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## Keywords

Cerebral palsy • Tetraplegia • Assessment • Functional deficit • Investigations  
• Surgery • Rehabilitation • Results

## Introduction

Spasticity is characterized by muscle hypertonia, caused by a hyperactive stretch reflex mechanism. It is linked to a central neurological impairment involving the pyramidal tract.

It may occur in several circumstances:

- in children, *cerebral palsy* is usually secondary to foetal or perinatal encephalopathy, and occurs less frequently than brain damage during childhood.
- in adults, it is usually related to *hemiplegia*, whether vascular (stroke) or traumatic (head injury)
- *tetraplegia* is often associated with spasticity of the lower limbs. Spastic involvement of the upper limbs is rare and is most frequently associated with incomplete tetraplegia.

Spasticity is rarely an isolated feature. The clinical picture generally includes other neurological and orthopaedic impairments which need to be carefully assessed together with the spasticity.

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## Cerebral Palsy

Cerebral palsy is a general term that includes all the sequelae of infantile encephalopathies occurring during the perinatal period, or during infancy.

In 2007, a group of experts in the field of CP gathered for an International Workshop on Definition and Classification of CP, and published the following definition of CP [1]: “Cerebral palsy describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing foetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication and behaviour, by epilepsy, and by secondary musculoskeletal problems”

Since Little [2], it was thought to be mostly linked to perinatal asphyxia, although recent studies indicate that this factor is responsible for only 5–10 % cases of cerebral palsy. Other causes may be related to the foetus itself (gestational age at birth, birth weight, growth restriction), or to the mother (neurologic disorders, infertility treatment, antepartum infection, thyroid disease) [3].

Postnatal acquired cerebral palsy accounts for 15 % cases, and is mostly due to meningoencephalitis, head injury, and cerebro-vascular accidents.

Infantile cerebral hemiplegia is the main type of cerebral palsy. It is characterized by unilateral cortical and subcortical involvement, particularly in the pyramidal tract, which causes various degrees of spasticity and motor deficit 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 in selected cases.

#### Clinical Pearl

Cerebral palsy encompasses a group of conditions which have resulted in the permanent disorder of the development of movement and posture, resulting in a diminution of function. They are non-progressive, but can also be associated with disturbances of sensation, perception, cognition and communication, as well as behaviour.

## Clinical Examination

The clinical picture may vary greatly from one individual patient to the other, depending on the amount and location of the initial brain insult.

Clinical examination is a critical part of the assessment. Its goal is fourfold:

- Evaluate spasticity.
- Evaluate possible muscle contracture and joint deformity
- Evaluate motor and sensory impairment in the upper limb.
- Evaluate existing function, and functional needs of the upper limb.

It is completed by a general examination in order to seek associated neurological disorders, and potential contraindications to surgery.

The data are recorded on standardised charts, which will allow intra- and inter-comparisons of the results of surgical treatment.

This examination is lengthy, and requires detailed knowledge of neurology, paediatrics, and orthopaedics.

It is best performed as a team, including all the specialists involved in the child's care (physical therapist, occupational therapist, paediatrician 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 cooperation 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 capacities, and cannot cooperate throughout the entire examination. Therefore it is not wise to decide on surgery after a single session, and assessment should be repeated before any decision making.

Video recording of each clinical session is most helpful, both for initial evaluation, for decision-making, and for evaluation of surgical outcome.

## Resting Posture of the Upper Limb

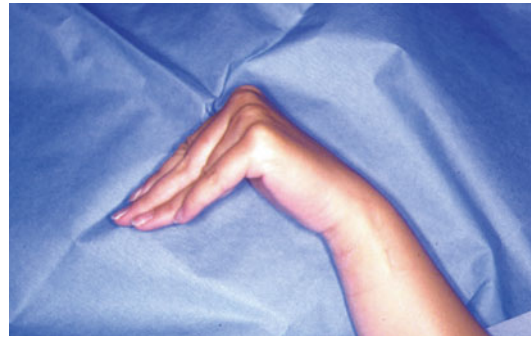
Inspecting the limb at rest prior to examination provides much information on spasticity. It usually predominates in the adductor, flexor and pronator muscles, leading to a typical resting posture in shoulder adduction and internal rotation, elbow flexion, forearm pronation and wrist flexion and ulnar deviation (Figs. 13.1 and 13.2).

The fingers may assume varied positions. Most frequently they are clenched into a tight 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), from spasticity of the interossei muscles (intrinsic swan-neck), or a combination of both.

The fingers may also occasionally assume an "intrinsic plus" deformity with flexion of the metaphalangeal (MP) joints and hyperextension of the interphalangeal (IP) joints, which is due to



**Fig. 13.1** Usual deformity of the spastic upper limb involving adduction and internal rotation of the shoulder, and flexion of the elbow, wrist and fingers



**Fig. 13.3** Spasticity of the interossei muscles: the “intrinsic-plus” deformity



**Fig. 13.2** Flexion and marked ulnar deviation of a spastic wrist

spasticity or contracture of the intrinsic muscles both (Fig. 13.3), 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 boutonniere deformity is less common.



**Fig. 13.4** Spastic adducted thumb

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 or even the middle finger, with the MP and IP joint extended (Fig. 13.4). 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.

Any factor that aggravates spasticity will increase these deformities.

### Evaluation of Spasticity

Spasticity is a muscle hypertonia, characterised by five classic clinical features:

1. It is selective. Predominantly involving the flexor, adductor and pronator muscles and

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 attempt is stopped. However if the opposing force is maintained long enough, the deformity 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 less frequent in the upper limb.
5. There may be an associated synkineses, described as ‘the phenomenon whereby paralysed 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 fingers extensors and abductors (Souques synkinesis).

It is assessed for each muscle or muscle group and is infrequent around the shoulder. At the elbow, it usually involves predominantly the biceps and brachialis muscles, but also the brachioradialis to a lesser degree. The triceps, classically spared, can occasionally be spastic.

Wrist flexors and forearm pronators are most frequently involved, leading sometimes to an extremely hyperflexed (100°) and hyperpronated (150°) position.

Spasticity is not easy to assess in the fingers because of the wrist deformity. It usually involves the finger flexors, and to a various degree, the interossei muscles.

In the first web space and the thumb, spasticity involves not only the thumb adductor, but also frequently the first dorsal interosseous and the flexor pollicis brevis. Associated spasticity of the flexor pollicis longus leads to the classical “thumb in palm” deformity.

Spastic involvement is recorded on the standard chart for each muscle or muscle group. Its importance is assessed on a 0–5 scale [4] (Table 13.1).

