



Anatomical and Surgical Basis for the Treatment of Paralytic Knees

**C. Elkassimi^a, S. Rouadi^a, M. Fadili^a, E. Bachkira^{b*},
A. M. Abdullah^b, I. Jadib^b, O. Hadad^b, K. Tabbak^b,
M. A. Kharoub^b, A. Rafaoui^b, A. Messoudi^b,
M. Rahmi^b and M. Rafai^b**

^a *Laboratory of Anatomy, Faculty of Medicine and Pharmacy of Casablanca, Morocco.*
^b *Orthopedic and Traumatology Department, Casablanca Hassan II University, Pavilion 32 of CHU Ibn Rochd, Morocco.*

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

Open Peer Review History:

This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: <https://www.sdiarticle5.com/review-history/94419>

Review Article

Received: 12/10/2022

Accepted: 21/12/2022

Published: 24/12/2022

ABSTRACT

This review is to highlight the anatomical and surgical basis for the treatment of paralytic knees that lead to a grave functional handicap because of its intermediate situation in lower limb.

Paralytic knee disease affects muscular balance and osteoarticular growth to the point of sometimes compromising the possibility of standing and walking.

Combined with a good knowledge of the main etiologies (Poliomyelitis, myelomeningocele and cerebral palsy), it enables the bases of the orthopedic treatment to be defined. Consequences related to paralytic knee disease may lead to neuroorthopaedic deformities in the joints.

Orthopaedic surgical treatment of the consequences of knee paralysis is varied both in terms of the diversity of the surgical techniques that can be carried out (tenotomies, lengthenings, transfers, osteotomies...) and the patient's profile. It must essentially meet functional and not morphological.

Keywords: *Paralytic knees; disorders; joint mobility; cerebral; spinal; muscular nerve; traumatology; orthopedic.*

*Corresponding author: Email: mehdibachkira@gmail.com;

1. INTRODUCTION

The aim of this review is to highlight the anatomical and surgical basis for the treatment of paralytic knees that lead to a grave functional handicap because of its intermediate situation in lower limb. Paralytic knee disease affects muscular balance and osteoarticular growth to the point of sometimes compromising the possibility of standing and walking. Combined with a good knowledge of the main etiologies (Poliomyelitis, myelomeningocele and cerebral palsy), it enables the bases of the orthopedic treatment to be defined. Consequences related to paralytic knee disease may lead to neuroorthopaedic deformities in the joints.

A good evaluation of the causes of these deformities (spastic or retracted muscles, joint stiffness), allows the therapeutic choice to be planned. Orthopedic surgical treatment of the consequences of knee paralysis is varied (tenotomies, lengthenings, transfers).

2. PARALYTIC KNEES

2.1 General

The assessment of Paralytic knees cannot be separated neither from the neurological context, which conditions the patient's lifestyle, nor from the rest of the musculoskeletal system because of its strategic place within the lower limb.

The study of these segmental disorders is complex and must be integrated into the overall analysis of the lower limbs and the subject being examined. The elements to be taken into account are the origin of the paralysis either central or peripheral, the limitation of range of movement of the joint and growth disorder observed on each joint, and the functional impact on ambulation.

There are several etiologies responsible of knee paralysis.

In our study, we will focus on the three main aetiologies namely: poliomyelitis, spina bifida and cerebral palsy (CP).

2.2 Polio knee

2.2.1 Generalities

Acute anterior poliomyelitis is an infectious endemic disease and is caused by Picornavirus. The most commonly affected muscles in the lower limb are the quadriceps, triceps and leg muscles.

- Muscle recovery depends on 3 elements:

- the number of undamaged motor neurons initially
- the number of affected motor neurons who return to normal function
- the number of motor neurons able to develop axonal extensions that re-innervate muscle fibres [1–4].

2.3 Consequences of Paralysis [1,3,5–8]

- Paralysis of the lower motor neuron with flaccidity of the muscles and the asymmetrical muscle forces exerted on a joint are responsible of creating muscle imbalances with retractions of the stronger muscles.
- In addition to these retractile tendencies, the effect of weight bearing creates deformities in the joints with deficient muscles, particularly in the knees, feet and spine.
- These problems are far more important within childhood where growth phases can dramatically worsen the deformities.

