Gait Analysis

An Essential Tool in the Treatment of Cerebral Palsy

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Gait analysis has radically changed the treatment of cerebral palsy. Preoperatively, it allows critical assessment of the specific pathologies of the patient. Postoperatively, it provides an accurate assessment of outcome. This assessment of outcome has in turn allowed the accurate critique of surgeries and has made it possible to discard treatments that are not useful or are perhaps even injurious. As a result of this continual reassessment of surgical techniques, several principles and insights regarding the treatment of cerebral palsy have been learned. These include (1) the importance of reestablishing normal gait prerequisites, (2) the methods of reducing the energy expenditure of the pathologic gait, (3) the importance of skeletal structures in providing the lever arm by which muscles produce moments around joints, (4) the role and importance of two joint muscles, and (5) the importance of separating abnormalities, which are emanating from the neurologic lesion, from secondary ("coping") responses. Through gait analysis, it has become apparent that diplegia and hemiplegia are noninclusive terms, each of which contain a variety of homogeneous patterns of gait. Eventually these patterns may be separated and identified and optimal treatment protocols for each pattern type developed.

Gait analysis began in California in 1872 when Leland Stanford, then governor of California, asked Eadweard Muybridge, a noted still photographer of his day, to produce a photograph of Stanford's trotting horse, Occident. The particular picture that he wanted, however, was one of the horse at full speed with all four feet off the ground at the same time. Several years later, after Muybridge had succeeded in developing faster photographic emulsions, the feat was finally accomplished by placing a series of stationary cameras with trip wires on the track. The amazing sequence of photographs that resulted from that unusual request not only marked the beginning of gait analysis but also landmarks in film history.[4]

Since then, much of the ongoing work in gait analysis has also occurred in California, with pioneers such as Vern Inman, David Sutherland, and Jacquelin Perry. Dr. Inman's work began immediately after World War II and related mainly to normal and amputee gait, whereas the work of Drs. Sutherland and Perry has been mainly concerned with neuromuscularly handicapped children and adults. Jacquelin Perry has inspired many students from a host of disciplines that includes orthopedic surgery, physical therapy, kinesiology, and biomedical engineering.

As a result of gait analysis, the treatment of cerebral palsy has changed dramatically. Motion analysis allows a more complete assessment of the patient's pathology before and after treatment. Before gait analysis, the outcome of surgical treatment in children with cerebral palsy was modest; patients retained a spastic gait, the only change being that for better or worse they walked differently. Further, surgical outcome could not be assessed critically enough to allow consistent improvements in treatment. Now, a new approach to cerebral palsy has resulted from gait analysis

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that is less empiric, more rational, and based on the pathophysiology of the condition.

IMPORTANCE OF REESTABLISHING GAIT PREREQUISITES

There are arguably five parameters that are used to establish normal gait. These include (1) stance phase stability, (2) swing phase clearance, (3) foot preposition in terminal swing, (4) adequate step length, and (5) energy conservation. Many or all of these are missing from the typical gait pattern of a patient with cerebral palsy; however, quality surgical treatment can restore many of them. For example, a child with a severely externally rotated, pes valgus foot does not have stance phase stability, but with an appropriate subtalar arthrodesis or a derotational tibial osteotomy (or both), it can usually be restored. Similarly, appropriate surgery and/or orthotics can often improve swing phase clearance, preposition of the foot in swing, and step length. Furthermore, when all of these parameters have been optimized, energy conservation is often improved as well. Gait analysis provides an awareness of these features that can eventually allow the orthopedist to note their presence or absence simply on clinical observation of gait. However, formal gait analysis is usually required to pinpoint the cause of the abnormality.

METHODS OF IMPROVING ENERGY CONSERVATION

Researchers are now becoming very aware of the energy demands of walking and are routinely measuring these costs pre- and postoperatively via oxygen consumption testing. In general, children with cerebral palsy have walking-energy demands that are 1.5 to three times normal, depending on their degree of involvement. There are at least three ways by which energy costs can be reduced in a child with cerebral palsy: (1) reduction of cospasticity, (2) restoration of stance phase stability of the hip and knee via the ground reaction force (GRF), and (3) elimination of foot drag. There are likely many others.

