Movement Disorders
Vol. 25, No. 9, 2010, pp. 1124–1130

Historical Review

Does the Historical Literature on Encephalitis Lethargica Support a Simple (Direct) Relationship with Postencephalitic Parkinsonism?

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Abstract: This article and the subsequent one suggest that the currently accepted view of a simplistic (direct) relationship between encephalitis lethargica (EL) and postencephalitic Parkinsonism (PEP) is based on a incomplete evaluation of the epidemic period literature. In this article we provide a detailed analysis of the literature from the period that demonstrates that Parkinsonism was not initially part of acute EL symptomatology, that PEP was not typically the prevailing type of chronic EL and that oculogyric crises were never part of acute EL symptomatology and not initially associated with PEP. The second paper uses these finding, and also examines the clinical justifications for concluding that all patients with PEP had prior acute episodes of EL, to reevaluate the presumed direct etiologic relationship between EL and PEP. © 2010 Movement Disorder Society

Key words: epidemic encephalitis; von Economo's disease; encephalitic Parkinsonism; oculogyric crises

Postencephalitic Parkinsonism (PEP) is currently perceived as having a very close etiologic relationship with encephalitis lethargica (von Economo's disease; EL), with PEP developing either immediately after the acute phase of EL or at some time (weeks to many years) later. This relationship between EL and PEP was based on the observation that EL patients could have a form of EL, the amyostatic-akinetic form, in which they showed many parkinsonian features, and on the perception that some EL patients seemed to pass seamlessly from the acute phase of EL to PEP.[1] This observation and perception led to the modern view that PEP is the predominant (and perhaps) only permanent sequel to EL.

In this article, we trace historically the development of PEP, the amyostatic-akinetic form of EL, and oculogyric crises (OCs; which are intimately associated with PEP; cf. below) to better understand the perceived relationship among them. The purpose of this analysis is to demonstrate that some of the currently accepted aspects of EL and PEP do not accurately reflect the epidemic period literature. Furthermore, the data detailed here provide critical support for the second article of this pair, which argues that the relationship between EL and PEP is more complex than currently believed, i.e., that the virus that presumably caused EL alone may not be the cause of PEP.

The selection of references that form the bases of these article was unbiased in that we searched our collection of EL literature (over 2500 publications) for

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Potential conflict of interest: Nothing to report.

Received 23 April 2009; Revised 17 July 2009; Accepted 11 December 2009

Published online 14 May 2010 in Wiley InterScience (www.interscience.wiley.com). DOI: 10.1002/mds.22991

TABLE 1. *Historical year-by-year analysis of major reports on EL pertaining to Parkinsonism during acute or chronic phase (1917–1929)*