Other key causes of contracture include gravity, articular flexions in bed and leaning on a weaker lower limb.

As a side effect of these causes, there is damage to the joints, bones and soft tissue as seen on the retracted joint capsules on the flexed side, flattened or deformed epiphysis, and retracted intermuscular partitions, nerves and vessels. At the flexed surfaces, the skin itself may show a retraction phenomenon

2.4 Poliomyelitis Knee Deformities

It is the paralysis of the quadriceps that dominates the clinical picture. The paralysis and retractions will combine to cause flessum, recurvatum, varum and valgum deformities.

2.4.1 The flessum knee

This is the most common deformity [9–11].

This deformity can either be the direct result of The retraction of the hamstrings and rarely of the gastrocnemians, or a compensation of a hip flexum or equinus of the foot.

Initially, the affected structures in a flexed knee are tendon then bones progressively due to the growth disturbances caused by this deformity.

In adults, flexion deformity of the knee can sometimes be very serious. This is usually associated with posterolateral tibial subluxation, and sometimes with external rotation of the tibial plateau, which will frequently result in a painful stiff knee.

Flexion occurs in two main situations

- If the subject walks autonomously, with a paralysis of the quadriceps, during the stance phase, the line of gravity passes behind the knee, making it necessary to stabilize the knee with the counterpart hand and flex the trunk
- In more severe flexion, the affected limb cannot support the body weight, and if this is combined with moderate paralysis of the contralateral lower limb, the individual loses the ability to walk. Knee flexion is then an obstacle to the achievement of an efficient walking device.

In all cases, correction of knee flexion is mandatory [3,12–14].

2.4.2 The recurvatum knee [10,15]

Knee recurvatum is defined by the degree of knee hyperflexion obtained beyond sagittal femorotibial alignment corresponding to 180° at baseline, combining a posterior capsulo-ligament distension and a bony recurvatum.

This deformity is the consequence of an active quadriceps associated with hamstring paralysis or, on the contrary, of a paralysed quadriceps associated with a fixed equinus. Poliomyelitis is the most common cause [16].

The initial paralysis of the quadriceps causes a recurvatum attitude when walking, which tends to increase the pressure on the anterior part of the tibial plateau due to the excess rolling of the femoral condyles, this results in damage to the tibial growth plate, a decrease in the tibial slope, and therefore a bony recurvatum. At the same time, posterior capsulo-ligament distension worsens, resulting in an increase in knee recurvatum.

2.4.3 The valgum knee [5,10,14]

It is relatively common (10 to 15 cases). It can be isolated or in association with flexum, recurvatum or external tibial torsion. The valgus knee may be static or dynamic, it may be directly related to

paralytic alterations of the knee or secondary to a deformity of the hip or foot.

In cases of total or subtotal paralysis of the knee, the dynamic valgum knee by paralysis of the internal flexors predominantly of the biceps muscle is not frequent, it is generally due to contracture of the tensor of the fascia lata, with the hip going into external rotation and abduction while the knee goes into flexion. This deformity can worsen by the contracture of the Sartorius.

The valgum knee is often the consequence of static alterations of the limb.

- a paralyzed hip is often positioned in external rotation which not only leads to its intrinsic deficit, but also to the need for the patient to ensure a wider polygon of sustentation, and if at the same time there is a paralysis of the knee, the static effort is concentrated on the medial collateral ligament which ends up distending.
- The medial space opens up while the lateral space pinches with an increase in overload, resulting in valgus.
- The valgus knee can be the compensation of a deformity of the hip or the foot. In the first case, it is the consequence of an adducted attitude of the hip. In the second case, it is the result of the patient's effort to compensate for a varus foot. As the support on the outer edge of the foot is unstable, the patient tries to set foot plantigrade on the ground and forces the knee into valgus.

The valgum knee does normally justify surgical treatment, except when it interferes with the orthopedic device.