In the ideal candidate, reduction of cospasticity between agonistic and antagonistic muscle groups is probably best accomplished with selective dorsal rhizotomy, although appropriate muscle lengthenings and/or transfers may reduce cospasticity to some degree. Some day this may be accomplished by means of appropriate pharmacologic agents. Currently, however, good tone-reducing drugs that do not have significant side effects are not available.

In normal gait, the quadriceps are used to stabilize the knee only during the first 20% of stance. Afterwards, the triceps surae retards the rate of forward progression of the tibia sufficiently to allow the GRF to move anterior to the knee and produce knee extension (Fig. 1). This mechanism can be lost in a number of ways, but the two principle ones are loss of stance phase stability (such as severe pes valgus in the example above) or iatrogenic lengthening of the heel cords. Once the mechanism is lost, the patient is forced to use his quadriceps to stabilize his knee joint throughout the entire stance phase of gait, which, in addition to increasing patellofemoral pressure to the point of causing pain, greatly increases the energy demands of walking (Fig. 2). Therefore, restoration of the normal mechanism of knee extension during the last half of stance is one of the primary goals of cerebral palsy treatment.

Because children with cerebral palsy have cospasticity around the knee, they usually have a rather pronounced loss of knee motion in swing. In addition, problems associated with foot clearance are often compounded by an equinus foot during the swing phase of gait. The latter problem can often be eliminated through the use of an appropriate ankle–foot orthosis. However, cospasticity often requires a combination of hamstring lengthening plus rectus femoris transfer, which converts the rectus femoris from a hip flexor–knee extensor to a hip flexor–knee flexor.[2,3]

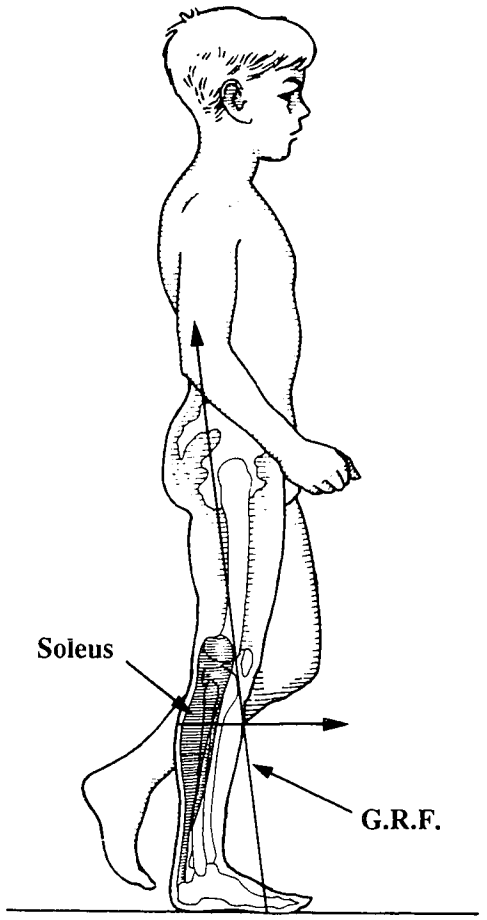

FIG. 1. Midstance. Using the soleus to slow the forward momentum of the shank, the GRF is brought in front of the knee. The GRF acting on the lever arm of the foot thereby generates an extension moment on the knee that provides the needed stability without the necessity of other muscle action. This extension moment is generally referred to as a plantarflexion/knee extension couple. (Reprinted with permission from Gage, J. R.: Gait Analysis in Cerebral Palsy. London, Mac Keith Press, 1991, p. 69.)

MUSCLE FUNCTION IN NORMAL GAIT

Physiologically, muscles can work in only three ways: as accelerators, as decelerators (shock absorbers), and as stabilizers. When a muscle acts as an accelerator, it always contracts concentrically (shortening contraction). Deceleration and shock absorption are accomplished via eccentric (lengthening) contraction. Finally, muscles acting as stabilizers act more or less isometrically (*i.e.*, they provide force without a significant change in length).

