In our experience of surgery in leprosy for many years, in Asia and in Africa, we have selected four procedures which are described here.

Transfer of the flexor superficialis of one finger to the extensor expansion of all four fingers (Figure 31.8)

The principle of this procedure is to reconstruct the function of the paralyzed intrinsic muscles and restore the balance of forces around the joints of the fingers. The following steps are used to perform this procedure:

- Exposure of the extensor expansions on the fingers: longitudinal incisions are made on the radial side of the little, ring, and middle fingers and on the ulnar border of the index finger at the level of the proximal phalanx. The extensor expansion and the lateral band are exposed sufficiently for tendon suturing.
- Release of the flexor superficialis tendon from the middle finger.
- Withdrawal of the motor tendon into the palm and its division into four slips.
- Tunneling the tendon slips into the fingers dorsal to the palmar transverse ligament to follow the route of the interossei.
- Suturing the tendon slips to the extensor expansion: the wrist is held in a neutral position or in mild flexion the MCP joints in 55° flexion, and the PIP joints straight.
- Bandaging and immobilization: for 4 weeks the hand must be held in a plaster cast with the wrist in a neutral position, the MCP joints at 10° more than at the time of suturing, and the PIP joints straight.
- Re-education and physiotherapy: after 4 weeks the stitches are removed, and re-education and physiotherapy exercises begin from the next day.

Transfer of the flexor superficialis tendon of one finger to the flexor pulley of all fingers (Figure 31.9)

In this operation, the motor tendon slip of the flexor superficialis is attached to the fibrous flexor sheath (flexor pulley), so that the transfer functions purely as a flexor of the proximal phalanx, and by countering extensor dominance at the MCP joint restores the balance of forces in the finger joint system.

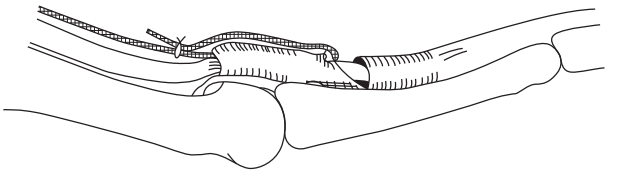


Figure 31.9

Lasso procedure.

Indications It is especially indicated in hands with long slender hypermobile fingers without significant hyperextension (or less than 20°) at the PIP joint when the finger is actively opened out fully (as when the MCP is held in neutral extension). When there is hyperextension, if the motor is inserted in the extensor expansion, hyperextension at the PIP joint becomes worse. In ulnar paralysis only the flexor superficialis is used for the ring and the little finger.

Operative procedure The two slips of flexor superficialis tendon of the middle finger are detached, by dividing the tendinous chiasma, and are withdrawn in the proximal palm. Each half is split into two slips in order to obtain four tendon slips. After this procedure the pulleys are exposed by a transverse palmar incision and, between the first and the second pulley, the slip of tendon is withdrawn. The tendon slip is folded back in front of the first pulley to create a lasso in which the first pulley is caught. For the index and middle fingers the MCP joints have to be fixed by about 30°, and for the ring and little fingers, by about 40°. Nylon sutures are tied, taking care to insure that the folded back part of the slip is included in the knot. For all fingers the procedure is the same.

Confirmation of the tendon suturing The tension is confirmed by observing the resting position of the MCP joint. On wrist extension, the proximal phalanges of all fingers should flex equally at their MCP joints.

Immobilization The position of the wrist and MCP joint is the same as for the previous procedure, but the PIP joints are left free to move and the patient is encouraged to flex and extend as soon after the operation as possible. This is very important to prevent any adhesions between tendons within the flexor apparatus. After 4 weeks the wrist and MCP joints are left free to move actively.

Capsuloraphy and pulley advancement (Figure 31.10)

In this operation, hyperextension at the MCP joint is prevented by shortening the anterior capsule of this joint. In addition, the leverage of the flexors at these joints is

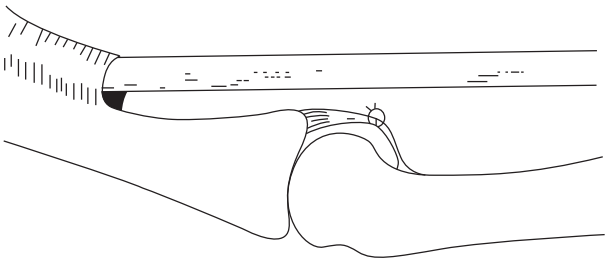


Figure 31.10
Capsuloraphy and pulley advancement.

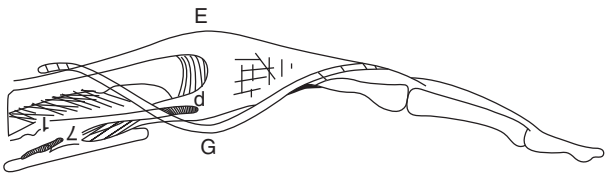


Figure 31.11
Diversion graft.

enhanced, by excision of the proximal part of the flexor pulley system.

Indication It is particularly indicated in hands with mild claw deformity of only one or two fingers due to ulnar paralysis.

Operative procedure A transverse palmar distal incision is generally used, the flexor apparatus is exposed in its sheath and the A1 and A2 flexor pulley. At about the level of the neck of the metacarpal, two longitudinal cuts in the fibrous flexor sheath are made at a distance of about 10 mm apart. The fibrous flap is cut and removed. The capsuloraphy (shortening of the joint capsule) can be done so that there is 20–30° flexion contracture at the metacarpophalangeal (MP) joint. At this time the little finger must be keep adducted in order to correct any ulnar deviation, and to prevent any future ulnar deviation of the fingers. Many other methods of shortening the anterior capsule have been described in the literature, e.g., intraosseous fixation of the capsule or the use of an anchor, but for leprosy, the procedure described is suitable in any case.