**Table 13.1** Modified Ashworth scale (from Bohannon and Smith D)

Grade	Description
0	No increase in muscle tone.
1	Slight increase in muscle tone, manifested by a catch and release, or by minimal resistance at the end of the range of motion (ROM) when the affected part(s) is moved in flexion or extension.
2	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM.
3	More marked increase in muscle tone through most of ROM, but affected part(s) easily moved.
4	Considerable increase in muscle tone, passive movement difficult.
5	Affected part(s) rigid in flexion or extension

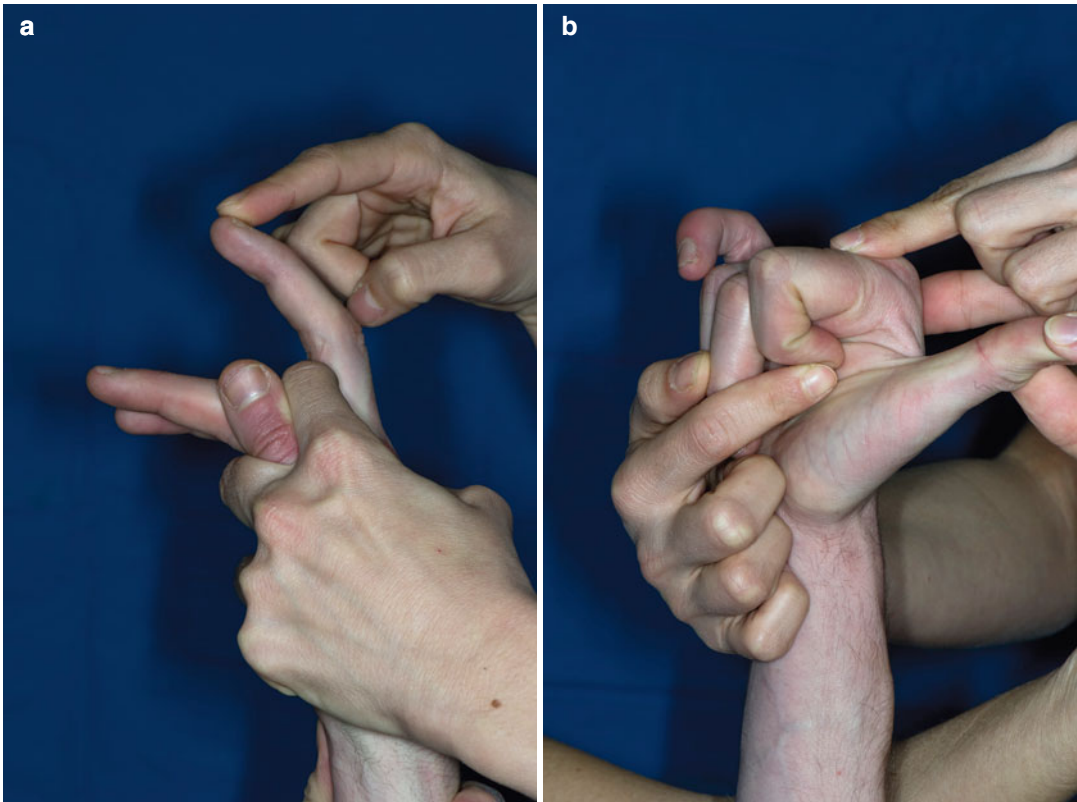
**Muscle Contracture**

Muscle contracture may involve spastic muscles. Unlike spasticity, it is permanent and cannot be overcome, although shortening the involved articular segment can alleviate it. For example posturing the wrist in flexion relieves contracture of the finger flexors. This is assessed by Volkmann’s angle, which is the degree of wrist flexion required to obtain full passive finger extension. Contracture of the intrinsic muscles of the fingers is assessed by the Finochietto test (Fig. 13.5a, b).

Clinical distinction between contracture and spasticity may be difficult to establish. In such cases nerve blocks with lidocaine are useful [5]. The anaesthetic may be injected either in the nerve trunk or in the motor point of the involved muscle(s). Spasticity yields completely whereas contracture persists [6]. Botulinum toxin yields the same result, with a much longer lasting effect.

**Joint Deformity**

Passive motion of the involved joints may be difficult to assess, not so much because of spasticity but because of muscle 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 assessment is so difficult that it is not until



**Fig. 13.5** Finochietto's test. (a) The PIP and DIP joints cannot be passively flexed when the MP joint is maintained in extension. (b) Flexion of the MP joint allows full passive flexion of the distal joints

immediate preoperative examination under anaesthesia that the actual range of passive motion can be evaluated.

Some joints of the fingers and thumb may have increased passive extension, resulting in joint instability. This occurs mainly at the thumb MP joint, and at the finger MP and PIP joints which can lead to a swan neck deformity (Fig. 13.6).

### Motor Assessment

Motor examination of the upper limb is not easy in children, especially under 5 years of age. The child should be provided with toys of different forms and colours, and be observed at play. Video recording is extremely helpful at this stage, as it avoids lengthy repetitions of tasks. Rather than individual muscles, it is easier to evaluate muscle groups contributing to the same function.



**Fig. 13.6** Spastic swan-neck deformity of the fingers

### Paralysed Muscles

The palsy (or 'pseudo palsy') usually predominates in the distal part of the upper limb, and involves the extensor and supinator muscles,



namely the 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.

Actually, rather than really paralysed, these muscles may be present, but made ineffective by the spastic antagonists.

Botulinum toxin has proved very helpful in assessing these muscles: when injected in the spastic antagonist muscles, it reduces dramatically their tone for several months, making it possible to evaluate and exercise the ‘paralysed’ muscles, which may end up demonstrating, in a number of cases, a satisfactory voluntary control.

This lack of control may also vary with limb position. For instance, voluntary movement of the thenar muscles is often facilitated by elbow extension.

### Active Muscles

The flexor, adductor, and pronator muscles, mostly spastic, usually retain some voluntary control. However, their examination is made difficult when severe deformities are present. For example, extreme flexion deformity of the wrist prevents evaluation of the strength of the finger flexors, which are mechanically shortened. If the deformity is spastic, botulin toxin may again be helpful in these cases. Synkineses and co-contractions, when present, may also complicate the motor examination. We have not found electromyographic (EMG) studies to be very helpful in evaluating either the pseudo-paralytic or the spastic muscles (see below).

Finally a **general motor assessment** is performed, in order to evaluate the global motor control of the upper limb.

Spontaneous involuntary movements are recorded: they usually indicate athetosis, which is generally a contra-indication to surgery.

Dystonia is an unadapted muscular response to command in which intermittent muscle contractions cause repetitive movements and/or abnormal postures. Again it is also usually a contra-indication to surgery.

Standard tests are performed such as the “head-to-knee” test where the patient is asked to

place his hand on his head, then to move it to the controlateral 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 classical “asymmetric neck reflex” is triggered when the head is turned actively or passively to one side, this produces abduction of the shoulder, and extension of the elbow, wrist and fingers of the ipsilateral upper limb while the controlateral limb all the joints flexed.

Once the motor examination has been completed, an attempt at classification can be made, using, for example, Zancolli’s classification [7] where:

- Type I includes the spastic ‘intrinsic-plus’ hands, in which spasticity of the intrinsic 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. Within type II there are three sub-groups classified by the degree of active finger flexion.
  - In Group 1, there is full active extension of the fingers with the wrist in neutral or near neutral.
  - In Group 2 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 Group 3 there is no active finger extension, even with maximum wrist flexion.

Goldner [8] has produced another classification in group I, 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 coordination, and minimal dexterity; in group II, there is weakness of wrist and finger extension, with a 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 stabiliser; in group III, the wrist and finger flexors are severely contracted. The primary goal of surgery is to improve cosmesis; in group IV, the hands are both spastic and athetoid.

