INTRODUCTION

Spasticity of the upper extremity in children most often is attributable to cerebral palsy (CP). It is a non progressive disorder of the central nervous system that has typical muscloskeletal manifestations in the arm and the hand. To treat the upper extremity in C.P properly it is important to have a clear understanding of the extent of the primary C.N.S. lesion, which often occurs through subspecialty consultation in multidisciplinary clinics ***(Water and Van Heest, 1998).***

Spasticity is found almost in every child with hemiplegic cerebral palsy and creates a major problem in the provision of care. Spasticity can seriously delay upper extremity motor development and function. If untreated, it results in a persistent flexion pattern in the upper limb with internally rotated shoulder; flexed elbow, pronated forearm and flexed wrist and fingers. This problem decreases the ability to use the hand and wrist in both supination and pronation. The malalignment of the joints and abnormal tone affect the quality and fine motor control needed for prehension and bilateral hand use. Early management of spasticity is therefore critical for the optimization of function ***(Yasukawa, 1990).***

Surgical procedures are not expected to transform a functionally poor palsied hand into one that approaches normal, but significant improvement in comparison with the pre-operative condition has been noted consistently ***(Goldner, 1975).***

Surgery of the spastic hand in cerebral palsy represents a very effective treatment when properly indicated and performed. Careful

examination testing and evaluation are of major importance in the selection of patients for surgical reconstruction. A reasonable degree of success can be obtained if the patients are carefully selected. Most poor surgical results are due to incorrect indication or poor execution of the surgical techniques ***(Zancolli & Zancolli, 1981).***

When considering surgical intervention for involvement of the upper extremity it is important to clearly define the therapeutic objectives and emphasize proper patient selection. The ultimate goal is an improvement in hand function. This is achieved by correcting deformity balancing muscle power, and stabilizing uncontrolled joint to obtain prehension, grasp and release. Other goals include improvement in hygiene and appearance. For achieving the best results a careful individualized preoperative assessment is crucial. This includes a thorough medical history focusing on birth and prenatal medical problems, developmental milestones, and demonstrated preoperative using of the upper extremity, and a complete physical examination, with attention to volunt ary control of pinch, grasp and release, the identification of spastic muscle units as shown by the stretch reflex and clonus, the persistence of the infantile postural reflex and the presence and degree of two-point discrimination, sterogensis, and proprioception ***(House et al., 1981).***

AIM OF THE WORK

The aim of this work is to assess the spastic pronation flexion deformity of the forearm and wrist and evaluation of the different methods of management trying to settle an optimum therapeutic approach to this problem.

CLASSIFICATION

Many classification systems have been proposed for cerebral palsy. The bases of these systems include neuropathology, aetiology, or clinical features. No system has been universally accepted because non has been able to capture the distinction between the subtypes of cerebral palsy without becoming overlay cumbersome.

One problem is that unlike adults, there is no specific correlation between the brain injury and its clinical presentation, because of the nature of the injurious agents and immaturity and plasticity of the developing brain.

The Swedish classification system, which divides cerebral palsy into spastic, dyskinetic, ataxic and mixed types is used ***(Hogberg et al., 1979).***

The most common form "spastic cerebral palsy", involves abnormalities of the pyramidal tract, manifesting as increased tone with a clasp knife quality, any voluntary muscle group theoretically can be affected, but the most common patterns are quadriplegia (27% of all cerebral palsy) diplegia (21%) and hemiplegia (21%).

Spastic diplegia implies minimal upper extremity involvement but spasticity of both lower extremities. Hemiplegia indicates involvement of one side of the body, with the upper extremity tending to be more involved than the lower extremity, quadriplegia means all four extremities are involved, lower extremity more than the upper.

The second type of cerebral palsy is dyskenitic and manifests as choreo-athetosis with variable tone or rigidity/ dystonia, in which the increased tone has a "Lead-pipe" quality. The dyskinetic form combined with the common ataxic forms of cerebral palsy population. Children with both spastic and dyskenitic features account for about 20% ***(Hagberg, 1979).***

The dyskenitic forms are classified by type of associated movement, not topography, because all four extremities are affected.

Choreo-athetosis involves slow rotatory movements, mostly involving distal muscles. The upper extremities are more involved than the lower extremities, and there is often prominent facial grimacing and or motor difficulties, these involuntary movements are accentuated by emotion, position change and volitional movement. Dystonia indicates the maintenance of a twisted, usually extended, positioning of the extremities.

The third type of cerebral palsy is the ataxic type. The patients with this type have impaired co-ordination and equilibrium and demonstrate ataxia. If they are ambulatory, they exhibit a wide base staggering gait. They are hypotonic and frequently have nystagmus and demonstrate "Post-pointing". They usually have normal deep tendon reflexes. These patients commonly have normal intelligence. This type of cerebral palsy is common.

