History of the emergence of seizure therapy

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10th NACT Meeting

May 21-23 2014 Tallinn

László Meduna (1896-1964)

- **Biography**

- Born: 1896, Budapest
- Catholic secondary school
- 1914 attended to the Faculty of Medicine, Pázmány Péter University
- 1915 volunteer on the Italian front
- 1921 medical degree
- 1922 employment in the Interacademic Brain Research Institute led by Károly Schaffer – began a reseach to the structure of corpus pineale
- 1925 moved to the Department of Psychiatry and Neurology together with Prof. Schaffer
- In one year time became the head of the outpatient service – interested in the neuropathology of idiopathic epilepsy
- Identified **glia cell proliferation** in excised epileptic brain tissue
- 1933 Béla Horányi (Hechst) described the **lack of glial reaction** in schizophrenia (neuropathological findings)

Antagonism between epilepsy and schizophrenia?

- Wagner-Juaregg: theory of antagonism between different disorders
- Antagonism between syphilis and malaria: fever therapy
  - (thermosensitivity of Treponema pallidum)

Clinical findings

Gyula Nyírő, Albin Jablonszky: Some data concerning the prognosis of epilepsy, with special attention to constitution. Orvosi Hetilap 1929;28:679-681.

176 patients with epilepsy	recovered	improved	no improvement	uncertain
Combined with schizophrenia	16,05%	65,43%	13,58%	4,94%
pure epilepsy	1,05%	23,16%	71,55%	4,24%

International reports in line

- Müller reported in 1930 two cases with schizophrenia whose psychotic symptoms disappeared after an accidental epileptic fit.
- In 1931 Glaus found only 8 patients with co-morbide epilepsy among 6000 schizophrenic patients
- In 1932 Steiner and Strauss, after reviewing the files of 6000 patients with schizophrenia concluded that in the presence of the illness the occurance of an epileptic fit is so rare, that its appearance raised a doubt concerning the accuracy of the diagnose

Finally Meduna concluded

'…if I can stimulate epileptic seizures in schizophrenics then these… will alter the chemical and humoral processes in the body in a way… that the suppression of the disease will be made physiologically possible.'

Next step: find an appropriate and safe agent for seizure induction

- Results according to safety

-strichnin, brucin, tebain: tetania

-coramin: gap between fit-inducing and lethal dose minimal

-caffeine, absinthe: cerebrovascular pathology (bleeding, inflammation)

-camphor

- Titration of the fit-inducing and lethal dose on guinea pigs (Meduna L.: Über experimentelle Campherepilepsie. Arch Psychiatr 1934;102:333-9.)
  - No cerebrovascular pathology

Conflict with Károly Schaffer

- Strong supporter of the heredodegeneration theory
  - Schizoprenia is a genetic disorder
  - untreatable
- Thus denied Meduna's new idea

Moving to Lipótmező

- Asylum with 500 beds
- Head of department position
- First human experiments

Sources of Meduna's recollections about the first human experiments

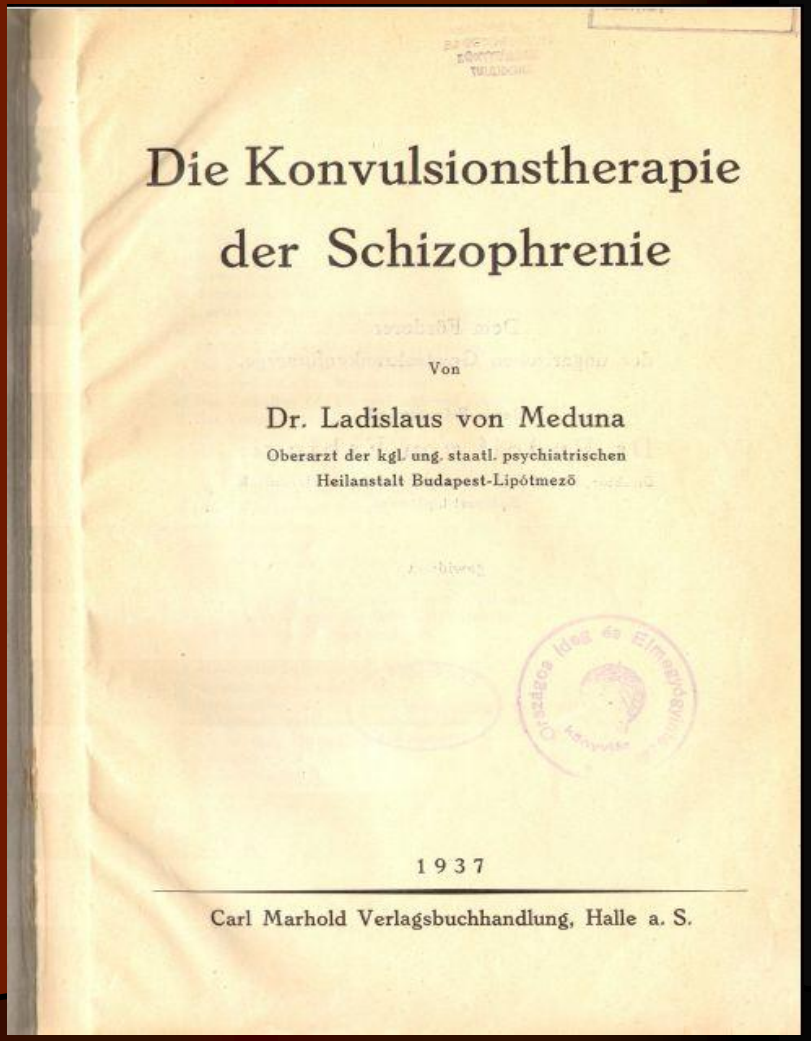
- Meduna L (1937) Die Konvulsionstherapie Der Schizophrenie. Carl Marhold Verlagsbuchhandlung, Halle.
- Meduna L (1954) The convulsive treatment: a reappraisal. J Clin Exp Psychopathol 15:219-33.
- Meduna L (1985) Autobiography. Convulsive Ther 1: 43-57, 121-138

First patient according to Meduna's autobiography:

- Name: Zoltán L.
- *"suffered from catatonic stupor for about four years"*
- *"He never moved, never ate, never took care of his bodily needs and had to be tube-fed"*
- was injected with camphor-in-oil in the morning of January 23, 1934.

Alternative source

- Monograph about the first 110 convulsive treated patients
- Published in 1937, in German
- Short vignettes about the successful cases
- Among them L Zoltan's case



Die Konvulsionstherapie der Schizophrenie

Von

Dr. Ladislaus von Meduna

Oberarzt der kgl. ung. staatl. psychiatrischen Heilanstalt Budapest-Lipótmező

1937

Carl Marhold Verlagsbuchhandlung, Halle a. S.

Discrepancy: L Zoltán-April?

hält er 1,2 g Cardiazol i.m. Nach der Injektion benommener Eindruck; Anfall tritt nicht ein. Am 4. 6. erhält er 1,3 g Cardiazol i.m.. Eine halbe Stunde nach der Injektion tritt ein 64 Sek. dauernder Anfall ein, wonach Pat. steif und unbeweglich im Bette liegt, nicht spricht, keine Nahrung aufnimmt. Am 5. 6. liegt er unbeweglich steif im Bette, muß gefüttert werden, spricht nicht. Am 7. und 11. 6. erhält er 1,3 bzw. 1,4 g Cardiazol i.m.; kein Anfall. Pat. macht einen benommenen Eindruck. Am 14. 6. erhält er 1,5 g Cardiazol i.m.; kein Anfall. Sieht benommen aus. Am 18. 6. 1,6 g Cardiazol i.m. Darauf tritt 67 Sek. langer Anfall auf. Nach diesem beträgt der Puls 96, die Atmung 20. Pat. ist nach dem Anfall ängstlich, stundenlang benommen, wälzt sich auf dem Bette, will aufstehen. Allmählich beruhigt er sich, auf Fragen antwortet er nicht. Am 21. 6. erhält er 1,6 g Cardiazol; kein Anfall, aber Pat. ist etwa 1 Std. lang benommen, spricht nicht, nimmt keine Nahrung zu sich. Am 22. 6. ist das Verhalten des Pat. schlagartig verändert. Er ist völlig klar, ruhig, zugänglich, spricht mit seiner Umgebung, antwortet verständig auf Fragen, zieht sich ordentlich und gewählt an und macht einen Spaziergang im Garten. Eine retrospektive Untersuchung unterlassen wir einstweilen aus psychischen Gründen. Am 24. 6. hält seine Remission unverändert an. Pat. wird entlassen.

Am 14. 8. erscheint Pat. zur Nachuntersuchung. Er ist völlig ruhig, geordnet und ausgeglichen. Er hat Einsicht in seine Krankheit. Bezüglich seiner Erkrankung hat er aber nur verschwommene, nebelhafte Vorstellungen bzw. Erinnerungsbilder. Seinen Negativismus kann er nicht erklären. Gegenüber seiner Individualität vor der Erkrankung kann er selbst keine Veränderung finden; seine Stimmung ist heiter, er geht gern in Gesellschaft und will später eine Stellung annehmen. Nach Angaben seines Vaters ist eine Änderung gegenüber der Zeit vor der Erkrankung nur insofern festzustellen, als seine früher sprudelnde Sprechweise ruhiger und flüssigerer Redeweise gewichen sei. Auch ihm scheint diese Veränderung günstig. Pat. hat an Gewicht zugenommen, sein Betragen ist offen, seine Fixiertheit, Starrheit und Halbheit sind völlig geschwunden. Er ist derzeit völlig gesund. Sein Urlaub wird bis 1. 9. verlängert.

Am 1. 10. meldet sich Pat. zur Nachuntersuchung. Er ist völlig geordnet, ist dicker geworden, seine Stimmung ist ruhig, heiter, er weist keinerlei Zeichen von Geisteskrankheit auf. An seine frühere Erkrankung kann er sich nur ungefähr erinnern. Er selbst stellte fest, daß sein Erinnerungsvermögen, soweit es sich auf den Ablauf und die Dauer seiner Erkrankung bezieht, mangelhaft ist. Er erinnert sich nicht gern an seine Krankheit, aber als wir ihn dazu aufforderten, gab er seine Erinnerungsbilder später bereitwillig bekannt. Er wurde als völlig geheilt entlassen.

Epikrise: An dem väterlicherseits belasteten Individuum kommt ein typischer katatonischer Zustand zur Ausbildung. Seine Behandlung erfolgt, genau genommen, in zwei Phasen. In der dazwischenliegenden Zeit macht Pat. eine Scharlachinfektion mit hohem Fieber durch. An seinem katatonen Zustand ändert sich dadurch nichts. In der ersten Phase erhielt Pat. 0,8 g Cardiazol und 133 g Kampfer und bekam dadurch insgesamt 7 Anfälle. Auf weitere 2 Cardiazol-Injektionen traten 2 Anfälle auf, nach denen der Zustand des Pat. sich zu bessern begann. Nach einem 6 Tage langen Zustande halber Remission erkrankte Pat. an Scharlach. Psychisch fiel er in seine Katatonie zurück. Nach Abklingen der Scharlachinfektion wurde die Krampfbehandlung neuerlich aufgenommen. Pat. remittierte nach 4 epileptischen Anfällen endgültig. Er erhielt im Laufe der Behandlung folgende Injektionen: 2 mal Cardiazol i.v. je 0,4 g; 25 Kampferinjektionen mit insgesamt 133 g (7 epileptische Anfälle); 3 mal Cardiazol i.v., zusammen 1,5 g (3 Anfälle); 7 mal Cardiazol i.m., zusammen 9,9 g (3 Anfälle).

