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Review

Intelligence and epilepsy: The early era

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ABSTRACT

This contribution to the commemoration of the 20th anniversary of *Epilepsy & Behavior* addresses the topic of epilepsy and intelligence, an early focus of interest in the history of the neuropsychology of epilepsy. The path through which the earliest measures of intelligence found their way to epilepsy research is reviewed followed by an overview of the subsequent themes of research, points of disagreement, advances in research using measures of intelligence, and developing awareness of the limitations associated with reliance on intelligence tests.
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> I would not, perhaps, be going too far to say that it [epilepsy] invariably exerts a prejudicial influence on the minds of those who are afflicted with it.... Unfortunately, we have as yet no test to gauge the scope and accuracy of the "mind's eye".
>
> [J. Browne, Medical Director, West Riding Asylum, 1873 [1]]

1. Introduction

This contribution to the 20th Anniversary Issue of *Epilepsy & Behavior* addresses the topic of epilepsy and intelligence, an area of interest now with a 100+ year history of empirical research. While intelligence was a key focus in the early to mid-1900s, interest has arguably declined over time in concert with a growing focus on specific cognitive domains including memory, language, and executive function. In point of fact, while there have been numerous narrative, systematic, and meta-analytic reviews of various aspects of cognition in epilepsy, the last focused review of intelligence in epilepsy appeared almost 50 years ago [2]. That review, entitled *"Intellectual and Adaptive Functioning in Epilepsy: A Review of 50 years of Research"*, serves as a basis for this expanded examination of the early attempts to apply evolving metrics of intelligence to epilepsy and the research trends that followed over the subsequent decades. As will be seen, research examining intelligence in epilepsy represents an early microcosm of the interests, concerns, and controversies that persist to this day in the broader field of the neuropsychology of epilepsy.

Conveyed here are the efforts of early investigators who struggled with an understanding of the intellectual correlates of epilepsy, the tension between new empirical findings versus the longstanding ingrained clinical views of epilepsy, the impact of sociopolitical beliefs (e.g., eugenics), and the evolving understanding of epilepsy and its emerging treatment options, which were informed by research on intelligence. What also became clear were the limitations of intelligence research defined by the use of commercially available test measures. In addition to interesting clinical publications, a series of cogent reviews appeared over the decades that very eloquently articulated the progress and the limitations of this literature, and they will be emphasized. While the focus here is on a reexamination of older ground, a piece to follow will target research on intelligence in epilepsy from 1972 to the present. Lastly, an acknowledged limitation of this particular review is reliance on English language publications.

To streamline this presentation, Fig. 1 summarizes the major variable sets that were examined in relation to intelligence as well as exemplars of factors within each of those broad categories. Tables 1 and 2 provide a very brief summary of studies addressing intelligence in epilepsy in this era [3–62], maintaining the format of Tarter's [2] review, separating investigations emanating from specialized institutions (e.g., epilepsy colonies) from other settings. These tables include investigations where intelligence was a prime, or among the prime, factors of interest as opposed to studies where intelligence was reported simply to characterize the cohort (e.g., in a "participants table"), or where intelligence was used merely as a covariate in other hypothesis-driven analyses. Papers not included were those that presented only intelligence quotient (IQ) distributions, focused on factor scores or pattern analyses

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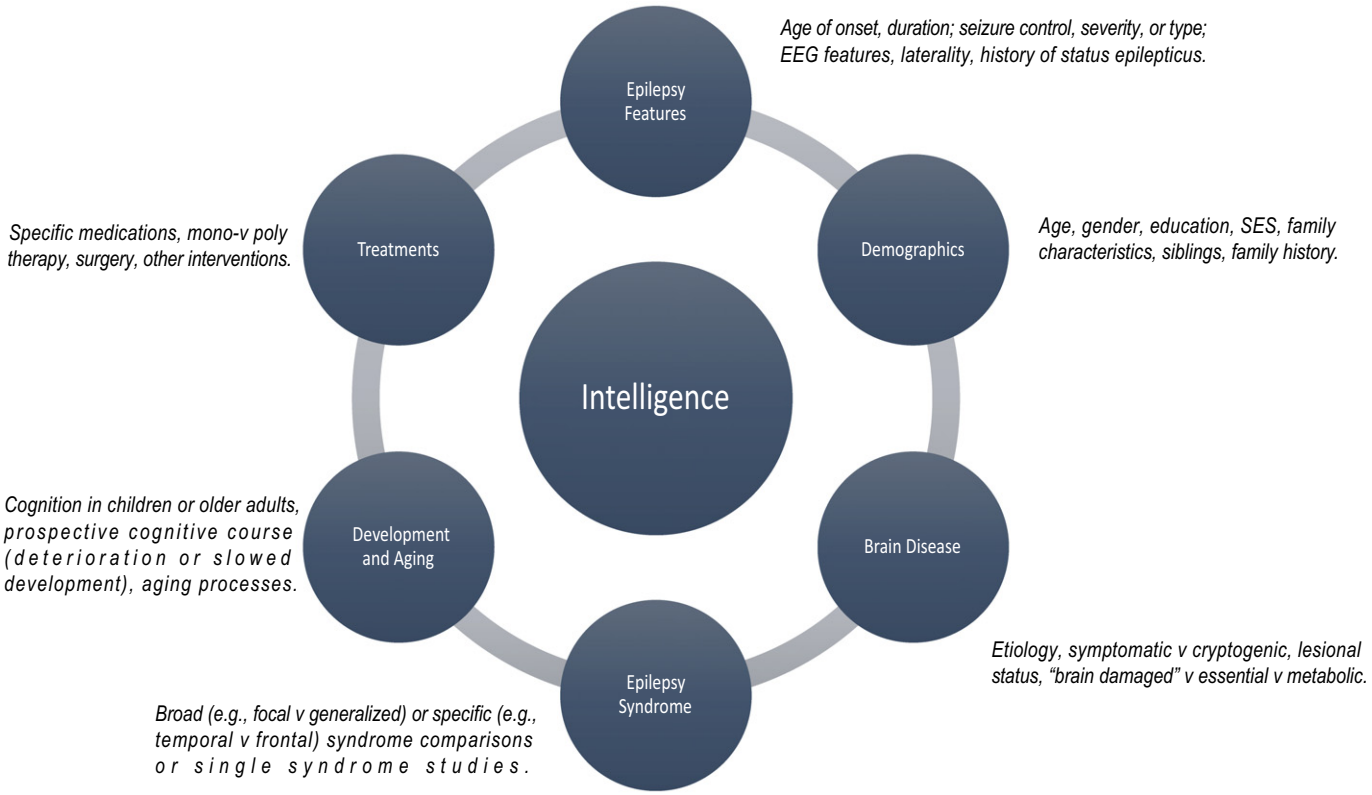

Fig. 1. Major variable sets examined in relation to intelligence in the studies reviewed.

without presentation of traditional IQ values, or those studies that focuses only on specific subtests from standard intelligence tests. The last columns in Tables 1 and 2 link to Fig. 1 by specifying the primary factors examined in relation to intelligence. Lastly, a recurring theme evident from before the onset of empirical research in this field to the present has been the issue of progressive deterioration in intellectual function, and publications with a test–retest paradigm (albeit some evaluating treatment outcomes such as medications or surgery) are underlined in the Tables. The topic of cognitive deterioration has been a subject of many prior reviews, both in this early era [2,63–67] as well as more recently [68–72], and the topic will not be relitigated here.

