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REVIEW ARTICLE

Reconstructing cerebral palsy

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Abstract

Forty years ago, a consensual definition of the cerebral palsy concept was suggested, delineating it as a disorder of movement and posture secondary to non-progressive pathological processes that affect the immature brain. Because this concept is pragmatic and based on function, it has survived unaltered many changes in pathophysiolgical knowledge, diagnostic technology and general nosology. However, its basis has appeared to be flawed. Its main justification remains management, for which the need to meticulously select patients, define adapted objectives, design appropriate management programs and evaluate results has been increasingly recognized. Fine movement analysis using recent technologies can provide a wealth of information about neurological functioning in cerebral palsy that can serve these purposes. Specific patterns of motor organization reveal different modes of motor control in individuals with developmental motor problems. The different motor patterns reflect individual adaptation to the impairment of the central nervous system. Taken phenomenologically these patterns can contribute to the clinical approach to cerebral palsy and redefine patients groups within this framework. (J Pediatr Neurol 2004; 2(2): 57-64).

Key words: cerebral palsy, history, motor control, movement analysis.

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Introduction

Certainly, there have always been individuals with developmental motor problems, but the idea of the current grouping and categorization of many of them under the label 'cerebral palsy' (CP) is relatively recent. This category is not a fixed and inevitable correlate of specific neuropathology or pathophysiology. Rather, it emerged in nineteenth century medicine as a heuristic product of contemporary epistemology. Little's seminal 1862 paper was entitled 'On the influence of abnormal parturition, difficult labours, premature birth, and asphyxia neonatorum, on the mental and physical condition of the child, especially in relation to deformities'. The term CP currently designates a group of conditions characterized clinically by chronic motor impairment due to early occurrence of a stable lesion to the brain. Given its inclusiveness the term implies a lot of heterogeneity in terms of etiology as well as types and severity of motor and associated disabilities. Kinnier Wilson (1) recognized that 'Little's disease' "is not an ailment of well-defined character, but a mere syndrome, and one of rather a wide range at that". However, the derived nosographic category has strong historical roots and the designation CP has remained in universal use by clinicians, therapists, epidemiologists and researchers. CP is a major public health issue in several respects. The incidence is 0.2-0.3% in industrialised countries, which is in the same order of magnitude as adult-onset diabetes and stroke. The current incidence of CP is similar to that recorded in the 1950s. The incidence decreased to 0.1-0.2% in the 1960s, apparently in relation to better control of obstetrical and neonatal risk factors. It subsequently increased, apparently in relation to increased survival of babies with very low birth weight. The motor disability in CP commonly includes several aspects of organization and control of movement and posture. The motor impairment may lead to orthopedic complications that further restrict motor abilities. Other clinical problems may be associated with the motor impairment because

of lesions caused by the same pathological process and because of the restricted motor development. Associated problems may include sensory and perceptual impairment, notably visual impairment, cognitive impairment, affective and behavioral disturbances, seizure disorder and failure to thrive.

Clinical recognition of CP is relatively recent, although it seems that Shakespeare gave an individual account of it in the words of the Duke of Gloucester, future King Richard III, perhaps hinting at prematurity and neonatal respiratory problems as the etiology: *"I, that am curtail'd of this fair proportion, cheated of feature by dissembling nature, deform'd, unfinish'd, sent before my time into this breathing world, scarce half made up, and [...] so lamely and unfashionable."* In his monograph on CP, Ingram (2) extensively reviewed the early medical literature on the subject. In the first medical report of the condition, in 1828, Delpech thought that his patient had suffered a spinal cord disease and that her brain, which he believed to develop from the cord, had failed to mature fully. The first large series of patients with CP was published in 1842 by Little, hence the term Little's disease. Thereafter, an increasing number of patients were recognized as having paresis and 'rigidity' with a paraplegic or hemiplegic distribution, or generalised involvement of the limbs and the trunk, since birth or the first years of life. However, largely consensual, explicit definitions of the CP concept were not formulated for more than a century after the initial medical descriptions.

