To provide an initial context, in Chapter 1 Mantovani and Scrutton detail those aspects of the history of cerebral palsy (CP) that are not only important in their own right but also provide insights into how the basis of current practice has evolved. History is not only the distant past. It also runs into the present day, so this chapter includes consideration of the contributions that have been made by those who were contemporaries not only of John Mantovani and David Scrutton but also of the Editors.

This is important as a background to the appreciation first of what constitutes CP, second as an illustration of what has been understood about the various relevant causal pathways, third as a demonstration of the approaches that have been used to guide therapy and other interventions, and finally as a context to current perspectives, which include family-centred care and the importance of maximizing functional abilities to foster societal participation.

Next in this section, Rosenbaum examines the issues of definition and classification. He has taken as a starting point the 2007 definition by Rosenbaum et al. By using what he describes as ‘a blind man and an elephant approach’, he illustrates that in any discussion on what constitutes CP it is important that consideration be given to who is asking the question and why. There are, for example, significant differences between the perspectives of considering CP as a biomedical condition or as a family predicament. For classification, both the editors and Rosenbaum have based their descriptions, not only in this chapter but also throughout the book, on those detailed by the Surveillance of Cerebral Palsy in Europe studies (SCPE). In his chapter, Rosenbaum has helpfully detailed this classification and correlated this with the range of neuroimaging abnormalities that may be seen.

In the chapter on family and social issues, Lach, Rosenbaum, Bailey, Bogossian, and MacCulloch examine in detail a range of topics that are relevant to parents and other family members when raising a child with CP. The diagnosis implies predisposition to varying levels of impairments related to mobility, cognitive processing, and communication. The authors emphasize the necessity for promoting an approach with families that conceptualizes possibilities for children’s functional achievement and for child and family development, and they recommend that there should be a clear focus on the long-term future as well as the immediate stages of function.

The next two chapters provide more information on living with CP. First, three personal perspectives are provided. These very helpfully portray what it means to have CP for an individual and, in addition, the account given by Lydia Ngwana, when describing her son Confidence, vividly illuminates the stresses of resource limitations.

By contrast Jamie Beddard details his childhood and adolescence before going on to describe his life as an actor, partner and father. Clearly resource limitations extend to life in the UK so far as employment and economic opportunities are concerned – but they can be overcome. Additionally, Karen Weinstein in her presentation adds the eloquent plea for health and other professionals to listen to disabled people and not to make assumptions without doing so.

Tosi and Rosenbloom then go on to provide a long-term perspective on CP. They emphasize first that there are more adults than there are children with CP. Next, having examined transition into adult life, they then describe the evolving impairments, especially pain, that can accompany ageing in CP. In their chapter they also review life expectation data for people with CP.

Palliative care and end-of-life issues and their management were beyond the remit of this chapter’s authors as, largely, these are not specific to people with CP. The editors recognize, however, that these are relevant issues, especially for very vulnerable children with profound disabilities who may have a short life expectancy but who nevertheless, together with their families, require sensitive, informed, and skilful multi-disciplinary care. Moreover, for some children with CP this requirement may continue for months or years rather than, as with adults who are nearing the end of their lives, be present only for a very short time.

This section concludes with Durkin’s chapter on global perspectives in CP. She emphasizes that CP is an invisible public health problem in most of the world. This is not because it is especially rare or because its impacts on the lives of individuals, families, and society are not measurable and profound. She draws this contrast by making clear that in high-income countries CP is considered the leading cause of motor impairment
in children, with prevalence estimates of approximately 2 per 1000 live births, or up to 3 to 4 per 1000 school-age children. However, globally, fewer than 10% of births occur and fewer than 18% of the world’s people reside in high-income countries. It follows that more than 90% of the public health impact of CP is borne by populations in low- and middle-income countries. She notes that there is a paucity of research on CP in low- and middle-income countries although the situation is slowly changing.

In this chapter, recent developments in concepts of disability and related international policies, and strategies for extending rehabilitation services in low-resource settings, are also discussed. Despite the progress that has been made, she makes the point that children in low- and middle-income countries are being compromised globally each day because of exposures and causes we know how to prevent. These include undernutrition, iodine deficiency, kernicterus, infections, and lack of access to newborn screening and primary health care, among others. She concludes that the extension of proven strategies to prevent CP to the majority of the world’s population living in low- and middle-income countries is essential and needs to be viewed as a global public health priority. This is no less important than the need to make new discoveries about the causes and strategies for preventing and managing CP in all populations.
Readers seeking a better understanding of where we are now may derive some benefit from the story of how we arrived here. Therefore this chapter provides an overview of the historical development of the concept of cerebral palsy (CP) and more detailed descriptions of some key individuals who led the changing interpretations and classifications of the condition. We have also included discussions on the definitions and various causal hypotheses that have been used along the way and a separate treatment of the often conflicting views of how various therapeutic interventions might contribute to making these children’s lives more fulfilling.

Pre-scientific views
References to congenital deformity are found in mythology in the person of Hephaestus (Roman name Vulcan), described as ‘the crook-foot god’ (Murray 1924), and in Hippocratic writings on clubfoot deformity (Adams 1886, p. 76). Other historical descriptions of deformity beginning as early as 4th century BC and extending to the 1500s describe various theories of causation such as inadequate infant swaddling, carelessness of the midwife, witchcraft, maternal ‘miswatching’ (seeing a person with deformity), and teething (Obladen 2011). By the mid-16th century, artistic representation of disability is evident in Raphael’s The Healing of the Lame (ca. 1515–16), and Shakespeare’s The Tragedy of King Richard the Third, which famously contains the protagonist’s self-description, ‘I, that am curtail’d of this fair proportion, cheated of feature by dissimulating nature, deform’d, unfinish’d, sent before my time into this breathing world, scarce half made up, and … so lamely and unfashionable’, denoting the deformity, disability, and a suggested relationship to preterm birth (Kail 1986, Raju 2006), although the recent discovery of the historical figure’s remains has provided new insights in the possible diagnosis. However, medical descriptions of presentations consistent with CP were extremely rare before the 19th century.

