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## History of Neurology

# Louis Tanquerel des Planches (1810-1862) and the history of discovering lead poisoning in the nervous system

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### ABSTRACT

Louis Tanquerel des Planches (1810-1862) only left us with one significant medical work, his *Traité des maladies de plomb ou saturnines* (treatise on lead or saturnine diseases), published in 1839. The work served as a reference for diagnosing and treating lead poisoning throughout the second half of the nineteenth century. The word “encephalopathy” that he coined at that time referred to toxic damage to the central nervous system. Whereas for two millennia and for most physicians, lead poisoning was considered lead colic, i.e. paroxysms of abdominal pain, Tanquerel collected seventy-two observations of damage to the central nervous system in workers exposed to lead in Parisian workshops (which no longer exist). He then inventoried and described forms of paralysis, delirium, coma, and convulsions related to lead poisoning. Having no qualms about stepping away from La Charité Hospital where he had treated patients with lead poisoning, he inspected their workplaces and unambiguously presented the deplorable conditions that caused so many patients to die. His “preservative” advice was an initial attempt at medical-social prophylaxis with the goal of helping the working class exposed in workshops without any respect for human life. With support from chemists and pharmacists, Tanquerel showed the presence of lead in brain tissue and thus demonstrated its neurological toxicity as early as 1839. This article is also an opportunity to note the contributions on this topic of some other physicians: François-de-Paule Combalusier (1713-1762), François Victor Mérat de Vaumartoise (1780-1851), Jean-Louis Brachet (1789-1858), Auguste Mirande (1802-1865), Vincent Nivet (1809-1893), Augustin Grisolle (1811-1869), and Ferdinand de Bernard de Montessus (1817-1899).

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The fire that ravaged the frame and spire of the Notre-Dame Cathedral in Paris on 15 April 2019 dispersed around 450 tonnes of lead on the ground and in the air. Shortly thereafter, various studies documented concentrations of lead in an initial radius of 1 km and up to 50 km, resulting in concentrations two to six times higher than previously [1]. The local

health authority, the Agence Régionale de la Santé d'Île-de-France, recommended preventive measures against lead poisoning in children. Blood lead level testing (BLL) was offered to families living close to the cathedral, specifically for children aged 0 to 6. According to a recent report, the environmental investigations revealed, for 13 children out

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of 1222 with BLL over 50  $\mu\text{g/L}$ , one or more sources of lead independent of the Notre-Dame fire. These investigations could not exclude exposure in connection with the cathedral fire. Sources of lead identified were sources usually found in old Parisian housing with a high frequency of balconies or terraces with laminated lead coatings (10/13 cases). The various sources were detected at home for 12 children, at school for 2 children, and in the street for one child. Thus, the fire of the Notre-Dame de Paris cathedral was not related in time and space with a BLL increase in children in the area resulting from the fallout of high-lead dust. However, the identification of 13 cases of lead poisoning should prompt health professionals to screen for lead poisoning in all Paris neighbourhoods and inform families of the poisoning risk [2]. According to Hanna-Attisha et al., “The science is now unequivocal. There is no acceptable level of lead. There is no safe threshold. Lead is pernicious at the lowest increments of exposure. The World Health Organization and the Centers for Disease Control and Prevention have declared that no level of lead in the blood is safe” [3]. At any rate, saturnism or lead poisoning thus re-emerged as a source of worry for the population from Paris and elsewhere who had forgotten the public health crisis around lead in Paris during the nineteenth century. While physicians had been familiar with lead colic since time immemorial, the neurological toxicity of this metal was only brought to light by a young physician, Louis Tanquerel des Planches (1810-1862), in his 1834 thesis which later became a thick treatise of more than 1100 pages published in 1839. After a biographical overview, I will present the history of knowledge on lead toxicity and in particular its damage to the nervous system, a finding for which Augustin Grisolle (1811-1869) laid the groundwork and Tanquerel went on to perfect.

## 1. Biographical overview

Louis Charles Marie Tanquerel des Planches was born on 17 August 1810 in Ambrières les Vallées, a city in Mayenne in northwestern France. He was the son of Louis Jean Tanquerel (1770-1822) and Marie-Louise Duval du Breil (1785-?). After completing two secondary school diplomas, Tanquerel was guided by a relative, René Nicolas Dufriche Desgenettes (1762-1837), who was chief physician in the imperial security force and later a public health professor in the medical school in Paris. Tanquerel started his medical studies and passed the competitive examination for entering the Ecole pratique (laboratories, classrooms, and practicums at the Paris medical school). He would never become a resident in the Paris hospital system. From 1831 on, he examined patients in the old wards of La Charité Hospital working alongside Pierre Rayer (1793-1867), Jean-Baptiste Bouillaud (1796-1881), Pierre-Joseph Rullier (17?-1837), and Théodorice Lerminier (1770-1836) [4]. He defended his thesis in 1834: *Essai sur la paralysie de plomb ou saturnine* (essay on lead or saturnine palsy) [5]. He failed both times he attempted the *agrégation* exam (to become a professor) in 1838 and 1844. Nicolas Adelon (1782-1862) presided over the jury in 1838 and evaluated the following: *Déterminer les caractères à l'aide desquels on peut distinguer pendant la vie et après la mort les congestions sanguines et les inflammations*

(determining the characteristics by which blood congestion and inflammation can be distinguished while the patient is still alive and after their death) [6]. In 1844, Tanquerel defended his thesis, *De la valeur de l'hydropisie dans les maladies; des indications thérapeutiques auxquelles elle donne lieu* (role of dropsy in diseases and its therapeutic indications) [7] before a jury presided over by Pierre-Eloi Fouquier de Maissemy (1776-1850). Thanks to his novel research on lead poisoning, he was raised to the rank of Knight of the French Legion of Honour on 25 April 1845.

