History of acute anterior poliomyelitis: observations in the 19th century and first half of the 20th century

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ABSTRACT

Humanity has been scourged by poliomyelitis for millennia; the great success of its prevention with vaccines is tempered only by the failure of its total eradication. It has been a long journey: in the early 19th century, the concept of paralysis still lacked a clear definition, and only the genius of Charcot demonstrated the relationship between the anterior horn of the spinal cord and atrophic paralysis, differentiating between acute forms, which are inflammatory, and chronic/progressive forms, which are degenerative. Poliomyelitis often appears in epidemic outbreaks, and authors in the early 20th century mention its supposed rareness. In the first years of the 20th century, Allen Starr gathered data on polio outbreaks in small populations, but also in large cities, with an outbreak in Copenhagen causing hundreds of deaths. Since the identification of the three poliovirus serotypes, the disease has been eradicated worldwide, with limited endemic foci in some specific countries. The long history of this persistent infection includes the failures of electrotherapy, the primitive "iron lungs," and hydrotherapy sessions, which were immortalised by the painter Joaquín Sorolla. Post-polio syndrome, in which the disease appears to progress with worsening of disability after a long period of stability, is a novel challenge. The re-emergence of the disease in impoverished populations, despite inoculation with live virus vaccines, represents another new challenge we face in our days.

KEYWORDS

Acute anterior poliomyelitis, healthcare, epidemics, history, Charcot, Sabin, intensive care, disability, post-polio syndrome

Messieurs, ici se présente le fait capital dans l'histoire de ces lésions systématiques de la substance grise […], le caractère anatomique de se circonscrire à la substance grise antérieures systématiques […]. Il faut ajouter encore le qualificatif aiguë ou chronique.
Charcot, 1876-1880

Introduction

Poliovirus is a small RNA virus with a genome of approximately 7500 nucleotides, and three serotypes of different levels of virulence. Infected individuals carry the virus in the throat and intestine, transmitting it through the faecal-oral route (eg, contaminated drinking water). Poliomyelitis has caused epidemic outbreaks, which were generally seasonal, and have sometimes resulted in massive infection of large populations. Historically, it remains an endemic disease in countries with deficient hygiene measures and infrastructure. For instance, in September 2024, 134 cases were reported in 5 different countries in the Sahel and Chad region (Cameroon, Mali, Nigeria, Central African Republic, and Chad), and the Gaza Strip, currently suffering a cruel war.

Acute anterior poliomyelitis has existed since deepest antiquity. A carved stone stele dated between 1403 and 1365 BCE depicts the priest Ruma with equinovarus foot

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Figure 1. Left: Egyptian stele depicting the priest Ruma with probable sequelae of poliomyelitis, worshipping the gods. © Ny Carlsberg Glyptotek, Copenhague. Right: amphora depicting the mythological hero Heracles (or Hercules, for the Romans) greeting Geras, portrayed as an old man with kyphosis and sequelae of poliomyelitis. © Museo Nazionale Etrusco di Villa Giulia

and a short, withered leg, walking with a cane (Carlsberg Museum, Copenhagen).[1] Merneptah Siptah, seventh Pharaoh of the 19th Dynasty of Egypt, whose mummy is displayed in the National Museum of Cairo, may also have had poliomyelitis. An even more realistic portrayal appears on a Greek amphora dated 480 BCE, found in Etruria, now central Italy. The scene shows the mythological hero Heracles (or Hercules) greeting Geras, the god of old age, portrayed as an old man with a shortened leg, equinovarus foot, and marked kyphoscoliosis (Figure 1).[2]

Material and methods

A total of 27 neurological works from the 19th century (n = 9) and early 20th century (n = 18) were reviewed (1843-1950). The review included textbooks, monographs, chapters, and excerpts written in German, French, Spanish, and English, from the personal library of one of the authors (SGR). We selected works that explicitly mention involvement of the anterior horn of the spinal cord, under one of the many names that have been used for acute anterior poliomyelitis over the years. The findings are analysed and compared to data from the modern literature.

Results

Poliomyelitis in the 19th century

The initial historical problem consisted of distinguishing between processes affecting the anterior horn of the spinal cord with neuronal depopulation and causing paralysis and muscle atrophy. The disease may present acute onset (Heine-Medin syndrome), in association with inflammatory pathological changes, or show a chronic-progressive course, sometimes familial, with neurodegenerative spinal cord alterations.[3]

In addition to Jean-Martin Charcot (1825-1893), other authors describing these processes in the 19th century include Jean François-Amilcar Aran (1817-1861) and Guillaume Duchenne de Boulogne (1806-1875), in


TRATADO COMPLETO
DE
PATOLOGIA GENERAL
POR A. F. CHOMEL,
CATEDRATICO DE LA FACULTAD DE MEDICINA DE PARIS &c.
Tercera edicion
enteramente refundida por el mismo.
NUEVA TRADUCCION CON NOTAS Y ADORNADA CON EL RETRATO Y BIOGRAFIA DEL AUTOR
POR DON ANTONIO SANCHEZ DE BUSTAMANTE,
ANTIGUO ALUMNO DE LA FACULTAD DE MEDICINA Y HOSPITALES DE PARIS, SOCIO DE MERITO DE LA ASOCIACION MEDICA DE JEREZ DE LA FRONTERA.
Obra adoptada por la Direccion general de Estudios para su respectiva asignatura.
MADRID:
LIBRERIA DE LOS SEÑORES VIUDA DE CALLEJA É HIJOS.
1843.

Figure 2. Left: Antoine François Chomel at around 30 years of age, according to the text. Right: title page of his treatise *Elements of general pathology*; Spanish edition printed in Madrid in 1843.

France, and by Guido Werdnig (1844-1919), in Austria, and, separately, by Johann Hoffmann (1857-1919) (types I and II), in Germany in 1886. The pathological findings enabled differentiation between acute poliomyelitis, in which neuron loss in the anterior horn is associated with inflammatory changes, and chronic-progressive forms, sometimes familial, in which neuron loss was neurodegenerative (Charcot, 1876-1880).