In the cerebral palsy gait, agonists and antagonists frequently contract concurrently. When this happens much of the muscle force that is produced is wasted in cocontraction, so the net accelerations generated are small. If further weakening of essential accelerators occurs during surgery, the patient may be rendered worse instead of better. For example, iliopsoas and triceps surae lengthening may cause inability to advance the swing limb or create poor foot clearance, or both, whereas excessive weakening of the hamstrings will usually cause excessive lordosis and loss of gait velocity. Therefore, it is imperative to identify the principle accelerators that are used in walking. Before gait analysis, there was really no way to do this. Through the use of kinetics (joint moments and powers), however, we can now measure the net muscle powers in the sagittal and coronal

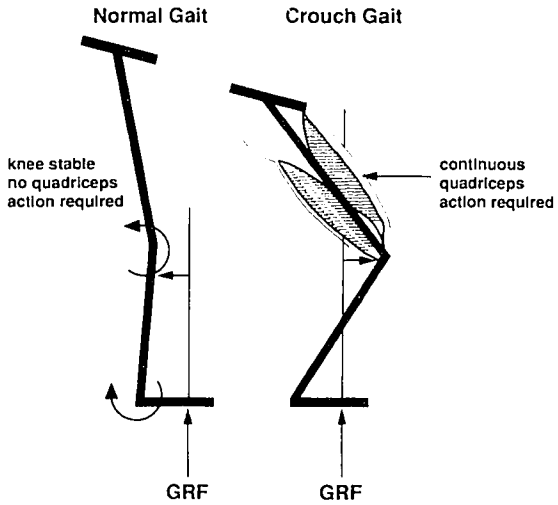

FIG. 2. Excessive knee flexion in stance. In normal gait, the knee is rendered stable in midstance and terminal stance via the plantarflexion/knee-extension couple. If there is excessive knee flexion in midstance, the GRF moves behind the knee and generates a flexion moment that must be resisted by an internal (muscle) moment. The quadriceps are the principal muscles employed, but hip extensors can assist to some degree.

planes at the hip, knee, and ankle (Fig. 3). In the normal gait, the triceps surae supplies roughly 40% of the total energy for propulsion, with the hip extensors, hip flexors, and quadriceps supplying approximately 30%, 20%, and 10%, respectively. Because of the loss of distal control, the situation is usually reversed in a child with cerebral palsy. Consequently, the triceps surae frequently provides the least amount of useful energy for propulsion, with most of the power coming from the hip extensors (including the hamstrings) and the hip flexors, respectively.

Because the net accelerations are greatly reduced in the cerebral palsied gait, decelerators as such are not as important as accelerators. There are notable exceptions, however. The soleus, for example, retards (decelerates) the forward progression of the tibia in midstance. This maintains the GRF in front of the knee and thereby generates an extension force to counteract the effect of the hamstrings and gravity. If the soleus is weakened excessively, particularly if the hamstrings and/or hip flexors are contracted or spastic, the patient will invariably develop a crouch