Delineation of the concept

Forty years ago, a workgroup proposed a construal of CP concept which has gained wide acceptance and is still in use (3). The group agreed that CP is one of a group of conditions due to cerebral dysfunction. They defined it as a disorder of movement and posture due a defect or lesion of the immature brain. They excluded from the label motor disorders of short duration, those caused by progressive disease and those "due solely to mental deficiency". The concept was a highly contingent outcome of the evolution of medicine in the early 1960s, and in particular of new views on development and early brain damage. It appeared as progressive, as predictions could be made within the framework of CP that were not covered by predecessor models based on late-acquired cerebral lesions. It is noteworthy that this definition has survived despite the many changes in pathophysiolgical knowledge, diagnostic technology and general nosology that have occurred since 1964. The remarkable robustness of the CP concept largely results from its accommodative properties that include the choice of the terms in the definition and the option that it is essentially based on function, defining patients with CP by what they do rather than by what they are. A similar tendency for favouring praxic aspects over ontological ones was contemporaneously noted in the field of sociology (4). In this way, Bax's definition pre-empted the World Health Organization approach to disabling conditions by more than 15 years.

Nevertheless, however stable in time and convenient in many contexts, the conceptual basis for CP is problematic. Limitations and difficulties are associated with most of the terms in the concept: a (a) disorder of (b) movement and posture secondary to (c) non-progressive pathological processes affecting the (d) immature (e) brain. (a) Any disorder category should be defined with respect to an appropriately documented normal situation. The very notion of a norm has been questioned over the last few years (5). This concept is often regarded as either representing an ideal model or a statistical reality. Modern nosology has tended to use various types of measurements to draw a precise line, sometimes assorted with 'confidence intervals', between normal and pathological. This is the case for many 'disorders', from flat feet and hypertension to AIDS and phenylketonuria. For CP no such measurements have been proposed, leaving individuals with clumsiness, poor balance or other mild motor difficulties in an area of diagnostic uncertainty. (b) The concept of CP is essentially based on the presence of motor manifestations. As just noted, no theoretical limit is given to this, although most clinicians would not regard signs like tremor, which is prevalent in groups at risk for unquestioned CP (e.g. children with very small birth weight), as sufficient for this diagnosis. Sensory, cognitive, behavioral and other associated impairments, though very prevalent and often significantly disabling, are not included in the concept. (c) It is usually accepted that progressive pathological processes, such as infection or tumour, may cause CP, provided their progression stopped while the brain was 'immature' (see (d)). A wealth of pathological and neurophysiological data now exist that indicate continuing aberrant development after the initial insult. This correlates with the Little Club's Memorandum of 1959, describing CP as a persistent but not unchanging disorder. However, it modulates seriously the notion of non-progressiveness. (d) 'Immature', which denotes the important functional difference between early and late lesion, is certainly the vaguest term in the definition. This vagueness was justified to prevent "administrative difficulties" (3). This likely echoes Milani-Comparetti's warning that excessively objective definitions in the context of CP might result in inadequately excluding numerous patients from assistance on legal grounds (6). Yet it has