Postoperative management For 5 weeks the MP joints are kept flexed at about 70°, the wrist is kept straight, and the PIP joints are not included in the cast. On the second day the patient starts to flex and extend the PIP joints of the fingers actively to prevent adhesions and to retain finger mobility. The cast is removed at 5 weeks and thereafter the patient can start using his hand for daily living activities. At 6 weeks different types of grasp exercises are started: e.g., grasping and playing with a rubber ball, grasping a glass of water. For 3 months the patient should not lift any heavy weights, not use the hand for heavy work, and must totally avoid the habit of forced extensions of the fingers. Most patients are able to achieve nearly straight fingers in the open hand position by 3 months after the operation.

Extensor diversion graft operation (Figure 31.11)

In this procedure, some of the force in the digital long extensor tendon is diverted to the volar aspect of the MCP joint, and returned to the extensor expansion distally by means of a tendon graft or fascia lata graft. The claw finger deformity is abolished by stabilizing the MCP joint. However, although it corrects claw deformity, it does not add power to the fingers and the proximal phalanx is flexed only by flexing the interphalangeal (IP) joints. Thus the fingers remain weak and the full intrinsic position is not restored after this operation. The procedure is particularly useful for cosmetic correction of clawing of one or two fingers, but it can be used for correcting all four fingers in patients in whom difficulties in postoperative re-education are anticipated.

Operative procedure If sufficient length (12 cm) of palmaris longus tendon is available, it may be used as a free graft; if not, a similar length of fascia lata from the thigh may be used instead. The grafts are transferred subcutaneously along the lateral band and lumbrical canal, anterior to the deep transverse metacarpal ligament. The lateral band is exposed on the radial side of the finger, and the extensor tendons are exposed on the dorsum of the hand up the neck of the metacarpal. The graft is cut into two or four pieces and the distal end of the graft is then sutured to the lateral band and extensor expansion at the level of each finger. The next step in the operation is to attach the proximal end of each graft to the corresponding digital extensor tendon in the dorsum of the hand. During this procedure, the wrist is kept in mild extension (about 15°), the MCP joints are held in some flexion (about 30°), and the PIP joints are straight. It is most important that the graft is held tight. Adequacy of extensor diversion is assessed by observing the resting posture of the fingers. If diversion is optimal, the resting posture of the hand is similar to that of the normal hand. If undercorrection or overcorrection is apparent, the graft is detached from the extensor tendon on the dorsum and reattached after the posture has been corrected.

For 4 weeks, the wrist is immobilized in mild extension (30°), the MCP joints in about 70° flexion, and the IP joints straight. At the end of the fourth week, wax baths and exercises are started. When full finger flexion and extension are restored, daily supervision is discontinued. The patient is forbidden to do heavy work or lift heavy weights for 3 months after surgery.

Many other procedures using free grafts and other motor tendons are possible (palmaris longus, flexor carpi radialis, extensor carpi radialis). These procedures are more difficult to perform and need a protracted and difficult course of physiotherapy. They can be performed only in specialist hand surgery units by very experienced surgeons.

Surgery for correction of claw thumb deformity[17,23–27] (Figures 31.12–31.16)

Paralysis of the ulnar nerve and combined paralysis of the ulnar and median nerves occur commonly in leprosy, and since these nerves supply the small muscles of the thumb, paralytic deformities of the thumb are frequently seen in these patients. Combined paralysis of the ulnar and median nerves results in paralysis of all small muscles of the thumb (abductor pollicis brevis, flexor pollicis brevis, opponens pollicis, and adductor pollicis). This gives rise to the deformity of 'claw thumb' and severe disability. When only the ulnar nerve is paralyzed the thumb is affected in a rather subtle manner. In this condition, the adductor pollicis becomes paralyzed, giving rise to weakness of grip and early fatigue of the thumb. Adduction of the thumb is still carried out using the extensor pollicis longus muscle. In about 60% of cases of ulnar paralysis, the flexor brevis muscle is also paralyzed. This weakens flexion of the proximal phalanx of the thumb, leading to buckling of the thumb with extension at the MCP joint. These conditions are corrected by suitable tendon transfer procedures.

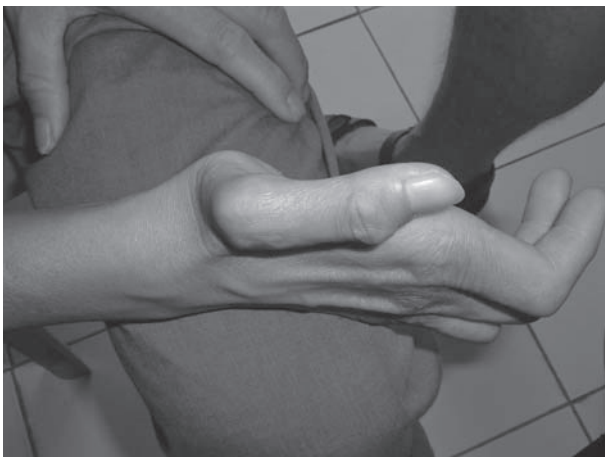
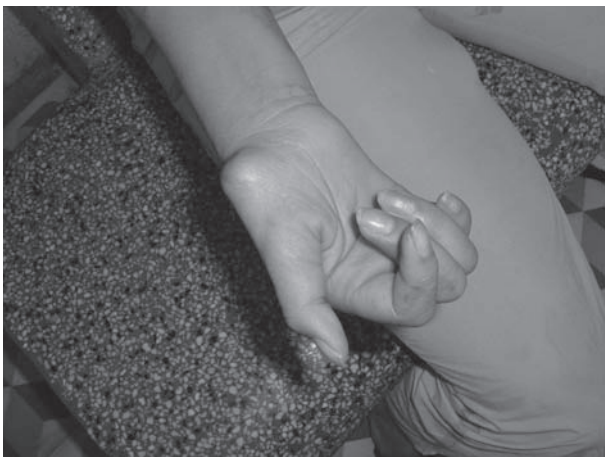