2. The road of intelligence to epilepsy: Binet–Simon to Goddard to Wallin and Fox

2.1. Alfred Binet and Theodore Simon

The path to the empirical investigation of intelligence in epilepsy begins with the work of Binet and Simon who were so influential in the measurement of individual differences [73–76]. As they stated [77],

> "…let us recall exactly the conditions of the problem which we are attempting to solve. Our purpose is to be able to measure the intellectual capacity of a child who is brought to us in order to know whether he is normal or retarded." (p. 37)

Their task was initiated in the context of variable nomenclature and a dearth of empirically driven taxonomies for understanding, measuring, and classifying mental handicap. There were instead a variety of clinical perspectives proposed by experts of the time, outlined and criticized in depth by Binet and Simon [77–78], to the likely chagrin of those reviewed. Examining the various ways that children with disabilities were classified, Binet carefully overviewed and criticized in detail the methods and models of his contemporaries. Disavowing the anthropometric approach current at the time, Binet instead focused on higher mental process, a decision antithetical to the era that he pursued with enthusiasm with Simon in their original and then revisions to the test [77–80].

> "The Binet–Simon Intelligence Scale represented a major paradigm shift for the young field of psychology. It tapped intelligence through assessment of complex mental abilities, as opposed to the narrow sensory and motor measures dominating the Galton–Cattel batteries. It was standardized, with explicit procedures for administration and objective scoring guidelines. It was norm referenced, permitting an individual's performance to be compared with that of his or her age peers. It was reliable, yielding consistent scores from one occasion to another. It was developmentally sensitive, recognizing that mental abilities in children develop in a meaningful progression and that the abilities of children differ substantially from that of adults… Finally, and most importantly, it seemed to work fairly well, providing an empirical foundation for the nascent study of intelligence and cognitive abilities." ([73], p.16–17)

While the measure was available it had yet to makes its way to the epilepsy community and for that aspect Henry Goddard played an indirect but important role.

2.2. Henry Goddard

Goddard's history and notoriety in the use and misuse of intelligence testing has been the subject of several reviews [81,82]. Here, the focus is on his role as a conduit in facilitating intelligence testing in epilepsy. In 1906, Goddard relocated from his teaching position at the West Chester State Normal School in Pennsylvania to the New Jersey Training School for Feeble-Minded Girls and Boys in Vineland NJ with a goal to develop a research laboratory to advance understanding of intellectual impairment. Goddard articulated the same frustrations that Binet and Simon

Table 1
Studies from specialized institutions.

Authors	Year	Sample	Test	IQ score	Factors
Wallin [3]	1912a	Children and adults w/ epilepsy (n = 333) and 378 Vineland residents (n = 378)	Binet–Simon	Higher mental age in epilepsy compared to feeble-minded participants	None
Fox[a] [4]	1924	Children, age 5–16 (n = 150)	Binet–Simon	Median boys = 71 Median girls = 65	Demographics, Development and aging
Collins et al.[b] [5]	1938	Children and adults from mixed sources, age 1–60+ (n = 229). Mdn age = 24	Stanford–Binet	Mean = 78	Epilepsy features
Kuglemass et al.[a,b] [6]	1938	Children from institution (n = 129) and private practice (n = 91), age 4–18	Stanford–Binet and others	Etiology (among improved pts) Mdn[f] primary institution = 56 Mdn secondary institution = 51 Mdn primary private pts. = 98	Development and aging, Epilepsy features, Brain disease
Collins[a] [b] [7]	1941	Children and adults, age 5–35 (n = 106)	Stanford–Binet	Mdn = 62	Epilepsy features, Brain disease, Demographics, Treatment, Development and aging
Shotwell & McCulloch [8]	1944	Children and adults, age 16–49 (n = 100)	Stanford–Binet	Mean = 49.2	None
Hilkevitch[a] [9]	1946	Children and adults, age 8–53 (n = 66)	Stanford–Binet	Mean = 75.3 Major seizures = 66.9 Minor seizures = 75.0	Demographics, Epilepsy features, Development and aging
Reed [10]	1951	Children and adults, age 5–45 (n = 295)	Stanford–Binet, Wechsler–Bellevue Kuhlman Tests	Mdn = 42[c]	Demographics, Epilepsy features
Balthazar [11]	1963	Adults, age ns[e] (n = 63) with EEG determined laterality of damage (L = 21, R = 15, I = 27)	Wechsler–Bellevue	Left hemisphere damage = 71.9 Right hemisphere damage = 70.9 Indeterminate damage = 71.3	Brain damage, Epilepsy features
Fitzhugh & Fitzhugh [12]	1964a	Adults, mean age = 40 (n = 179)	Wechsler-Bellevue and Wechsler Adult Intelligence Scale	Wechsler Bellevue FSIQ = 74.56 WAIS FSIQ = 72.18[d]	
Fitzhugh & Fitzhugh [13]	1964b	Adults left hemisphere (mean age = 37.3), right hemisphere (mean age = 35.75) and diffuse damage (mean age = 37.8) (n = 98)	Wechsler Adult Intelligence Scale	Left hemisphere damage = 69.8 Right hemisphere damage = 69.2 Diffuse damage = 69.5	Epilepsy features
Fitzhugh et al. [14]	1964c	Adults, mean age young = 24.2, mean age older = 49.7 (n = 283)	Wechsler–Bellevue	Young = 72.7 Older = 79.9	Demographics

Note: For Tables 1 and 2, summary IQ values are presented in the "IQ score" column (e.g., WAIS Full Scale as opposed to including Verbal and Performance IQ scores as well). In many cases, the primary aim of a study was not simply to provide an overall IQ value, for instance the study may have focused on examining test–retest course, but baseline IQs were provided and are presented in the Tables, with broader discussion of the paper in the text. Not included in the Tables are studies presenting or using only factor scores, subtest scores, IQ distributions, or other partial presentations of intelligence test data. Some readers may have preferred to have these studies included here. Given those limitation it is clear there are other investigations from this era that use intelligence test data.

[a] Study involves test–retest paradigm.
[b] Subjects from mixed sources (institutionalized and noninstitutionalized).
[c] Based on 197 patients administered Stanford–Binet.
[d] All subjects took both tests and all IQ and subtest scores compared.
[e] ns = not specified.
[f] Mdn = median.

expressed involving the classification of children with mental deficiency [81]. He, however, was not one to burn bridges early on, and his observations of the needs of the field were less cantankerously expressed. In an ongoing search for improved methodology and taxonomy, Goddard felt that he had exhausted potential sources and applications in America and in 1908 traveled to Europe. As he later said,

> "It was not until the Spring of 1908 when I made a visit to Europe in the interests of the work that I learned about the tests. On that trip a visit was made to Dr. Decroly in Brussels. Dr. Decroly and Mlle. Degand had just completed a try-out of tests by Drs. Binet and Simon of Paris. Upon my return home I began at once on the children of the Training school, employing Decroly's article as the source of information. Later I obtained Binet's article." ([83], p.5).

The original publications of Binet and Simon appeared in French journals, and only later a 1916 English translation was made possible through Goddard and the Vineland Training School [84]. The Binet and Simon papers were translated from French by Elizabeth Kite, an extremely bright "field worker" in the eugenics tradition located at the Training School. In the preface of the text, Goddard summarized his unawareness of Binet and Simon's 1905 work until 1908, appreciating its promise when testing it at Vineland, and especially stimulated by the 1910 revision [73,81].