In the mixed type of cerebral palsy, the patients have both spastic and athetoid types. The athetoid motion disorder is more common in the upper extremities, and the spasticity is more evident in the lower

extremities. It's important to recognize that each type of cerebral palsy has its own course, and the clinical presentation may evolve over time.

For example, children with spastic diplagia may be hypotonic as infants, with the appearance of increased tone later in the first year of life.

Children with dyskinetic cerebral palsy typically do not manifest choreoathetasis until the second year of life ***(Hagberg, 1979).***

PATHOLOGY

Neuropathology:

Because many cases of cerebral palsy reach middle age and die of some quite unrelated condition, pathological confirmation of the initial diagnosis is rarely obtained. It has been customary state that spasticity is the result of damage to the motor cortex or pyramidal tracts, that athetosis is due to a lesion of the basal ganglia, and that ataxia is the end result of damage to, or disease of cerebellum or of its connections.

Tremors and rigidity are though to follow minor degrees of more widespread injury to the basal ganglia. This "watertight" localization of the pathological lesion, however, can be challenged by the description of several exceptions to this route. Accurate information is being acquired slowly to show that cerebral palsy is associated with developmental defects such as the absence of the cerebellum or of the basal gangalia, and polymicrogyria. The last is a condition in which there are other neurological abnormalities in the central nervous system with visual defects, deafness and perceptual difficulties, speech problems, mental retardation and hyperkinetic behaviour ***(Tablan, 1970).***

In ***ataxic*** cerebral palsy, the most congenital ataxia have a prenatal origin that is often genetic although acquired hemorrhagic cerebellar lesions have been described. In many analyzed autopsy cases of cerebral palsy, macroscopically, cerebral palsy brains are classified into microgyria-pachygira type, thin cerebral mantle type, hydrocephalus type, and lissencephalic type. The cerebral palsy brains show narrowed white matter with dilated ventricle compared with the controls. Microscopic analysis show that ectopic gray matter and disorganization of the cerebral

cortex, the neuronal depletion. These findings suggest that some of the cerebral palsy brain resulted from disturbance of neuronal migration ***(Tsutsui and Mizutani, 1997).***

In ***diplegia*** the underlying mechanism are those of periventricular leukomalacia and periventricular haemorrhagic venous infarction. The diplegic syndromes are characteristically seen in infants born prematurely with haemorrhagic infraction of the periventricular areas being seen especially in the increasing numbers of very preterm survivors. Radiological support for this concept is available from magnetic resonance imaging studies ***(Kodea; Guganuma; Kohono et al., 1990).***

In ***tetraplegia***, the neuropathology and neuro-imaging may show a widespread multicystic encephalopathy often with cortical and subcortical atrophy and gliosis. In ***dyskinetic*** cerebral palsy, from the neuro-pathological and neuro-radiological perspectives there is ample evidence of basal ganglia pathology as being the basis of the clinical abnormalities that result. There are both basal gangalia and cortical and sub-cortical damage demonstrated with a resulting mixed clinical picture ***(Pasternak, 1993).***

The neuropathology of heamorrhagic and hypoxic-ischaemic peri-natal encephalopathies and their effects on the post-natal development of the brain has been studied in children who survived with these lesions for days, weeks, months and years and eventually developed C.P. it is emphasized that the post-natal development of the gray matter next to these lesions is altered in a specific manner. The post natal resolution (scarring) of the sub-pial haemorrhage causes structural changes in the superficial layers of the cortex and permanent leptomeninginal

heterotopia. The pyramidal cells of layers II and III whose dentrites had been partially amputated by haemorrhage became star cells.

Myopathology:

A histologic and morphometric study of spastic muscle from many "children with cerebral palsy, comparing muscle structure with the gait parameters of energy expenditure index and dynamic electro myography was performed. This study revealed that there are no abnormalities in fiber ultra-structure were found in the subjects with cerebral palsy. Children with cerebral palsy had abnormal variation in the size of muscle fibers and altered distribution of fiber types ***(Maren Padillia, 1997).***

BIOMECHANICAL CONSIDERATIONS

Movements in the region of the elbow, forearm and wrist:

Two sets of movements occur in the region of the elbow:

I- Flexion extension: at the elbow joint.

IIa- Pronation and supination: at the superior radio-ulnar joint.

IIb- Supination and pronation: at the inferior radio-ulnar joint.