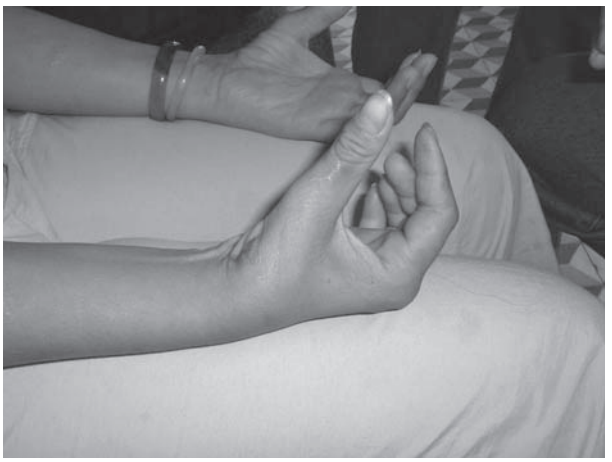
Figure 31.12
Resting position of thumb with extensive paralysis.

Figure 31.13
Instability of the thumb in ulnar paralysis.



(a)

(b)



(c)

Figure 31.14
(a) Paralysis of the thumb before surgery; (b) results of a bilateral transfer of the flexor sublimis of the third finger; (c) results of a transfer of indicid proprius.

Evaluation of the thumb

The thumb must be examined carefully, noting its resting position and recording all the muscles moving the thumb and its three joints (the carpometacarpal (CMC) joint at the base, the MCP in the middle, and the IP distally). The CMC must be checked for subluxation and contracture in the extended and externally rotated position, the MCP must be checked for arthritis and instability in flexion, and the IP for joint contracture. Further, the first web must be examined for contracture. When there are joint problems such as subluxation or arthritis, radiographs must be taken to confirm the suspicion.

General considerations

When all the muscles of the thumb are paralyzed, the thumb is unable to abduct, flex, or rotate inward, but it can adduct with the extensor pollicis longus.

Aim of surgery

The aim of surgery is to restore voluntary abduction-opposition of the thumb by transferring the tendon of a normal muscle.

Case selection

Cases without complications, such as thumb web contracture, IP joint contracture of more than 30°, arthritis of the MCP joint, or subluxation of the CMC joint, are selected for surgery. If any of these complications are present, many other procedures have to be used, e.g., IP arthrodesis, first web release, stabilization of the CMC joint by ligament reconstruction. In a few cases (e.g., contracture in extension of the MCP joint), arthrodesis of the MCP joint is necessary.

Procedure

The tendon of the flexor digitorum superficialis from the ring finger is detached and rerouted to the thumb to function as its abductor and opponens. Many techniques are available for this, of which one using the ulnar canal for a pulley is the most frequently used. The procedure is performed in several steps (Figures 31.15 and 31.16):

- The superficialis tendon is detached from its insertion and withdrawn from the forearm.
- The tendon is tunnelled through the ulnar nerve canal into the palm.
- The tendon is tunnelled through to the medial side of the thumb.
- The short adductor tendon and the medial joint capsule of the MCP joint are exposed.

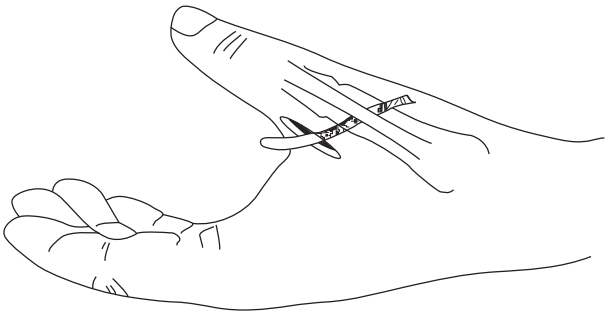


Figure 31.15
The FDS tendon is brought to the ulnar side of the MCP joint of the thumb.

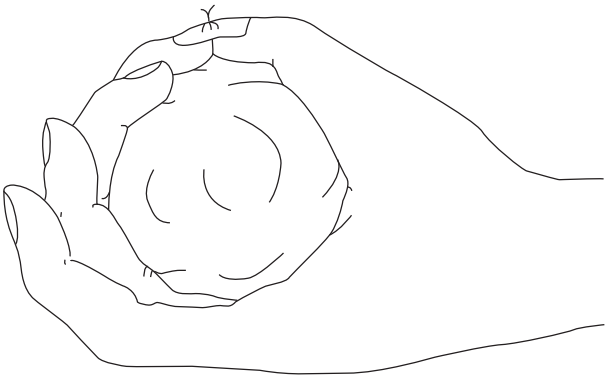


Figure 31.16
One easy method for postoperative positioning of the thumb.

Suturing the transfer

During suturing, the position of the thumb is such that the thumb nail is almost parallel to the palm. The position of the thumb can be easily maintained by placing a sterile gauze roll in the thumb web. The two tendon slips of the transferred flexor superficialis are now sutured to the joint capsule, including in the suture any fibers of the paralyzed adductor pollicis. Afterwards the position of the thumb must be checked. It should remain in 80% abduction-rotation. If not, the stitch must be removed and the tension readjusted. An alternative method of tendon fixation is possible, whereby the second slip of the transferred tendon is fixed distally through the extensor pollicis longus while the first slip is fixed to the medial capsule and the tendon of the adductor pollicis.