5. Fall. Zoltan L., 33 jähriger Elektroinstallateur. Anamnese: Ein Onkel väterlicherseits und eines seiner Geschwister sind gemütskrank.

Aufnahme ins Krankenhaus am 18. 10. 1930. Bei der Aufnahme zeigt er das Bild der Schizophrenia simplex, die langsam in katatonischen Stupor übergeht. Von April 1931 bis Januar 1934 liegt er in völligem Negativismus zu Bett, muß künstlich

ernährt werden, spricht nicht, läßt Harn und Stuhl unter sich. Im April 1934 begannen wir die Konvulsionstherapie. Pat. bekam 33 Kampfer- und Cardiazol-Einspritzungen und reagierte mit 25 Anfällen. Völlige Remission. (Diesen Fall habe ich in meiner ersten Veröffentlichung als „1. Fall L. Z." ausführlich beschrieben.)

6. Fall. Anton S., 14 jähriger Schüler. Ein Onkel mütterlicherseits ist gemütskrank. Wichtigere anamnestische Daten sind unbekannt.

Anstaltsaufnahme erfolgte am 5. 6. 1935. Bei der Aufnahme ist Pat. unruhig, verwirrt. Bei der Untersuchung zeigt er Logorrhöe, verwirrte Assoziationen, die allerdrastischsten Flüche fließen sozusagen in ununterbrochenem Strom aus ihm heraus. Sein Benehmen ist unruhig. Er hält keinen Augenblick still, will öfters fortgehen; dann wieder erschlafft seine Muskulatur völlig, so daß er auf dem Stuhle festgehalten werden muß, um nicht herunterzufallen. Einer genaueren Untersuchung ist er unzugänglich. In den ersten 5 Tagen muß er wegen hochgradiger psychomotorischer Unruhe unter Morphin-Scopolamin gehalten werden.

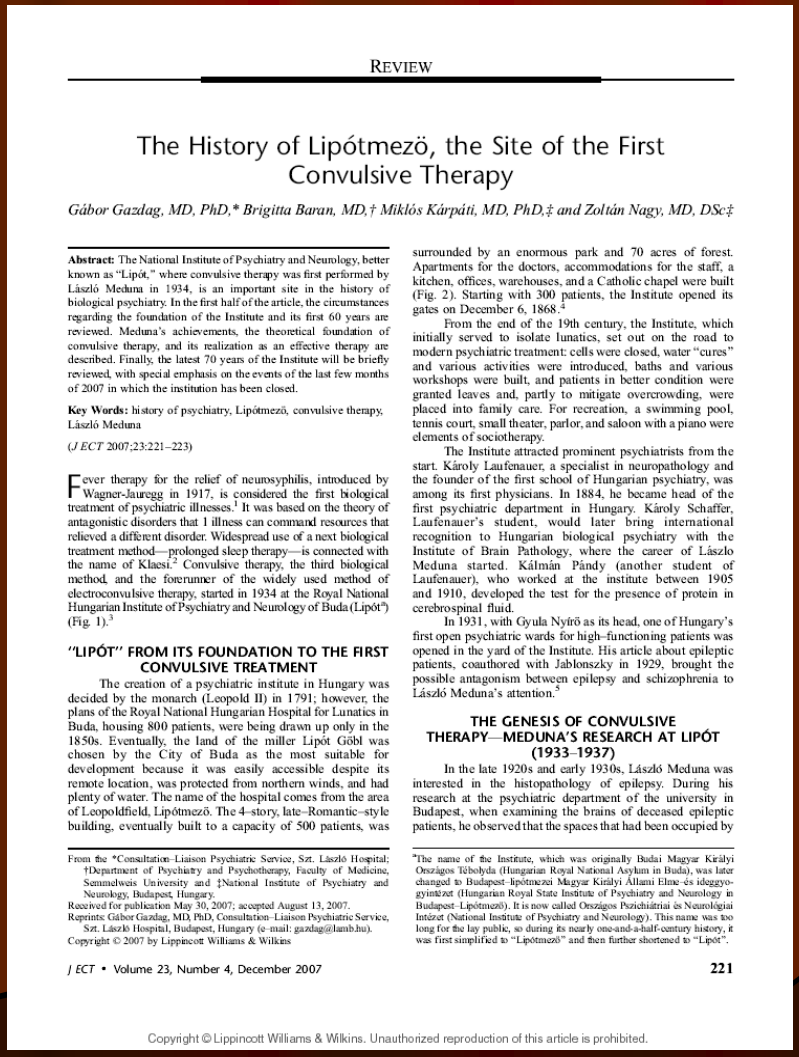
Am 11. 6. erhält Pat. die 1. Cardiazol-Injektion in der Dosis von 0,5 g i.v.; Anfall tritt nicht auf. Zustand unverändert. Am 14. 6. 0,6 g Cardiazol i.v. Darauf macht Pat. benommenen Eindruck, Anfall tritt nicht auf. Am 18., 21. und 25. 6. und am 2. 7. bekommt Pat. Cardiazol i.v. in folgenden Dosen: 0,7, 0,8, 0,9, 0,9 g. Am 5. 7. ist der Zustand unverändert; Pat. ist unruhig, lärmend, läßt sich nicht säubern, den ganzen Tag stößt er schwerste Flüche und Beschimpfungen hervor. Er erhält 0,95 g Cardiazol i.v., worauf er mit einem 74 Sek. dauernden epileptischen Anfall reagiert. Nach diesem ist er 10 Min. lang benommen und unruhig. Am 7. 7. ist Pat. unruhig, von Zeit zu Zeit schreit er anhaltend, zeitweise stößt er einzelne Schreie aus. Er geht hin und her, liegt auf dem Fußboden herum. Am 9. 7. 0,95 g Cardiazol i.v. Danach ist er benommen, seine Oberlippe zuckt einige Male, nach der Injektion liegt er ruhig. Nachmittags ist er ruhig und still, nachts schläft er. Am 10. 7. benimmt er sich ruhig, still, zieht sich an, führt Gespräche mit seinen Verwandten.

Am 18. 7. oberärztliche Untersuchung. Das Benehmen des Pat. ist ruhig, gesittet, keinerlei pathologische Symptome festzustellen. Fragen beantwortet er liebenswürdig, lächelnd und unmittelbar. Linkisches Wesen, Starrheit, Manieriertheit sind nicht mehr zu bemerken. Bezüglich seines früheren Zustandes zeigt er völlige Amnesie, retrospektive Daten sind deshalb nicht zu erhalten. Pat. ist als völlig geheilt anzusehen. Am 25. 7. nimmt ihn sein Vater, dem ärztlichen Rat entgegen, nach Hause.

Am 7. 8. wird Pat. von seinem Vater wieder eingeliefert. Der Vater erzählt, daß sein Sohn seit einer Woche völlig verwandelt sei. Er will nicht essen, ist dazu nur mit Überredungskünsten zu bewegen. Er vernachlässigt sich vollständig; wenn man ihn nicht dazu zwingt, wäscht er sich nicht, schläft seit Tagen nicht, spricht nicht, murmelt höchstens einige unverständliche Worte, jede Nacht springt er plötzlich aus dem Bett und untersucht die Türen. Bei der Untersuchung ist Pat. in hohem Maße gehemmt, sitzt fast unbeweglich auf einem Fleck, von Zeit zu Zeit macht er kleine Bewegungen, bei deren Ausführung er unsicher erscheint und häufig nicht zu Ende kommt. Sein Blick ist leer, er starrt gedankenlos in die Luft, die Vorgänge um ihn herum interessieren ihn nicht, sein Ausdruck verrät eine gewisse Angst. Auf Fragen antwortet er meistens nicht, nur gelegentlich nach langwierigem Zureden antwortet er kurz mit ja oder nein.

Am 9. 8. erhält Pat. 0,9 g Cardiazol i.v., worauf er mit einem 60 Sek. dauernden Anfall reagiert. Am 13. 8. ist sein Zustand unverändert. Pat. ist verschlossen, gehemmt, gibt auf Fragen flüsternde, zusammenhanglose, verwirrte Antworten. Er erhält 0,9 g Cardiazol, worauf ein 55 Sek. langer Anfall eintritt. Am 15. 8. ist Pat. schweigsam, mißgestimmt, blickt träumerisch um sich. Auf Fragen antwortet er nur stockend mit einzelnen Worten. Auf diesbezügliche Fragen berichtet er, er habe Angstzustände gehabt, verschiedene Stimmen gehört, höre aber jetzt noch Stimmen. Während der Untersuchung wird Pat. immer wortkarger, schweigsamer, versagt

Decision on the closure of Lipótmező in 2007



REVIEW

The History of Lipótmezö, the Site of the First Convulsive Therapy

Gábor Gazdag, MD, PhD, Brigitta Baran, MD,† Miklós Kárpáti, MD, PhD,‡ and Zoltán Nagy, MD, DSc‡*

Abstract: The National Institute of Psychiatry and Neurology, better known as "Lipót," where convulsive therapy was first performed by László Meduna in 1934, is an important site in the history of biological psychiatry. In the first half of the article, the circumstances regarding the foundation of the Institute and its first 60 years are reviewed. Meduna's achievements, the theoretical foundation of convulsive therapy, and its realization as an effective therapy are described. Finally, the latest 70 years of the Institute will be briefly reviewed, with special emphasis on the events of the last few months of 2007 in which the institution has been closed.

Key Words: history of psychiatry, Lipótmező, convulsive therapy, László Meduna

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Fever therapy for the relief of neurosyphilis, introduced by Wagner-Jauregg in 1917, is considered the first biological treatment of psychiatric illnesses.[1] It was based on the theory of antagonistic disorders that 1 illness can command resources that relieved a different disorder. Widespread use of a next biological treatment method—prolonged sleep therapy—is connected with the name of Klaesi.[2] Convulsive therapy, the third biological method, and the forerunner of the widely used method of electroconvulsive therapy, started in 1934 at the Royal National Hungarian Institute of Psychiatry and Neurology of Buda (Lipót[a]) (Fig. 1).[3]

"LIPÓT" FROM ITS FOUNDATION TO THE FIRST CONVULSIVE TREATMENT

The creation of a psychiatric institute in Hungary was decided by the monarch (Leopold II) in 1791; however, the plans of the Royal National Hungarian Hospital for Lunatics in Buda, housing 800 patients, were being drawn up only in the 1850s. Eventually, the land of the miller Lipót Göbl was chosen by the City of Buda as the most suitable for development because it was easily accessible despite its remote location, was protected from northern winds, and had plenty of water. The name of the hospital comes from the area of Leopoldfield, Lipótmező. The 4–story, late–Romantic–style building, eventually built to a capacity of 500 patients, was surrounded by an enormous park and 70 acres of forest. Apartments for the doctors, accommodations for the staff, a kitchen, offices, warehouses, and a Catholic chapel were built (Fig. 2). Starting with 300 patients, the Institute opened its gates on December 6, 1868.[4]

From the end of the 19th century, the Institute, which initially served to isolate lunatics, set out on the road to modern psychiatric treatment: cells were closed, water "cures" and various activities were introduced, baths and various workshops were built, and patients in better condition were granted leaves and, partly to mitigate overcrowding, were placed into family care. For recreation, a swimming pool, tennis court, small theater, parlor, and saloon with a piano were elements of sociotherapy.