However, disappointed by his failure to obtain a university position, Tanquerel left Paris in 1848 and gave up his practice of medicine. He retired to the castle he had built in northwestern France (Sarthe) near his native Mayenne, known as Château Rochefeuille (Fig. 1). “As soon as he moved to the countryside, Tanquerel took a passionate interest in agriculture. He published numerous agricultural reports and every year was selected by his region as member of the jury for the Poissy competition. His reports and research were of great use and led to much merited praise. He was able to show that agriculture was the most routine of arts because it lacked science” [8]. His castle has remained linked to agriculture. In 1962 it became a school for agricultural and environmental occupations.

On 15 April 1837, Tanquerel married Marie Euphrasie Tanquerel des Uzachères (1798-1872). They had a daughter in 1839. Tanquerel died suddenly at age 53 in his castle on 27 May 1862.

## 2. Lead colic

The toxic properties of lead were described at the beginning of the common era by the Greek physician Pedanius Dioscoride (Πεδάνιος Διοσκορίδης 40-90 AD): “Est autem letalis ejus potus (this drink is lethal)” [9]. François Citoys (Franciscus Citesius 1572-1652), physician to Cardinal Richelieu, described a colic epidemic in the west-central region of Poitou in 1616, often referred to the “Poitou colic” [10]. The synonyms coined



Fig. 1 – Château de Rochefeuille built by L. Tanquerel des Planches in Mayenne (Wikipedia).

over the years include painters colic, plumbers colic, colica pictorum, Devonshire colic, Damnoniens colic of de John Huxham (1694-1768) [11], endemische Kolik, Madrid colic, Surinam dry colic, bilious colic, and dry bellyache in North America. Whatever the name, the condition involves paroxysms of abdominal pain.

Lead colic was often confused with colic related to scurvy. Théodore Tronchin (1709-1781) is considered the first to have formally identified, in 1757, the toxic aetiology by the inhalation of lead vapours or lead ingestion, notably in the form of beverages kept in lead casks [12]. In 1762 Pierre-Joseph Bonté (1730-1806) from Normandy distinguished mineral colic attributed to "saturnine salts" from *colique végétal* (vegetal colic) due to beverages "rich in tartaric acid" [13]. This inaccurate distinction was a source of confusion and was abandoned by the end of the eighteenth century when it was recognised that these "lithargyric" beverages in fact contained lead oxide used empirically to reduce bitterness in wine, ciders, and rum from the West Indies and French Guiana.

Following in the footsteps of François-de-Paule Combalusier (1713-1762) by publishing in 1761 *Observations et réflexions sur la colique de Poitou ou des peintres* (observations and commentary on the Poitou and painter forms of colic) [14], François Victor Mérat de Vaumartoise (1780-1851) collected more than one hundred observations in a treatise 300 pages in length with over one hundred observations, published in 1812: *Traité de la colique métallique vulgairement appelée colique des peintres, des plombiers, de Poitou avec une description de la colique végétale et un Mémoire sur le tremblement des doreurs sur métaux* (treatise on metallic colic commonly referred to as

painters colic, plumbers colic, or lead colic with a description of vegetal colic and a dissertation on shaking in metal gilders) [15]. This work was subsequent to his thesis defended on 11 July 1803 entitled *Dissertation sur la colique métallique, vulgairement appelée colique des peintres, des plombiers, du plomb* (dissertation on metallic colic, more commonly known as painters' colic, plumbers' colic, or lead colic) [16]. The works of Combalusier and Mérat (Fig. 2) indicate that the clinical picture of lead colic and its cause were perfectly recognised at that time. However, disorders affecting the nervous system were only marginally mentioned. Mérat, who authored the most thorough work in the early nineteenth century, noted that shaking, convulsions, paralysis, and delirium were rare and thus accorded them only a few lines. For him, these disorders were not very significant in relation to extreme abdominal pain and rapid deterioration of general health sometimes leading to death.

In 1850, Jean-Louis Brachet (1789-1858) [17] published his *Traité pratique de la colique de plomb* (practical treatise on lead colic). He began with a detailed historical overview starting with antiquity which provided information on the history of knowledge in the area of lead colic [18].

### 2.1. Clinical picture of lead poisoning in the middle of the nineteenth century

White lead is the basic lead carbonate occurring naturally as a mineral, also known as hydrate of cerussite. It was formerly used as an ingredient for lead paint because of its opacity [19]. In his thesis defended on 18 June 1845 (Fig. 3), Ferdinand de