Bandaging and immobilization

After skin closure, dressing and bandaging are done as usual, followed by immobilization for 4 weeks in a cast with the wrist in the neutral position, the thumb in full abduction and rotation, its MCP joint in 30° of flexion, and the PIP in full extension. The sutured incision of the ring finger is dressed and bandaged. Alternatively, the nails

of the thumb and the little finger are stitched together over a bulky palmar gauze roll.

Contraindications

Thumb web contracture, flexion contracture of the PIP joint, CMC joint instability, and non-availability of the flexor superficialis of the ring finger because of weakness of the flexor profundus muscle are contraindications, and the patient must be treated by other procedures.

The medial dislocation of the extensor must be checked. In fact, in longstanding ulnar–median nerve paralysis, the key pinch, by which objects are held between the adjacent sides of the thumb and index finger, is constantly used. This leads to medial dislocation of the extensor pollicis longus at the level of the wrist. Correction of this deformity is essential to erase from the patient's mind this adaptive adduction function of the extensor. Therefore, when such a condition is present, the operation of restoration of opposition should always be combined with a simple correction of this condition.

Other procedures

Lateralization of the extensor pollicis longus tendon at the wrist

If preoperative contraction of the extensor pollicis longus during the key pinch occurs with medial dislocation of the tendon, the procedure must be done together with the other procedures. A short dorsal incision is made across the line of the extensor tendon. The tendon is identified at the pulley of Lister's tubercule. Next the aponeurosis overlying the tendon is cut and the tendon shifted radially. The flaps of the pulley must be sutured in order to insure that the tendon will not return to its original course. Another simpler procedure is possible: removal of Lister's tubercule.

Transfer of the extensor indicis proprius around the ulnar border of the wrist

This is a suitable procedure in leprosy. The extensor indicis proprius is detached from the ulnar border of the extensor apparatus of the index finger at the MP joint, withdrawn at the posterior side of the wrist, and transferred subcutaneously through the insertion of the abductor pollicis brevis and the long apparatus of the thumb. The postoperative program is the same as for the previous procedure.

Transfer of the flexor superficialis of the ring finger and rerouting at the ulnar border of the palmar aponeurosis

This transfer, commonly known as the Thompson transfer, is suitable when there is an isolated paralysis of the adductor pollicis. The flexor superficialis is withdrawn into the palm and rerouted subcutaneously to the thumb, where the fixation is carried out as in the transfer through the ulnar nerve tunnel.

Transfer of one half of the flexor pollicis longus onto the extensor apparatus (Figure 31.17)

This is the preferred procedure, together with the flexor superficialis transfer procedure, in all cases of ulnar–median

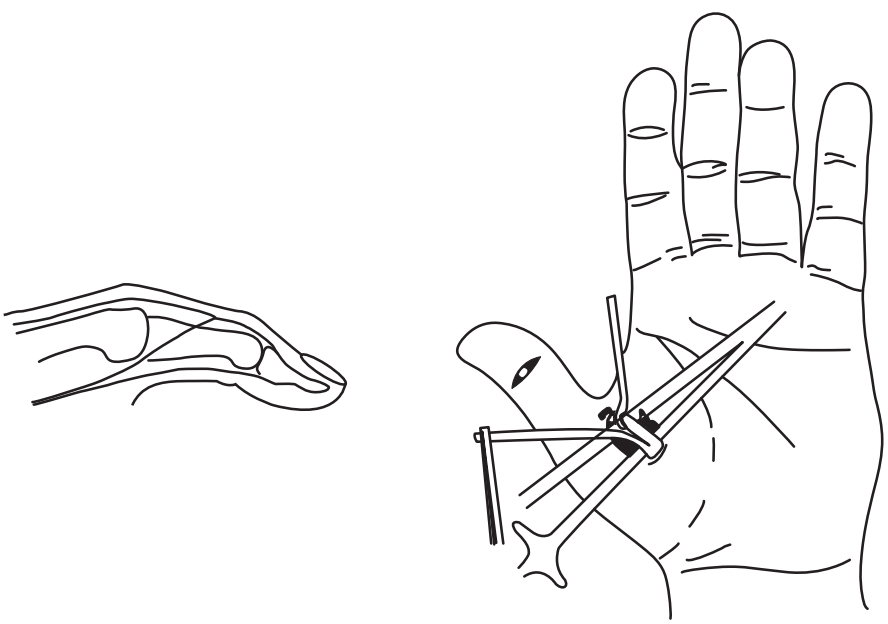


Figure 31.17
Transfer of one half of the flexor pollicis longus.

