



Jean-Noël Seignot (1925–2022): Pioneer in the treatment of Gilles de la Tourette syndrome

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ABSTRACT

In 1961, Jean-Noël Seignot (1925–2022) published a clinical observation in the *Annales Médico-Psychologiques* of a patient with a severe form of Gilles de la Tourette syndrome. He included the results of his clinical test with R.1625, a new compound then in an experimental phase, developed by Janssen Laboratories in Belgium—that is, haloperidol. Seignot's article is constantly cited in the bibliographies on the treatment of this syndrome. Here, I present the first-ever complete translation of Seignot's publication, followed by his biography, based on family archives kindly made available to me. This account is accompanied by a brief history of the discovery of haloperidol. Seignot saw that Gilles de la Tourette syndrome was not just a disease but “a whole mode of being.” His foresight in the care and treatment of his seriously ill patient deserves a commemorative biography.

KEYWORDS

Gilles de la Tourette;
haloperidol; Jean-Noël
Seignot; Tourette syndrome;
tics

In their seminal work, *Gilles de la Tourette Syndrome*, Arthur K. Shapiro (1923–1995) and Elaine Shapiro (1925–2014) divided the history of the description of this condition into seven periods (Shapiro et al. 1978). The first period, from 1825 to 1900, corresponds to the earliest descriptions, notably that of the Marquise de Dampierre (Ernestine Émilie Prondre de Guermantes, 1800–1884) by Jean-Marc-Gaspard Itard (1775–1838) (Walusinski 2020), followed by the descriptions in the seminal work Georges Gilles de la Tourette (1857–1904; see de la Tourette, 1885), completed one year later by Georges Guinon, who added obsessive-compulsive symptoms to the clinical picture of this new nosographic entity (Teive et al. 2025; Walusinski 2026).

The second period began around 1900, when Henry Meige (1866–1940) and Eugène Feindel (1862–1930) published their book on tics, which would serve as a reference for nearly half a century (Meige and Feindel 1902). The third period corresponds to the 1921 proposal by Hungarian Sandor Ferenczi (1873–1933) of an etiological interpretation of the syndrome using psychoanalysis (Ferenczi 1921).

The fourth period, in the 1950s, involved a series of clinical case studies published in various European countries and the United States. The fifth period, in the early 1960s, was marked by the first clinical drug tests, notably in the seminal publication by Jean-Noël Seignot (1925–2022) (Figure 1), which we will examine here. Shapiro wrote, “The chemotherapy phase began in 1961 with the successful treatment with haloperidol of a single



Figure 1. Jean-Noël Seignot at the beginning of the 1960s (Archives N. Seignot).

patient by Seignot in France.” This led to the sixth period, when different psychotherapeutic approaches were tested.

The seventh period, which Shapiro regarded as pertaining to psychiatry—his own specialty—spanning from the 1970s to the present day, could best be described as neurological. As our understanding of the functional substrates of the neural networks involved in the symptoms is becoming increasingly precise and detailed, it is more and more clear that Gilles de la Tourette syndrome is a neurodevelopmental disorder involving a combination of genetic, immune, and neurobiological factors.

Why is Jean-Noël Seignot considered a pioneer?

On December 19, 1960, Jean-Noël Seignot was elected a corresponding member of the French Medical-Psychological Society, the oldest learned society in French psychiatry, founded in 1852 and still active today. In his first presentation to the society’s members on February 27, 1961, he outlined his observation regarding a patient he had treated with a new drug. Thanks to this treatment, the patient—who suffered from a severe form of Gilles de la Tourette syndrome—saw a significant reduction in his motor and vocal tics. The summary of Seignot’s treatment was published in January 1961 in *Annales Médico-*

Psychologiques, the oldest journal of psychiatry, founded in 1843. It was titled “*Un cas de maladie des tics de Gilles de la Tourette guéri par le R. 1625*” (“A case of Gilles de la Tourette tic disease cured by R. 1625”). Seignot prescribed haloperidol, also known as R. 1625, which was not yet on the market at the time, but Seignot had samples for clinical tests.