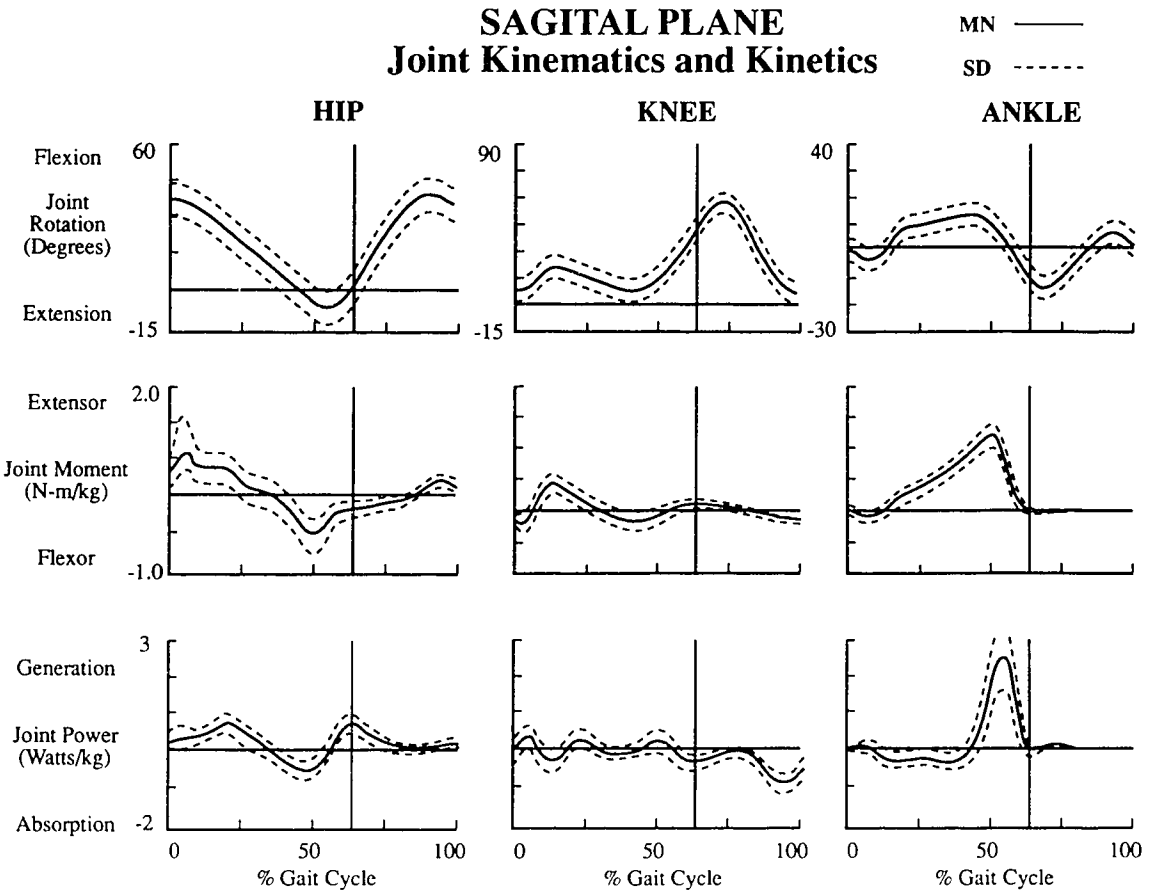

FIG. 3. Kinetics (moments and powers). The graphic output of sagittal plane moments and powers used at the author's institution. The top row is actually the kinematics of the hip, knee, and ankle (degrees of rotation). The middle row displays the moment of force around each of these joints in newton-meters per kilogram, and the bottom row displays the joint power, which is the product of the joint's moment times its angular velocity. The units of the abscissa are watts per kilogram. (Solid line = mean; broken line = standard deviation.) (Reprinted with permission from Gage, J. R.: Gait Analysis in Cerebral Palsy. London, Mac Keith Press, 1991, p. 31.)

gait. Furthermore, many of the decelerators such as the hamstrings and the rectus femoris are biarticular muscles that act eccentrically as decelerators at one end and concentrically as accelerators at the other. Thus, weakening the hamstrings will not only reduce their deceleration effect at the knee but will also reduce their ability to act as accelerators at the hip, thus reducing the net extension force at the hip.

In general, stabilizers are slow-twitch muscles that function more or less isometrically. Thus, from a standpoint of cerebral control, their function is much less complex than that of the fast-twitch, biarticular muscles. It is probably because of this fact that stabilizers are not usually involved in cerebral palsy. However, they may appear weak because they have an inadequate or insufficient lever arm to generate an effective joint moment or because they are relatively overpowered by antagonists.

ORTHOPEDIC VERSUS MOMENT ARM DEFORMITY

Muscle forces can be much more easily understood if they are always pictured as part of a force couple or moment. As a simple example of moments of force, think of two children on a teeter–totter. Each child is creating a moment around the axle to which the teeter–totter is attached, and since the moments are in opposite directions, they are tending to balance each other (Fig. 4). Muscles produce internal moments, which resist the inertial forces and/or external moments produced by the GRF. In the case of both the muscle and the GRFs, the lever arm on which they act is the bone and their axis of