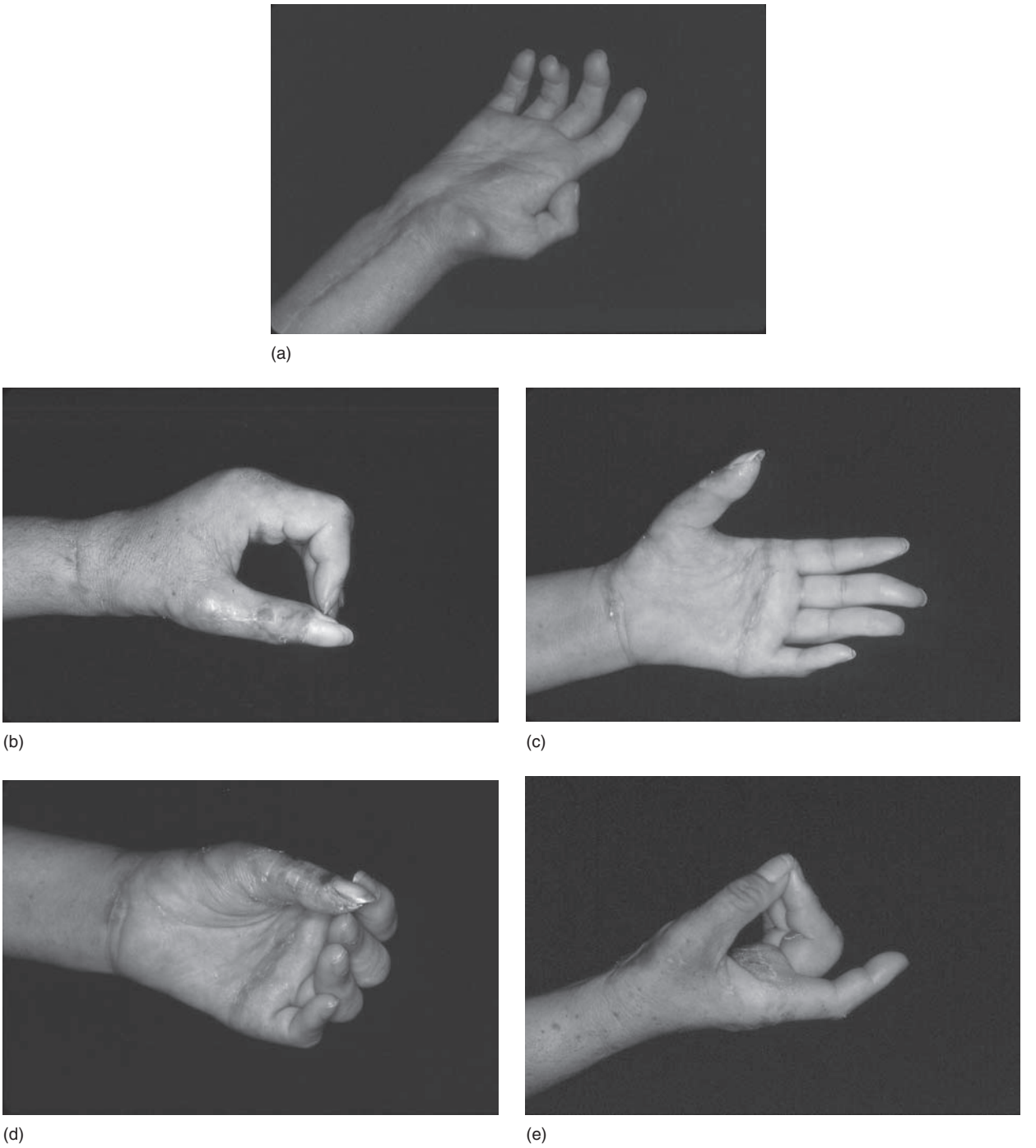


Figure 31.18

(a) Claw hand with stiffness of the thumb; (b) same patient; (c) result after the lasso procedure and transfer of the extensor indicis proprius and lateralization of the extensor pollicis longus; (d) same patient, with very good opposition; (e) same patient, other side.

paralysis, except when there is no buckling of the proximal phalanx when it is forced to resist extension. The procedure is carried out before the superficialis transfer, at the same session, thus restoring opposition of the thumb as well as stability of the proximal phalanx during resisted pinch grasp. The aim is to restore stability to the MCP–IP joint system of the thumb by stabilizing the IP joint, thus balancing the forces and preventing MCP joint hyperextension.

Indications

- presence of Z deformity either at rest or when the terminal phalanx is forced to resist extension
- when the terminal phalanx is flexed by more than 40° during a resisted pinch grasp, with or without hyperextension of the proximal phalanx (Froment's sign).

Limitations of surgery This surgical procedure stabilizes the proximal phalanx and facilitates independent flexion at the MCP joint, but at the cost of restriction of the flexion of the terminal phalanx to only 20°, which can be a disability. The advantages as well as the limitations of the procedure must be explained to the patient.

Operative procedure

- The radial slip of the flexor pollicis longus (FPL) is released by a short incision along the flexor crease of the IP joint.
- The tendon slip in the thenar region is withdrawn.
- The extensor expansion is exposed and the radial slip of the FPL is taken to the dorsum of the proximal phalanx of the thumb.
- The radial slip is fixed to the extensor expansion. Suturing is done with the wrist in neutral position, the proximal phalanx flexed by 20°, and the distal phalanx flexed by 25°. After suturing is completed, the resting position of the thumb must be 20° at the MCP joint and 25° at the IP joint.

Postoperative management The thumb must be immobilized by a plaster cast in the same position with the wrist in neutral flexion. After 4 weeks the thumb should be mobilized – at first only in active movements without resistance, but after 4 weeks the patient can increasingly use the hand in activities of daily living.

Summary

Hand and nerve surgery in leprosy is a difficult challenge that needs qualified surgeons for specific leprosy-related problems. Many procedures are available, but each surgeon, experienced with these particular problems, must choose the best procedure for the patient, with the aim always to improve the function of the patient and help their rehabilitation (Figure 31.18).

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32 Restoration of function of the upper limb after tendon transfer

Giorgio A Brunelli

Tendon transfers at the upper limb are a common type of reconstructive surgery, used when an important motor function is irretrievably lost and one or more neuromusculotendinous units, of minor functional importance, are available, to substitute for the lost function. This is often the case in:

- partial recovery of brachial plexus injuries
- severe lesions of nerve trunks (e.g., the musculocutaneous, median, radial, or ulnar nerves) incurable by nerve surgery because of avulsion of the roots from the spinal cord, a long time has elapsed since the injury, or of failure of previous nerve repair.
- loss of substance or irretrievable damage to a muscle or a group of muscles having a similar function.

Tendon transfer in these cases can restore a function that will be more or less effective according to the number and strength of the transferable muscles (be they spared by the palsy or healed by previous nerve repair). The aim of tendon transfers is:

- to compensate for the loss of movement
- to counteract the deforming forces of the antagonist muscles
- to improve the balance and stability.