Movements of superior radio-ulnar joint: ***Kapandji (1970)***

Fig. (1) the main movement is rotation of the head of the radius (1) about its axis within the fibro-osseous ring (2), formed by annular ligament and radial notch of the ulna. This movement (II) is limited by tension developed in the quadrate ligament, which therefore acts as a brake (3). The head of the radius is not quite cylindrical but slightly oval: It's great axis lying obliquely antero-posteriorly (IIIa) measures 28 mm and its short axis 24 mm. This explain why the annular cuff of the radial head cannot be entirely bone and rigid. The annular ligament, which constitute about three quarters of the cuff is flexible and allow some stretching, while holding the head in perfect fit.

Fig. (1): Re-drawn from Kapandji, I.A. (1970). The physiology of the joint 2nd edition. Churchill Livingstone, Edinburgh, London, New York (Movements at superior radic-ulnar joint).

There are four accessory movements related to superior Radio-ulnar joint:

1- The cup-shaped surface of the radial head rotate in relation to the capitulum. (Fig. 1-IV).

2- The beveled ridge of the head of the radius (2) glides in contact with capitulo-trochlear groove of the humerus. (Fig. 1-IV).

3- The axis of the radial head is displaced laterally during pronation (Fig. 1-V) because of the oval shape of the head. During pronation (b) the great axis of the radial head comes to lie transversely so that the long axis of the radius (x) is displaced laterally by a distance equal to half the difference between the two axis of the radial head i.e. 2mm. This lateral displacement has great mechanical significance. It allows room for the medial movement of the radial tuberosity, into which the biceps is inserted. (the white row 1-Ib).

4- The plane of the proximal surface of the radial head is tilted distally and laterally during pronation (VI). This is due to rotation of the radius about the ulna during pronation as follows:

   - At the beginning of pronation, i.e. while still in supination (a) The long axis of the radius is vertical and paralleled to that of the ulna.
   - At the end of pronation, the long axis now runs obliquely distally and medially. So that the plane of the radial head, which is perpendicular to this axis, is now tilted distally and laterally at an angle y with the horizontal plane.
   - During pronation, the long axis of the radius has swept over part of the surface of a cone whose axis is the same as the common axis of the radio-ulnar joints. The carrying angle of the arm, which is prominent during supination (c) become negligible during pronation (d) owing to change in direction

of the radial axis with the result that the long axis of the arm and forearm become continuos.

Movement of the inferior radio-ulnar joint: (Fig. 2)

Kapandji (1970), stated that if the ulna remains stationary and only the radius moves (Fig. 2-1). In this case (I) the axis of rotation runs through the small finger and medial edge of the ulna. This is the case when the forearm lies in contact with the table all the time during rotation.

The main movement then is rotational displacement of the lower end of the radius about the ulna. Fig. (2-II) shows supination from the neutral position (dotted line), the radius and ulna seen from below after removal of the wrist and the articular disc. Fig. (2-III) shows pronation from the position of supination (dotted line), note that the styloid process of the ulna constitutes an obstacle in the extreme position of pronation and supination.

Fig. (2): Re-drawn from Kapandji, I.A. (1970). The physiology of the joint 2nd edition. Churchill Livingstone. Edinburgh, London, New York (Movements at inferior radio-ulnar joint).

Axis of the forearm motion:

Axis of pronation-supination:

It is the line which joins the center of the radial head and the base of the styloid process of the ulna. This axis is fixed in relation to the ulna, but the axis of the forearm itself is not fixed in relation to the ulna throughout the range of pronation and supination ***(Last, 1984).***

Axis of the forearm:

Last (1984), indicated that it runs form the mid point between the epicondyles of the humerus down to the mid point between the styloid process of the radius and ulna. In pronation the axis of the forearm crosses the ulna obliquely and lies parallel with the axis of pronation-supination, it is in line with the shaft of the humerus.

Carrying or cubital angle:

Last (1984), defined it as an angle between the long axis of the humerus and the long axis of the ulna.

It is a valgus angle measuring 15-20 degrees ***(Gray, 1973) Cunningham, (1976)*** stated that the angle appears maximally in full extension and supination (Fig. 3).

Factors share in carrying angle formation are:

1- Obliquity of the distal end of the medial edge of the trochlea which lies 6mm distal to lateral edge ***(Gray, 1973).***
2- The shaft of the ulna is angled some what laterally from the line of the trochlear notch to form the carrying angle ***(Last, 1990).***

Fig. (3): Photograph from ***Last, R.J. (1990)*** (Anatomy Regional and Applied, 7th edition, English Language Book Society, Churchill Livingstone): The carrying angle of the elbow. The bones of the left upper limb viewed from in front. The axis of pronation-supination is shown; it is identical in both supination and pronation if the humerus does not move. In supiration the third metacarpallies parallel with the shaft of the humerus; the forearm itself oblique to these, making the carrying angle at the elbow. In pronation, the axis of the forearm comes into line with the shaft of the humerus and with the axis of pronation-supination; the carrying angle at the elbow apparently disappears, though in fact the ulna has not moved.