For Charcot, the founder of the anatomical-clinical method in neurology, the disease he called *paralysie spinale de l'enfance* was interesting not only for its peculiar clinical course, with abrupt onset, as described by Duchenne de Boulogne and Heine, but also for its prototypical nature, which enabled him to relate paralysis with muscle atrophy to specific lesions to the anterior horn of the spinal cord.[3] Thus, he identified chronic adult forms, often combined with degeneration of the corticospinal tract,[4] an entity he designated amyotrophic lateral sclerosis, and distal forms of familial muscle atrophy, which he described together with Pierre Marie in 1886, the same year that the English physician Howard H. Tooth characterised a similar condition as peroneal muscle atrophy.[5,6]

Another problem was outstanding at the starting point: the concept of "paralysis," which, before Charcot, was far from clear. For instance, in the work *Elements of general pathology* by the eminent physician Auguste François Chomel (1788-1858) (Figure 2), whose third edition was published in Madrid in 1843,[7] syncopal loss of consciousness and coma are jumbled confusingly with hemiplegia and paraplegia. In one excerpt, Chomel writes that:

> The complete abolition of muscular contractility and voluntary motion constitutes paralysis; a term applied to the loss of sensation as well as of motion […]. Paralysis is general in comatose affections, syncope, asphyxia, etc. If it affect but one side of the body, it is called hemiplegia; if the lower half, paraplegia, or paraplexia […].

We may suppose that the latter would be the label applied to children with acute infantile paralysis.

The Madrid edition features a curious full-page dedication by the translator, Antonio Sánchez de Bustamante of the Medical Association of Jerez de la Frontera, an institution to which he dedicates his work "to show my great gratitude and respect, as a meritorious member." The work, published over 180 years ago, is notable due to its semiological refinement, despite medicine's weak conceptual foundation at the time. Surprisingly, there is no specific allusion to such a striking entity as acute spinal paralysis of infancy. Chomel benefited from the teachings of great French masters of the time, including Pinel, Corvisart, Laënnec, and Boyer at Paris' Hôtel-Dieu hospital, as well as lessons at Hôpital de la Charité. He is considered a follower of Bichat due to his studies in anatomical pathology.

Others failed to identify such a dramatic morbid process as acute anterior poliomyelitis. In their work *Handbuch der inneren Medizin* (Manual on internal medicine), published as late as 1922, Mohr and Staehlin were not aware of the disease, unlike Charcot-Marie-Tooth disease and other degenerative muscular atrophies.[8] It is indeed surprising that a disease like acute anterior poliomyelitis, which condemned thousands of children to live with terrible paralyses and deformities, should have escaped the awareness of such distinguished instructors as Chomel in 1843, and Mohr and Staehlin as late as 1922. In the final years of the 19th century (1894), Möbius, in Leipzig, continued to consider it a "rare disease," though he did not suggest that it was non-existent or unknown.[9]

From "rare disease" to limited epidemic outbreaks

Paul Julius Möbius, in Leipzig, wrote in 1894 that: "The sudden appearance of inflammation of the anterior horn in children, generally with febrile symptoms, is so characteristic that it is easily recognised." What is surprising is that he should dedicate only 18 lines to *poliomyelitis acuta* (original italics) in a volume of over 400 pages.[9] A similar phenomenon occurs with the heavy volume (830 pages) on diseases of the nervous system published the same year by Debove and Achard.[10] In a chapter on paraplegias, they openly confess that "acute spinal paralyses in children and adults are not the most common." They do not describe the rarity of acute anterior poliomyelitis due to lack of knowledge; in fact, they are well familiarised with the disease and accurately describe its symptoms. The answer is simply that, in the late 19th century in Leipzig and Paris, there may not have been polio epidemics.

A different perspective is presented by Allen Starr (1854-1932), professor of neurology at Columbia University, New York, who in 1903 reached the conclusion that anterior poliomyelitis was not a hereditary but rather an epidemic process. In other words, clustering of cases was explained not by the existence of a shared gene in certain populations, but rather by more or less circumscribed epidemic outbreaks, which were particularly frequent in the summer.[11]

The data in the literature were accurate; for instance, we may cite the pioneering observation reported by Oskar Medin in Umeå, Sweden, in 1881. The German internist Kindborg deduced that "there is probably a specific source of infection in that country," that is to say, infection by one of the three responsible poliovirus-es.[12] Starr himself witnessed a widespread epidemic in 1895, which affected the small rural town of Otter Creek Valley (Vermont, United States). The 144 cases detected between July and September were concentrated within a radius of barely 12 miles (19.3 km).[11] Today, we would suspect a source of infection near these populations, and a contamination of drinking water by one of the three strains of poliovirus.

The 20th century saw large poliomyelitis outbreaks in Europe and North America. Historically, the calamitous outbreak that shook the population of Copenhagen in 1952 was particularly relevant, not only due to its gigantic proportions: 30-34 patients were admitted per day to the city's Blegdam hospital; of these, some 300 patients presented bulbar involvement and respiratory difficulties. Mortality in this subgroup was 85%-90%. Shifts of up to 250 students were mobilised to inflate ventilation bags. This would not be the first time in history that hospital wards were overrun with paralysed children. The negative pressure system known as the "iron lung," in which the child was shut in a claustrophobic capsule (Figure 3) gave way to positive pressure systems using an endotracheal tube. This system, proposed by Björn Ibsen, was crucial in dramatically reducing mortality rates. This development is considered to represent the origin of intensive care units.[13]

In an excellent review article, Zafra Anta et al.[14] signal the main historical milestones of poliomyelitis in Spain: the first communication by Vilches at the institute created