The beginnings: Little, Osler, Freud, and initial treatment ideas
During the first half of the 19th century the French school began describing anatomic localization for neurological impairments. Cazauvielh and Billard reported cases of congenital paralysis associated with cerebral atrophy, and Cruvielhier, Breschet, Lallemand, and Rokitansky described isolated cases of cerebral atrophy in children. In 1842 Henoch described cerebral atrophy in a young woman with childhood-onset bilateral hemiplegia in his dissertation (Longo and Ashwal 1993).

The first comprehensive description of the clinical manifestations of spastic motor conditions is to be found in the lectures and writings of William John Little, an English orthopaedic surgeon. Born in London in 1810, Little was a sickly child who developed a neuromuscular illness, perhaps poliomyelitis, at age 4 years and was left with a talipes equinovarus deformity (Accardo 1989). This experience ultimately led him to attend London Hospital Medical School. In 1835 he travelled to Berlin for further study, and while there persuaded Louis Stromeyer to perform a subcutaneous Achilles tenotomy on his left leg. The procedure was successful, and Little returned to London where he joined the faculty of the London Hospital Medical School and adapted the tenotomy procedure. At the age of 27 he published a comprehensive treatise on clubfoot and related orthopaedic deformities, which established his reputation as a world authority on these conditions. His professional prominence enabled him to found the Hospital for the Cure of Deformities in Bloomsbury Square in 1840, which was ultimately granted a royal charter as the Royal Orthopaedic Hospital in 1845.

In 1843 Little gave a series of lectures on ‘Deformities of the human frame’ in which he described ‘a peculiar distortion which affects newborn children which has never been elsewhere described, … the spasmodic tetanus-like rigidity and distortion of the limbs of the newborn infants, which … [he had] traced to asphyxia neonatorum, and mechanical injury to the fetus immediately before or during parturition’ (Anonymous 1854). This was a new conceptualization on the causes of orthopaedic deformity, which set the stage for Little’s landmark presentation to the Obstetrical Society of London in 1861. In this lecture Little provided insightful clinical descriptions and therapeutic suggestions, but it was his novel correlation of orthopaedic deformities with preterm birth and perinatal difficulties that was most noteworthy (Little 1862). He described individuals who had developed rigid hemiplegia, paraplegia, or generalized rigidity following a preterm or traumatic birth and whom he believed had experienced a ‘third termination’ to be distinguished from neonatal death or full recovery. Little suggested
that these outcomes had resulted from a ‘delay of only a few moments in the substitution of pulmonary for ceased placental respiration … (which) may imprint a lasting injury …’ (Little 1862, p. 297). He thereby established a new hypothesis that related long-term disability to the adverse metabolic effects of peripartum hypoxia. The published audience reactions to his lecture include notes of surprise, denial, and disagreement. Little conceded in follow-up discussion that there was great variability in the outcomes of children with difficult births stating ‘that perhaps for one [with motor disability] that depended on abnormal or preterm birth, there were twenty or more from other causes incidental to later life’ (Little 1862, p. 343).

Little resigned his faculty position 2 years after this lecture and its publication which so challenged the prevailing views on causation of disability. Nonetheless, he remained an active teacher and practitioner for the next quarter of a century. His name gradually came to be associated with various forms of spastic motor syndromes, most frequently spastic diplegia, which was termed Little’s disease well into the 1960s.

A notable physician who contributed to the literature during this same period was Jakob von Heine, who reported that symmetrical paralysis of the lower extremities could result from cerebral rather than spinal causes in the 1860 edition of his textbook. This was an important contribution, which conflicted with the views of Broussard and others who had localized the pathology to the spinal cord (Longo and Ashwal 1993). Other contributions included Cotard’s review of the clinical and pathological causes of cerebral paralysis while working with Charcot in 1868 and case series of infantile hemiplegia, cerebral sclerosis, and cerebral paralysis which were published by Kundrat, Gaudard, Wallenberg, Bourrneille, Jendrassik, and Pierre Marie between 1860 and 1880 (Longo and Ashwal 1993). None of these authors added to the understanding of the pathophysiological mechanisms of cerebral injury. In 1865 Virchow recognized periventricular gliosis, which he attributed to inflammation, and in 1885 Strümpell also noted a cerebral inflammatory process which he believed was caused by in utero infection analogous to poliomyelitis, coining the term ‘polioencephalitis’ (Ashwal 1990). In that same year McNutt published an influential paper ascribing infantile-onset spastic hemiplegia to ‘meningial haemorrhage’ (Longo and Ashwal 1993). William Gowers, among others, agreed with this hypothesis and supported Little’s view on the importance of a history of difficult parturition. It was also Gowers who introduced the term ‘birth palsies’ as a category of deformity and described several patterns of dyskinesia (Gowers 1888). The growing interest in this condition caught the attention of two major figures in the history of medicine—one in the new and one in the old world.

William Oder was born in remote Bond Head, Ontario, Canada, in 1849 and spent a year at Trinity College School in Ontario where he came under the influence of William Arthur Johnson, a noted naturalist who introduced the young Oder to biology and the microscope, which was to remain a lifelong preoccupation. Oder then transferred to Toronto Medical College and onto McGill University in Montreal from which he received his MD degree in 1872. He spent two subsequent years studying in Europe and another two teaching back at McGill before moving to Philadelphia in 1884 to become Professor of Medicine at the University of Pennsylvania. The following year he became a member of the Philadelphia Neurological Society and was appointed to the staff of the Infirmary for Nervous Diseases where he consulted both on adults and on children with neurological conditions.

In 1888 Osler delivered a series of lectures which were subsequently published as a monograph, The Cerebral Palsies of Children (Osler 1889). This treatise established the term CP although both Wallenberg in 1886 and Gowers in 1888 had used it previously (Gilles 2007, Ashwal 1990). Osler distinguished three clinical patterns of CP: spastic hemiplegia, bilateral spastic hemiplegia, and spastic paraplegia, and provided correlative descriptions of postmortem findings. He also associated the clinical patterns of CP with various pathogenetic mechanisms while noting the potential limitations of determining causation from postmortem analysis. He agreed with Gowers that bilateral hemiplegic and paraplegic cerebral palsies might be appropriately termed ‘birth palsies’ and with McNutt that traumatic haemorrhage was one of the chief causes. He acknowledged conflicting theories for the cause of hemiplegia and put forward several potential aetiologies including postnatal infectious and vascular mechanisms.