proved more useful clinically than arbitrary time limits. As regards functional implications, we feel that 'immature' may be taken to mean "before function has developed" for each considered function (e.g. walking, manipulation, etc.). However, the very concept of maturity has been challenged in the last few years (7,8). (e) The term 'brain', which includes the cerebellum (and perhaps the brain stem), usefully excludes motor disorders of spinal, peripheral nerve, muscular or mechanical origin. Early reports described cases consistent with CP either together with other cases of pediatric motor disorders under semantically neutral terms, such as essential infantile paralysis (9), or included within pathologically defined groups, such as fatty atrophic paralysis of infancy (10), without presuming the precise neurological cause. Although Duchenne de Boulogne (10) demonstrated that bilateral paralysis of the lower limbs could be due to brain damage, many late nineteenth century neurologists believed that it could only result from spinal pathology. The spinal origin of Little's disease was thus supported by Erb, Pierre Marie and Charcot (who used the term 'tabès spasmodique' for what would now be 'spastic diplegia'). On the basis of different clinical reasoning and pathological evidence, other neurologists argued for a cerebral origin, like Gowers (who called the condition 'central birth palsies'), Osler and Freud (who coined the term 'Diplegie'). At the beginning of the twentieth century the debate was not settled yet, as Déjerine and André Thomas (11) included Little's disease in their treatise on spinal cord disorders. As late as 1936 Van Gehuchten (12) relayed clinical, histological and pathophysiological arguments aiming to demonstrate the spinal origin and dismiss the cerebral origin of Little's disease "proprement dite", while recognizing that lesions in the cerebral motor areas result in a cerebral form of the disease. Several older classification schemes for CP include a category labeled 'paraplegia' (13-15) and some authors still refer to paraplegia when the arms are 'minimally' as opposed to 'perceptibly' affected in spastic diplegia (16). This is a source of confusion as the term paraplegia classically indicates involvement of the motor pathways in the thoracic or upper lumbar cord. However, some cases of cord injury with slight neurological deficit are probably wrongly diagnosed as CP. A recent study suggests that there may be differences in locomotor control between developmental spasticity of cerebral and spinal origin (17). Distinctive features of the latter include relative preservation of head orientation, trunk instability, preserved arm swing and lack of selectivity in lower limb movement. There may be a case for proposing a germane concept of spinal palsy defined as abnormal movement and posture secondary to non-progressive pathological processes affecting the immature spinal cord.

The exclusion of disordered movement and posture "due solely to mental deficiency" is interesting but cannot be useful as the mechanism underlying this association is currently not clear. In many instances (e.g. Down syndrome, Prader-Willi syndrome) factors causally unrelated to cognition likely play an important role in motor dysfunction. And certainly, if motor disorder can be due directly or indirectly to mental deficiency, the latter must contribute significantly to the clinical manifestations that typify forms of CP that are consistently associated with cognitive impairment. Little recognized the 'mental condition' of the affected children and from another standpoint Ireland suggested 'paralytic idiocy' as one of 12 types of intellectual disability in one of the first modern textbooks on this subject (18,19). One hundred years ago, however, Brissaud and Souques proposed to exclude cases of congenital spastic rigidity with mental defect from the context of Little's disease, but the constructed entity proved dubious (1). More recently, other autors have attempted to revise or update the definition of CP, though only minor qualifications were added, as by Mutch et al. (20): "an umbrella term covering a group of non-progressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of the brain arising in the early stages of its development". Still, the comparability between the use of the term CP in different places and times has been shown to be poor (21). In this context, Ferriero (22) discussed the difficulties in diagnosing CP, "something that is not one thing", while Niemann and Michaelis argued that it cannot be considered as a diagnosis and the term should not be used in medical and scientific communication (23).

Implications for management

The CP concept thus appears to be pragmatic but flawed and confused. However, analytic thinkers acknowledge that *"what is confused is sometimes more useful than what has been clarified"* (24). Their approach would not consist of refuting the concept but rather of exposing the function it serves. The main justification for the CP concept has been management. Various treatment programs for patients with CP have been proposed over the years. Awareness of the necessity to evaluate the effect of treatment has increased gradually. Five years ago, the American Academy for CP and Developmental Medicine suggested guidelines for classifying treatment outcome (25). These guidelines are based on dimensions of disablement inspired by the World Health Organization recommendations on levels of evidence. They are consistent with the concept of evidence-based medicine as applied in child

health issues and have provided a highly valuable framework for assessing the results of studies conducted in patients with neurodevelopmental disabilities. However, no similar guidelines have been proposed for designing treatment programs. This may result in a contrast between the rigour applied in checking answers and in asking questions. Specific quality standards for framing questions may be crucial to achieve a better outcome for patients. It is recognized that the complexity of many clinical problems calls for specific questioning of their different elements. In the case of CP, multi-level reappraisal would seem timely given the rapidly increasing availability of promising treatments such as intrathecal baclofen, botulinum toxin injections, selective dorsal rhizotomy, pallidal stimulation and other medical and physical interventions.