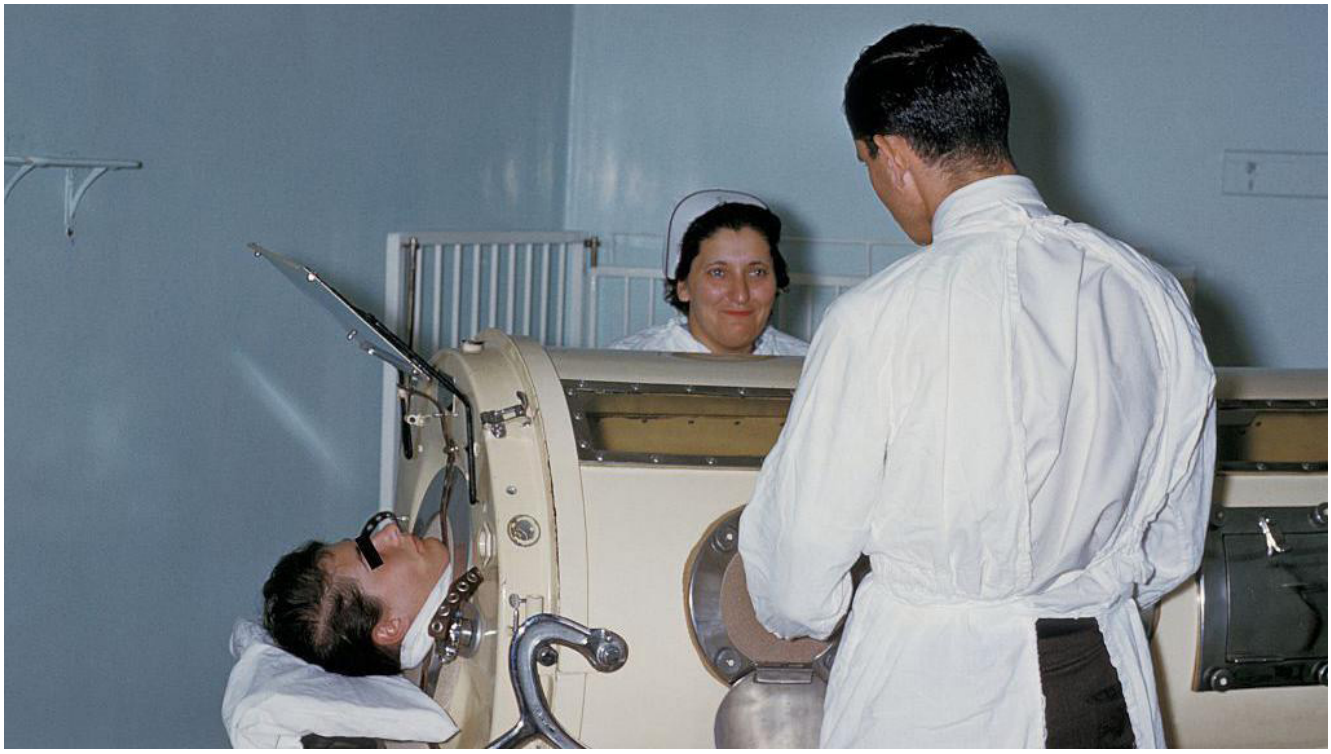

Figure 3. Patients with severe respiratory failure due to poliomyelitis were treated with the "iron lung," according to Kelly.[13] © Centers for Disease Control and Prevention's Public Health Image Library.

in 1827 by the Cádiz physician Federico Rubio y Galí in Madrid, and published in the institute's official publication, *Revista Iberoamericana de Ciencias Médicas*, in 1904; the inauguration of the first iron lungs at Hospital del Niño Jesús, Madrid, in 1951; and the first mass vaccination campaign in Spain in 1963.

Electrotherapy

One of the pioneers of electrotherapy was Eduard Hitzig, who with Gustav Fritsch in 1870 had used electrical stimulation to demonstrate the excitability of the canine motor cortex. After the end of the Franco-Prussian War, Hitzig worked as a general physician and psychiatrist in Berlin, applying the anatomical and physiological experience from his experimental research to his own patients.[15] Strikingly, Debove and Achard dedicate considerable space in their extensive 1894 work to electrodiagnosis and electrotherapy, which occupy 133 pages of the total 782.[10] Electrotherapy was becoming fashionable. In the early years of the 20th century, true treatises on electrotherapy were published, such as the 1908 work *Electrical treatment*.[16] For instance, we may cite the complicated, insightful monograph *Éléments d'électrothérapie clinique* published in 1906 by Adolphe Zimmern, a former hospital physician from Paris. Zimmern presented the work at the French Academy of Sciences with the intention of being admitted to the academy, but was unsuccessful. He used costly apparatus for electrodiagnosis (seeking a "degeneration reaction") and electrotherapy, which he particularly applied in general neuromuscular diseases. Jaime Vera López (1858-1918), a renowned politician and eminent neuropsychiatrist from Salamanca, was enthusiastic about electrotherapy, which he called the "true pharmacopoeia." In 1890, he developed an electrotherapy clinic at the Hospital Provincial de Madrid, with the

Figure 4. Joseph Charrière's electrotherapy machine, similar to that used by Duchenne de Boulogne to treat neuromuscular disease. © Le Compendium. Albert Balasse.

support of the local authorities.[17] This reflects the enormous popularity of the technique, which was doubtless born of social need, as was the case of poliomyelitis.

Charcot created an electrotherapy section at his department, which was perhaps intended more for his patients with hysteria at La Salpêtrière, placing Romain Vigouroux (1821-1911) in charge. He was the official successor of Duchenne de Boulogne, who used the system developed by Joseph Charrière.[18] In Spain, Barraquer Roviralta offered excellent clinical outpatient treatment for neurological patients at his Electrotherapy Dispensary at Hospital de la Santa Creu in Barcelona in 1882, which was subsequently converted into a mixed neurology and electrotherapy service.[19] The enormous popularity of electrotherapy (its real efficacy has never been demonstrated) may perhaps be attributed to the magic of observing the jerking of patient's paralysed muscles after receiving a shock from the mysterious apparatus that Duchenne showed off in the wards of La Salpêtrière ("the little man with his box," as the interns called him) (Figure 4).