2.4.4 The varum knee [14]

It is always observed in association with a flexion deformity. Its origin is often dynamic, due to the traction exerted by the internal flexors of the knee while the biceps is paralysed. It can also represent the compensation of a valgus of the foot.

2.5 Spina Bifida Knee

2.5.1 General

There are three anatomical lesions of increasing severity, spina bifida occulta, meningocele and Myelomeningocele, the most severe and frequent.

The lesion level corresponds to the last root responsible for muscular activity. Below the lesion level there is an absence of innervation above the lesion, there is in principle a normal spinal cord.

Gait prognosis: depends mainly on the quadriceps (L2-L3-L4), key to standing. Above L3, the quadriceps is paralysed and independent walking without aid is not possible. Below L5, independent walking is theoretically possible but sometimes difficult due to weakness of the gluteal muscles.

Between L4 and L5, the intermediate stage, the aggravation is even more marked since the quadriceps weakens as it struggles against knee flexion, a consequence of the absence of the gluteus maximus and triceps surae [10,17,18].

2.5.2 Orthopaedic deformities

They depend on the level of injury and the consequences of the paralysis on the locomotor system notably muscle imbalances aggravated by fatigue, retraction and osteoarticular growth disorder. These deformities occur mainly in the sagittal plane, and the quadriceps is an important element of the orthopaedic prognosis in this case. If its force is sufficient, it allows autonomous walking and a good locking of the knee, if on the contrary the quadriceps is weak or limited, it cannot oppose neither the automatic activity of the hamstrings, nor the weight of the body when standing up.

2.5.2.1 Flexion deformations [18–21]

They are common. It is possible to observe

- external rotation flexion of both hips and flexion of the knees secondary to spina bifida at the thoracolumbar level which, in addition to the flaccid paralysis, can generate reflex activities often involving the external rotators of the hip and the hamstring muscles.
- Valgus external rotation: The weakness of the gluteal muscles, more or less associated with that of the quadriceps, leads to a flexion attitude which begins as soon as the child starts to walk. This flexion deformity of the knee invariably leads to a valgus deviation. Patients with weakness in the hip adductors and plantar flexors of the ankle aggravate this external valgus deviation when walking.

External rotation is the consequence of a lesion which is located in particular at the L4 level. When it is bilateral, it leads to a so-called mirror deviation: valgus flexion on one side and varus flexion on the other.

2.5.2.2 Stiffness in extension

They are less frequent than bending deformations.

It may be a genu recurvatum or, more often, a stiff knee in hyperextension.

The genu recurvatum is not the main issue if the irreducible extension is very troublesome for sitting and walking. Knee extension stiffness is frequently associated with other homolateral deformities such as flexion and external rotation, dislocation or subluxation of the hip, equine varus of the foot and internal rotation of the leg. It is mainly L3, L4 paralysis with fixed knee extension.

It may be due to fibrosis of the quadriceps or bone deformities such as a malunion of a distal femoral fracture [19,21,22].

2.5.2.3 Valgus deformity

It can be observed either isolated or in association with external rotation of the tibia after retraction of the tensor fascia lata. It occurs especially in the case of injury at L1, L2 level. If this deformity hinders the fitting of orthopedic device, it may justify surgical correction [10,19].

2.6 Knee of the Cerebral Palsy

2.6.1 General

The brain injury results in variable neurological forms that greatly influence the existence of orthopaedic disorders:

- Spasticity: it is especially in this most frequent form that orthopaedic problems will arise. This term covers multiple entities: Isolated exaggeration of the stretch reflex, abnormal postural reaction, resting hypertonia, a combination of reflexes and pathological reactions, excessive resistance to stretching of both active (contractile) and passive (viscous retraction) origin. For the orthopaedic surgeon, spasticity is the opposite of athetosis as the former does not cause excessive contractions at rest.

- ✓ The diplegic form: is a particular form where spasticity mainly affects the lower limbs with flexion-adduction attitude of the hips, flexum of the knees and variable deformity of the feet (equinus valgus or talus), associated with internal rotation of both lower limbs [23,24].

Other topographic forms can be observed: Hemiplegic and quadriplegic.