When dealing with the lesion of a single nerve trunk, and we have at our disposal good donor musculotendinous units, the prognosis of the surgery is good (provided all the rules for tendon transfer are observed). If the lesion involves two main nerve trunks, the loss of function is more severe and there are fewer available muscles to transfer, whereas if more function must be restored, the prognosis is less good. In cases of partial recovery of brachial plexus injuries, the result of the tendon transfer will be fair (or poor) according to the state of relevant muscles. However, in these cases, as well as in late proximal median–ulnar nerve palsy, even if minimal muscle balance is restored, the hand will rarely be used for precision activity.

Atrophy or dystrophy of the finger pads will reduce the grip (both in power and precision). So in these cases, the aim of the surgeon will be limited to restoration of a key pinch, especially if the contralateral hand is normal. In order for tendon transfer of the hand to be possible, the following conditions should apply:

- the shoulder and elbow must have reasonable function
- the joints to be moved by the transfer should be movable and soft
- the donor unit should be good (M5 or M4+)
- the mind of the patient is normal
- the patient is not too elderly.

When planning one or more tendon transfers it must be remembered that:

1. Each musculotendinous unit has its own 'course' according to the type of the muscle: long, large, short.
2. The route of the tendon must be as direct as possible.
3. If a pulley is needed it must be firm and must not cause adhesions.
4. A transferred musculotendinous unit can supply only one action.
5. The transferred muscle will lose strength (approximately one-fifth of its strength).
6. The strength depends on the type of the muscle and particularly on its mass).
7. The bed where the tendon will lie has to allow sliding.
8. The transferred tendon has to give a new action by sacrificing its previous one, but the loss of the latter should not create a new disability.
9. In the recorticalization of the movements of the various muscles of the hand, the automatization and synkinesias that occur in the lower limb do not apply. Therefore it is possible to transfer a synergistic (or even an antagonistic) muscle that will be effective due to the selective cerebral control.

As a rule, some extension of the wrist is necessary to give strength to the flexors so that wrist extensors are synergic with flexors of the digits, whereas the flexors of the wrist are synergic with the extensors of the digits.

Any tendon transfer (as well as motor nerve transfer) needs and introduces a brain rearrangement, which is possible due to the 'plasticity' of the brain (Figure 32.1). This rearrangement requires a different amount of alteration of the brain diagram and of the effort on the part of the patient. The difference depends on the functional discrepancy of the muscles, and on the distances between the cortical areas of the donor and receiving muscles, as well as on their synergistic or antagonistic functions. The rearrangement of the brain motor pattern must restore the (motor) corporeal image by transferring to an area of the brain deprived of its effector the function of another area. However, this is not as simple as it looks. The function of a brain area is strictly related to the neighboring areas that have different functions: the areas of synergistic or antagonistic muscles that condition, check, restrict, and modulate the function of the area in question. Not only must the area of the donor muscle substitute for the area of the lost muscle, but its modulating connections must also be substituted by the connections that previously modulated the area of the missing muscle. This is only possible by means of the formation of new synapses connecting the donor area with the areas surrounding that of the missing muscle. New synapses must be intensely exerted in order to obtain their long-term potentiation and consequent new memories that will be integrated in the corporeal image and in the motor diagram. This rearrangement implies the formation of new neuronal circuits connecting neurons of areas distant from each other. These neurons have to learn to fire together like the neurons of the circuits close to the original area did before the injury.

The most difficult adjustment is that between the neurons of the new motor area with the synergistic and antagonistic neurons that previously modulated the missing muscle, that after the transfer must change their connections. This alteration is driven by repetitive inputs from the proprioceptive corpuscles of the muscle by the tactile sensations and by the visual control of the new movement.

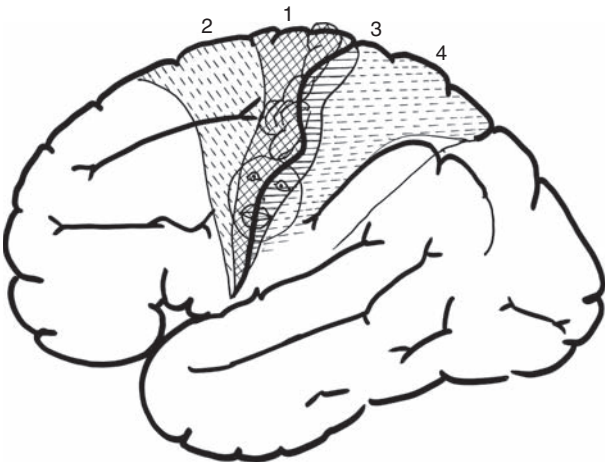

Figure 32.1

The motor and sensory areas of the brain cortex: 1, voluntary motor area; 2, psychomotor area; 3, sensory area; 4, recognition area.

In the brain cortex there are many different areas (Figure 32.2), which have been numbered by Brodman according to their cellular architecture, the differences in which imply different functions. Thus the sensory somatic areas (3,2,1) form and stimulate the cells of the motor area (4), but the responses to the sensory inputs are modulated by special modulatory areas: 5 for manipulation, 17,18, and 19 for visual control, and 7 for integration of the visual and proprioceptive inputs and for sending this to the supplementary motor area (6). The conscious commands for the response of the motor area come from areas (8) 9, 10, and 11 (multimodal association areas), which integrate in their pattern the memory information coming from areas 20, 21, and 22 that store new memories by means of the unavoidable connections with the hippocampus. Functional MRI shows the simultaneous or subsequent activation of these areas in normal brain.