The viral aetiology of poliomyelitis. Román Alberca's neuroaxitis ectotropas

The biography of the Spanish researcher Román Alberca Lorente (Alcázar de San Juan, Ciudad Real, 1903 – Valencia, 1966) is similar to those of other neuropsychiatrists of his day, such as Gonzalo Rodríguez Lafora and Dionisio Nieto Gómez: they trained at the Hospital Provincial de Madrid, a breeding ground of neurology and psychiatry, with José Sanchís Banús at the neuropsychiatry clinic and Del Río Hortega at the neuropathology laboratory, at a time when the latter had moved to the Residencia de Estudiantes after his forced departure from the Laboratorio de Investigaciones Biológicas.[20] Alberca's interest in infectious diseases led him to travel later in his career to the Institut Pasteur in Paris, where he studied with Constantin Levaditi and wrote

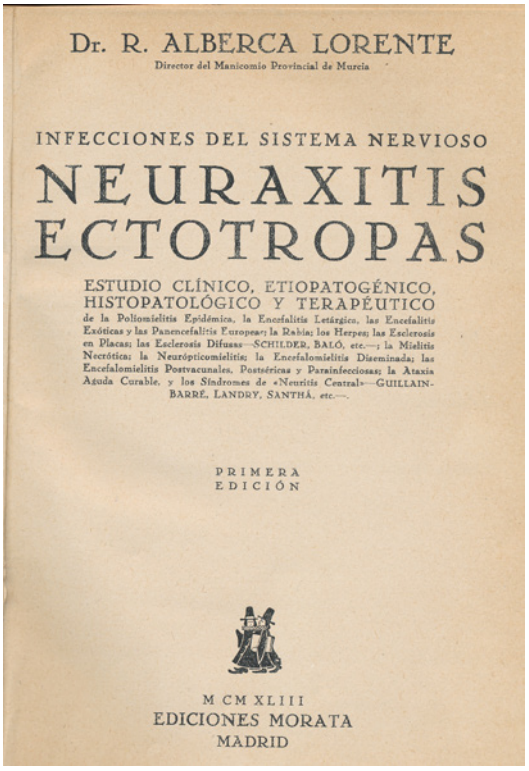

Figure 5. The neuropsychiatrist Román Alberca Lorente (1903-1966), circa 1940. Image taken from Fernández Villalba and Herrero Esquerro.[20] His book *Neuroaxitis ectotropas* (1943) is an essential reference in the history of poliomyelitis.

his monumental work *Neuroaxitis ectotropas*, published in 1943. He wrote 17 dense pages on acute anterior poliomyelitis, saying that it was "still a young disease." He was concerned with forms whose manifestations were thought to go beyond the anterior horn of the spinal cord, which manifested with isolated Babinski sign, and cerebellar and mesodiencephalic forms, which presented with tremor, parkinsonism, or narcolepsy, all of which could give rise to misdiagnosis (Figure 5).[21]

In the first third of the 20th century, experimental studies with monkeys had proved beyond all doubt that poliomyelitis was an infectious disease. Intraperitoneal injection of spinal cord fragments from children who had died with poliomyelitis caused the same clinical progression and identical anatomical pathology lesions in the animals, after a progression time of 4-33 days. The only known predisposing factor was age, with children aged younger than 8-9 years being particularly vulnerable; it came to be known popularly as "morning paralysis" due to the appearance during those hours of flaccid paralysis after a prodromal febrile period.[22] The acute onset and epidemiological characteristics of poliomyelitis led Constantin von Economo to consider a potential relationship with encephalitis lethargica,[23] which appeared in Vienna in the winter of 1916-1917 and subsequently spread across Europe. This work was translated from German to Spanish by López Ibor, who at the time was a physician at the Provincial Mental Institute of Valencia. Though prodromes with flu-like symptoms were common, a viral origin could not be demonstrated, despite its coinciding with the severe influenza epidemic of 1918. Furthermore, the three clinical forms of acute encephalitis lethargica (somnolent-ophthalmoplegic, hyperkinetic, and hypokinetic) bore no resemblance to poliomyelitis.

The oropharyngeal route of infection, through ingestion of contaminated water or food, is accepted as the most common, with flies typically acting as a vector in

the latter case. The hygiene measures implemented in the 1940s and 1950s led to an increase in subclinical infections and a change in the population most frequently affected, with cases now appearing in older children and even adults. Through neutralisation with specific antiserums, three strains of the poliovirus were identified, known as Brunhilde, Lansing, and Leon (type 1, 2, and 3 strains, respectively), although coxsackievirus A7 may cause identical symptoms. New epidemics appeared in the 20th century, and researchers became interested in bulbar forms, which were associated with high mortality and posed a great challenge in the nascent intensive care units. The selective infection of the anterior horn in poliovirus, and its frequent asymmetry, remain a mystery.

Clinical course and prognosis

The essential traits of infantile poliomyelitis or infantile spinal atrophic paralysis were already known in the early 19th century. The eminent internist Paul Georges Dieulafoy (1839-1911), a disciple of Armand Trousseau, acquired considerable experience at the Hôtel-Dieu hospital in Paris.

> After an initial febrile period coinciding with gastrointestinal symptoms, patients suddenly developed generalised muscle pain and a meningeal syndrome, which gave way to "sudden" paralysis of highly irregular distribution, followed by muscle atrophy.