The pathophysiology of the deformities is under the dependence of both the brain lesion and bone growth. Spasticity progressively leads to growth disorders of the muscles, which become short, and to osteoarticular deformities [10,25,26].

2.6.2 Cerebral palsy deformities

2.6.2.1 Genu flexum

Knee flexion is very common. The spastic state leads to a disturbance of the motor pattern with an imbalance between agonist and antagonist muscles, which is reflected in the knees by the hyperactivity of the hamstrings and the weakness of the quadriceps which struggles to maintain joint balance.

The genu flexum may also be secondary to hip flexion (insufficient gluteus maximus, hyperactivity or shortness of the rectus anterior or femoral anteversion) or to a foot deformity (equinus or talus).

If the flexum deformity is balanced through efficient hip extensors and ankle flexors, it can be compatible with independent walking.

On the other hand, if the flexum deformity is no longer balanced, it worsens because the spasticity is more significant, the hamstrings are therefore shorter, which results in an ascension of the patella and stretching of the patellar tendon [10,27,28]

2.6.2.1.1 Genu recurvatum

Less frequent than flexum deformity, it is the consequence of an importance spasticity of the quad. By Genu recurvatum, the hamstrings are too elongated and quad are shortened, which limits the passive flexion. This deformity can also be due to excessive contraction of anterior rectus associated with weak gastrocnemius muscle and flexum hip deformity [29].

2.6.2.1.2 Genu valgum/varum

It is rare because in the spastic patient the muscle imbalance is mainly in the sagittal plane with a predominance of the hamstrings. They may be secondary to abnormalities in other joints including excessive femoral anteversion and valgus instability of the astragalo-calcaneal joint [29].

3. OTHER ETIOLOGIES OF PARALYTIC KNEE

There are other causes of knee paralysis. These include

- Cerebral and spinal cord injuries with multiple aetiologies: traumatic, infectious, vascular tumour, degenerative, malformative.
- Myopathies [30]: Duchenne's and becker's dystrophies, myotonic syndromes, congenital myopathies and metabolic myopathies.
- Arthrogryposis [31]
- Isolated crural paralysis [32]

4. PRINCIPLE OF SURGICAL TREATMENT OF PARALYTIC KNEES

4.1 Orthopaedic Lengthening Procedures [9]

Different types of musculo-tendinous lengthening gestures can be proposed, the choice takes into account three elements:

- a) The importance of the elongation sought
- b) the functional or non-functional purpose
- c) the existence of antagonist muscles, their strength, their mode of expression

4.1.1 Simple tenotomy

It consists of a pure section of the tendon which can, in certain cases, be carried out under local anaesthetic when it is isolated. It allows an elongation which has no limit in the correction of the deformity, but it makes the muscle lose its functional possibilities.

It can only be proposed if the antagonists are absent.

4.1.2 Tendon lengthening

- Intramuscular lengthening: It consists of performing a tendon tenotomy at the emergence of the tendon within the muscle

fibres or myo-tendon junction, thus the elasticity of the muscle fibres allows for an elongation evaluated on average at around 15 mm. This procedure preserves all the functional capacities of the muscles, without the phenomenon of sideration, with the possibility of immediate active rehabilitation.

- Intra-tendon lengthening is based on the principle of Z-shaped tenotomies, either percutaneous or open, allowing the desired lengthening. Tenotomy-suture is preferred when the status of the antagonists remains uncertain to avoid any risk of hypercorrection.
- Muscle disinsertion The principle is to remove all muscular insertions and to ensure a mobilisation of the musculo-tendinous body while preserving the vascular-nervous bundle. The degree of lengthening depends on the possibility of mobilising the vascular-nervous bundle.

4.2 Tendon Transfers

Their purpose is to compensate for absent antagonists. There are two types of transfers: active tendon transfers and passive tendon transfers (or tenodeses).

4.2.1 Active tendon transfers

Treatment consists of transferring the distal insertion of a muscle to enable it to perform the desired movement. These transfers can only be performed if the muscles have a strength rating of at least 4.

Unlike tendon transfers in peripheral paralysis, tendon transfers in central injury are performed on muscles with impaired control. It is important that the analytical or synkinetic contraction occurs during the movement being sought.