When a tendon transfer is performed, the location of the cortical motor area is shifted to a new place (Figure 32.3) and all the modulating connections have to be shifted as well (Figure 32.4). In children and young patients, the brain plasticity is greater, i.e., the functional pattern of the cortex is more easily changed, and the firing of motor neurons for different tasks is easier and quicker. The same brain rearrangement is necessary for successful motor nerve transfer.

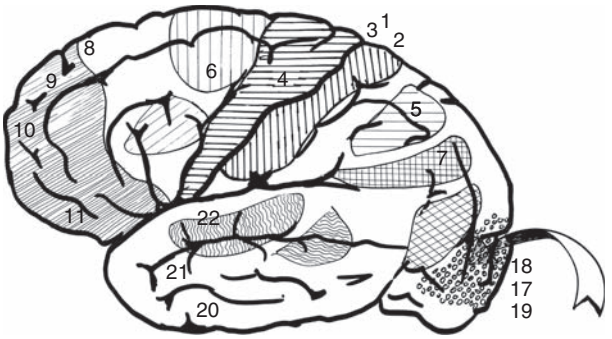

Figure 32.2

Associative areas: interpretation of some of Brodman's areas. In the brain cortex different associative areas can be distinguished. Every input received by the primary somatosensory area (1,2,3) or stimulus to the primary motor area (4) is subject to connections with other areas that recognize and elaborate them and send back the response to the sensory or motor area or to other associative areas for further elaboration. The association areas are area 5 that modulates the manipulative ability; area 7 that integrates the proprioceptive inputs with the visual ones coming from areas 17,18, and 19, and relays this to the primary and supplementary motor areas (4,6). Area 6 is for supplementary motion modulation. Areas 9,10, and 11 are for multimodal associative processes and for ideation of movements. Areas 20, 21, and 22 are involved with the memory through their connections with the hippocampus. New memories established by means of long-term potentiation of new synapses are fundamental for the patient to integrate the new function of the transferred musculotendinous unit into his motor pattern.

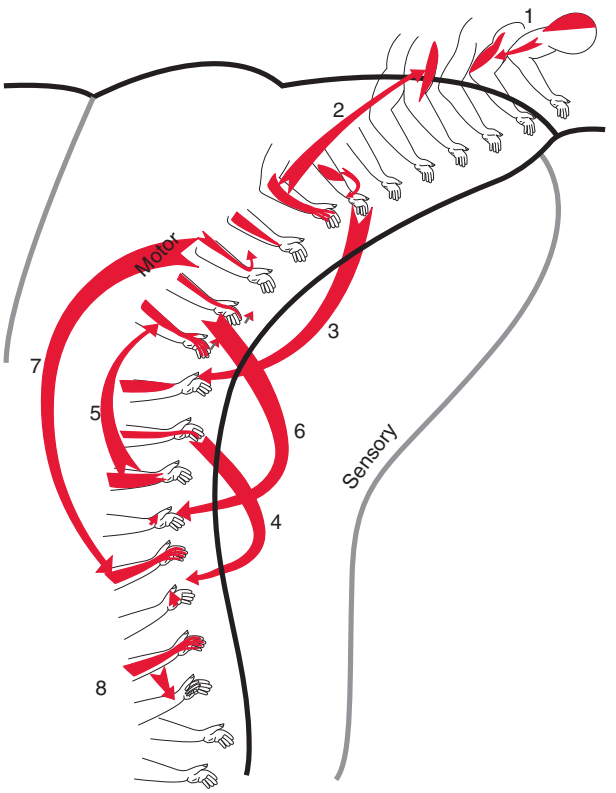

Figure 32.3

Arbitrary example of the displacement of the commanding areas of the motor cortex after tendon or nerve transfers: 1, The deltoid commands the function of the triceps. The neurons firing for the new function (triceps) are different from the original ones. 2, The epitrochlear muscles substitute for the function of the biceps and brachialis muscles. 3, After the transfer, the pronator teres area commands the function of the extensors of the wrist. 4, The adductor pollicis is activated by the area of the FDS of the ring finger. 5, The extensor digitorum communis is activated by the area of the FCR. 6, The opponens pollicis is activated by the EIP. 7, The FDPs are activated by the ECRL. 8, The interossei are commanded by one or more areas of FDS. (See also Figure 32.4.)

If the transfer of a motor nerve is possible (because the receiving muscle is still trophic and shows fibrillation due to stretching by antagonists or to electrotherapy) after the rearrangement of the brain cortex the results will be better (at least from the theoretic point of view). In fact, the impulses coming from the new 'commander area' reach the original muscle that has the same mass, course, strength, and direction and will not be hindered by adhesions. If the pad of the reanimated digit (particularly the thumb and index finger) is insensate, a sensory nerve transfer or a sensory island flap will greatly improve both the function and its re-education.

Re-education is at least as important as the surgery in the final result. Postoperative treatment cannot finish by demonstrating to the patient that, in attempting to move the donor muscle, the receiving one contracts! As mentioned above, to have a valid function this must become an integrated part of a coordinated functional pattern, including various sensory, ideatory, and modulating motor centers.

First, assisted movement must be repeated many times a day with increasingly complex tasks, and the patient must pay attention to the tactile, proprioceptive, and visual sensory feedback. The patient then continues to practice until the movement is performed easily and with minimal attention. However, if another flexor tendon is used for flexor tendon transfer, rehabilitation will be almost spontaneous and will not require a special effort from the patient. The movement is activated by an area of the brain close to and functionally similar to the missing area, and it can become selective without requiring special attention from the patient due to subconscious learning of the brain. However, in contrast, considerable initial concentration by the patient is required for the transfer of antagonists.

As operations are now becoming increasingly sophisticated, patients are becoming more and more demanding, and surgeons are increasingly under threat of legal action, the aid of a psychologist may be useful in certain cases.