He described his personal experience with the infrequent bulbar forms: "A patient I observed with Joffroy […] presented bouts of suffocation and continuous and paroxysmal dyspnoea; evidently, the bulbar nuclei were affected."[24]

Pierre Marie and André Léri addressed some of the semiological features of infantile spinal paralysis, with the majority of cases occurring in early childhood, "between 12 and 16 months of life." In the flaccid and amyotrophic form, sphincters are nearly always spared. Patients rarely present paraplegia; rather, one limb or section of a limb is typically involved in isolation. Due to muscle degeneration and laxity of the ligaments, the legs show abnormal mobility in their different segments ("jambe de polichinelle" in French). Certain vasomotor and trophic signs may be observed, such as cold skin, cyanosis, reddening, mottled skin, atrophic skin, or calluses at contact points. Limb growth stops, whether in the legs or the lower part of the trunk; sometimes only the arms are affected. The semiological conclusion is decisive: "The clinical picture is sufficiently characteristic for this variety of paraplegia to be recognisable at first sight" (Figure 6).[25]

For the Belgian neurologist Arthur van Gehuchten, the most useful semiological signs for differentiating poliomyelitis from forms of acute polyneuropathy, such as Guillain-Barré syndrome, were the marked asymmetry of paralysis and the more intense involvement of proximal muscles,[19] with sparing of distal mobility.[22] Two physicians from Berlin, Curschmann and Kramer,[26] gathered data from Duchenne on the prognostic value of electrical stimulation: "Muscles that are not stimulated by faradaic current at three weeks after onset remain permanently paralysed." These authors considered the absence of excitability from galvanic current to be a more reliable sign.

Prognosis, which was so unclear in the early stages, and the great distress of the patients' parents, were intelligently addressed by the Danish neurologist Knud H. Krabbe, head of the nervous diseases department at Copenhagen's Kommunenshospitalet. He wrote a short manual, without figures, presenting the conventional data found in any treatise of the 20th century. However, we should highlight two original aspects: according to Krabbe, some epidemics of poliomyelitis coincided with encephalitis outbreaks; furthermore, the two diseases could manifest with similar symptoms, suggesting a common virus. However, perhaps the most original contribution was his way of informing the distressed parents, to whom he gave the following explanation:

> Firstly, there is no risk of relapse; furthermore, the paralysis existing at the moment will ostensibly improve in the first year or even 18 months. Finally, the improvement is much faster in the early stage of the disease, and becomes increasingly slower[27]

In the first third of the 20th century, Mikhail Kroll, professor of nervous diseases at the University of Minsk in Belarus, defended a surprisingly nihilistic position (the Spanish translation was published in 1931, at the height of the Soviet period; the German edition was published in 1929). Guillain, Barré, and Strohl published their article in 1916, during the First World War:

> Diagnosis of anterior poliomyelitis is based on clinical signs of limited value: if cranial nerve palsy and sensory deficits are observed, the diagnosis would not be poliomyelitis but rather Guillain-Barré syndrome.[28]

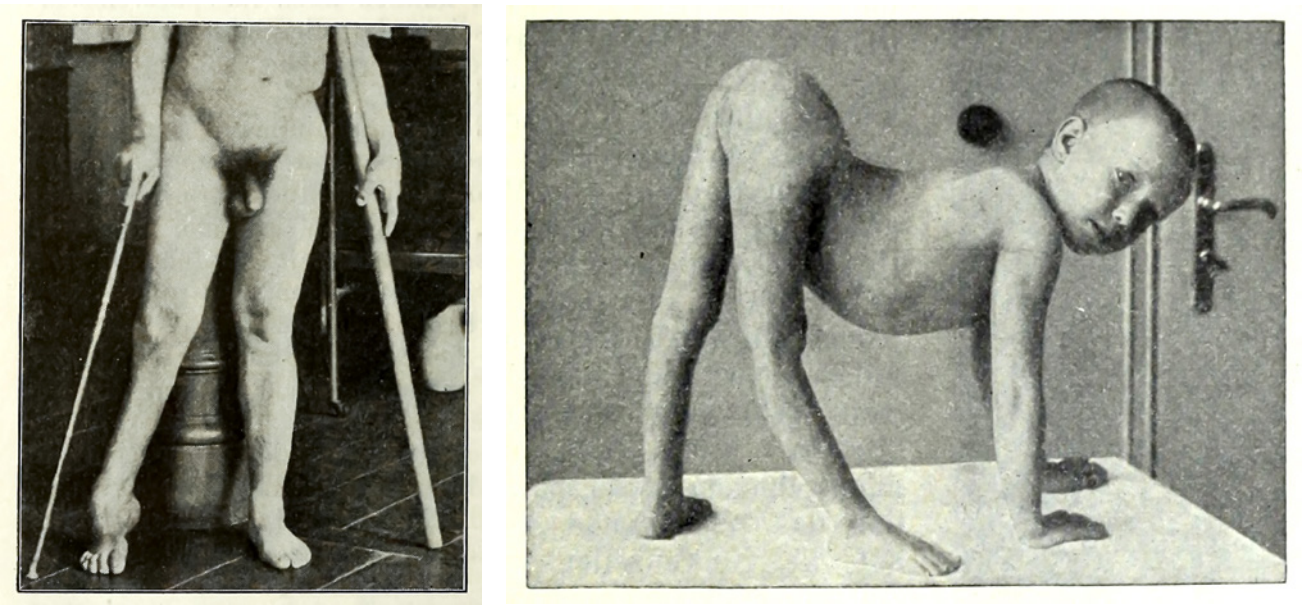

Figure 6. Left: sequelae in adulthood of acute infantile poliomyelitis, with marked shortening of the right lower limb with equinovarus foot (figure 106 in Oppenheim[25]). Right: bilateral paralysis of the legs with hip flexion contracture and lumbar hyperlordosis (figure 209 in Oppenheim[25]). This posture with marked quadrupedal gait is seen today in some beggars in India.

With the dramatic reduction in poliomyelitis in North America and Europe following the development of vaccines, less emphasis was placed on the disease in textbooks. The monumental work by Vicente Gilsanz, chair of medicine in Madrid, and his numerous collaborators, dedicated only 15 pages (of a total of 727) to the disease, citing only 4 references.[29] In 1971, the neuromuscular disorders expert John N. Walton published an excellent summary in English of acute anterior poliomyelitis, covering only two pages.[30]

The first description of CSF analysis in poliomyelitis was published in 1919, in a 230-page book specifically addressing the usefulness of this examination in different diseases.[31] In polio, the colloidal gold curve is flat or shows a slight tilt to the right, and CSF shows moderate mononuclear pleocytosis. After considering various hypotheses, the author concludes that: "The bacteriology of poliomyelitis remains an unresolved question."