4.2.2 Passive tendon transfers

They are based on the realization of a proximal fixation of a tendon to ensure a positional balance of the joint. The proximal tenodesis can be either osseous or ligamentary.

4.3 Osteoarticular Gestures

4.3.1 Arthrolysis

The aim is to free the joint by cutting through the capsular, ligamentary and sometimes muscular formations. They are quite rare in deformities of

neurological origin which are by nature extra-articular.

In all cases, arthrolysis must respect the lateral ligaments or the central pivot, in knee arthrolysis, to avoid joint instability.

4.3.2 Arthrodesis

According to some authors, they have no indication in the proximal joints: hip, knee. For others, however [9], arthrodesis can be proposed as a first-line treatment in a regulated surgery, to treat a paralytic knee.

4.3.2.1 There are 2 types of arthrodesis

- extra-articular arthrodesis, which has become rare, in which the joint is fused without approaching it, using a bone graft.
- Intra-articular arthrodesis, the most frequently practiced, during which the joint is approached openly for the bone elements in direct contact so that they fuse together.

4.3.3 Osteotomies

It is defined by the resection of a bony wedge. In paralytic knees, the osteotomy can be low femoral or high tibial.

Generally, it is performed at the level of the deformity itself. If the deformity does not dictate the location, it is performed at the metaphyseal level.

4.3.4 The arthroplasties

They are part of the orthopaedic possibilities for the treatment of spasticity within the framework of vicious circles associating joint arthrosis and an increase in spastic hypertonia. Associated musculotendinous or neurotomy procedures should exceptionally be performed at the same time.

5. SURGICAL TECHNIQUES

5.1 Musculo-Tendon Surgery

5.1.1 Lengthening surgery

5.1.1.1 Hamstring tenotomy [19,33–36]

The treatment of knee flexion depends on its extent. If it is moderate (below 30°), it can be corrected by re-education and orthopaedic means, posture or standing braces.

Above 30°, it may require a hamstring release.

- Technique: A tenotomy of the semitendinosus and rectus internus is performed, combined with a lengthening of the semimembranosus and, outside, a lengthening of the biceps. The types of lengthening, intramuscular or Z-shaped, depend on the amount of lengthening required. A lengthening is commonly performed when the muscle strength of the hamstrings is at least 3.

Tenotomy is performed in case of weakness of hamstrings

Sometimes an additional procedure is performed on gastrocnemius. This depends on whether they are retracted or not.

The evaluation will be possible after having extended the hamstrings at the maximum extension of the knee, to appreciate the tenodesis effect of the gastrocnemius with the appearance of an equinus. In this case, a proximal disinsertion of the gastrocnemius will be performed.

When full knee extension is not achieved, a posterior capsulotomy is performed.

In the most severe cases, a section of the posterior cruciate ligament is associated.

Postoperative immobilisation is essential to maintain the result obtained.

5.1.1.2 Re-energising the extender [10,36]

Re-tensioning of the extensor apparatus may be warranted if there is hyper elongation of the patellar tendon and a patella alta. It consists of tenotomy-resection of the rectus abdominis at the level of the anterior inferior iliac spine and plication of the patellar tendon, or distal transfer of the anterior tibial tuberosity.

- Technique: The patella is lowered so that its lower pole is level with the joint space, with knee extension. In close to or mature skeletal patients, distal transfer to the anterior tibial tuberosity with screw fixation of a bone block containing the patellar tendon is performed. In skeletally immature patients, the insertion of the patellar tendon at the inferior pole of the patella is cut and the tendon shortened (approximately 2cm).

The distal tip of the patella is excised; the tendon is then reconnected to the patella.

In all cases, it is imperative to correctly evaluate the tone of the quadriceps in order to avoid hypercorrection.

5.1.2 Tendon transfers

There are some basic common principles for successful tendon transfer:

- Each muscle to be transferred must be strong enough to do the work of the paralysed muscle properly.
- A weak muscle always becomes weaker after transfer.
- The path of the transferred muscle must be as direct as possible and as close as possible to the ideal path of the replaced muscle.
- The route must also allow for sliding and therefore use the subcutaneous fatty tissue or even the sheath of the replaced muscle. The innervation and vascularisation of the transferred muscle must not be compromised during the tendon transfer.