Ideally, design of management of motor dysfunction in CP should be directed at predefined objectives. With respect to the dimensions of disablement suggested by the World Health Organization, it may be relevant to give attention to an additional domain that links motor impairment to functional limitations, namely motor control (26). This would question how a task is performed rather than if it is realized. This approach might lead to a better understanding of the pathophysiology underlying the disability in selected groups of patients. It might also directly contribute to determining optimal management.

In this context, patient selection is particularly critical (27-29). Since Little, classification schemes have been proposed to account for different types of CP. Although some of them derive from attempts to integrate presumed etiological and pathophysiological aspects together with clinical elements (13,14), the more commonly used classifications are based solely on the predominant muscle tone abnormality, eventual involuntary movements and the gross topological distribution of these neurological abnormalities. Most classification systems represent descriptive compromises that may serve to propose adapted management programs. This is reflected in differences between classification systems suggested by different practitioners, whether pediatricians, orthopedic surgeons, neurologists or pediatric neurologists. Currently the systems in regular usage include the following categories: spastic diplegia, spastic quadriplegia, spastic hemiplegia, extrapyramidal or dyskinetic CP and ataxic CP. The use of pathophysiological terms for describing clinical features may be criticised. However, neurological semiology has consecrated many of such connotations. As the clinical definitions of these terms have been based on adult disorders and different types of abnormalities found simultaneously in a single child may be difficult to identify, a Task Force on Childhood Motor Disorders has recently proposed clinical definitions of several types of tone abnormalities in children (30). For example, the term 'spasticity' is used when resistance to externally imposed movement increases with increasing speed of stretch and varies with the direction of joint movement, or when it rises rapidly above a threshold speed or joint angle. 'Dystonia' refers to involuntary sustained or intermittent muscle contractions that cause twisting and repetitive movements or abnormal postures. Distinction between spasticity and dystonia occurring in the same limb therefore requires determining the velocity-dependent, action-induced and posture-responding components.

More functional systems have been developed, such as the Gross Motor Function Measure (31), a widely used validated test of basic movement skills that was designed specifically for clinical and research use in children with CP. Together with a gross motor function classification system, this tool may serve to classify children into categories of severity and evaluate gross motor changes over time. Quantification is made on the basis of how much of the tasks the patient can realize independently, without any reference to the quality of the performance.

Tridimensional recording of movement

In contrast, other approaches are based on fine analysis of movement. Human movement study has long been limited by technical difficulties concerning both kinematic recording and analysis of recorded data. These difficulties have gradually been overcome thanks to technological advances. A major breakthrough was achieved in the early days of photography. This new technique for capturing instances in time allowed examination of movement in the split second when action was artefactually (virtually) stopped. In the second half of the nineteenth century the American photographer Eadweard James Muybridge and the French medical doctor and physiologist Etienne-Jules Marey succeeded in producing motion pictures of human locomotion by using iterative photography. Muybridge used multiple cameras which could be triggered electrically to obtain an order series of shots. Marey used a single camera with a revolving shutter, the *fusil photographique*. Their work set the basis for all subsequent progress in this area of research, leading to the current technologies for tridimensional quantification of movement using multiple rapid digital cameras. Usually movement recording systems include an array of digital cameras that track the displacement of skin markers. The precise position of cameras is calibrated into the computer software. In most currently available systems, the markers are either passive, reflecting