Tonkin [9] has described for the thumb deformities, a classification of three types, modified from House [10]:

- type I (intrinsic deformity) where spasticity of the medial thenar muscles (AP, FPB, and first DIO) associated with paresis of the thumb abductor and extensor muscles cause adduction of the first metacarpal, flexion of the MP joint and extension of the IP joint.
- 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.
- 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.

Aside from Tonkin’s classification, we have not found any of the available classifications easy to utilise, 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. Moreover these classifications are not very helpful in the process of decision-making.

### Sensory Examination

Sensory examination is practically impossible before the age of 4–5 (and two-point discrimination before age 6–7). Furthermore, besides the child’s cooperation, it requires a certain level of intellectual capacities and language ability [11]. In cerebral palsy the basic sensory functions (light touch, pain, temperature) are essentially intact, while complex sensations (fine sensibility, proprioception, stereognosis) are more readily affected.

Light touch is explored using a smooth point or a finger, pain with a needle, and temperature with tubes of hot and cold water.

Fine sensibility is explored with 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. Stereognosis is tested by placing an object in the child’s hand and asking him to identify it. 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 recognise large figures drawn in the palm, and has a two-point discrimination of no greater than 5–10 mm [12].

It has been shown [13] that severely impaired sensation often goes along with upper limb discrepancy in hemiplegic CP children.

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 [14].

### Functional Assessment

A large variety of tools are utilised to assess the functional value of the spastic upper limb.

Whichever tools are used, the assessment should be video-recorded, as the videos can be viewed as many times as necessary, thus shortening the actual duration of the test.

The same tests will be repeated and recorded after surgery, and will then serve as a comparison for evaluation of the results of surgical procedures.

### Functional Tests

A great diversity of functional tests are available. Some tests are analytic, assessing a single function, other are purely functional, assessing the use of the upper limb in ecologic situations (AHA).

- The pick-up and release test evaluates not only hand prehension, but also the contribution of the whole limb to that function. Objects of

different sizes and volumes are placed in front of the child. Him/her are asked to pick them up, and then to move them to a different place. Computerised systems have been used more recently in an attempt to quantify hand grasp and release. They allow three dimensional analysis of the movement, and provide a repeatable protocol for objective evaluation of upper limb motor performance [15, 16].

- In the “box and block test”, the patient is asked to move as many wooden blocks as possible from one compartment of a box to another in 1 min.
- 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. Questionnaires are adapted to the child’s age, and 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 some potential functional capacity. In these cases, the child may persist in ignoring it even when functional ability can be improved through surgery.

There are several validated non-specific questionnaires evaluating hand function. To the best of our knowledge, currently there is no validated questionnaire adapted to the cerebral palsied child.

### Grading Scales

Many scales have been designed to quantify the functional value of the upper limb, although few are specifically designed for the spastic upper limb [8, 11]. Among them :

- Hoffer [12] tests dressing, personal hygiene, feeding, bimanual activities, grasp and release, as well as the lateral pinch.
- House provides an eight grade classification, based both on the grasping capacities, and on

the contribution of the hand to bimanual activities.

- The Shuue [17] scoring system mixes analytic measurements, and functional measurements (grasp and release, spontaneous functional analysis, and dynamic positional analysis), adding up to a numerical scoring.
- The MACS score is designed for quadriplegic patients

### General Preoperative Assessment

The aim of this general examination is to evaluate the real benefit the child could gain from surgery, taking into account other neurological impairments, the patient’s age, intelligence, motivation and environment.

### Other Neurological 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 limbs, they are usually undertaken before any upper extremity surgery.

Associated extrapyramidal signs should also be detected. These include the following:

- Athetosis, which is characterised 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 often make activities of daily living impossible.
- Parkinson syndrome is characterised 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 surgery, as the child is unable to use his hand because of these non voluntary movements.

The capacity of the child to communicate must be evaluated, seeking for visual, hearing



and language problems. Behavioural 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 functional surgery is not indicated when the IQ is lower than 70, although this is not an absolute rule, as a number of surgical procedures aimed at improving comfort, cosmesis and personal hygiene are still indicated [18].

### Age

Because the neurological deficit in cerebral palsy does not evolve, early surgery can be undertaken. Sometimes it is necessary to operate very early because of an increasing deformity. However, in most cases one prefers to wait until the child is old enough that their motor and sensory capacities can be evaluated accurately and he or she can cooperate with surgery and more importantly postoperative rehabilitation.

In the adult, the surgeon should be more cautious when recommending surgical intervention, as many of these patients have often adapted functionally and socially to the handicap and surgery may be a bit more detrimental than beneficial.

### Motivation and Environment

Any assessment of motivation should take into account the patient's ability to understand the goal, the modalities and the expected benefit of the proposed treatment, and to participate actively in the postoperative regimen. Understanding and motivation from the child's parents are also mandatory. Environmental factors during the surgical period are also important, such as a rehabilitation centre with an integrated school system and access to physiotherapists experienced in treating children with cerebral palsy.

According to Tonkin [19], "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, although satisfactory views may not always be obtained when there is a severe deformity such as wrist hyperflexion. Contralateral views in the same position may be helpful.

They may reveal growth disturbances of the distal radius, ulna, carpus and occasionally avascularity of the lunate [14], or dislocation of the radial head [20].

EMG studies provide information on the spastic muscles (voluntary control, phasic control), as well as on possible co-contractures of the antagonist muscles. In pseudo-paralytic muscles, they may identify voluntary control that is not clinically detectable (because of spastic and/or retracted antagonists, joint deformity and/or stiffness). However, they do not provide quantitative information on the power of the tested muscle.

Dynamic EMG studies, although difficult to perform in young children, may be particularly useful in determining the most appropriate donor muscles when planning a tendon transfer. Most of the potential donors are spastic to some degree. They can be utilised only if they are capable of relaxation at rest or during the antagonist movement (phasic control). A muscle that fires continuously is not a good candidate for use as a transfer [21, 22].

### Clinical Pearl

Surgical intervention should only be undertaken after a thorough and if necessary sequential evaluation by all clinicians, including the surgeons, occupational and physiotherapists, neurologists, etc. The use of video recording is often helpful.

The use of dynamic EMG's and particularly botox as a diagnostic tool can be extremely useful.

The ideal candidate is a cooperative 6 year old child, who has a predominantly spastic upper limb deformity, with satisfactory hand sensibility and with little other significant neurological deficit.

## 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 and follows a flaccid phase. There are usually few active muscles, and the wrist is often paralytic, both in flexion and in extension. Sensation is often severely impaired in the hand, involving mainly deep sensation and stereognosis. It can persist despite dramatic motor improvement, thus improving functional recovery. Trophic changes, such as reflex sympathetic dystrophy and vaso-motor changes, are frequently associated. These features, are, usually a contraindication to surgery. However, procedures aiming at reducing spastic contractures, and improving hygiene and nursing may be helpful in some cases.

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## Head Injury

The initial trauma may have involved various portions of the brain and cerebral trunk and the clinical features will vary accordingly. Thus the clinical picture is extremely varied in head injury patients. Motor impairment depends upon the extent of brain damage. It may regress rapidly in some patients, or remain in others.

Other neurologic disorders are often predominant, e.g., cerebellar syndrome or frontal impairment. Many of which contra-indicate surgical attempts at improving function to the upper limb.