Transplant harvesting should at best remove a bony or cartilaginous film that will allow for a more solid anchorage after transfer [5,37]

5.1.2.1 Hamstring transfer

Transferring the hamstrings to the patella can (only partially) supplement the strength of the deficient quadriceps, provided that both the biceps and the semitendinosus are transferred for greater strength and better balance (preventing the risk of patellar decentration). The result is better if the quadriceps is not totally paralysed and if the gluteals and triceps surae provide effective additional stabilisation [8,10]

In the case of a complete quadriceps deficit, to avoid a recurrence of flexum, the medial rectus can also be transferred. The transfer does not allow the quadriceps to actively lock the knee, but rebalances the flexor/extensor tension.

In case of hip extensor deficits, the rectus femoris, semitendinosus and biceps can be transferred on the femoral condyles (Eggers' procedure) [9].

5.1.2.2 Tendon transfer of the tensor fascia lata

The tensor fascia lata has the advantage of having fibers whose direction and length are similar to those of the quadriceps as well as being synergistic.

In addition, distal disinsertion of the tensor fascia lata reduces the hip flexion often present and removes the flexor and valgator effect of this muscle on the knee.

Technique described by Riska [27]: Riska proposes another formula for using the iliotibial band, which he detaches completely from its tibial insertion and, after having detached it very high, sutures it to the patella after creating a bone tunnel through which he passes the terminal tabs.

5.1.2.3 Anterior rectus tendon transfer [27,38]

The transfer of the anterior rectus is proposed as a palliative treatment for the hyperactivity of the hamstrings due to spasticity. The aim is to reduce the flexion forces during the stance phase, and thus strengthen the movements in the sagittal plane during the swing phase.

5.1.3 Correction of knee flexion

Its correction is imperative.

The extension of the knee will thus lead to an automatic stabilisation of the joint during the stance phase by resorting to postural locking even in the absence of the quadriceps;

The body's axis of gravity will shift in front of the knee, making walking possible without the need for external stabilisation.

A Supra-condylar femoral extension osteotomy can be carried out [13,39]

In cases where the flexion is less than 30 degrees, the supra-condylar femoral extension osteotomy represents a simple and effective surgical technique, on the other hand, when the flexion is greater than 30 degrees, it requires firstly a release of the soft parts (lengthening of the hamstrings), then a slow and progressive correction thanks to the installation of an Ilizarov apparatus which allows, on the one hand, to control the tension of the posterior vascular-nervous elements and, on the other hand, to

carry out a supra-condylar osteotomy in order to gain the last degrees of residual flexum.

- Trepano-osteoclasia: It is a simpler technique and easier to perform than osteotomy. It was first proposed by Brandes in 1932 for the correction of spinal deformities; its indication was extended to deformities of other pathologies such as poliomyelitis.

It consists of performing a manual osteoclasty after having weakened the chosen area and then correcting it with a series of perforations

5.1.3.1 Correction of the recurvatum knee

The recurvatum must sometimes be respected in the case of a major quadriceps deficit; it helps to lock the knee for standing and walking. In this case, the only thing to do is to monitor the deformity and to prescribe a cast with the knee slightly flexed.

But one can also observe an irreducible extension, very awkward for the sitting position. Surgery is the rule by lengthening the quadriceps tendon. The difficulty is, here again, to maintain the result acquired by regularly mobilising the knees and alternating postures in flexion and extension. Bone interventions are still the most commonly practiced.

- Supra-condylar femoral flexion osteotomy [15,39,40]: Difficult to perform, it aims at correcting the sagittal axis defect, but also very often the associated rotation or valgus anomalies.

This is a posterior subtraction osteotomy.

- Arthroplasties: The principle of these procedures is to prevent hyperextension by means of a bone block acting much like the olecranon in the elbow.

5.1.3.2 Valgum knee correction

The supra-condylar femoral varus osteotomy, described as the simplest, is the most used technique [39].