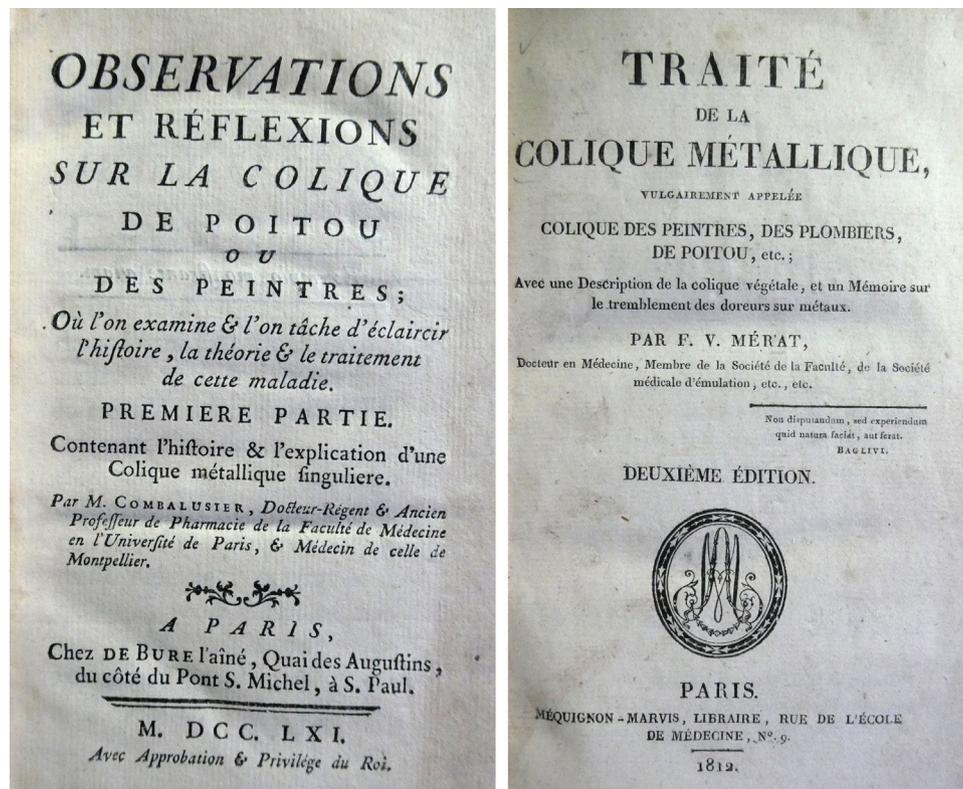


Fig. 2 – Covers of the books by Combalusier and Mérat (OW collection).

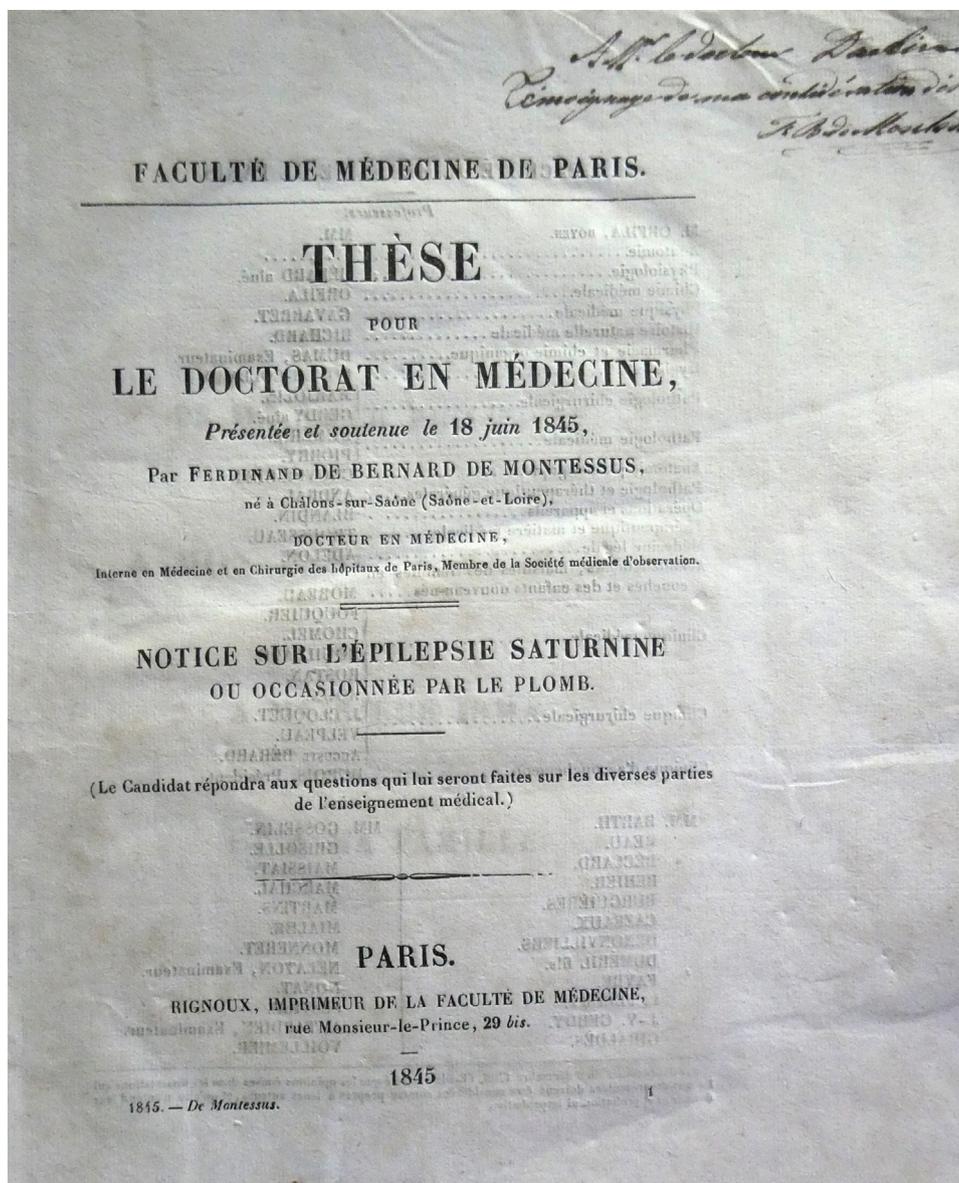


Fig. 3 – Covert of the thesis by Ferdinand de Montessus (OW collection).

Bernard de Montessus (1817-1899) summarised the clinical picture for lead poisoning, laying out the fate of workers in white lead preparation workshops: “Immediately upon arriving in the workshops, their portliness drained away, they were pale, their faces took on a sullen or pallid hue and a waxy complexion, and often their eyes, and in some cases the entire surface of their bodies, became yellow. Their teeth, gums, and less often the inner side of their lips, cheeks and tongue took on an ashen, brown, or violet colour. Their breath was fetid, and their salivation increased or decreased. There was also anorexia, constipation, diarrhoea, and colic that in some cases were atrocious, with intolerable pain in the continuity and contiguity of the limbs. Such are the symptoms that these unfortunate workers could rarely avoid. Happy are those not overtaken by this furious maniacal delirium that arises from idiocy, or this coma so close to death, or

these general or epileptoid convulsions, which are even more fatal!” [20].