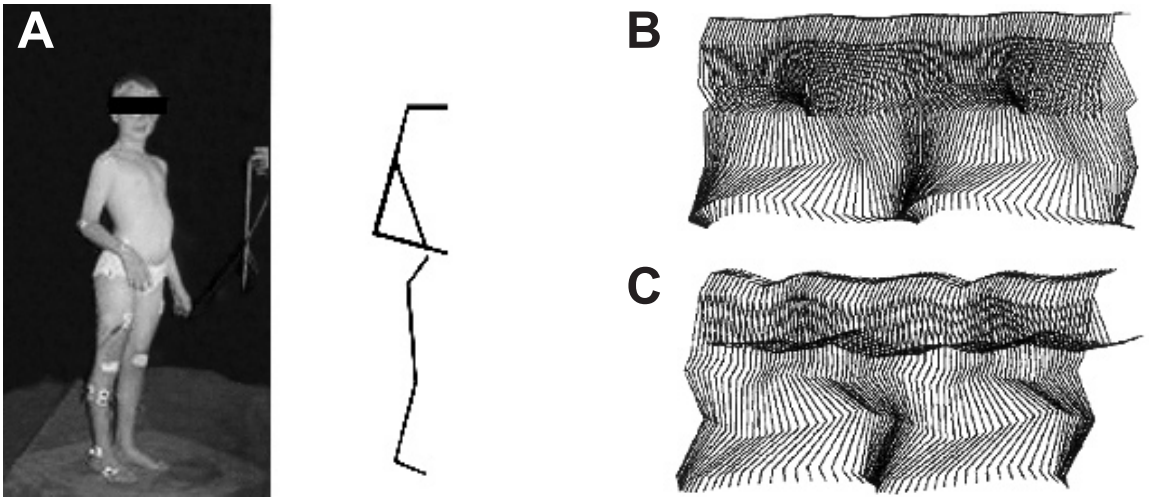

Figure 1. A: Positioning of reflective markers for kinematic recording and surface electrodes for electromyography. The stick diagram on the right shows the links between the markers. **B:** Kinogram (stick diagram) representing sagittal projection of locomotion, showing two gait cycles of an unimpaired child. Two consecutive segments represent 20 ms. **C:** Kinogram representing sagittal projection of locomotion, showing two gait cycles of a child with leucomalacic spastic diplegia [locomotor control strategy I (44)]. Two consecutive segments represent 20 ms.

light emitted towards them, or active, emitting light themselves. In accordance with Marey's suggestion, these markers are placed at specific anatomical landmarks. They define segments that form a model for the body or body parts which is approximated to the actual body by a mathematical model included in the computer software (Figure 1). Data recorded synchronously by several cameras are processed to give the tridimensional displacements of the markers with respect to themselves and the external space. Processed data form the base for kinematic calculations. Additional recording modalities may be used in synchrony with that of markers displacement. Electromyography can be performed simultaneously in different muscles, usually by means of surface electrodes. Force plates may also be included in comprehensive systems. Several devices have been used to approach energy expenditure associated with the recorded movements. These technological developments give wide and reliable access to multiple aspects of movements. Such objective, quantifiable and standardizable information can be followed up with respect to natural history or intervention. In particular, it can be practically applied at a gait and posture laboratory before and following specific treatments. Synchronous acquisition of kinematic parameters and muscle activity has revolutionized the physiology of movement.

Naturally, this sophisticated approach to movement has been largely applied in the field of movement disorders where experience in observational movement analysis already existed as well as a need to further refine observation and measurements of intervention results. As walking is the movement *par excellence* for which such an experience existed, it became the main focus for analysis (32). Therefore, most movement recording facilities used for clinical purposes are currently referred to as 'gait laboratories'. In the last few years clinical interest in movement analysis has increased markedly, particularly with regard to the management of CP (33). The hope has been that this technology might optimize treatment procedures: *"Its use has transformed the treatment of gait disorders from an art into a science"* (34). However, the place of laboratory movement analysis in the management of patients with CP is currently uncertain. A survey of published results of treatment of gait problems evaluated by computerised gait analysis led to the conclusion that available evidence of efficacy of the technology is currently insufficient, possibly in relation to the type of data collected (35).