5.1.3.3 Arthrodesis [14,41]

Knee arthrodesis represents a surgical technique that resolves, in a safer way, the problem of knee stabilisation. However, stabilisation by arthrodesis must take into account the patient's

choice, as it is sometimes more incapacitating in functional terms than a device blocked in extension for walking and locked in the sitting position.

The main indication is complete paralysis of the knee muscles.

It would also be an interesting alternative technique in cases where arthroplasty is formally contraindicated.

6. CONCLUSION

Paralytic knee disease affects muscular balance and osteoarticular growth to the point of sometimes compromising the possibility of standing and walking. The clinical picture is essentially dominated by neuro-orthopaedic deformities, the most frequent of which are flexum deformities.

A complete and precise neuro-orthopaedic examination allows the assessment of segmental disorders which must be integrated into the global analysis of the lower limbs and the subject to be examined. Combined with a good knowledge of the aetiologies, it enables the bases of the orthopaedic treatment to be defined.

Orthopaedic surgical treatment of the consequences of knee paralysis is varied both in terms of the diversity of the surgical techniques that can be carried out (tenotomies, lengthenings, transfers, osteotomies...) and the patient's profile. It must essentially meet functional and not morphological criteria.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Dizien O, Held JP. Acute anterior poliomyelitis. *Encycl Med Chir, Treaty of Neurology*. 1993;17-070-A-10.
2. Houeto JL, Gout O. Neurological manifestations of infections. *Encycl Med Chir, Treaty of Neurology*. 1999;17-049-A-10.
3. Huckstep RL. Poliomyelitis: A simple guide. World Ortho Inc; 1997.
4. Jubelt B, Agre JC. Characteristics and management of polio syndrome. *Jama*. 2000;284(4):412-4.
5. Chataigner H, Onimus M, de Billy B. *Neuromuscular diseases in children* Ed Sauramps, Montpellier; 1991.
6. Lebarbier P. The foot of the cerebral palsy: Diagnostic problems and therapeutic principles. *Cah Teaches SOFCOT*. 1996;55:159-68.
7. Giori NJ, Lewallen DG. Total knee arthroplasty in limbs affected by poliomyelitis. *JBJS*. 2002;84(7):1157-61.
8. Shahcheraghi GH, Javid M, Zeighami B. Hamstring tendon transfer for quadriceps femoris paralysis. *J Pediatric Orthop*. 1996;16(6):765-8.
9. Denormandie P, Kiefer C, Mailhan L, Even-Schneider A, Sorriaux G, MARTIN JN. Orthopedic treatment of the consequences of spasticity in the lower limb. *Neuro-Chir Paris*. 2003;49(2-3):339-52.
10. Lebarbier P. Paralytic lower limb. *Encycl Med Chir, Musculoskeletal Treatise*, 1998;14-301-A-10.
11. Onimus M, Mandaba JL. Possibilities of verticalization and walking in children in the sequelae of poliomyelitis in an African environment. *Int Orthop*. 1992;16(2):196-201.
12. Bunch W. Biomechanical principle and application. Ed. Atlas of orthotics CV Mosby. 1985:76-111.
13. Leong JC, Alade CO, Fang D. Supracondylar femoral osteotomy for knee flexion contracture resulting from poliomyelitis. *J Bone Joint Surg Br*. 1982;64(2):198-201.
14. Mezzari ML, Poliomyelitis A-La. Diagnosis and treatment of sequelae. Univ lille ii faculty of medicine 86 rue de paris, 59000 Lille, France; 1967.
15. Bussière C, Aït Si Selmi T, Neyret P. Genu recurvatum. *Encycl Med Chir. Locomotor Apparatus*. 2001;14.
16. Rainault JJ. Serious poliomyelitis knee recurvatum. *Rev Chir Orthop*. 1962;48: 561-77.