Montessus did not use the eponym Burton’s line. However, Henry Burton (1799-1849), a physician at Saint-Thomas’s hospital in London, had in 1840 described this pathognomic gingival line indicating lead poisoning [21]. Burton’s line is a blue-purplish line on the gums seen in lead poisoning. It is caused by a reaction between circulating lead with sulphur ions released by oral bacterial activity, which deposits lead sulphide at the junction of the teeth and gums [22].

## 2.2. Lead poisoning in Paris in the middle of the nineteenth century

The ravages of saturnism at the beginning of the nineteenth century were considerable. Its victims filled hospital wards,

notably in the department of François Martin-Solon (1794-1856) at Beaujon Hospital, where Grisolle collected the materials for his thesis on lead colic. He defended it one year after Tanquerel's thesis [23]. Joseph Récamier (1744-1852) also treated these patients at Hôtel-Dieu Hospital. Tanquerel worked on the Saint-Michel and Sainte Marthe wards at La Charité Hospital, directed by Rayer, but also in the departments of Gabriel Andral (1797-1876) and Stanislas Sandras (1802-1856). All of these hospitals cared for patients from the suburbs of Paris, notably Le Pecq, Clichy, and Courbevoie, cities in which, since the end of the eighteenth century, workshops had been set up for the white lead industry [24]. There were no health or prophylactic measures for workers at that time. In 1850, Brachet noted that "in Paris alone, five to six hundred victims [were] treated in hospitals" [18] each year, resulting in twenty to thirty deaths among them.

### 2.3. Thesis of Tanquerel des Planches

Des Genettes presided over the jury for Tanquerel's thesis; the other members were Adelon, Bouillaud, and Auguste-François Chomel (1788-1858). The candidate defended *Un essai sur la paralysie de plomb ou saturnine* (essay on lead or saturnine palsy) [5] on 6 February 1834, after having collected twelve observations in Rayer's department at La Charité Hospital. From the beginning, prior to his thesis, Tanquerel wrote that "the paralysis, whose single cause was the action of a saturnine agent, has been barely studied... Why has this paralysis always been studied so carelessly? This negligence is probably based on the fact that we are used to considering saturnine colic, not as the main disease produced by lead, but as the single condition due to this poison, and paralysis as a secondary effect or symptom of colic". He only found mention of paralysis during colic in the writings of three authors: Jean Boucher-Beauval de La Rochelle in 1673 [25], Anton de Haen (1704-1776), a Dutch physician writing in 1759 [26], and Auguste Mirande (1802-1865) in 1825 who was the first in this thesis to describe saturnine amaurosis [27].

Tanquerel noted that this pathology was more frequent than physicians of the day believed it to be. He defined lead palsy as follows: "Abolition or significant weakening of voluntary movement and sensitivity, or one of these faculties, as caused by lead and its various compounds". With his thesis, he set out to accurately describe the symptomatology, progression, prognosis, and pathological anatomy of this disease. Undoubtedly to foreground his observations, he published some of them in the *Gazette des Hôpitaux* a few days after his defence [28].

Tanquerel started by presenting the pathways by which lead was absorbed; that is, digestive and respiratory routes and cutaneous routes as in the application of "Goulard's water" (an aqueous preparation of lead acetate used at the time on tuberculous tumours). He asserted that only lead explained the paralysis and not mercury or other metals. He noted that rats living in white lead workshops developed paralysis in their hind quarters. He did not fail to perform experimental pathophysiological research, hoping to reproduce convulsions and paralysis in rabbits by introducing grains of lead acetate under the skin. He failed to produce any pertinent results with this chemical form and this route.

In addition to those who worked with white lead and in minium workshops, painters were sometimes affected, and in rare cases women, who did not work in these occupations. Lead palsy usually occurs later than colic, after a few months' exposure to lead. But Tanquerel highlighted the fact that this paralysis could occur in the absence of lead colic; thus it was not in most cases linked to lead poisoning.

Tanquerel focused on providing the most accurate clinical picture possible from the history of lead palsy. The prodromes included a sensation of swelling in the limbs, most often in the upper limbs, with weakness, pins and needles, and tingling. Insomnia and nightmares set in together with a state of anxiety before the occurrence of shaking and severe cramps when the patient actively bent their legs. Over the course of the condition, paralysis was always predominant in the upper limbs and first affected the finger extensors, the supinators, and the abductors and adductors of the thumb. It was more or less complete and flaccid. Some muscles could maintain contractility whereas other neighbouring muscles no longer responded. Amyotrophy set in rapidly. Sensitivity was maintained for quite some time but pain could appear. Then the muscles of the larynx became paralysed, making it impossible to speak. Finally, deficient respiration led to the patient's death.

Those poisoned could be identified by Burton's line, grey-slate in colour, and also by the blackening of the teeth. Subcutaneous fat disappeared and the victims' faces were marked by characteristic emaciation. The skin seemed glued to the facial bones. Memory weakened and dementia set in. Tanquerel used the term "marasma" to describe the terminal state. The length of progression varied; the impairment was gradual, progressive, and irregular. Remissions could give the illusion of a possible recovery. Only the absence of contact with lead and, for Tanquerel, the treatment developed at La Charité Hospital could save the patients. The duration of exposure to lead was key to the prognosis. He went on to review other forms of neurological damage such as anaesthesia, amaurosis, and deafness. Tanquerel performed two autopsies of deceased saturnine patients who had become paralysed. He did not observe any macroscopically visible lesions of any consequence in the central and peripheral nervous systems.