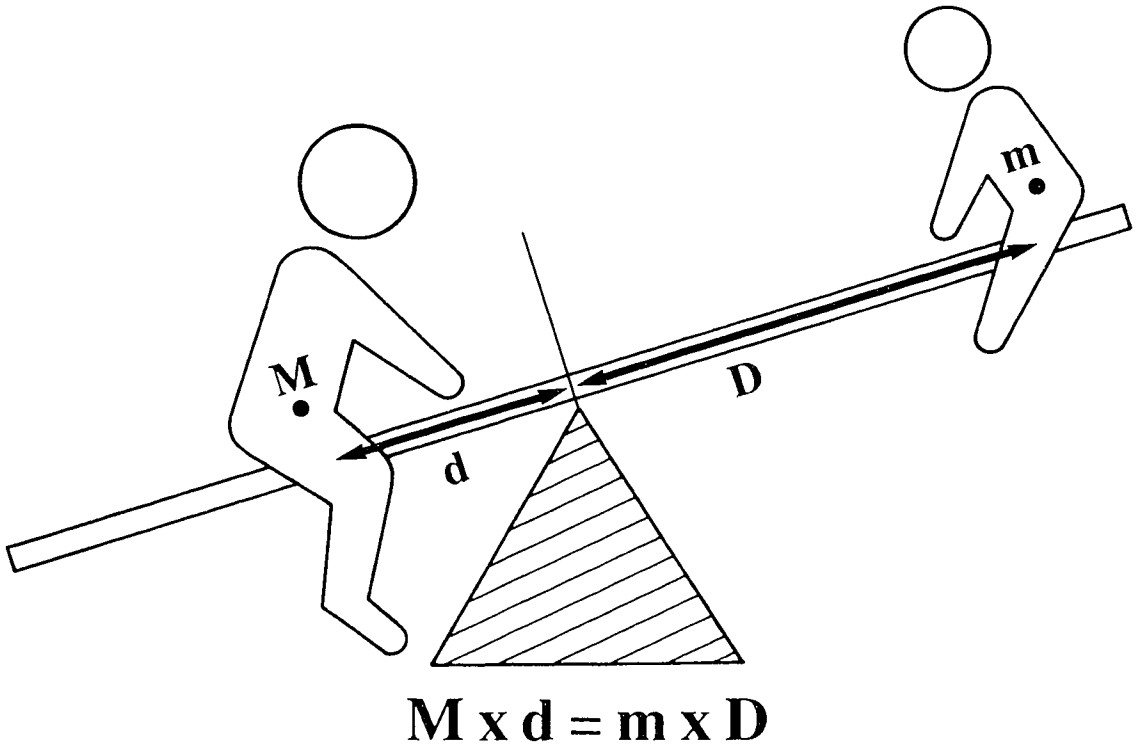

FIG. 4. The relationship between an adult and a child on a teeter-totter is exactly the same as that of the muscle and the GRFs at each of the lower-extremity joints, and the pivot point or fulcrum is always the joint center. (Reprinted with permission from Gage, J. R.: Gait Analysis in Cerebral Palsy. London, Mac Keith Press, 1991, p. 80.)

rotation is the joint center. If the muscle is acting perpendicular to the axis of rotation, the moment of force produced is always equal to the muscle force times its distance from the axis of joint rotation. If one remembers that a muscle always acts as part of a force–couple or moment, it should be apparent that an inadequate moment arm (lever) will produce apparent weakness in the face of an adequate muscle. In the past, orthopedic surgeons have concentrated on the muscles and have tended to ignore bony lever arms. However, restoration of appropriate lever arms is critical to restoration of function. Two examples of this are the foot with pes valgus deformity and the externally rotated foot. In the case of the pes valgus foot, the lever arm has no rigidity, so the GRF cannot generate an adequate extension moment against the knee. In the case of the externally rotated foot, the plane of the foot is external to the plane of the knee, and the force generated against the knee by the GRF produces a combination of extension, valgus, and external rotation (Fig. 5). Depending on the pathology, either subtalar arthrodesis or a derotational tibial osteotomy might rectify the situation without requiring muscle surgery.

THE ROLE AND IMPORTANCE OF TWO-JOINT MUSCLES

In general, the two-joint muscles function as energy transfer straps in gait, and as such