Source/type of study	Year	Content
von Economo[4,5]	1917	No suggestion in either paper that there are sequelae to EL or described any constellation of symptoms that could be viewed as parkinsonian; in 1929, monograph[1] notes that first EL epidemic "was practically free from sequelae."
Netter[6]	1918	No mention of a parkinsonian type of EL or of any type of sequelae.
Newsholme et al.[7]	1918	Mentioned sequelae but only paresis and contractures; also listed no type of EL that appeared to be related to the basal ganglia.
Buzzard[8]	1918	Described a 59-yr-old patient with EL who was initially "mentally diagnosed" with paralysis agitans as the patient entered the consulting room because of gait and mask-like face; 2 mo later, the patient had greatly recovered, suggesting that the signs of Parkinsonism constituted a transitory phase of EL.
Hall[9]	1918	Remarked that facial expression of an encephalitic patient is, "so suggestive of Parkinson's mask that I thought at first she must have paralysis agitans," but also noted that symptoms disappeared in a few days.
Wilson[10]	1918	Presents seven cases, each ideally representing a type of EL including paralysis agitans type; this patient showed bradykinesia, tremor and a masked face; after 5 wk symptoms markedly decreased in severity.
Bramwell[11]	1920	No mention of parkinsonian or amyostatic type of EL.
Walshe[12]	1920	No mention of parkinsonian or amyostatic type of EL.
Franck (cited in Catola[13])	1920	Parkinsonian forms of EL are very rare, although Catola noted that this is debatable.
Tilney and Howe[14]	1920	Described five cases of EL as "paralysis agitans" type.
Sicard and Paraf[15]	1920	Described five patients suffering from PEP who showed rigidity, bradykinesia, and a shuffling gait; all initially suffered from the somnolent-ophthalmoplegic form of EL and the authors stated that they have never seen PEP develop from the myoclonic form of EL; in commenting on this work, von Economo[1] noted that this article was the first to describe the high frequency of this sequel.
Marie and Levy[16]	1920	Concluded that the Parkinson syndrome is not unique to PD but can occur in other morbid entities and perhaps in time still other parkinsonian entites may emerge.
Achard[17]	1921	"... If the features [in paralysis agitans] are, in effect, immobility, if the muscle structures of the face take part in general hypertonia, the eyes usually are not at all half-closed, but often are on the contrary bulging. Yet in EL, the eyelids are almost always drooping, which is quite the opposite." Achard further commented that he has only seen cases of acute EL and had not seen a case in which PD appeared to be a remote sequel of EL.
Symonds[18]	1921	Listed the major EL types: lethargic, neuralgic, choreiform, psychic, myoclonic, meningitic, and polyneuritic; but later in review mentioned, in relation to sialorrhea, that there was also a parkinsonian type; pertaining specifically to sequelae, noted that most interest has been aroused by involuntary movements.
Parsons et al.[19]	1922	Table 47 in this volume detailed EL sequelae; Parkinsonism is not listed as a separate category in any of the 271 cases of EL that had sequelae 2–18 mo after the acute episode; tremors were reported for 45 patients, but rigidity for only 9; psychic changes were the most frequent sequelae (47 cases). These authors also specifically stated, "Express references to *symptomatic paralysis agitans* do not occur, but mention is made of the mask-like face, stiffness, festinant gait, and other features of the parkinsonian picture." In other words, there were no references relating EL directly to PD, but there were definite reports of parkinsonian symptomatology.
Grossman[20]	1922	Described the sequelae of EL based on 92 American cases followed for 1–3 yr after acute EL; 49 were considered to be "of the paralysis agitans type;" noted, however, "A striking fact was that in some of our patients, in whom during the acute stage of their illness a most intense picture of the paralysis agitans syndrome was noted, there was no evidence pointing to involvement of the basal ganglions when re-examined.
Souques and Mouquin[21]	1922	"We know that these parkinsonian syndromes often present phases of spontaneous amelioration; often, in particular, some weeks after EL one sees a remission rather prolonged from spasms and trembling at the beginning, until the day where, some months or some year much later, the syndrome reappears more intense and more complete."
Drysdale[22]	1922	Questioned whether some of his patients might have developed PD regardless of whether they had EL; raised this issue because uncertain whether these patients had definitely had a previous phase of EL.
British Medical Association Meeting[23]	1923	Buzzard noted that the parkinsonian syndrome is one of the most common sequelae of EL and stated that he saw and reported his first case in 1918 (cf. above); Howell reported that mental changes or alterations in character constituted the most common sequelae, next were those patients with parkinsonian symptoms; noted, however, that the symptomatology was variable, with some patients having only the face affected; was certain that the parkinsonian sequelae of EL would stimulate research in paralysis agitans; Feiling in Riddock et al. presented the most detailed information on PEP of the meeting, noting that PEP may arise from any type of EL either immediately or after a delay of 3 yr or more.
Naville[24]	1923	For Geneva, reported that after 1918 and 1921 EL epidemics, patients with PEP sequelae are half those with psychomotor and mental inertia, and their prognosis for recovery is better.