Here is the translation of the paper written by Seignot:

Although this is a single case, I felt that the outcome in this patient was worth reporting, since until now tic disorder has been regarded as resistant to treatment. Here is the summary of the medical history of this 35-year-old man, who presented with a typical tic disorder:

The first tics appeared around the age of ten: they consisted of sudden sideways movements of the head accompanied by an inarticulate cry, known as a barking tic. These tics persisted for several years; however, following a stay in the countryside in 1940, they ceased for a few years. He passed his “certificat d’étude” [primary schooling certificate] and was studying accounting. But around the age of 18, just as he was starting work as an assistant accountant, the tics returned; they quickly became more complex and forced him to stop working. Each time, he would let out one or more shouts, accompanied by frightening grimaces and sudden, violent movements; he would grab his ears and pull at them forcefully, or he would pound his face with all his might using both fists, thereby breaking his teeth and dentures.

For fifteen years, all manner of treatments were tried on him, to no avail; Eunocet, meprobramate, Largactil, and reserpine, amongst others, failed to bring about any improvement, despite being administered in the highest possible doses. In 1957, he underwent a topectomy; his tics disappeared for a few days following the operation, but then returned, and when he was discharged from the hospital where he had been operated on, he was in the same condition as when he was admitted. In 1958, he struck his eyeballs so violently that he went blind as a result of vitreous hemorrhage and retinal detachment.

In November 1960, his condition was particularly alarming; he had an average of one or two tics per minute; each lasted about four seconds, during which he would repeatedly strike himself and thrash about in all directions; his coprolalia was very pronounced, consisting of a mixture of insults and foul language. What’s more, he was very unhappy, as he was constantly plagued by an impulse phobia that drove him to bang his head against the sharp edges of furniture and doors. I decided to put him on R. 1625, starting with a dose of 1 mg a day; by the very next day, his tics had almost completely stopped. Only a few remained, and they only surfaced when someone mentioned his illness to him. In an attempt to achieve a complete remission, the dose was increased, but almost immediately he felt very tired; the treatment, which was interrupted for a few days, was resumed at a dose of 6/10 mg per day; with this extremely reduced maintenance treatment, he now had only two or three tics per day. Furthermore, the intensity of these few residual symptoms was greatly reduced; in particular, the coprolalia had completely disappeared.

The effect of R. 1625 was thus remarkable because the daily number of tics, previously near one thousand, was reduced to a few units. The maintenance dose was minimal and thus tolerated without any difficulty. Finally, his condition has remained stable, as there has been no recurrence of tics over the last three months, despite two difficult experiences for him: the news that his son has epilepsy, and the news that he himself is now permanently blind. The only thing we cannot verify, unfortunately, is how he would cope with returning to work.

This single observation of a rare condition does not allow us to draw general conclusions; however, there is reason to hope that R. 1625 will prove useful, not only in this severe form of tics, but also in all other, more common, and far more widespread forms, in adults or even in children.

In 1957, the patient had undergone a topectomy, a neurosurgical procedure involving “bilateral ablation of the rostral-medial cortex of each frontal lobe, considered part of Brodmann areas 9 and 10,” a technique developed by the American James Lawrence Pool (1906–2004; see Pool 1949) and tested in France at the Hôpital Lariboisière in Paris by Jacques Le Beau (1908–1998; see Le Beau 1951). This form of psychosurgery was proposed as an alternative to lobotomy. It did not improve the condition, but Seignot did not report any adverse effects.

In the year following the publication of Seignot’s work, Giovanni Caprini et Vincenzo Melotti (at Verona’s hospital) published two clinical cases in Italy in which haloperidol was used, achieving a similar symptomatic improvement to that reported by Seignot (Caprini and Melotti 1962). In 1963, two psychiatrists from the University of Iowa published two further observations of patients with Gilles de la Tourette syndrome who had shown just as much improvement as Seignot’s patient (Challas and Brauer 1963). Haloperidol became the first-line treatment for this syndrome. Seignot had opened the way forward.