During this period Bernard Sachs (who was to publish the first textbook on child neurology in 1895) and Frederick
Historical Perspective

Peterson were working at Columbia University in New York. In 1890 they published a detailed review of 140 cases of CP using a similar classification to Osler’s but adding a new term, ‘diplegia’, instead of ‘double hemiplegia’ (Sachs and Peterson 1890). They divided their cases into congenital and acquired forms, provided clinical descriptions, and correlated the various clinical forms with pathological findings and historical details to provide the likely time of onset and putative aetiologies, ‘The child is either born with or in its early life develops some forms of paralysis; a hemiplegia, a diplegia or a paraplegia. In the congenital cases there has been some disturbance during the pregnancy or labour has been tedious and difficult a or definite cause cannot be given. In the acquired cases we have seen that the onset of paralysis may occur after acute infectious diseases, during convulsions, or from causes that cannot be fathomed’ (p. 303).

Sachs and Peterson also emphasized the potential role for vascular insults as frequent causes of the cerebral palsies (Sachs and Peterson 1890, p. 319). In congenital cases, they agreed on the aetiological importance of haemorrhage as well as infection and maternal trauma and hazarded a caution to obstetricians, ‘… we wish to repeat our warning to the obstetrician: Hasten a protracted labour for the skillful use of forceps and careful manipulation are less apt to do injury than the prolonged compression of skull and brain in the pelvic canal’ (p. 329).

Sigmund Freud was another major historical figure of this era. He is best known as the dominant force in early 20th century psychiatry but began his professional life as a neurologist working with children (Longo and Ashwal 1993). Born in 1856, in Freiberg in Mähren, at that time part of the Austrian Empire and now part of the Czech Republic, he was a gifted student who developed proficiency in German, French, Italian, Spanish, English, Hebrew, Latin, and Greek. He entered the University of Vienna planning to study law but switched to medicine and received his doctorate in 1881. He pursued several years of additional study including time in Paris and Berlin before accepting the position as head of a new neurological department at the Institute for Children’s Diseases in Vienna in 1886.

Between 1891 and 1897 Freud published three monographs on CP; the first, written with Oscar Rie, a paediatric colleague from the Institute, reported a group of children with hemiplegic CP. Two years later Freud introduced the concept of ‘cerebral diplegia’ by describing 53 children with CP and combining them under this single term although he commented on the frequency of transitional forms and mixed cases (Accardo 1982). Freud agreed with Little that preterm birth was frequently associated with paraplegic rigidity and that difficult deliveries often preceded the development of generalized rigidity, and with Osler about the questionable validity of using postmortem findings to determine the causes of CP, but

Fig. 1.2. Sir William Osler (1849–1919). Courtesy of the Wellcome Foundation (creativecommons.org/licenses/4.0/legalcode).

Fig. 1.3. Sigmund Freud (1856–1939). Courtesy of the Wellcome Foundation (creativecommons.org/licenses/4.0/legalcode).
importantly challenged their views on causation. Freud suggested that intrinsic vulnerabilities of the child might predispose to brain injury. He further emphasized the variability in outcomes following similar circumstances in stating that ‘… the majority of premature infants and infants with asphyxia secondary to difficult delivery are not damaged by it (‘recover unharmed from that condition,’ according to Little). Thus to the accidental factor of birth trauma must be added a second predisposing factor which combines with the former to produce the diplegia: if this predisposition were sufficiently exaggerated it might suffice by itself to produce diplegia or do so in association with head compression during the normal delivery’. He went on to say ‘… Thus in many cases Little’s aetiology only reinforces the action of congenital factors …’ (Accardo 1982).

Freud’s final monograph on CP, *Die infantile Cerebralähmung*, was published in 1897. Again he stressed the value of classifying the cerebral palsies on a clinical basis and divided CP into subgroups of infantile hemiplegia and cerebral diplegia. He emphasized the frequent lack of an apparent cause for CP and the lack of specificity of perinatal risk factors in its occurrence. He also described separate aetiological categories for congenital, birth-related, and postnatally acquired forms and described familial and hereditary forms of CP, comorbid conditions including epilepsy and intellectual impairment, and treatment approaches.

In general, Freud’s work provided a more theoretical perspective of causation and pathogenesis. His conceptualization of the potential importance of intrinsic biological vulnerabilities to brain injury and resultant outcomes was unique at the time and remains a thought-provoking area of investigation and discussion to the present.

‘Treatments’ for CP were not developed until the end of the 19th century. It was probably a gymnast, Jennie Colby, who began what could be seen as a practical and applied ‘paramedical’ interest when noting that children’s nursery rhymes (rhythm with content and familiarity) helped some of these children move more easily. She also noted that movement difficulty could be related to the child’s intellectual as well as motor disability (Slominski 1984). Later, her work with Mary Trainor (one of the first graduates from a physical therapy school), came to the attention of Bronson Crothers and Winthrop Phelps and was incorporated into what became the physical treatment of children with CP at the Children’s Rehabilitation Institute in Cockeysville, Maryland.

**Broadening the discussion: aetiology, comprehensive care, and Winthrop Phelps**

Entering the 20th century, historical benchmarks become somewhat more subjective. We note the significant contributions of Frederick Batten who described ‘cerebellar diplegia’, the ataxic form of CP, Robert McCarrison who described the occurrence of cerebral diplegia from endemic cretinism (McCarrison 1909), and Cornelia de Lange who described the association of hyperbilirubinemia with intermittent rigidity and opisthotonus (Batten 1903, McCarrison 1909, Ashwal 1990, p. 25). Another noteworthy individual in the early 1920s was British neurologist James Collier. In his Presidential Address before the Neurological Section of the Royal Society of Medicine in October in 1923, Collier expressed his disagreement with Little and Osler on the importance of birth factors in causing cerebral diplegia. He advanced an alternative theory of pathogenesis based on the earlier observations of Brissaud that associated preterm birth and diplegia with the pathological findings of ‘arrested development’ in the pyramidal tract of the spinal cord. This view held that CP was caused by primary degeneration of cerebral neurons of unknown cause (Collier 1923).