Biomechanical aspects of cerebral palsied upper limb:

The tenodesis effect of joint motion of pluri-articular muscles is of great importance. Release of grasp may be possible only if the wrist is in full palmer flexion, thus exerting a tenodesis effect on the finger extensors. To arthrodese such a wrist without first supplementing fingers extensor power by tendon transfer would be disastrous problem ***(Samilson, 1966).***

The biomechanical results of neurophysiologic change in cerebral palsy are contracture, malalignment, instability and imbalance.

Reciprocal innervation is lacking, and normal agonist antagonist relationships are lost. The loss of phasic activity and asynchronous electrical potentials in agonist and antagonist helps to explain the so-called "Spasmus mobilis" occasionally following flexor ulnaris transfer to wrist or finger extensors, where continued electrical activity in the new position results in reverse deformity ***(Cooper, 1995).***

One of the characteristics of alterations in muscle action in cerebral palsy is the observation that contracture of a muscle, obviating full passive joint motion, results in dissipation of antagonistic muscle force on joints proximal or distal to the point of contracture. For example, with wrist flexion contracture, finger extensors do not help to extend the wrist but dissipate most of their force on the metacarpophalyngeal joints, resulting in attempted hyperextension of these joints ***(Stack, 1962).***

PATIENT EVALUATION

Cerebral palsy may be defined as a non-progressive disturbance of the cerebral cortex prior to the age of two years, manifested by altered motor, sensory and frequently intellectual functions ***(Hillel Skoff et al., 1985).***

It is important to get a tough history from the child's family, teacher's and therapists for a more detailed view of the child's function ***(Waters & Van Heest, 1998).***

First and foremost consider the age of the patient under evaluation. Remember that he or she is a growing child and his or her physical, cognitive and psychosocial development issues are changing constantly. Although the general pattern of spasticity may remain the same, the degree of deformity and limitation in function may change with age. Second understand that the degree of spasticity and limitation of function may be different with each examination. In a calm setting with familiar people, the child will have less reactive spasticity and better function. There will be inconsistencies in range of motion, individual muscle strength and degree of deformity with serial examinations. Trends are clearly evident with multiple examinations however. Each examination should be thorough with documentation of:

1- General clinical evaluation.
2- Type of deformity.
3- Active and passive range of motion in all joints.
4- Assessment of voluntary ability of grasp and release .
5- Muscle strength.

6- Hand sensibility.

7- Size measurements of circumference and length of the arm, forearm, and hand.

(Waters & Van Heest, 1998)

I- GENERAL CLINICAL EVALUATION

Which should determine the following:

a- Aetiology of C.N.S. lesion:

The causes of cerebral palsy are numerous. They include insults in the prenatal and postnatal periods. The etiological diagnosis is basically made through data elicited by the clinical history ***(Taft, 1984).***

Table (1): Aetiology of cerebral palsy.

Time of insult	Causes
Prenatal: 1- 1st trimester 2- 2nd + 3rd trimester	Teratogens Genetic syndromes Chromosomal abnormality Brain malformations Intrauterine infections Problems in fetal/ placental Functioning
Labor + delivery	Pre-eclampsia Complication of labor and delivery
Perinatal	Sepsis / C.N.S. infections Asphyxia Pre-maturity
Childhood	Meningitis Traumatic brain injury Toxins

(Hagberg and Hagberg, 1984)

b- Type of neuromuscular disorder:

there are three main types of neuromuscular disorders produced by the lesion of the C.N.S.

i- Spastic or pyramidal (50%) characterized by:

1- Muscular spasticity:

The most common posture of the spastic U.L. is one of elbow, wrist and finger flexion with forearm pronation and the thumb in adduction or flexion adduction. These deformities depend on the spasticity of the extrinsic muscles of the forearm and hand and most often on that of the flexor-pronator mass, occasionally spasticity produce some internal rotation contracture of the shoulder ***(Zancoli, 1983).***

2- Synchroniaus activity or contraction:

This represent abnormal reaction of the spastic muscles during rest or when the muscles are acting as antagonists ***(Samilson; Morris, 1964).***

Antagonist muscles remain electrically active on both flexion and extension of the involved part of the limb ***(Zancoli, 1979).***

A typical finding is the persistent activity of the flexor muscles of the wrist when complete finger and wrist extension is attempted specially at the level of the flexor carpi ulnaris ***(Zancoli, 1983).***

3- Over active stretch reflex:

Hyperactivity of the normal stretch reflex is produced by failure of the normal muscle lengthening reaction on elongation by sudden stretching ***(Swanson, 1968).***

4- Hyper reflexia or increase in tendon jerks.