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## Tetraplegia

Spasticity in tetraplegic patients usually occurs in the lower limbs. According to Zancolli [7], 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 [23]

Spasticity, when moderate, does not interfere with surgical rehabilitation of the tetraplegic

upper limb. When it is significant, it causes deformities which must be corrected prior surgery. When it is predominant, tendon transfers may be impossible [24].

## 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 consists primarily of physiotherapy and splinting, occupational therapy, and pharmacological treatment as needed.

Any decision-making should include the patient and his/her family, as well as all the physicians and care-givers 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 earlier in the patient's life, preferably during childhood [19]. Later on, the patient can develop 'actions or tricks' allowing them to undertake various activities. Any surgery whilst undertaken with the best intention can result in deterioration in function. Another advantage of performing surgery in children is that there is some evidence that improved use of an extremity can improve cortical representation of the extremity and hopefully decrease the development of neglect. Beach et al. evaluated 40 patients with CP who underwent a tendon transfer to improve wrist extension (FCU to ECRB) with an average follow-up of 5.2 years, and found that the best results were seen in patients who had the transfer between the ages of 7 and 12 years [25]. In addition, early surgery may decrease the formation of contractures.

## Rebalancing the Forces

The goals of surgical treatment can vary greatly, depending on the extent of the initial cerebral trauma. Whenever possible, it is to improve function. In some cases, however, it will be limited to improving nursing and comfort, or to correct a severe deformity.

In any case, the surgery will have to address all the deforming causes, in order to rebalance the forces exerted around the involved joint.

Three types of procedures may thus be indicated, in isolation or together:

1. Those which aim at reducing spasticity
2. Those which aim at reducing muscle and/or joint contracture
3. Those which aim at reinforcing paralysed muscles

## Reducing Spasticity

### Local Pharmacological Agents

Besides systemic medications such as Baclofen, which are used in severe and generalised 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 difficult cases to differentiate between spasticity and contracture [6].

Alcohol blocks have also been widely used in the past [26, 27]. Injected either into the nerve trunk or the motor point of the involved muscle(s), they would produce a reduction of spasticity lasting up to several months and even longer if the antagonist muscles were active [5].

Lastly, phenol, which is more effective, may be toxic for the surrounding tissues, and must be applied surgically within the epineurium [28].

Botulinum toxin A is a neurotoxin produced by the bacterium *Clostridium botulinum*. When injected into a muscle, it blocks the release of acetylcholine at the neuro-muscular junction, resulting in the denervation of the involved muscle. This denervation is dose-dependant, and reversible. Its effect starts 10–15 days after the injection, is maximal at 2–3 months, and usually lasts for 4–6 months.

Used initially for blepharospasm and strabismus, it is now routinely used in spastic limbs [29, 30] with measurable and reproducible effects in cerebral palsied children [31, 32]. Charts are available which indicate the effective dose for each age group and for each individual muscle [33].

While yielding the same result as the previously mentioned agents, it is much easier to use, because it is injected into the muscle body instead of the motor point, which often proves

difficult to locate. Canulated stimulation needles or ultra-sound localisation have rendered the injection even easier and more effective.

### Indications

Besides its diagnostic use mentioned earlier, botulinum toxin (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 should be exercised. The stronger they get, the longer lasting the effect of BT will be. Muscle contractures and stiffness of the involved joint(s) may also be efficiently reduced 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 improve their strength accordingly, thus balancing the spasticity more effectively. 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 injections of BT. If spasticity recurs after each injection, a more definitive procedure can subsequently be performed. BT also plays, in such cases, an educational role in simulating the effect of surgery. This is usually very much appreciated by the patient, who understands exactly what to expect from the surgical procedure.

BT is also indicated in spastic upper limbs secondary to head injury, where spasticity may be temporary. It is particularly useful in spasticity of the elbow flexors which may be 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 protect and facilitate education of the transferred muscle.

## Tenotomy

Tenotomy of a spastic muscle will obviously permanently relieve spasticity. However, it will also permanently suppress muscle function. As a consequence, one must be certain that the muscle to be tenotomised would not be better employed as a transfer in order to rebalance the deforming forces.

In the upper limb, it is mostly used for the wrist flexors (FCU), and the pronator teres (PT). For the FCU, tenectomy with dissection of the fascial connections of the muscle is advocated by de Bruin et al. [34], who have demonstrated that this is much more effective than simple tenotomy in reducing the wrist flexion torque.

## Selective Neurectomy (“Hyponeurotisation”)

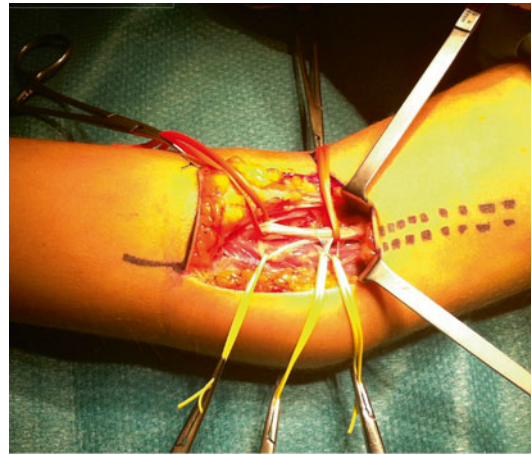
Selective neurectomy, like tenotomy, suppresses both spasticity and function.

It may be indicated in non functional upper limbs with severe spasticity, in order to facilitate hygiene and nursing, and to improve cosmesis. 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, had been suggested as early as in 1913 by Stoffel [35] This technique has gained some popularity after Brunelli and Brunelli [36] published a series of clinical cases in 1983, coining the term “hyponeurotisation”. The procedure is performed at the entry point of the nerve into the muscle, where it usually divides into three or four fine fascicles. Under magnification loops, part of the fascicles are resected (Fig. 13.7).

Brunelli initially advocated removing 50 % of the fascicles, but because he experienced some recurrence of the spasticity (“adoption” phenomenon), he then recommended resection of two thirds of the fibres.

In order to simplify the procedure, some have chosen to perform a “partial” neurectomy at the level of the nerve trunk, without approaching the target muscle(s). The motor fascicles are identified using a stimulator and partially resected.



**Fig. 13.7** Selective neurectomy of the motor branches of the median nerve to the wrist flexors and pronator teres: each motor branch is dissected down to its entry into the muscle, then partially resected

While faster and more limited in exposure, this technique is quantitatively less accurate, with potential injury to sensory fibers.

## Indications

Selective neurectomy is indicated when one wishes to reduce spasticity permanently. Resection of the motor fibres must include at least 2/3 of the fascicles, knowing that it will reduce the strength of the muscle in the same proportion as spasticity.

In our hands, this technique has proved more effective on large muscles with a single or mostly predominant motor pedicle. It has been less satisfactory in small intrinsic muscles such as the thumb adductor and first dorsal interosseous muscles.

Hyponeurotisation and partial neurotomy have no effect on muscle or joint contractures; if either are present, these must be addressed by another procedure.