TABLE 1. *Historical year-by-year analysis of major reports on EL pertaining to Parkinsonism during acute or chronic phase (1917–1929) (Continued)*

Source/type of study	Year	Content
Mott (in preface to Wimmer[25])	1924	"A considerable portion of these cases must pass into the chronic stage, and they will fall into different types according to the location of the pathological process in the central nervous system, the common form having the parkinsonian syndrome."
Wimmer[25]	1924	In "Author's Preface"noted that PEP by this date has become well-known to "medical men."
Cruchet[26]	1925	Had observed the immobile face in some of the earliest cases of the disease in 1917 and that the parkinsonian type of EL was evident in the 1919–1920 EL epidemic; also stated that whereas the parkinsonian type of EL is "pretty often curable," the prognosis of PEP, which appears between 6 mo to more than 2 yr after an apparent cure of EL, does not have a good prognosis.
Hunt and Cornwall[27]	1925	Stated about PEP, "... unlike any of the other sequelae of encephalitis, the parkinsonian syndrome has nothing about it to suggest that it is a recrudescence of the original malady."
Alpers and Patten[28]	1927	Described a 15-yr-old boy who developed "acute encephalitic Parkinsonism" but then fully recovered after 2 yr; noted that this type of disorder is almost always considered a late sequel of EL.
Parsons[29]	1928	Suggested mask-like face in acute EL often produced by lethargy and/or facial paresis, and thus differs from that during PEP.
von Economo[1]	1929; 1931	Clearly described the sequelae of EL, including PEP, and also described a primary type of EL that he called the amyostatic-akinetic type, which had many parkinsonian features.

relevant articles from the epidemic period (1917–1929) and later that presented some information/view on the amyostatic type of EL, EL sequelae, OCs and/or PEP. We emphasize here that our review includes articles published in the three major languages of the time, English, French, and German. Furthermore, to facilitate readability, we have placed much of the supporting literature in tabular form; thus, the reader is free to consult the tables for these data but the article is fully comprehensible without reference to the tables. Finally, in the tables, where we include quotes from non-English articles, we only include the English translation (to conserve space).

HISTORICAL REVIEW OF EL REPORTS PERTAINING TO PARKINSONISM

In two 1980s article on EL/PEP, parkinsonian signs were considered to be important for diagnosing EL.[2,3] However, parkinsonian signs were not part of von Economo's original description of the acute disease[4] and some authors initially considered them to be transitory (Table 1).

Similarly, parkinsonian signs were not considered part of the chronic syndrome until about 3 years after von Economo's 1917 initial description and not firmly associated with chronic EL until about by 1924 (see Wimmer in Table 1). And, during this period there were consistent questions as to the relationship between PEP and idiopathic Parkinsonism (PD), with some clinicians of the time (especially the French clinicians) insisting that PEP and PD were identical (including etiology).[17,30,31]

Additionally, the amyostatic-akinetic form of EL was not a consistent form of EL. Von Economo[1] stated that the amyostatic form of EL was particularly prevalent in some EL epidemics, such as that in London in 1918, and Hamburg in 1919. And, Cruchet[26] indicated that the greatest number of patients with this type of EL was observed between November 1919 and April 1920. We also note in Table 1 a comment by Franck that the parkinsonian form of EL may have been rare, but Franck was not a well-known EL authority.

Lastly, virtually all recent reviews of EL only mention the three types of EL highlighted by von Economo (somnolent-opthalmoplegic, hyperkinetic, and amyostatic-akinetic).[32–34] However, there were actually many more types categorized; we listed 28 types in a previous publication[35] including some that would seem very unrelated to Parkinsonism (e.g., cerebellar, hemiplegic, spinal, polyneuritic, autonomic, tabetic, myelitic, thalamic, and juvenile pseudo-psychopathia). It is