The Institute attracted prominent psychiatrists from the start. Károly Laufenauer, a specialist in neuropathology and the founder of the first school of Hungarian psychiatry, was among its first physicians. In 1884, he became head of the first psychiatric department in Hungary. Károly Schaffer, Laufenauer's student, would later bring international recognition to Hungarian biological psychiatry with the Institute of Brain Pathology, where the career of László Meduna started. Kálmán Pándy (another student of Laufenauer), who worked at the institute between 1905 and 1910, developed the test for the presence of protein in cerebrospinal fluid.

In 1931, with Gyula Nyírő as its head, one of Hungary's first open psychiatric wards for high–functioning patients was opened in the yard of the Institute. His article about epileptic patients, coauthored with Jablonszky in 1929, brought the possible antagonism between epilepsy and schizophrenia to László Meduna's attention.[5]

THE GENESIS OF CONVULSIVE THERAPY—MEDUNA'S RESEARCH AT LIPÓT (1933–1937)

In the late 1920s and early 1930s, László Meduna was interested in the histopathology of epilepsy. During his research at the psychiatric department of the university in Budapest, when examining the brains of deceased epileptic patients, he observed that the spaces that had been occupied by

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[a]The name of the Institute, which was originally Budai Magyar Királyi Országos Tébolyda (Hungarian Royal National Asylum in Buda), was later changed to Budapest–lipótmezei Magyar Királyi Állami Elme–és ideggyógyintézet (Hungarian Royal State Institute of Psychiatry and Neurology in Budapest–Lipótmező). It is now called Országos Pszichiátriai és Neurológiai Intézet (National Institute of Psychiatry and Neurology). This name was too long for the lay public, so during its nearly one-and-a-half-century history, it was first simplified to "Lipótmező" and then further shortened to "Lipót".

- Inspiration to start a work aiming at the finding of the medical documents of the first convulsive treated patients.

Method of the research

- Patients' files in the Archives of the National Institute of Psychiatry and Neurology, Budapest
- the records of those patients who had died or left the institute between 1933 and 1945.
- Found among those who died in 1945

L Zoltan’s chart

A budapesti I—III. kerületi királyi járásbiróságnak 19 30. évi X. hó 30 .... számú végzése alapján az intézetbe véglegesen felvétetett.

Pk IV 71170/1930

Budapest, 19 30. évi I. hó 30.

igazgató.

Értesítendő hozzátartozóinak címe

Gondnokság alá helyezve

gondnok.

Attila-nyomda r.-t. Buda, I., Szent János-tér 1/a. Tel. 355—77.

BUDAPEST-LIPÓTMEZEI M. KIR. ÁLLAMI ELME- ÉS IDEGGYÓGYINTÉZET

Kórrajz.

5 K uet.

Férfi/Női felvételi szám: 337.

Beteg neve: L [redacted] Zoltán

Kor 33. vallás ref. állapot nős

Foglalkozás villanyszerelő

Születési helye Illava Trencsén m.

Illetőségi hely Bp.-

Utolsó lakhely Bp. I. Hidegkúti út 2/b.

Szülei Mózes, F [redacted] Mária

Neje B [redacted] Rózsi

Felvétetett 19 30. évi okt. hó 18-n

Élelmezési osztály III.

Felvétel alapja Gy. eup.

Honnan szállították be? lakásáról

Ki szállította be? mentők

A büntetőtörvénnyel összeütközésbe jött betegsége előtt/után

által és mely biróságnál?

Hatósági elhelyezésnek minősül-e?

Testsúly a felvételkor

Testsúly az elbocsátáskor

Kórisme:

Schizophrenia

Betegség oka anyagi, terheltség (egy nagynénje és atyjának egy testvére elmebeteg volt)

Betegség kezdete

Volt-e már intézetben?

Sérülések, külső rendellenességek, vagy szervi betegségek, trachoma? scabies nincsen

Apadásba jött 19 45. év február hó 24.-n. d. u. 3/4 órakor.

Ápolási időtartam .... év .... hó .... nap.

~~Mint nem elmebeteg, gyógyultan, javultan~~ ~~gyógyulatlanul~~, elhalálozás folytán

tenosülése Gluczwar következtében

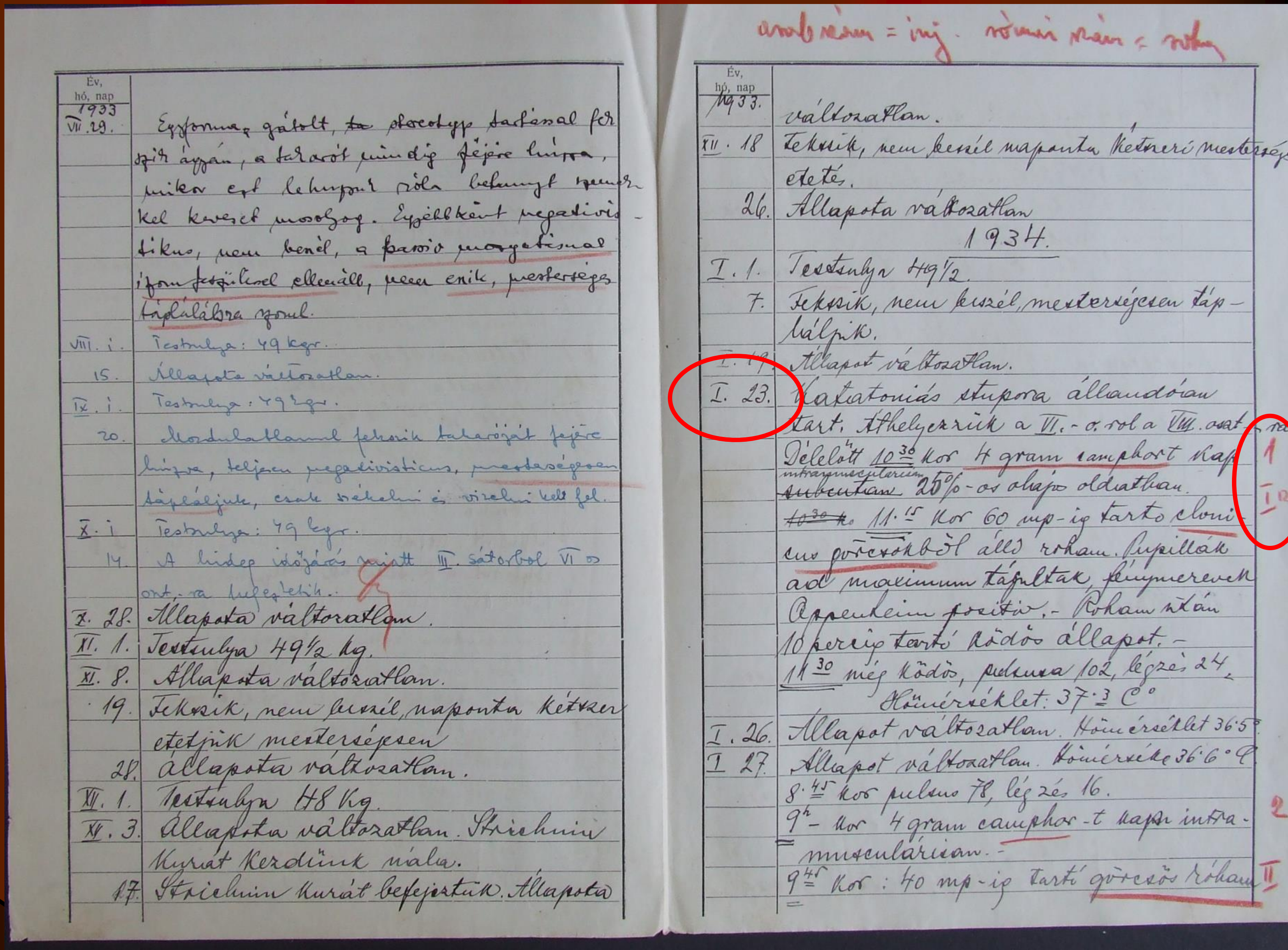
L Zoltán - history of illness, reason for admission

- About 3-3.5 years ago saw “strange people waving to him”, sneaking past him. Felt his body to be either very hot or very cold. Claimed that he is not the father of his child, heard voices coming from within his body.
- In the month before his admission, he asked his wife to give him a knife to kill himself.
- Treated for luetic infection about 9 years ago.
- Family history: a sibling and an uncle were mentally ill.

L Zoltán - symptoms at admission, course of illness in the Institute

- When examined, he did not respond to questions instead talked with his wife about their child. Tells that the voice coming from within his body saying: "Tell him who baby Böbi's father is!"
- Before convulsive therapy, stuporous, mute and required tube feeding.