## Neurosurgical Procedures

Treatment of spasticity by posterior rhizotomy was initially recorded in the late nineteenth century literature. It is now frequently used for spasticity of the lower limbs. In the upper limb, however, results have been very variable, with a number of respiratory complications [37]. Bertelli et al. [38] has recently reported a significant reduction in 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

Besides FCU and PT (see above), this technique may be useful in severe contractures occurring in a non-functional upper limb, particularly for hygiene or nursing purposes (fingers permanently flexed in a tight fist, for example).

### Muscle Release

The classical flexor-pronator release procedure, described by Page in 1923 [39], consists of releasing the proximal insertion of the wrist flexors and pronator teres muscle from their medial epicondylar origin. The muscles are allowed to slide 4–5 cm distally. It has also been recommended that reinserting them distally to the ulnar periosteum will avoid a secondary supination deformity [40].

This procedure can be extended to the finger flexor muscles, as described by Scaglietti. It is then referred to as the “Scaglietti-Page” procedure. The skin incision is extended 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 haemostasis and post-operative suction drainage mandatory.

Finally, Zancolli and Zancolli [41] has described an ingenious and more limited release of the medial epicondylar muscles consisting in a transverse resection of the inter- and peri-muscular fascia of all the involved muscles, performed 6 cm distal to the medial epicondyle (“flexor aponeurotic release”) (Fig. 13.8).

### Tendon Lengthening

Goldner [8] advocated performing a Z lengthening of each individual tendon. This procedure, simple when there is only one or a few tendons involved, becomes more complicated and time



**Fig. 13.8** Zancolli’s flexor aponeurotic release: a transverse band of the muscular fascia is resected

consuming if it is to include all the flexors of the wrist and fingers.

### Fractional Lengthening

When multiple lengthening are required, this is preferably performed at the muscle-tendon junction. This technique consists in performing multiple transverse incisions of the tendon in the area where muscle and tendon overlap (Fig. 13.8). Careful passive extension then allows the muscle fibers to lengthen, while retaining muscle-tendon continuity.

Post-operatively, no immobilisation is required, and early active motion is initiated.

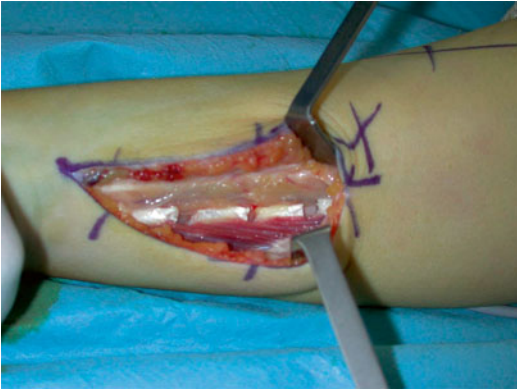
### STP

For severe finger flexor contractures, Braun et al. [42] has described an ingenious procedure of transfer of the flexor digitorum superficialis to the profundus (STP). It consists in sectioning all the finger superficialis (FDS) tendons distally at the wrist, then sectioning all the flexor profundus (FDP) 5–7 cm more proximally, extending the fingers, and suturing as a whole the proximal stump of the FDS to the distal stump of the FDP. This predictable procedure reduces finger flexors contracture and pain, while improving hygiene [43], but suppresses independent finger flexion.

### Bone Shortening

Shortening the skeleton of both bones of the forearm does reduce muscle contracture, and has been advocated in non-cooperative adults.





**Fig. 13.9** Fractional lengthening of the flexor carpi ulnaris muscle

Omer and Capen [44] performed proximal row carpectomy together with muscle transfers. This procedure reduces slightly muscle contracture, while improving wrist mobility in cases of joint contracture. It may also be indicated in those spastic patients who develop a symptomatic Kienbock's disease [14].

### Indications of Muscle Release Procedures

Mild contracture of the wrist 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 [45].

Muscle contracture involving only one tendon, or a limited number of tendons, may be treated by Z lengthening of the individual tendons. Multiple contractures of the wrist and finger flexors are best treated by fractional lengthening. This procedure has progressively replaced the classical Scaglietti-Page release, which is much more invasive, with potentially more severe complications (Fig. 13.9).

In non or poorly functioning hands where contracture interferes with whatever function remains and/or with hygiene and nursing care, the simple STP procedure is best indicated.

Indications for bone shortening are uncommon.

None of these procedures are effective in the presence of joint contracture. If, after all muscle contractures have been eliminated, there is still a limitation of passive motion, a joint procedure must be added.

### Joint Contracture

Conventional arthrolysis procedures may be required to treat the spastic upper limb, taking into account that there is often combined muscle and joint contractures, both of which need to be addressed, and that the contracture is likely to recur if either the spasticity is not relieved or if the antagonist muscles are not active (or activated).

A fixed pronation deformity may require a release of the interosseous membrane. Severe longstanding deformities in adults may only respond to osteotomy of the forearm, or arthrodesis in a more favourable position.

### Tendon Transfers

Tendon transfers are required when the paretic or paralysed muscles require augmentation. They are usually performed to improve forearm supination, wrist extension, thumb extension-abduction and finger extension.

They differ from classical tendon transfers in several ways:

- Muscles available for transfer vary with each patient. They are often difficult to select because spasticity, co-contracture, muscle weakness and lack of coordination may render individual muscle assessment extremely difficult. Careful and repeated muscle evaluation, EMG study and botulinum toxin may all be helpful with this decision.
- The transferred muscle is often spastic to some degree. It has been stressed that a spastic muscle that does not have a phasic control should not be used as a transfer [21, 22]. Dynamic EMG studies are very helpful in selecting adequate muscles.
- Tendon transfers will be successful in activating a paralysed muscle, only if the spastic and/or contracted antagonist muscles are attenuated and/or released (prior to or) at the time of 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. Any surgical planning adapts to each specific situation, bearing in mind that all

deforming elements should be treated, and preferably at the same time in order to rebalance local forces, for an optimal result.

We will describe here our most frequent procedures.

### At the Shoulder Level

Shoulder retraction in adduction and internal rotation, which impairs function of the hand should be treated. If necessary a prior injection of botulinum toxin will differentiate muscular contracture from spasticity. Muscle contracture is released by releasing subscapularis and lengthening pectoralis major (and minor if required). Postoperative physiotherapy is initiated immediately, and continued for 8 weeks at least.

### At the Elbow Level

The deforming forces at this level are spasticity and/or contracture, whereas paralysis is rare.

In head injury patients, spasticity of the biceps may be isolated and temporary. In these patients, severe muscle hypertonia may lead to early and irretractable flexion of the elbow [46]. If conservative therapy fails to improve it rapidly, botulinum toxin is indicated, followed by intensive physiotherapy combining active and passive extension of the elbow. If spasticity recurs, selective neurectomy (“hyponeurotisation”) of the musculo-cutaneous nerve is indicated. It is particularly effective in this large muscle which has a single and easily accessible motor branch [47].

In a number of cases, however, the other elbow flexors may also be spastic. Each spastic muscle must be treated, whether either botulinum or hyponeurotisation is used.

In CP patients the flexion deformity may be complex, combining spasticity and muscle (and joint) contracture of the elbow flexors, and not infrequently spasticity of the triceps muscle.

Botulinum toxin is a very effective diagnostic tool at this level: it allows assessment of the degree of spasticity of each individual elbow muscle. EMG studies by Keenan et al. [48] have shown that even brachioradialis may be severely affected.