2.3. J. E. Wallace Wallin

Wallin was the first to report results of an application of the Binet–Simon Scale to persons with epilepsy, his history reviewed in detail previously [85,86]. Briefly, in 1910, Goddard invited Wallin to offer a summer course in functional psychology at the Training School in Vineland while Goddard was again traveling in Europe. During that summer, Wallin observed administration of the Vineland translation of the 1908 Binet–Simon and other measures. After the end of the term, Wallin was encouraged by Edward Johnstone, Director of the Training School, to accept an offer to work at the nearby New Jersey Village for Epileptics in Skillman NJ, beginning in October 1910, where he would serve as the first staff member of a newly established Laboratory of Clinical Psychology, "…the first laboratory of clinical psychology in an institution for epileptics anywhere in the world" [87]. Wallin quickly undertook systematic assessment of the residents, reporting the results of the first administration of the Binet–Simon Scale to persons with epilepsy

Table 2
Studies from noninstitutionalized (NI) sources.

Authors	Year	Sample	Test	IQ score	
Dawson & Conn[a] [15]	1929	Children, age 4–12 (n = 49), siblings (n = 20), and general hospital children (n = 991)	Binet–Simon	Epilepsy mean = 80.6 Sibling mean = 91.2 Hospital controls mean = 90.6	Development and aging, Demographics, Epilepsy features
Fetterman & Barnes[a] [16]	1934	Children and adults, age ns[b] (n = 105)	Stanford–Binet	Mean = 74 Idiopathic etiology = 77 Alcoholic etiology = 70 Organic etiology = 69	Epilepsy features, Development and aging, Brain disease
Sullivan & Gahagan[a] [17]	1935	Children with epilepsy, age 2–14 (n = 103), children with allergy (n = 45), LA city school students (n = 63,147)	Stanford–Binet	Median epilepsy = 92.4 Median allergy = 103 Median LA schools = 105	Demographics, Epilepsy features, Development and aging, Brain disease
Somerfeld-Ziskin & Ziskin[a] [18]	1940	Children and adults, age 3–58 (n = 100) —"virtually untreated at study entry"	Stanford–Binet	Mean = 93 Mean cryptogenic = 97 Mean symptomatic = 88	Brain disease, Treatment, Development and aging
Arieff & Yacorzynski[a] [19]	1942	Adolescents and adults with organic epilepsy, age ns (n = 27)	Stanford–Binet	Organic = 74.3 Nonorganic = 85.1[c]	Brain disease, Development and aging,
Yacorzynski & Arieff[a] [20]	1942	Youth and adults, nonorganic epilepsy (n = 54)	Stanford–Binet	Idiopathic = 84.0 Focal = 86.6	Treatment, Epilepsy features, Treatment
Lennox & Collins [21]	1945	Child and adult twins, age 4–61 (n = 93 twin pairs, 60 no epilepsy, 33 one or both with epilepsy)	Stanford–Binet and Wechsler–Bellevue	Nonepilepsy controls = 108 Epilepsy w/o brain injury = 96 Epilepsy with brain injury = 77	Brain disease, Demographics (twin)
Sands & Price [22]	1947	Adults, mean age = 28.9 (n = 77)	Wechsler–Bellevue	Idiopathic = 104.5 Organic/symptomatic = 94.8 Idiopathic/personality = 107	Brain disease
Collins & Lennox [23]	1947	Children, age 4–15 (n = 100) Adults, age 16+ (n = 200)	Stanford–Binet and Wechsler–Bellevue	Mean males = 103.1 Mean females = 105.1 Mean males = 110.8 Mean females = 111.8	Demographics, Brain disease, Epilepsy features
Lewinski [24]	1947	Adolescents and adults, age 17–35 (n = 25, all males)	Wechsler-Bellevue	Mean = 99.7	
Sheps [25]	1947	Adolescents and adults, age 17–44 (n = 133 Form A, n = 237 Form B, all males)	Canadian Army Intelligence Test (Examination M) (Forms A and B)	Form A mean Controls = 124.3[d] Epilepsy = 110.9 Form B mean Total sample = 126.4 Epilepsy = 120.1	No comparison to IQ
Collins [26]	1951	Adolescents and adults, age13–65 (n = 400)	Wechsler-Bellevue	Mean = 108.04	Demographics, Brain disease, Epilepsy features
Winfield [27]	1951	Male veterans, age 22–34 (n = 60)	Progressive Matrices (Forms C,D,E)	Normal controls = 23.7 Cryptogenic epilepsy = 22.9 Symptomatic epilepsy = 18.6 Post traumatic nonepilepsy encephalopathy = 18.4	Brain disease, Epilepsy features
Zimmerman et al. [28]	1951	Children, age 3–16 (n = 100) Adults, age 16–71 (n = 200)	Stanford–Binet and Wechsler–Bellevue	Mean children = 92.6 Idiopathic petit mal = 100.5 Idiopathic petit/grand = 91.5 Idiopathic grand mal = 91.3 Symptomatic = 89.0 Traumatic = 89 Mean = adults 100.4 Idiopathic petit mal = 108.7 Idiopathic petit/grand = 108 Idiopathic grand mal = 98.5 Symptomatic = 98.1 Traumatic = 92.5	Brain disease, Epilepsy features
Davies-Eysenck[a] [29]	1951	Children and adults, age 6+ (n = 161)	Progressive Matrices	Median = 94	Development and aging, Clinical seizure features, Demographics
Meyer & Yates[a] [30]	1955	Youth and adults, age 15–43 (n = 18)	Wechsler–Bellevue	Pre- to PostOp (3–4 wks) Dominant = 92.7 to 84.4 Nondominant TL = 101.2 to 99.4	Clinical seizure features, Treatment, Epilepsy syndrome
Hovey & Kooi [31]	1955	Epilepsy, age ns (n = 50), organic brain disorder, age ns (n = 54), functional, age ns (n = 65)	Wechsler–Bellevue	Epilepsy = 101 Organic nonepilepsy = 99.2 Functional = 104.4	Clinical seizure features
Quadfasel & Pruyser [32]	1955	Male veterans, mean age 33.1 (n = 38)	Wechsler–Bellevue	Psychomotor = 107 Grand mal = 107	Epilepsy syndrome
Tenny	1955	School age children attending special school for epilepsy (n = 690)	Detroit Intelligence Test Series	Mdn = 84	
Halstead H [35]	1957	Children with epilepsy, median age 11 [girls) and 11.5 [boys] (n = 68) selected from three sources (normal, residential, and physically handicapped). Controls (n = 54), mean age 10.5 for boys and girls	Stanford–Binet	Controls = 99.8 Epilepsy = 87.5 Normal schools = 95.9 Residential school = 79.2 Physical handicap = 60.0	Demographics, Epilepsy features, Brain disease Behavior
Halstead W[a] [36]	1958	Tested before and after anterior temporal lobectomy (n = 17, 8 dominant, 9 nondominant). Mean age of all 21 operated	Henmon–Nelson Test	Pre- to PostOp Verbal IQ Dominant = 86.0 to 85.9 Nondominant = 89.2 to 91.3	Epilepsy syndrome, Epilepsy features, Treatment,

Table 2 (*continued*)