Henry Cameron, a London paediatrician quickly challenged Collier’s view in *The Lancet* (Cameron 1923). He argued that CP resulted from brain injury in at least some cases based on his own experience, and he distinguished traumatic and asphyxial causes. Cameron also provided a prescient emphasis on the importance of neonatal symptoms including encephalopathy and seizures in predicting which infants would have adverse outcomes from perinatal events. His descriptions of clinical features and evolution of findings over time can still be read profitably.

In 1927, American neurologist Frank Ford co-authored a monograph on Birth Injuries of the Central Nervous System with Bronson Crothers and Marian Putnam. Ford emphasized the importance of subdural haemorrhage in autopsies of children dying in the neonatal period, attributing those haemorrhages to trauma, and described ‘pallid asphyxia’ calling attention to its relationship to adverse outcomes including CP. He further expressed his opinion that ‘It does not seem probable that asphyxia alone without gross haemorrhage can be of much importance’. Ford concluded that ‘the congenital diplegias which constitute by far the largest group of infantile spastic palsies as seen in the pediatric department (235 out of 280 in all) … are the result of various pathological processes of intrauterine origin’ (Ford 1927, p. 24).

There was limited interest in treatment approaches for those with CP through the first quarter of the 20th century, but this began to change quickly in the late 1930s. Several treatment centres, including the multi-disciplinary clinic at Children’s Hospital in Boston, Massachusetts, were starting, and it was in Boston that physical therapy approaches were pursued in earnest once they were introduced by Bronson Crothers and colleagues (see below). Crothers was one of the first to spend his academic career in the nascent field of child neurology, and he was influential in promoting the value of psychological support for children with disabilities, authoring an early monograph on the subject (McLean 1990). Crothers worked with Richmond Payne during this period to document the natural history of various forms of CP as well as the benefits of physical therapy, and he mentored Winthrop Phelps, an orthopaedic surgeon who was to play a major role in the
development of American views on classification and treatment of CP over the next 30 years.

Winthrop M Phelps, born in Bound Brook, New Jersey, in 1894 was educated at Princeton University and the Johns Hopkins University School of Medicine, graduating in 1920. After postgraduate training in Baltimore and in Boston at Massachusetts General and Boston Children’s Hospitals, he spent a year in a research teaching fellowship in orthopaedic surgery at Harvard Medical School where he encountered the innovative therapy approaches of Crothers and colleagues. In 1931 Phelps joined the medical faculty at Yale University. Phelps’s seminal paper ‘Cerebral birth injuries: their orthopaedic classification and subsequent treatment’ was published the following year and is a landmark in the history of care for those with CP. In 1936, Phelps returned to Baltimore where he established a practice specializing in the treatment of children with CP. He founded the Children’s Rehabilitation Institute in Cockeysville, Maryland, later moving the facility to Reisterstown. Phelps’s institute was both a treatment and training facility, and he worked tirelessly to establish clinics for similar purposes in other major US cities.

Although he acknowledged the neurological basis of CP, Phelps classified all patterns of movement disorders under the rubric of dyskinesia with subgroups of spasticity, athetosis, and others (Phelps 1941). Phelps was a conservative surgeon. His approach to care connected diagnostic criteria to specific modalities of therapy including bracing, nerve blocks, and adaptive equipment as well as ‘muscle education’, and he emphasized physical therapy as a keystone of intervention (Phelps 1950). During the late 1940s and through the 1950s there was an added emphasis on interventional approaches resulting from experience with rehabilitation approaches to casualty management during the Second World War. Phelps and like-minded clinicians used this growing knowledge base to the advantage of the present time as the Kennedy Krieger Institute.

Another important leader in American childhood disability during this period was Meyer Perlstein. A Chicago-educated paediatrician and neurologist, Perlstein was an energetic teacher and early proponent of interdisciplinary diagnostic and treatment teams who cared for thousands of children and adults with CP. One of his significant contributions was a new multimodal characterization of children’s disability based on neuroanatomy, clinical symptomatology, muscle tone, severity, and aetiology (Perlstein 1952). Perlstein also established a CP brain registry in 1949 which was later incorporated into the Armed Forces Institute of Pathology Neuropathology Registry (Ashwal 1990). Crothers, Phelps, and Perlstein, along with Earl Carlson and George Deaver from New York, and Temple Fay from Philadelphia, founded the American Academy for Cerebral Palsy (AACP) as a professional organization devoted to interdisciplinary physician education in 1947.

The challenge of classification: the American Academy for Cerebral Palsy and The Little Club

A survey of the membership of the AACP in the 1950s produced a classification scheme for CP which incorporated multiple categories including motor patterns, topography, aetiology, neuroanatomy, functional capacity, and therapeutic need (Minear 1956). A related aetiological perspective was summarized by Nicholas Eastman in 1954 at a St. Louis meeting of obstetricians in a setting reminiscent of Little’s nearly a century before (Eastman and DeLeon 1955). In his lecture Eastman presented his data from 96 infants born at Johns Hopkins Hospital which indicated that 40% of children later diagnosed with CP had ‘poor’ condition at birth compared with only 2% of more than 11,000 control infants. Eastman called for a collaborative CP registry with the participation of the American Academy of Obstetrics and Gynecology and the AACP, and later served as President of the AACP, the only obstetrician to have done so.