TABLE 2. *Number of cases of PEP and other sequelae of EL*

Year reported	Years included	Region	#PEP (%)	#others	Comment	Reference
1922	Unstated	France	68 (67)	34	Cited in Wimmer 1924	Souques[31]
1922	Unstated	US	49 (53)	43		Grossman[20]
1922	Unstated	US	36 (37)	61		Bing and Staehlin[36]
1922	Unstated	France	39 (41)	55	Cited in Wimmer	Reys[37]
1922	Unstated	England	0 (0)	271	See Table 1	Parsons et al.[19]
1922	1919–1922	US	16 (10)	145	Includes, acute, chronic, and fatal cases	House[38]
1925	Unstated	US	11 (14)	67	Only 27% of acute EL showed sequelae	Neal et al.[39]
1927	1924–1927	Belfast	83 (59)	58	2.5 yr after acute EL	Robb[40]
1928	Unstated	US	586 (89)	75		Ziegler[41]
1928	Unstated	Germany	28 (82)	6		Stern[42]
1928	1919–1925	Newcastle-on-Tyne	6 (10)	54	Major report on the "after-histories" of EL in England; some data may have been used more than once	Parsons[29]
	1924	Birmingham	28 (15)	155		
	1926	Birmingham	36 (30)	64		
		Glasgow	50 (25)	150		
	1919–1925	London	56 (20)	224		
	1919–1926	England	168 (39)	266		
		England	334 (36)	591		
1934	1923–1924	Sheffield	64 (32)	135	Notes that since 1925 more have become parkinsonian	Hall[43]
1931	Unstated	London	129 (49)	136		Borthwick[44]
1937	1917–1926	Boston	100 (54)	84	Notes that only children with sequelae can be expected to show some recovery	Holt[45]

unclear how often any of these "types" including the amyostatic-akinetic type led to PEP or even if all of these types represented the same condition.

The importance of these data (Table 1) is to demonstrate that EL was not initially associated with Parkinsonism and, even later, some doubted this relationship or believed PEP and PD to be the same. Why this is so is not entirely clear but it suggests that there were some fundamental changes in the syndrome over time and thus the relationship between EL and PEP may not be as direct or consistent as it is currently perceived to be.

PEP VERSUS OTHER FORMS OF CHRONIC EL

Table 2 presents all the available actual numerical data for the number of PEP cases versus other forms of chronic EL that were reported from 1922–1937. The reported percentage of EL patients who immediately or eventually developed PEP symptomatology is very variable, ranging from no cases to the vast majority. Certainly, this variability related to definitional aspects of PEP as well as to the timeframe used to record the patient's history. On the basis of the data in Table 2, it is difficult to accept the premise that the vast majority of EL cases eventually developed PEP as advocated by Duvoisin and Yahr in 1965[46] or the statement by Dourmashkin in 1997[47] that, "the outstanding motor manifestation [of the chronic form] was the parkinsonian syndrome, present in almost every case." Rather, non-PEP sequelae (especially psychiatric) were common during the epidemic period and will likely still be prevalent should EL recur. The lack of attention to these non-PEP sequelae[47] have undoubtedly skewed the perception toward accepting a direct relationship between EL and PEP.

OCULOGYRIC CRISES

OCs are now considered almost a pathognomonic sign of PEP[48] and it is doubtful that they were described prior to the EL epidemic period (but see Jeliffe in Table 3). Nevertheless, this sign was not clearly identified until 1921 (4 years after EL was defined), and even then only putatively (Table 3). Thus, OCs were not part of the constellation of signs that were associated with the earliest descriptions of PEP and certainly were not considered part of acute EL symptomatology (von Economo never listed them as being associated with acute EL). Accordingly, considering OCs to be a sign consistent with a diagnosis of acute EL is incorrect (see Refs. [2] and [3]), although most modern putative cases of EL consider them inherent to the disease (e.g., Refs. [65] and [66]). That OCs are not part of acute EL symptomatology but are considered part of PEP symptomatology[48] raises some questions as to the continuity between the two conditions. Furthermore, that OCs were not initially recognized in PEP also raises questions about whether there were some changes in EL and/or PEP during the epidemic period, suggesting perhaps that these were not unitary syndromes. Accordingly, Wilson in the EL chapter in