Several articles followed, showing a response in nearly 80% of cases. Gradually, the side effects and adverse reactions would be documented. In 1973, Faruk Abuzzahab and Floyd Anderson of the University of Minnesota published a study of 430 patients treated with haloperidol, detailing for each symptom of the syndrome the benefits achieved through treatment with haloperidol: “Haloperidol is superior to any other treatment and must be considered the treatment of choice in Gilles de la Tourette syndrome” (Abuzzahab and Anderson 1973).

Who was Jean-Noël Seignot?

Seignot was born in Paris on September 13, 1925, and studied at the Paris Medical School. In 1949, he passed the examination to work in psychiatric hospitals as a resident. Since Philippe the time of Pinel (1745–1826) and Jeanne Étienne Dominique Esquirol (1772–1840), the founders of psychiatry in France, mental illness was the first medical specialty to exist in its own right, with a specific training program following the completion of medical studies. This is why there were residents in psychiatric hospitals, distinct from the residents in the Paris hospitals.

During his residency, Seignot was successively taught by neurosurgeon Marcel David (1893–1986) at Saint-Anne Hospital, and by psychiatrists Paul Guiraud (1882–1974), Xavier Abély (1890–1965), Pierre Menuau (1893–1985), Baptiste Chanes (1893–1971), and Georges Daumezon (1912–1978). He finished his residency at the Enfants-Malades hospital in the department of Georges Heuyer (1884–1977), who introduced him to child psychiatry. Heuyer also presided over his thesis, which Seignot defended on March 20, 1953: *Le sommeil, étude physiologique et psychologique* (Sleep, a Physiological and Psychological Study; see Seignot 1953). He subsequently turned down the post of senior resident offered to him by Heuyer, preferring to move to Calais in June 1953 to practice as a private neuropsychiatrist, the first specialist in neuropsychiatry to serve a vast area in northern France. He worked there for some 40 years before passing away on May 21, 2022, in Nice, at the age of 96.

First publication

While working as a resident under Heuyer, Seignot published a paper with him and Serge Lebovici (1915–2000) in *La Revue neurologique* titled “*Paraplégie spasmodique familiale avec affaiblissement intellectuel chez deux sœurs, maladie de Schilder?*” (“Familial Spastic Paraplegia with Intellectual Disability in Two Sisters: Schilder’s Disease?” Heuyer, Leibovici, and Seignot 1951). Two sisters, having had a normal early childhood, gradually developed paraplegia at the age of three, followed by quadriplegia with pyramidal spasticity; this was soon complicated by significant intellectual decline, accompanied by athetoid movements of the upper limbs and generalized epilepsy. Although the mother took quinine during her pregnancies to treat malaria, the authors ruled out the possibility that the condition was caused by fetal toxicity, given the timeline of the disease’s progression. The demential state ruled out the possibility of Strumpell-Lorrain disease (Walusinski 2020). That is why they considered Schilder-Foix disease.

Charles Foix (1882–1927) and Pierre Marie (1853–1940) described their first case in 1913, including the anatomopathological examination (Foix 1923, 168–175). In Leipzig, Paul Schilder (1886–1940) described this pathology in 1912, then again in 1913 (Schilder 1912, 1913). In 1925, the Polish physician Edward Flatau (1868–1932) revisited and detailed the description and proposed replacing the term “Encephalitis periaxialis diffusa,” coined by Schilder, with “*Encephaloleukopathia scleroticans progressiva*” (Flatau 1925).

In 1927, Charles Foix and Julien Marie (1899–1987) sought to define this familial condition more precisely within pediatric nosology by distinguishing it from other central and peripheral atrophic disorders, viewing it as an explanation for many cases of childhood paraplegia, which were considered idiopathic at the time (Foix and Marie 1927). This rare, progressive, and invariably fatal disease is characterized by adrenal atrophy and diffuse cerebral demyelination. The main clinical features are progressive dementia accompanied by spastic quadriplegia, cortical blindness, deafness, and a condition resembling a pseudobulbar palsy. The disease begins in childhood and leads to death within a few months or years. Schilder-Foix disease belongs to a heterogeneous group of disorders, and it is necessary to rule out the following diagnoses: acute disseminated encephalomyelitis, subacute sclerosing leukoencephalitis, rubella panencephalitis, metachromatic leukodystrophy, Churg-Strauss syndrome, Krabbe disease, and adrenoleukodystrophy. Its cause remains unknown, but a mitochondrial pathology is currently under discussion (Synofzik et al. 2023). The eponym seems to be no longer in use.