The severe sequelae of poliomyelitis

Acute anterior poliomyelitis presents a low mortality rate, but may cause terrible sequelae, resulting in disability and deformity. One of the authors (SGR) recalls childhood companions who wore complicated orthopaedic contraptions that joined in play, grinding and creaking. He also remembers four physicians with significant sequelae of the disease who specialised in various branches of neuroscience, perhaps dreaming that they could one day understand or overcome their situation.

Due to the high prevalence of the disease in the period reviewed in this article, it was possible to observe the sequelae, in the form of muscular atrophy of variable severity, generally affecting one limb, with lesser growth of the affected limb compared to the uninvolved side. Fatal cases, which were not exceptional, showed unilateral haemorrhagic necrosis of the anterior horn in the acute phase, followed by glial scars in cases with sequelae, with a reduction in size of the anterior horn and absence of ganglion cells (Figure 7).

Treatment of acute poliomyelitis and sequelae

In the 20th century, the available treatments were essentially symptomatic: antipyrine for fever, sodium bromide to alleviate pain, and small doses of strychnine in the hopes of facilitating activation of the paralysed muscles. After the acute phase, treatment was based on

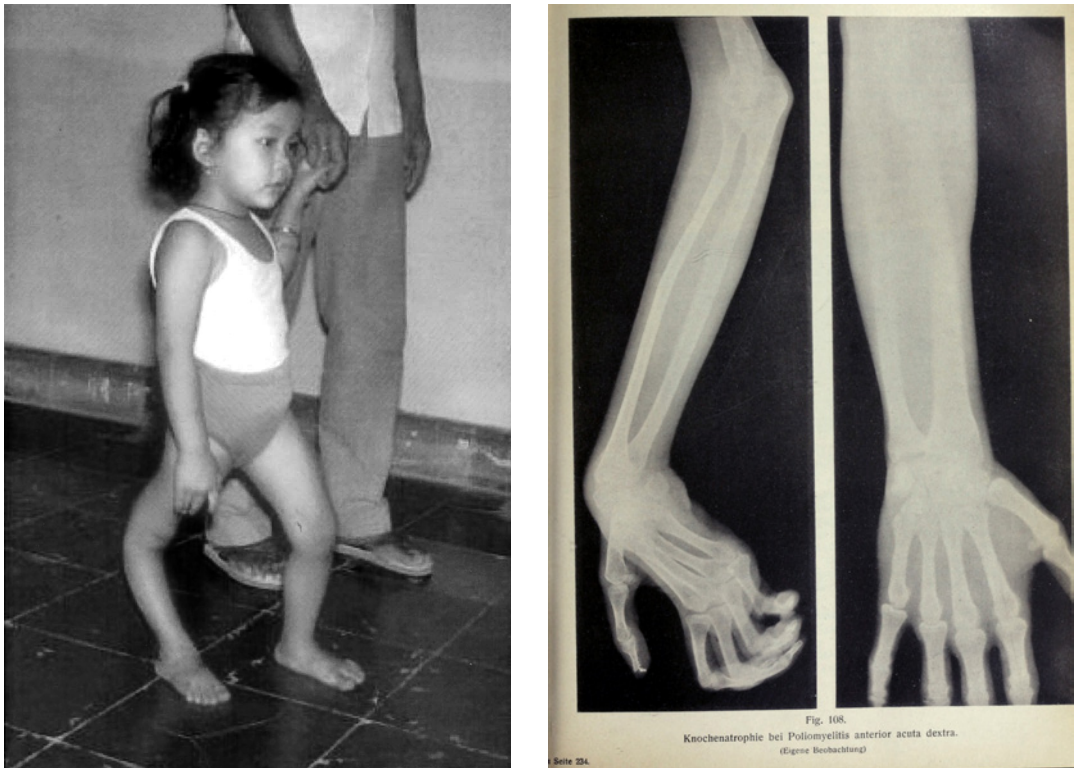

Figure 7. Left: sequelae of acute anterior poliomyelitis in a girl with severe *genu recurvatum* secondary to atrophy of the quadriceps femoris muscle. Image taken from Billings and Collins.[36] Right: the first radiographies demonstrating severe muscle retraction with claw hand deformity (figure 108 in Oppenheim[25]).

sessions of hydrotherapy in warm baths, where it was easier to move the paretic limbs, either in swimming pools or "sea baths." The Valencian painter Joaquín Sorolla Bastida (1863-1923) painted a large group of boys with terrible poliomyelitis sequelae bathing at the sea shore under the attentive gaze of a monk. He erroneously entitled the painting *Hijos del placer* (Children of pleasure), mistakenly believing that their terrible limb deformities were caused by congenital syphilis. After being made aware of the error by his friend Vicente Blasco Ibáñez, he changed the title to *Sad inheritance!*, which was also rather inaccurate (https://historia-arte.com/obras/triste-herencia) (Figure 8). The painting, made on the beach of El Cabanyal in summer 1899, represents the culmination of Sorolla's works on social issues. It suggests that the vices of the parents may lead to severe consequences for their children. The painting received the Grand Prix at the 1900 Paris Exposition. Sorolla himself proposed that the Spanish state purchase the work for 40 000 pesetas.