The day of the first treatment

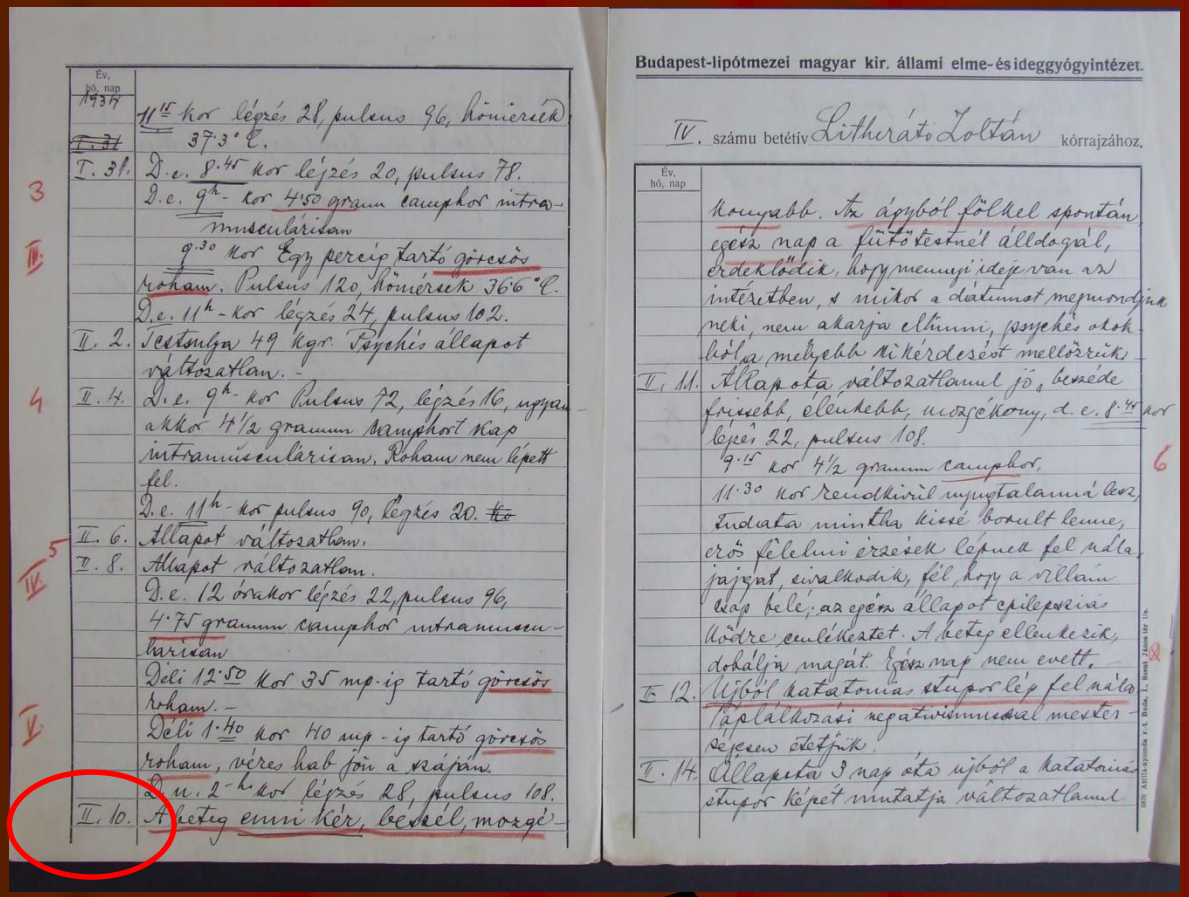


arab szám = inj. római szám = roham

Év, hó, nap	
1933 VII. 29.	Egyforma, gátolt, [illegible] stereotyp tartással fekszik ágyán, a takarót mindig fejére húzza, mikor ezt lehúzzuk róla befelé fordul [illegible] kel keveset mosolyog. Egyébként negativisztikus, nem beszél, a passiv mozgatásnak ugyanolyan ellenáll, nem eszik, mesterséges táplálásra szorul.
VIII. 1.	Testsulya: 49 kgr.
15.	Állapota változatlan.
IX. 1.	Testsulya: 49 kgr.
20.	Mozdulatlanul fekszik takaróját fejére húzza, teljesen negativisticus, mesterségesen tápláljuk, csak széklet és vizelet kell fel.
X. 1	Testsulya: 49 kgr.
14.	A hideg időjárás miatt III. sátorból VI os ont.-ra helyeztetik.
X. 28.	Állapota változatlan.
XI. 1.	Testsulya 49½ kg.
XI. 8.	Állapota változatlan.
19.	Fekszik, nem beszél, naponta kétszer etetjük mesterségesen
28.	állapota változatlan.
XII. 1.	Testsulya 48 kg.
XII. 3.	állapota változatlan. Strichnin kurát kezdünk nála.
17.	Strichnin kurát befejeztük. Állapota

Év, hó, nap		
1933.	változatlan.	
XII. 18	Fekszik, nem beszél naponta kétszeri mesterséges etetés.	
26.	Állapota változatlan	
	1934.	
I. 1.	Testsulya 49½.	
7.	Fekszik, nem beszél, mesterségesen táplálják.	
I. 19.	Állapot változatlan.	
I. 23.	Katatoniás stupora állandóan tart. Áthelyezzük a VI.-o.-ról a VIII. osztályra. Délelőtt 10[30] kor 4 gram camphort kap ~~subcutan~~ intramuscularisan 25%-os olajos oldatban. ~~10[30] ko~~ 11.[15] kor 60 mp-ig tartó clonicus görcsökből álló roham. Pupillák ad maximum tágultak, fénymerevek Oppenheim positiv. - Roham után 10 percig tartó ködös állapot. - 11[30] még ködös, pulsusa 102, légzés 24. Hőmérséklet: 37.3 C°	1 I roh
I. 26.	Állapot változatlan. Hőmérséklet 36.5 C°.	
I 27.	Állapot változatlan. Hőmérséklet 36.6 C°. 8.[45] kor pulsus 78, légzés 16. 9[h] - kor 4 gram camphor-t kap intramuscularisan. - 9[45] kor: 40 mp-ig tartó görcsös roham	2 II

The first sign of the improvement
(10th February)

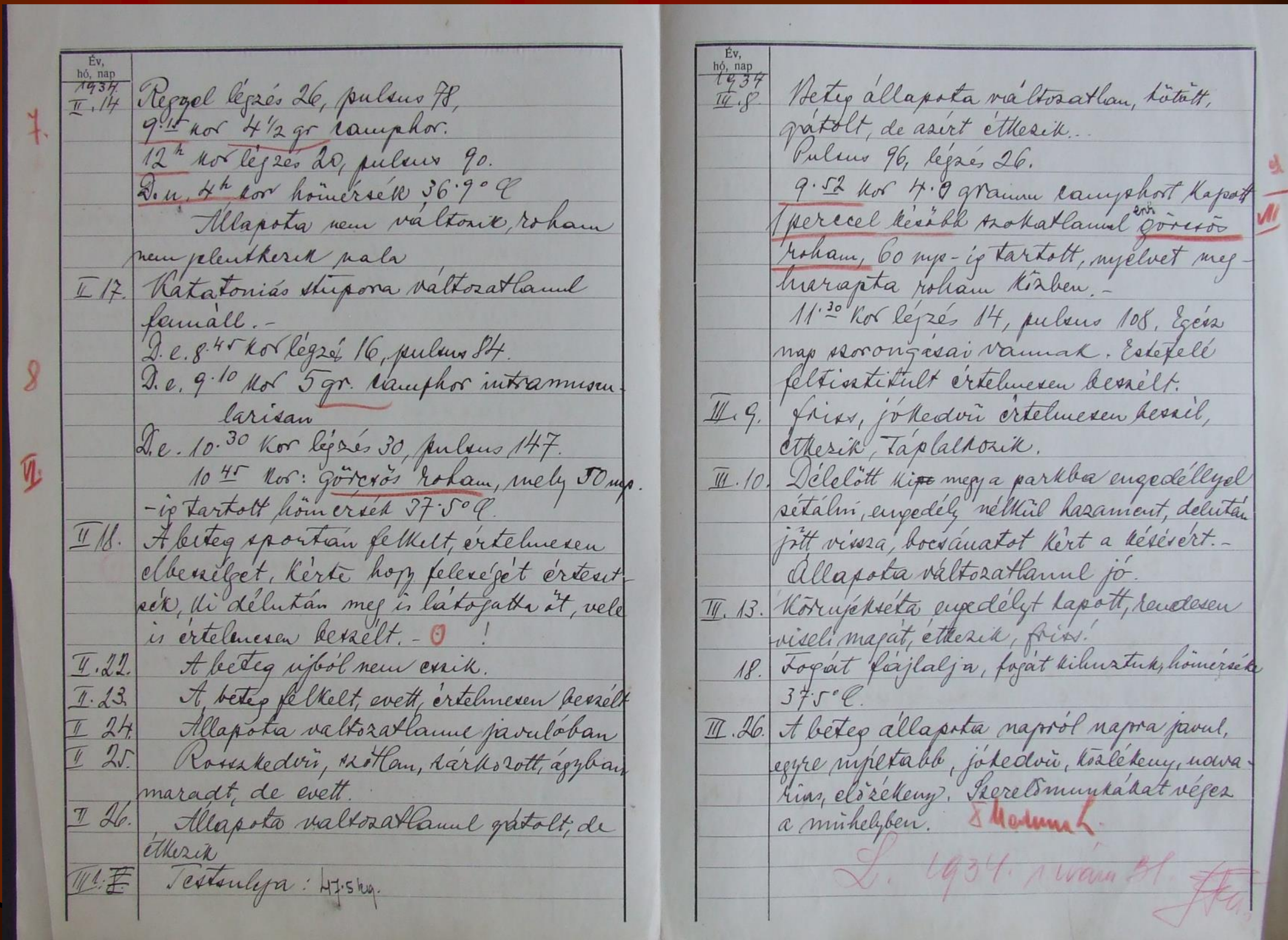


Budapest-lipótmezei magyar kir. állami elme- és ideggyógyintézet.

IV. számu betétív Litheráti Zoltán kórrajzához.

"He asked for some food, spoke and has been more active. He would get up and stand by the radiator the whole day... he inquired how long he had been here..."

End of the first course
(8th March)



Év, hó, nap	
1934 II. 14	Reggel légzés 26, pulsus 78, 9.15 kor 4 ½ gr camphor. 12h kor légzés 20, pulsus 90. D. u. 4h kor hőmérsék 36.9° C. Állapota nem változik, roham nem jelentkezik [illegible]
II. 17.	Katatoniás stuporja változatlanul fennáll.– D. e. 8.45 kor légzés 16, pulsus 84. D. e. 9.10 kor 5 gr. camphor intramuscularisan. D. e. 10.30 kor légzés 30, pulsus 147. 10.45 kor: görcsös roham, mely 50 mp.-ig tartott hőmérsék 37.5° C.
II. 18.	A beteg spontán felkelt, értelmesen elbeszélget, kérte hogy feleségét értesítsék, ki délután meg is látogatta őt, vele is értelmesen beszélt. – !
II. 22.	A beteg újból nem eszik.
II. 23.	A beteg felkelt, evett, értelmesen beszélt
II 24.	Állapota változatlanul javulóban
II 25.	Rosszkedvű, szótlan, zárkózott, ágyban maradt, de evett.
II 26.	Állapota változatlanul gátolt, de étkezik
III. 1.	Testsúlya: 47.5 kg.
1934 III. 8.	Beteg állapota változatlan, kötött, gátolt, de azért étkezik.. Pulsus 96, légzés 26. 9.52 kor 4.0 gramm camphort kapott 1 perccel később szokatlanul erős görcsös roham, 60 mp.-ig tartott, nyelvét megharapta roham közben.– 11.30 kor légzés 14, pulsus 108. Egész nap szorongásai vannak. Estefelé feltisztitult értelmesen beszélt.
III. 9.	friss, jókedvű értelmesen beszél, étkezik, táplálkozik.
III. 10.	Délelőtt ment a parkba engedéllyel sétálni, engedély nélkül hazament, délután jött vissza, bocsánatot kért a késésért.– Állapota változatlanul jó.
III. 13.	Környékséta engedélyt kapott, rendesen viseli magát, étkezik, friss.
18.	Fogát fájlalja, fogát kihúztuk, hőmérsék 37.5° C.
III. 26.	A beteg állapota napról napra javul, egyre nyíltabb, jókedvű, közlékeny, udvarias, előzékeny. Szeretőmunkákat végez a műhelyben.