Spasticity leads to impaired elbow flexion not only during voluntary movements, but also while standing, walking or running. It is also

frequently perceived as cosmetically unacceptable by children and their families. If targeted botulinum toxin is effective, selective neurectomy usually leads to a satisfactory permanent result.

If the contracture 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 myo-fasciotomies at the muscle-tendon junction if the contracture is mild [49]. If it is severe, a long Z plasty of the biceps tendon is more effective, requiring postoperative immobilisation for 4–6 weeks.
- the brachialis is lengthened by multiple transverse fasciotomies, after dissection and protection of the vascular bundle [50].
- the brachioradialis may require release from its proximal insertion, after dissection and protection of the radial nerve and its motor branches.
- 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, however, may be disappointing in the long term, especially if the triceps muscle is weak, or has a poor voluntary control.

Tendon transfers are very seldom indicated at the elbow, and only for the triceps muscle. The choice of donor muscles is large, as many proximal muscles are usually active.

In non-functioning limbs with a severe elbow deformity, a simple procedure, such as a neurectomy of the musculo-cutaneous nerve [51], or a biceps tenotomy, extended as required to the other elbow flexors, usually improves elbow extension immediately by 40° [46]. Successive postoperative plaster casts usually further improve the result.

### At the Forearm Level

A symptomatic pronation deformity can be improved surgically. Available procedures include:

- selective neurectomy or tenotomy of the pronator teres
- lengthening of the pronator teres if the muscle is contracted, with release of the interosseous membrane when required
- tendon transfer, usually by pronator rerouting [52, 53] if the supinators are paretic or paralysed
- rotational osteotomy of the forearm bones if the deformity is fixed.

Gschwind and Tonkin [54] have established a classification of pronation deformities and a proposed surgical plan for each group:

- Group I, where there is active supination beyond neutral, does not require surgery
- Group II, where active supination is limited to neutral or less, 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 (rerouting), or the FCU
- In 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 [45]

### At the Wrist Level

The wrist is the most frequent site of surgical treatment in the spastic upper limb. Here again, the deformity occurs as a result of a combination of spasticity and muscle contracture of the wrist and finger flexors, and muscle imbalance due to paresis or paralysis of the antagonist wrist extensors.

If there is no muscle contracture (i.e. full passive extension of the wrist and hand), botulinum toxin is injected in the spastic muscles, 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, then selective neurectomy of the spastic muscle together with tendon transfer to the paralysed

extensors (usually to the ECR muscles) is an option. 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 muscles group should be treated as described earlier. It has been our experience that this release usually attenuates spasticity of the involved muscles and as a consequence there is usually no need for any complementary procedures aimed at reducing spasticity.

Paralysis or paresis of the wrist extensors is treated by tendon transfers, usually involving the ECR muscles. The FCU (“Green transfer”) is the most appropriate muscle when it demonstrates adequate relaxation at rest (phasic control), provided the FCR is active. Other motors may include brachioradialis [55], pronator teres or a finger flexor superficialis.

The ECU is often paretic, although when the flexion deformity of the wrist is severe, it may sublux volarly, and become a wrist flexor. Rerouting it dorsally and radially will help rebalance the wrist, by decreasing ulnar deviation, and enhancing wrist extension.

In cases of a non- or poorly functioning hand with a severe wrist flexion deformity, fusing the wrist may be a reasonable option for improving cosmesis and nursing care [56, 57]. The procedure involves bone shortening, usually through a proximal row carpectomy, or a dorsal wedge osteotomy, and wrist and finger flexors lengthening as required, in order to avoid a permanently clenched fist. Van Heest and Strothman [58] recommended to use plate and screws, although Hoffer and Zeitzew [59] obtained the same rate of fusion with K wires.

Mid-carpal fusion is an interesting option when the deformity is not too severe and one wishes to retain some mobility in the wrist joint.

### At the Fingers Level

Flexion contracture of the fingers should be treated in conjunction with the wrist deformity.

Isolated spasticity of the finger flexors theoretically responds to either botulinum toxin or selective neurectomy. 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 fractional lengthening, which has supplanted the muscle slide procedure (Scaglietti). By means of its weakening effect, it is also indirectly effective on spasticity of the finger flexors, and we now tend to use it more frequently in this indication. STP is effective in poorly or non-functioning hands for cosmetic or nursing purposes. Carlson's experience [60] is that fractional lengthening of the digital flexors will be sufficient if the fingers can be extended fully with the wrist in the flexed position. Otherwise, if the fingers cannot be extended fully, then a superficialis to profundus transfer is indicated. Release of the finger flexion deformity may unmask an intrinsic spasticity, which will require additional treatment.

Tendon transfers to augment active finger extension are not frequently indicated, although there is no consensus in the literature [61, 62]. It was formerly recommended [63] 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. The generalised use of botulinum toxin has rendered this precaution unnecessary in most cases.

#### 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, it can be corrected surgically.

If the swan neck is due to muscle imbalance, a combination of intrinsic muscle spasticity and over activity of the finger extensors, this may be corrected by one of the following procedures: tenodesis of the flexor superficialis [64], tenotomy of the central band of the extensor tendon [63], lateral band tenodesis as performed by Littler and Cooley [65] or by Zancolli, spiral oblique ligament reconstruction [66].

In spastic patients, one must be extremely careful not to overcorrect the deformity, as this may lead to a flexion contracture.

#### 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 [7].

If the swan-neck deformity is severe, IP joint hyperextension must be treated as described above at the same time as the intrinsic release. Severe spasticity of the intrinsic muscles can be addressed by neurectomy of the motor branch of the ulnar nerve in Guyon's canal [67].

#### At the Thumb Level

Available procedures aimed at rebalancing the thumb, by means of reducing spasticity, include releasing the contracted muscles, stabilising thumb joints, and augmenting paretic or paralytic extensor muscles by tendon transfers.

#### Reduction of Spasticity and Muscle Contracture

As mentioned earlier, nerve procedures are not reliable enough when addressing intrinsic muscle spasticity.

An isolated contracture of the adductor muscle may be corrected by distal tenotomy at the sesamoid level, associated with a stabilisation of the MP joint to avoid the development of an hyperextension deformity. Most frequently, however, there is a combined contracture involving other thenar muscles (APB, FPB), the first dorsal interosseous together with the adductor muscle. In such cases, Matev [68] described an extended palmar release including adductor pollicis, flexor pollicis brevis, and the distal two-thirds of abductor pollicis brevis. The first dorsal interosseous muscle may also be released as required through the same incision. This procedure is technically demanding as one must release these muscles completely while protecting their motor branches [69].

When the flexor pollicis longus is contracted, it can be released by a fractional lengthening in the forearm.

Muscle contracture, especially in cerebral palsy, may be associated with a skin contracture of the first web space. This is treated by a Z plasty.

#### Joint Stabilisation

MCP joint hyperextension deformities can be treated by either a volar tenodesis or a capsulodesis, although both of these have a tendency to slacken with time. As a consequence, a sesamoid-metacarpal fusion, as described by Zancolli, or a simple MCP joint fusion may be more appropriate, particularly in severe cases.