Thesis

Seignot’s thesis, prepared in Heuyer’s child psychiatry department, provides insight into hospital practice during the postwar period, focusing on the study of children’s sleep—including that of premature infants—using electroencephalography, a technique still relatively new and innovative. Seignot referred to American research, notably that of Frederick Bremer (1892–1982) and Nathaniel Kleitman (1895–1999) from before the war, and to the report presented by Charles Kayser (1899–1981) of Strasbourg at the Congress of Physiologists in 1949. His primary source of inspiration, which he wished to build on, remained his reading of the scientific thesis of Henri Piéron (1881–1964), defended in 1912 (see Piéron 1912). Seignot’s thesis, which remains of historical interest, highlights the

hypotheses put forward at the time regarding the physiology of sleep. Neurotransmitters were not yet widely known; the concept of REM sleep was unknown. Seignot acknowledged the still-recent discovery of the anatomical localization of the centers of wakefulness and sleep, identified by Constantin von Economo (1876–1931; see von Economo C 1917) during the epidemic of encephalitis lethargica (1917–1939; see Foley 2018).

Seignot's thesis appears, in part, to be a preliminary draft of a handbook for general practitioners, intended to provide guidance on the management of sleep disorders in children—one of the favorite subjects of his teacher, Heuyer. He devoted a lengthy chapter to conditioned reflexes, because “we must attribute the induction of sleep in animals—and, by extension, in humans—to training, education and, more generally, conditioning.” Seignot finished with a long chapter on dreams: “Everyone is always interested in their dreams.” He dwelled on the difficulty of distinguishing “the transition from dream to reality,” curiously citing sleepwalking as an example. He drew on Sigmund Freud (1856–1939), according to whom one should always be able to trace the origin of each element of a dream back to the subject's past, using the method of free association. With regard to clinical treatment, and discussing only implications, he accepted the formula devised by Freud whereby a dream is the disguised fulfillment of a repressed desire. Seignot's work concludes with an extensive bibliography, reflecting the care he took in compiling his substantial thesis.

Discovery of haloperidol

Paul Janssen (1926–2003), a Belgian chemist, had initially synthesized R. 875, or dextromoramide—a morphine-derived analgesic (Palfium)—as one more than 1,300 synthetic molecules; he and his team searched for new, even more potent analgesics using molecular screening techniques. Instead of continuing along the path he had taken with dextromoramide, Janssen decided to synthesize phenylpiperidine derivatives. He synthesized the first butyrophenone, R. 1187, in 1957, and went on to develop a whole family of related compounds. The 45th molecule, R. 1625, was synthesized on February 11, 1958. It bore no chemical resemblance to pethidine. Its analgesic effect was weaker than that of morphine but, curiously enough, whereas morphine derivatives stimulate mice, butyrophenones—and in particular R. 1625—induce a cataleptic state and sedation reminiscent of the effects of chlorpromazine (Granger and Albu 2005).

Following a month of animal trials, human trials began on March 17, 1958, at the Brugmann Hospital in Brussels. The first publication by the team at the University of Liège's psychiatric clinic appeared in January 1959: *Étude et expérimentation cliniques du R. 1625 ou halopéridol, nouveau neuroleptique et neurodysléptique* (Study and Clinical Trials of R. 1625 or Haloperidol, a New Neuroleptic and Neurodysleptic; Divry et al. 1959). Haloperidol was administered intravenously to 18 patients suffering from psychomotor agitation. This compound calmed their agitation even at minimal doses, whereas other sedatives had failed to achieve this result. Increasing the doses “cause[d] pronounced parkinsonism,” which was in fact acute dyskinesia; the authors termed it “neurodyslepsia” (Divry, Bobon, and Collard 1960).