Margin notes (red): 7., 8, VI, VII

L. 1934. április 31. [signature]

The end of the first course

9.	March 8 9:52 a.m.	4 g camphor i.m.	9:53 a.m.: unusually strong, 60-second seizure with a tongue bite	"*was distressed the whole day; by evening his mind became clear, he spoke intelligently*"
	March 10	"In the morning he went to walk in the park with permission; he went home without permission, came back in the afternoon, and apologized for being late."		
	March 26	"*His condition has been improving day by day; he is becoming outgoing, cheerful, communicative, polite... He does repairs in the workshop*." (Dr. Meduna)		
	April 3	"*Under supervision, he does repair jobs in the workshop... he can move freely in the institute, goes out to the city twice a week and always returns in time... he parries questions concerning his illness with an 'I don't know'...*"		

Outcome (Meduna's autobiography)

- In Meduna's account, Zoltán's illness was in remission until 1939 as a result of convulsive therapy

Convulsive treatment courses according to Zoltán's chart

- First course of convulsive treatment: from 01/23/34 until 03/08/34 (camphor)-9 inj.
- Second course: from 07/12/34 until 08/28/34 (camphor and cardiazol)-7 inj.
- Third course: from 10/18/34 until 10/31/34 (cardiazol)-3 inj.
- Fourth course: from 02/22/35 until 04/02/35 (camphor)-11 inj.
- Fifth course: from 08/16/35 until 09/27/35 (cardiazol)-6 inj.
- Sixth course: from 01/23/37 until 01/30/37 (cardiazol)-3 inj.

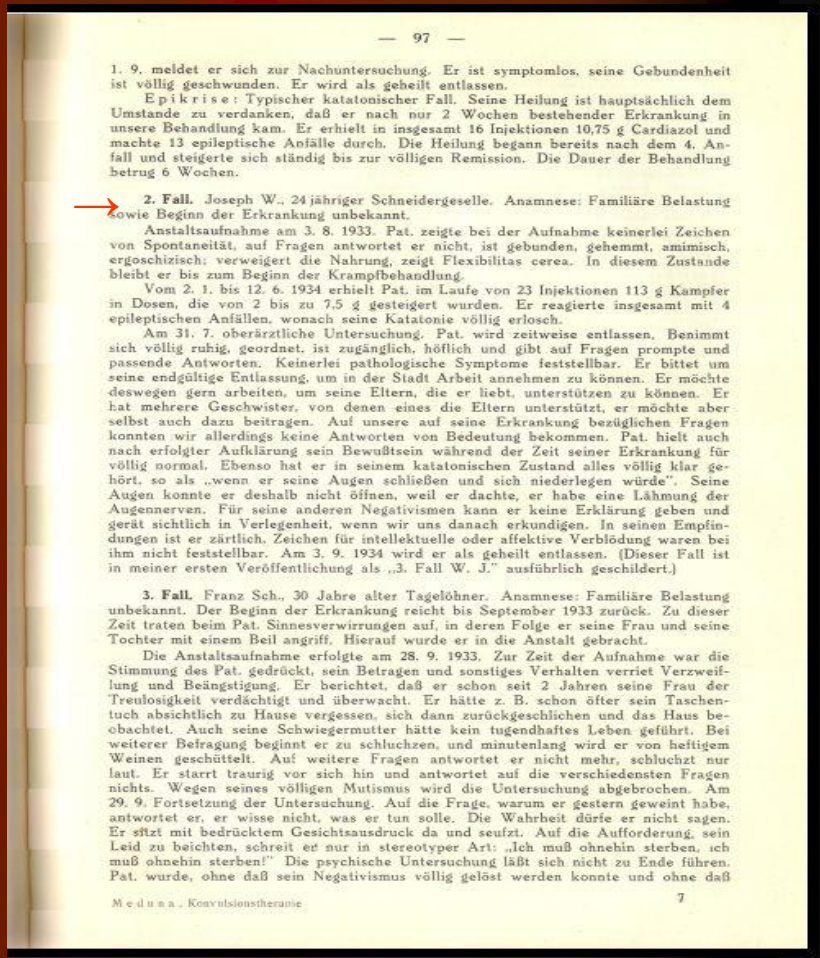
Course of Zoltan's illness until his death

- Between 1940 and 1945, there was a further gradual decline in both his mental and physical condition, though tube feeding was no longer required nor did severe stupor develop. Zoltán was never discharged from the institution after his admission in 1930.
- The cause of his death remains unclear. No autopsy information is available. Starvation and the poor hygienic conditions caused by the siege of Budapest might explain his death, although there were no symptoms of infection recorded in his medical history. On January 1, 1945 his weight was only 43 kg.

If Zoltán was the 5th then who was the real first?

Monograph: 2nd case

- W József
- Treatment started: January 2
- Catatonic schizophrenia
- improved



— 97 —

1. 9. meldet er sich zur Nachuntersuchung. Er ist symptomlos, seine Gebundenheit ist völlig geschwunden. Er wird als geheilt entlassen.

Epikrise: Typischer katatonischer Fall. Seine Heilung ist hauptsächlich dem Umstande zu verdanken, daß er nach nur 2 Wochen bestehender Erkrankung in unsere Behandlung kam. Er erhielt in insgesamt 16 Injektionen 10,75 g Cardiazol und machte 13 epileptische Anfälle durch. Die Heilung begann bereits nach dem 4. Anfall und steigerte sich ständig bis zur völligen Remission. Die Dauer der Behandlung betrug 6 Wochen.

2. Fall. Joseph W., 24 jähriger Schneidergeselle. Anamnese: Familiäre Belastung sowie Beginn der Erkrankung unbekannt.

Anstaltsaufnahme am 3. 8. 1933. Pat. zeigte bei der Aufnahme keinerlei Zeichen von Spontaneität, auf Fragen antwortet er nicht, ist gebunden, gehemmt, amimisch, ergoschizisch; verweigert die Nahrung, zeigt Flexibilitas cerea. In diesem Zustande bleibt er bis zum Beginn der Krampfbehandlung.

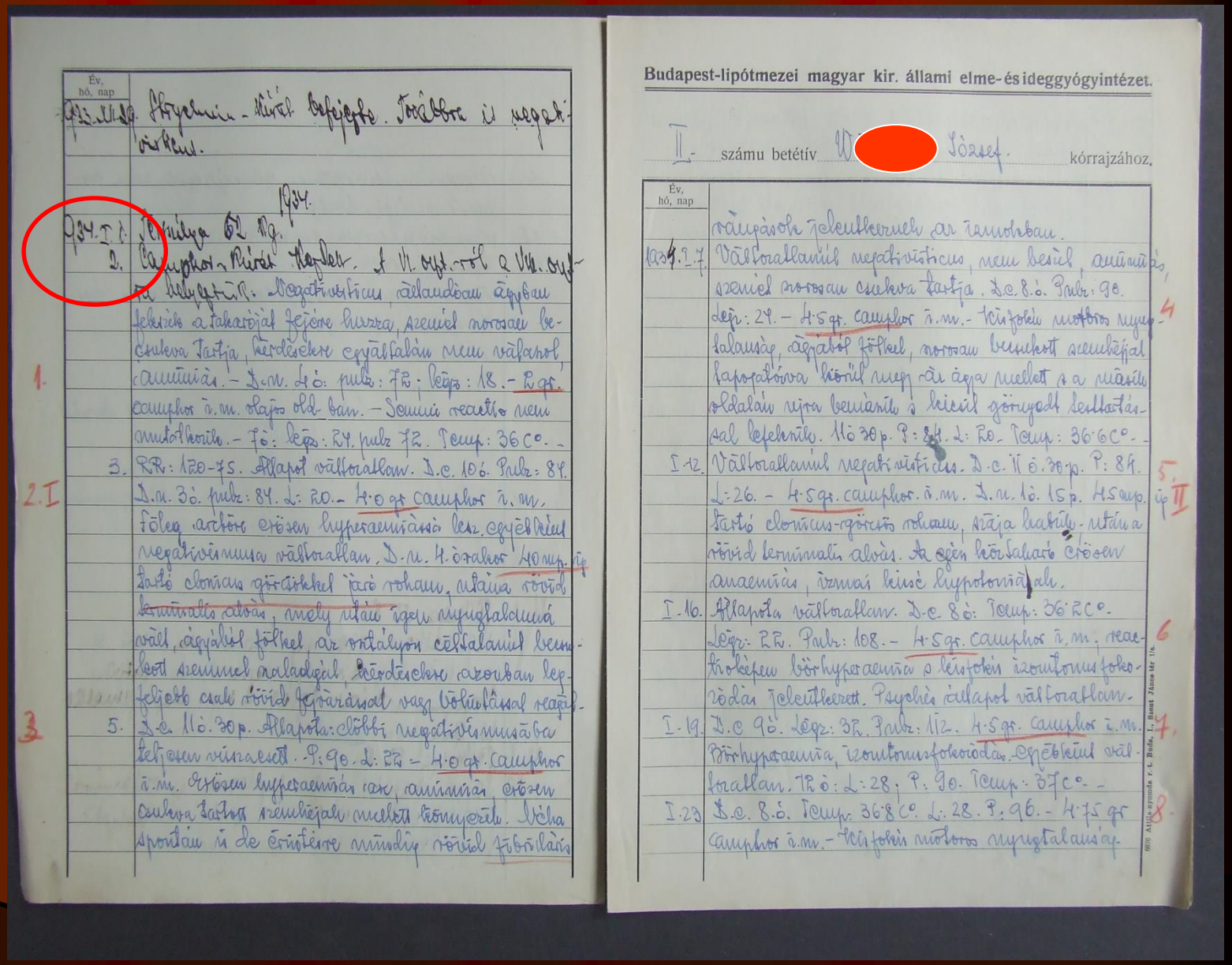
Vom 2. 1. bis 12. 6. 1934 erhielt Pat. im Laufe von 23 Injektionen 113 g Kampfer in Dosen, die von 2 bis zu 7,5 g gesteigert wurden. Er reagierte insgesamt mit 4 epileptischen Anfällen, wonach seine Katatonie völlig erlosch.