Around 1903, some authors doubted the potential benefits of galvanotherapy, despite admitting that it produced "chemical changes in the muscle that are essential to growth and nutrition."[11] Given the evidence that the disease spreads through secretions from the nasal mucosa, proposed in Sweden in 1905, isolation of patients and local application of mentholated solutions was a desirable approach.[22]

In the 1940s, the so-called iron lung or artificial respirator began to be used in patients with bulbar forms of poliomyelitis who were unable to breathe independently, who typically also presented tetraplegia. With limited availability, the ethical implications of applying the system "in all patients with respiratory difficulties" were considered with great press impact.[32] The iron lung consisted in a capsule that exerted suction (negative

Figure 8. Joaquín Sorolla. *Sad inheritance!*, 1899, oil on canvas (212 × 288 cm)

pressure) on the chest (Figure 3). A prominent case was that of Paul Alexander, who lived in an iron lung from the age of 6 years until his death at 78 years, as reported by the BBC (https://www.bbc.com/mundo/articles/cyxz9xkeq19o). Today, tracheostomy and positive pressure systems are used. In the 1950s, the Spanish newspaper *ABC* published a photograph of the new iron lung at Hospital del Niño Jesús in Madrid (https://www.abc.es/archivo/fotos/en-el-hospital-nino-jesus-ninos-metidos-en-pulmones-de-acero-son-1202458734.html).

Belarmino Rodríguez-Arias, one of the founding members of the Spanish Society of Neurology, together with other neurologists interested in diseases of childhood, published an interesting article in 1961, reviewing new proposed treatments for patients attended for "neurovirosis" at the Municipal Neurological Institute of Barcelona.[33] In addition to epidemic infantile paralysis, they discussed a diverse range of infectious processes attended at the Institute, including herpes encephalitis, rabies encephalitis, sequelae of encephalitis lethargica, and benign lymphocytic meningitis. The authors were reluctant about the supposed benefits of hypodermic serum therapy, consisting in the administration of serum from convalescent patients or immunised horse. One of the authors (SGR) vividly recalls the prescription of horse serum after a diagnosis of diphtheria at the Instituto Antidiftérico, located on Calle Blasco de Garay (Madrid) at the time. An enormous bulge in the abdomen, resembling a mushroom, was followed by polyarthralgia accompanied by generalised papules and a distressing pruritus. Warm towels applied to the joints and rubbing with talcum powder, administered by parents, uncles, and aunts, were the treatment for anaphylactic shock at the time, along with scraping of the buccopharyngeal membrane, while awaiting spontaneous resolution.

Disappointed by the supposed innovations (non-specific vaccine therapy, insulin therapy, concentrated gamma globulins, and sterile casein [Caseosan]), Rodríguez Arias supported prolonged bed rest ("both mental and physical"), avoiding unsuitable body positioning, physiotherapy for muscle contractures, aspiration of exudates, and alertness to bulbar extension with respiratory paralysis, which should preferably be treated with positive pressure devices.[33]

Post-polio syndrome

With increasing frequency, we are consulted by patients who have suffered for many years with sequelae, which are generally severe. Despite this, and with admirable self-sacrifice and strength of will, they are able to adapt to the limitations of their disease, living happy, productive lives. These patients generally consult around the age of 50 years, when they realise that the affected muscle groups are gradually becoming weaker. Spared muscles remain the same, and patients present no new symptoms, such as sensitivity disorders or cramps. Rheumatologists and physiotherapists confirm the known osteoarticular disorder, but it is challenging to objectively establish the increased muscle weakness so clearly perceived by the patient. An important contribution in Spain was the experience with 310 diagnosed cases of post-polio syndrome at Barcelona's Instituto Guttman. The study underscores the variability of symptoms and the need for multidisciplinary teams.[34]

Post-polio syndrome currently represents a severe social problem in Norway, where thousands of people present sequelae of acute anterior poliomyelitis, a disease first described in the country in 1869, the year of the first outbreak in Norway.[35] The European Neurological Federation published a set of consensus criteria for its diagnosis in the journal *European Neurology* in 2006.

Important neuropathological data and suggestions on the disease mechanism were contributed in a study performed at the Vanderbilt Hospital by Billings and Collins; the latter was a survivor of poliomyelitis, contracted 60 years earlier. In 2005, these authors reported a unique experience: in 1941, five members of a college fraternity had presented poliomyelitis, and similar symptoms appeared in members of another fraternity, which appeared to be a "second epidemic." One of them was almost apnoeic at admission and, despite treatment with artificial respiration, eventually died. Albert Sabin flew from Cincinnati to personally perform an autopsy of the spinal cord. The anterior horn had disappeared on one side. On the contralateral side, some neurons displayed neuronophagia, suggesting an active inflammatory process, whereas others showed abnormal somas and intact nuclei. Although the survivors displayed CSF pleocytosis, it was not possible to transmit the disease to a group of monkeys (Figure 9).[36] The frequency and mechanism of post-polio syndrome are unknown. One hypothesis is that not all neurons in the affected segments of the anterior horn are destroyed. Surviving neurons undergo a process of age-related attrition, with degeneration of axon sprouts that would have partially reinnervated the muscles.

The development of post-polio syndrome raises questions about the concept of poliomyelitis as a static disease, and represents a challenge for healthcare professionals in different fields of medicine. Due to its insidious onset, age of onset, non-specific symptoms, and controversial aetiopathogenesis, this syndrome is difficult to diagnose and confirm. Furthermore, new symptoms and the consequent reduction in functional capacity give rise to new needs and healthcare demand, fed by the anxiety felt by poliomyelitis survivors.

Some studies suggest that submaximal aerobic training alongside low-intensity muscle stretching may be useful,[37] but results are variable and the improvement in muscle strength is sometimes transient. This is a complex syndrome requiring multidisciplinary management by experts in physiotherapy, occupational therapy, neurology (for carpal tunnel syndrome and radiculopathies), rheumatology (for tendinitis and degenerative arthropathies), and pulmonology (for sleep apnoea syndrome).[38]

Towards eradication: mass vaccination of the population

Albert Sabin (1906-1993) and the Russian Mikhail Chumakov (1909-1993) were the protagonists of the long, complex development of oral vaccines against all three polioviruses, based on inactivated viruses cultured in monkey kidney tissue. Albert Bruce Sabin was born to a Jewish family in Bialystok (Poland); born Abraham Saperstein, he changed his name after emigrating in 1921 to the United States, where he graduated from New York University with a degree in medicine. He had his own laboratory from 1939, where he developed experimental models in rhesus monkeys.[39] The development and commercialisation of the models developed by Jonas