### Tendons Transfers

Tendons transfers are necessary when thumb extension-abduction is paralysed or weak. Suggested motors include the extensor carpi radialis if the wrist extensors are spared [68], brachioradialis [64, 41], flexor carpi radialis [70] or a flexor superficialis [71].

Inglis suggested rerouting the APL through the APB in order to increase thumb abduction.

Rerouting the EPL so as to change its adduction component into one of abduction, was performed by Manske through the first dorsal compartment, whereas Carlson used a pulley harvested from the most volar slip of the APL

Tonkin et al. [9] advocated a specific treatment for each group (see above for classification):

- type I (intrinsic deformity): treated using a combination of adductor/FPB release, and first dorsal interosseous release if necessary, rerouting EPL to EPB, BR transfer to APL, and stabilisation of the MP joint by either capsulodesis, sesamoido-metacarpal, or MP fusion as required.
- type II (extrinsic deformity): treated by a FPL tendon slide, together with a release of the other contractures as required.
- type III (combined deformity): treatment includes the same procedures as in Type I, associated with a FLP tendon slide, and IP joint fusion in recalcitrant flexion deformities.

#### Clinical Pearl

The fundamental of any surgical treatment for spasticity is in the first instance to reduce the spasticity, then address either muscle or joint contracture, followed by rehabilitation and strengthening, particularly of the antagonist muscles.

The fundamental of any surgical treatment for spasticity is to reduce the spasticity, and address at the same time all deforming forces, including muscle or joint contracture and muscle weakness, followed by rehabilitation and strengthening, particularly of the antagonist muscles.

### Conclusion

Not many spastic patients are candidates for surgery of their upper limb, because of the many other neurological problems frequently associated.

Surgery should be decided upon only after several examinations of the upper limb. One should also take into account the other neurological 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, and augmentation of weak or paralysed muscles, together with the stabilisation of unstable joints. It is best performed early in CP children. Finally, there is no such thing as "standard procedures" in this group of patients, because each case is different.

### References

1. Rosenbaum P, Paneth N, Leviton A. A report: the definition and classification of cerebral palsy. *Dev Med Child Neurol Suppl.* 2007;109:8.
2. Little WJ. Course of lectures on the deformities of the human frame. *Lancet.* 1843;41:350-4.
3. Lawson RD, Badawi N. Etiology of cerebral palsy. *Hand Clin.* 2003;19:547-56.
4. Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. *Phys Ther.* 1987;67:206-7.
5. Roper B. Evaluation of spasticity. *Hand.* 1975;7:11-4.
6. Braun RM, Mooney V, Nickel VL. Flexor-origin release for pronation-flexion deformity of the forearm and hand in the stroke patient. *J Bone Joint Surg.* 1970;52A:907.
7. Zancolli E. Surgery of the hand in infantile spastic hemiplegia. In: Zancolli EA, editor. *Structural and dynamic bases of hand surgery.* 2nd ed. Philadelphia: JB Lippincott; 1979. p. 263-83.
8. Goldner JL. The upper extremity in cerebral palsy. *Orthop Clin North Am.* 1974;5:389-414.
9. Tonkin MA, Hatrick NC, Eckersley JRT, Couzens G. Surgery for cerebral palsy: part 3: classification and operative procedures for thumb deformities. *J Hand Surg Br.* 2003;26B:465-70.
10. House JH, Swathmey FW, Fidler MO. A dynamic approach to the thumb-in-palm deformity in cerebral palsy. *J Bone Joint Surg Am.* 1981;63A:216.
11. Tardieu G. *Le dossier clinique de l'IMC: méthodes d'évaluation et applications thérapeutiques.* 3rd ed. Paris: Masson; 1984.



12. Hoffer MM. The upper extremity in cerebral palsy. AAOS Instruct Course Lecture: 133. 1979a.
13. Van Heest AE, House J, Putnam M. Sensibility deficiencies in the hands of children with spastic hemiplegia. *J Hand Surg Am.* 1993;18A:278–81.
14. Leclercq C, Xarchas C. Kienböck's disease in cerebral palsy. *J Hand Surg Br.* 1998;23(B):746–8.
15. Fitoussi F, Diop A, Maurel N, el Laassel M, Pennecot G. Kinematic analysis of the upper limb: a useful tool in children with cerebral palsy. *J Pediatr Orthop B.* 2006;15:247–56.
16. Butler EE, Ladd AL, Louie SA, LaMont LE, Wong W, Rose J. Three-dimensional kinematics of the upper limb during a Reach & Grasp Cycle for children. *Gait Posture.* 2010;32:72–7.
17. Davids JR, Peace LC, Wagner LV, Gidewall MA, Blackhurst DW, Roberson WM. Validation of the Shriners Hospital for Children Upper Extremity Evaluation (SHUEE) for children with hemiplegic cerebral palsy. *J Bone Joint Surg Am.* 2006;88A(2):326–33.
18. Mital MA, Sakellarides HT. Surgery of the upper extremity in the retarded individual with spastic cerebral palsy. *Orthop Clin North Am.* 1981;12:127.
19. Tonkin MA. The upper limb in cerebral palsy. *Curr Orthop.* 1995;9:149–55.
20. Sneineh AKA, Gabos PG, Miller FMD. Radial head dislocation in children with cerebral palsy. *J Pediatr Orthop.* 2003;23:155–8.
21. Hoffer MM. Dynamic electromyography and decision-making for surgery in the upper extremity of patients with cerebral palsy. *J Hand Surg Am.* 1979;4:424.
22. Van Heest AE. Functional assessment aided by motion laboratory studies. *Hand Clin.* 2003;19:565–71.
23. Maury M, Audic B, Guillaumat M, Francois N. L'évolution du traitement de la spasticité dans les lésions médullaires. VIème Congrès International de Médecine Physique, vol. II. Barcelone: 1972. p. 543–49.
24. Hentz VR, Leclercq C. Surgical rehabilitation for the unusual, incompletely injured tetraplegic patient. In: Hentz VR, Leclercq C, editors. *Surgical rehabilitation of the tetraplegic upper limb.* London: W.B.Saunders; 2002. p. 211–4.
25. Beach WR, Strecker WB, Coe J, Manske PR, Schoenecker RL, Dailey L. Use of the green transfer in treatment of patients with spastic cerebral palsy: 17 year experience. *J Pediatr Orthop.* 1991;11:731–6.
26. Tardieu G, Tardieu C, Hariga J, Gagnard L. Treatment of spasticity by injection of dilute alcohol at the motor point or by epidural route. *Dev Med Child Neurol.* 1968;10:555–68.
27. Carpenter EB, Seitz DG. Intramuscular alcohol as an aid in management of spastic cerebral palsy. *Dev Med Child Neurol.* 1980;22:497–501.
28. Braun RM, Hoffer MM, Mooney V, et al. Phenol nerve block in the treatment of acquired spastic hemiplegia in the upper limb. *J Bone Joint Surg Am.* 1973;55(A):580–5.
29. Das TK, Park DM. Botulinum toxin in treating spasticity. *Br J Clin Pract.* 1989;43:401–2.
30. Memin B, Pollak P, Hommel M, Perret J. Traitement de la spasticité par toxine butulique. *Rev Neurol.* 1992;148:212–4.
31. Koman LA, Mooney JF, Smith BP, et al. Management of spasticity in cerebral palsy with botulinum A toxin: report of a preliminary randomized double blind trial. *J Pediatr Orthop.* 1994;14:299.
32. Fehlings D, Rang M, Glazier J, Steele C. An evaluation of botulinum-A toxin injections to improve upper extremity function in children with hemiplegic cerebral palsy. *J Pediatr.* 2000;137:331–7.
33. Graham HK, Aoki KR, Autti-Ramo I, et al. Recommendations for the use of Botulinum toxin type A in the management of cerebral palsy. *Gait Posture.* 2000;11:67–79.
34. deBruin M, Smeulders MJC, Kreulen M. Flexor carpi ulnaris tenotomy alone does not eliminate its contribution to wrist torque. *Clin Biomech (Bristol, Avon).* 2011;26:725–8.
35. Stoffel A. Treatment of spastic contractures. *Am J Orthop Surg.* 1913;10:611.
36. Brunelli G, Brunelli F. Partial selective denervation in spastic palsies (hyponeurotization). *Microsurgery.* 1983;4:221–4.
37. Fraioli B, Nucci F, Baldassarre L. Bilateral cervical posterior rhizotomy for severe spastic syndromes with dyskinesias. *Appl Neurophysiol.* 1977;78:26–40.
38. Bertelli JA, Ghizoni MF, Frasson TR, Borges KSF. Brachial plexus dorsal rhizotomy in hemiplegic cerebral palsy. *Hand Clin.* 2003;19:687–99.
39. Page CM. An operation for the relief of flexion contracture in the forearm. *J Bone Joint Surg.* 1923;5:233–4.
40. Braun RM. Stroke rehabilitation. In: Green DP, editor. *Operative hand surgery.* New York: Churchill Livingstone; 1982. p. 195–211.
41. Zancolli EA, Zancolli Jr ER. Indications opératoires et traitement de la main spastique infantile. *Ann Chir Main.* 1984;3:66–75.
42. Braun RM, Vise GT, Roper B. Preliminary experience with superficialis to profundus tendon transfers in the hemiplegic upper extremity. *J Bone Joint Surg Am.* 1974;56(A):466–72.
43. Keenan MA, Todderud EP, Henderson R, et al. Management of intrinsic spasticity in the hand with phenol injection or neurotomy of the motor branch of the ulnar nerve. *J Hand Surg Am.* 1987;12A:734–9.
44. Omer GE, Capen DA. Proximal bone carpsectomy with muscle transfers for spastic paralysis. *J Hand Surg Am.* 1976;1:197–204.
45. Gschwind CR. Surgical management of forearm pronation. *Hand Clin.* 2003;19:649–55.
46. Hoffer MM, Waters RL, Garland DE. Spastic dysfunction of the elbow. In: Morrey BF, editor. *The elbow and its disorders.* Philadelphia: Saunders; 1985. p. 616–26.
47. Cambon Binder A, Leclercq. Anatomical study of the musculocutaneous nerve branching pattern: application for selective neurectomy in the treatment of