Am 31. 7. oberärztliche Untersuchung. Pat. wird zeitweise entlassen. Benimmt sich völlig ruhig, geordnet, ist zugänglich, höflich und gibt auf Fragen prompte und passende Antworten. Keinerlei pathologische Symptome feststellbar. Er bittet um seine endgültige Entlassung, um in der Stadt Arbeit annehmen zu können. Er möchte deswegen gern arbeiten, um seine Eltern, die er liebt, unterstützen zu können. Er hat mehrere Geschwister, von denen eines die Eltern unterstützt, er möchte aber selbst auch dazu beitragen. Auf unsere auf seine Erkrankung bezüglichen Fragen konnten wir allerdings keine Antworten von Bedeutung bekommen. Pat. hielt auch nach erfolgter Aufklärung sein Bewußtsein während der Zeit seiner Erkrankung für völlig normal. Ebenso hat er in seinem katatonischen Zustand alles völlig klar gehört, so als „wenn er seine Augen schließen und sich niederlegen würde". Seine Augen konnte er deshalb nicht öffnen, weil er dachte, er habe eine Lähmung der Augennerven. Für seine anderen Negativismen kann er keine Erklärung geben und gerät sichtlich in Verlegenheit, wenn wir uns danach erkundigen. In seinen Empfindungen ist er zärtlich. Zeichen für intellektuelle oder affektive Verblödung waren bei ihm nicht feststellbar. Am 3. 9. 1934 wird er als geheilt entlassen. (Dieser Fall ist in meiner ersten Veröffentlichung als „3. Fall W. J." ausführlich geschildert.)

3. Fall. Franz Sch., 30 Jahre alter Tagelöhner. Anamnese: Familiäre Belastung unbekannt. Der Beginn der Erkrankung reicht bis September 1933 zurück. Zu dieser Zeit traten beim Pat. Sinnesverwirrungen auf, in deren Folge er seine Frau und seine Tochter mit einem Beil angriff. Hierauf wurde er in die Anstalt gebracht.

Die Anstaltsaufnahme erfolgte am 28. 9. 1933. Zur Zeit der Aufnahme war die Stimmung des Pat. gedrückt, sein Betragen und sonstiges Verhalten verriet Verzweiflung und Beängstigung. Er berichtet, daß er schon seit 2 Jahren seine Frau der Treulosigkeit verdächtigt und überwacht. Er hätte z. B. schon öfter sein Taschentuch absichtlich zu Hause vergessen, sich dann zurückgeschlichen und das Haus beobachtet. Auch seine Schwiegermutter hätte kein tugendhaftes Leben geführt. Bei weiterer Befragung beginnt er zu schluchzen, und minutenlang wird er von heftigem Weinen geschüttelt. Auf weitere Fragen antwortet er nicht mehr, schluchzt nur laut. Er starrt traurig vor sich hin und antwortet auf die verschiedensten Fragen nichts. Wegen seines völligen Mutismus wird die Untersuchung abgebrochen. Am 29. 9. Fortsetzung der Untersuchung. Auf die Frage, warum er gestern geweint habe, antwortet er, er wisse nicht, was er tun solle. Die Wahrheit dürfe er nicht sagen. Er sitzt mit bedrücktem Gesichtsausdruck da und seufzt. Auf die Aufforderung, sein Leid zu beichten, schreit er nur in stereotyper Art: „Ich muß ohnehin sterben, ich muß ohnehin sterben!" Die psychische Untersuchung läßt sich nicht zu Ende führen. Pat. wurde, ohne daß sein Negativismus völlig gelöst werden konnte und ohne daß

W József



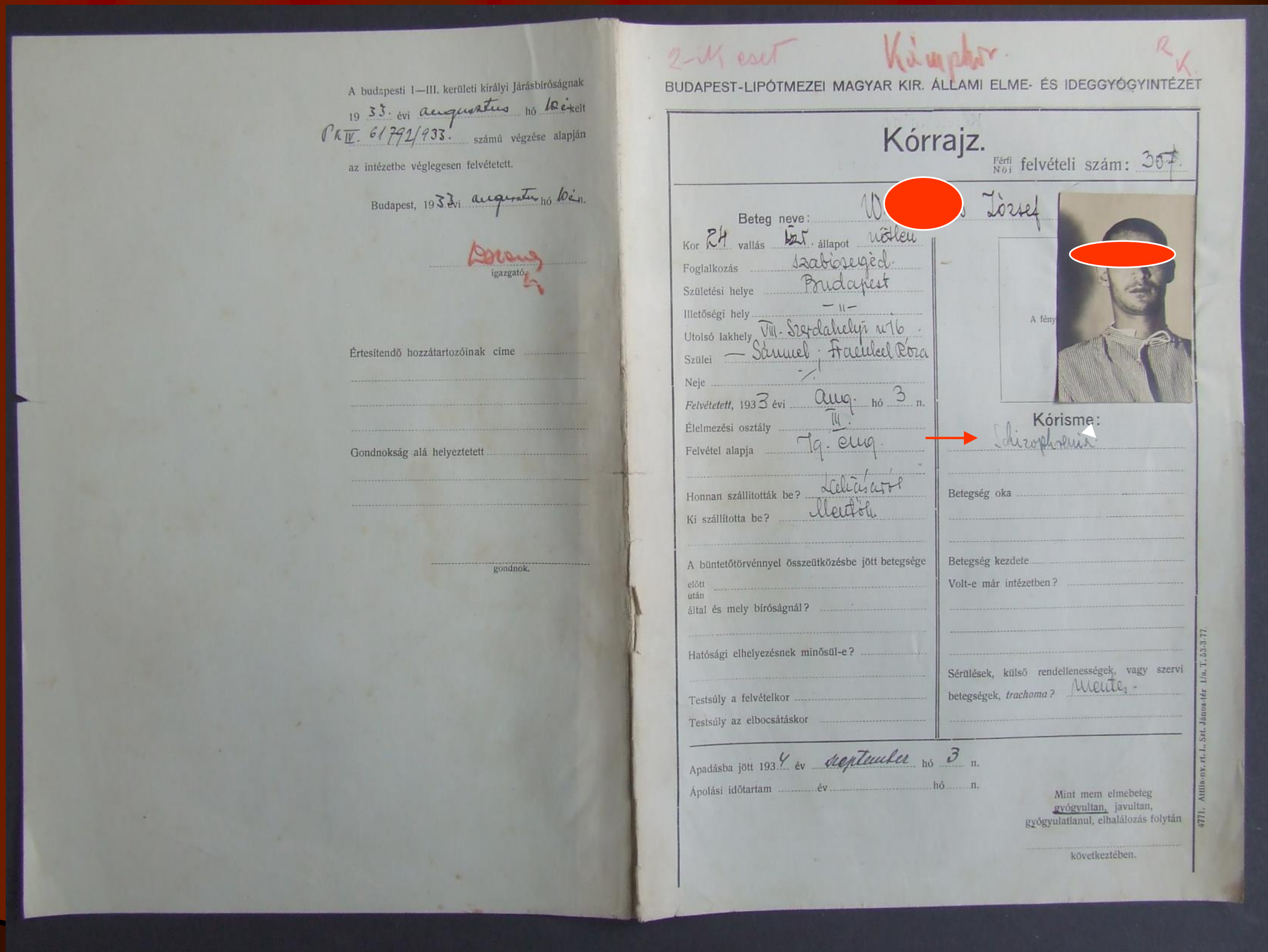
Budapest-lipótmezei magyar kir. állami elme- és ideggyógyintézet.

II. számu betétív W [redacted] József kórrajzához.

W József – symptoms at admission, course of illness in the Institute

- When admitted, he did not respond to the questions he was asked. It took repeated requests to make him oblige, and even then, stopped the sequence of actions on several occasions. On other times, gave the examiner an imploring look, opened his mouth to say something but did not eventually say a word. When was made to sit down, buried his face in his hands, cried without tears, then looked around with a beseeching look on his hands face, with his hands clasped. Upon leaving the room, began to cry, leaning against the door post.
- Later developed flexibilitas cerea, did not respond to stimuli. In the remaining part of the year, defecated and urinated on himself from time to time. Had to be fed, then required artificial nutrition.

W József – Dg.: Schizophrenia



A budapesti I—III. kerületi királyi Járásbíróságnak 19 33. évi augusztus hó 10-én kelt PK IV. 61792/933. számú végzése alapján az intézetbe véglegesen felvétetett.

Budapest, 1933. évi augusztus hó 10-én.

igazgató.

Értesítendő hozzátartozóinak címe

Gondnokság alá helyeztetett

gondnok.

2-ik eset Kámpfer. R.K.

BUDAPEST-LIPÓTMEZEI MAGYAR KIR. ÁLLAMI ELME- ÉS IDEGGYÓGYINTÉZET

Kórrajz.

Férfi / Női felvételi szám: 307.

Beteg neve: W[redacted]s József

Kor 24 vallás izr. állapot nőtlen

Foglalkozás szabósegéd

Születési helye Budapest

Illetőségi hely —"—

Utolsó lakhely VIII. Szerdahelyi u. 16.

Szülei — Sámuel ; Frankel Róza

Neje —/—

Felvétetett, 1933 évi aug. hó 3 n.

Élelmezési osztály III.

Felvétel alapja Ig. eng.

Honnan szállították be? Lakásáról

Ki szállította be? Mentők

A büntetőtörvénnyel összeütközésbe jött betegsége előtt / után

által és mely bíróságnál?

Hatósági elhelyezésnek minősül-e?

Testsúly a felvételkor

Testsúly az elbocsátáskor

A fény...

Kórisme: Schizophrenia

Betegség oka

Betegség kezdete

Volt-e már intézetben?

Sérülések, külső rendellenességek, vagy szervi betegségek, *trachoma?* Mentes. -

Apadásba jött 1934 év szeptember hó 3 n.

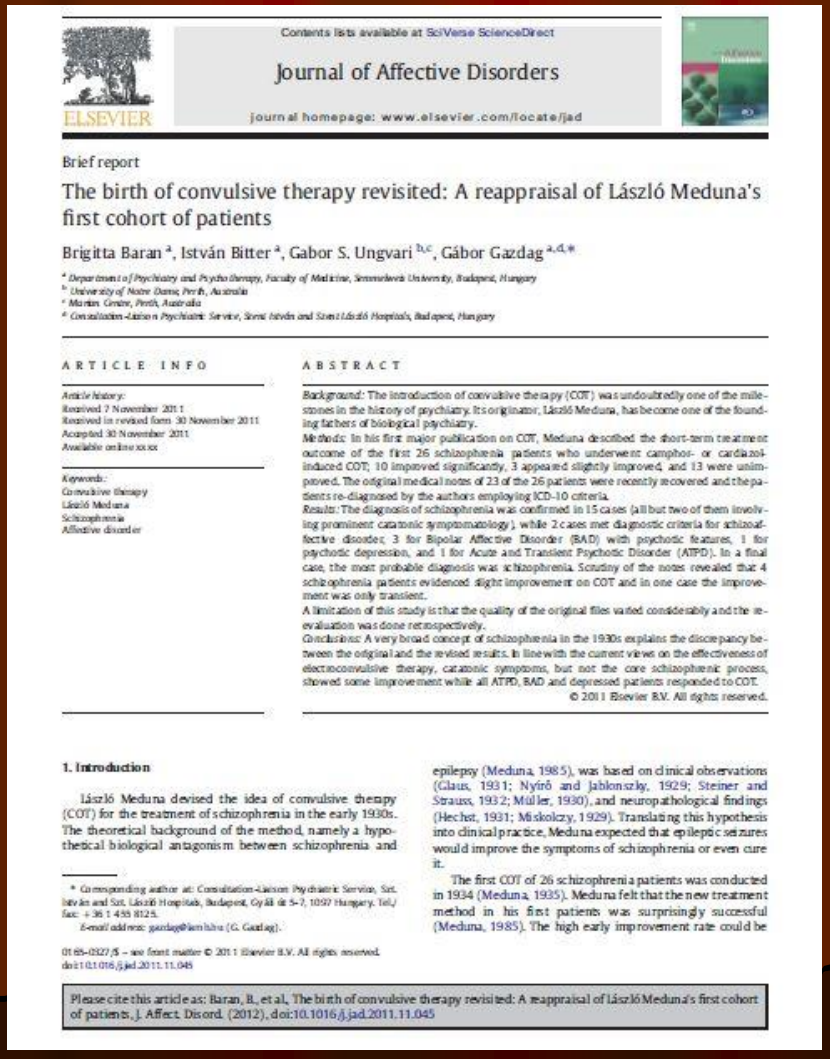
Ápolási időtartam ...... év ...... hó ...... n.