5- The positive hoffman reflex.

6- Diminished superficial reflexes.

7- Clonus.

8- Slowness and weakness of voluntary contraction and loss of control of fine movement.

9- Myostatic contractures:

After long standing flexion contracture, the spastic muscles have the tendency to secondary retraction owing to fibrosis ***(Swanson, 1982).***

10- Flaccid paresis or paralysis:

The extensor and supinator muscles of the hand and forearm are usually affected by flaccid paresis or paralysis according to the severity of the deformity ***(Goldner, 1975).***

ii- Extrapyramidal type or dystonic 40%:

This type of neuromuscular disorder may be represented by athetosis, ataxia, tremors and rigidity.

Athetosis: See Fig. 4.

Characterized by:

1- Abnormal, involuntary and poorly coordinated movement with varying of tension.
2- Delayed postural development.
3- Hypotonus in infancy which may change with growth to increased tonus in stress situations.
4- Decreased reflexes in infancy which may become hyperactive later.
5- No tendency to muscular contracture.

6- Absence of Hoffman reflex.

7- Frequently preserved sensibility of the hand.

(Goldner, 1975)

Tremor:

Represented by involuntary movements that follow a regular rhythmic pattern in which flexor and extensor muscles contract alternatively.

Fig. (4):: Athetotic- involuntary movements of the digits.

Ataxia:

It is characterized by disturbed balance and equilibrium, muscle incoordination and hypotonia.

Rigidity:

It is demonstrated by resistance to movement through the hand entire range of movement with no exaggerated stretch reflex ***(Zancoli et al., 1983).***

iii- Mixed type 10%:

It is a common form, most frequently spasticity is present with athetosis as characterized by the association of hypertonus and abnormal movement ***(Zancoli et al., 1983).***

c- Presence of general neurological disorder:

The general neurological disorders found in cerebral palsy include the following:

i- Speech impairment: delayed speech, aphasia, voice disorders.

ii- Visual impairment: strabismus, optic atrophy, cataract.

iii- Hearing impairment: perceptive deafness, conductive deafness, central deafness.

iv- Convulsion disorders.

v- Behavior disorders resulting from brain injury.

vi- Emotional instability.

vii- Psychological problems.

viii- Mental deficiency.

ix- Perceptual and conceptual disturbance.

(Zancoli et al., 1983)

d- Topographic involvement:

Cerebral palsy can involve four extremities as following:

i- Tetraplegia: both arms and legs.

ii- Hemiplegia: one side of the body (usually with greater involvement of the upper extremity).

iii- Diplegia: all four limbs (with lower limbs much more involved than upper limbs).

iv- Triplegia: three limbs (usually with both leg and one arm affected).

v- Monoplegia: one limb involved.

(Hagberg, 1979)

e- Evaluation of intellectual maturity by psychological testing I.Q.:

Children falling within the first standard deviation of normal are termed normal, between the first and second standard deviation are termed educable (I.Q. 50-70) and between (I.Q. 30-50) children below this level are severely or profoundly retarded. In general only those individuals with educable or higher level can expect functional results from upper extremity surgery ***(Mark Hoffer, 1982).***

II- TYPE OF DEFORMITY

The elbow and forearm:

Elbow flexion is due to spasticity of the biceps muscle. Also pronation deformity of the forearm is common and disabling in children with cerebral palsy and is caused by spasticity of the pronator teres and at times the pronator quadratus. At times it may be aggravated by lengthening of the biceps tendon for elbow flexion contracture ***(Lovell and Winter, 1986).***

The wrist and fingers:

The most common deformities of the upper extremity in spastic paralysis are those of flexion of the wrist and fingers. These deformities are usually accompanied by pronation of the forearm, flexion of the elbow, and the thumb-in-palm deformity ***(Lovell and Winter, 1986).***

III- ACTIVE AND PASSIVE RANGE OF MOTION OF ALL THE JOINTS

When evaluating motor impairment the affected limb is examined first for passive range of movement of each joint. Lack of full passive mobility precludes full active mobility. Long standing muscle spasticity with joint influence can lead to joint contractures most often seen as wrist flexion contracture. Joint hypermobility also can be seen as a result of long standing muscle imbalance hyperextensibility of the thumb metacarpo-phalengeal joint, for example, is common from a metacarpal adduction contracture and secondary compensatory M.C.P. joint hyperextension ***(Waters & Van Heest, 1998)*** see Fig. 5.