- elbow flexors spasticity. *Surg Radiol Anat*, (in press 2014).
48. Keenan MAE, Haider TT, Stone LR. Dynamic electromyography to assess elbow spasticity. *J Hand Surg Am*. 1990;15:607.
  49. Waters PM, Van Heest A. Spastic hemiplegia of the upper extremity in children. *Hand Clin*. 1998;14:119–34.
  50. Mital MA. Lengthening of the elbow flexors in cerebral palsy. *J Bone Joint Surg Am*. 1979;61A:515–522.
  51. Garland DE, Thompson R, Waters RL. Musculo-cutaneous neurectomy for spastic elbow flexion in non functional upper extremities in adults. *J Bone Joint Surg Am*. 1980;62A:108–12.
  52. Colton CL, Ransford AO, Lloyd-Roberts GC. Transposition of the tendon of the pronator teres in cerebral palsy. *J Bone Joint Surg Br*. 1976;58(B):220.
  53. Sakellarides HT, Mital MA, Lenzi WD. The treatment of pronation contractures of the forearm in cerebral palsy. *J Hand Surg*. 1976;1:79–80.
  54. Gschwind CR, Tonkin M. Surgery forcerebral palsy: part 1. Classification and operative procedures for pronation deformity. *J Hand Surg Br*. 1992;17B:391–5.
  55. McCue FC, Honner R, Chapman WC. Transfer of the brachioradialis for hands deformed by cerebral palsy. *J Bone Joint Surg*. 1970;52A:1171–80.
  56. Pinzur MS. Carpectomy and fusion in adult acquired hand spasticity. *Orthopedics*. 1996;19:675–7.
  57. Rayan GM, Young BT. Arthrodesis of the spastic wrist. *J Hand Surg Am*. 1999;24A:944–52.
  58. Van Heest AE, Strothman D. Wrist arthrodesis in cerebral palsy. *J Hand Surg Am*. 2009;34A:1216–24.
  59. Hoffer MM, Zeitzew S. Wrist fusion in cerebral palsy. *J Hand Surg Am*. 1988;13A:667–70.
  60. Carlson MG, Athwal GS, Bueno RA. Treatment of the wrist and hand in cerebral palsy. *J Hand Surg Am*. 2006;31A:483–90.
  61. Hoffer MM, Lehman M, Mitani M. Long term follow-up on tendon transfers to the extensors of the wrist and fingers in patients with cerebral palsy. *J Hand Surg Am*. 1986;11A:836–40.
  62. Tonkin M, Gschwind C. Surgery for cerebral palsy. Part 2. Flexion deformity of the wrist and fingers. *J Hand Surg Br*. 1992;17B:396–400.
  63. Smith RJ. Surgery of the hand in cerebral palsy. In: Pulvertaft RG, editor. *Operative surgery – the hand*. London: Butterworth; 1977. p. 215–30.
  64. Swanson AB. Surgery of the hand in cerebral palsy and muscle origin release procedures. *Surg Clin North Am*. 1968;48:1129–38.
  65. Littler JW, Cooley SGE. Restauration of the retinacular system in hyperextension deformities of the proximal interphalangeal joint. *J Bone Joint Surg*. 1965;47(A):637.
  66. Thompson JS, Littler JW, Upton J. The spiral oblique retinacular ligament (SORL). *J Hand Surg Am*. 1978;3A:482–7.
  67. Keenan MA, Korcchek JI, Botte MJ, et al. Results of transfer of the flexor digitorum superficialis tendons to the flexor digitorum profundus tendons in adults with acquired spasticity of the hand. *J Bone Joint Surg Am*. 1987;69:1127–32.
  68. Matev I. Surgical treatment of spastic “thumb in palm” deformity. *J Bone Joint Surg*. 1963;45(B):703–8.
  69. Withaut J, Leclercq C. Anatomy of the adductor pollicis muscle. A basis for release procedures for adduction contractures of the thumb. *J Hand Surg Br*. 1998;23(B):380–3.
  70. Inglis AE, Cooper W. Release of the flexor-pronator origin for flexion deformities of the hand and wrist in spastic paralysis. *J Bone Joint Surg*. 1966;48(A):847–57.
  71. Smith RJ. *Tendon transfers of the hand and forearm*. Boston: Little, Brown and Co.; 1987.