The treatment used at La Charité Hospital was developed in 1818 by Pierre-Joseph Pelletier (1788-1842) and Joseph-Bienaimé Caventou (1795-1877). It included galvanic currents applied to the paralysed parts or electropuncture and "sulphuric baths and several sorts of stimulants", such as strychnine "extracted from Saint Ignatius beans and nuxvomica". Finally, he mentioned "prophylactic treatment", suggesting that the authorities implement regulations concerning the working time spent in the white lead workshops, e.g. one month of exposure out of three. The workshops were to be properly ventilated with dry air. The workers were to wear masks; lead must not come into contact with their skin. The workers were to wash their hands and faces frequently, and they were required to do so before leaving the workshop. Eating and napping in the workshops were to be prohibited.

In 1836, in addition to his thesis, Tanquerel published an observation of a former painter treated for heart failure using

lead acetate. This treatment caused the recurrence of lead colic that the patient had suffered from during his career. Paralysis developed in the upper limbs followed by the lower limbs and accompanied by violent pain. Thus the iatrogenic use of lead led to his death. The macroscopic autopsy did not reveal any anomalies [29].

In 1855, in his famous *Traité de l'électrisation localisée* (treatise of localised electrification), Guillaume Duchenne de Boulogne (1806-1875) added to Tanquerel's observations with the results of the electrification of paralysed patients: "I studied the state of muscular properties using localised faradisation in eighteen cases of lead palsy... Paralysis due to lead is generally limited to the upper limbs". And later on: "A very bizarre phenomenon occurs such that the lesion of electromuscular contractility always seems to affect certain muscles whereas the entire limb is paralysed. If the progression of this dynamic lesion is studied, as well as the order in which the muscles are successively damaged, it can be observed that the common extensor of the fingers then the extensors of the index and the pinky, and after that the long thumb extensor, are the first to be impaired in their electrical contractility". Closing the debate around the origin of so-called vegetal colic, he wrote: "The perfect similarity of the electromuscular phenomena observed [in vegetal colic] with those that characterise lead palsy is a new and powerful argument for those who see vegetal palsy and saturnine palsy as having a common cause, i.e. lead poisoning" [30].

In her remarkable thesis defended on 26 July 1889, Augusta Dejerine-Klumpke (1859-1927) interpreted the history of paralysis related to lead as follows: "The history of saturnine palsy can be divided into several periods: a purely clinical period that includes the studies of Tanquerel des Planches and Duchenne de Boulogne, and a much more recent period, in which pathologic anatomy was used to elucidate the pathogeny of these forms of paralysis" [31]. She did not fail to pay homage to Tanquerel: "We should mention the memorable work of Tanquerel des Planches and Duchenne de Boulogne leading to the magisterial clinical description of lead palsy. We owe everything that could be said on lead palsy before the use of electrical currents to Tanquerel des Planches, and his description laid the groundwork for all later clinical studies."

## 2.4. Treatise on lead or saturnine diseases

After his thesis on lead colic, defended on 10 July 1835, in which he referred to convulsive episodes and lead palsy, Grisolle described in 1836 "cerebral accidents produced by saturnine preparations" [32]. He noted the rarity of the cases described up to that time: "Having no knowledge, in all the cases of the cerebral condition caused by lead, I can propose no new expression to describe the disease. By using the term 'saturnine cerebral accidents', I am in no way suggesting anything about its nature that eludes our investigations." In response, Tanquerel coined the word "encephalopathy" in 1836, a term that is still in use today [29,33]. He explained his reasoning in his defining work, the two-volume *Traité des maladies de plomb ou saturnines* (treatise on lead or saturnine diseases) (Fig. 4), noting that this name was immediately adopted by many authors, such as Pierre-Adolphe Piorry in his

1837 *Traité du diagnostic et sémiologie* (treatise on diagnosis and semiology). Encouraged by the interest that his thesis generated, Tanquerel continued to collect clinical cases of lead poisoning in the departments of La Charité Hospital. Some one hundred observations enabled him to present the complete clinical picture of lead poisoning as it was understood in the middle of the nineteenth century.

In the first volume, he made an initial study of lead colic and its premises, notably the changes of colour in the teeth and the mucous membranes of the mouth, the impairment of taste, the characteristic fetid breath, and the mustiness of the patients of living spaces. In addition to the toxicity studies for the various chemical forms, Tanquerel presented the routes of lead absorption in the organism and made a long list of exposed occupations (he did not fail to mention lapidaries, crystal cutters, mirror cutters, pharmacists, etc.). He described in detail the clinical aspects of colic and noted concomitant icterus, the disease's progression, and the proposed treatments. Using comparisons on series of patients who had been treated and others who had not, he showed the ineffectiveness of most therapies used at the time, notably "limonade sulphurique" (sulphuric lemonade). He recommended the "La Charité treatment" combining purgations with senna, ricin oil, and Sedlitz water (magnesium). But his preference was oil of croton which he combined with purgations using "sudorific infusions", belladonna, and opiates for pain. Tanquerel mentioned the attempts of Mérat, with the help of the pharmacist Ernest Barruel, to establish doses "in the management of individuals suffering from lead colic" but his efforts were in vain. He did not fail to note that Alphonse Devergie (1798-1879) and Osmin Hervy (1815-1841) obtained the first tissue doses for lead in 1838 [34].

The second, more original volume focused solely on damage to the central nervous system in lead poisoning. He referred to his thesis to treat patients with lead palsy by using statistics to describe the symptoms in detail, notably the localisations of the paralysis. Among his observations, he cited the case of a painter with paralysis of the fingers, resembling Aran-Duchenne hand, combined with "trembling" of the muscles of the lower arm where he suspected lead as the cause. Although he did not describe amyotrophy, was this a case of amyotrophic lateral sclerosis? As in his thesis, Tanquerel was surprised that no lesion visible to the naked eye had been observed during autopsies on the central and peripheral nervous systems: "It is impossible for us to say how the poison works on the spinal cord to determine this disturbance in the production of the motor agent; this inner action is unknown to us even though it is as demonstrated as that of strychnine". It is clear here that Tanquerel does not situate the control of motricity in the brain but rather in the spinal cord.