Eastman’s work paralleled Virginia Apgar’s research which used a 10-point rating scale to assess the delivery room status of the infant (Apgar 1953). She correlated delivery room scores at 1 and 5 minutes of more than 1000 infants with outcomes including neonatal death, encephalopathy, and long-term development. This post-delivery scoring system, quickly named the Apgar score, became a standard metric for evaluating the relationships between maternal care and newborn status and led to improvements in maternal, infant, and neonatal care. An unintended consequence of this focus on delivery room status was its apparent support for the notion that intrapartum events were frequently responsible for adverse long-term outcomes including CP—echoing Little’s perspective.
During this period in the UK, Evans, Asher and Schonell, and Wylie brought forward several classification proposals for CP using various combinations of topographic and motor impairment paradigms (Morris 2007). Ingram and coworkers also developed a system using neurological and topographical characterizations and a tripartite severity index which emphasized the need for continuing assessment because of the changing clinical manifestations of CP over time (Ingram 1966).

In the late 1950s Ronnie Mac Keith and Paul Polani organized the ‘Little Club’ in London. This was an international group of physicians who met regularly to discuss issues related to CP (MacKeith et al. 1959, Polani 1959). They made several attempts at definitions, culminating in the 1964 characterization of CP as ‘a disorder of posture and movement due to a defect or lesion of the immature brain’ (Bax 1964). Even the Little Club, however, acknowledged the complexities and inconsistencies in attempts at any classification scheme, concluding that it was ‘impossible to proceed definitively with classifying cerebral palsy’ (Bax 1964).

The aetiological debate continues: the impact of the Collaborative Perinatal Project and beyond

Although treatment options and cultural awareness expanded during the 1950s and 1960s, the debate on causation continued. Lilienfeld, Pasamanick, and others working in the field of epidemiology suggested a new hypothesis to explain the cause of childhood disability. Their influential view was conceptualized as ‘a continuum of reproductive casualty, consisting of brain damage incurred during prenatal and perinatal periods as a result of abnormalities during these periods leading to a gradient of injury extending from fetal and neonatal death through cerebral palsy, epilepsy, behavior disorder, mental retardation’ (Pasamanick and Lilienfeld 1955). This hypothesis fostered the view that prevention of CP and other developmental disabilities could be achieved through maternal health and obstetrical improvements, and culminated in the introduction of electronic fetal heart rate monitoring during labour in the late 1960s. This technological advance has continued until the present, without delivering on its promise to reduce the prevalence of CP (Graham et al. 2006).

Between 1959 and 1966, ‘The Collaborative Study on Cerebral Palsy, Mental Retardation and Other Neurological and Sensory Disorders of Infancy and Childhood’ began enrolling pregnant women in 12 university centres in the USA. Unofficially shortened to the Collaborative Perinatal Project (CPP), this multidisciplinary effort identified more than 54,000 pregnant women, followed them from their first prenatal visit through gestation and delivery, and sequentially evaluated their offspring until age 7. This study led to the identification of 189 children with CP and allowed extensive evaluation of causal and associated factors. The major conclusions of the study, which have appeared in many publications over subsequent years, included two that are directly relevant to the CP causation debate: (1) the events of labour and delivery are not major contributors to the occurrence of CP because most cases have their origins before labour begins and (2) intrauterine inflammation is a major cause of adverse pregnancy outcomes (Klebanoff 2009).

In the laboratory, the research of Windle, Myers, and Brann and others from the late 1950s into the 1970s provided evidence about the mechanisms and neuropathological patterns of cerebral injury resulting from asphyxia in experimental animals. This work established clinical–metabolic correlations associated with focal ischaemia and cytotoxic cerebral oedema (Brann 1988). During the 1970s and early 1980s, a series of influential epidemiological papers using data from the American CPP and the Western Australia CP Registry appeared which challenged the status quo (Nelson and Ellenberg 1986, Blair in Stanley et al. 2000). These and many other publications provided compelling data that only a small minority of cases of CP were causally related to intrapartum events. The parallel realization that the nearly universal use of electronic fetal heart rate monitoring through the 1980s and 1990s, and the resultant marked increase in labour interruption by Caesarean sections, had had no measurable impact on the prevalence of CP also strengthened the view that intrapartum events, or at least those amenable to facilitated delivery, were not the dominant factors in the causation of CP (Graham et al. 2006). The data also indicated that preterm birth and its comorbidities remained the leading risk factor for CP despite the fact that most children with CP were born at or near term and did not have intrapartum risk factors for brain injury. Causation is clearly not a unitary phenomenon, nor is it currently known for most individuals with CP.

The physical treatment of the motor disorders

With respect to the children’s physical management, there was a growing international awareness of children with CP and their

Fig. 1.5. Ronnie Mac Keith (1908–1977).
need for help during the period between the World Wars (1919–39), but what sort of help and by whom was not immediately obvious. Worldwide individual therapists, local parent groups, local charities, hospitals, and medical (usually orthopaedic) clinics adopted a variety of treatment programmes. In the USA, prominent among them was the Children's Rehabilitation Institute regimen. It was probably the most purposefully organized and became a favoured training for occupational and physical therapists wishing to treat children with CP.

So place-by-place intervention would vary to meet the perceived needs and the available local facilities; for example, by the mid-1930s in the USA there were already clinics or hospital-based programmes specifically for CP in 24 States (Report of Committee on Research and Efficiency 1937). Therapists too were creating a demand for structured information on how to treat these children (McPeek 1929, Rogers and Thomas 1935, Girard 1937, Phelps 1941), but research on treatment effect was not so in evidence, although the need for a structured assessment was clearly emerging (Sirkin and Whitfield 1936).

However, at that point the Second World War (1939–45) put a hold on most development, although in 1943 Eirene Collis, a physical therapist and occupational therapist, had set up the UK's first CP clinic at Queen Mary's Hospital for Sick Children, Carshalton, Surrey, and by 1947 published A Way of Life for the Handicapped Child. In the same year, Gesell and Amatruda published their second and strongly revised edition of Developmental Diagnosis, which became one foundation stone of the examination of the infant and young child. In general, treatment had more discrete aims and so used more longstanding and general intervention methods: massage, passive movements, gross movement synergies, strengthening individual muscles and functional movement patterns, balance training, stretching tight soft-tissue structures (including plastering, and orthotics), modified seating, walking aids, and equipment and training for daily living activities.