In 1959, numerous authors published the results of their experiments and specified their indications and side effects. At the 1959 international symposium in Beerse, the first French

series was presented by Pierre Pichot (1918–2020) on behalf of Jean Delay (1907–1987) and by Thérèse Lempérière (1925–2013), then a senior resident (Delay et al. 1960). This series consisted of patients with schizophrenia and those suffering from chronic delusions. These authors considered that haloperidol “had an effect qualitatively similar to that of neuroleptics, but quantitatively more potent.”

It should be noted that when Seignot prescribed haloperidol to his patient suffering from Gilles de la Tourette syndrome, he was undoubtedly aware of the experiments mentioned, as well as others, but at that time no one had yet prescribed this drug to alleviate abnormal movements. Although we do not know what prompted him to attempt this experiment, moving from thought disorders to movement disorders, it is perhaps due to the patient’s obsessive-compulsive disorder or certain repetitive delusions such as mental tics. It is worth noting that Jean-Martin Charcot (1825–1893) referred to “mental tics” when describing what we now call obsessive-compulsive disorder. Seignot’s empathy for the patient’s severe distress—given that conventional treatments had reached an impasse—likely also played a decisive role. This was not, however, his first innovation.

Seignot: Pioneer of the psychiatric use of Largactil

Seignot’s family kindly passed along an unpublished text he wrote in 2021. In it, Seignot explains how, while working as a resident under Paul Guiraud in 1952, he came across an article by Henri Laborit (1914–1995), Pierre Huguenard (1924–2006), and Raymond Alluaume (1923–1974) published in *La Presse médicale* (Laborit, Huguenard, and Alluaume 1952), discussing the clinical trials of 4560 R.P.—that is, Rhône-Poulenc’s future Largactil (Barral 1997). Taking Laborit’s conclusion at face value—that this sedative held immense potential for use in psychiatry—Seignot requested and obtained samples of the new product in 1952. Faced with episodes of acute delirium causing potentially fatal severe disturbances in the autonomic nervous system (hyperthermia and hyperazotaemia), Seignot immediately realized that this drug might inhibit them. He thus treated two patients, who quickly became calm and survived. This was a dramatic turn of events for which Seignot, who feared he might be held responsible for any fatal outcomes,

Guiraud encouraged him to publish his findings, but the latter’s retirement and other unspecified events prevented him from publishing the seminal description, “much to my disappointment!” noted Seignot. Guiraud chose instead to give priority to Jean Delay (1907–1987), Pierre Deniker (1917–1998), and Jean-Marie Harl (1924–1965)—who published their own findings a few weeks later in *Annales Médico-Psychologiques*—believing that their international reputation would ensure the rapid dissemination of this innovative new treatment (Delay, Deniker, and Harl 1952). Seignot nevertheless believed that a few patients might have been saved had his own paper been published sooner. This previously unpublished account established Seignot as one of the pioneers of the therapeutic use of Largactil in psychiatry, alongside Jean Sigwald (1903–1975) and Daniel Bouttier (1917–2007; see Sigwald and Bouttier 1953).

Conclusion

Haloperidol was the first antipsychotic proven to be effective in the treatment of Gilles de la Tourette syndrome and is, in Europe, still one of the agents approved for this pathology. It is

a potent dopamine receptor antagonist, acting specifically against D2 receptors. It also blocks muscarinic acetylcholine receptors as well as adrenergic receptors and has a well-documented effectiveness in tic reduction. However, due to considerable adverse effects, the use of haloperidol as treatment of Gilles de la Tourette syndrome has decreased in the last three decades, from being a first-line agent to being used only in carefully selected, severely affected, and otherwise treatment-resistant patients (Roessner et al. 2022). Aripiprazole has become the most commonly used agent for the pharmacological treatment of Gilles de la Tourette syndrome across the Western world. In any case, neurologists have made enormous advances in the “hard science” of Gilles de la Tourette syndrome—but Seignot saw that it was not just a disease but “a whole mode of being.” His perspicacity in taking charge of and treating his seriously ill patients deserves to be remembered today.

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