Mint mem elmebeteg gyógyultan, javultan, gyógyulatlanul, elhalálozás folytán

következtében.

4771. Attila-ny. rt. I., Szt. János-tér 1/a. T. 53-3-17

Doubts acording to the accuracy of the diagnoses



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Brief report

The birth of convulsive therapy revisited: A reappraisal of László Meduna's first cohort of patients

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ABSTRACT

Background: The introduction of convulsive therapy (COT) was undoubtedly one of the milestones in the history of psychiatry. Its originator, László Meduna, has become one of the founding fathers of biological psychiatry.

Methods: In his first major publication on COT, Meduna described the short-term treatment outcome of the first 26 schizophrenia patients who underwent camphol- or cardiazol-induced COT; 10 improved significantly, 3 appeared slightly improved, and 13 were unimproved. The original medical notes of 23 of the 26 patients were recently recovered and the patients re-diagnosed by the authors employing ICD-10 criteria.

Results: The diagnosis of schizophrenia was confirmed in 15 cases (all but two of them involving prominent catatonic symptomatology), while 2 cases met diagnostic criteria for schizoaffective disorder, 3 for Bipolar Affective Disorder (BAD) with psychotic features, 1 for psychotic depression, and 1 for Acute and Transient Psychotic Disorder (ATPD). In a final case, the most probable diagnosis was schizophrenia. Scrutiny of the notes revealed that 4 schizophrenia patients evidenced slight improvement on COT and in one case the improvement was only transient.

A limitation of this study is that the quality of the original files varied considerably and the re-evaluation was done retrospectively.

Conclusions: A very broad concept of schizophrenia in the 1930s explains the discrepancy between the original and the revised results. In line with the current views on the effectiveness of electroconvulsive therapy, catatonic symptoms, but not the core schizophrenic process, showed some improvement while all ATPD, BAD and depressed patients responded to COT.

1. Introduction

László Meduna devised the idea of convulsive therapy (COT) for the treatment of schizophrenia in the early 1930s. The theoretical background of the method, namely a hypothetical biological antagonism between schizophrenia and epilepsy (Meduna, 1985), was based on clinical observations (Claus, 1931; Nyírő and Jablonszky, 1929; Steiner and Strauss, 1932; Müller, 1930), and neuropathological findings (Hechst, 1931; Miskolczy, 1929). Translating this hypothesis into clinical practice, Meduna expected that epileptic seizures would improve the symptoms of schizophrenia or even cure it.

The first COT of 26 schizophrenia patients was conducted in 1934 (Meduna, 1935). Meduna felt that the new treatment method in his first patients was surprisingly successful (Meduna, 1985). The high early improvement rate could be

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- Reappraisal of the patients (23 from the 26 patients)
- Meduna, L., 1935. Versuche über die biologische Beeinflussung des Ablaufes der Schizophrenie. Zeitschrift für die gesamte Neurologie und Psychiatrie 152, 235–262.
- 3 researcher independently
- According to ICD-10 criteria
- Reevaluation of improvement

Results

Table 1
Diagnoses and outcome ratings by Meduna and the re-assessment.

No. of cases[a]		Number of prior hospitalizations	Diagnoses from the original records	ICD-10 diagnoses	Outcome judged by Meduna	Outcome judged by the raters
1st case	LZ, 33 years	–	Catatonic schizophrenia	Catatonic schizophrenia	Improved	Improved
2nd case	HGY, 21 years	–	Schizophrenia	Schizoaffective psychosis	Remitted	Remitted
3 rd case	WJ, 24 years	–	Schizophrenia	Catatonic schizophrenia	Remitted	Remitted
4th case	BI, 21 years	–	Schizophrenia	ATPD	Remitted	Remitted
5th case	LP, 24 years	–	Schizophrenia paranoid type	Major depression with psychotic features	Remitted	Remitted
6th case	Dr. PSt,				Remitted	
7th case	TJ, 20 years	3	Catatonic schizophrenia	Catatonic schizophrenia	Transient improvement	Unchanged
8th case	KJ, 15 years	–	Schizophrenia	BAD-manic phase	Improved	Remitted
9th case	WJ Jr., 23 years	–	Schizophrenia paranoid type	BAD-mixed phase	Improved	Improved
10th case	SzI, 19 years	–	Schizophrenia	BAD-mixed phase	Improved	Only transient improvement
11th case	TJ, 36 years	–	Schizophrenia	Catatonic schizophrenia	Unchanged	Unchanged
12th case	SzB, 22 years	3	Schizophrenia	Schizophrenia paranoid type	Unchanged	Unchanged
13th case	SI, 28 years	1	Schizophrenia	Catatonic schizophrenia	Unchanged	Unchanged
14th case	NP, 24 years	–	Catatonic schizophrenia	Catatonic schizophrenia	Unchanged	Unchanged
15th case	KA, 18 years	1	Schizophrenia	Catatonic schizophrenia	Unchanged	Unchanged
16th case	HV, 31 years	1	Schizophrenia	Catatonic schizophrenia	Unchanged	Unchanged
17th case	BI, 19 years	–	Schizophrenia	Catatonic schizophrenia	Unchanged	Unchanged
18th case	NO, 21 years				Transient improvement then relapse	
19th case	RE, 18 years	–	Schizophrenia	Catatonic schizophrenia	Unchanged	Unchanged
20th case	KF, 40 years	1	BAD	Schizoaffective psychosis	Unchanged	Unchanged
21st case	KD, 19 years	1	Schizophrenia	Schizophrenia paranoid type	Unchanged	Unchanged
22nd case	PI, 30 years	–	Catatonic schizophrenia	Catatonic schizophrenia	Unchanged	Improved
23 rd case	FO, 23 years	–	Schizophrenia paranoid type	Catatonic schizophrenia	1 month full remission then relapse	Only transient improvement
24th case	CsL, 19 years	–	Schizophrenia	Schizophrenia doubtful	Remitted	No data
25th case	KJ, 22 years	–	Schizophrenia	Catatonic schizophrenia	Improved	Improved
26th case	KN, 16 years				Improved	

ATPD = Acute and Transient Psychotic Disorder.
[a] Cases are numbered as in Meduna's publication (Meduna, 1935). Files of cases 6th, 18th and 26th were not found.

Improvement rates according to ICD-10 diagnoses

Table 2
Rates of improvement according to major ICD-10 diagnostic groups.

	Total number of patients	No. of improved/ remitted patients	%
Schizophrenia	15	4	27%
Affective disorder	6	5	83%
ATPD	1	1	100%
Uncertain psychosis	1	?	
Total	23	10	43%

ATPD = Acute and Transient Psychotic Disorder.

Who should have been the first treated patient?

- Treatments initiated before L Zoltán's:
  - 02/01/1934: 6 patients (1 with oligophrenia+5 with schizophrenia)
  - 03/01/1934: 1 patient (with schizophrenia)
  - 07/01/1934: 1 patient (with schizophrenia)
  - 19/01/1934: 1 patient (with schizophrenia)
  - 23/01/1934: 2 patients (L Zoltán and another patient with Propf schizophrenia)

Treatments initiated on January 2

- Nándor F – oligophrenia
- Gyula H – schizophrenia (improved Feb 11)
- István P – schizophrenia
- Elemér E – schizoprenia
- József T – schizophrenia
- József W – schizophrenia (improved March 2)

ORIGINAL STUDY

László Meduna's Pilot Studies With Camphor Inductions of Seizures

The First 11 Patients

Gábor Gazdag, MD, PhD, István Bitter, MD, PhD, DSc,† Gabor S. Ungvari, MD, PhD,‡ Brigitta Baran, MD,† and Max Fink, MD§*

Summary: In his autobiography, László Meduna described the first session of convulsive therapy using intramuscular camphor as occuring on January 23, 1934 at Royal National Hungarian Institute of Psychiatric and Neurology at Budapest-Lipótmező in Hungary. Unearthed records of the patients treated at this institution reveal that Meduna's dose-finding experiments began on January 2, 1934. The symptomatology and history of illness, diagnosis, socio-demographic data, the seizure characteristics, and immediate and long term outcomes of the first 11 patients are described. These first trials elicited seizures in less than half the injections. Seizures of various durations (including missed seizures) and double (tardive) seizures were recorded. Mutism, refusal to eat requiring tube feeding, and other signs of catatonia dominated the psychopathology of 7 of the first 11 patients. Two improved sufficiently to be discharged from the hospital and third patient became fit for occupational therapy. These records exhibit the meticolous systematic nature of the first human trials with induced seizures and the fortuitous nature of the first human trials with induced seizures and the fortuitous nature in patient selection of catatonic patients – an illness that is most responsive to induced seizures.

Key Words: László Meduna, electroconvulsive therapy, history of electroconvulsive therapy

(*J ECT* 2009;25: 3–11)

In his autobiography, László Meduna describes the first seizure that he induced in a patient with the use of intramuscular camphor as occurring on the morning of January 23, 1934 at the Royal National Hungarian Institute of Psychiatry and Neurology at Budapest-Lipótmező in Hungary.[1] Meduna wrote his autobiography in the mid 1950s after he had migrated to the United States in 1939. In his reminiscences, Meduna described Zoltán L. (Z.L.) as the first patient who received camphor-induced convulsive therapy. An opportunity to examine the records of the patients treated at Lipótmező found that Meduna's first experiments with camphor inductions of seizures had begun on January 2, 1934 and that Z.L. was the 10th patient in whom seizures had been induced. The availability of the case notes permits a more detailed picture of Meduna's first experiences in selecting patients and developing a dosage schedule.[2]

Meduna's hypothesis was developed on neuropathological grounds. Autopsy studies had shown that patients with schizophrenia had a paucity of brain glia, whereas patients with epilepsy had a surfeit.[3] Clinical observations supported an image of a clinical antagonism between schizophrenia and epilepsy because few seizures occurred in patients with schizophrenia and few patients with epilepsy exhibited the schizophrenic form of psychosis.[4,5] Meduna chose camphor as a seizure-inducing agent based on animal experiments with various chemical agents.[6]

MEDUNA'S REPORTS ABOUT THE RESULTS OF THE CONVULSIVE TREATMENTS

In 1935, Meduna published his first clinical experience with 26 patients.[6] Remission of illness was reported in 10 of the patients. The mean dose of camphor was 5.6 g. He administered 128 injections eliciting 62 seizures in the 10 patients who showed improvement and 229 injections with 25 seizures in the 13 patients who had shown no benefit. He found camphor to be an inefficient agent for seizure induction. Finally, Meduna concluded that "patients with short duration of illness benefited most from convulsive therapy."[6]

In 1937, Meduna published his monograph, *Die Konvulsionstherapie der Schizophrenie*, describing an experience with 110 patients and offering vignettes of the 53 who were successfully treated.[7] The vignettes include the dates of the treatment sessions, permitting an insight into his actual results. The 53 cases include Z.L. and 2 patients whose treatment commenced earlier on January 2, 1934.