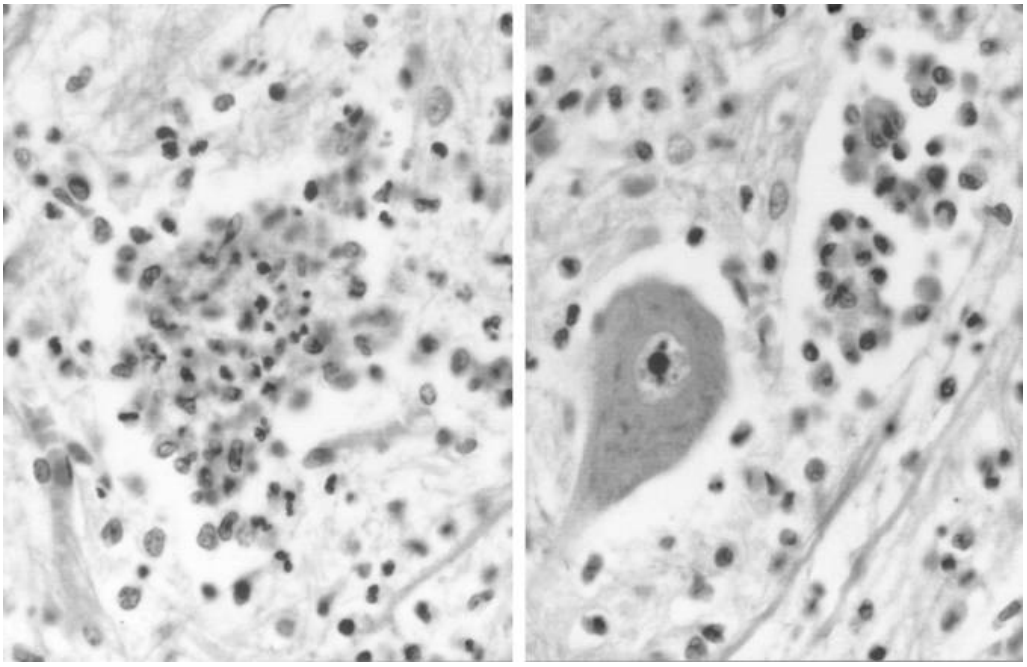

Figure 9. A spinal cord studied by Albert Sabin, taken from a patient with poliomyelitis. This image was published 60 years after the acute episode. Some neurons present intense neuronophagia (left), whereas others (right) appear intact. Image taken from Billings and Collins.[36]

Salk (subcutaneous injection) and Albert Sabin (oral route) were followed by conflict due to economic considerations, which was eventually mitigated by a certain degree of understanding and collaboration between the two great political blocs of the Cold War.[40] In Spain, the first oral dose of polio vaccine was administered in 1959, although mass vaccination campaigns only began in 1963, and some individuals are known to have been missed. One of the authors (SGR) attended a child with poliomyelitis in 1967 at the neurology department of Gran Hospital de la Beneficencia General del Estado (today, Hospital de la Princesa) in Madrid. The child may have been one of the last patients with acute poliomyelitis attended in Spain. In five years, prevalence fell from around 2000 to 62 cases per year. The last autochthonous case in Spain was recorded in 1988, although an imported case was reported in 2021 (online data from the Ministry of Health, 2024).[41]

Final stages in the eradication of poliomyelitis

The project to eradicate poliomyelitis, accepted by all countries in 1988, was a spectacular success, with a 90% reduction in cases thanks to the preventive administration of the triple oral vaccine with attenuated live virus. In the countries where the disease was endemic for various reasons (poorly prepared healthcare staff, remote populations, denial, and military conflict), incidence has decreased dramatically. In low-income countries, the disease has reappeared in a small number of vaccinated individuals during the first 2-3 years of life due to insufficient immunogenesis, secondary to infection by viruses from the vaccine itself or circulating viruses. Nonetheless, prevalence has fallen by over 99%.[42]

Recent media reports (August 2024) alert to the need for an "urgent ceasefire in Gaza to vaccinate children against polio." Some 1.3 million doses are needed, as only 80% of children are vaccinated. To date, only one case has been reported, but poliovirus has been isolated in waste water. UNICEF estimates that sufficient vaccine stocks to meet Gaza's needs will be available by October. The ancient plague on humanity is still with us. The persistent circulation of wild poliovirus in endemic regions, and the detection and circulation of vaccine-derived poliovirus

Table 1. Main contributions to the history of poliomyelitis in the 19th and the first half of the 20th century.

Year	Author	Location	Scientific contribution
1843	Chomel	France	Clinical differentiation from other types of paralysis
1876	Charcot	France	Pathological description of atrophic paralysis
1881	Medin	Sweden	Geographical differences
1894	Möbius	Germany	Characterisation of polio as a "rare disease"
1894	Debove	France	Electrodiagnosis and electrotherapy
1903	Starr	USA	Classification as an epidemic disease
1904	Dieulafoy	France	First precise clinical description
1922	Mohr and Staehlin	USA	Failures of clinical identification
1943	Román Alberca	Spain	Treatise on polio and other viral diseases
1952	Kelly	USA	Use of the iron lung in bulbar forms
1959	Sabin	USA	First oral polio vaccine
1988	Spanish Ministry of Health		Last autochthonous case of the disease
2005	Billings and Collins	USA	Post-polio syndrome

in several regions, including Europe, mean that there is a real risk of importation of poliovirus.

In conclusion, this study explores poliomyelitis using publications from the 19th century and early 20th century, many of which are difficult to access. This is the long story of humankind fighting against a cruel disease, mainly affecting children, and resulting in a dreaded, lifelong disability (Table 1 shows the main milestones in the history of the disease). The discovery of an efficacious vaccine represents the greatest triumph to date. However, the fight is not yet won: whether due to poverty, war, or both, the process has re-emerged in our days in some populations. The appearance of the mysterious post-polio syndrome continues to represent a challenge for researchers.

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Conflicts of interest

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