Authors	Year	Sample	Test	IQ score	
		pts. = 30.9		Pre- to PostOp Performance IQ Dominant = 110.5 to 118.1 Nondominant = 103 to 114	
Milner[a] [37]	1958	Adolescents and adults, age 14–45 (n = 30)	Wechsler–Bellevue	Left temporal pre-op = 108.5 Right temporal pre-op = 103.0	Treatment, Clinical seizure features, Epilepsy syndrome
Wilson et al. [38]	1959	Adult veterans (n = 38)	Wechsler Intelligence Scale	Mean = 88	
Meyer[a] [39]	1959	Youth and adults, age 12–51 (n = 25)	Wechsler–Bellevue	Pre-Op to Post-Op (1 yr) Dominant TL = 98.2 to 95.2 Nondominant TL = 103.4 to 113.1	Epilepsy features, Demographics, Treatment, Epilepsy syndrome
Mirsky et al. [40]	1960	Adults with TLE, age 31 (n = 39), FLE, age 31 (n = 18), Generalized, age 28.1, n = 19), Controls, age 31.5 (n = 25)	Wechsler–Bellevue	TLE = 100.6 FLE = 91.6 Generalized = 85.7 Control = 100	Epilepsy syndrome
Parsons & Kemp [41]	1960	Hospitalized male veterans. Control mean age = 31.5 (n = 16), psychomotor mean age = 32.1 (n = 16), generalized mean age = 32.2 (n = 16)	Wechsler Adult Intelligence Scale	VIQ PIQ Psychomotor: 96.0 91.5 2 Generalized: 91.1 86.3 Controls: 93.7 94.2	Epilepsy features, Epilepsy syndrome
Shalman [42]	1961	Left TLE right handed, age = 34.5 Left TLE left handed, age = 27.1. Nonfocal left handed, age = 32.4 (total n = 21)	Wechsler-Bellevue	Pre-Op IQ TLE right handed = 103.8 Left TLE left handed = 104.6 Nonfocal left handed = 103.4	Demographics, Epilepsy Features, Epilepsy syndrome
Chaudrey & Pond[a] [43]	1961	Youth, age 2–18 who do (n = 28) or do not (n = 28) exhibit progressive cognitive deterioration, selected from two sites (hospital, institution). All reportedly brain damaged.	Wechsler Intelligence Scale for Children, Stanford–Binet, and others.	Baseline and follow-up IQ ranges provided for subjects by ascertainment location.	Clinical seizure features, Demographics, Treatment, Development and aging Other (behavioral complications, premorbid ability, social/family)
Lansdell [44]	1962	Left speech dominant with left or right hemisphere epilepsy (n = 18) and right speech dominant (n = 9) with left hemisphere epilepsy (n = 9) matched for IQ.	Wechsler–Bellevue	Right speech dominant left hemisphere epilepsy cases have more preserved verbal subtests than left speech dominant left hemisphere epilepsy.	Clinical seizure features
Dennerell et al. [45]	1964	Children, mean age 10.3 (n = 100) and adults, mean age 28.4 (n = 100)	Wechsler Intelligence Scale for Children Wechsler Adult Intelligence Scale	Children = 89.0 Adults = 96.9	Demographics
Dennerll [46]	1964b	Children and adults, psychomotor epilepsy, mean age = 20.6 (n = 60)	Wechsler Intelligence Scale for Children Wechsler Adult Intelligence Scale Wechsler–Bellevue	95.2 Factor scores examined	Demographics
Lansdell & Mirsky[a] [47]	1964	Centrencephalic, mean age 22.8 n = 19) and focal epilepsy, mean age 31.4 n = 65) [33 patients post ATL re-evaluated 3 weeks later]	Wechsler–Bellevue	Centrencephalic = 100.1 Focal = 98.5 Left TLE cases showed a drop in Verbal IQ (scores not provided), no PIQ changes	Epilepsy features, Epilepsy syndrome, Treatment
Meir & French [48]	1965	Temporal lobectomy subjects (n = 53). Unilateral mean age = 27.8, bilateral mean age = 28.3	Wechsler–Bellevue	Bilateral EEG abn = 97.8 Unilateral EEG abn = 98.6	Syndrome, Epilepsy features
Klove & Matthews [49]	1966	Adults (mean age) Control = 33.5 Brain damage w/o epilepsy = 34.9 Brain damage w epilepsy = 34.7 Epilepsy unknown etiology = 31.7	Wechsler Adult Intelligence Scale	Control = 109.1 Brain damage w/o epilepsy = 91.7 Brain damage w epilepsy = 94.9 Epilepsy unknown etiology = 100.29	Epilepsy features, Brain disease
Dennerll et al. [50]	1966	Employed age = 29.3 (n = 43) Unemployed age = 27.6 (n = 46) Prevocational age = 17.5 (n = 32)	Wechsler Adult Intelligence Scale	Employed = 103.6 Unemployed = 89.0 Prevocational = 102.3	Epilepsy features
Small et al. [51]	1966	Temporal lobe epilepsy, mean age 37.4, Centrencephalic, mean age 26.0, Focal Nontemporal, mean age 33.2 (n = 50).	Wechsler Adult Intelligence Scale	No temporal lobe vs nontemporal lobe epilepsy differences on WAIS VIQ, PIQ, FSIQ. TL group significantly better on Arithmetic, Comprehension, Similarities scales.	Epilepsy syndrome, Epilepsy features, Other (psychiatric diagnoses)
Meier & French[a] [52]	1966	Adult temporal lobe epilepsy before and after surgery (n = 56). Mean age left TLE = 26.3, right TLE = 26.8	Wechsler–Bellevue and Wechsler Adult Intelligence Scale	PreOp = 94 LTLE, 99.1 RTLE PstOp1 = 95.8 LTLE,101 TLE PostOp2 = 92.3 LTLE, 95.9 RTL	Epilepsy features, Treatment, Epilepsy syndrome
Blakemore et al. [53]	1966	Youth and adults, age 11–45. Left hemisphere, mean age 25.9 (n = 19). Right hemisphere, mean age = 28.2 (n = 13)	Wechsler–Bellevue, Wechsler Adult Intelligence Scale,	Left hemisphere = 90.63 Right hemisphere = 105.3 All patients	Epilepsy syndrome, Epilepsy features, Brain disease

(*continued on next page*)

Table 2 (*continued*)

Authors	Year	Sample	Test	IQ score	
			Wechsler Intelligence Scale for Children	Max Sz/Max = 95.6 Min Sz/Min Path = 96.4 Max Sz/Min Path = 91.0 Min Sz/Max Path = 104.0	
Blakemore & Falconer[a] [54]	1967	Anterior temporal lobectomy patients (n = 86, left [dominant] = 54, right [nondominant] = 32, age ns)	Wechsler Intelligence Scales	Pre-Op Verbal/Performance LH: 96.7/103.3 RH: 102.3/97.4 Postop (10 + years) LH: 97.5/104.3 RH: 104.9/97.2	Epilepsy syndrome, Treatment, Epilepsy features
Matthews & Klove [55]	1967	Adults (mean age) Known etiology (n = 65) Major motor = 32.4 Psychomotor = 29.0 Mixed = 30.1 Unknown etiology (n = 69) Major motor = 30.7 Psychomotor = 28.3 Mixed = 26.9 Control (N = 99) Nonneurological = 33.5 Brain damage w/o seizures = 33.3	Wechsler Adult Intelligence Scale	Known etiology Major motor = 93.4 Psychomotor = 95.1 Mixed = 96.6 Unknown etiology Major motor = 97.2 Psychomotor = 105.9 Mixed = 94.1 Control Nonneurological = 108.1 Brain damage w/o seizures = 93.2	Epilepsy features, Brain disease
Fedio & Mirsky [56]	1969	Children, age 6–14 (n = 60) Mean ages: left/right temporal = 10.2/10.8, controls = 10.2 centrencephalic = 10.8	Wechsler Intelligence Scale for Children	Left temporal = 96.9 Right temporal = 102.8 Centrencephalic = 97.5 Controls = 104.9	Epilepsy features, Epilepsy syndrome
Needham et al. [57]	1969	Patients with familial epilepsy (n = 73) Family member abn EEG/no sz (n = 38) Family members nrml EEG/no sz (n = 186) Ages ns	Wechsler Intelligence Scale for Children, Wechsler Adult Intelligence Scale, Stanford–Binet	WAIS Patients = 89.5 Family EEG abn/no sz = 107 Family nrml EEG/no sz = 107.3	Epilepsy features, Demographics
Schwartz et al. [58]	1970	Children age 9–15 (n = 140) Mean ages: epilepsy = 12.1, questionable epilepsy = 11.9, controls = 12.7	Wechsler Intelligence Scale for Children	Epilepsy = 91.2 Questionable/uncertain epilepsy = 101.0 No epilepsy = 102.5	Demographics, Epilepsy features
Singh & Virmani [59]	1970	General (n = 20), focal (n = 9) and psychomotor (n = 6), mean age = 23	Wechsler Adult Intelligence Scale	General = 96.6 Focal = 93.2 Psychomotor = 94.8	Epilepsy features
Rutter et al. [60]	1970	Children age 5–14 (uncomplicated epilepsy n = 58, with structural brain disorder n = 22, cerebral palsy n = 33, other brain disorder n = 20)	Abbreviated WISC (2 verbal and 2 performance subtests)	Epilepsy = 102 Cerebral palsy = 78 Other brain disorders = 74	Brain disease
Stevens et al. [61]	1972	Adults, age ns (n = 29)	Wechsler Adult Intelligence Scale	Temporal lobe = 102.7 Centrencephalic = 91.7	Epilepsy syndrome, Treatment (surgery),
Hartlage & Green [62]	1972	Children and adolescents, 6–16 mean age = 11)	Wechsler Intelligence Scale for Children	Grand mal = 78 Petit mal = 85 Psychomotor = 80 Focal = 83 Mixed = 81 Overall = 82	Epilepsy features