TABLE 3. *Historical year-by-year account of oculogyric crises*

Source	Year	Comment
Oeckinghaus[49]	1921	Reported that in October 1920, after sleeping for 8 wk uninterrupted because of EL, a farmer's 15-yr-old daughter complained of her eyes rolling up.
Lemos[50]	1924	First description of OCs in France.
Fischer,[51] Meyer,[52] and Ewald[53]	1924	First description of OCs in Germany; Fischer noted that he could find no prior references to this type of ocular movement.
Geimanowitsch et al.[54]	1924	First description of OCs in Russia.
Hohman[55]	1925	Presented the first description of OCs in the US (four cases) and noted that he could find no prior references to this type of ocular movement; considered OCs to be a late onset sign of PEP, although they occurred as soon as 1 yr after the onset of parkinsonian signs in one case.
Barkas[56]	1926	Earliest British accounts of OCs; almost all cases were associated with chronic EL, almost always of the parkinsonian type.
Wimmer[57]	1926	Indicated that OCs occur 4–7 yr after the development of chronic EL.
McCowan and Cook[58]	1928	Reported that the earliest occurrence of OCs was 6 mo after EL onset, but they consistently appeared at least 1 mo after the onset of parkinsonian signs; also noted that the incidence of OCs was increasing; reported an incidence of 17% among 136 institutionalized encephalitics and saw their first OCs in 1923; stated that there was no relationship to any specific epidemic; their patients developed their initial EL episodes from 1919 until 1925.
Collier[59]	1928	OCs virtually exclusively in the "parkinsonian syndrome of lethargic encephalitis."
Bramwell[60]	1928	Stated that there was no mention of OCs in the literature before 1923 and noted that, in 11 cases, they were consistently associated with manifestations of Parkinsonism; described the sign as *pathognomonic* (our italics) and was sometimes of diagnostic importance because the accompanying Parkinsonism signs were too slight to be recognizable.
Critchley[61]	1928	Commented on the rarity of OCs, which occurred in only 5.6% of his 72 cases; noted that they are only found as part of the parkinsonian syndrome and that they may be becoming more frequent; observed that during a crisis the patient does not lose consciousness, but may have hallucinations.
Taylor and McDonald[62]	1928	OCs only occur in PEP.
Bennett and Patton[63]	1930	Reported that OCs, although a postencephalitic residual, may occur without any other parkinsonian sign, and presented one case as an example.
Jelliffe[64]	1932	Postulated that OCs had been observed before the epidemic of EL, were not solely present in EL/PEP patients, were not an isolated phenomenon, and were typically associated with some affective disturbances; hence, viewed them as secondary (functional) phenomena associated with psychiatric processes; also noted that OCs tended to occur late in the disease process, not during acute EL but during the chronic phase; although he maintained that OCs had occurred earlier than the 1920s, the descriptions he presented from earlier times are not convincingly representative of OCs.

his 1940 book, *Neurology*,[67] stated that, "... there is reason to believe the 'encephalitis' [EL] is not identical at all times and locations, and it may cover states of dissimilar etiology."

CONCLUSIONS

As might be expected, the relationship among EL, PEP, PD, and OCs during the 1920s was clearly "muddled," not only by diagnostic problems, but presumably also by language, communication issues (some associated with World War I) and the presumed delay in onset of PEP. PEP was not considered a sequel of EL by all epidemic period clinicians, and some considered it virtually the same as PD. These factors plus the lack of any clear diagnostic criteria for EL led us to question the assertion that EL led directly to PEP. In the next article we follow-up on this one, using the presented data and information on the post hoc rediagnosis of EL after the development of PEP, to suggest that the relationship between EL and PEP is not as direct as currently perceived and that PEP, similar to other parkinsonian disorders, had multifactorial causation.

Acknowledgments: Support for this research was provided by the Sophie Cameron Trust (www.thesophiecamerontrust.org.uk). Ms. Hope Owens and Dr. Roger Duvoisin provided the French translations, and Dr. Paul Foley and Mr. Kris Kallmeyer provided the German translations. Mr. Richard Steele provided constructive criticism on prior versions of this manuscript and reviewers provided very detailed and helpful criticisms of earlier versions of this manuscript.

Financial Disclosures: Nothing to report.

Author roles: All three authors contributed to all aspects of conceiving, writing and revising this manuscript.

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