His second chapter deals with "saturnine anaesthesia". Combined or not with paralysis, this superficial anaesthesia affects the sense of touch. Tanquerel gave no indication concerning the perception of hot/cold or of pain. He distinguished "saturnine anaesthesia of the retina" causing a more or less complete, uni- or bilateral amaurosis. The disturbance took a few hours to develop then disappeared as quickly, after only a few days.

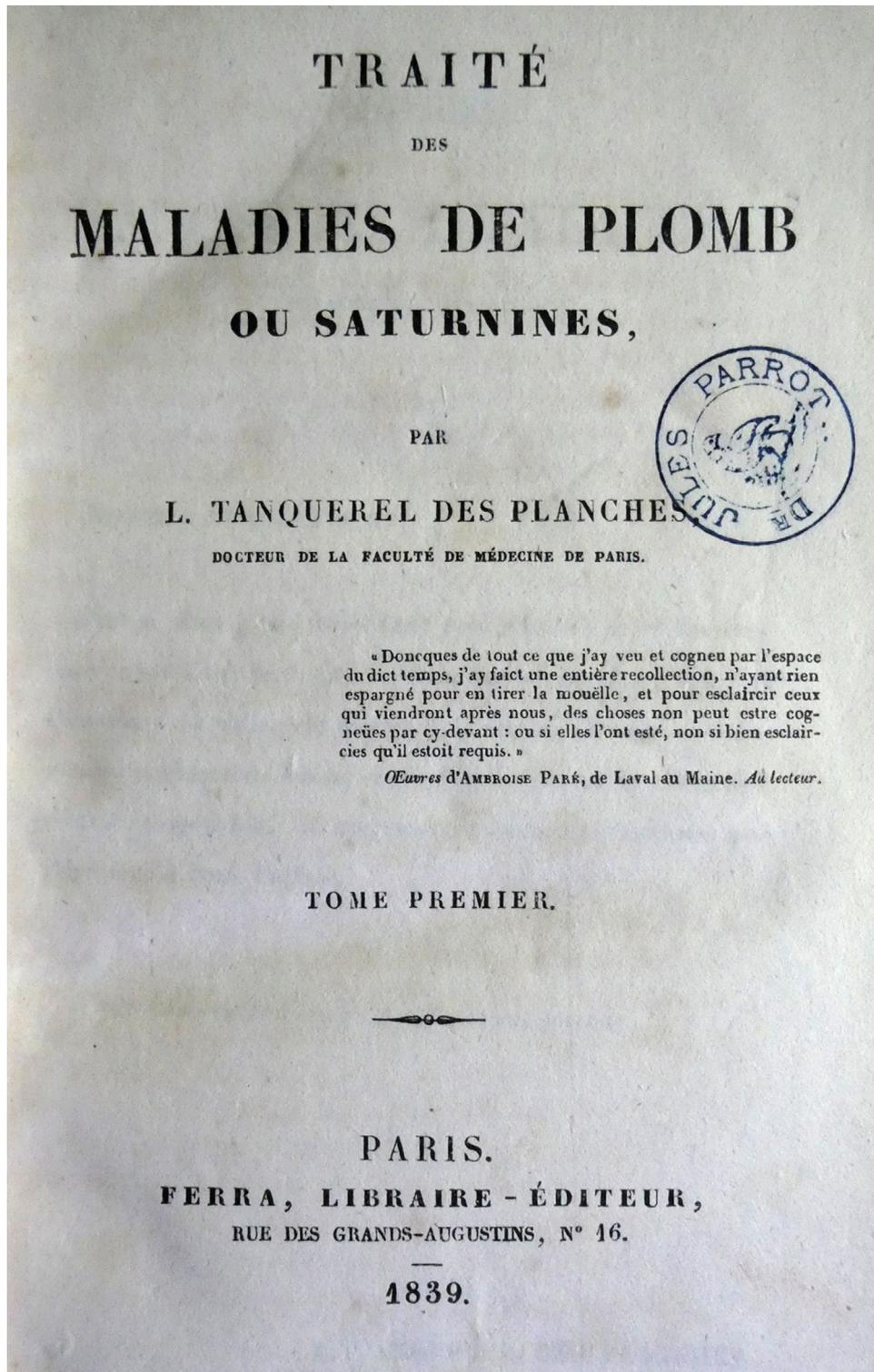


Fig. 4 – Cover of the treatise on lead disease by Tanquerel des Planches (OW collection).

## 2.5. Saturnine encephalopathy

“The poisoning results in various morbid phenomena: delirium, coma, or convulsions which are combined or not with the loss of one or more senses” [33]. This was Tanquerel’s definition of saturnine encephalopathy. One of the characte-

ristics that he interpreted as a diagnostic element was the rapid variability of the symptoms; paralysis of a limb could last several hours, as could blindness or delirium. He mentioned that many authors from antiquity spoke of lead colic, noting simultaneous epileptic seizures. Lithargyric wines specifically caused convulsions (the additive result of alcohol is probable).

Tanquerel's starting point was the descriptions provided by Vincent Nivet (1809-1893), who was then a resident and would go on to a professorship at the medical school in Clermont-Ferrand. These descriptions were published in 1836 in the *Gazette médicale de Paris* [35]. Tanquerel considered Nivet as the first author to provide complete, coherent clinical descriptions for the interpretation of symptoms, attributing the neurological disorders specifically to lead poisoning.

Tanquerel based his position on seventy-two observations that he had compiled himself. Most frequently, the patients with saturnine encephalopathy had never shown neurological disturbances before coming into contact with lead. Their symptoms disappeared once they no longer had contact with this metallic poison, but they fell out of remission when they were once again around lead. For Tanquerel, this argument was the irrefutable proof of the damaging role of lead absorption at that time, almost exclusively by digestive mucous membranes. His other argument was as follows: "Cats and other animals that spend time in the white lead and minium workshops are frequently affected by circling disease, horrible convulsions, and soporific accidents, causing rapid death" [33].