[a] Study involves test–retest paradigm.
[b] ns = not specified.
[c] The nonorganic patients reported to come from same clinic but specifics/demographics not presented.
[d] The mean and standard deviation for the Canadian Army Intelligence Test (Revised examination M) was not specified and comparability to standard IQ metrics is unclear.

in his 1912 paper published in *Epilepsia* [3]. In that paper, he presented the results of a comparison of residents with epilepsy (n = 333) to "feeble-minded" residents from Vineland (n = 338) on the Binet–Simon. He reported that residents with epilepsy were not as impaired as traditional "feeble-minded" persons.

In his paper, Wallin referred to more comprehensive assessment of the residents that included tests of perception, memory, reasoning, and other cognitive abilities, as well as the use of inventories designed to assess social, personal, industrial, motor, and school status. He referred to his efforts to obtain a control group of children attending normal school who through serial testing would facilitate normative data that could be used to characterize both abnormal neurodevelopmental and degenerative patterns in residents with epilepsy. The results of these efforts were reported in subsequent publications including texts devoted to cognition in epilepsy [88,89]. Overall, Wallin undertook the first systematic serial assessment of residents with epilepsy who were tested 5 times in 5 months to track intellectual development. That said, Wallin left the New Jersey center after only 8 months because of personality/interpersonal clashes with the superintendent (Dr. Weeks) [87].

2.4. J Tyler Fox

For unknown reasons, it took 12 years for the second publication to appear examining intelligence in residents with epilepsy, but as will be shown later, intelligence testing was underway at other sites as early as 1916 [5]. Fox [4], at the Lingfield Epileptic Colony in the UK, reported the results of administration of the Binet–Simon, again in the context of a more comprehensive array of cognitive and academic achievement tests to 150 children between the ages 5–16 years, characterizing their status as well as examining changes in intelligence over 2 years in 130 children. Fox also arguably examined the ecological validity of the

tests by comparing psychometric scores to staff ratings of the potential adaptive living skills of the youth. He also characterized a variety of other cognitive and academic abilities and the degree to which they lagged behind intelligence-based expectations. Fox was prescient in articulating a need to investigate the role of etiology and clinical manifestations of this "diverse disorder", as well as the need for more prolonged prospective investigations of intelligence in epilepsy.

The thoughtful and empirically driven nature of the early contributions by Wallin and Fox set the stage for the work to follow. They saw the need for assessment of cognition beyond intelligence, incorporated prospective assessments, and struggled with the meaning and limitations of their findings. At the time of their work, a number of specialized facilities for epilepsy had been and were continuing to be established [90,91]. Some became laboratories for investigation that, as we will see, became lightning rods for criticism.

3. Intelligence takes hold: 1920–1940

A few early papers examined specific tasks from the Binet–Simon such as the digit span task [92] (not included in Tables), but more commonly the entire Binet–Simon or the later developed Stanford–Binet [93] were administered. Seven publications appeared in this time frame with 3 of the 7 emanating from institutions, in whole or in part.

These early studies revealed a number of interesting findings that included the following: a) inclusion of siblings of children with epilepsy whose higher IQs (91.2 siblings vs 80.6 patients) were felt to argue that children with epilepsy came from families of "normal intellectual stock" [15], b) studies of persons with epilepsy from sites outside of institutions exhibited better intellectual status which spoke to the issue of ascertainment bias associated with epilepsy colonies [6,17], c) presumed symptomatic etiology was more associated with compromised intellectual ability than so-called essential epilepsy (e.g., [16]), d) comparison to other disease groups showed the relative standing (generally lower) of the intelligence of persons with epilepsy [15,17], e) examination of cognition in relation to the primary medications of the time, including phenobarbital, revealed no adverse effect on intelligence in a prospective investigation [18], and f) early detailed analyses of Stanford–Binet response patterns/subtest scatter occurred, which were felt to provide a sense of the cognitive strengths and weaknesses associated with epilepsy (e.g., strong vocabulary, weak practical judgment) [5].

The first review of this developing literature appeared in 1941, written by Molly Harrower-Erickson [63], then at the Montreal Neurological Institute (MNI), where she summarized:

> "There is not a single group among hospitalized patients in which the average or mean IQ is normal. The results vary from markedly subnormal findings (IQ as low as 65), to near normal (IQs of 92 and 93)…. while no large group of extramural patients has been studied psychometrically, the evidence that exists points to the fact that many such patients may have average or above average intelligence". (p. 549)

The last sentence is perhaps difficult to understand given the existing published literature, but Harrower-Erickson worked at the MNI for a period of time and saw other than institutionalized patients with epilepsy, which may have served as a basis for her opinion.

Other themes which were to become recurring topics in the neuropsychology of epilepsy were raised in this review including whether there was evidence of cognitive deterioration (no fixed evidence per Harrower-Erickson), the relation of mental status to the frequency, severity, cause and age of onset of the seizures, the relationship between intelligence and medication treatment, and the psychological effects of epilepsy. As she summarized,

> "When judged in terms of scores in *psychometric tests*, therefore, it would seem that, to date, no significant relationship has been shown to exist between the severity and frequency of attacks, and the mental condition of the patient. There is a suggestion that the cause of the attacks is in some way connected with the intelligence level, particularly when the patients who comprise the organic or symptomatic groups are known to have diffuse cerebral damage. There is, also, evidence that in some types of attacks, the earlier the onset the more likely is there to be some degree of mental deterioration." (p.552)

Another obvious question asked by early researchers was why intelligence scores were lower. Were there particular tasks that were more challenging/impaired than others, and here opinion was mixed, ranging from "no" [17] to "yes" with a focus on memory span [for digits] [92], or memory, attention and language [18]. So in this early window (1911 to 1940), the broad outlines of many questions that had been asked, and would be repeatedly examined over decades, were laid out.