17. Samuelsson L, Skoog M. Ambulation in patients with myelomeningocele: A multivariate statistical analysis. *J Pediatric Orthop.* 1988;8(5):569-75.
18. Walter BG, Columbia, Missouri treatment of hip and knee problems in myelomeningocele.
19. Langlais J. The spina bifida knee. Childhood surgery and orthopedics, Ed Sauramps, Montpellier; 1993.
20. Mcrae R, Kinninmonth WG. Orthopedics and trauma. General topics and vertebral diseases. Ed Momento Med; 1997.
21. Wright JG, Menelaus MB, Broughton NS, Shurtleff D. Natural history of knee contractures in myelomeningocele. *J Pediatr Orthop.* 1991;11(6):725-30.
22. Brinker MR, Rosenfeld SR, Feiwell E, Granger SP, Mitchell DC, Rice JC. Myelomeningocele at the sacral level. Long-term outcomes in adults. *JBJS.* 1994;76(9):1293-300.
23. Cahuzac M. The child with cerebral palsy. Mason; 1980.
24. Sussman MD, Aiona MD. Treatment of spastic diplegia in patients with cerebral palsy. *J Pediatr Orthop B.* 2004;13(2): S1-12.
25. Khouri N. The hip of children and adolescents with cerebral palsy. *Cah Teaches SOFCOT.* 2001;78:123-36.
26. Lespargot A, Renaudin E, Robert M, Khouri N. Muscles and tendons of the IMOC: Clinical review and experimental data. *Cerebral Word Paris.* 1999;20(2):69-90.
27. Gage JR. Gait analysis in cerebral palsy. *Clin Dev Med.* 1991;121.
28. Normand X, Dubousset J. Tensioning the extensor mechanism of the knee in the triple flexion gait in children with motor disabilities. *Rev Orthopedic Restorative Surgery Appar Mot.* 1985;71(5):301-10.
29. Morrell DS, Pearson JM, Sauser DD. Progressive bone and joint abnormalities of the spine and lower extremities in cerebral palsy. *X-rays.* 2002;22(2):257-68.
30. Bonnard CH. Myopathies Current diagnostic and therapeutic classification. SOFCOT Teaching Conferences, 1991; 40:1-14.
31. Georges F. Arthrogyposes *Encycl Med Chir, Treatise on Musculoskeletal System,* 1998; 15-201-A-10. Neurosurgery, Masson Paris, 2003; 49:339-352.
32. Boulu P. Electromyography and evoked potentials in musculoskeletal pathologies. *Encycl Med Chir,* 1997;14-001-R-10.
33. Abraham E, Verinder D, Sharrard W. The treatment of flexion contracture of the knee in myelomeningocele. *J Bone Joint Surg Br.* 1977;59-B(4):433-8.
34. Keenan MAE, Ure K, Smith CW, Jordan C. Hamstring release for knee flexion contracture in spastic adults. *Clin Orthop Relat Res* 1976-2007. 1988;236:221-6.
35. Martin JN, Sorriaux G, Judet T, et al. Knee flexion by retraction of the hamstrings in brain-damaged adults: Apropos of a series of 37 patients treated by distal tenotomy of the hamstrings *Rev. Chir. Orthop.,* Masson Paris. 2003;89:3587.
36. Beals RK. Treatment of knee contracture in cerebral palsy by hamstring lengthening, posterior capsulotomy, and quadriceps mechanism shortening. *Dev Med Child Neurol.* 2001;43(12):802-5.
37. Samilson RL. Tendon transfers in cerebral palsy. *J Bone Joint Surg Br.* 1976;58(2):153-4.
38. Defebvre L, Duba F. Clinical and functional assessment of motor disability *College of Neurology Teachers,* 2003; FAQ (motor disability).
39. Lambotte JC, Langlais F. Osteotomies of the distal femur *Encycl Med Chir, Treatise on Surgical Techniques,* 1999; 44-825.
40. Mehta SN, Mukherjee AK. Flexion osteotomy of the femur for genu recurvatum after poliomyelitis *The Journal of Bone and Joint Surgery,* 1991; 73-B: 200-2021.
41. Acquitter Y, Hulet C, Souquet D, Pierre A, et al. Arthrodesis of the knee under arthroscopy.

© 2022 Elkassimi et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:

The peer review history for this paper can be accessed here:

<https://www.sdiarticle5.com/review-history/94419>