By the early 1950s several authors had discussed the therapeutic use of various fundamental locomotor reflexes and postural responses (Obholzer 1954). Of these, subsequently, the most influential was possibly Temple Fay (1948), who included the concept that ontogeny needed to replicate phylogeny, and that the phylogeny included sequential modes of locomotion.

A desire for change: treatment insights, rationales, and hypotheses

It was around this time that among health professionals there appeared a readiness for change as CP came to be viewed as a disorder demanding a new and a radically different habilitation approach and several (mainly individual) clinical insights, rationales, and hypotheses were published. Karel and Berta Bobath (1950) described their use of reflex-inhibition, Margaret Knott (1952) wrote about the proprioceptive neuromuscular facilitation approach to the treatment of CP, and András Petö established the National Institute for Motor Therapy in Budapest, Hungary, subsequently publishing his educational approach to CP (Petö 1955). Temple Fay (1954) published on the use of pathological and unlocking reflexes (the precursor of Glenn Doman's and Carl Delacato's, and Vaclav Vojta's, treatment techniques). These were followed by Margaret Rood (1956) describing her topical muscle facilitation techniques and Signe Brunnstrom (1956) her methods of muscle facilitation. The following year Karel and Berta Bobath (1957) described their treatment of CP.

While therapists began to absorb these challenges to their rationales, Vaclav Vojta (1965) published his methods of relaxing spasticity, Jean Ayres (1966) her views on the interrelationships among perceptual motor functions in children, and Adriano Milani-Comparetti and Anna Gidoni (1967) showed the significant differences within hypertonic CP, describing a locomotor examination that highlighted the different postural reactions (and so potential to function) of those with spastic and ‘dystonic’ hypertonia.

However, therapy is nothing if not practical and the written word was never enough. One of the major influences on the overall management of CP from around 1960 was the increased availability and widespread uptake of postgraduate training courses. Some taught a treatment to achieve a specific and topical aim (e.g. facilitating a muscle or muscle group’s activity). Others, while having their specific techniques, carried with them an ethos such that, knowing a therapist’s allegiance, one could assume their response to almost any clinical situation or treatment option. Foremost of these were Bobath and Vojta, and an adherent of the one would find little of value in the other. The widespread influence of the Bobaths’ courses (Scrutton 1991), in advocating that all physical management had to relate to the whole child and to involve the parents, gradually and helpfully infiltrated all parts of the health team, allowing a more rounded discussion of the child’s total management.

Medical and social changes and their effects on the management of cerebral palsy

What had led to this widened outlook and exploration? Between about 1945 and 1965 several important and relevant changes were occurring. First, knowledge of many aspects of child development was growing rapidly and its relationship to treatment was becoming blatant. Everyone now needed a broader knowledge base. Second, knowledge of how early upper motor neuron lesions influenced the development of voluntary and involuntary movement during infancy was advancing. Next, during the 1950s, neonatal care was significantly advanced by more structured examination of the newborn infant (Appgar 1953) followed by risk-related neonatal follow-up (Eastman and DeLeon 1955). Together these introduced a change in the clinical presentations of CP, resulting in different treatment problems. At
Thus discussion, which had previously been about achieving specific aims, changed to which school of thought was ‘correct’; and it rapidly became little more than ‘ours is better than yours’. Had it not been so negative and time-consuming, it might have been amusing; instead it was frustrating. Reading publications of that era now with their measured language gives little insight into the antagonism displayed in many meetings. Facts were few and so opinions thrived as each group strove to ensure that the children received what each considered the most appropriate treatment. It may have been a small world, but it was a vibrant one, which helped create where we are today.

Those who combined these new therapies with a family-oriented approach did so from their earlier treatment experience or naturally out of themselves; for advising the family of easier and perhaps more therapeutic ways of handling their child in day-to-day situations seemed to be no great part of these new methods, which, in the main, were clinic-based. Those new treatments that were strongly home-based were designed so as to use the opportunity for intensive parental treatment sessions, rather than adapting the child’s therapeutic care within a more harmonious family life.

None of these new treatment ‘methods’ was in overall advance of the others. A method advocating treatments that would be considered acceptable today might combine them with practices seen as outdated or unacceptable even at the time. No one treatment held all the good cards, and many experienced therapists mixed and matched parts of these therapies to suit the child, the family, or their own training, experience, and circumstances. It was this need to recognize some ‘good’ in many, but not all, treatments that was exemplified by much of Sophie Levitt’s work (Levitt 1977). Some alternative ways to view treatments were put forward (Shepherd 1974, Scrutton 1976) but it was stony ground, one trod gently, and little grew.

Another aspect of this overall uncertainty was that there were several treatments that were frankly abhorrent to many health workers, who considered their regimens at best disadvantageous to the child or family and at worst abusive. There still are such ‘treatments’, as well as some with untenable rationale and well-documented problems.

**The emergence of disunity over aims and means**

However, quite separate from these background changes, there arose open and frank disagreement about the treatment these children required. The wide variations in these treatment methods was caused in part by their differing treatment aims; but underlying that was a disagreement (sometimes open, but often implied) about what it was possible for treatment to achieve. These differences were wide, from the ability effectively to remove the CP completely, to ameliorate it, to accommodate life to it, or to do no more than to prevent its worst developmental effects. Allied to this was the question of how to judge treatment success.

**HOW STRICTLY SEQUENCED IS GROSS MOTOR DEVELOPMENT?**

There was strong disagreement about the use of primitive patterns of movement, mass patterns, and primitive reflexes/responses. Was stimulating creeping movements, for instance, just that and no more, or was it helping, some would say essential for, an infant to progress through to a more developed gross locomotor stage? This latter view was held quite dogmatically as a fundamental truth about how gross movement and postural ability usually develops. But there was also a more general undercurrent of adherence to the concept that there is a ‘correct’...
sequence of movement development and that children must, for example, fully develop crawling before 'being ready' or even being 'allowed' to walk; this influenced some treatments more than others. How important is it for a child to follow the usual gross locomotor developmental sequence? Those who observed infants saw a predominant (usual) sequence but not an essential one, as there are several other pathways to the same end. In addition, locomotor development could be seen, not only as sequential neurological maturation but also as a natural biomechanical one of learning to cope with a rising centre of gravity and a narrowing base.