A surgeon should do his best to explain the rearrangement phenomena to his patient, making him understand the rules and the tricks of re-education. In the case of severe lesions in particular, where the transferred muscle was less than M4+, the psychologic assistance of the surgeon is paramount both to teach the patient what to do and to help him to be satisfied with a partial result.

We should not forget that, even if the patient has been warned that the lesion is severe and that a modest result is anticipated, after surgery he will often expect a perfect result!

Experience-based forecasting for different tendon transfers

Transfers aiming for flexion of the thumb and fingers

The donor musculotendinous units most commonly used are the ECR, the ECU, or both. If the transferred units are M5, the ECR is passed across the membrane through a large 'window', the tension given to the transfer is correct, and the ECR animates the FDP of the fingers whereas the ECU passed around the ulna animates the FPL, then the results are very good. However, if these conditions are not all met then the results are less good, as, for instance, when the ECR is transferred to the FDP around the radius.

With regard to the alteration of the brain cortex pattern, this transfer is one of the best because the extensors of the wrist are synergic with the flexor digitorum muscles. The same reasoning is valid for the transfer of the FCR and FCU to the EDC and EPL.

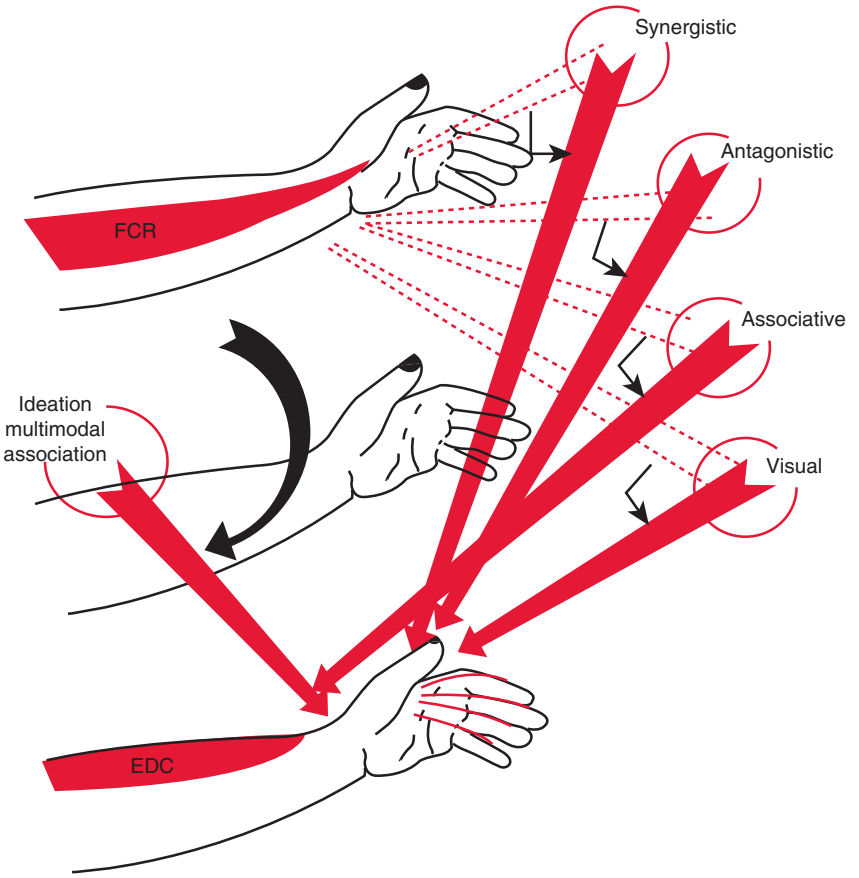

Figure 32.4

Arbitrary explanation of the alterations of the modulatory connections that have to occur after the transfer of every single function. This figure illustrates transfer of the FCR for the function of the EDC.

Transfers aiming to restore opposition-abduction of the thumb

Techniques with this aim are numerous. In principle, if the new motor has an individualized function, has a strength of M5, is brought to the thumb in the correct direction, is sutured with a correct tension on the dorsal aspect of the MPJ of the thumb (also to provide pronation), then the result is generally very good. It will be less good if its strength is M4 and if it is combined with other transfers.

Results are always poorer whenever more than one or two functions are looked for. This is for two reasons: the hindrance of the tendons that cross over each other and the inherent difficulty for the patient's brain to learn two different new movements with all their modulatory connections.

Transfers for thumb adduction

Such transfers may also be done with different motors and different techniques. The best results are given by those using a single motor for adduction alone (or those aiming to restore adduction simultaneously with flexion of the finger, because the grasping ideation involves both flexion of the digits and adduction). The brain pattern for adduction will be more easily restored if the flexor digitorum superficialis tendon of the long or of the ring finger, passed deep to the FDP, is used.

Reconstructive surgery for the interossei

Reconstructive surgery for the interossei due to irreparable ulnar nerve palsies also has an easier brain pattern integration if flexor digitorum superficialis tendons are used, as the MPJ flexion is part of the pattern of grasping so that the ideation of flexing the PIPJ is easily transferred to that of the MPJ flexion. This does not mean that transfer of motors that originally have a different function does not work. However, a greater effort for re-education will be necessary, and the results will be less good, especially in adults.

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Restoration of Function in Upper Limb Paralyses and Muscular Defects

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The definitive study of the surgical management of muscular defects and neurobiological disorders of the upper extremity, this textbook details the restoration of essential functions in this area, whatever the etiology of the disability. With a lead editor who has devoted a lifetime to surgery of the hand and upper extremity, and contributions from experts across the globe, *Restoration of Function in Upper Limb Paralyses and Muscular Defects* draws on extensive practical clinical experience to present the reader with an authoritative and insightful resource for difficult but common operations.

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