One cautionary note in historical reviews such as this, where disorders have benefitted from substantially increased medical understanding over time, is that the meaning of frequently used terms related to important aspects of the disorder, for instance, etiology (idiopathic, symptomatic, genetic), or seizure type, may have decidedly different meanings and implications in the older literature (for example, see Shorvon [94] for a discussion of the changing meaning of "etiology" over time).

4. Moving forward and out of institutions — 1941–1949

During this interval an increasing proportion of publications emanated from noninstitutional ($n = 7$) compared to institutional ($n = 3$) sources. The papers from institutions all using the Stanford–Binet again exhibited much lower IQ scores, which ranged from a low of 42 to a high of 75 [7–10]. The core features of several of these investigations were as we might see today, taking a metric of cognition, intelligence in this case, and examining performance and/or performance change over time in relation to presumed etiology, seizure frequency/severity, presence of status epilepticus, medication, onset age, duration of disorder, aspects of family background, and often with examination of individual test items in an attempt to identify relatively spared versus more adversely affected abilities [7]. It was in this window that the first attempt to devise a short form of the Stanford–Binet for use in patients with epilepsy appeared [8].

The powerful effect of ascertainment bias became strikingly evident with institutional studies continuing to show very low IQ scores while patients from outpatient and private practice settings, hospital-based locations, and military sources [24,25], garnered in part to demonstrate that the institutional studies were biased, revealed much higher IQs ranging from 91 to over 100 [21–23].

Pertinent to this theme was the notable collaboration between psychologist A. Louise Collins and epileptologist William Lennox, who investigated intelligence in epilepsy with a series of interlocking papers with increasingly larger sample sizes over the years. Collins' first publication on epilepsy in 1938 emanated from the Boston Psychopathic Hospital followed by a publication from Monson State Hospital in 1941. An important publication from their collaboration appeared in 1946 in the published proceedings of a scientific society meeting [23]. Here, they reported results from 300 private practice patients (100 children and 200 adults), stating that the results were being presented predominantly in charts as they anticipated a later formal publication. One hundred participants were 4–15 years of age and were tested with the Stanford–Binet, and 200 participants were 13–50 years of age and were tested with the Wechsler-Bellevue [95]. The mean IQ for all 300 participants was solidly average (108.9); mean IQ for the younger group was average (104.2) and high average for the adult group (111.4). Examined was the impact of the absence/presence of brain injury in both children (106.5 vs 96.7) and adults (113.9 vs 102.1). In an interesting subanalysis, they examined child IQ as a function of father's occupation, which ranged from a mean child IQ of 99 when father was a

"skilled laborer" to a mean child IQ of 114 when father was a "major executive". More generally, they summarized,

> "We arrive at the general conclusion that private patient epileptics—as represented by this 300 sample—have better than average intelligence, and that genetic (essential) epilepsy is preferable to acquired (symptomatic) epilepsy. Verbalized and block design tests are best performed and rote memory and concentration fare the worst. Pattern analyses resemble the normal more closely than the psychotic or neurotic. ([23], p. 600)

As will be seen immediately below, in 1951, Collins was the sole author reporting results from an expanded sample of their private practice adult cohort, expanding the 1946 adult sample by another 200 patients. The pediatric data were not presented independent of the 1946 paper. By 1960, their total sample size reached 600 (400 adults and 200 children) because of the addition of another 100 children with epilepsy, the very brief summary of the pediatric data presented by Lennox in his text *Epilepsy and Related Disorders.*

5. The pace quickens — 1950–1959

The 1950s were a period of increasing research addressing cognition in epilepsy. Fifteen papers appeared with only one from an institution — so the ascertainment trend had shifted in a major way. This period saw the continuation and development of several interesting and important clinical and research trends.

5.1. A major empirical contribution to intelligence in epilepsy

In a definitive 1951 paper entitled "Epileptic Intelligence", Collins [26] made a singular contribution by intensifying the focus squarely on intelligence in noninstitutionalized persons with epilepsy.

> "Past studies of epileptic intelligence have seldom been based on extramural patients. Only the records of instructional or public clinic patients have been available for such studies, and these have often included a large proportion of mental defectives. The intellectual deficits were usually concomitants or precedent conditions and not resultants of epilepsy, but the low intelligence was often assumed to be that of epileptics in general". ([26], p. 392)

> "The present study is based on a sample of the records of the neglected majority, private patients who have come or have been sent by their physicians to the Seizure Unit of the Children's Medical Center in Boston for treatment under its director, Dr. William Lennox." (p 392)

The sample size was large (n = 400) with a wide age range (13–63), and the mean IQ was 108, in fact,

> "Our distribution is not that of a normal curve, due to the large proportion of the group in the three highest grades" (bright normal, superior, very superior)." (p 393)

Collins was very interested in pattern analysis of intelligence test performance. On the Wechsler–Bellevue, she found that the three lowest scores (though still average) were digit symbol, arithmetic, and digit span, the latter two considered "concentration tests". An earlier age of onset but not duration of disorder was significantly associated with lower intelligence, intelligence was associated with clinical seizure types (highest IQ in petit mal epilepsy), electroencephalography (EEG) classification (highest IQ in association with normal EEG), and etiology (higher IQ in essential epilepsy).

Collins pointed out that "early writers usually maintained that the constitutional 'idiopathic' type of epilepsy had a more deteriorating effect on the patient's mentality than did epilepsy due to brain injury" (p 305), presumably because of the long proposed "tainted" constitutional/hereditary nature of their epilepsy. Collins found the reverse — IQ was higher in essential (111.2, n = 299) than traumatic (98.95, n = 101) cases. Traumatic was not used in the limited technical sense — but referred to any form of brain damage. As late as 1951, it is somewhat surprising that Collins said,

> "The most definite and probably most significant of our findings is the superiority of the constitutional group over the subjects whose disorder is due to brain damage" ([23], p. 398)

She went on to state,

> "…mental defect or low intelligence is not necessarily a concomitant or a resultant of epilepsy. The disorder may occur in individuals of all degrees of intelligence." (p. 398)

> "Environmental causes, particularly those that operate to prevent normal educational opportunities and normal social contacts, may be a potent factor in causing the apparent deterioration, formerly considered inevitable." (p. 398)

5.2. An important critical review of epilepsy and intelligence

The nextmajor review of this literature appeared in 1952 by Angela Folsom [64], and it was interesting inmany respects. In her review Folsom addressed key issues related to the notion of intellectual "deterioration" in epilepsy, selection bias, and a view of the most important core findings to date.

Regarding the general relationship between epilepsy and deterioration Folsom said,

> "Whatever its cause, deterioration with seizures was supposed to constitute epilepsy as a disease syndrome. Repeated findings of low IQ in institutional groups similar to those studies by clinicians only appeared to confirm the clinical findings of deterioration." (p. 15)

But Folsom took a direct critical look at the use of this term:

> "Deterioration, as clinically used, is essentially a descriptive term for severe cognitive deficit. Whatever else it may include, it implies a progressive falling off or loss in intellectual abilities. …What is important here is how this term has been used in epilepsy…. "Cognitive deficit" may be used in one of two ways, implying a level of function below the population average, or referring to an actual or implied drop from an earlier level of functioning. A low IQ carries the first meaning, but it can never in itself be a measure of the second." (p. 16)

> "The most direct measurement of loss of mental efficiency is by means of serial retesting on the same subjects. The results of serial retesting in epilepsy by no means indicate the progressive decrease in IQ that is consistent with the idea of deterioration." (p. 16)

Folsom was another person very critical of the degree of research emanating from select populations (colonies, institutions) and how that selection likely characterized the view of epilepsy.