**Strengthening and Spasticity**

There was also strong disagreement about the effect of muscle strengthening. It had long been known that by resisting hip and knee flexion the flaccid ankle dorsiflexors could be activated. But was that desirable? The inactive muscle was being taught to work, but was that detrimental because it reinforced the early developmental flexor synergy? Spastic muscles are also weak muscles. Does strengthening them make them more spastic or fitter for function? From the 1930s, strengthening muscles and whole functional movement synergies were a part of most treatment programmes. Then, in the early 1960s, mainstream opinion changed; strengthening muscles became 'facilitating spasticity' and became a minority activity for about the next 25 years. Growing research was already gradually changing this when Damiano and Abel (1998) demonstrated strengthening's considerable benefits to functional status and overall health and well-being.

**Deformity**

What was the correct treatment for fixed deformity? Should the shorter muscle/tendon be stretched or did that increase spasticity and encourage further deformity? Would 'night splinting' be more effective because the child was relaxed and it was for a longer period? If it should be stretched, how much stretch and for what length of time (Tardieu et al. 1988)? Would more permanent 'splinting' from infancy be preventive, or was deformity inevitable and to be corrected only by surgery when and where it is advantageous? For many until the late 1960s the answers to these questions were considered to be 'known', as the upper motor neuron deformities of CP, about which there was little experience, were equated to those of lower motor neuron poliomyelitis, which at that time were considered to be well understood (Sharrard 1979). The realization and understanding that, unlike polio, lengthening one muscle could change the mass patterning of a limb or throughout the body took a little time, and the treatment aims and methods needed to adapt. Words, too, dictated how the problem was viewed: 'contracture', a word still commonly used in CP, fails to describe what happens in the child, implying that the muscle/tendon, which had been long enough, has shortened rather than failed to have grown proportionately with bone length. It also allows us to forget the deformity of the opposing muscle group: overgrowth.

**Polypropylene Changed Orthotic Usage**

In the late 1960s orthoses changed radically: polypropylene ankle–foot orthoses (Yates 1968), light and hidden, replaced the metal and leather 'below knee irons'. Although these had been easily re-shaped and adjusted for growth, they were costly, slow to make, heavy, and socially stigmatizing. Drape-formed polypropylene ankle–foot orthoses were soon further improved by vacuum forming. They brought many advantages, their lightness and look made them more acceptable to school-age children, and they could markedly improve gait, particularly that of children with hemiplegia. Their disadvantage was that they could not be altered and had to be re-made for adjustment or growth.

**The Family, Society, and the Educational Establishment**

There were different views about the role of the family. The Children's Rehabilitation Institute and Eirene Collis had advocated 'parent advice clinics' for discussion and advice about what each family could contribute to the treatment programme. These were an integral part of treatment in many clinics throughout the world, often combined with home visits; and later on, two programmes were constructed around the parents being trained by a therapist to be the therapist and do all the treatment. Some therapists disagreed strongly, considering that treatment should be an entirely clinic-based activity because a therapist's skill was essential to an effective treatment. A balance was needed and perhaps Nancie Finnie's book *Handling the Young Child with Cerebral Palsy at Home* (Finnie 1968) helped more rational discussion as better-informed parents felt able to demand they had a role and choose its extent. Her book also opened the eyes of many therapists to how much of their treatment was a combination of experience and applied common sense. How much can be expected from each family or school will always be an important discussion, some aspects of treatment being more readily transposed to the home or school setting, and perhaps this is the dividing line between treatment per se and physical management.

History has also shown us that the straightforward export of a treatment system from one society to another may be inappropriate. How people actually live day-to-day is markedly different from place to place throughout the world, and although the aims and specific treatments for certain aspects of CP can be universal, the day-to-day physical management needs are unlikely to be similar. Once shown what is required and why, a community based around village life can be far more supportive, therapeutic, and socially integrating for the child and family than, say, the environment of a high-rise apartment block in a modern city. A lecture on seating is not easily taken back to a society without chairs.
However, perhaps the most challenging and enduring question for the entire health team arose in the 1950s when András Pető put forward that these children’s habilitation programmes should be planned and performed by educationalists rather than a health team; health having an important role where its various skills were needed, but not the primary one. His concept of the ‘conductor’, being part teacher, part therapist, and part something new, brought to mind that in the 1940s Eirene Collis had advocated that children with CP needed ‘CP therapists’ who would combine the skills of occupational and physical therapists and perhaps a bit more besides. For an educationalist to assume the leading role remains a challenging concept to most health professionals and one possibly in need of clearer definition.

**Change, confidence, and a lack of evidence**

The greatest influence in this story has been the changing ‘climate of professional opinion’. In very broad terms, from beginning of the 20th century up to around 1950, everyone was united in trying empirical treatments with a few assessing the outcomes (Crosland 1951, Karlsson et al. 1960); however, the assessment methods used what would now be considered unsuitable. Then, for about the next 30 years, several eponymous treatments (certainly no better tested and proven than what had preceded them) somehow held sway. Not for all therapists of course, but it was a hard fight to oppose their evangelistic dogmatism. What goes around comes around and by the end of the 1980s the balance had tipped and for health workers an acceptable treatment was becoming an evidence-based treatment. Parents of children with CP, however, will always be vulnerable to those with charisma and the unfounded belief that they know how to cure CP.

‘Cure’ is a strong word, often carefully avoided; but there were those who claimed explicitly that they could rid a child of their CP if they could start treatment ‘early enough’; and, more circumspectly, several others who implied something very close to that for aspects (but not the entirety) of CP. How could such a situation have arisen?

Inexperienced therapists seeking an effective treatment could easily be impressed by demonstrations of patient handling that could show immediate and marked effects: producing limb and trunk movement synergies, reducing spasticity, or allowing better postural control. These were impressive demonstrations of ‘effect’ and it could be churlish to ask ‘so what?’, even though no evidence of long-term effect was ever forthcoming. More ‘traditional’ treatments, based only on having long-term results, could show few such immediate effects, and a treatment demonstration followed by ‘… and we do this for 15 years and it prevents equinus deformity’ was never a winner, particularly as there was no good evidence for that treatment either.