He established statistics for the exposed occupations which primarily indicated white lead and minium. The contact time varied considerably, from eight days to nine months, before the first symptoms developed, without necessarily being preceded by colic (6 out of 72). The seriousness of earlier colic leading to encephalopathy did not in any way predict the seriousness of the disturbance. The treatment used at that time at La Charité Hospital to alleviate colic had no effect on preventing the appearance of neurological disorders. "All these facts indisputably prove that encephalopathy is one of the distinct forms of saturnine poisoning and is independent of colic and other forms of lead disease" [33]. Tanquerel identified poor health in individuals and alcoholism as concomitant factors that favoured the condition. The significant variability of several factors made it impossible for him to predict with certainty the risks for a worker in particular to be a victim of encephalopathy when he was hired: "The existence of a constant, unknown relationship between the production of this disease and the current mechanism of the organism in individuals under the influence of lead preparations" [33].

Certain prodromes generally signal more serious disorders a few hours beforehand: headaches, trembling, bizarre hallucinations, dizziness, amaurosis, vacant gaze, anxiety and sadness, and night terrors. The main symptoms combine mental and neurological disorders that can occur in isolation or simultaneously, without any precise order or chronology. Tanquerel drew from the descriptions written by alienists and described four forms of saturnine encephalopathy: with delirium, coma, convulsions, and a form combining these three clinical pictures.

The delirium could be calm. The expression of emotions in the patients was not correlated with the circumstances; a state of vacancy could be interrupted by fits of laughter, and a melancholy appearance was associated with delirious speech marked by megalomania, a sense of familiarity with strangers, etc.

The delirium could be tied to rage: "A general exaltation could be seen in all the patients' acts; they cried out,

vociferated, swore, threatened, stormed about, ripped their clothing, broke the straps that bound them to their bed, ran through the wards, attacked for no apparent reason anyone that got in their way, tried to fight with them, injure them, bite them, while subjecting them to the most crude insults" [33]. He observed cases of defenestration and self-mutilation. Speech was delirious and incoherent. All of this could last several hours, in the daytime as well as at night, for a prolonged period that was complicated by a worsening general condition with dehydration and loss of weight: "The mouth dries out and the arid tongue may even become encrusted". Remissions were followed by relapses; both types of delirium could alternate and lead to somnolence preceding a coma. "Individuals with saturnine encephalopathy (delirious form) were sent to Bicêtre Hospital for the insane. A few patients very promptly recovered but were cruelly held for months on end in this establishment to ensure there would be no relapses" [33]. For Tanquerel, delirium tremens caused by alcoholism was the only pathology that could result in a diagnostic error.

Coma could also set in after a few hours: "Some automatic movements of the head, trunk, or limbs can be observed from time to time. The pupils are dilated and sometimes narrowed; they persist in this state in the presence of bright light or may slowly narrow" [33].

"Convulsions are saturnine accidents encountered frequently, but very rarely in isolation" [33]. In addition to partial or generalised forms, the episodes could also appear to be tetanus or catalepsy. One of the particularities noted by Tanquerel was generalised trembling as a precursor to the episode. The convulsions were marked in the face and the upper limbs but frequently without loss of consciousness because, according to Tanquerel, the patients bore witness to their full experience of the episode once I had stopped and the postictal somnolence had dissipated. Thirty-six of the seventy-two cases involved convulsions, either right away, after a phase of delirium, or during their coma. The extremely detailed descriptions of the episodes take up several pages of Tanquerel's treatise; they could be said to replace the videos we now have access to.

Tanquerel did not classify the forms of paralysis, with or without anaesthesia or amaurosis, and regarding encephalopathic symptoms. He did note, however, that they could appear before, during, or after the episodes of delirium, convulsions, or coma. In all cases, it remained difficult to establish a prognosis. After hours or days of episodes, at most lasting seventeen days in his observations, death at times occurred unexpectedly, in any phase of saturnine encephalopathy. In other cases, after a coma lasting a few days, consciousness slowly returned after a recovery of cerebral functions that appeared to be complete. Prolonged sleep could also terminate an episode with a return to normal prior to waking. Tanquerel also observed what he called "saturnine dementia", a state in which mental condition fluctuated from one hour to the next, without progression after several weeks of development.

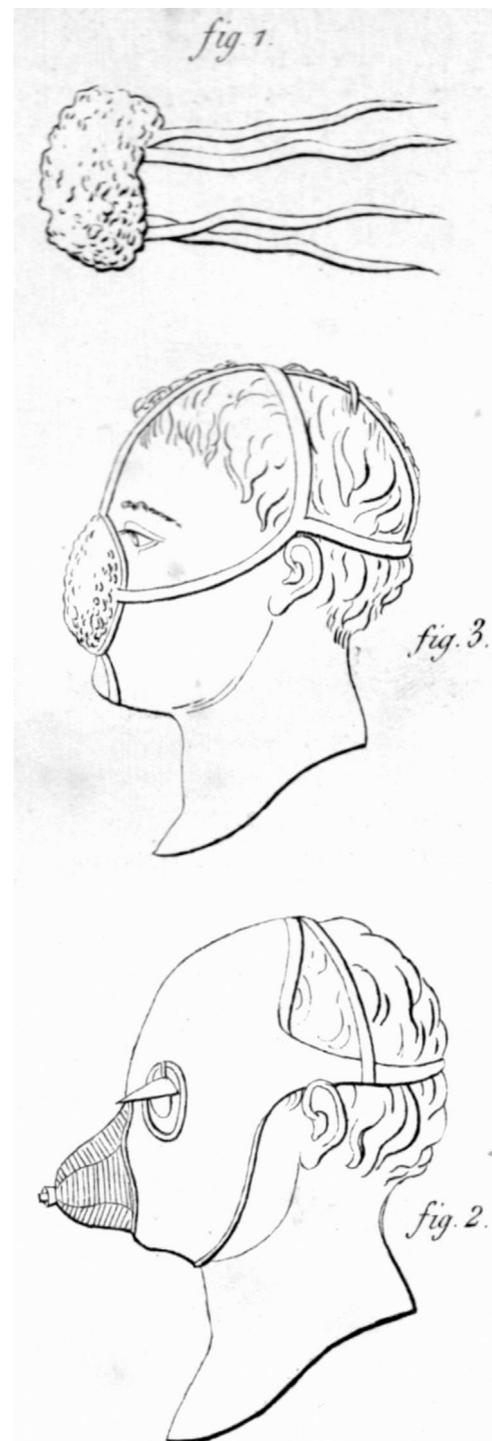
At the autopsy of twenty-one cases, only one macroscopic examination of the brain was performed. Tanquerel reached the same conclusions as Grisolle and Nivet: "Following saturnine epilepsy, there was hypertrophy of the brain mass,