The range of movement should be recorded on the principle that the natural position equals 0º***(Waters & Van Heest, 1998)*** .

The wrist, fingers and thumb are evaluated by asking the patient to straighten his fingers and to make a fist in order to obtain a general idea of motion of all digits and to note any limited motion or abnormal attitude ***(Zancoli et al., 1983).***

Fig. (5): To test for spastic wrist flexor: flex the wrist then gradually extend: palpate contracted and spastic tendon (flexor carpi ulnaris or radialis) ***(Water and VanHeest, 1998)***.

Fig. (6a): Test for spastic and contracted flexor digitorum superficialis.

Fig. (6b): Test for spastic or contracted flexor digitorum profundus.

(Water and Van Heest, 1998)

After evaluation for fixed deformity, the limb is evaluated for dynamic deformity, as for any disorder of the motor system observing the child move the limb in purposeful activities is imperative to define the dynamic component. The specific spastic muscle. The voluntary control of the muscles, and the status of the antagonist muscles need to be documented. By observing the joint malpositioning during activities, the spastic muscles predominantly responsible for the dynamic deformity can be identified. Excessive first metacarpal adduction, for example, identifies the adductor policies as the primary spastic deforming force, excessive thumb C.M.P. flexion implicates the flexor pollicus brevis and excessive thumb interphalangeal flexion identifies the flexor pollicus longus as the primary cause of the thump- in-palm deformity ***(Water & Van Heest, 1998).***

Videotaping therapy sessions while patients perform the standardized tasks has been helpful because it lessens the stress and anxiety of performance-on-demand that children can feel with hurried examination in the physician's office. Ideally the videotaping is on a split-screen recorder with simultaneous frontal and lateral views, which provides the best three-dimensional representation of the child's upper extremity limitations and function ***(Waters & Van Heest, 1998).***

Dynamic electromyography also has been used to assess dynamic deformity in older children ***(Waters & Van Heest, 1988).***

IV- ASSESSMENT OF VOLUNTARY ABILITY TO GRASP AND RELEASE

Voluntary control of the spastic muscles and some ability to open the fingers by active wrist flexion are important to obtain good post-operative functional results ***(Swanson, 1982).***

Different tests are used to evaluate the ability to open and close the hand, and the patterns of prehension are noted:

a- The ability to open the hand is evaluated by:

i- Measuring with disks of graduated sizes- 6, 8, 10, 12, 14,16 and 18 cm.

ii- Determining hand placement over the table.

iii- Measuring the degree of wrist flexion that will enable the patient to extend the fingers.

b- Grasp and pinch patterns are studied: see Fig. 7, 8 & 9.

The ability to grasp is evaluated through the use of spherical balls of graduated sizes (5, 8, 10 and 12 cm) and cylinders of graduated sizes (3, 5, 8, 10 and 12 cm) pinch patterns including tip, pulp and lateral pinches chuck or three-digit pinch and pinch to separate fingers and evaluated with the use of different objects.

c- Speed, skill, voluntary control and coordination for prehension and function that are evaluated by the pickup test and by manipulation of different objects.

Fig. (7): Pinch testing.

Fig. (8): Grasp testing.

Fig. (9): Pinch; left; thumbs and index extended. Right; thumb and index flexed. These represent terminal positions for digits when executing this pattern of movement.

The use of manipulative toys in evaluating children can help the surgeon to make a decisions on the indication of re-constructive procedures to improve both function and cosmetics. Coordination between both hands is observed and recorded ***(Zancoli et al., 1983).***

Classification of voluntary grasp and release patterns:

The classification of patterns of hand deformity and of voluntary grasp and release patterns of the fingers can help the examiner to determine a program of treatment and it's prognosis ***(Goldner, 1975).***

The degree of sensory impairment may vary considerably from one pattern to another:

Pattern I:

The fingers can actively extend with the wrist in less 20° of flexion. This represents a mild deficiency to open the fingers and the appearance of the extremity is satisfactory. These patients can benefit from reconstructive surgery. Particularly if the proprioceptive sensation is good.

Pattern II:

The wrist and fingers are maintained in flexion active finger extension is possible only with more than 20° of wrist flexion.

These are sub classified into two groups according to their ability to voluntary extend the wrist when the fingers are flexed.

Pattern II-A:

The patient have positive voluntary extension of the wrist if the fingers are flexed; the extensors of the wrist are active and have good voluntary control.

Pattern II-B:

The subject is unable to extend the wrist when the fingers are flexed; the extensor of the wrist are very weak or paralyzed.