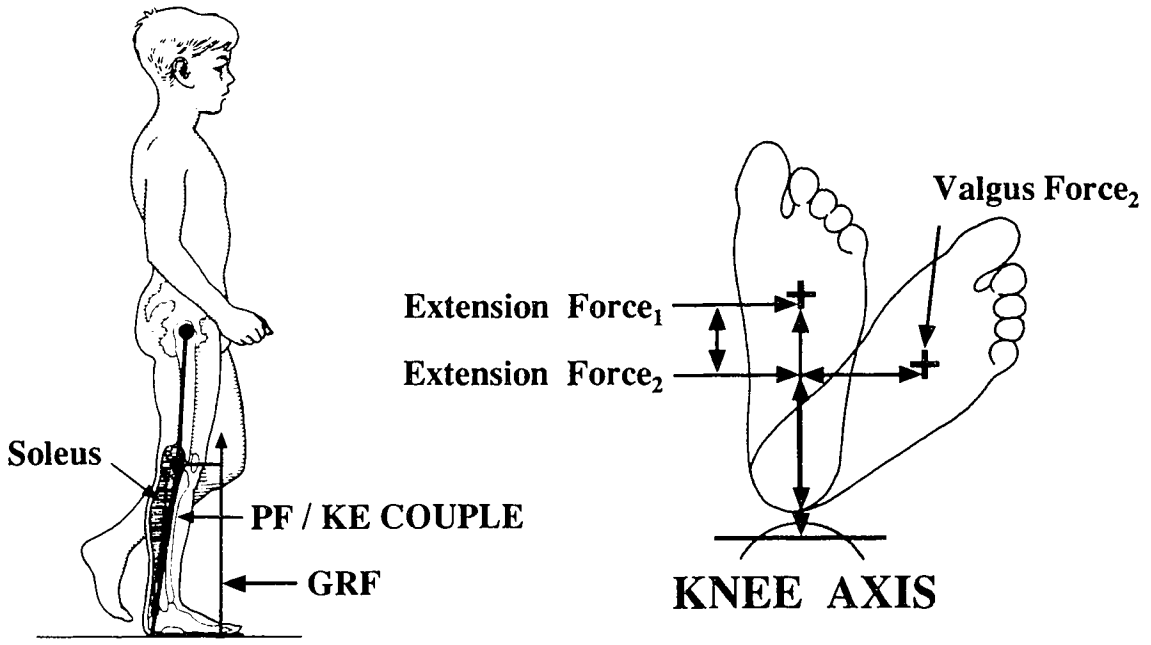

FIG. 5. The plantarflexion/knee-extension couple. An illustration of the effect of external foot rotation on the GRF. In normal gait at midstance, the GRF generates a pure extension moment at the knee as seen in the figure on the left. If pes valgus and/or external tibial torsion is present, the moment vector of the GRF moves posterior and lateral to its normal position as shown on the right. This means that the knee-extension moment is reduced and additional moments are introduced, generating valgus and external rotation forces at the foot, shank, and knee. With time and growth, these abnormal forces will produce bony deformity such as progressive pes plano valgus, external tibial torsion, and genu valgum. (Reprinted with permission from Gage, J. R.: Gait Analysis in Cerebral Palsy. London, Mac Keith Press, 1991, p. 106.)

they have been estimated to reduce energy consumption by about 20% during normal walking.[6,8] For example, with rapid walking the rectus femoris is used as a concentric accelerator to augment hip flexion power during preswing and initial swing phases. However, distally this muscle acts as an eccentric decelerator of the shank by limiting knee flexion via its patellar insertion. In this way, the rectus is actually absorbing energy at the knee and generating energy at the hip. Thus, as it transfers energy from the shank to the thigh, the resultant muscle contraction may be nearly isometric (Fig. 6). However, precise control of the timing and intensity of action is critical to normal function. Because of the complexity of their action and the deficiencies of the cerebral control system, these muscles probably never function appropriately in patients with cerebral palsy. Therefore, function can often be enhanced if the action of these muscles can be simplified or modified. An example of this is transfer of the distal end of the rectus femoris to the gracilis or the sartorius, which converts the muscle from a hip flexor/knee extensor to a hip flexor/knee flexor.[2,3,5]