characterised in the following manner: flattening and piling of cerebral circumvolutions and anfractuosités eliminated for the most part, such that the surface of the hemispheres was almost uniform". He added, "Increase in the density of the soft part of the brain and reduction in the capacity of the ventricles." [33] He thus observed cerebral oedema that probably caused intracranial hypertension that led to death. He discussed the moment at which this state was established. Did the poisoning lead to and cause convulsions? Or was the oedema the result of the convulsions? In certain cases he did not see any apparent anomaly; "if the symptoms were produced by anatomical lesions, they should always be evident" [33]. He concluded that the lesions observed were secondary and not the cause of the symptoms of saturnine encephalopathy.

One of the important influences of Tanquerel's work that distinguished it from some of Grisolle's writings is that he submitted two brains to chemical analysis performed by Devergie and the pharmacist Gaston Guibourt (1780-1867) to detect the presence of lead in the cerebral tissue. In two observations, the presence of lead was established whereas "this famous chemist had never encountered lead as a constituent element of this organ" [33]. Observation XI concludes with the technique these pharmacists used: "After drying the animal material, it is fired to reduce coal. It is then calcined in a porcelain crucible and the coal is washed off several times as calcination proceeds. Once the material is incinerated, it is treated with hydrochloric acid. Part of the acid is evaporated, water is added, and the aqueous liqueur is then treated with hydrogen sulphide. It forms a chocolate brown or black precipitate, depending on the predominance of copper or lead. It is allowed to deposit, then collected in a small porcelain cup, followed by nitric acid treatment. The mixture is extended with water, filtered, and evaporated at low heat. The filtration product contains both metals as salts. Regarding lead revivification, it entails using a blowtorch on the sulphur" [33].

Tanquerel included in his treatise the same therapeutic propositions as those described in his thesis. He added a long chapter on "prophylactic treatment or methods for protecting against saturnine diseases" which began as follows: "For some time, authorities and all of society have been moved by the story we and other observers have told of the serious accidents that so often and so cruelly strike a large portion of the working class and individuals who work with lead preparations, to meet the needs and provide the amenities of life" [33]. For this reason, Tanquerel was called upon by the council in charge of public health for the city of Paris to propose prophylactic measures. His in-depth inquiry led him to visit many workshops. He questioned owners, workshop supervisors, and workers, artists, and artisans. He believed that the government had an obligation to enforce the "simple and inexpensive" precautions that he recommended.

Here are few of his propositions: promotion of individual health, continuous ventilation in workshops, wearing facial sponges and masks that he had designed (Fig. 5), using oilcloth coats, providing a dressing room so that soiled clothes would not be taken home, washing mouth and teeth after work, and prohibiting meal preparation and eating in the workshops. Tanquerel estimated that the number of exposed workers could be drastically reduced if their organisation was



**Fig. 5 – The prophylactic sponges and masks designed by Tanquerel (OW collection).**

improved. The fight against alcoholism, the obligation to eat before starting to work (he noticed that fasting hastened poisoning), and separating workshops from living spaces rounded out the recommendations he made. He discouraged all forms of spirits which were supposedly protective and were thus recommended, but they had no demonstrated effectiveness and could mask the consumption of alcohol in the

workplace. Finally, he completed his book by reviewing the various exposed occupations and stipulating the rules specific to each one.

### 3. Conclusion

Tanquerel's unique area of medical interest was lead poisoning at a time when its devastating effects were of little interest to the medical profession. By naming and identifying the neurological symptoms of this occupational disease, which went much further than the lead colic known for over two millennia, Tanquerel enabled real progress in medical knowledge. This opened the way to identifying a toxic occupational impairment of the central nervous system. Not stopping there, he expressed concern over the working conditions of the proletariat who were exploited in the white lead workshops of the nineteenth century. This made him a forerunner of public health specialists and occupational physicians. The term encephalopathy, which we owe to him, has remained so common among neurologists that they have forgotten its inmarcescible creator. It is appropriate to shine new light on Tanquerel at a time when the silent epidemic of lead poisoning continues to claim its victims. Because lead is not biodegradable, it demonstrates remarkable environmental persistence. Despite the fact that the amount of lead in paint intended for use in or on residential buildings, furniture, or children's toys has been forbidden, lead-based paint continues to be a major source of lead exposure in young children. This is partially due to the fact that several million young children live in older homes in which lead-based paint was previously used, and as this old paint ages, it peels, flakes, and crumbles into dust that settles on the interior surfaces of homes and in the soil surrounding the exterior of the home. The dust and soil containing these tiny paint particles inevitably make their way into children's mouths as a result of normal childhood exploratory hand-to-mouth behavior. The monitoring of children living not far from Notre-Dame is a good example, recalled here. Children are at greater risk of developing manifestations of lead toxicity because more of their lead body burden is stored in metabolically active sites. It must be remembered that no safe level of lead exposure has been determined [36].

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### Statement of ethics

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