Pattern III:

There is a severe flexion deformity of the hand. There is no active finger extension even with maximal wrist flexion and there is no active wrist extension even with the finger flexed. Passive extension of the fingers and thumb is possible when the wrist is flexed. Hand sensibility is usually poor in these cases it is very difficult to obtain a satisfactory hand function and prognosis after surgery is poor ***(Zancoli, 1987).***

V- MUSCLE STRENGTH OF GRASP AND PINCH

- The grip strength is evaluated with a dynamometer.
- A pinch meter is used to evaluate pinch strength.

(Waters and Van Heest, 1998)

The Elliott and Connolly classification of hand movements (1984) is a very recent innovation, based upon intrinsic movements of the digits in the hand. The descriptive terminology is appealing e.g. “pinch”, “dynamic tripod” (holding a pencil), squeeze (compressing the barrel of a syringe) “twiddle” (rolling a nut on a threaded bolt), “rock” (holding a thin disc in the palm and rotating it), “radial roll” (as in snapping the thumb), “index roll” (rolling a pea-sized object between the pulp of the thumb and the index finger), plus combinations of manipulative movements termed “steps” and “slides” (Fig. 10).

Fig. (10a): Radial roll: in this example the thumb is abducted throughout; in other instances it may be partially adducted, consequently operating radial index more distally. Left; index less flexed, right: index more flexed.

Fig. (10b): Rotary step: a schematic representation of positions in which the digits are placed (left) and successive postures of the hand (right). Sequence (right, top to bottom) shows successive phases in clock wise rotation totaling approximately 120° of object rotation. This occurs between transitions top to center and bottom to top, as indicated by the rotatory arrows.

Fig. (10c): Dynamic tripod: Small, simultaneous flexion and extension movements of the thumb, index finger and digit manipulate pen.

Fig. (10d): Squeeze.

Fig. (10e): Twiddle: Left: thumb in full abduction. Right: thus in partial adduction, with concurrent index flexion. Extent of thumb adduction is quite variable, depending on extent movement required in alternating between postures illustrated.

Fig. (10f): Interdigital step: From center of left the object is rotated by extension of ulnar digits, especially digit 3. Flexed thumb passes under rotating object to assume its position shown left. From left to right, thumb and ulnar digits flex to grasp object and index extend and may lose contact with it. From right to center, index flexes to preserve position of object against thumb, while ulnar digits flex to reposition below object in readiness for next cycle.

Fig. (10g): Linear step: From center to left, ulnar digits extend inter-phalangeally, but flex at carpo-metacarpal joints, sliding along object. From left to right, thumb abducts to oppose ulnar digits. From right to center, index flexes to restore initiation posture.

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Fig. (10h): Palmar slide: The movement illustrated in change from left to right, involves extension of thumb and radial deviation of index, with some extension.

Fig. (10-I): Rock: illustrated with pencil held transversely in radial-ulnar axis. Movements of thumb and digit 3 are much reduced compared with movements of other digits. Left: ulnar digits relatively extended. Right: ulnar digits relatively flexed.

Fig. (10-j): Index roll: Top left: slight reciprocal flexion of thumb and extension of index, and top right: the reverse. Full roll (bottom left and right) as for index roll, but with involvement of additional digits. The object rocks about the radio-ulnar axis as a result of movement between the positions illustrated.

Fig. (10-k): Rock: Illustrated in ventral view with a patridish. Left: thumb in partial abduction. Right: thumb in partial adduction.

VI- HAND SENSIBILITY

It is recommended that sensory testing should be an integral part of therapy, because tactile sensations and streognosis are needed to successfully perform tasks requiring finger and tactilegnosis ***(Bolanos et al., 1989).***

Tachdjian and Minear performed 13 sensory tests in 96 children with cerebral palsy, 88 of whom had spastic hemiplegia, not only were sensory deficits present in 41.7% of the children tested but also a high correlation was found between the functional ability of the hand and the severity of sensory deficits. Therefore, moderate to sever motor disability was associated with sever sensory dysfunction ***(Tachdjian and Minear, 1958).***

Studies evaluating the performance of children with cerebral palsy reveal the existence of sensory deficits in 40% to 73% of patient population. In all of these studies, sterognosis, two-point discrimination and position sense were the sensory modalities chiefly impaired ***(Jones, 1989).***

Sensory assessment battery:

a- Pressure sensitivity:

This is measured using the Semmes-Weinstein monofilaments. The psychophysical method of limits is used to determine threshold, that entails the filaments being applied in an ascending and a descending order. The filament first perceived in the ascending order and the filament last perceived in the descending order were noted, when applying the filaments the hand is occluded from sight by screen. Areas tested are

distal phalanex of the thumb, little finger and index finger for 3 times each and the highest threshold obtained is scored ***(Cooper et al., 1995).***

b- Two-point discrimination:

The discriminator is used which contain fixed pairs of prongs from 1mm to 12mm apart, with the subject's vision occluded by screen. The prongs is moved along the fingers in a proximal to distal direction. One or two points is randomly applied to the skin surface and the subjects is asked to identify whether they felt one or two points the smallest distances between two points that the subjects identify without an error is taken. Areas tested include the volar surfaces of the distal phalanx of the thump, index finger and little finger ***(Cooper et al., 1995).***

c- Sterognosis:

This modality is tested via the method of visual matching different shapes (circle, triangle, square, diamond and octagon) and different objects of daily use (toothbrush, tennis ball, 4-inch comb, large cup and candy in wrapper) is presented to the subject in random order with vision occluded the subject then is asked to point to the object from a selection of drawings containing all objects the total number of objects correctly matched out of 12 was taken as the subject's sterognosis score (Fig. 11) ***(Cooper et al., 1995).***

d- Proprioception:

The child's hand is supported by the examiners hand with eyes occluded. The metacarpophalangeal joints of the thumb and index and little fingers were removed holding the proximal phalanx laterally either up or down with a maximum of 30 degrees of movement on a trail. The

subjects is asked to identify the direction of movement. The number of correct responses out of five is scored ***(Cooper et al., 1995).***

e- Directionality:

A semmes-weinstein monafilament 4 degrees higher than the pressure threshold obtained is selected and moved along the volar surface of the distal phalanx of the thump, index finger, little finger in random order if application. The direction of the monofilament movement (toward or away from the body) is required to be identified by the subject. The number of correct responses out of five is scored ***(Cooper et al., 1995).***

Fig. (11): Stereognosis test objects.

Classification of upper Extremity functional use according to ***House et al. (1981).***

Level	Category	Description
0	Does not use	Does not use
1	Poor passive assist	Uses as stabilizing weight only
2	Fair passive assist	Can hold object placed in the hand
3	Good passive assist	Can hold object and stabilize it for use by other hand
4	Poor active assist	Can actively grasp object and hold it weakly
5	Fair active assist	Can actively grasp object and stabilize it well
6	Good active assist	Can actively grasp object and manipulate it
7	Spontaneous use, partial	Can perform bimanual activities and occasionally uses the hand spontaneously
8	Spontaneous use, complete	uses hand completely independently without reference be the other hand.

At the time of evaluation the results of correction were graded based on:

Excellent **:** Category 6, 7, 8

Good **:** Category 4, 5

Fair **:** Category 2, 3

Poor **:** Category 0, 1

(Michelle Gerwin, 1999)

Gshwind and Tonkin (1992)Assessment scale of pronation deformity.

Type	*Description*
I	Active supination beyond neutral
II	Active supination to less than neutral
III	No active supination, free passive supination
IV	No active supination, tight passive supination

At the time of evaluation the result of correction of pronation deformity were graded based on the following criteria:

Excellent: Active supination to 65 degrees or more and pronation to 90 degrees.

Good: Active supination to between 35 and 65 degrees and pronation to 60 degrees.

Fair: Active supination of less than 20 degrees

(Michelle Gerwin, 1999)

Aids to assessment of upper limb:

1- Electromyographic evaluation:

The role of E.M.G. in the evaluation and planning of tendon transfer in cerebral palsy is well established. Muscle transfers that, according to E.M.G. evaluation, are in phase with recipient muscles have a better chance of performing as desired. There is evidence, however, that non-phasic muscles can be transferred and still be effective ***(Mowery et al., 1985).***

Although phasic transfers are more reliable, selected transfers of continuously firing muscles can provide improved hand and wrist function. Out of phase transfers of the flexor carpi ulnaris to the wrist extensors cause excessive wrist extension and diminished function ***(Koman et al., 1990).***

2- Myoneural block:

The use of myoneural blocks is possibly the only mechanisms that allows determination of potential function before surgical intervention. In addition, the use of long-acting neuro-muscular blocking agent may be of direct therapeutic and diagnostic benefit. Nerve blocks using appropriate, safe doses, based on the weight of the patient and employing 1% lidocaine or 0.25% bupivacaine, eliminate or weaken motor and sensory function distal to the block and allow potential antagonist capacity to be estimated. However, the duration of the block (one to six hours) limits repetitive evaluation and the ability to estimate the potential recovery of strength over time with the antagonist eliminated.

In using selective nerve blocks, the surgeon must remember that doses of lidocaine and bupivacaine have an additive effect; thus the dosage must be carefully administered. Excessive intramuscular doses of either of these agents can result in systemic toxicity ***(Koman et al., 1990).***