> "With increasingly intensive study of non-institutionalized groups, it has become clear that the IQ level of epileptics varies over the same range as that of the non-epileptic population, and that whatever deficit may be characteristic of the epileptic, it is not a low IQ". (p. 15)

> "It is by no means improbable that, had representative samples of epileptics been available for intensive study at the start of psychometric evaluation of cognitive deficit, many of the psychological studies reported in the literature would not have been carried out." (p. 15)

Like Binet, Goddard, Collins, Lennox, and others, she was critical of "clinically" derived [nonpsychometric] pronouncements of cognitive

function given the subjective and bias-prone nature of such expert opinions.

When sorting through the potential causes of depressed intelligence, Folsom pointed to the relevance of etiology and credited Fox [4] as suggesting that etiology be considered as a grouping factor for epilepsy as persons with epilepsy may differ in degree or kind of psychological deficit as a function of this factor. She pointed out that it was not until 1942 that Arieff and Yacorzynski [19,20] revealed that "…brain damage, rather than seizures, is a significant factor in psychological deficit in epileptics" (p. 16), noting that these findings were confirmed by others [23,22,27]. That said, she understood that as knowledge developed the proportion of persons in the idiopathic category would decrease while the symptomatic proportion would increase.

Another variable of interest to Folsom was age of seizure onset and associated developmental issues:

> "These findings suggest that when seizures occur after intellectual growth has been completed, intelligence level is not likely to be affected. Early onset may interfere with intellectual growth in at least three ways: by interfering with normal schooling, seizures in childhood may prevent the acquisition of skills tapped by intelligence tests, particularly by the Stanford–Binet: secondly, grand mal seizures may produce permanent changes in the immature brain, whereas they may have no appreciable effect on the physiology or structure of the fully developed brain; or, early brain damage, of which seizures are one of the symptoms, may result in early mental defect."(p.18)

Finally, Folsom was one of the few to point explicitly to the potential detrimental effects of institutionalization on mental status — certainly pertinent to the topic of cognitive course. She also mentioned verbal versus performance IQ (PIQ) differences as well as specific deficits in epilepsy (memory, verbal).

In addition to the major papers by Collins and Folsom, this decade saw the development and/or extension of several important trends using intelligence tests that addressed and advanced topics of interest.

5.3. Epilepsy surgery

In this decade, began the systematic use of tests of intelligence and other cognitive abilities in the evaluation of epilepsy surgery candidates. Hebb [96–98] published early interesting case reports examining patients who underwent right temporal or left or right frontal resections for treatment of epilepsy. He noted minimal intellectual anomalies or changes in intelligence, but at times with detectable weaknesses in more specific cognitive abilities — raising concern about the sensitivity of intelligence tests. Systematic group studies began appearing in the 1950s, emanating from London [30,34,39], Montreal [37], and Chicago [36], involving patients operated upon by Falconer, Penfield, and Bailey. In these studies, Meyer and colleagues published systematic short- and longer-term follow-up assessments of surgical candidates and, in addition to IQ, pioneered the use of tests of paired-associate learning and recall; IQ for nondominant cases was largely unaffected while dominant resections were associated with interval decline in verbal IQ but more striking verbal learning and memory change, which persisted over time. Ward Halstead [36] administered a less commonly used test of intellectual ability (Henmon Nelson Test) as well as his measures of "biological intelligence" [99], which revealed a minimal impact of conservative temporal lobe resection on pre- to postoperative cognition. Milner [37] demonstrated the nature of pre to acute postoperative change on measures of memory with limited change in intellectual status and, consistent with Hebb's early concerns, was the largely unaffected intelligence performance in the setting of profound memory loss such as was observed in patient HM [100]. These early studies set the stage for an extensive literature to follow (e.g., [101,102]).

5.4. Comparison of epilepsy syndromes

As understanding of epilepsy developed, reports began to compare intellectual as well as other cognitive differences among patients with discrete epilepsy syndromes with a particular focus on patients with temporal lobe epilepsy (TLE)/psychomotor seizures and centrencephalic epilepsy [32]. The surgical investigations of course focused primarily on TLE as well [101,102].

5.5. EEG and cognition

An increasing niche of papers examined the relation of EEG abnormalities to intelligence test performance [26,31,103], research that had been undertaken earlier [23] and continued forward [41,48]. These reports using metrics of intelligence followed the antecedent discovery of affected cognition in association with subclinical and clinical EEG abnormalities [cf. 104 for review]. A clever and clinically notable finding was demonstrated by Hovey and Kooi [31] where the relationship between so-called "nonanswer responses," or NRs, was hypothesized to reflect a momentary deviation from an established goal idea during test performance (e.g., during the Comprehension subtest). Nonanswer responses were considered atypical in that a patient might have been providing correct answers but then suddenly made a careless error or appeared as if not engaged, subsequently resuming previously appropriate performance. Using subtests of the Wechsler–Bellevue, a higher proportion of epilepsy patients demonstrated NRs (44%) compared to brain impaired (17%) and psychiatric groups (9%). They hypothesized that this variability reflected transient subtle performance changes attributable to abnormal EEG discharges, a hypothesis that they tested using simultaneous EEG during cognitive testing [103]. Electroencephalography burst activity was noted during some part of the question and answer period in two-thirds of the NRs, which were the most common in the group with epilepsy. While they pointed out that the EEG activity would be considered "subclinical," it was associated with what they called "intellectual automatisms".

6. 1960–1972: concluding the early era

In this interval, 27 papers appeared, 4 from institutions and 23 from noninstitutional sites.

6.1. Epilepsy and related disorders

In a 1960 review by Lennox in his text *Epilepsy and Related Disorders* [105], the topic of estimated (by clinicians) mental status was discussed followed by review of objective assessment of intelligence. In the chapter entitled *"The Mind and Personality of the Epileptic"*, Lennox' longstanding interest in mental status was evident. He again was critical of the antecedent overreliance on institutionalized patients and their inherent biases, and restated what he considered to be the "*five foes of mental competence*" in epilepsy, those being genetic influence, organic abnormality of the brain acquired before the onset of seizures, the epilepsy itself, sociopsychologic isolation and repression, and overdosage with sedative antiseizure medications.

Several previously noted themes were extended and developed further in this time frame with the addition of new ideas.

6.2. The intersection of epilepsy syndrome and cognitive deficit

Allan Mirsky and colleagues undertook a series of studies comparing patients with focal (predominantly but not exclusively TLE) and nonfocal and especially centrencephalic epilepsies, with particular interest in group differences on measures of intelligence, memory, and attention [40,47,56]. These studies were performed with both adults and children with epilepsy as well as with a subset of anterior temporal lobectomy (ATL) cases. Children showed no difference between Verbal,

Performance or Full scale IQ scores as a function of epilepsy type; however, selective attention problems were observed in centrencephalic epilepsy and memory difficulties in TLE [56] — similar to their reported findings in adults. This work again suggested a limitation of intelligence testing compared to specific cognitive abilities hypothesized to be related to the basic pathophysiology of the disease.