Motor control

An exclusive focus on gait is probably an excessive reduction of a subject's motor behavior. Also, the relevance of recorded parameters has not been determined. However, physiological studies of motor organization and control using this technology could have major implications pertaining to its clinical use. A growing body of such studies of motor control have provided physiological insights into CP. Patients with early lesion to the brain control movement and posture by using more global rules of motor organization than unimpaired subjects as an adaptation to the central nervous system impairment. This should be taken into account when defining targets for intervention.

The question of whether patients with CP organize their motor behavior in a 'primitive'

or 'immature' fashion has long been a matter of debate with regard to pathophysiology, diagnosis and management of CP. Recommended clinical examination procedures (15) and passive observation (36) have focused on some features of otherwise "primitive normal movement patterns" for their value in indicating CP. Conversely, many authors have considered the motor behavior of unimpaired infants and young children in terms of neurological deficit, likening it to pyramidal, extrapyramidal or cerebellar disorders. A few similarities between unimpaired toddlers and children with CP have been reported, most strikingly a tendency to use agonist-antagonist muscle co-activation (37,38). A dominant co-activation pattern has been documented for various tasks in small children (39,40). More generally, this has also been described at the early stage of acquiring a new skill, whereas reciprocal inhibition is seen when the skill has been learned. However, the children with CP cannot be considered as inexperienced as they had been able to perform the studied movements for several years before the studies. Furthermore, greater variability is found in unimpaired toddlers than within the groups of CP, suggesting adaptability based on diversity, competition and choice in the former and predominance of stereotyped, 'fail-safe' mechanisms in the latter. Finally and most notably, head angular orientation is more stable in toddlers than in any of the CP groups, suggesting a mode of control relying on a head-centered referential as further refined in older normal children but not in CP.

Since Freud coined the term 'diplegia', it has been used universally to designate a self-contained entity despite the clinical heterogeneity such a basic descriptor is expected to cover. This is likely related to an epidemiological bias as the vast majority of cases of spastic diplegia are due to periventricular leucomalacia secondary to hypoxic-ischemic insult in preterm neonates. However, motor functioning is not uniform in spastic diplegia (41-45). In the group of children with leucomalacic spastic diplegia, four distinct strategies of gait control have been identified (44). Furthermore, comparison between leucomalacic spastic diplegia and Angelman syndrome underlines the importance of qualifying groups of patients with spastic diplegia with other elements than the topography and predominance of tone alteration (45) in a context where diagnosis of both conditions is ultimately based on clinical elements including the motor impairment.

Recent approaches have thus striven to redefine patients groups within the clinical framework of CP according to the presence of characteristics that reflect specific modes of motor control rather than deficiency (42-44,46-48). Further studies may identify more characteristics to achieve a more complete description. In this context, particular attention should be given to functional tasks as well as the environmental factors relating to the patients. The main objective of this type of approach is change in motor organization of patients through alteration of motor control. This is consistent with the demonstration of new patterns in patients with various conditions following unsupervised (49) or guided practice (50). The relatively late emergence of these motor patterns indicates that they are not univocally determined by neural factors but more likely represent experiential modification of neuronal subgrouping within populations tuned to the encoding of the previous patterns.

The theories of motor control underlying these approaches predict that early management (i.e. commencing before function has developed, for each considered function, as in the above discussion on 'maturity') might result in improved motor control in CP. In this view, for example, special emphasis on early control of head orientation or achievement of good axial control before independent walking should be encouraged. Furthermore, early improvement of motor control might affect other domains of neurological functioning. For example, early head stability should enhance early visual experience, which might improve visual function and related cognitive processes.

Conclusion

Perhaps individuals who are now diagnosed as having CP are different from individuals to whom this term has been applied over the course of the last 40 years. This might be due in part to epidemiological reasons, that have been termed the 'changing panorama' of CP (51). In addition, it might be because of the theories held about these individuals and the remedies that have been put in place around their abnormal behaviors, leading to the emergence of so-called 'classical' and 'modern' forms (52). Conversely, the resulting changes in the individuals have significantly contributed to the evolution of ideas about physiological and pathological motor development.

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