METHOD

We reviewed the case notes of patients in whom Meduna induced seizures using intramuscular injections of camphor and were discharged from the Royal Hungarian Institute of Psychiatry and Neurology at Lipótmező or died between 1933 and 1945. These records were selected from the archives of the National Institute of Neurology and Psychiatry. In this report, we summarize the case notes of patients whose treatments preceded or were concurrent with those of Z.L.

The demographic characteristics and case histories of the 11 patients are shown in Table 1. Details of the treatments of each patient are offered in Table 2, and their posttreatment follow-up information is summarized in Table 3.

OBSERVATIONS

The first attempt to induce a seizure was recorded on January 2, 1934 when Meduna injected 2 g of camphor intramuscularly to

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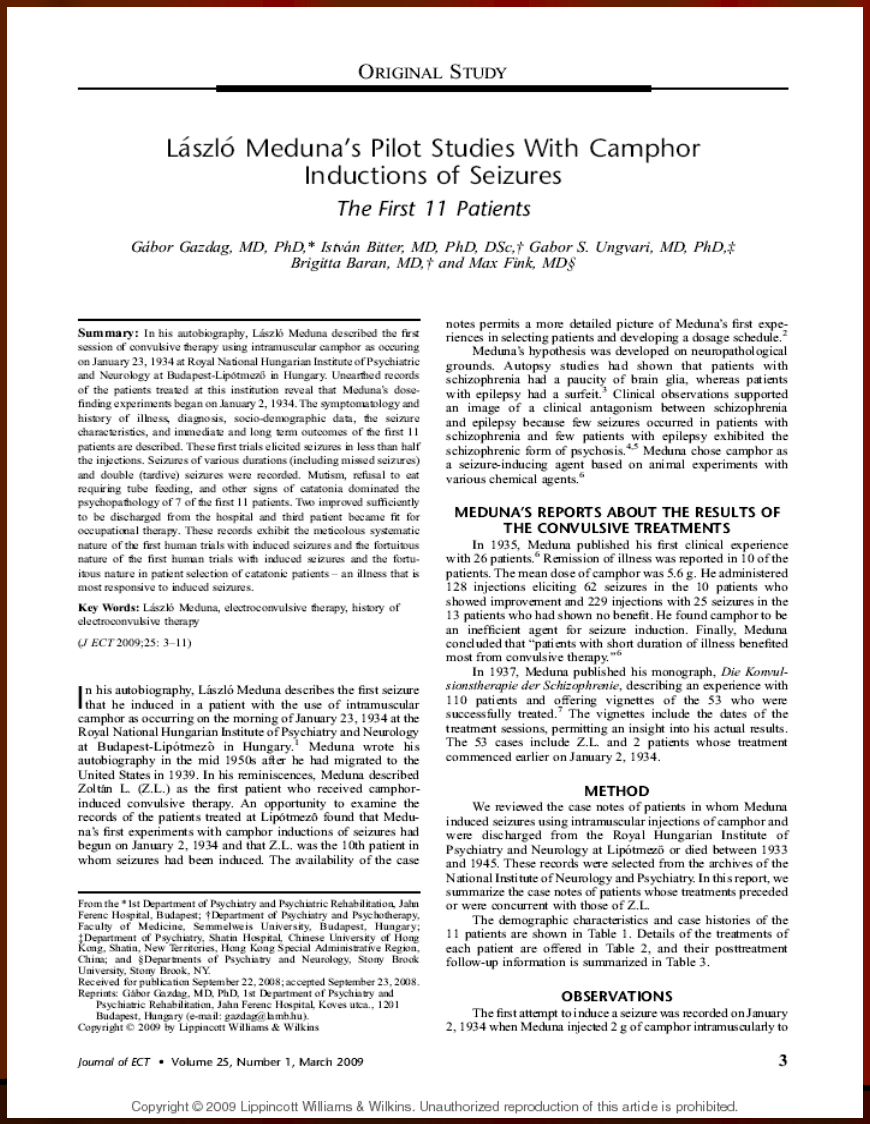
Review of the first 11 cases (2009)



ORIGINAL STUDY

László Meduna's Pilot Studies With Camphor Inductions of Seizures

The First 11 Patients

Gábor Gazdag, MD, PhD, István Bitter, MD, PhD, DSc,† Gabor S. Ungvari, MD, PhD,‡ Brigitta Baran, MD,† and Max Fink, MD§*

Summary: In his autobiography, László Meduna described the first session of convulsive therapy using intramuscular camphor as occuring on January 23, 1934 at Royal National Hungarian Institute of Psychiatric and Neurology at Budapest-Lipótmezõ in Hungary. Unearthed records of the patients treated at this institution reveal that Meduna's dose-finding experiments began on January 2, 1934. The symptomatology and history of illness, diagnosis, socio-demographic data, the seizure characteristics, and immediate and long term outcomes of the first 11 patients are described. These first trials elicited seizures in less than half the injections. Seizures of various durations (including missed seizures) and double (tardive) seizures were recorded. Mutism, refusal to eat requiring tube feeding, and other signs of catatonia dominated the psychopathology of 7 of the first 11 patients. Two improved sufficiently to be discharged from the hospital and third patient became fit for occupational therapy. These records exhibit the meticolous systematic nature of the first human trials with induced seizures and the fortuitous nature of the first human trials with induced seizures and the fortuitous nature in patient selection of catatonic patients – an illness that is most responsive to induced seizures.

Key Words: László Meduna, electroconvulsive therapy, history of electroconvulsive therapy

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In his autobiography, László Meduna describes the first seizure that he induced in a patient with the use of intramuscular camphor as occurring on the morning of January 23, 1934 at the Royal National Hungarian Institute of Psychiatry and Neurology at Budapest-Lipótmezõ in Hungary.[1] Meduna wrote his autobiography in the mid 1950s after he had migrated to the United States in 1939. In his reminiscences, Meduna described Zoltán L. (Z.L.) as the first patient who received camphor-induced convulsive therapy. An opportunity to examine the records of the patients treated at Lipótmezõ found that Meduna's first experiments with camphor inductions of seizures had begun on January 2, 1934 and that Z.L. was the 10th patient in whom seizures had been induced. The availability of the case notes permits a more detailed picture of Meduna's first experiences in selecting patients and developing a dosage schedule.[2]

Meduna's hypothesis was developed on neuropathological grounds. Autopsy studies had shown that patients with schizophrenia had a paucity of brain glia, whereas patients with epilepsy had a surfeit.[3] Clinical observations supported an image of a clinical antagonism between schizophrenia and epilepsy because few seizures occurred in patients with schizophrenia and few patients with epilepsy exhibited the schizophrenic firm of psychosis.[4,5] Meduna chose camphor as a seizure-inducing agent based on animal experiments with various chemical agents.[6]

MEDUNA'S REPORTS ABOUT THE RESULTS OF THE CONVULSIVE TREATMENTS

In 1935, Meduna published his first clinical experience with 26 patients.[6] Remission of illness was reported in 10 of the patients. The mean dose of camphor was 5.6 g. He administered 128 injections eliciting 62 seizures in the 10 patients who showed improvement and 229 injections with 25 seizures in the 13 patients who had shown no benefit. He found camphor to be an inefficient agent for seizure induction. Finally, Meduna concluded that "patients with short duration of illness benefited most from convulsive therapy."[6]

In 1937, Meduna published his monograph, *Die Komvulsionstherapie der Schizophrenie*, describing an experience with 110 patients and offering vignettes of the 53 who were successfully treated.[7] The vignettes include the dates of the treatment sessions, permitting an insight into his actual results. The 53 cases include Z.L. and 2 patients whose treatment commenced earlier on January 2, 1934.

METHOD

We reviewed the case notes of patients in whom Meduna induced seizures using intramuscular injections of camphor and were discharged from the Royal Hungarian Institute of Psychiatry and Neurology at Lipótmezõ or died between 1933 and 1945. These records were selected from the archives of the National Institute of Neurology and Psychiatry. In this report, we summarize the case notes of patients whose treatments preceded or were concurrent with those of Z.L.

The demographic characteristics and case histories of the 11 patients are shown in Table 1. Details of the treatments of each patient are offered in Table 2, and their posttreatment follow-up information is summarized in Table 3.

OBSERVATIONS

The first attempt to induce a seizure was recorded on January 2, 1934 when Meduna injected 2 g of camphor intramuscularly to

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- Frequency: 2-3/week
- Induction agent: camphor
- Dose: 2-5 g (4, 8 g)
- Number of injections: 9-32
- Injection/seizure rates: 2/1, 13/1(HGY), 20/4, 32/2!, 23/3, 23/4(WJ), 19/1, 25/3, 2/0, 9/6(LZ), 10/1
- Reevaluation of efficacy:
  - remission: 2+1
  - no improvement: 8

Shared characteristics of the improved patients

- Illness history < 1 év (2) (3/11)
- Indication: tube feeding (3)
- Highest injection/seizure rate (LZ)

Conclusions: introduction of the method

- Theoretical background (antagonism)
- Animal experiments
- Established methodology
- Inclusion criteria (stupor, refusal to eat)
- Titration of seizure inducing dose

Conclusion: question of priority

- First patient uncertain, 6 patients received camphor injection for therapeutic purposes on 02/01/1934
- First improvement (10th February): L. Zoltán

Conclusion – Zoltán's case

- Meduna was unrealistically optimistic about the effectiveness of convulsive therapy. (only transient improvement)
- Lack of more prolonged remission in Zoltán's case may have been due to the relatively few treatment sessions (only 9)
- In one aspect Meduna's therapeutic enthusiasm seemed to have been justified: once convulsive therapy had been started, Zoltán never became severely stuporous in need of tube feeding. (convulsive therapy is the most efficacious treatment for catatonia and stupor of various origins)

Final conclusion

- Convulsive treatment initiated a paradigm shift in the approach to the whole concept of schizophrenia because it successfully challenged the dogmatic view of therapeutic nihilism in the endogenous psychoses.
- The introduction of convulsive therapy initiated biological research in psychiatry and also boosted the morale and professional self-esteem of psychiatrists.

Thank you for your attention!