6.3. Epilepsy surgery

Interest in surgical outcomes continued both within the same clinical centers mentioned previously as well as at new centers. In regard to the former, Blakemore and Falconer [54] continued to follow the London series out to 10+ years postoperatively, but with markedly declining subject numbers as the length of follow-up progressed (e.g., n = 86 preoperatively 2 and 12 months postoperatively, declining to n = 15 at 8–9 years postop and n = 11 at 10+ years postop). They reported that the decline in verbal IQ following dominant ATL seen at two months postop gradually recovered by one year and was stable out to 10+ years, with little change in PIQ following nondominant resection. Also examined were the changes in verbal learning ability originally reported by Meyer and colleagues — these showing a much slower and gradual pattern of recovery. Blakemore et al. [53] also investigated the relation of seizure frequency and underlying neuropathology to preoperative intellectual capacity where the findings were characterized as "generally inconclusive". In America, Meir and French [52] examined intellectual function before and approximately 1- and 3-year intervals after epilepsy surgery. No significant intellectual declines were seen following left ATL but a late decline in PIQ in right TLE (Picture Completion and Object Assembly) was reported.

6.4. Etiology

The decades long recurring theme of the relation between etiology and intellectual status was revisited in increasingly sophisticated investigations, extending the analyses to include not only intelligence but adaptive abilities reflected in Halstead's measures [99]. Comparing controls to patients with verified brain damage with epilepsy, brain damage without epilepsy, and epilepsy of unknown etiology, the less adversely affected performance of the latter group across measures of intellectual ability and Halstead's measures was reported, while the more abnormal performance of the brain damaged groups, regardless of the presence of epilepsy, agreed with prior authors that underlying etiology was a major factor compared to idiopathic epilepsy [49]. These results were extended by Matthews and Klove [55] by breaking the epilepsy groups into psychomotor and major motor and mixed seizure type groups into known and unknown etiology groups. They noted an orderly progression of impairment with the most intact but still depressed compared to control performance in the patients with psychomotor epilepsy of unknown and then unknown etiology.

6.5. Epilepsy IQ in population-based research

Evident has been a general lack of population-based knowledge of intellectual ability. While cited in the epilepsy literature primarily for the results of behavioral assessments, Rutter et al.'s [60] epidemiological Isle of Wight study also included a brief measure of intelligence (4 subtest Wechsler Intelligence Scale for Children) where intelligence was found to be average (106) in the 64 children with uncomplicated epilepsy, and lower in children with other neurological conditions (cerebral palsy = 77, other = 78). More population-based studies were to follow in the modern era.

6.6. Psychopathology

There is current interest in the impact of depression and/or anxiety on cognition in epilepsy. Anticipating this interest, Stevens et al. [61] examined the relationship of psychopathology to intellectual status as well as a variety of neuropsychological tests among patients with temporal and centrencephalic epilepsy. Stevens et al. were unable to find an adverse impact of increasing (abnormal) Minnesota Multiphasic Personality Inventory (MMPI) scale scores on the Wechsler Adult Intelligence Scale (WAIS), save a positive association between increasing hypomania scale scores and increasing Performance and Full Scale IQ scores.

6.7. Psychosocial problems

Under- and unemployment are known problems for those affected by epilepsy. Examining the neurological and psychosocial factors related to employment status in epilepsy, Dennerll et al. [50] examined measures of intelligence, cognition, and personality and were able to demonstrate reliable differences between the groups — an area of research that developed further with the establishment of comprehensive epilepsy centers in the US, but the Epilepsy Center of Michigan headed by the epileptologist Ernst Rodin was an early leader in this area of research [106].

6.8. Idiopathic epilepsy and its familial impact

As noted earlier, idiopathic epilepsy had a curious path over the decades. Originally posited to be at heightened risk of deterioration because of its "constitutional" (or presumed "genetic") nature with hypothesized propensity to deteriorate intellectually, idiopathic epilepsy was ultimately found to be comparable to controls (e.g., [26]) or among the least impaired etiologic groups (e.g., [55]), but interest in their status persisted, including in their relatives. Needham et al. [57], interested in familial idiopathic epilepsies (e.g., petit mal), studied affected patients (n = 73), relatives concordant for EEG abnormality but discordant for seizures (n = 38), and relatives discordant for EEG abnormality and seizures. Intellectual ability was examined across these groups as well as between patients and their own family members administered the identical intelligence test. Affected patients scored lower than the other groups (and specifically 70% of patients were below the family mean) while those with EEG abnormality but no seizures showed test scores comparable to unaffected family members.

6.9. Test investigations

Interest in the meaning of subtest scatter, profile patterns, and IQ splits (Verbal vs Performance) associated with neurological markers of disease (e.g., laterality of "brain damage", EEG patterns) continued, even in institutional samples [11,13]. Interesting was serial administration of the Wechsler–Bellevue and WAIS to a cohort of institutionalized patients, which questioned the appropriateness of combining data from the two tests [12] which had been the practice in some studies. Comparison of younger and older institutionalized patients with chronic epilepsy on the Wechsler–Bellevue and Halstead tests revealed significantly poorer performance on age-adjusted scores in the older patients [14] — an interesting way to examine chronicity effects, which on its face appeared consistent with a hypothesis expressed by Lennox [66] ("The mental power of a healthy adult declines with increasing age. We might anticipate a hastening of the process for epileptics whose seizures continue".)

7. Closing out the early era

The 60-year interval from the time of Wallin's [3] initial contribution to the review by Tarter [2] saw examination of the general intellectual status of children and adults with epilepsy alone as well as compared to healthy controls, relative intellectual performance associated with various seizure types, the initiation of comparative studies of epilepsy syndromes, and the relationship of intellectual ability to characteristics

of the disease (age of onset, duration) and markers of its severity (seizure frequency, severity, time without seizures, episodes of status epilepticus). Also examined were the relationship of intelligence to various EEG patterns and abnormalities, and the relative standing of epilepsy compared to other patient groups (brain damage, other medical disorders). Intellectual ability was examined in relation to the outcomes of medical treatments including medications and surgery, the status of persons with epilepsy relative to siblings and other family members, as well as intelligence in twin pairs, work that was far antecedent to the current renewed interest in the cognitive and behavioral status of siblings and other family members — although much of the earlier work was likely undertaken for reasons related to eugenics. The limitations of reliance solely on intelligence measures to characterize cognition became evident in studies of epilepsy surgery and syndrome comparisons. From the onset of this literature other cognitive measures were included with the metrics of intelligence, although this varied across studies. The ingrained early clinical view that epilepsy was a deteriorating disorder captured interest from the beginning with a substantial number of prospective studies of intelligence that have been reviewed elsewhere and continue to this day.

From a psychometric perspective, of interest was the development of short forms for more efficient assessment, inspection of performance scatter to identify particular cognitive strengths and weaknesses inherent in the intellectual abilities of persons with epilepsy, early use of "hold" measures such as a vocabulary test to provide an estimated premorbid baseline with which to quantify estimated deterioration, characterization of the underlying factor structure of intelligence tests and its relation to clinical epilepsy variables, the potential influences of psychopathology on intellectual performance, and, in the early studies, concern regarding the impact of limited or disrupted education and institutionalization on intelligence. As new tests of intellectual ability became available, they were adopted and at times contrasted to earlier versions. Similarly, as new classifications of seizures and syndromes were developed, research interests followed apace.

Students of the neuropsychology of epilepsy could likely apply a similar summary to a substantial proportion of contemporary research, not only in regard to intellectual ability but also in relation to specific cognitive domains. Such constancy over time may suggest the presence of a respected tradition-bound approach to understanding cognition in epilepsy. Another view may be that this constancy reflects a relatively inflexible tradition that finds it hard to accept repeated answers to the same questions posed over decades.

In the follow-up to this paper, greater attention will be paid to developments in general research in intelligence and the degree to which epilepsy has benefitted, and could benefit further, from those advances and related research trends.

Declaration of competing interest

The author has no conflicts of interest.

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