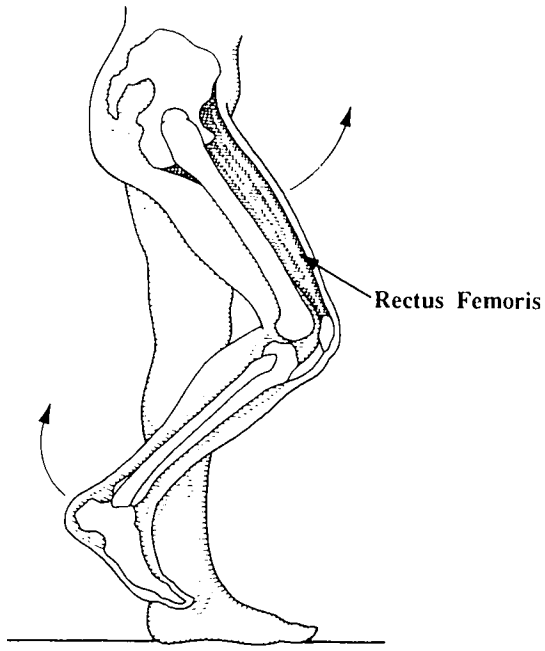

FIG. 6. During initial swing, the rectus femoris limits the rate of knee flexion. Rapid walking is achieved through increased power of the plantarflexors and the hip flexors. Therefore, the eccentric restraining action of the distal end rectus femoris limits knee flexion. Rather than wasting this energy, the rectus femoris acts concentrically at its upper end to augment hip flexion. Thus, the total action of the muscle is more or less isometric, and its function is that of an energy transfer strap, carrying the inertial energy of the shank up to the hip. (Reprinted with permission from Gage, J. R.: Gait Analysis in Cerebral Palsy. London, Mac Keith Press, 1991, p. 69.)

SEPARATION OF PRIMARY AND SECONDARY (COPING) DEFORMITIES

It should be remembered that in the cerebral palsy gait, abnormalities rarely occur in isolation. Rather they are multiple and consist of both primary anomalies (*i.e.*, those directly attributable to the damage to the central nervous system) and secondary anomalies, which are those compensations the individual uses to circumvent the primary problems of gait. Thus, the secondary compensations can be thought of as "coping responses." For example, cospasticity of the rectus femoris and hamstrings commonly produces a stiff-knee gait, which in turn leads to problems with foot clearance in swing. This is frequently compensated by hip abduction to circumduct the swinging limb. The primary deviation of gait in this example is the stiff knee that results from the rectus femoris and hamstring cospasticity. The secondary, or coping, response is the circumduction. Much of the difficulty encountered in studying pathologic gait is the separation of the primary and coping responses. Since normal gait is the most efficient, any deviation from the normal, whether primary or secondary, results in excessive energy consumption. Good treatment demands their separation, because to optimize the efficiency of gait, we need to correct the primary abnormalities and not interfere with the coping responses, which will disappear spontaneously when no longer required. However, because

many of these gait deviations are extremely subtle, separation often cannot be accomplished without formal gait analysis. Dynamic electromyography (EMG) is not particularly helpful in this separation, since primary and secondary abnormalities will result in aberrant muscle firing times and thus in abnormal EMG. However, if we study joint kinetics (moments and powers) we will be provided with an objective method to differentiate between primary and coping response, particularly if these are used in conjunction with dynamic EMG.

EMERGING HOMOGENEOUS PATTERNS OF INVOLVEMENT

Diplegia and hemiplegia are "wastebasket" terms, as each contains a variety of homogeneous patterns. In spastic hemiplegia, researchers have found four principle types of involvement; Type I is the least and Type IV is the most severely involved pattern.[7] These types can be differentiated by gait analysis and treatment tailored to the specific pattern type (Fig. 7). With appropriate treatment, the patient with Type III or IV involvements can be significantly improved so that his/her gait approaches that of a patient with Type I involvement. The four subtypes of spastic hemiplegia are relatively simple and often can be recognized with slow motion videotape alone. However, spastic diplegia is much more complex and probably contains many subtypes. However, once the subtypes of diplegia have been identified, criteria for their identification can be established and optimal