A therapist is faced day-to-day with the complex and multiple problems these children present together with the questions, demands, expectations, and disappointments expressed by worried and often exhausted families. Many therapists, without facts or research to guide them, needed something or somebody with ‘authority’ to assure them that they were treating correctly. Some were fortunate to find that within their team. Many could find it only in one of the eponymous treatments, usually one that matched their skills, personality, or practice. It was not a good solution, but it may have been the only one possible at that time. Unfortunately, these systems/methods/approaches became so well established that together they stifled much original thought and set back research for possibly 30 years. Today it is no longer acceptable that a paper on treatment, without any established evidence of efficacy, should be accepted for publication and be widely taught as being the ‘correct’ treatment, allowing its founder and adherents to hold others to public scorn as being ‘less enlightened’. That happened and thankfully that is history.

The process of more rigorous searches for evidence-based medicine (Sackett 1996) and the development of measurement tools are, for CP, epitomized by the American Academy for Cerebral Palsy and Developmental Medicine’s Methodology for Developing Evidence Tables and Reviewing Treatment Outcomes Research approved by their Treatment Outcomes Committee in 1999, setting a long-term and, inevitably, continuous process.

Throughout this story one feature has been consistently present: peer pressure. It should serve to remind us that, perhaps in a different guise, the sieve of dominant opinion is probably as much a part of today’s world as it ever was of yesterday’s.

**Changing views on disability, the impact of recent research, and current thinking on an aetiology**

Significant changes about perceptions of disability have occurred over the past several decades. This is based on the paradigm shift from a deficit-centric model of description to the World Health Organization conceptualization focused on body structure and function, activity limitation, and participation with contextual influences of environmental and personal factors (World Health Organization 2001). This has been operationalized in the International Classification of Functioning, Disability and Health. Frameworks for functional measurement and family-centred care for children with CP have entered the mainstream through the influential contributions of the McMaster University CanChild group and their disciples, and a concomitant focus on evidence-based and best practice approaches that have assumed primacy around the world (Rosenbaum et al. 1998, King et al. 2004, Palisano et al. 2004). Major progress has occurred in the standardization of clinical assessment of motor function using descriptive and evaluative (i.e. change detecting) measures like the Pediatric Evaluation of Disability inventory (PEDI) and Gross Motor Function Measure (GMFM) (Haley et al. 1992, Russell et al. 2002). Another instrument, the Gross Motor Function Classification System (GMFCS), is a descriptive tool that is now used.
worldwide to classify the functional status of individuals with CP using a five-level scoring system based on functional motor levels (Palisano et al. 1997, 2008). More generally, there have been important developments in appreciation of the need for relevant outcome measures both in the short and longer term (see Chapter 20).

Increased access to high-quality neuroimaging has moved us away from postmortem material, which has become of limited availability, to real-time assessment of brain structure. In addition, advances in biochemistry and molecular biology have improved the understanding of mechanisms of injury and malformation, and opened the door to preventive treatment strategies (Krageloh-Mann et al. 1995, Ferriero 2004, Bax et al. 2006).

Nonetheless, the lack of universal agreement about definition and classification of CP has continued. Within the past decade, another consensus-based definition has been published (Rosenbaum et al. 2007). The new definition represented the efforts of a large multinational committee working for several years to achieve consensus. The annotated monograph, published with attached commentaries including some dissenting opinions from major researchers and clinicians, provided a definition premised on the World Health Organization perspective and emphasizing the importance of comorbid impairments: ‘Cerebral palsy describes a group of permanent disorders of the development of movement and posture causing activity limitation that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication and behaviour, by epilepsy and by secondary musculoskeletal problems.’

A proposed classification statement was attached and included four primary components: (1) motor abnormalities, (2) accompanying impairments, (3) anatomical and neuroimaging findings, and (4) causation and timing (Rosenbaum et al. 2007, p. 12).

With regard to the centuries-long debate on the pathogenesis of CP, the quest for understanding continues. Extensive epidemiological research over the past 30 years has led to an understanding of aetiology based on appreciation of risk factors for CP, particularly with respect to prenatal, intrapartum, and immediate postnatal circumstances (O'Shea et al. 1998, ACOG Task Force 2014). The cause of CP in most individuals remains unknown. Rather than a single cause it is now clear that CP results from causual pathways involving multiple aspects of gestational risk factors including in utero environmental exposures and intrapartum stresses and genetic vulnerability (Stanley et al. 2000, Mantovani 2007). Future research using descriptive epidemiology, evolving neuroimaging techniques, and newer histopathological, genetic, and immunochemical technologies continues to promise enhanced understanding and the hope for the ultimate prevention of CP.

The history of CP is a history of the gradual recognition that there exists a group of different but not disparate disorders which have enough in common to claim a single name, but enough differences in causation, presentation, and intervention needs to be the source of endless discussion. Its definition is debatable, its classification difficult, its movement disorders various, its causes heterogeneous, and its social impact considerable. There is hardly any area of expertise in health, education, family dynamics, or social need it does not touch. Nevertheless, ‘history’ continues and never before have we had such investigative tools, research methodology, and means of communication to allow us to unravel, bit by bit, what we need to do: wherever possible to prevent it, to discover what we can and cannot achieve through treatment, and to make society aware of the need to organize essential care, family support, and social opportunity for all those who need it.

The early part of the 21st century has seen further developments in the promotion of function and participation for people with CP. Examples detailed in other chapters in this book include consideration of a lifelong perspective, with specific regard to transitions and ageing, specific use of technology to enhance each of the domains of the International Classification of Functioning, Disability and Health, as well as societal changes that relate to the lives of those with disabilities.

Acknowledgements
We acknowledge the assistance of Peter Blasco, who provided historical materials which were helpful in preparing this chapter.

REFERENCES
Historical Perspective