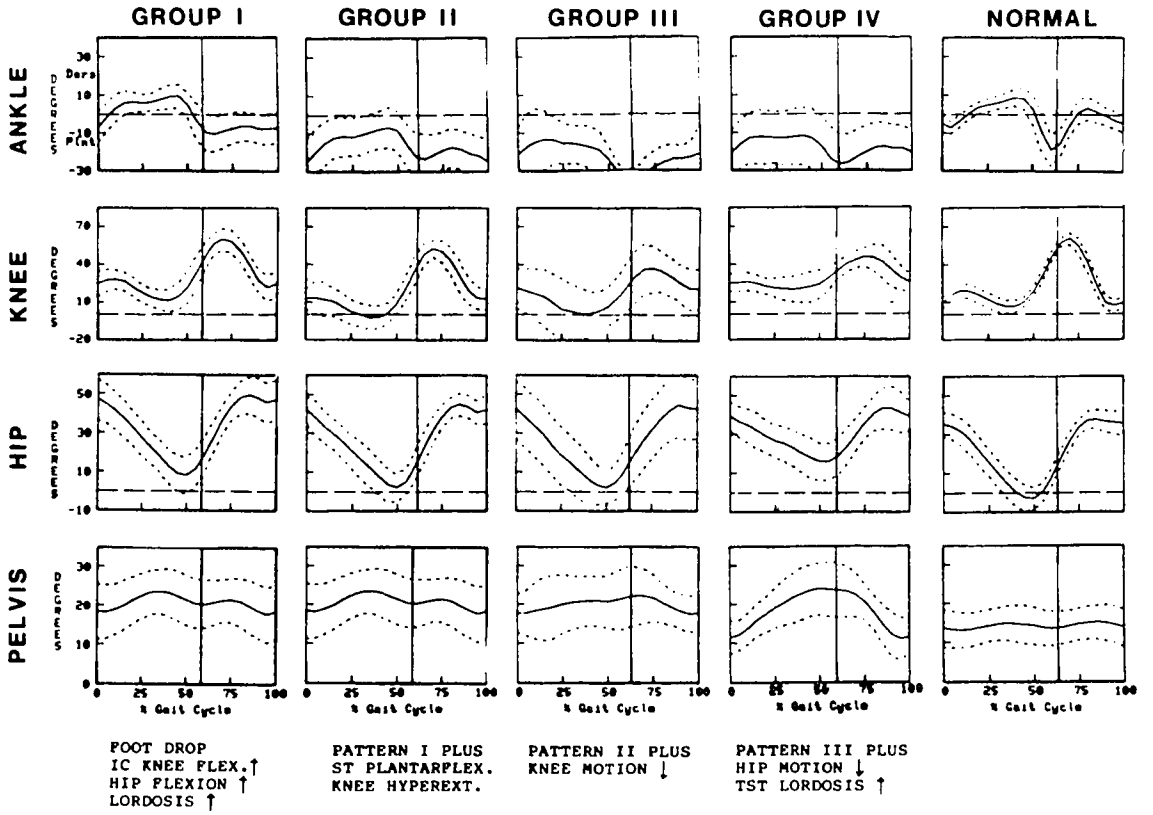

FIG. 7. A summary of the kinematics of the sagittal plane in the four types of hemiplegia. The charts indicate the mean (solid line) and the standard deviations (dotted lines) of rotation of the joints in the sagittal plane for each and for normal values. (Reprinted with permission from Winters, T. F., Gage, J. R., and Hicks, R.: Gait patterns in spastic hemiplegia in children and young adults J. Bone Joint Surg., 69A:438, 1987.)

treatment protocols determined for each subtype.

Until the advent of clinical gait analysis laboratories, the treatment of cerebral palsy was an art, not a science. Beyond the clinical examination, there was no attempt to precisely define the pathology; very little thought was given to how the treatments would affect the dynamics of that pathology in either the short or long term, and not much effort was made to assess the outcome of the intervention. Since the priorities of normal gait were not well understood, treatments were not aimed at establishing these objectives. The importance of muscles as prime movers in gait and bones as the levers on which they act was known only in a very general way, and little effort was made to define and preserve muscles that were functioning as accelerators or to restore their lever arms. As a result, iatrogenic injury was often added to the physiologic burden inflicted by the cerebral palsy, and the child was worse after the intervention than before. Our physical therapy colleagues recognized this and therefore often argued against surgical treatment. Further damage resulted from prolonged immobilization, which left the child weak and stiff. And finally, frequent interventions converted childhood from a time of play to a perpetual state of recovery.

Fortunately this is starting to change. Thanks to computerized motion analysis, there is a much better understanding of the physiology of gait, and in a very rudimentary way, the neurophysiology of motor control is beginning to be understood. Because of gait analysis, treatments have changed a great deal during the past ten years. In a few more years, with the widespread dissemination of gait analysis, orthopedic surgery will be based routinely on objective evidence of dysfunction and sound hypotheses. Computer simulation of treatment is now close to reality. In fact, a paper has recently been published in which this technique is already being used.[1] Because today's technology has opened new doors of opportunity, tomorrow's orthopedists will need to be true "bioengineers" of the human frame if they wish to treat their patients well.

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